THE PALGRAVE HANDBOOK OF BIOLOGY AND SOCIETY

Edited by Maurizio Meloni, John Cromby,
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The Palgrave Handbook of Biology and Society

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Introducing the New Biosocial Landscape

Maurizio Meloni, John Cromby, Des Fitzgerald, and Stephanie Lloyd

For many decades, the study of society and the study of biology have been estranged from one another. There are complex reasons for this estrangement. Those reasons are rooted partly in the ways that, for a long time, biologists configured the relationship between their epistemic objects (particularly genes) and those objects' environmental influences; they are also partly rooted in the way that social scientists insisted, for an equally long period, on a strict division of labour between the sciences of society and the sciences of life. Yet many social scientists have now shown that a neat demarcation between the social and the biological has been largely illusory given the intense proliferation of objects, practices, and cultures that have persisted along a supposedly rigid biology/society border (Haraway 1991; Kroenfeldner 2009; Meloni 2016, reprinted here as Chap. 3). Nevertheless, the distinction between the biological and the social has become part of our everyday conceptual fabric—an inescapable metaphysics to which, to various degrees, all of us have more or less succumbed.

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When considered from an historical perspective, the estrangement between knowledge of biological life and knowledge of social processes has arguably been a necessary step. Richard Lewontin famously pointed out that Darwin had to propose an impoverished model of the relationship between organism and environment in order to overcome 'an obscurantist holism that merged the organic and the inorganic into an unanalyzable whole' (2000, 47). However, as Lewontin further noticed, often the epistemological presumptions 'that are necessary for progress at one stage in history become bars to further progress at another' (ibid.). The model suggested by Darwin is in fact nowadays enriched by models (for instance, niche-construction, Odling-Smee et al. 2003) that point to a more complex relationship between organism and *milieu*.

A similar development has occurred in the relationship between knowledge of life processes and knowledge of society, where an initial estrangement may have been, *inter alia*, a productive process. If we compare the holism of nineteenth-century sociologists like Herbert Spencer, for whom there is no social advancement without corresponding biological growth, to the rejection of biological explanations proposed by turn-of-the-century social scientists such as Émile Durkheim or Alfred Kroeber, it is arguable that this rejection was an important step on the way to a more potent understanding of social life. Today, however, that well-known self-sufficient entity, the social fact, has become an obstacle for a broader comprehension of the world in which we live, in all its inextricably biosocial or biocultural dimensions. This *Handbook* is an attempt to wedge us across that obstacle. It is motivated by an intuition (and it is hardly alone in this) that the time has come to reposition this historical legacy and to move beyond the acrimonious controversies that have characterized twentieth-century thought as it traversed the biology/society border.

This *Handbook* provides the first comprehensive overview of the extent to which, and how quickly, we are moving beyond the charged debates that characterized much 'biosocial' thought in the twentieth century. Bringing together a compelling array of truly interdisciplinary contributions, the *Handbook* shows how nuanced attention to *both* the biological sciences *and* the social sciences opens up novel perspectives on some of the most significant sociological, anthropological, philosophical, and biological questions of our era. Our central assertion is that the life sciences, broadly conceived, are currently moving toward a more social view of biological processes, just as the social sciences are beginning to reincorporate notions of the biological body into their investigations.

We are perfectly aware that others have mapped this terrain before us (Fox Keller 2011; Lock 2015; Rose 1997, 2013). Nonetheless, there is work to be done to bring together the burgeoning but too often fragmented work that has powerfully emerged within that terrain. That work, in turn, has rested on some striking developments across a range of intellectual domains. We think here of work in social neuroscience, which shows not simply that the capacity for interaction is instantiated in the brain, but that brain structure and function are themselves part-produced through particular sets of environmental and social relations (see e.g., Cacioppo 2002); we think also of the discovery of adult neurogenesis in humans, the realization that parts of the adult brain continue to produce new cells through the lifetime, that these cells may have functional significance, and that they may be affected by developmental and environmental impacts (see e.g., Gould et al. 1999); and we think of the renewed emphasis on neuroplasticity, which suggests that the brain continues to change and develop as a person ages and lives (see e.g., Draganski et al. 2004). Similar developments occur in what, in molecular biology, is called the postgenomic moment—the increasing awareness of a profound malleability of genomic functioning and a recognition of its dependence on time and place, biography and milieu, social institutions and experiences, with profound implications for the notion of biological heredity that we have received from the century of the gene (Lappé and Landecker 2015; Stallins et al. 2016; Meloni 2016). Today, we know that DNA expression is influenced by factors including toxins, work stress, nutrition, socio-economic status, early childhood care, perhaps even the lifestyle of one's mother, father, or grandparents—all factors that at least partially exceed the traditionally biological. This new understanding, with DNA always ready to respond to environmental cues, is, somewhat paradoxically, a product of scientific advances that were expected to deepen and confirm pre-existing theories of the fixed gene.

These developments have come at a propitious time for the social sciences, and especially for social theory. As Nikolas Rose points out, 'over the last decade a number of social theorists and feminist philosophers have come to realize that it is not reactionary to recognize the reality of our fleshly nature, and to examine the possibilities and constraints that flow from it' (2007, 4). We have thus seen, in feminist theory especially, in related trends such as the 'affective turn' and, more recently, in a body of work going under the sign of a 'new materialism' (Coole and Frost 2010), a growing and often contested assemblage of turns to materialities, affects, ontologies, and bodies—all of which have contributed to a corpus of theoretical work that no longer accounts for itself in terms of its distance from biology—and, indeed, sometimes moves

in quite the opposite direction (Wilson 2004, 2015; see Pedwell and Whitehead 2012, for an important overview of some of these developments). Scholars such as Donna Haraway (1997) and Karen Barad (2007), for example, have edged social scientists away from taking the natural sciences in general, and the biological sciences in particular, as mere objects or resources—as only practises that might be looked *at*, rather than *with*. At the risk of flattening out important distinctions between diverse perspectives, these trends undo binary oppositions between biological influences and social forces, and so have begun to legitimate social research that unpicks the separation between natural and social science.

Given the forms of erasure often built into claims to novelty (see Ahmed 2008), we are reluctant to hail only the newness of such developments. Nevertheless, it does seem, today, that there are many opportunities to do deeply consequential sociological and anthropological work with, and through, bioscientific knowledge and practice. And perhaps this should not be surprising. No matter the hyperspecialization of contemporary scholarship, with its sharp policing of disciplinary boundaries (an actuality partly concealed by rhetorics of 'interdisciplinarity'), human life remains stubbornly biosocial through and through. Whether it is the disproportionate distribution of certain diseases in lower socio-economic groups (Marmot 2010), or the visceral reactions that hate speech may provoke (Zembylas 2007); whether it is the way in which socio-economic and scientific activity modifies bacterial life (Landecker 2016) or gets physically recorded into the outer environment, or in genomic expression; whether it is the way in which normative views of gender, class, and race imbue the materiality of scientific findings with meaning and thereby transform them (Haraway 1989); or the way in which political forms and institutions affect how bacterial diseases take form and circulate (Nading 2012), few central objects of either the social *or* biological sciences today can be understood other than with complex biosocial, biocultural, or biohistorical rubrics.

The aims of this *Handbook* are twofold. First, to demarcate an epistemic space in the relationship between the life sciences and the social sciences. This space stands orthogonally to previous sociobiology-biosociety debates, especially those that took shape in the last quarter of the last century. Thus, we were exhorted either to pit the biological *against and before* the social (sociobiology, evolutionary psychology), or to promote the social *against and above* the biological. This *Handbook* aims to undermine this symmetrical hostility. In so doing, we don't want to oversimplify the complex and disparate (if interdependent) matrices of method, theory, and knowledge at stake on both sides of these divides—nor indeed to gloss the dense networks of power and status

in which they are enmeshed. While perhaps these contributions are only first steps, the biosocial that emerges from this assemblage of 38 chapters, at least, no longer depends upon an original separation of biological and social forces, organism and environment, agent and milieu, that have then to be awkwardly recomposed in a secondary, additional moment (see Fitzgerald and Callard 2015, reprinted here as Chap. 19).

This has clear implications for knowledge production. In part, this is because the entanglements our contributors identify challenge the neat separation between content and context that favours 'entrenched ways of conceiving causation and agency' (Alder 2013, 97) wherein humans are conceived largely independently of their circumstances. But it is also because these entanglements go well beyond now-established social constructionist claims that biological knowledge is shaped by meaning, power, and norms. Rather, biological matter itself, be it genomes, brains, diseases, or viruses, is simultaneously irremediably social, not only in its form but also in its content. And vice versa: the very fabric of sociality is always enabled, mediated, and modulated by fleshy substrates—be they genetic or epigenetic, nutritional, metabolic, hormonal, behavioral, or toxicological. At all levels, the biological and the social are *in* one another.

Our second aim is to avoid dissipating this knowledge through the too-many rivers and trickles of the contemporary academy. The very gesture of bringing together research that is otherwise largely fragmented and isolated is part of a performative gesture of creating new spaces. In so doing, this *Handbook* offers a relatively stable research platform, and functions as a teaching tool to help foster a new generation of scholars who are more capable of thinking in complex, critical ways: about the nuances of our irreducibly hybrid, entangled, biosocial world, and about the benefits and costs of the prevailing metaphysics that drives a wedge between biology and society, and which still primordially structures much academic work.

Overview of the Chapters

Handbooks, we suggest, are epistemic things of a sort—they are contingent, hard-to-grasp, generative objects; they are set out into the world, and worked upon; they unfold, under examination, in multiple ways; and if they never achieve their final definition, still it is only later we come to realize that the always-in-process work of *defining*, and of *being defined*, is where the epistemic and ontological magic happens (Rheinberger 2010). Perhaps it would be better to say that handbooking is an epistemic practice (Knorr Cetina

2001)—which is to say that it is a dynamic activity of nudging, moving, and sometimes disrupting the objects and practices of knowledge that it comes into contact with. This volume, perhaps more than most, is generated, assembled, worked on, and distributed as a dynamic intervention into an emerging space: the six sections are conceived precisely in the spirit of intervening in key hotspots of the biology/society debate. Two additional notes before we describe the chapters: (1) While this volume is for (indeed, founds its contribution on) a certain kind of comprehensiveness, such a goal always, and of necessity, remains in the distance; we would not have it otherwise, and do not wish to exert any totalizing force here. Nonetheless, more prosaically, there are gaps in what follows, some of which we are aware of (although we will not compound the error by naming them!); other gaps will have to wait for our readers to gently point them out. Such gaps can be variously attributed to the exigencies of time and space, bad fortune, or the blindnesses and prejudices of the editors. Without wishing to disavow responsibility for our own omissions, we truly hope to see, in future years, other volumes, from other authors and editors, making good where we have erred. (2) There are authors who write both from 'inside' and 'outside' different practices in what follows. Which is to say: there are those describing some elements (either in hope or in concern) of their own practice here, and there are those (both encouragingly and critically) accounting for the practice of some *other*. (And there are more, probably the majority, awkwardly straddling such logics of inside and outside.) In any event, we have chosen not to mark these distinctions; where disciplinary and other divisions are bureaucratically real enough already, we have no desire to make them more so. If this strategy will occasionally confuse the reader, we are nonetheless convinced that the convivial intentions of the volume are not well served by marking, in advance, who wishes to be in and who out.

A final note: in this time of ascendant protectionist nationalisms and racisms, we also wish to highlight the *Handbook*'s pluralism, not only of approaches and disciplines but also of places. With nearly 50 contributors representing a wide diversity of cultures and geographical regions, from Israel to Brazil, from Australia to Europe, from South Africa to North America, the *Handbook* is an invitation to think biology and society always in the plural, as *biologies* and *societies* (and perhaps this should have been a more appropriate title for this endeavour). After all, among the strongest legacies of the social studies of science is the reminder that 'all scientific knowledge-claims have a provenance: they originate at some place, and come from there' (Gieryn 2002). This *Handbook*, albeit in its own minor way, is deeply committed to caring for scenes that foster a plurality of ways of being-there, of coming-from-there, of going-there.

Outline of the *Handbook*

We start with a historical section ('History of the Biology/Society Relationship'), since we believe that history (if not historicism) is an obligatory passage point for anyone who wants to take seriously the notion that the estrangement between the social and the biological is less a fact of nature, and more a sedimented effect of long-term strategies and decisions involving various disciplinary bodies, authors, institutional settings, and other agencies. In the two first chapters, Snait Gissis and Maurizio Meloni cover a similar historical period, examining the transactions between biology and sociology in the second half of the nineteenth century, including the impact of those transactions in terms of debate on fixedness and plasticity, individuals and social groups, heredity and wider notions of inheritance and tradition. These two chapters, while sharing an historical period, focus on different reverberations of biological knowledge on the making of a modern social science. Gissis looks at the significance of a Lamarckian framework in the work of Spencer and Durkheim, whereas Meloni points to the subtle influence of the German founder of the modern hard view of heredity—August Weismann—on Durkheim's writings in the 1890s as a foundational step toward erecting a neat separation between the social and the biological.

In the next chapter, Chris Renwick focuses on one of the key terms at the crossroads of the social and biological—population. The chapter explains how the emergence of population thinking in biology and social science in late-nineteenth-century and early-twentieth-century Britain were related, with research at the intersection of the two fields helping to construct shared ideas and practices. As the chapter shows, eugenics played a major part in this story 'featuring a space that some researchers considered to be a genuine third sphere between biological and social science'. In the fifth chapter, Antonine Nicoglou focuses on another of the key concepts in twenty-first-century biology—plasticity. She provides an historical account of the role of this concept as a key means of navigating the space between nature and nurture. Nicoglou argues that a comprehensive understanding of the concept of plasticity will assist us in divesting ourselves of this dichotomous opposition.

In Chap. 6, Jonathan Marks traces the transformation of the field of biological anthropology from a science of race to a science of human spatiotemporal variation. He focuses on two major misconceptions in anthropology, each with a long historical legacy: that the human species is composed of zoologically meaningful taxonomic entities, and that human groups think differently in ways that are significantly innate. As Marks writes: 'both of these propositions have been falsified about as thoroughly as young-earth

creationism, but their political value is sufficient to continually resurrect them'. Marks then investigates the complex moral and political dimension associated with these epistemic questions and the inescapable moral side of debates on race and racism. In the next chapter, Will Viney focuses these debates into one epistemic object: the culture of twinning as it emerged between history, biology, and literature. As a sort of 'natural experiment', twin research has been used to think through the divisions between biology and environment, and its history has lessons for our understanding of how human groups interact with scientific endeavours. Viney outlines a history of the conceptualization of twins in an account that is concerned less with the validity of findings generated by twin studies, and more with the ways that this research exemplifies the interweaving of different assumptions (medical, sociological, psychological, ideological, methodological) vis-à-vis the possibility of neatly separating genes from environment. This, argues Viney, is what captures the imaginations of medical researchers and the general public alike in relating to twin studies.

Finally in this section, in Chap. 8, Tatjana Buklijas sketches one of the very first histories of the rise to public prominence of epigenetics, which is among the most rapidly expanding fields in the life sciences, and increasingly seen by many as a potential bridge between the social and the natural sciences. Buklijas looks at competing interpretations of epigenetics as paradigm-shifting, or as just a scientific-cultural trend reinforcing genetics, contrasting views that, she claims, 'go along with opposing historical narratives and understandings of future promise of epigenetics'.

The second section 'Genetics, Postgenomics, Epigenetics, and Society' focuses on some key changes in contemporary molecular biology that have shifted our view of the gene as an autonomous master of development to the 'reactive genome' of molecular epigenetics—now unfolding in specific social and historical *milieux* (Gilbert 2003; Griffiths and Stotz 2013; Keller 2011, 2014). In their chapter, Maurizio Meloni and Giuseppe Testa critically analyse the 'epigenetics revolution', with its claims to herald a new epoch both for gene-based epistemology and for the wider discourse on life that pervades knowledge-intensive societies of the 'molecular age'. Meloni and Testa scrutinize the fundamentals of this revolution, highlighting in particular how the very contours of what counts as 'epigenetic' are often blurred, something that crucially contributes to its success.

In the next chapter, Frances Champagne focuses on the potential of environmental epigenetics research for understanding risk of health and illness, as well as its role in documenting the effects of life experiences. As an epigenetics researcher, Champagne provides insight into how 'hard' scientists might be

both concerned and eager to see how environmental epigenetics research—including Champagne's own—will be translated into new understandings of animals (including humans) and their environments, and eventually into new clinical approaches and interventions. Amy Hinterberger, in her chapter on "Molecular Multicultures", examines what has happened to the politics of multiculturalism in light of the molecularization of biology. Hinterberger argues that a conceptual framework of 'molecular multicultures' may be helpful to highlight how the cultural politics of heredity in bioscience draws together the classificatory practices of the nation-state, the naming practices of identity-based social movements, and the segmenting techniques of genome science.

In her chapter on 'The First Thousand Days', Michelle Pentecost provides an introduction to a movement that is taking an increasingly important space within public health, namely, studies of the first thousand days of life. Pentecost documents this understanding of child development, from conception to two years of age, which suggests that experiences during this period of life set children on paths for the rest of their lives. Using a South African case study of the global 'first thousand days' initiative, Pentecost examines how the DOHaD (Developmental Origins of Health and Disease) paradigm and epigenetic knowledge, as 'biosocial' objects of enquiry, are embedded in global discourses that come to bear on the everyday.

In the next chapter, which takes an educational focus, Deborah Youdell proposes a biosocial understanding that conceives of learning as the folding together of multiple intra-acting forces and processes within which possibilities for social justice are mediated biologically, physiologically, and neurally, as well as affectively, intellectually, and interpersonally. To make this argument, the chapter foregrounds developments in epigenetics and the entanglement of the social and the biological, and Youdell makes a case for thinking about socially just education in a biosocial way. Through an engagement with research in education and the biosciences, she argues that biosocial education research can bring into view 'molecular, neuronal, metabolic, biochemical, social, cultural, affective, psychic, and relational processes operating across multiple scales and temporalities'. Any contemporary ambition for socially just education, Youdell claims, must now attend to this complexity and to its biosocial character.

Finally, in her chapter on the challenge of assembling biomedical big data, Sabina Leonelli examines the issues involved in disseminating, integrating, and analysing large datasets collected on human subjects and non-human experimental organisms, and within both clinical and research settings. Leonelli highlights some of the technical, ethical, and epistemic concerns

underlying current attempts to portray and use 'Big Data' as a revolutionary tool for producing biomedical knowledge and related interventions. When bringing together data collected on human subjects with data collected from other organisms, significant differences in the experimental cultures of biologists and clinicians emerge which, if left unnoticed, risk compromising the quality and validity of large-scale, cross-species data integration. Leonelli highlights the complex conjunctions of biological and clinical practice, model organisms and human subjects, and material and virtual sources of evidence, emphasizing the fragmented, localized, and inherently translational nature of biomedical research.

The third section, 'Neuroscience: Brain, Culture, and Social Relations', is devoted to neuroscience, including the intersections of the diverse practices that term now implies with/in psychiatry and psychology. If we were producing a handbook on relations between the biological and social sciences as little as 15 or 20 years ago, it is difficult to imagine there being much to be said about the neurosciences. Today, the situation is quite different: for many now working in the neurosciences, what makes this area so appealing is precisely the fact that so much of the social and cultural world in which our brains develop *cannot* be reduced to bare neurological material. Authors in this section explore that realization and seek new ways to develop it. But the section leads with two chapters that urge continuing caution about naïve celebration.

We begin with Jan Slaby and Suparna Choudhury's 'Proposal for a Critical Neuroscience'—one of a suite of papers published in the mid-2000s, in which these authors, with their colleagues and interlocutors, set out a compelling vision for how new relations between the neurosciences and critical social sciences might take shape. In this programmatic contribution, Slaby and Choudhury account for their own attempt to 'respond to the impressive and at times troublesome surge of the neurosciences, without either celebrating them uncritically or condemning them wholesale'. The chapter seeks to show what, precisely, an ethos of 'critique' can offer to the neurosciences and how it can help to open out the range of practices and intuitions through which neuroscientific facts are made.

In a complementary chapter, Fernando Vidal and Francisco Ortega zero in on the neuroscience of culture, where they argue that 'in spite of an emphasis on the two-way processes that turn brain into culture and culture into brain, a common feature of the neurodisciplines of culture is their belief in the ontological primacy of the brain'. Working through some of the key techniques and approaches through which neuroscientists have tried to get at culture, Ortega and Vidal show how the field relies on quite traditional neuroscientific

methods and tropes. The chapter pays special attention to the way that cultures of 'individualism' and 'collectivism' are conjured in this field, and how the image of culture that emerges around it, for all the methodological novelty, turns on a surprisingly conventional image of discrete and bounded 'cultural' entities.

The chapter that follows is in quite a different mode. Here, Christian von Scheve takes seriously the notion of a 'neurosociology', proposing that 'many neuroscience studies and paradigms as well as their hypotheses and results are directly adaptable to and relevant for the processes and mechanisms traditionally studied by sociologists'. To consider this potential, von Scheve focuses on the paradigmatic case of affective neuroscience, a field that concerns itself with the processing of emotions. Offering a thick account of how neuroscientific work might then help to hook emotional processes into social situations, von Scheve proposes that a neurobiological perspective on emotion could help sociologists to move away from accounts of instrumental reason when they consider moments of decision-making and thus help us to understand, in a much more fine-grained way, the deeply embodied nature of such social scenes.

The next chapter, by anthropologist Rebecca Seligman, joins that of von Scheve in her intuition that there is something important to be gained from running neurobiological and social scientific problems through one another. This time, the argument focuses on the relationship between physiological and cultural states, through a study of religious devotion in Brazil. Focusing on the phenomena of spirit possession in Brazilian Candomblé, Seligman uses ethnographic and psychophysiological interventions to explore this religious practice, and to show how religious states recruit particular forms of psychophysiological regulation. Drawing on the concept of bio-looping, Seligman's chapter 'draw[s] attention to the ways in which embodied processes, including biological ones, are implicated in the continuous and mutually reinforcing relationships among meaning, practice, and experience'. For Seligman, such an attention has the capacity to tell us something very new about the concept of embodiment—and allows us to get a grasp of moments in which psychological, cultural, and physical states seem strikingly inseparable from one another.

The next chapter, by Des Fitzgerald and Felicity Callard, tries to take a metaperspective on the space between neuroscience and social science. Fitzgerald and Callard argue that there is much scope, now, for reanimating collaborative relationships between the social sciences and neurosciences, but that this potential is squandered by arguments (both for and against such a development) that significantly misunderstand what is at stake. Setting themselves

against what they call 'the regime of the inter-', a space of thought that insists on understanding neuroscience and social science as very different kinds of thing, whether in service of 'integrating' them or keeping them apart, Fitzgerald and Callard instead call for thicker attention to, and situation of researchers in, *experiments*, as sites of novel exchange and practice.

The final two chapters in this section expand these debates through attention to two very specific sites. First, in his chapter on neuroscience and schizophrenia, John Cromby uses the development of the diagnostic category of schizophrenia to show, in its past, present, and future, how schizophrenia has been developed through symbiotic relationships to the brain and neuroscience. Beginning with the foundational work of Kraepelin and Bleuler, and tracing this work into contemporary neuroscience, Cromby shows how 'conceptualisations of mental health and illness, concepts and images of brains, their parts and their functions, practices of treatment and intervention, and the somewhat disparate interests of multiple professions … are continuously circulated and exchanged, and mutually, dynamically and contingently related'. Sketching out a range of possible futures for the scientific study of schizophrenia, the chapter shows, for example, a renewed interest in social and relational approaches; through this and related attentions, argues Cromby, committed and serious neuroscientific work need not be wedded to the traditional rubrics of biological psychiatry.

In the final chapter in this section, Stephanie Lloyd and Eugene Raikhel examine the emergence of a style of thought that connects work in environmental epigenetics to the 'suicidal brain'. Lloyd and Raikhel propose that epigenetics be analysed as a 'style of reasoning', a particular mode of biologically construing both the environment and time in a way that, for some, has 'led to a new vision of the relationship between society and biology, while for others they have bolstered long-held ideas about biosocial complexity'. They draw on epigenetic research on suicide as a way of showing how, in this space, social contexts can get molecularized, drawing connections, for example, between early social and environmental experience and suicide risk. The (often explicitly) political ramifications of such a thought-style become apparent in the case of aboriginal suicide in Canada, where a blanket insistence on 'early adversity' often occludes the complexities of structural violence, as well as 'highly specific social, political and economic contexts'. Perhaps returning us, then, to where we began this section, Lloyd and Raikhel conclude that mere 'engagement' cannot simply override the deep epistemological differences between social scientists and neuroscientists.

Section IV is devoted to *social epidemiology*, a discipline that began to emerge in the 1960s, and which has, since then, gained considerable stature

and reach generating evidence for, and interest in, the social causation of illness and health. Social epidemiology's concern with the societal determinants of patterns of disease points to a quintessentially biosocial dimension. Nevertheless, the mere existence of a subdiscipline called *social* epidemiology is already suggestive of the various epistemic tensions along the biology/society border. As Nancy Krieger (e.g. 2011) in particular has repeatedly highlighted, what is at stake in the separate constitution of social epidemiology is the tendency of epidemiology to fall prey to a taken-for-granted 'just biological' presumption, according to which attempts to identify social determinants of illness and health tend to be seen as somewhat additional, optional, adjunctive, or marginal. From different angles, the five contributions in this section all challenge this established way of thinking.

First, Michelle Kelly-Irving and Cyrille Delpierre consider embodiment in relation to social epidemiology, focusing on the incidence of cancer. Their chapter traces some of the intertwined conceptual and methodological issues with which coherent empirical research into embodiment and epidemiology must contend. A life course approach is suggested whereby DNA mutations in cancer are at least partially initiated by immune and inflammatory system processes, processes, that are in turn open to social influence. Finally, evidence is presented from a prospective study suggesting that, at least among women, an accumulation of 'ACEs'—adverse childhood experiences—is associated with a subsequently increased incidence of cancer.

In the next chapter, Silvia Stringhini and Paolo Vineis outline some of the evidence regarding the connections between socio-economic status (SES) and health, before presenting candidate processes, most notably epigenetic ones, that might mediate these connections. Stringhini and Vineis describe a conceptual framework within which epigenetic processes in relation to health and SES might be understood, summarize some of their own research exploring the connections between epigenetic changes and SES, and then draw out some policy implications of their studies (including those that flow from the potential reversibility of some epigenetic changes).

From a different angle, Jonathan Wells and Akanksha Marphatia consider how maternal capital could mediate the associations between health and social inequality. Drawing on evidence for both plasticity and critical periods in development (periods during which environmental influences might have more marked or enduring consequences), the concept of 'maternal capital' describes how offspring are differentially enabled to thrive during development by (largely unintentional) variations in the somatic or behavioral 'investments' of mothers. While maternal interventions designed to benefit offspring might seem to treat mothers as little more than passive vehicles, Wells and

Marphatia suggest that this problem might be avoided if the chosen interventions are ones that also benefit mothers themselves.

In Chap. 25, Mike Kelly and Rachel Kelly provide a narrative overview of the character of, and synergies between, the new 'omic' biological subfields. They suggest that these projects can be integrated with sociological accounts of the dynamism that characterizes structure–agency relationships, in order to more precisely answer questions about the relationships between disease and environmental stressors. Kelly and Kelly draw on Giddens's structuration theory to understand how the repetitive, recursive character of much human activity gets realized within socioculturally normative practices with both social and biological aspects. Hence, practices constituting activities such as eating, drinking, loving, working, and child-rearing have societal origins and, simultaneously, 'drive' the human interactome.

Finally, in the last chapter of this section, some of the intricate associations between socio-economic variables and health inequalities are explored empirically by Rasmus Hoffmann, Hannes Kröger, and Eduwin Pakpahan. Life expectancy differentials of 5–10 years between the most and the least wealthy (and differences in healthy life expectancy of up to 20 years) starkly illustrate the force of social influence, as do related differentials associated with gender and ethnicity. Nevertheless, as this analysis demonstrates, empirical studies that compare social causation models of these inequalities with social selection models (i.e. models presuming that health inequalities drive socioeconomic status) produce a more complex picture where different influences predominate at different stages of the life course.

In the fifth section of the book, 'Medicine and Society', attention turns to the institutions and people affected by, and shaping, emerging knowledge and practices in the postgenomic era, as life, risk, and vitality are measured and interpreted in new ways. Conceptually, these movements attempt to reach into and beyond individual bodies, producing data that aims to quantify individual profiles whilst also situating bodies in specific environments. These practices embed specific goals and values in emerging forms of surveillance in the ongoing reconception of human bodies and biosocial spaces. Assumptions are made about what forms of data can be compared, and what forms of data count—with 'the environment', interior or exterior to the body, often reduced to one or two key factors, commonly measured with brief questionnaires or checklists to be linked to biomarkers. This represents what social scientists have referred to as 'pragmatic' or 'methodological reductionism', conceptualizing environments as a set of molecular inputs. This logic requires the abstraction of inputs, with distinctions in content or derivation flattened and rendered incidental. Amongst other concerns, observers worry that the

reduction and flattening of environmental contexts to molecular mechanisms will make it more likely that potential interventions are solely conceived on this scale.

Opportunities for collaborations between bio-scientists and social scientists are opened by these conceptualizations of humans, environments, health, and disease, yet questions remain over how multiple forms of data might be brought into conversation with one another. Potential studies raise questions about how research might be carried out in such a way that it avoids beginning with 'the social', 'the psychological', and 'the biological' as distinct domains. Beyond largely rhetorical invocations of 'the' biopsychosocial model (which in fact was never developed coherently as such) lies a clear need instead to view these processes, and the data produced about them, as symmetrical, with no branch of evidence considered more 'real' or foundational than another.

In Chap. 27, Patrick Bieler and Jörg Niewöhner provide a portrait of the ways in which the relationships between the human material body and social practices are currently being explored. In their account, Bieler and Niewöhner argue that the epistemological space opened by these interests and molecular understandings of humans provides an opportunity for social scientists to engage with social differentiation as a complex biosocial phenomenon, rather than as measurable variables. They propose a study of the 'body-in-action' as a boundary object in emerging research in both biological and social sciences. This body-in-action 'implies that it must be ethnographically accounted for in its complex entanglements with the assembled environment instead of trying to measure clearly defined, decontextualized variables'. An understanding of context nevertheless remains a significant challenge in studies of individual biomedical and molecular profiles, and Nadine Levin explores this challenge in Chap. 28. Levin explicates some of the issues raised by making, and making sense of, 'big data' in biomedicine, as scientists attempt to construct molecularized, personalized accounts of situated risk. Proposing an 'anthropology of data', Levin aims to question the norms, politics, and values that get wrapped up in data.

In Chap. 29, Barbara Prainsack provides an historical overview of personalized medicine, tracing it from its original focus on matching drug therapies to patients' specific genetic profiles, to its current instantiation which is concerned more broadly with a consideration of patients' profiles—molecular and otherwise—in order to improve medical care and research. Within this historical shift, Prainsack focuses most particularly on the implications of one of the central goals of this research in its current form of 'precision medicine'—comprehensive individual data capture. This data capture seeks to

produce the most detailed profile possible of individuals' lives, bodies, and environments in order to reach 'personalization', a point that would putatively permit improved patient care as well as continue to inform biomedical researchers' future interventions. Yet, as has often been the case in the history of medical research, patients who contribute information, time, and self-monitoring ultimately have little influence on how their bodies and lives are represented and 'datafied' in this process.

In Chap. 30, Megan Warin and Aryn Martin explore the construction of the uterus as a social space in epigenetics research. Warin and Martin situate this process within the broader reconsideration of the environment within epigenetics. Through case studies of reproduction (fetal origins and microchimerism), they explore the rearticulation of environments, not only in terms of the limits of binaries (nature/nurture; self/other; time and space) but also in terms of postgenomic capacities to reduce the environment to individual risk in gendered and sexed bodies—rather than open research agendas to a consideration of the complexity of biosocial spaces. In Chap. 31, Ayo Wahlberg then considers the growth of interest in biomedical research in the study and management of morbid living. Through this research, 'quality of life' becomes the focus of data collection as disease-specific clinical trials are carried out, and patients and caregivers are taught to 'live with' sickness as optimally as possible. The result is a 'novel analytics' of what Wahlberg refers to as the 'vitality of disease'.

In Chap. 32, Elizabeth F. S. Roberts and Camilo Sanz provide a methodological intervention, describing their efforts to develop a new research platform that combines ethnographic and biological data—'bioethnography'. Bioethnography is a response to the criticisms of big data, in which the potential wealth of 'comprehensive profiles' is often lost in reductive forms of data collection, management, and analysis. By contrast, bioethnography aims to 'arrive at a better understanding of the larger histories and life circumstances that shape health and inequality'. These authors' approach emerged from collaborations with environmental health scientists involved in a longitudinal pregnancy birth cohort and chemical exposure study in Mexico City. Now in a phase of analysis, Roberts and Sanz reflect on the process that entails the 'epistemic, temporal, and logistical coordination of disparate, and differently positioned intellectual research ecologies', in order to provide a preliminary guide for social scientists engaged in biosocial collaborations.

The *Handbook* ends with a section on 'Contested Sites/Future Perspectives', of which there are many that the emerging biosocial world is likely to provoke or is already provoking. Of the many dangers to be circumvented in a volume like this, perhaps most urgent is to avoid covering over the many pressing

political, conceptual, methodological, and evidential objections (each intertwined with, and sometimes masquerading as, the other) that have dogged the history of 'biosocial' approaches. We think here not only of critiques launched at such crass endeavours as sociobiology and evolutionary psychology, but also more recent critical approaches taken to social neuroscience, socio-genomics, epigenetics, and so on. We take very seriously the responsibility of a handbook of biology and society to not only 'memorialize' these contests, but to contribute in whatever minor way it can to keeping them in view—to insist, indeed, that it will have no truck with any 'biosocial' space wherein this history of contestation around biosocial approaches is rendered invisible. While the deep historical and political debates that have structured the division between the biological and the social are present, in some way, in all of the contributions in this section, we nonetheless here explicitly foreground discussions of how the biosocial intercedes—and not always in welcome or happy ways—at the intersections of race, gender, class, science, and justice (Reardon 2013).

We begin with a chapter from Catherine Bliss that, drawing on interviews with leading figures in genomic science, foregrounds discussions on racial politics in the postgenomic age. The chapter shows how 'struggles over the characterization of race, and the amelioration of racial inequality, have come to be drivers of large-scale global research programs'. Bliss focuses in particular on the relationship between 'science activism' and 'mass activism', to highlight how some genomic scientists actually take on the mantle of racial activism. While this mobilization has similarities to the kinds of mobilization we are more familiar with in the political mainstream, it ultimately fails to support a politics of mass movement around racial inequality. In the postgenomic age, Bliss argues, the political mobilizations of scientists in fact results in a reinforcement of a deterministic understanding of race.

In the following chapter, Kenney and Müller turn their attention to environmental epigenetics research on maternal care, arguing that while, on the one hand, this research is exciting and offers possible opportunities for collaboration between molecular biology and the social sciences, it is also necessary to consider its political dimensions. Through their research, they underscore how common-sense assumptions about sex, gender, sexuality, and class are present in the design, interpretation, and dissemination of experiments on the epigenetic effects of maternal care. As these experiments come to support claims about human motherhood through a dense speculative cross-traffic between epigenetic studies in rodents and psychological and epidemiological studies in humans, Kenney and Müller argue that current research trends work to illustrate, rather than interrogate, existing stereotypes about maternal agency and responsibility. Through their work they aim to

offer a cautionary perspective regarding the potentials and challenges for new forms of collaborative biosocial knowledge-practices emerging out of environmental epigenetics.

The next chapter in this section, by Jessica Bardill, is about the reconfiguration of ancestors and identities in biosocial times. Bardill's chapter focuses on genetic ancestry testing and American Indian peoples—showing how accounts of Native American ancestry and identity that variously go under the sign of 'scientific' or 'indigenous' depart from one another in politically potent ways. To gain analytic purchase on these tensions, Bardill draws on the concept of 'story', in order to 'to promote alternative understandings and ultimately another narrative by which to move thought forward in a variety of spaces'. Drawing on specific examples—such as the relationship between the Uros people of Bolivia and the National Genographic Project—Bardill shows how indigenous concepts and resources have been drawn on by genomic scientists, while potentially disrupting embodied notions of inheritance and identity. Drawing on the work of Gerard Vizenor, however, Bardill concludes by offering a new vision of indigenous-led genomic science—a way of producing knowledge in which 'another kind of partnership' becomes possible.

The next chapter, by Stefan Helmreich, and with a postscript from Nicole Labruto, is about the intersection of capital and biotechnology. The text develops out of a book review—of Kaushik Sundar Rajan's *Biocapital* (Duke, 2006) and Nikolas Rose's *The Politics of Life Itself* (Princeton, 2007)—but here turns the labour of reviewing into a wider reflection on, and taxonomy of, accounts of 'biocapital' and its cognates. Helmreich offers both a comprehensive listing of the family of concepts that centre on 'biocapital' *and* a more-or-less literal genealogy of its taxa. Picking through his own labour of speciation, and the different forms of capitalization implicated in the various taxa, Helmreich begins to wonder what would happen if 'we asked not what happens to biology when it is capitalized, but asked rather whether capital must be the sign under which all of today's encounters of the economic with the biological must travel'. Nicole Labruto then takes up this discussion as it has torqued in the last decade, showing, for example, how analyses from the 'global South,' or those working at very different scales, might further shift our sense of how (and where) we might travel with this term.

Ed Cohen's chapter, which follows, stays at a broader and more programmatic level. Cohen analyses the genealogical entanglements of species and population as the reigning figure of the human. The chapter shows how species/population emerged at the nexus of eighteenth-century natural history and political economy, and argues that this emergence informs both the ways that political economy provides the bio-logic of capitalism, and the ways that

the 'human species' makes this bio-logic make sense—as the dominant calculus though which, today, we partition and participate in the world.

The final chapter in this section, by Samantha Frost, appropriately pushes us (and not always gently) into the future, constituting a sort of rough guide to the emerging politics of converging human and non-human worlds. Rather than rooting her contribution in one specific case, Frost develops a set of theses for the intersection of biology and politics—a series of concepts, principles, and practices that are not intended to *govern* the entanglement of human worlds, material environments, and historical cultures, including the subjects and organisms that are emergent *in* those tangles. Frost offers a kind of political guidebook to the biosocial present, one that ultimately builds toward 'a concept of humans that demands different, and differently detailed, figures of movement and interchange between body and environment'. Across ten substantively distinct theses, Frost offers a compelling account of how we might make sense of the ontological situation in which we find ourselves, and how we might learn to dwell in it as (still!) resolutely political creatures.

These chapters are unalike one another in important ways—politically, substantively, and methodologically—but what holds them together is an insistence that thinking biosocially is thinking about histories of contest and exploitation, and that these histories remain very present in current genomic and postgenomic projects. At stake in this section—and throughout the volume—is our insistence that we are not here blindly promoting some would-be novel biosocial confabulation, but are rather working to think through new (and old) configurations of the biological and social, as these are coming to inhabit one another in multiple political and research contexts today. And if we are willing to risk broadly thinking *well* of such configurations, or to *wish* them well, or to want to *do well*, we do not do so without an awareness that there is active political work required here too—and, indeed, that such work is not evenly distributed, and also that it might yet fail. The chapters in this section remind us that, whether we like it or not, we are going to figure out what it means to live in postgenomic worlds; at some point, we will need, together, to figure out a cosmopolitics of cohabitation, if we are ever going to make good sense of the present.

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Part I

History of the Biology/Society Relationship

2

Models, Metaphors, Lamarckisms and the Emergence of 'Scientific Sociology'

Snait B. Gissis

The truth is Lamarckism never was a real system … it is far more difficult to define than Darwinism … it is not so much a system as a point of view, an attitude towards the main biological questions.
Delage and Goldsmith, 1912 [1909]

In the latter part of the nineteenth century evolutionary biology of a particular bent—Lamarckian-Spencerian, neo-Lamarckian—played a crucial role for a number of sociologists in Europe and in the USA, molding their programs, methods, practices and even their self-image as scientists.[1]

This transfer established both similarity/suitability and difference between the biological and the social sciences domains. The 'logic of transfer' depended on the cultural-scientific-political context of that transfer, on whatever appeared plausible for specific individuals and did not appear arbitrary to their audiences, since both domains were based on similar social-institutional practices. It could take place only within contexts which allowed for the assumption that there was a fundamental correspondence between organic nature and social life, and between the mechanisms of development, modes of heredity, foundational units and general types of lawfulness in these domains. Thus, sociologists could present their emerging field as fundamentally similar and yet as uniquely distinct. The character of this migration/transfer gradually changed towards the end of the nineteenth century and the early twentieth century.

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The aim of my short chapter is to answer the question: 'How and why were models, mechanisms, analogies, metaphors and assumptions which could generally be characterized as Lamarckian-Spencerian, neo-Lamarckian, be perceived to be especially congenial to an emerging sociology seeking to become a scientific discipline in the latter part of the nineteenth century, and even in the early twentieth century?'

In answering this question I shall touch cursorily upon the following issues: determinism and plasticity, individuals and collectivities, heredity, inheritance, and deal primarily with Herbert Spencer and Émile Durkheim.

Let me start with a broad generalization. Four interconnected but distinct scientific objects relevant to the discussion emerged at the end of the eighteenth century and the beginning of the nineteenth century by virtue of economic, political, colonial and social contexts: 'society/the social', 'living nature', 'the self' and 'race'. Throughout the nineteenth century, these four scientific objects were analyzed, articulated, elaborated upon, diversified and deployed as foci of empirical research and theoretical investigations, and gradually crystallized into bounded scientific fields, into disciplines, became academized and institutionalized. These four scientific objects also became intertwined in multiple ways throughout that period. Investigating 'living nature' was considered a combined empirical/theoretical endeavor from its inception as a separate field of investigation. Later on in the nineteenth century, this gave the field a privileged position vis-à-vis the three others and helped shape a context, both scientific and social-political-cultural, that allowed transfers from it. The biological reservoir at large, and the evolutionary biological specifically, supplied styles of thinking, presuppositions, conceptual tools, materials used as evidence in theory construction, rhetoric and contents, and patterns of investigative practices. It suggested divisions to subfields, provided scientific legitimation and at times even temporary institutional incorporation.

Lamarck and Lamarckisms[2]

From the early nineteenth century on, 'Lamarckism' is a term that has come to cover a broad spectrum of theoretical positions on the nature of the mechanisms of evolution, and the controversies concerning Lamarckism, though at times very generative, were often conducted with little attention paid to the actual original arguments of Lamarck.

Lamarck (e.g. Philosophie Zoologique, Histoire Naturelle des animaux sans vertèbres) attributed an all-significant role to the changing environmental circumstances and to the resultant pressure on organisms. Changing environmental contexts gave rise to new needs in organisms, creating new

behaviors, which in turn brought about changes in functions, forms and sizes of organs. Thus, behavior and repetitive use or disuse that gave rise to cumulative minor changes in organisms could, through a gradual process, bring about the transformation of existing species and the creation of new varieties and species. Lamarck's doctrine not only accounted for individual variations within a given species, but since he looked upon species as changing/transforming over vast amounts of time into new ones, and as having only a relative stability in a particular environment, his doctrine broke down the conceptual boundaries among varieties and species and among genera. Accumulation of minor changes could be effected because they were inherited not as characteristics but as changed patterns of specific biological activities, namely, changes in the properties and motions of fluids of the organism. However, it is to be noted that the ability to pass on changes to the next generation depended on the age of the changing organisms involved. Most importantly, Lamarck was understood to claim also that the evolutionary processes involved the development of levels of increasing complexity in organisms. Lamarck considered organization and self-organization principal features of living forms, and thus viewed complexification as an inherent property of life. However, ontological 'reality' was attributed in an absolute sense only to individuals. Contrary to accepted historical lore, his views were widely disseminated and influential all over Europe, particularly in the late 1810s and the 1820s.

From the 1820s on, numerous explanations, some of which Lamarckian, were proffered throughout the nineteenth century to explain the history of life forms, their succession, their transformations and the mechanisms of their change. These explanations were disseminated not only through medical, biological and natural history channels but also through wider cultural media. Scientific, cultural and national contexts and styles formed and shaped modes of understanding, thus producing different images of Lamarck and Lamarckism. After the publication of Darwin's *Origin of Species* in 1859, later appropriations of Lamarck conspicuously used particular elements that were then deployed in conjunction with other explanatory components to produce new theories that were still discussed under the general heading of Lamarckism.

Nonetheless, there were certain features that served as a common foundation for that historical diversity. Foremost among them were the following:

(a) The environment was considered as an active causal agency in adaptive change. However, organisms were perceived to interact with it rather than just being acted upon by it in the gradualist evolutionary process;
(b) Evolution was seen as progressive, giving rise to an increase in the complexity of organisms;
(c) Self-organization was conceived as constitutive;

(d) Development was seen as central in the analysis of organisms;
(e) Behavior was considered a central explanatory mechanism in changes acquired within the lifespan of developing organisms (use—inheritance mechanism). These changes were perceived to be preserved and transmitted intergenerationally, thus combining development and heredity.

Many Lamarckists used some model of recapitulation—which Lamarck had not assumed—often based on an analogy between fetal development and the development of organic complexity throughout the animal series. Hardly any role was assigned to competition, conflict, struggle, nor to chance. Lamarckists used the available wealth of scientific resources, drawing on field and experimental data from disciplines as varied as chemistry, geology, entomology, paleontology, embryology and botany. This diversity partially accounts for the different emphases within loose groups of Lamarckists.

Until the 1880s, Lamarckism seemed compatible with Darwinism. There was enough room for positions that would be perceived as 'hybrid' (by today's historians), as well as for exclusively 'pure' neo-Lamarckian and neo-Darwinian positions. Thereafter, until after World War I primarily in the USA, France, Germany, Austria, and Great Britain, reflecting the various national and political frameworks of the life sciences, varieties of Lamarckism, some of which using the epithet 'neo-Lamarckism', played a significant, though at times an ambivalent, role side by side with other non-Lamarckian ones, such as Weismannian and, later on, Mendelian positions. The polarized varieties which emerged in the last decade of the nineteenth century persisted through the first two-thirds of the twentieth century. Neo-Darwinism crystallized as an exclusively selectionist, anti-Lamarckian view of evolution, while the hallmark of Lamarckism for many became the inheritance of acquired characteristics.

Spencer—Psychology, Biology, Sociology[3]

Spencer should be seen as a pioneering creator of a biological-social intertwining/enmeshing whose mechanisms for the amalgamation had an enormous impact. I shall briefly elucidate Spencer's Lamarckian brand of evolutionary biological core concepts and proceed to show how they were both embedded and embedding in his psychology and sociology, thus producing an intertwining which could and did serve in differing scientific-political- cultural contexts both as enabling vehicle for the emergence of new entangled fields and

for their disentanglement and bounding. During the period under consideration, the sociological point of view was beginning to emerge, and by the time Spencer was writing the many volumes of his *Principles of Sociology* (*PS*), the struggles to establish social science, also called 'sociology', as an autonomous discipline were widespread in Western Europe and in the USA. From the mid-1860s and until at least the late 1880s, Spencer's writings were translated and disseminated all over Europe and the USA, and reviewed, discussed and emulated.

In the mid-1850s, Spencer crystallized his view that Evolution (with a capital e) was the all-encompassing framework of living nature, a general process and principle whose main feature on all levels was a growing complexity of relationships between environments and organisms and their evolving effects. Spencer enunciated this radically innovative stance when introducing evolution to the analysis of the mental apparatus in the 1855, first edition of his *Principles of Psychology* (*PP*i). He there articulated what I consider to be his basic epistemological and methodological frame of analysis of living nature, namely, the changing interrelations of the changing interactions between organism and environment, and the changing effects they induce.

In all his works, Spencer was groping towards an evolutionary framework which, first and foremost, would provide a rich and detailed account of the complexity of the living world and its biological, psychological (mental) and social diversity, and give meaning to structure, functionality, adaptability, intentionality and other aspects of life as these evolved. All these were characterized by interactions, openness and directionality; all these were time- and history-bound. Furthermore, the role played by 'organism' in Spencer's three-place model did not have to be an individual, but could be fulfilled by both individuals and collectivities, and indeed became so. Within the Spencerian theoretical framework, the description of the process, which would apply at all levels and in all fields (but primarily in biology, psychology and sociology), was 'the movement from homogeneity to heterogeneity'. Thus 'heterogeneity' came to stand for 'complexity' and vice versa. Within his theoretical framework, the hierarchical feature of living nature was evidenced by increased heterogeneity, by progressing from a lesser to a greater degree of complexity, particularly in the relations among the tripartite elements of the model. The connections between 'the internal and the external', and in psychology between physiological and mental states, were encompassed under the same conceptualization (*PP*i 1855, 482–83, 485–86). Within Spencer's framework, 'physiological division of labor'—that is, the combined processes of 'differentiation', 'specialization' and 'integration'—functioned as a generalized

appellation and feature of mechanisms that were to explicate the process whereby complexity increased. The same applied to 'use and disuse' and 'inheritance of acquired modifications'. They provided what Spencer wanted: change and continuity, especially intergenerational continuity but with a conceptualization which rejected the metaphysics of isolatable, unchanging objects and/or entities and emphasized the evolvement of an ever-changing, increasingly complex order within these realms (e.g., *PP*i 1855, 368, 381, 382). For Spencer, organisms could be considered only within some environment. Thus, rather than positing a dichotomy, his theoretical efforts were directed at establishing a strong coupling between the two. Within Spencer's system, in order for 'environment' to have reference, there had to be an 'organism-environment-evolving effects' frame of analysis. Spencer considered 'environment' as the sum of all—physical, chemical, biological, social—external conditions and circumstances in which organisms found themselves, including the existence of other organisms. Spencer commented in various places that the effects of the environment on the lowest and on lower organisms were limited, and that these effects would become more diversified and more complex as the organisms themselves became more complex. Conversely, organisms impacted differentially on their environment: the lowest organisms did so to a minimal degree and the most complex organisms to a maximal degree, in accordance with the hierarchies of individuation (*PB*, 1867 I, 426). However, the boundary between environment and organism did not signify zones of internal cooperation versus zones of external competition.

Thus, because the reciprocal effects were not fully predetermined, there was latitude; the framework contained a measure of openness towards the future. This allowed for 'emergence'—for emergent new structures, and emergent new means of interaction. This is conspicuous in Spencer's sociology. He thought that states of homogeneity were inherently unstable, and that states of heterogeneity produced webs of interdependencies, rather than relations marked by dominance and subordination. He also conceived of changes of characteristics rather than of accumulated variations. Spencer's commitment to his basic tripartite frame of analysis and to complexification implied a hierarchical view of individuation and of living entities at large. It was hierarchical in that the progressive development Spencer was committed to required stable component entities, stable (on a specifiable timescale) but also endowed with a measure of plasticity that would allow for some change in structure, properties or functions. For Spencer, an individual was a consequence of evolutionary processes, and not a foundational unit in a description of living nature. Stability did not inhere in particular components of biological individuals, but in the sameness of mechanisms and patterns and in the continuity of their

workings. Viewed from Spencer's evolutionary perspective, the entities of his wide-ranging investigations manifested—to put it anachronistically—extensive phenotypic plasticity, tangled hierarchies, complicated classifications and controversial developmental histories.

Already in 1855 when dealing with psychology (in *PP*i), Spencer explained in great detail how experiencing produced neural changes through the mechanism of accumulative-repetitive experience, which he regarded as developmental-evolutionary. These changes were to be passed on hereditarily, that is, as a biological transmission of habituated psychological patterns of experiencing, acquired through the life of an individual. In *PP*i and *PP*ii, human competences, 'tendencies' or 'latencies' were neither solely biological nor solely cultural-mental. They were pre-organized molds, inherited as neural modifications functioning as neural-psychological patterns for organizing experience. By that experiencing, they were open at the same time to further functional-adaptational modification, which in turn would become biologically inherited.

Consequently it became possible for Spencer to intentionally blur the demarcation between biological heredity and social-cultural-psychological inheritance in order to posit hybrid categories—neither solely physiological nor solely mental. This whole edifice could be coherently sustained if the underpinning of the blurring-hybridizing became collectivist, while the rhetoric—political, ideological and scientific—remained individualistic, and thus could provide legitimation and induce acceptance for a new science. Thus, heredity was conceptualized on the borderline between the individual and the collectivity (the species, the 'race').

The central role of environment and adaptation to its changing conditions could, theoretically, be interpreted in two major ways:

1. A facet of a hardcore biological determinism that would overrule whatever effects social changes of environment could have. The capabilities of the individual would then be conceived as merely a reflection of the conditions of existence of the species and thus be bindingly hereditary. Biological determinists could then apply concepts of biological purity to race, to class.
2. The role of environment would be expressed through the weight of the formation and the transmission of social and cultural functionally adaptational patterns such as habits, customs, traditions, and thus it could highlight 'progress' as an open-ended endeavor.

As Spencer conceived of biological evolution as a movement from homogeneity to heterogeneity, and understood it as complexification, it could, to a

large extent, be identified with 'progress' when applied to humans. Lamarckism offered a double perspective on ethics and on society at large. It emphasized the overall importance of the milieu in shaping present and future generations through the inheritance of acquired characteristics, with 'use–repetition-habituation' as a major explanatory mechanism. This implied the possibility of shaping the future: present changes could be bequeathed as prospective biological traits to be further elaborated in the future. The seeming determination of the present by the past was one consequence of such thinking, the other the molding of the present in light of a projected utopian future. Note that 'socially' and 'mentally' collective inheritance was transmitted as biological heredity. It formed the molds which fashioned and shaped the individual's experiencing, behavior and thought. The changes were truly feasible only on the level of collectivities because although modifications were inherited by individuals, they were effected and inherited as social habits and psychological patterns. Thus, the Lamarckian 'future perspective' was dependent on its being a social/collective rather than an individual construction.

Spencer had first considered the discipline of sociology and its constituting elements in his essay 'The Social Organism'.[4] The guiding thread in 'The Social Organism', an analogy between individual (biological) organism and human society, continued to inform Spencer's work on social phenomena until the very end. He there asserted that 'society is a growth and not a manufacture'.[5] Societies and organisms were considered to be distinct from all other entities in the universe by virtue of two fundamental features: the continual increase in complexity of their structure, and the functional interdependence of their component parts. Though individuals and societies were mutually formed and fashioned, yet society was likened to an individual organism, or more precisely hierarchies of differing societies were likened to hierarchies of differing species of organisms. Societies and organisms were both conceived to grow by an increase of structural complexity, and by an increase of the mutual interdependence of parts, resulting in the 'whole' being more stable and longer-lasting than its component parts. The biological analogy of society to organism was helpful to Spencer in preventing outright teleological explanations. Instead, he deployed concepts such as 'function', 'harmony' and 'equilibrations', where the latter implied an inbuilt feedback or looping mechanism for system maintenance. In this manner, whatever appeared to the members of the collectivity as goals and ends could be looked upon as a consequence of the way the feedback mechanisms were arranged. However, Spencer posited human societies in contradistinction to the material bounding and material continuity, which were the most basic attributes of individual organisms. Their component elements, human individuals, were ordinarily seen as dis-

crete and separate, and materially, though not functionally, mobile even in the most elementary forms of society. Humans were all endowed with sensation, feeling and with consciousness, though differentially so.

Given the above, does it follow that for Spencer society was 'more than' an organism or that a biological organism was 'a society manqué'? In all of Spencer's writings on the social, a generalized, enhanced notion of biological organisms was mapped unto or projected upon human society in such a way so as to enhance certain projected features which would enable Spencer to claim that societies and biological organisms were different from anything else; and that at that level of resolution, society was an organism. These projected features were a hierarchically graded structural complexification, a growing functional division of labor, a gradual growth of internal structures of coordination and an evolvement of internal structures for the regulation and control of component units. Concurrently there was a marked effort to minimize other features in a manner that allowed him to argue that society was in fact distinct from an organism, and to enable him to call for a new mode of description, explanation and argumentation when dealing with societies. This new mode would constitute a new discipline, sociology, which would deal with sociocultural evolution. One of the most striking differences in Spencer's biology and sociology is found in the modes of classifying societies as compared to organisms. He classified societies solely

(a) By their degree of organization and
(b) By the predominant features of their overall institutional systems, and in particular, by the impact of these institutional systems on the modes of life of the component units of the society in their interactions with these systems.

I believe that Spencer deployed the analogy in the way metaphors are often used, namely, to project an affinity while at the same time to create a distance. That constituted distance opened up space for an analysis of societies as *sui generis* and thus for an analysis of the relations between individuals and collectivities within a non-biological conceptual framework. On the surface, the units of human individuals seemed to be the bearers of evolution, who transformed the collective evolutionary framework, but deep structure analysis would reveal them as predominantly transformed intergenerationally by that framework.

Even though he held a fairly deterministic view of evolutionary laws, laws in which the environment always played an all-important role, nonetheless Spencer's mechanisms for change included an in-principle contingency. The

plasticity of organisms, the existing diversity, and contingency, became key elements when trying to account for the enormous diversity in the biological world. And the same was true for societies as superorganisms: their plasticity, diversity and their ever-growing complexity—the latter an inherent feature of all the living—together with contingency were key factors in accounting for the growth and stability of societies. Evolutionizing meant accepting the priority of the collectivity at least on the methodological and epistemological levels. As we shall see, in that sense, Durkheim made explicit the implicit cost of Lamarckian evolutionizing in the human sciences. The later crisis of neo-Lamarckism both in biology and in the human sciences turned collectivist assumptions into a methodological anathema.

The Spencerian-Lamarckian 'amalgam' made it possible to deploy the biological reservoir of problématiques, models, metaphors and analogies both for legitimation and for constitution. One of the main assets of this 'amalgam' was its hereditary mechanism which in its many contemporaneous versions allowed for cultural inheritance to be biologized and vice versa. It helped offering social diagnoses, suggestions for potential cure, 'prescriptions' for the future, based on 'scientific analysis'.

Durkheim: Models, Metaphors and Transfers in the First Two Phases of His Sociological Writings[6]

Durkheim was one of the central and most important figures in the emerging field of sociology. His writings from 1885 till 1892 and from 1893 till 1897, which are usually characterized as early and middle-period works, are one of the foci of my paper. During that time span, Durkheim undertook to conceptualize and define sociology, positing it as a nascent science. In addition during the middle period he created an influential work-collective—the Durkheimians—producing intense quasi-disciplinary activities. Later on, they became one of the most forceful and determined contender groups claiming for the position of 'French sociology'.

The sociological narrative woven by Durkheim was continuous and causal. During his early and middle period, that exposition was based on an evolutionary plot into which the various historical and institutional layers of human societies and their timelines converged. Its method was claimed by Durkheim to be scientific, because it was presented either as a variation on, a continuation of, or an analogue to, methods used in the various branches of biology. However, I believe that the connection did not rest upon an *actual* similarity

but upon the cultural and scientific discourse of similarity between the social and the biological during that period. This *constructed similarity* could in another context be construed as its opposite, and it was.

Differences in the choice of an evolutionary matrix among sociologists stemmed from the specific society and concrete national culture within which they lived and worked, and from the varying interpretations given to those evolutionary matrices in the cultural and political discourses of those societies. And Durkheim was no exception in using biological evolutionary rhetoric as he sought to don a scientific garb to his work and to the new discipline he was attempting to constitute.

From the late 1870s until sometime in the late 1890s, there was *a particular intertwining of factors in France as the Third Republic was being shaped* which affected the processes I am concerned with, namely, the emergence and crystallization of neo-Lamarckian positions and the emergence of intense, quasi-disciplinary activities in various social sciences, particularly in sociology.

Although modern society was virtually the sole object of investigation in the various strands of the emerging sociology, deploying an evolutionary matrix enabled practitioners to utilize materials relating to both past and present western and non-western societies in constructing a single continuous narrative.

I contend that Durkheim adopted biology, and more specifically particular versions of *evolutionary* biology, as a model science and as a reservoir of models, metaphors and analogies for his new 'scientific sociology'. During the early period, he was vacillating between a Spencerian version of Lamarckism and the evolving French neo-Lamarckisms, as well as the uses of biology and organicism in general offered by other social scientists, primarily German ones, e.g., Schäffle. Taking these oscillations into account allows one to analyze a richer and more variegated spectrum of transfers from the evolutionary-biological onto the social and to better understand the more consistent use of neo-Lamarckian models and metaphors found in Durkheim's work from the mid-1880s until around the mid-1890s.

Spencer's *Principles of Psychology*, his *First Principles* and his *Principles of Biology* had all been quickly translated into French in the 1860s and 1870s. But Spencer's books and their byproducts also produced works utilizing/applying components of the Spencerian evolutionary synthesis to advance a realist analogy between an individual organism and society. Thus, natural selection was regarded as a secondary mechanism. The organic world was regarded as a graded continuity, and the evolutionary process as gradual. It was assumed that there was a directionality to evolutionary change from elementary, homogenous states towards heterogeneous states of greater

differentiation and complexification arrived at by way of adaptation as the environment effected changes directly, and a major role assigned to the use/disuse mechanism. These changes were conceived of as transmitted intergenerationally through the back-and-forth mechanism between the social-psychological and the biological. It also meant that 'environment', together with a metaphorical deployment of the 'battle for life', would easily be moved from biological to social contexts and become instrumental in fashioning social policies. However, in the 1880s, the Spencerian option came under focused criticism from different directions.

French neo-Lamarckian principal problematics, conceptual vocabulary, methodological strictures and constraints, commonalities and divergences gradually crystallized in the late 1870s and the early 1880s. Towards the end of the century, there appeared more synthetic overviews of the neo-Lamarckian evolutionary theory and practice, placing it within a more general history of the rise of evolutionary thought. One can point to certain 'transferable' commonalities:

(a) The central and all-important causal role of '*environment*' in bringing about adaptation to changes, and in the production of variations.
(b) The subsidiary, negative role of natural selection, with an emphasis on the unacceptable fortuitous character of the mechanism.
(c) The defining of 'life', 'organized beings', 'organisms' by invoking the notion of '*protoplasm*': a programmatic effort to emulate in physiological work the extraordinarily successful reductionist procedures of the physical-chemical sciences.
(d) The decision to isolate the *individual* organism—that most elementary entity in evolutionary terms—and make it the basic epistemological category. This, with its implicit reductionism, was considered as enabling a bottom-to-top deterministic causal explanation, which was considered the only one acceptable scientifically. Such an explanation was then perceived as mechanistic. Concurrently though, there were strong methodological emphases on the notion of *function*, and on functional unity.
(e) The efforts, variously formulated, to construct a narrative which would provide for novelty *within* the framework of organismal *plasticity*.
(f) The formulation and the dealing with 'heredity' in rather generalized terms, as a generalized intergenerational transfer of 'acquired features' or as something made concrete by the discussion of environment.

Also of relevance is the fact that already by the 1880s, acceptance of transformism—with various particular assumed mechanisms—had been perceived

by members of the new elites to be a shibboleth to membership in the forward-looking sector of the newly forged civil society and its functionaries. To be secular, radical (in the republican sense), to hold some form of faith in a materialist world view, to support some progressive solidarity as the moral-social ideal for one's society were attitudes that seemed to be significant features in the presentation of self common to neo-Lamarckists and to other members of the new elites, e.g., 'solidarist' positions. These attitudes were interlaced with concrete scientific positions that had been adopted within the transformist camp at that stage. They were looked upon as providing scientific underpinnings for both conserving/preserving national traditions and for initiating novel reforms. The 'futurity' of Lamarckism was wholly dependent on its being a social rather than an individualistic construction. Given the strong amalgam of biological and moral-political assumptions at that period, the implications of giving this up were considered intolerable in the early 1890s.

In the contemporaneous discourse, object, method, conceptual tools, projected practices, problematics were deliberated upon within a 'double-barreled framework': on the one hand, social and evolutionary, and on the other, biological.

In the 'early period', Durkheim fashioned his sociological project on some hybrid model of versions of evolutionary biology, *but parted company with the accepted commonalities and agreements of the contemporaneous 'double-barreled' framework on some important issues*, e.g., the concept of an individual and the notion of a separate discipline, while still adopting models and metaphors its adherents used.

The basic unit of French neo-Lamarckian evolutionary biological theories of that period was *the individual*, whether the organism itself or its component organs, since one could pursue the reductive process further to cells, to the protoplasm or its otherwise named equivalents, and to its 'individualized' chemical components. A principal feature of such an individual was the possibility of conceiving of it as singular, distinct and isolated, seemingly 'independent'.

Spencer was construed at the time as using in a realist manner the individual organism as a model for society within a particular version of the evolutionary plot. I maintain that Spencer was then understood as follows: wherever he drew the analogy between larger organisms (at times called 'colonies') and society, or seemingly between an individual and a collectivity, the latter was to be seen as a superorganic individual.

The basic unit and the focus of causal explanations *of most sociological theories of that period* was also *the individual*. The descriptions, analyses and explanations of collective situations then became those of the aggregated reactions of each individual to a state of affairs deemed common. Thus, for Perrier, only

by virtue of putting this 'individual-association' concept within a neo-Lamarckian evolutionary framework and assuming Lamarckian mechanisms could it hold. Alfred Espinas developed the realist analogy of the organism to society to the extreme. Thus, in Espinas the demarcation between society and multileveled individuals and between the organic/biological and the social were consistently blurred. The blurring allowed for a multitude of concepts that could be applied equally to both, and also made possible formulating a theoretization of the collectivity on the psychological level.

During his eight-month visit to Germany in the mid-1880s attending the universities in Berlin, Leipzig and Marburg, Durkheim became familiar with 'direct translations' of model to reality, and 'direct translations' of the biological to the social, such as the 'translations' effected by Paul von Lilienfeld. Durkheim also became aware there of the more widespread practice of mediated analogical transfer by which human society became turned into an individual organism as in the writings of Albert Schäffle.

There thus existed a number of influential, widely read, alternative, though closely related, modes of conceptualizing individual, organism, and collectivity/society, and Durkheim was well aware of these alternative theoretizations. But then*, very early on, he overturned the table by deciding to consider the collectivity as the basic unit both methodologically and epistemologically*:

> Society is not a simple collection of individuals, it is an entity which precedes those of whom it is now composed, and whom it will survive, (it is that) which acts upon them more than they act upon it, that which has its life, its consciousness, its interests and its destiny. (*RP* 1885, 19:8)

He thereafter had to face the difficulties of accounting for the role and status of individuals.

In somewhat later writings (*De la division du travail social, DTS*), Durkheim suggested that in sociology it was not individuals of any kind which could implement the conceptual sociological function that the 'protoplasm' or its equivalents fulfilled within biological evolutionary theories, but rather that it was a fictionally backwards-projected unit which, by definition, was a homogenous collectivity. *The object of sociological inquiry* was posited as the relationships rather than the related 'individuals'. However, these social relationships, '*les liens sociaux*', were conceptualized within a historical-evolutionary framework, whose mechanisms for change, for transformation and preservation were fashioned using the French neo-Lamarckian models.

In 1883–4, Durkheim taught a philosophy course in a college-lycée in Sens. Very little in it suggested the social science path that Durkheim chose

later. However, his determination to constitute sociology as a scientific discipline can perhaps be discerned in his lectures when noting the importance he attached to explicating what constituted science. When doing so he explicitly followed Claude Bernard. In the 1883–4 Sens course, the main features of the evolutionary mechanisms were a constantly changing environment and thus an ongoing adaptation of the organism which could bring about its further 'perfection'. In the very long run, it was heredity that fixed the organism's continued modifications as an attribute of the species. The survival of the most successfully adapted was presented as 'election by chance (hasard)'.

The early Durkheim insisted that the diversity and multiplicity of nature were basic, irreducible features of biological reality. In his introductory lessons in the University of Bordeaux in 1887–8, he began to delineate the later arguments to distinguish between deep structures and surface phenomena, the latter being indicators to the deep structures. This distinction, which seemed to work in biology, began to be analogously constructed in sociology.

The early writings were marked by sharp vacillations between strongly expressed statements of distinction and separation between the sociological and the evolutionary biological and also by tentative transfers from one field to another in order to provide projected methods and practices. Here too Spencer and the French neo-Lemarckisms offered differing possibilities.

When considering transfers, Durkheim argued that sociology as a new science should turn to evolutionary biology for ideas, models and hypotheses. He believed that evolutionary biology's approach should serve as a guide for studying social types together with the general features of social life. The same applied to the notion of environment, internal and external, and its transferred role in the construction of the collectivity and its social environment. The taking apart and then bringing together of the individual organism should serve as a methodological guide for the sociologist. All explanations of social processes were formulated so as to bring out this aspect which was deemed central to French neo-Lamarckisms, that is, that 'tout se passe mecaniquement' (*RP*, 1887).

When concerned with the separation of the social and the biological and drawing a distinction between the two, Durkheim put forth the example of Claude Bernard, whose role as model had been significant to the French neo-Lamarckists. This example was advanced to emphasize that sociology should follow biology in the latter's striving for scientificity, and in its efforts for a separation of the biological from the physical, this in order to posit an autonomous biological method. Applying it to sociology, Durkheim stated: 'There must be something in the social realm which escapes biological investigation.'[7] And in reaction to Schäffle's book mentioned above, Durkheim

remarked that any straightforward and direct application of biological practices to sociology would only hamper its progress.

Already at that early stage, perhaps while vacillating and not being fully aware of it, Durkheim was performing the double movement that characterized his later work: on the one hand, the close transfer in order to constitute a discipline and legitimize it, and on the other hand, the distancing, bounding and distinguishing in order to establish its autonomous status.

I would argue that considering the range of his works during the early and the middle period, Durkheim's innovative claim to explain the social by the social was to a significant extent made possible, seemingly paradoxically, by the sophisticated manner in which fundamental tenets and terms of contemporaneous, mostly French neo-Lamarckian, evolutionisms were transposed to the field of society. This Durkheim did by two modes: one strategic, the other theoretical. I consider Durkheim's discussion of the concept of 'social life' both in *De la Division du travail social* (*DTS*) and in *Les Règles de la method sociologoique* (*RMS*) as elucidating examples of the strategic-rhetorical usage. Durkheim's main argument was that a history of forms of social life was a necessary component, just as a history of forms of life in biology had been. Through this in *RMS* he incorporated sociology among the sciences that dealt generally with 'forms of life', and consequently also with the speciation of such forms. 'Forms of life' were posited as equivalent in signification to 'forms of organization', thus implying a methodology of 'the whole and/versus its parts'. In *RMS* Durkheim also used the analogy between 'life' in its biological signification and 'sociability' in its signification as 'social life', in order to distinguish between psychology and sociology. Life in both significations depended on the association of components, on their organization and structuration. It could not inhere in any separate component alone. The distinction between the living and the non-living within biology by analogy could serve to distinguish the subject matter of sociology from other disciplines, and draw its boundaries as a new discipline.

The earlier mentioned discussion by Durkheim of what should be considered the elementary unit of society—the 'protoplasme social, le germe d'ou seraient sortis tous le types sociaux' (*DTS*, VI:149)—paralleled the contemporary discussions by Lamarckians (in obvious contradistinction to Darwinians) in search of the most elementary, most primitive unit from which all life had evolved. Given Durkheim's presuppositions that homogeneity was prior to and conditional for heterogeneity, given his presupposition that the collectivity was prior to the individual and conditioned the individual, this 'protoplasme' had to be a homogeneous group, that is, an aggregate with no internal differentiation. Durkheim was fully aware that this was a

hypothetical unit, a construction he needed in order to draw the complete outline and genealogy of a series. In biological theorizing, this methodological device was employed only when the other links in the series had already been delineated. Durkheim's series, however, was yet to be constructed. Nevertheless, he deemed such a hypothetical construction, in analogy to the biological one, was theoretically necessary, in order to posit an evolutionary tree with its accumulating social traits and patterns.

Durkheim deployed 'Organism' as a necessary component in his argument about the relations between individuals and their collectivities. These relationships—but not the actual individuals, nor the actual collectivities—could be viewed, in some of their aspects, as analogous to parts of the organism in relation to its totality. He insisted on a very partial application of this analogy to individuals, and admonished against any literal understanding of it. In contradistinction to most of his contemporaries, Durkheim's usage of the analogy purported to make the collectivity epistemologically real, to construct its 'visualization' or 'observabilty' at a time when the visibility of 'Nature' had become a significant scientific issue. Thus, he transposed the differentiation in the economy of the organism into the social differentiation resulting from the division of labor in society. Social plasticity meant that when a primary process of adaptation to the environment occurred, deemed as either biological or social, it resulted in a certain practice, a *social* pattern. This pattern would then be transmitted as a habit and custom, that is, through processes of socialization and acculturation. Adaptation of individuals could be understood only within their culture. This was considered by Durkheim to be the *sociological equivalent of the inheritance of acquired traits*. The division of labor as a mechanism of change could induce alterations in the moral, ideational and civilizing practices and beliefs of societies, and thereby transform those of individuals.

Furthermore, in contradistinction to many readings of Durkheim, I contend that his thought can be better understood methodologically as an evolving, at times problematical and contradictory construction of classificatory social continua. These were differentiated by their time scales and by whether they reflected individual or collective attributes. One such important continuum related to 'habit', a central concept in any Lamarckian scheme whether biological or social, that stemmed from the central role of the concept of use and disuse in Lamarck's work. Another continuum, perhaps the most important in Durkheim's social theory, was the social continuum between acts-and-states of individuals and collective institutions. The variety of social life revealed a movement from states of acting and behaving and from states of thinking and feeling, states that were transient, unstable and completely

unfixed, applicable for short periods of time to individuals or to limited segments of the collectivity, to patterns that were more solidified, lasted for longer periods of time, became fixed and even permanent. The latter were always typical of the entire collectivity, and were reflected in the social behavior of an individual. These patterns could be socially transmitted between generations. Durkheim called this process 'crystallization', which I interpret as *an effort to establish a continuity* between the beliefs, and experiential states of individuals and the persistent, more enduring, collective practices, patterns and institutions, and their material products (which also constituted an integral part of that continuum). The distinction between crystallized social institutions that could put constraints, impose obligations, even coerce, and the processes of consolidation of social phenomena related to the position of phenomena along the social continuum.[8] Most contemporaneous sociological and ethical theories assumed that 'doing' involves a specific individual who was the 'doer' or 'the owner of the doing'. Durkheim's unique position vis-à-vis his contemporaries was his insistence that the 'real owner' was a collectivity, that the 'doer' in this sense was a social group. This assumption was to serve as a methodological lever in discarding explanations given in terms of motives, intentions, meanings and ends of individual doers, and thus was instrumental in transposing the concept of causality from the natural sciences to sociology, another significant move in the effort to establish sociology as a scientific discipline. However, his position on these problematics underwent significant transformations which reflected severe tensions regarding foundational issues.

Durkheim's position throughout the 1890s was that society should be explained by social categories, but that the conceptual tools for this analysis were to be borrowed from another science. This was done while remaining acutely aware of the difference between the two fields. Self-consciously and intentionally Durkheim advocated 'a two-layered practice'. On the one hand, a transposition which changed the referents and dissolved the coherence of the original model, and on the other, a procedure through which the singular aspects of sociology were made conspicuous, particularly those in which the transposition stood out.

Durkheim's attitude to transfer from evolutionary biology had been crystallized in the mid-1880s, while writing review essays. What he had already criticized mildly in the 1880s he totally rejected in the 1890s, specifically: the direct transfer in which organisms and social organisms were discussed identically. He invested great efforts in drawing fine distinctions between this practice and other modes of transfer and transposition he held to be legitimate and generative. This was done in order to present the new field to be constituted—scientific sociology—as autonomous, with its object distinct from

that of biology, its methods unique to it, and its boundaries—that is, the legitimate problems it considered and its modes of explanation—clear-cut.

I believe this had to do with the particular 'mechanism' of metaphorizing—that is, establishing similarity and marking and positing distance—that Durkheim deployed in delineating the boundaries of the new discipline. He constructed a gap of dissimilarity between the two disciplines, which could not have been constructed unless similarity had been formerly assumed and established. In his three books of the 1890s, *DTS*, *RMS*, and *S(Le Suicide)*, Durkheim pointed out a group of differences that he thought would single out sociology as distinct and separate from biology.

Their common feature was that the object of biology was characterized as being determined, while the object of sociology was presented as partially undetermined environmentally, that is, context-wise so, and relatively flexible, with a different 'logic' (in the sense of types of order and of relations such as those between functions and structures). This distinction served as a way of drawing the line between what could and could not be transferred over the boundary thus posited between biology and sociology.

The French neo-Lamarckian evolutionary reservoir, a significant resource for Durkheim and his group during those years, disappeared before the turn of the century, because it became superfluous or hampering in more than one sense. The particular Durkheimian modes of transposition created a certain common familiarity among those who either passively accepted them or actively employed them in their own work. It turned them into 'a community of practice'. However, with the 'getting together' to publish *L'Année sociologique* and with their alignment around the Dreyfus Affair, new ways and means of forging this 'work-collective' became available. Moreover, the Durkheimians became engaged in another intense activity of bounding through their overall plan to sociologize the sciences dealing non-biologically/medically with humans. This work of bounding was carried out by recurring mappings of the social sciences, particularly in their annual journal, *l'Année Sociologique*. Furthermore, in order to distance sociology from its most closely related rival at the time, philosophy, and at the same time divest philosophy of its authoritative position in the systems of high school and higher education, the Durkheimians 'annexed' to sociology some of philosophy's traditional topics. Moreover, some key members of the group called for a non-biological mode of dealing with the problematics of collectivity-individuals, and started to elaborate diverging modes for doing that. This, it turned out, presented the possibility of excluding from the general field of 'the social sciences' groups and individuals who deployed a biological 'race' category.

Throughout the late 1890s, a heated and prolonged debate raged within the French educational system, which at the time was undergoing thorough ongoing reforms.

One of its more far-reaching results was that competing sociologists were compelled to delineate the boundaries of their discipline more sharply, to mark more conspicuously the differences between the object and method of sociology and those of other disciplines and to constitute it as a discipline with its own unique epistemic assumptions and its own rhetoric. Note however, that these considerations and examinations did not result in social thought and sociological activities at large, thereby becoming biology-free in France.

A supporting factor in that bounding process of emerging social sciences in France was the gradual decline of the scientific status of the French neo-Lamarckisms, resulting from the impact of the publication and translation of August Weismann's works and even more importantly the gradual realization of the implications of his theory not only for experimental biology but also for the transfer from evolutionary biology to other spheres. Thus, from the mid-1890s on, there followed/evolved a sharp polarization of the French community of biologists into neo-Darwinians and neo-Lamarckians.

The adoption of sophisticated transpositions and transfers from French neo-Lamarckisms had at one stage played an important role in the success of Durkheim and his group in creating a new space for themselves within the cultural field. The gradual bounding of sociology, its later presentation as an autonomous discipline, completely distinct from any transfer or transposition from biology, and the shedding of the French neo-Lamarckian garb was done at a critical moment for its legitimation in the fast-changing political and cultural context of ideologically controversial educational reforms, of the Dreyfus Affair and particularly of its aftermath. I believe that Durkheim and his group were groping at that stage to posit another 'common ground' for the then deeply divided French society, a social-cultural 'common ground' for which no such transfer was needed.

Conclusion

During the second half of the nineteenth century, both biologists and social thinkers coped methodologically and epistemologically with issues related to heredity, inheritance, individuals, collectivities, collective frameworks, sociality. In the newly emerging 'fields of sociology', various influential figures, starting with Herbert Spencer, assumed some relations of dependency between

individuals and collectivities in their explanatory mechanisms of selected properties of individuals. They posited mechanisms involving concurrently modes of biological heredity and of social inheritance and were concerned with questions connected with the extent of determination and plasticity involved in intergenerational transmission. They struggled with supplying the grounds for making claims of scientific legitimacy and universality for their developing fields. I have looked at Spencer and Durkheim, two figures, who were widely and deeply influential with constituting the discipline of sociology in their respective countries, Britain and France. At an important stage in their activities, they embraced Lamarckian/neo-Lamarckian modes of biological description and explanation, and these were instrumental in fashioning their sociological views. Both were members of communities that were situated within cultures that were rapidly being transformed and were being shaped by pressures and needs in them.

When biology was contributing to constituting sociology, biologists shaping evolutionary perspectives were struggling with the relevance to biology of the successful example of science-making presented by the causal mechanisms prevalent in the physical and chemical sciences of the period, and in particular, presented by the reduction of these sciences of the macro to the micro and their analysis of wholes in terms of interactions of the smallest entities assumed to compose them. It is within this context that the transfer from the evolutionary biological to the sociological took place, and shifting forms of dependence between individuals and collectivities were being shaped. I contend that the Lamarckian/neo-Lamarckian mechanisms and assumptions used by Spencer and Durkheim were adopted to 'collectivize' the individuals through shared components that coupled individual development and an evolutionary history of the human species, the social and the biological, and also related past determination and future-oriented possibilities. These Lamarckian/neo-Lamarckian mechanisms were looked upon as forming an all-important component of the enabling conditions for sociality for both the species and the individual and were also considered relevant to the issue of making universal the formulation of sociology as a scientific field, a sociology that could serve as a foundation for reforming society.

Notes

1. Given that this is a handbook chapter, the number of references to both the primary and the secondary literature have been kept to a bare minimum.
2. See, e.g., Burkhardt 1977; Corsi 1988; Bowler 1988; Conry 1974; Gissis 2009; Loison 2010.

3. See, e.g., Gissis 2005; Francis 2007; Francis and Taylor 2015; Jones and Peel 2004; Peel 1971.
4. *Westminster Review* 1860.
5. See p. 269, also p. 266.
6. See, e.g., Alexander and Smith 2005; Gissis 2011; Lukes 1973; Schmaus 2004; Turner 1993.
7. *Cours* 1888.
8. See detailed quotations in Gissis 2002.

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3

The Transcendence of the Social: Durkheim, Weismann and the Purification of Sociology

Maurizio Meloni

Introduction: Weismann and the Possibility of the Social

This chapter addresses the emergence during the late nineteenth century of a certain way of thinking that came to be seen in the twentieth century as self-evident for many social scientists and biologists alike. According to this way of thinking, "If something *is not biological* in origins, *it must be social*" or, alternatively, "*If not social*, it *must be biological*". The many possible versions of this fundamental way of thinking can be easily found in hundreds of articles discussing behavioural, medical or developmental issues. A slightly more sophisticated refinement introduces the view that traits, disease or behaviours are actually a bit of both, or rather the result of an "interaction" or "combination" among the two kinds of cause, namely the biological and the social, nature and nurture, heredity and environment. However, as Evelyn Fox Keller has noticed, this apparent synthesis creates more problems than one may think: "the notion of interaction presupposes the existence of entities that are at least ideally separable—i.e., it presupposes *an* a priori *space between* component entities" (2010, 6, my italics).

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The argument of this chapter is not to offer an alternative way of thinking to this *a priori disjunction*; nor is to critique its possible epistemological shortcomings. My interest is instead genealogical: How did we come to think this way? When and how did posing biological and social factors, blood and civilization, heredity and environment, as alternative domains start to make sense, up to the point to become a sort of truism? In this chapter I build on Keller's acute genealogy of the nature-nurture opposition as located in a certain specific social, cultural and political history in the late nineteenth century (2010), to bear on a broader problem: the making of a really modern (i.e. non-biological) sociology, which emerged with its idea of a purely social (i.e. non psychobiological) level of causation nearly at the same time as this "hard disjunction" (Keller 2010) between heredity and the environment, nature and nurture, was made.

Is there any connection between the emergence of the social as a non-biological and non-psychological source of causation and the making of the modern view of heredity (Johannsen 1911)? Is just a coincidence that sociology—as we are told in nearly all textbooks—started to emancipate itself from biologism in the very last years of the nineteenth century, exactly in the arc of time between Weismann's publication of his seminal compendium on heredity (1893a) and the rediscovery of Mendel (1900)? This relationship between history of sociology and history of science remains in my view one of the most overlooked in intellectual history. Building on an existing scholarship, in a previous article, I have already argued how Alfred Kroeber, a key figure in American anthropology, crucially depended on the incorporation of Galton and Weismann to purify anthropology from the "vitiated mixture" of organic and superorganic explanations, that is., Lamarckism (Meloni 2016a, see also Kroenfeldner 2009). However, people may think that Kroeber is just an idiosyncratic case not generalizable to other cultural contexts and disciplines. In this chapter I will argue for a parallel, though subtler, role of Weismann in the making of Durkheim's sociology. I want to claim, rather provocatively, that the transcendence of the social in Durkheim (truly Durkheim's trademark) is *entirely isomorphic* to Weismann's transcendence of the germplasm: in both cases, they aimed to construct objective realities radically independent and exterior from individual tendencies and peculiarities. The collective nature of the social is perfectly analogous to the collective nature of hereditary tendencies established by Weismann (and Galton before him). As we shall see, Weismann offered Durkheim an important scientific support to make boundaries between sociology and biology. Since the discovery of Weismann in a footnote in the *Division of Labor* (1893), Durkheim borrowed from and capitalized on Weismann's epistemic revolution, founding a scientific pendant to

his idea of an ontological break between the social and the individual domain, that is, that a social fact is not the sum of many individual facts. This ontological hiatus, as I will show, was impossible to conceive under a nineteenth-century Lamarckian framework, and instead corresponded perfectly to the modernization of heredity started by Galton and consolidated by Weismann, and later by genetics. To understand this, however, it is important to go back to what happened before the bifurcation between the social and the biological took place, when this broadly Lamarckian framework was at its peak. I will focus here (summing up some of the themes of my recent *Political Biology*; see Meloni 2016a) on two different disciplinary bodies of knowledge in the nineteenth century: social theory and philosophy on one side; medical writings on heredity on the other.

The Complicity of the Social and the Biological Before the Big Dichotomy

Social Theory and Philosophy

Before the word biology was coined in the early 1800s, there are obviously many predecessors of a dichotomous understanding of the relationship between "nature" and "society" (if we want to use nature as a proxy for what will be later called the biological). In early modernity, Rousseau's name comes easily to mind for his radical disjunction between nature and society. Rousseau, who was the first to use "social" as the adjective of society (Heilbron 1995), was also the most original representative of a trend that opposed the *social order as non-natural* to nature *as non-social*. Part of this naturalism transited into the next century in the form of an ethic and aesthetic celebration of nature among the Romantics. However, Rousseau's point was mostly normative, not aimed to parse human behaviour in biological or social explanations as antagonist causes. Moreover, if we look at things later in the nineteenth century, when the notion of the social starts to be more intensely theorized, an utterly dichotomous framework is indeed rare. For instance, the young Marx's view in the *Economic and Philosophical Manuscripts* (1844) contains a holistic understanding of the social in which "the social character" is not something opposed to "the natural", but it is rather the totality of human relationships (natural ones included). In a different context and decade, John Stuart Mill's view has been elegantly analysed by Fox Keller (2010), and I can simply repeat her point here. In his *Utilitarianism* (1863), Mill considers "moral feelings" as "not innate, but acquired", a statement that provoked

Darwin's distress in his *Descent* a few years later. Mill's sentence may seem to reflect a twentieth-century antagonism between nature and nurture. However, as Keller observes, in claiming that moral feelings are acquired, Mill was making an entirely non-dualistic point. If moral feelings are acquired, he wrote, "*not for that reason* [they are] *the less natural*. It is natural to man to speak, to reason, to build cities, to cultivate the ground, though these are acquired faculties" (Keller 2010; see also Paul and Day 2008).

Spencer provides an even clearer illustration here, given his recognized dependence on Lamarckism (Peel 1971; Bowler 1983; see also Burrow 1966; Offer 2010). In a Lamarckian context, as I have argued elsewhere (Meloni 2016a, b), the social is always on the verge of turning into the biological, that is, in a nineteenth-century language, habits via use-inheritance are progressively fixed and transmitted by heredity to the next generations. Use-inheritance necessarily undermines any strict boundary between the social and the biological, the mind and the body, as well as the acquired and the innate. As Spencer said in his very Lamarckian *Factors of Organic Evolution* (1887), acquired characteristics "may, in the successions of individuals, *generate innate tendencies* to like or dislike such actions" (my italics). The sentence is obviously troubling for a twentieth-century understanding of the innate as fixed and therefore *impossible to be generated* by the influences of previous generations: but Spencer, as a Lamarckian, did not see a contradiction at all in looking at the *innate as something generated* from the deeds of previous generations.

However, the function of Lamarckism was not only to confuse (if not make utterly impossible) the distinction between the innate and the generated, the social and the biological. It was essential for nineteenth-century sociology that Lamarckism offered a key mechanism to connect organic and social evolution, biological and moral progress, thus making sociology coterminous with social evolutionism in a teleological and linear view (see Weinstein 1998; Gissis 2003). It is at the conjunction of three key arguments that Spencer makes organic and social progress indistinguishable. Firstly, that morality has a physiological basis or rather is "a development of physiological truth" (1851/1883, chapter 31, par. 6). Secondly, that human characteristics are malleable by the environment—something especially true for higher civilizations deemed more plastic than others (thus establishing a hierarchy of civilizations based on plasticity). Thirdly and finally, that use-inheritance is true, thus making moral progress, as a physiologically based feature, cumulatively transmissible across generations (Weinstein 1998). Spencer is not shy to make the case for a clear sociological implication of his broader biological view: "If functionally-produced modifications are inheritable, then the mental associa-

tions habitually produced in individuals by experiences of the relations between actions and their consequences, pleasurable or painful, may, in the successions of individuals, generate innate tendencies to like or dislike such actions. *That our sociological beliefs must also be profoundly affected by the conclusions we draw on this point, is obvious*. If a nation is modified *en masse* by transmission of the effects produced on the natures of its members by those modes of daily activity which its institutions and circumstances involve; then we must infer that such institutions and circumstances mould its members far more rapidly and comprehensively than they can do if the sole cause of adaptation to them is the more frequent survival of individuals who happen to have varied in favourable ways" (Spencer 1887, 5–6, my italics).

This and similar worldviews were extremely widespread in the nineteenth century. Such philosophies opposed the scary randomness of Darwinian variation, with a reassuring teleological view of *biological*-cum-*social* progress, a steady advancement (as in biological ontogeny) from the homogenous to the complex, with little or no space chance. Regression and degeneration were definite possibilities, but faith in "perfectibility" was far stronger (Gissis 2003). Before coming to the destruction of this Spencerian worldview (as a consequence of the emergence of hard heredity and later genetics), it is important to look quickly at a second body of scholarship: medical hereditarianism before hard heredity.

Heredity Before Modern (Hard) Heredity in Medical Writings[1]

It is very telling that until the eighteenth century the word heredity had mainly a juridical meaning (Müller-Wille and Rheinberger 2012; see also Johannsen 1911), while in a medical context, it was used only as an adjective (López-Beltrán 2004). A significant change occurred only from the early nineteenth century, when the notion of *hérédité* or *heredity* started to be nominalized and investigated as a phenomenon in itself in medical writings especially in France and Britain. However, what is meant by this hereditarian literature is very different from our post-twentieth-century understanding. Heredity meant in the early and mid-nineteenth century a complex entanglement of social and biological factors, innate and acquired characteristics. It envisioned a blurred mechanism "beginning with conception and extending through weaning" (Rosenberg 1974). A case in point is the Enlightenment polymath Erasmus Darwin, Charles's grandfather who viewed heredity "as the result of a malleable admixture of nature and nurture causes" (Wilson 2007).

Erasmus believed that exciting external causes produced structural changes in the organism and were then fixed into heredity. These views were very visible for instance in his poem *The Temple of Nature: Or, The Origin of Society* (1806): "The clime unkind, or noxious food instills to embryon nerves hereditary ills" Erasmus wrote; "The feeble births acquired diseases chase, Till Death extinguish the degenerate race" (cited in Wilson 2007, 137). Erasmus's citation is very early in the nineteenth century, but Charles himself, Erasmus's grandson, still until 1868 (*Variation of Animal and Plants Under Domestication*), held to a mechanism (which he named "pangenesis") whereby direct communication existed between body cells and reproductive organs. This would be in flagrant violation of what we know today as genetics. A good illustration of the gap between Darwin and the modern view of heredity can be found in the quarrel between Darwin and his younger half-cousin, Francis Galton, on the empirical validity of pangenesis. Galton tested the gemmules hypothesized by Darwin and showed no circulation in the blood of these "reproductive elements". After that episode, which we can consider as a sort of parting of the ways, Galton's view of heredity developed autonomously and originally. A new view of heredity radically close to environmental inputs was made after Darwin thanks to the converging effort of two different traditions of thought, one mostly statistical and anthropological championed by Galton, the other embryological represented by August Weismann (Churchill 2015). The two views had much in common, and their conceptual impact went well beyond history of science as I will try to show next about Durkheim. However, it is important to focus quickly on the significance of the making of the modern knowledge regime of heredity (Müller-Wille and Rheinberger 2007, 2012).

The Making of Hard Heredity in the Late Nineteenth Century

The making of hard heredity in the last three decades of the nineteenth century is an event of immense importance, in science and beyond it. As a significant body of scholarship has shown (Bowler 1989), the same rediscovery of Mendel in 1900 can be considered a delayed effect of the making of hard heredity. Hard heredity, or the modern notion of heredity (Johannsen 1911), is the notion that heredity is fixed at birth and is not affected directly by changes in the environment (Bonduriansky 2012). It was the making of this notion that created the epistemic space within which the Mendelian notion of a particulate and stable (unchangeable) hereditary material (later christened the gene) could be situated, and Mendel "rediscovered". As Weismann proudly

claimed, "Mendel's law is an affirmation of the foundation of the germ-plasm theory" (quoted in Churchill 2015, 540).[2] Leaving aside Galton's key contribution, I will restrict my analysis to Weismann, for reasons of space, but also to advance my thesis about the structural analogies with Durkheim's thought.

August Weismann

Celebrated by Mayr as "the greatest evolutionist after Darwin" (1985), August Weismann (1834–1914) stood for a transformation from the "original, flexible Darwinism" that could still make room for a "Lamarckian component in addition to natural selection" to a more "dogmatic" one (Bowler 1983, 75; see also Mayr 1982) in which natural selection was the exclusive and omnipotent source of individual variation. In the 1880s, Weismann tested Lamarckian inheritance by amputating the tails of more than 20 successive generations of mice. Their offspring all had intact tails. These experiments were intended to disprove the theory that acquired mutilations could be inherited across generations. Lamarckian inheritance, according to Mayr (1988), "never regained full credibility after Weismann's attack", though Lamarckians have disagreed on the significance of these experiments. Weismann put on much shakier grounds the inheritance of acquired traits that was, at the time, nearly considered a common-sense view.

Since 1880s, Weismann was understood as the proposer of a crucial turn in the reconceptualization of heredity and evolution, "striking at the very root" (Wallace 1889, 411) of all theories claiming for direct effects of the environment on heredity. Given these expectations, Weismann soon became a polarizing figure that could be embraced or fought against, but could not leave things as they were before. Before Weismann, natural selection and Lamarckian inheritance were seen as concomitant factors in the process of selection differing only by degree, not kind (Romanes 1899). Heredity was a pluralistic mechanism. After Weismann, the polarization between these two mechanisms—natural selection and the inheritance of acquired characters—became extreme, giving rise to a series of ideological fights. The term neo-Lamarckians and neo-Darwinians were both created after Weismann's first important works, between 1885 and 1888. The heated debate with Spencer in the early 1890s (e.g. Spencer 1893a and b; Weismann 1893b) is very representative of this clash between what, after Weismann, emerged as two irreconcilable worldviews. Weismann's idea of heredity was known as the theory of the "continuity of germplasm" and was based on the assumed "existence of a special organized and living *hereditary substance*, which in all multicellular organisms,

unlike the substance composing the perishable body of the individual, is transmitted from generation to generation" (1893a, xi). The doctrine of the continuity of germplasm is a fundamentally dualist one, based on a "contrast between the *somatic* and the *reproductive* cells" (1893a, 183). As Mary Jane West-Eberhard in a now classic work explains, "The cells of the soma participate in growth and differentiation, but then they die, while the germline cells, set aside early in development, serve as an uncontaminated bridge to the next generation" (2003, 331). What was destroyed by this view was any bridge between the individual and the race. This is the opposite of a Lamarckian-Spencerian view in which individual acquisitions are passed on and become fixed into the heredity of the group. As I shall argue next, *this ontological hiatus between the race and the individual*—made possible by the destruction of the Lamarckian bridge of use-inheritance—was understood by Durkheim *as a scientific pendant of his also dichotomous view* of the hiatus between social and individual life. To convey this idea of an impossible communication between "characters acquired by the adult body" and germplasm (Bowler 2009), Weismann used a metaphor that Durkheim would have probably liked: to suppose communication between what is acquired during a lifetime and the hereditary substance "is very like supposing that an English telegram to China is there received in the Chinese language" (Weismann 1904).

Weismann, Weismannism and a Legacy to Re-evaluate

Underneath the image of Weismann as the man who destroyed Lamarckism, a more nuanced historiographical tradition has established that the German embryologist pioneered elements of a radically new vision of heredity while adhering to old developmental and even environmentally induced views of heredity that persisted until his last publications (Churchill 2015; Bowler 1989; Winther 2001; Novak 2008). There is no doubt that broader political pressures (Winther 2001) hardened the Weismannian dichotomy between non-heritable somatic variations and germplasm heredity into a broader ideology, "Weismannism". This ideology was at the heart of swelling eugenic and hard-hereditarian schools of thought in the early twentieth century. Weismann was seen as buttressing a conservative racial argument, bringing support to the racial hygiene movement in or the militaristic ideology of the ruling elites in Germany (Crook 1994). Nevertheless, beneath the more ideological uses, if not caricatures, of Weismann's thought, his profound and long-lasting impact as an original thinker has to be entirely re-evaluated, especially in its implications for the social sciences and ideas of social reform and progress. Beyond

politics, the rise of Weismannism (or neo-Darwinism) was seen as a huge intellectual catastrophe for the social sciences as well. Herbert Spencer understood Weismann as a menace for "Education, Ethics, and Politics" (1893a, 488). Lester Frank Ward (1841–1913), the prominent neo-Lamarckian and first president of the American Sociological Association, was similarly perturbed by Weismann, the new "great prophet of science". If hard heredity were true, he surmised, social progress would be lost. How could it be otherwise if each generation's political, moral and educational efforts were erased with the rise of the next?

> If nothing that the individual gains by the most heroic or the most assiduous effort can by any possibility be handed on to posterity, the incentive to effort is in great part removed. If all the labor bestowed upon the youth of the race to secure a perfect physical and intellectual development dies with the individual to whom it is imparted why this labor? (…) In fact the whole burden of the Neo-Darwinian song is: Cease to educate, it is mere temporizing with the deeper and unchangeable forces of nature. And we are thrown back upon the theories of Rousseau, who would abandon the race entirely to the feral influences of nature. (Ward 1891, 65)

Thus, Weismannism was initially received as a reactionary and exclusionary doctrine in politics, supporting fatalist and nationalist views, while from the perspective of the social sciences, it seemed to offer few if any advantages. However, Weismannism inspired less intuitive political corollaries as well, both in politics and in terms of knowledge production. Before looking in detail at what Durkheim borrowed from Weismann, the subtle and richer implications of Weismannism have to be emphasized. I will focus here on two points.

Firstly, in politics, there is an obvious consequence of Weismannism that was seriously overlooked by Lamarckians. As various neo-Darwinists have claimed in different contexts, from Alfred Russel Wallace to Yuri Filipchenko, and Julian Huxley, the degenerative effects of the environment would be contained and even neutralized by an impervious hereditary substance (Meloni 2016, chapter 4). After all, if the good effects of education could not be attached to heredity, then the ill-effects of unequal social structures would also be kept at bay. As heirs of the twentieth century, we struggle to understand how hard heredity could be progressive because we tend to associate the emphasis on the environment as typical of social reform movements. However, in a period where claims of the degeneration of races and classes were so widespread because of their repeated exposure to pathogenic environments,

Weismannism had a liberating potential. Alfred Russel Wallace (who was a Weismannian and an anti-eugenist) claimed that it was a "relief" to know, after Weismann, that all the "evil and degradation" of human history will leave no permanent traces once "a more rational and more elevating system of social organization is brought about" (1892).

A second point regarding knowledge production is vividly exemplified by Kroeber's use of Galton and Weismann to challenge the confusion of organic and superorganic in Lamarckian explanations (Kroenfeldner 2009; Meloni 2016b). It is on this point in particular that we need to reflect to see what sort of potential Durkheim saw in Weismann. The separation of heredity from individual lifetime acquisitions allowed Weismann to draw out three consequences of the utmost importance for the social sciences. The first was to radically separate the connection between biological and social development, making Spencerian social evolutionism impossible and driving a "wedge" (Peel 1971) between the evolution of life and that of society. After Weismann, social evolution as a whole is no longer there, but split into two. The second was to radically separate individual actions from their hereditary substance, freeing the individual from the yoke of their ancestors' deed, and making heredity a much less personal force—a generic one, as Durkheim clearly saw. The third consequence, in delimiting heredity to the germplasm, was to release the whole body (sexual elements excluded) and above all its environmental influences from hereditarian mechanisms, with general emancipatory effects for the sciences that aimed at studying this environmental and now extra-hereditarian dimension, as Kroeber saw better than anyone else. After Weismann, what connects human generations across time belongs to two utterly separated domains: an internal perpetuating germplasm, subject of biological and evolutionary investigations; and cultural, educational and social processes, now disentangled from the vicissitudes of biological heredity. Such drawing of boundaries could not be missed by someone like Durkheim who, as a good follower of Boutroux, was looking for epistemic fences to delimit and anchor each science to its own purified domain.

Durkheim as a Weismannian

There are only two citations of Weismann in Durkheim's work, to my knowledge. Both are in footnotes, the first in *Division of Labour* (DL, 1893), and the second in *Suicide* (S, 1897). This paucity of explicit references may justify the fact that all commentators have overlooked the significant way in which Durkheim borrows from the hard heredity revolution to make ontological

room for his transcendence of the social. My key thesis is not only that, as we shall see in more detail below, *Weismann supplies Durkheim with a powerful scientific companion to make the social transcendent, but more importantly that the structure of Durkheim's theory is entirely isomorphic to Weismann's*. Durkheim's dichotomy of society and individual maps perfectly onto Weismann's dichotomy of germplasm and transient individual bodies. Both challenge some form of empiricism in their own field. For both, it is not individual experience (contra Spencer, a common enemy) that makes general categories (Durkheim 1915, 13), such as society or heredity: the social and the germplasm have a flavour of immortality that is certainly not allowed in the individual. Beyond this morphological symmetry, Durkheim and Weismann have much in common: both portrayed themselves as initiator of an epistemological break in their disciplines; both were passionate boundary-makers (in the sense of Gieryn 1983, 1999), aiming to clearly demarcate a positively founded science from the vestiges of long-held opinions; both were great modernizers (in the sense of Latour 1993) who deployed a largely dichotomous vocabulary to restructure their scientific fields: any spurious element, be it individualism or Lamarckianism, had to be zealously rejected to achieve a purified view of social or biological heredity. Both had a profound faith in positive science and were aware they were situated at a critical juncture in their respective disciplines' transition to a more mature stage. Finally, in their political implications, they were very ambivalent creatures, whose legacy had the common destiny of being interpreted in opposite directions, conservative and progressive, romantic and positivist.

Even a superficial knowledge of both authors and their scientific context invites one to draw parallels. However, the critique has wholly overlooked any connection between the two: no references to Weismann can be found in any of the key scholarship texts (see, e.g., Lukes 1985; Mucchielli 1998). Among mainstream interpretations of Durkheim, we are told that he belonged to the 1890s' generation who "were nurtured in a Republican milieu and were influenced by neo-Lamarckian theory of evolution and heredity" (Fournier 2005, 60) but the way in which he used or rejected this milieu is not addressed. In his vast reconstruction of the discovery of the social (1998), Mucchielli uses the category of "antinaturalistic reaction" or "critique of biological determinism" to trace the evolution of sociology from the biological to the social, though never addressing whether this reaction could be done without any relationship to epistemic changes within biology itself. A few authors analyse Durkheim's theory of race (Fenton 1980; Lehmann 1995; Paligot 2006), which is connected to his view of heredity, but once again very scant details appear on Durkheim's knowledge of biology. Mainstream interpretations

recognize that some key notions in Durkheim were "strongly moulded by nineteenth-century biology and medicine" (Lukes 1982, 146; see also La Capra 2001), but we are left in doubt about the specific quality of this knowledge.

Among non-mainstream interpreters of Durkheim, there are a few authors who have looked more extensively (and bravely) at the importance of biological themes in the development of Durkheim's thought, challenging the stereotype of Durkheim's antibiologism. Hirst, for instance (1973), analyses in-depth the way in which Durkheim's sociology borrows from biological themes either in terms of metaphors or analogies. Although Hirst overlooks the influence of Weismann, he offers a convincing argument that Durkheim sees in Darwin (with his concept of random variations) the possibility to break away from the teleological progressionism of Lamarckians like Spencer. This is, in my view, a first important wound to the notion of a simple neo-Lamarckian influence on Durkheim because of the cultural landscape in which he was immersed. Robert Nye (1982, 1984) has highlighted the reliance of Durkheim on a Lamarckian repertoire in the early phases of his work, especially in Durkheim's 1888 article on mental pathology that features quasi-degenerationist themes. However, as Hawkins has pointed out (1999), Durkheim's later trajectory can be seen as a progressive break with this degenerationist, Lamarckian model. Hawkins emphasizes the emancipatory value of Durkheim's notion of non-dysfunctional criminality to depathologize its figure, but does not perceive Weismann as important in this abandoning of Lamarckian views of heredity. Finally, Gissis has written by far the most extensive treatment of the influence of Lamarckism on French sociology, and in this light, she analyses the relationship between Durkheim and biology (2003; see also Chap. 2 this volume). Gissis's argument is that the Lamarckian idiom was quintessential in grounding Durkheim's solidarist perspective and finding an alternative to the individualism and organicism endorsed by Worms and his supporters. According to Gissis, Durkheim and the Durkheimians naturally inclined to Lamarckian explanations given Lamarck's "methodological and epistemological (but not ontological) priority of the collectivity". Although Lamarckism was a very flexible conceptual repertoire, I am not entirely persuaded by this point. For instance, it is rather difficult to enrol Lamarckism entirely on the side of collectivity against individuality: Spencer, a Lamarckian *and* an individualist, provides the clearest counterexample here. But there is a more important argument in my view: that Durkheim understood the neo-Darwinian view of race and heredity (i.e. Weismann) exactly as a denial of individuality, which is instead central in a Lamarckian view of race (shared by Spencer) where race is made by the accumulation of *individual*

modifications. Looking at the chronology, Gissis's detailed analysis is in fact less at odds with my argument than it may seem. As Gissis recognizes, Lamarckism was first used and then abandoned by Durkheim; when the credibility of Lamarckism started to wane, Durkheimians moved away in search of other theoretical models (2003). My argument can be seen as an addition to Gissis: I want to highlight how important the understanding of Weismann was in 1890s for this transition out of Lamarckism. Even more subtly, the incorporation of Weismann did not just allow a move away *from Lamarckian biology* but, given its dualistic framework, *from biology as such*, thereby allowing the emergence of the social as something transcending the organic (and of sociology as its epistemologically bounded disciplinary domain).

Reading Durkheim's Weismann

The very few references to Weismann in Durkheim's work provide a partial justification for the gap in scholarship addressing Weismann's legacy and influence on Durkheim. However, those references that do exist are significant and strategic, not merely ornamental.

In fact, they are all the more important considering the time and disciplinary context of Durkheim's work: time, because Weismann's key compendium on heredity appeared in 1892, and was translated into French the same year (*Essais sur l'hérédité et la sélection naturelle*) just one year before *DL* was published; and discipline, given the usually angry responses that Weismann and neo-Darwinism obtained by sociologists (with Spencer and Ward being rather typical of this frustration). In this difficult context, it is evident that Durkheim looks at Weismann with eyes that are different from other sociologists or fellow nationals (who often depicted him as "a German menace to French biological research": Gissis 2003). Instead, Durkheim saw in Weismann a methodological brother-in-arms, as it is evident from the three key works of Durkheim in the 1890s.

Division of Labour in Society (*DL*, 1893)

The first reference to Weismann's work in Durkheim's writings appears in a footnote of *DL*. Weismann's work is introduced in the conclusion of chapter 4. To give some context, Chaps. 4 and 5 are a long detour devoted to the study of various "secondary factors" that have, beyond social causes as such, a role in explaining, hampering or speeding the division of labour. Chapter 5 in

particular is devoted to one of those non- or pre-social factors—heredity. The knowledge that Durkheim displays of the heredity debate in the mid- and late nineteenth century is more than erudite: it shows a real engagement with an issue that has important sociological consequences. Durkheim discusses at length key authors such as Prosper Lucas, Galton, Lombroso and de Candolle. Here Durkheim advances a notable argument, which will recur again in *Suicide*, about the waning of heredity both in human evolution and as a social institution. In this latter case, it is the progression of the division of labour to more complex and specialized forms that results in a decline of the social significance of heredity. As Durkheim writes, "the importance of heredity in the social organization of labour is all the greater when that labour is less divided up" (*DL*, 258). Before proceeding to an analysis of the reference to Weismann, two things are worth remarking here in Chap. 5. Firstly there are no ambiguities in Durkheim's usage of heredity as a modern concept: heredity is about the fixed and the innate, something that is opposed to the social environment and can't be generated by it, as in Erasmus Darwin or Spencer. The lesson of Galton, cited at length, seems to be fully internalised. Secondly, Durkheim makes a clear connection between the emergence of a contemporary science of heredity and its waning significance in society. It is just because heredity declines as an article of faith, replaced "by a faith that is almost its opposite" (i.e. the power of the individual in shaping his destiny), that we are now in the conditions to study it. Heredity, Durkheim adds, "did not come into the purview of science until the moment when it had almost vanished from that of belief. Yet there is no contradiction here. For what, finally, the common consciousness affirms is not that heredity does not exist, but that its importance is less great, and *science, as we shall see, reveals nothing that contradicts this view*" (*DL*, 250). I want to argue here that the "science" that reveals a diminution of the significance of heredity is exactly Weismann's "hereditarianism". It is precisely in the context of an argument for which "the individual is tied less strongly to his past" and "it is easier for him to adapt to new circumstances as they occur", that the reference to Weismann is introduced in a long footnote which ends the chapter.

What Durkheim says is extremely interesting. In spite of striking a (diplomatic?) note of cautiousness about the conclusive anti-Lamarckian evidence produced by Weismann, he is in no doubt about taking Weismann on board as a champion of his diagnosis of the diminishing power of heredity. This interpretation is brilliant, original and nearly unique at the time. Durkheim understands Weismann in a way that goes against the grain of how Weismannism was generally understood in right-wing, racial hygiene quarters (Weindling 1989): not race and heredity as fate, but exactly the breaking of

fate, because individual variations (the legacy of the past) no longer have direct impact on future generations. Weismann, the scientist of heredity par excellence, is also in sum the liquidator of the burden of heredity. Why? Because from Weismann, Durkheim gets a twofold lesson that will become increasingly relevant in his future work.

Firstly, that what is transmitted in biological heredity, after Weismann and contra Spencer, is not the individual type but *a broader and therefore vaguer* "generic type" (the germplasm for Weismann or the stirp for Galton). "Not so easily affected by individual variations, as has on occasion been supposed", Durkheim writes (*DL*, 268); this generic type implies that what heredity transmits is not the specific determinations resulting from individual actions and tendencies but *a generic substratum of faculties and propensities*. As a collective property of the race rather than the result of the individual actions, heredity is radically depersonalized. What results is that "the more indeterminate and plastic this [generic] type, the more also the individual factor gains ground" (*DL*, 268), making heredity's yoke lighter. The passage could have been stronger, as if Durkheim is just starting to realize the importance of this shift in the view of heredity. But it is clear that this paragraph has to be read against the background of a series of passages in the chapter where it is emphasized again and again that "what heredity transmits consists more and more in indeterminate predispositions, general ways of feeling and thinking" that only at the social level do they become then specialized "in a thousand different ways". Although the reading of de Candolle may have played a role in this interpretation, such a quintessentially sociological way of thinking is the natural ally of Weismann's view of heredity as being confined to the collective level and not affected by individual variations. One can compare several passages of *DL* to what Weismann himself writes to deny the inheritance of *a specific artistic talent*: "The Bach family shows that musical talent, and the Bernoulli family that mathematical power, can be transmitted from generation to generation, but this *teaches us nothing as to the origin of such talents*. (…) Gauss was not the son of a mathematician; Handel's father was a surgeon, of whose musical powers nothing is known; Titian was the son and also the nephew of a lawyer, (…) a man is not born a physicist or a botanist, and *in most cases chance alone determines whether his endowments are developed in either direction*" (1893a, 96–97, my italics).

It is this way of thinking in which the idea of a specific musical talent is dissolved and replaced by a broader faculty that put Weismann's thought very much in line with Durkheim's idea of a generic force of heredity that can then take many social forms. This Durkheim-Weismann line is obviously very different from a strictly Lamarckian view in which musical talent, as an inherited

characteristic, is passed interpersonally in families of musicians, as Spencer wanted. But it is also very different from the crude hereditarianism of the first generation of eugenicists who believed in a specific "wandering impulse" running in families of sailors (Davenport 1915).

However, there is a second lesson from Weismann, even more important than the first, contained in this dense footnote, a lesson that will become the true mark of Durkheim's hidden Weismannism. It is the key notion that between the individual and the "collective type" or race there is in Weismann *an ontological gap*, exactly the chasm existing in Durkheim, between the social and the individual. The passage is worth citing entirely:

> From another viewpoint also these theories [of Weismann] are of interest to us. One of the conclusions of our work to which we attach the most importance is this idea that social phenomena derive from social and not psychological causes. Also, the collective type is not the mere generalisation of an individual type, but on the contrary the latter arises from the collective type. For a different order of facts Weismann likewise shows that *the race is not a mere prolongation of the individual; that the specific type*, from the physiological and anatomical viewpoint, is not an individual type that has perpetuated itself over time, but that has its own course of evolution. Also the individual type has detached itself from the collective type, far from being its source. *His views are, like ours, it seems, a protest against the simplistic theories that reduce the composite to the simple*. (*DL*, 268, my italics)

Durkheim is here rejecting the empiricism of Spencer where categories are made by individual actions (see similarly Durkheim 1915, 13), and Weismann is doing exactly the same in his own field. The resonance between the two views is exceptionally vivid here: just as in Durkheim the social is outside the reach of individual, so in Weismann the germplasm is situated outside the reach of any "variation that takes place in individuals of the species". Exactly as in Durkheim the social transcends the will and consciousness of the individuals, so in Weismann the germplasm is a transcendent entity "on which individuals get attached as excrescences" (Ansell-Pearson 2003, 6). Durkheim certainly had in mind the following passage from William Platt Ball, a scientific popularizer quoted in the same footnote, who in his 1890 anti-Lamarckian text *Are the effects of use inherited?* made (correctly) a similar analogy to describe the new view of heredity:

> Galton compares parent and child to successive pendants on the same chain. Weismann likens them to successive offshoots thrown up by a long underground root or sucker. (Ball 1890, 66)

It is likely that this type of analogy struck more than one chord in Durkheim. As we shall see in *Suicide* in particular, this new view of heredity became a sort of implicit scientific legitimation for Durkheim's primacy of the social as ontologically irreducible to individual actions, a *sui generis* thing. This ontological gap was the same Weismann had in mind for his own theory of heredity where "all parts of the body do not contribute to produce a germ from which the new individual arises, but (…) on the contrary, the offspring owes its origin to a peculiar substance of extremely complicated structure, viz., the germ-plasm" (1893a, 11–12).

The Rules of Sociological Method (*R*, 1895)

Let us come now to *the Rules of Sociological Method* (1895). Here there are no explicit references to Weismann, nor is heredity a particular central focus of this classic book. Durkheim's conceptual engagement with heredity can be considered transitional in this work, between the central treatment it takes in *DL* (an entire chapter) and the critical analysis of race and heredity in *Suicide* which I shall address next. Nonetheless, in a text that is foundational in establishing boundaries between the social and other domains, and between sociology and other disciplines, from my hypothesis it should follow that this could not happen without any evaluation of the parallel status of the biological. As is well known, the *Rules* is the book where social facts are defined in their exclusivity, as a self-standing category on which the professional monopoly of the sociologist can be fully exercised. As Durkheim writes, there is "a category of facts which present very special characteristics", something that "cannot be confused with organic phenomena, nor with psychical phenomena", a "new species" to which the term social "must be exclusively assigned". These special facts are "consequently the proper field of sociology" (*R*, 52). This is a seminal moment for the emergence of the social as a purified category and it would be a significant challenge to my argument if this delimitation of the social could occur regardless of any take on biology.

Once again, radically overlooked by commentators, a passage in Chap. 5 clearly illustrates how Durkheim had, two years after *DL*, fully assimilated the Weismannian lesson. It is because of this incorporation that Durkheim, I want to argue, can establish a radically dichotomous mode of functioning between the social and the biological that breaks at its core any temptation to establish a synthetic social evolutionism as in Spencer. Let us offer a bit of context first. Chapter 5, the "Rules for the Constitution of Social Types", is the place where Durkheim lays out his "social morphology" aimed "to constitute and classify

social types" (*R*, 111). Here Durkheim introduces the key concept of social species as intermediate entities between the extreme nominalism of historians (with their "confused multitude of historical societies") and the realism of philosophers (with their "unique, although ideal, concept of humanity": *R*, 109). The notion of social species is foundational to the production of a system of social classification, a "complete scale of social types" starting from the simplest, the horde ("protoplasm of the social realm") and then, via a system of combinations and differentiation, the clan and more complex social forms and structures. The use of morphology in a taxonomic sense is obviously in analogy with its biological usage (Hirst 1973). Are we then back to organicist sociologies that can't distinguish the social from the natural? Durkheim seems to dance dangerously on the border of biological analogies, exactly in a text where the demarcation has to be neat and unambiguous. If morphology is a way of conducting research that is available to both sociologists and biologists, as commentators highlight (Lukes 1985), from where can a radical difference emerge? How can confusion be avoided in the use of the cross-disciplinary notion of species? As Durkheim recognizes, by using the notion of species we are moving on slippery terrain. As he claims, "there are social species for the same reason as there are biological ones. The latter are due to the fact that the organisms are only varied combinations of the same anatomical unity" (*R*, 116). This is obviously a situation of potential confusion, very much in need of a boundary that may help avoid any transgression of field. It is at this point that Weismann comes to hand. The passage is here worth citing in its entirety for the way in which it can dramatically separate social from biological species making use of the core anti-Lamarckian argument:

> "However, from this viewpoint, there is a great difference between the two domains. With animals, a special factor, that of reproduction, imparts to specific characteristics a force of resistance that is lacking elsewhere. These specific characteristics, because they are common to a whole line of ancestors, are much more strongly rooted in the organism. They are therefore not easily whittled away by the action of particular individual environments but remain consistently uniform in spite of the diverse external circumstances. An inner force perpetuates them despite countervailing factors in favour of variation which may come from outside. This force is that of hereditary habits. This is why biological characteristics are clearly defined and can be precisely determined". With the social domain, instead, things are radically different. As he continues: "In the social kingdom this internal force does not exist. Characteristics cannot be reinforced by the succeeding generation because they last only for a generation. (*Ils ne peuvent être renforcés par la génération parce qu'ils ne durent qu'une generation*). In fact as a rule the societies that are produced are of a different species

> from those which generated them, because the latter, by combining, give rise to an entirely fresh organisational pattern. (….) The distinctive attributes of the species do not therefore receive reinforcement from heredity to enable them to resist individual variations. But they are modified and take on countless nuances through the action of circumstances". (*R*, 116–117)

This text is extraordinarily dense and complex. It deserves to be analysed carefully to see the different ways the modern view of heredity is incorporated and used for Durkheim's own sociological goals. Firstly and more visibly, the difference between the biological and the social kingdom is made possible by the fact that inherited characteristics are impossible. The Weismannian lesson, still cautiously approached two years before, is now no longer in question, *at least for the social domain*. If acquired characteristics are heritable, as Spencer believes, the social would be coterminous with the biological, subject to the same regime of functioning: in biology as in culture, the next generation would inherit the acquisitions of the earlier one. Instead we have here two very distinctive domains. The first is a domain of biological perpetuation based on an inner force that is insensitive to external signals: in this first domain, it has to be noticed, Durkheim uses an ambiguous language of hereditary habits, but obviously he is referring to ideas of heredity as interiorized and hard, "not easily whittled away by the action of particular individual environments", "consistently uniform in spite of the diverse external circumstances" (*R*, 116). A few years later, genetics will come to occupy this space of unresponsiveness to external signals. Out of this kingdom of biological reproduction, dominated by the inertial force of ancestral heredity, a second domain—the social—emerges, that lacks this inner force and is completely determined by "the action of circumstances" (*R*, 117). Here, once again supporting a minority interpretation of Weismann, what Durkheim emphasizes in the destruction of use-inheritance is emancipation from the yoke of heredity: "the societies that are produced are of a different species from those which generated them, because the latter, by combining, give rise to an entirely fresh organisational pattern" (*R*, 116). This resonates profoundly with Weismann's own interpretation of his work, that is, that "the hypothesis of the continuity of the germ-plasm gives an identical starting-point to each successive generation" (Weismann 1893a, 168). To go back to the main point, what we have here is a polarized scenario, in which the force of heredity is confined to the biological, and the freedom of change at each generation becomes to trademark of the social. What is missing? Nothing important to our eyes, but a substantial certainty for nineteenth-century authors: the Lamarckian third way, an inner force of heredity, but shaped by the action of circumstances, a

plastic heredity. In destroying this third way, the link connecting the social and the biological is also destroyed. Two different stories can commence, no longer at risk of *liaisons dangereuses*.

Suicide (*S*, 1897)

With *Suicide*, I come to the third and last stage of the incorporation of the hard hereditarian revolution as a key scientific support for the transcendence of the social. *Suicide* is the book where the social, in its autonomy and self-standing authority, finds "a new and especially conclusive proof". From this point of view, *Suicide* does not present a new argument but puts the insights anticipated in *DL* and *Rules* on a firmer base. The two key intuitions emerging from the two previous books are confirmed with a higher level of confidence by Durkheim. To reiterate, these are: firstly, that heredity is narrowed, delimited and restricted to just the transmission of generic characteristics. It therefore loses the level of penetration and personalization given to it by the inheritance of acquired characteristics; secondly, that the social fully transcends individual deeds, exactly as race in Weismann's "positive science" stays with its individual members.

The first of these points is made repeatedly in the book on "the extra-social causes" of suicide. Heredity is not denied, so much as generalized to lose its penetrative force: "heredity", Durkheim writes,

> plays an important role; but it is no longer the heredity of suicide. What is transmitted is the general mental affliction, the nervous weakness of which suicide is a contingent result, though one always to be apprehended. In this case heredity has nothing more to do with the tendency to suicide than with hemoptysis in cases of hereditary tuberculosis. (*S*, 45)

The genericity of heredity is a key way to deny a direct and whole passage of "the tendency to self-destruction (…) from parents to children and which, once transmitted, gives birth wholly automatically to suicide" (*S*, 42) as in a hereditarian-psychological view of heredity. This is no longer possible because "what is transmitted is not the affliction itself but only a field such as to favor its development" (*S*, 43). A Lamarckian view would be open to a similar critique, whereby it is the personal experience of the previous generation that shapes the instinct of the next, making the relation between self-destruction in parents and in children more intimate. Once again, only hard heredity (in the sense of this less common reading of Weismann) may favour the secularization of heredity, making it a generic and less invasive force.

The second point repeats, in a more assertive fashion, the interaction with Weismann already highlighted in *DL*. It is worth following the text strictly because this is one of the key passages in the invention of Durkheim's sociologism, where Tarde is taken as the main target. Exactly at the end of this long passage, Weismann is called upon to offer scientific validation to the autonomy of the social as a transcendent, collective force. To offer again further context: this occurs in the chapter discussing the social element of suicide, part of Book II (*Social Causes and Social Types*). What Durkheim is arguing is that *Suicide* offers an empirical confirmation of his key intuition that the social is not merely a manner of speaking, an innocuous metaphor nor a cover for the reality of individual communication. The naïve common-sense view in which only individuals exist and the social is ethereal has to be entirely reversed: "The individuals making up a society change from year to year, yet the number of suicides is the same so long as the society itself does not change." The individual, not the social, is the transient reality; Tarde is the enemy here:

> It has been thought that this conclusion might be avoided through the observation that this very continuity was the work of individuals and that, consequently, to account for it there was no need to ascribe to social phenomena a sort of transcendency in relation to individual life. (*S*, 272)

However, this is not how things work. What Tarde would like to persuade his readers is that anything regarding the social is about personal transmission "from an individual parent, teacher, friend, neighbor, or comrade to another individual".

If we think of this model vertically, we can see how Tarde's interindividual approach is entirely isomorphic to the inheritance of acquired characters, where transmission is personal, from the experience of one generation to that of the next. This is for Durkheim the most flagrant misunderstanding of what the social (as a collective tendency) is; exactly as for Weismann, personal heredity is the misunderstanding par excellence of how heredity as a collective entity works. Both forms of transmission, the social for Durkheim and heredity for Weismann, have instead a "very special nature" (*S*, 272), which must be recognized in its entirety.

A few pages later, Durkheim recapitulates the theme of the whole chapter and finds a scientific validation (or at least, a companion) to this anti-Tarde strategy. Again, it is worth citing the passage in its entirety:

> Such a way of considering the individual's relations to society also recalls the idea assigned the individual's relations with the species or the race by contemporary zoologists. The very simple theory has been increasingly abandoned that the

> species is only an individual perpetuated chronologically and generalized spacially. Indeed it conflicts with the fact that the variations produced in a single instance become specific only in very rare and possibly doubtful cases. The distinctive characteristics of the race change in the individual only as they change in the race in general. The latter has therefore some reality whence come the various shapes it assumes among individual beings, far from its consisting simply of a generalization of these beings. We naturally cannot regard these doctrines as finally demonstrated. But it is enough for us to show that our sociological conceptions, without being borrowed from another order of research, are indeed *not without analogies to the most positive sciences*. (*S*, 285, emphasis added)

The first text Durkheim cites is Delage (*Structure du protoplasme*), a Lamarckian author, but the reference is specifically to the pages where Weismann is discussed. The second reference is explicitly to Weismann, "and all the theories akin to Weissmann's [sic]", Durkheim writes. Durkheim appears here like a solitary runner who raises his head at the end of a hard event to look for some support. Here he finds Weismann: no matter the concession to the criticisms of his fellow nationals ("We *naturally* cannot regard these doctrines as finally demonstrated"), no matter the denial of any subordination or weakness of its sociological empire ("*without being borrowed* from another order of research"), Durkheim is content to have found an analogy in the positive sciences for his sociological anti-empiricism.

Conclusion

In this chapter I have illustrated the strategic uses of Weismann's work in Durkheim. Although I am not claiming that the German embryologist was his only scientific inspiration to purify sociology, what Weismann certainly offered to Durkheim was a precious scientific ally to get rid of the empiricism of Lamarckian theory in which heredity resulted from the *accumulation of individual variations*. After reading Weismann, this Lamarckian view was seen by Durkheim as completely analogous to the various sociologies that understood the social as *accumulation of individual actions*. In a Latourian sense (1993), the *purification* strategy of Durkheim actually depended on *a (hidden) hybridization* with Weismann's biology.

It is obviously important to delimit this claim of a radical purification to Durkheim's own work, rather than the whole post-Durkheimian tradition (for instance, Mauss), or even the late Durkheim of *Elementary Forms* (1912) where the society–individual cleavage is somehow more nuanced. Nonetheless,

with all the necessary qualifications and caveats, my reading of a profound hybridization of Durkheim on the Weismannian stock may contribute to offer an alternative reading of the schism between the social and the biological, from which I started.

According to a mainstream historiography, which informs handbooks and teaching materials, the social sciences at some point broke with outmoded biologistic models, making themselves free for more sophisticated, non-organic ways of explanation. The social was finally discovered. However, why was this emancipation from outmoded ways of thinking possible, or even necessary, after a certain point? Historian Dorothy Ross argues, for instance, that, with reference to the American context, "from about 1880 to 1905 the social sciences did not appear to feel that their free borrowings [from biology] placed them under threat (…) After 1905 however there is evidence of greater sensitivity to, and defensiveness against, both biology and psychology, in the face of new currents within these subjects—Mendelian genetics" (1993, 100). The argument here is that (to limit my analysis at the relationship with biology), when the pressure from the biological got worse, after 1900 (when Mendel was rediscovered and, one can assume, eugenics started), the social sciences no longer felt comfortable sharing their epistemic premises with biologists. High tensions were emerging and a peaceful coexistence was now at risk. While the chronology of this interpretation is (more or less) correct, I think that the relationship between cause and effect is reversed. My article on Durkheim and Weismann, as my previous one on (Meloni 2016b), illustrates how the social sciences *were not put under any greater threat* by biological arguments when Mendel was "rediscovered", courtesy hard heredity. It is rather that *now for the first time the social sciences found a way out from biologism*. Why? Because courtesy of Galton, Weismann and genetics, biology made possible for the first time the circulation of a concept of heredity that was utterly separated from the social environment. In this way, the latter was freed from any direct connection with the biological. Heredity was secluded away in the germplasm (later, in the gene) becoming less invasive than in previous Lamarckian forms. It was now possible to *distinguish neatly and for the first time* between heredity and sociocultural transmission. Durkheim's sociological explanations of the reproduction of criminality in families is perfectly in line with what a geneticist like Thomas Hunt Morgan would say, three decades later, with regard to the epistemological shortcoming of the eugenicist's pedigree. For Durkheim "we cannot determine the relative contribution of heredity among all criminal vocations, (…) [If] the son of a thief becomes a thief himself it does not follow that his immoral nature is a legacy bequeathed him

by his father. To interpret the facts in this way we would have to be able *to isolate the effects of heredity* from those of circumstances, education, etc." (*DL*, 257, emphasis added). In 1925, Morgan wrote similarly that. "The pedigrees that have been published showing a long history of social misconduct, crime, alcoholism, debauchery, and venereal diseases are open to the same criticism [i.e., conflating biological and social heredity] from a genetic point of view; for it is obvious that these groups of individuals have lived under demoralizing social conditions that might swamp a family of average persons. It is not surprising that, once begun from whatever cause, the effects may be to a large extent *communicated rather than inherited*" (quoted in Allen 2011, 201–2, emphasis added). What seems a sociological gift, that is, distinguishing communication from heredity, is in fact also perfectly in tune with the hard heredity revolution promoted by Weismann that culminated with genetics. Morgan named it "the two-fold method of human inheritance" (in Allen 2011), which clearly converges with Durkheim's view of a homo duplex (*S*, 171: "man is double") and Kroeber's dualism between the organic and superorganic. Rather than being enemies, sociology and genetics have shared a certain epistemic contiguity in the twentieth century, where *a radical separation of heredity and heritage* was made possible (mostly via Weismann). Whether this will continue to be the case in the current century is a different matter that I cannot address in the limited space of this chapter.

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Notes

1. This section reproduces a few passages of my *Political Biology* (2016), chapter 2.
2. I am well aware that this simplifies a complex debate on the transition from a speculative view of heredity in Weismann to the experimentalism of geneticists. This, however, has to be left aside in this chapter.

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4

Biology, Social Science, and Population in Late Nineteenth- and Early Twentieth-Century Britain

Chris Renwick

In his landmark book, *The Growth of Biological Thought* (1982), the eminent German-American biologist, Ernst Mayr (1904–2005), argued that there was a significant difference between popular perceptions of scientific development and the deeper shifts that were characteristic of real change.

> Discoveries are the symbol of science in the public mind. The discovery of a new fact is usually easily reportable, and thus the news media also see science in terms of new discoveries. When Alfred Nobel wrote out the conditions for Nobel prizes, he thought entirely in terms of new discoveries, particularly those useful to mankind. Yet to think of science merely as an accumulation of facts is very misleading. In biological science, and this is perhaps rather more true for evolutionary than for functional biology, most major progress was made by the introduction of new concepts, or the improvement of existing concepts. Our understanding of the world is achieved more effectively by conceptual improvements than by the discovery of new facts, even though the two are not mutually exclusive. (Mayr 1982, 23)

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For this reason, Mayr did not think the widespread acceptance of the idea of evolution since the publication of Charles Darwin's *On the Origin of Species* (1859) was necessarily the most important thing to happen to modern biology. According to Mayr, a closely related, hugely significant, but underappreciated development was a transformation of the way biologists understood the relationship between individual organisms and species or groups. The shift away from what he called "typological thinking", a way of interpreting the world dating back to Plato, was slow. But after almost a century its successor, "population thinking", had revolutionised how we make sense of what is around us (Mayr 1982, 35–47).

Despite being one of the most famous arguments in the field of history and philosophy of biology, few scholars have connected Mayr's take on biology after 1859 with similar and contemporaneous developments in the social sciences. Indeed, even the eminent British sociologist and enthusiast for the population idea John H. Goldthorpe (2016, 10–11) has admitted to being unaware of Mayr's work until relatively recently. This oversight is surprising, not least because of the enthusiasm amongst social scientists for the work of the French philosopher and social theorist Michel Foucault, who wrote a number of influential pieces on the emergence of population as a concept since the early modern era. Population was important for Foucault (2007) because he thought it enabled new forms of knowledge and was therefore an important component of governmentality during the past 400 years. To be sure, some Foucauldians have explored the role of population in social science since the late nineteenth century. Thomas Osborne and Nikolas Rose (2008), for example, have argued that the early 1900s saw "population" become a common investigative problem in British sociology. In the process, population provided what the French sociologist Bruno Latour (1987) calls the "set up" in which "inscription devices"—ways of making objects knowable—became meaningful for many social scientists, whether they now realise it or not.

Not even Rose and Osborne, however, have considered the relationship between developments in social science and Mayr's account of biology. Yet if population entered both biological and social science at around the same time and brought about similar changes, surely they had something in common? This chapter explains how the emergence of population thinking in biology and social science during the late nineteenth and early twentieth century were indeed related, with research at the intersection of biological and social science helping to construct shared ideas and practices. Eugenics is a topic that casts a long shadow over this topic but, as we will see, it was only part—albeit a very important part—of a story featuring a space that some researchers

considered to be a genuine third sphere between biological and social science. Although that sphere broke down, its influence has continued to be felt right through to the present, not only by supplying some of the intellectual and institutional materials on which the post-1945 disciplinary ecology was built but also helping change the way we have come to know the biological and social worlds.

Nineteenth-Century Origins

According to Mayr, typological thinking was a form of essentialism that had a vice-like grip on the Western mind from Plato through to the twentieth century. As Jonathan Hodge (1996)—a former student of Mayr's—has explained in his elaborations on and critiques of Mayr's argument, the turning point for typological thinking came in the Middle Ages when the natural philosopher and theologian Thomas Aquinas synthesised Plato's notion of a craftsman creating forms according to an abstract recipe or set of instructions with the Judeo-Christian account of creation in six days, as set out in Genesis. For thinkers working within the Thomist paradigm, the universe was a place in which there were a fixed number of forms. All variation, including that in the organic world, was a consequence of the reproductive process failing to replicate divine instructions precisely. The underlying truth about the organic world was discontinuity between beings in space and time (Mayr 1982, 38).

Although Mayr admitted that his account of philosophy and intellectual life before *On the Origin of Species* was monolithic and contained obvious straw men, he was adamant it also illustrated a number of key points. The most important, according to Mayr, was that Darwin's contemporaries failed to grasp his most challenging idea: that species are merely well marked varieties; hence why there was what Peter Bowler (1988) called a "non-Darwinian" revolution during the late nineteenth century, when there was great enthusiasm for the idea of evolution but the mechanism of natural selection was widely rejected. On Mayr's account, the radical consequence of Darwin's worldview, which took until the second quarter of the twentieth century to emerge fully, was "population thinking". This was profoundly different from typological thinking because it stressed

> the uniqueness of everything in the organic world. What is important for [population thinkers] is the individual, not the type. They emphasize that every individual in sexually reproducing species is uniquely different from all others, with much individuality even existing in uniparentally reproducing ones. There is no

> 'typical' individual, and mean values are abstractions. Much of what in the past has been designated in biology as 'classes' are populations consisting of unique individuals. (Mayr 1982, 46)

As Mayr conceded, however, Darwin was not the only contributor to the construction of this way of thinking. Statisticians, whose technical and conceptual innovations made sense of phenomena that seemed to fall short of law-like behaviour, were also important (Hacking 1990). Darwin's cousin, Francis Galton (1822–1911), was a central figure in these developments. Inspired by *On the Origin of Species*, Galton had devoted himself to the study of heredity and was convinced, as he explained in *Hereditary Genius* (1865), his first book on the subject, that intellectual ability was passed through families. In addition to acquiring huge amounts of new data, Galton developed a large number of hugely important statistical tools, including regression analysis and correlation. These endeavours left a significant legacy for a wide range of disciplines (Renwick 2012, Chap. 2; Gillham 2001; Hacking 1990, Chap. 21).

Two aspects of Galton's work stand out as significant when it comes to the development of population thinking in biology. The first is that he was a saltationist—a believer in evolution through large jumps rather than gradual steps—meaning he also had one foot in the past, according to Mayr's account. The second is that Galton only made significant and rapid progress in his quest to discover the laws of inheritance once he had given up on the questions and methods that concerned most biologists during the three decades after the publication of *On the Origin of Species* (Renwick 2012, Chap. 2). Frustrated by his failure to discover the physiological mechanisms behind heredity, Galton had redoubled his efforts with statistics, specifically on human and social data, leading eventually to his landmark book *Natural Inheritance* (1889), which was a formative influence on both the biometricians and the Mendelians, whose split defined biology for the first quarter of the twentieth century (MacKenzie 1981, Chaps. 5 and 6; Provine 1971, Chaps. 2 and 3).

Equally important, however, was the way Galton's work helped establish population as a bridge between biology and the social sciences. Confronted by a constantly expanding body of information about society and the economy, social researchers faced many of the same struggles as biologists when it came to relating individuals and groups. The poor, for example, had always been a visible part of society. But as people moved from the countryside, where they had toiled on the land, to the cities like Manchester, Leeds, and Birmingham—where they searched for better paid but less secure work in mills and later

factories—the dynamics of the problem changed. Social investigators had all manner of questions about the lives of people who crowded into overflowing and unsanitary slums during the nineteenth century, not least whether industrial society had created more poverty than earlier social and economic systems.

The site for the most important developments in connection with these problems was social surveying—the field many sociologists and historians consider the UK's only major contribution to the sociological tradition (Abrams 1968; Bulmer 1985; Platt 2014). The usual starting point for histories of social surveying in Britain is the Liverpudlian businessman, Charles Booth, who set out in the mid-1880s to establish the extent of poverty in the British capital. Bankrolled by his private fortune, earned from the shipping industry, Booth's project employed a team of investigators and resulted in the 17-volume *Life and Labour of the People of London* (1889-1902) with its famous colour-coded maps that showed where different social classes could be found. Drawing on testimony from public officials, his investigators' observations of day-to-day life in London's slums, and information about the cost of living—Booth claimed that 30% of London's residents were poor. Yet this was a static picture of life in late nineteenth-century Britain with rigid social classes, populated by people with particular types of character. An alternative account was offered by Seebohm Rowntree, a member of York's famous Quaker and chocolate-manufacturing family, who carried out his own survey eventually published as *Poverty: A Study of Town Life* (1901) (Briggs 1961). Whilst Rowntree utilised the concept of a "poverty line" and drew a distinction between "primary" and "secondary" poverty, his most important innovation was the "poverty cycle" (Rowntree 1901, Chaps. 4 and 5). Thirty per cent of the population might have been poor at any one time, Rowntree argued, but they were not always the same people. Events from illness, to the birth of children, to old age—dragged people below the poverty line only for them to rise above it again once they had passed (Rowntree 1901, Chap. 5). The British population was a dynamic place where individuals' life cycles were often out of sync with social structures.

Although *Life and Labour of the People of London* and *Poverty: A Study of Town Life* did not seem to make much use of biology or biological ideas—neither Booth nor Rowntree ignored biology, whatever the impression one might get from their status as founding figures in the history of thinking about the structural causes of poverty. Both saw character and social status as having a natural, if not wholly determinate, connection of some kind, which was unsurprising, given the easy flow of ideas between biological and social science during the late nineteenth and early twentieth centuries. Indeed, when Galton

(1909, 8–12) explained his ideas about improving the human race to wider audiences, he often took Booth's classification of London's population as a reference point and suggested ways in which their findings overlapped. The big question, however, was who owned the sphere where biological and social science intersected. For many people, Galton's eugenic programme seemed like the most obvious starting point, when it came to looking for an answer.

Biosocial Science and Population During the Early Twentieth Century

Population entered scientific and political discussions as a problem and remained a prominent issue for around half a century in two different forms (Renwick 2016a; Soloway 1990). During the early 1900s through to the late 1920s, most commentators were focused on overpopulation: the old Malthusian idea that the total number of people in the country was growing faster than the UK could handle. With a century's worth of census data showing that the number of people in Britain had increased from around 7 million to more than 30 million, these observers thought they had plenty of evidence to support their concerns about the country's future economic and social stability. In so doing, they connected population growth with Booth and Rowntree's studies of poverty, as well as the myriad of more impressionistic accounts of life in the slums, to argue that an increasingly large section of society was not cut out for the contemporary struggle for existence. As they did so, a new biologically tinged language also began to spread in wider culture. Words such as "degeneration", which had been given its modern meaning by the biologist E. Ray Lankester in the 1880s, became popular as some people imagined Britain as a social organism that was slowly but surely becoming less well adapted to the conditions of modern industrial life (Lester 1995; Pick 1989, Chaps. 6 and 7).

The British eugenics movement, which had floundered during Galton's early efforts to promote his project, also benefitted from these ideas and events. The Galton Laboratory and chair in National Eugenics at University College London were both founded within a few years of the Eugenics Education Society at the end of the first decade of the twentieth century, giving some people hope that a new era of social investigation and policy making was on the horizon. Yet, interest in the connection between overpopulation and social problems was broader and more complex than we might suspect from the family pedigrees that captivated so many of the founding members of the Eugenics Society (Mazumdar 1992, Chap. 2). Long before he was famous for theories about total aggregate demand and counter cyclical spend-

ing, for instance, John Maynard Keynes (1919, Chap. 2; 1923) argued that Britain's struggles with unemployment might be a consequence of there being too many people in the country (Toye 2000).

By the mid-1930s, though, the focus for discussions about population had shifted. In part, change was generational. Biologists and social scientists whose careers began to take off after the First World War had new priorities. One was to tackle the problems they saw as corrupting eugenics—a project they agreed with broadly but struggled to reconcile themselves with thanks to the more outlandish pronouncements of members of the Eugenics Education Society. Scientists including Julian Huxley, Alexander Carr-Saunders, and J. B. S Haldane, who became known as "reform" eugenicists, were certain that biological knowledge was a source for social progress but they worried deeply about the reliability of the family pedigree methods that underpinned demands for things like forced sterilisation of people perceived as threats to national "stock" (Kevles 1984, Chap. 11; Soloway 1990, Chap. 8). Reform eugenicists wanted biosocial science to be sophisticated and modernising, and they tried to build a project that changed the problems that people worried about as well as the tools they used to investigate them (Renwick 2016).

Having analysed the data on population growth in greater depth, this new generation of biological and social scientists argued that the real concern was under, not over, population. They pointed out that population growth had been caused almost entirely by increased life expectancy, which they suggested had obscured another worrying trend: a declining birthrate (Szreter 1996, Chaps. 6–8). If the young were not having children at the rate their parents and grandparents had, then the country would reach a point where population numbers fell off a cliff edge, perhaps declining to as little as four million people by 2035 (Charles 1935, 6; see also Charles 1936). Eugenicists and social reformers needed to stop fixating on what were known as negative eugenic policies, which aimed to restrict or prevent breeding among specific groups, and instead focus on positive eugenics, which would encourage people to have more children.

In this respect, population provided a meeting point for biologists worried about reproduction patterns, and social scientists who were interested in the capacity of economic and social structures to support those within them. As a consequence, fertility was one of the most important points of intersection between biological and social science. The major focus for many researchers was differential fertility: divergences in rates of reproduction between social classes (Soloway 1990; Szreter 1996). Karl Pearson, Galton's biographer and the first Professor of National Eugenics at UCL, was among the first to try to make sense of this problem as part of his project to make formal statistical

sense of Galton's various claims about heredity (Pearson et al. 1899; Porter 2004, Chap. 9). The thinker who made the subject their own, however, was the Cambridge-educated statistical geneticist Ronald A. Fisher (1890–1962), who spent the most important 14 years of his career at the Rothamsted Experimental Station in Hertfordshire (Box 1978). Building on conceptual and technical insights, first aired in a paper published shortly after the First World War (Fisher 1918), Fisher's most celebrated contribution to biology was *The Genetical Theory of Natural Selection* (1930), a book that bridged the gap between the warring biometricians and Mendelians by demonstrating a mathematical relationship between the gradualism of Darwinian natural selection and the discontinuity of Mendelian genetics.

Yet, as contemporaries who reviewed the book, not to mention historians who have studied it since, noted, one of the most striking things about *The Genetical Theory of Natural Selection* is the final third, which is about human evolution and eugenics—something that highlights the inseparability of biology, society, and politics during the period in question (Hodge 1992; Esposito 2016). In a technical exposition of an argument familiar to members of the Eugenics Society—an organisation he was an enthusiastic and leading member of—Fisher claimed that fertility was not constant but varied, first because of social conditions but then because it was inherited (Fisher 1930, Chap. 9; Mazumdar 1992, Chap. 3). These dynamics were one of the important reasons civilisations declined, he claimed, and if the large numbers of middle class and professional people who put off starting a family until they had made progress in their chosen careers was anything to go by, Britain would be the next one to fall by the wayside (Fisher 1930, Chaps. 8–12). High fertility would become an insurmountable barrier to success unless the country's social and economic structures were reformed in light of biological knowledge.

Although Fisher's work was highly influential and central to the formation of what Julian Huxley (1942) called the "modern evolutionary synthesis", Fisher's arguments about fertility, as well as the assumptions that underpinned them, were widely contested, most notably by his great rival Lancelot Hogben (1895–1975). A Cambridge-educated biologist, a Quaker, and a socialist who was married to fellow radical and demographer Enid Charles (1894–1972), Hogben first came to prominence during the early 1920s when he moved to the University of Edinburgh. Along with Huxley and Francis Albert Eley (F. A. E.) Crew, Hogben became a central figure in the campaign to encourage biologists in Britain to embrace experimental methods (Erlingsson 2009a, b). After a spell abroad, first in Montreal and then Cape Town, Hogben returned to Britain in 1930 when Beveridge, the director of the London School of Economics (LSE), invited him to lead a new Rockefeller Foundation funded

interdisciplinary project at the college: a department of social biology (Renwick 2014).

Hogben's main aim at the LSE was to integrate his laboratory-based work on biological mechanisms, through which he and Charles helped make the *Xenopus* clawed frog a model experimental organism in biology, with social science population research (Renwick 2014; Tabery 2014, Chap. 2). In so doing, he and his colleagues, including his doctoral students, Charles, and the eminent German Jewish refugee Robert Rene Kuczynski (1876–1947) had a clear target. As Hogben made clear in *Genetic Principles in Medicine and Social Science* (1931), he believed eugenicists vastly exaggerated the amount that could be attributed to genes, especially when it came to things like intelligence, which were not only woolly concepts but also measured with problematic tools. The environment had a huge role to play too, meaning those who claimed the working classes were inherently inferior were being guided by prejudice more than sound scientific reasoning (Renwick 2014, 2016; Werskey 1978, 60–6 and 101–14).

As James Tabery (2014, Chap. 2) has shown, Hogben and Fisher's disagreement was more complex than whether to attribute most importance to nature or nurture in an organism's development. Fisher's statistical innovations were designed to quantify the contribution of both genes and environment as causes of variation. In this respect, he had genuine reform eugenics credentials. But merely attributing a causal role to the environment was not good enough for Hogben, who argued that the interaction of nature and nurture was a distinctive and crucial cause of variability (Hogben 1931, 99-103; Tabery 2014, Chap. 2). Fisher thought this interaction was simply background noise—something relatively unimportant and safe to ignore—and he grew frustrated with Hogben's insistence that any proposed connection between eugenic policies imposed on individuals and improvement at the population level be demonstrated experimentally. The result was what Tabery (2014, 36) calls an "explanatory divide", akin to Thomas Kuhn's (1962) "paradigms" or Ian Hacking's (1992) "styles of reasoning", which had important consequences for the future of reasoning about gene/environment interactions in biology.

Population, Politics, and Society

In addition to being an argument about the causes of variability in populations, Hogben and Fisher's argument was also a deeply political dispute that was emblematic of the stakes when it came to relating individuals and society

in a biosocial context. Their divergence reflected the political meanings of individuals' choices in scientific theories and practices. Whilst Hogben was a radical socialist pacifist who had been imprisoned as a conscientious objector during the First World War, Fisher was much more conservative and had volunteered to fight in 1914 but been rejected because of his poor eye sight (Tabery 2014, Chap. 2). It was therefore little surprise that one was more willing than the other to ignore issues that complicated the case for eugenic policies. Yet, these individual differences were embedded in a broader intellectual ecology in which fundamental questions about the kind of society people wanted to live in were deeply contested. Perhaps surprisingly, these debates and the ideas that stemmed from them were important sources of inspiration for those who helped construct the reforming ideologies and movements, including neoliberalism and social democratic welfarism, that dominated politics during the late twentieth century.

An underappreciated source for many of these changes was the Oxford school of biology, through which some of the most important population thinkers passed during the early twentieth century. Home to the marine biologist Alistair Hardy (1896–1985) and the zoologist Vero C. Wynne-Edwards (1906–97), and therefore particularly strong in zoology, biology at Oxford had helped keep Darwinism alive among evolutionists and natural scientists during the late 1800s and early 1900s, meaning adaptationist and selectionist ideas were a key part of the intellectual environment (Depew and Weber 1995, 320–4). A particularly strong strand in Oxford biology was the effort to understand the relationship between individuals and groups from genetic, cultural, and behavioural perspectives. A shared idea was that things like behaviour and customs are not only selected for but also capable of regulating wider aspects of group experience. On the one hand, this meant Oxford biologists believed behaviour was something that could produce germinal change; on the other, it meant they thought customs, traditions, and other social factors helped determine things including population size.

Along with Julian Huxley, whose most notable contribution to the modernising project in biology and biosocial science was his population-framed argument that race is a political not a biological category, Alexander Carr-Saunders (1886–1966) was one of the leading figures in biosocial science with a background in biology at Oxford (Huxley and Haddon 1935; UNESCO 1952). After studying and teaching at the university during the first decade of the twentieth century when he specialised in zoology, Carr-Saunders headed to University College London (UCL) where he learned about biometrics from Pearson and become involved with the Eugenics Education Society, as well as the university settlement at Toynbee Hall in London's East End, where a host of

social reformers, from the economist William Beveridge to future Labour Prime Minister Clement Attlee, had learned about the social issues they were keen to solve. From that point onwards, and after a short spell when he considered a career in law, Carr-Saunders became known as a social scientist working at the intersection with biology. In 1923, he was appointed the first Charles Booth Professor of Social Science at the University of Liverpool—another British university that was able to expand during the interwar years thanks to financial support from the Rockefeller Foundation (Ahmad 1987; Seim 2013).

Like many of his contemporaries, Carr-Saunders' initial interest in biosocial science was spurred by eugenics, about which he wrote the Home University Library volume (Carr-Saunders 1926). No doubt thanks in part to his association with Pearson, who maintained Galton's scepticism of the Eugenics Education Society's populist activities, Carr-Saunders became a leading reform eugenicist. Aligning himself with Huxley, Charles, and other scientists who were warning about the spectre of population decline, Carr-Saunders' carved out a reputation for himself as an expert on social structure who had a significant interest in bringing those interests to bear on social policy and politics (Osborne and Rose 2008; Renwick 2016). Embracing the cause of positive eugenics, he told reforming members of the renamed Eugenics Society that they would not make any progress towards their ultimate goals until they stopped worrying about individuals, particularly individuals they believed to be spreading bad genes or traits, and became population thinkers instead (Carr-Saunders 1935).

Drawing on what he had learned at Oxford, he developed an historical explanation of change at a population level that took environment seriously. In *The Population Problem* (1922), the book that established his reputation as a biosocial thinker and earned him the chair at Liverpool, for example, he presented society as a complex mix of interrelated and shifting parts, including economic, psychological, anthropological, and biological elements, through which traditions as well as genes were altered, stabilised, and perpetuated through time. Social groups had different customs, he argued, and these were crucial in explaining why some succeeded and others failed in a process of constant competition. Central to his account was the idea that customs and traditions determined population size within a given society. More specifically, Carr-Saunders explained that success was associated with forms of social organisation that were capable of maintaining a population of an optimal size. A good society was one where people reproduced at a rate high enough to sustain its existence and enable all its members to stake a claim to its rewards but not so high that its natural and social resources would come under too much pressure.

Building on this foundation, Carr-Saunders became renowned for his expertise when it came to questions about social structure and social reproduction. He co-wrote a hugely important study of the professions in Britain since the medieval era and entered a long-term intellectual and administrative collaboration with his University of Liverpool colleague David Caradog Jones, which began with a statistical survey of England and Wales and went on to include a hugely important but now neglected survey of Merseyside (Carr-Saunders and Wilson 1933; Carr-Saunders 1926; Carr-Saunders and Jones 1927, 1937; Carr-Saunders et al. 1958; Jones 1934). Carr-Saunders also succeeded Beveridge as the director of the LSE in 1937, where he remained for the next 28 years—a position that cemented his status as one of his generation's most important and influential, albeit now largely forgotten, social scientists.

One of the most notable thinkers to fall under Carr-Saunders' influence was the Austrian economist and author of *The Road to Serfdom* (1944), Friedrich Hayek (1899–1992)—his colleague at the LSE for more than a decade. As a staunch individualist, Hayek struggled to explain how order appeared and was maintained in social systems, especially when it came to questions about continuity and change in ideas, beliefs, and actions over time. Carr-Saunders' account of how customs were selected at the group level turned out to be extremely useful. Drawing on *The Population Problem* as well as Wynne-Edwards' work, Hayek developed a theory of cultural evolution in which regularities in behaviour were produced by competition between groups. A committed liberal, Hayek argued that customs most likely to produce wealth and freedom, such as respect for private property, were advantages in that process. The result when everything was functioning properly was the "spontaneous order" he wrote about during the two decades after the Second World War (Hayek 1952, 1967, 1973; Angner 2002; Beck 2012, 2016). Unlike Carr-Saunders, however, Hayek saw these things not as means of restraining a population and its resources, but as mechanisms of constant expansion (Beck 2016, forthcoming).

Hayek's individualist political economy, which now goes by the name "neoliberalism", failed to capture the imaginations of politicians in Europe and North America during the third quarter of the twentieth century, when unfettered free markets seemed a long way from the answer to the problems confronting countries that were recovering from war and the Great Depression that had preceded it (Mirowski and Plehwe 2009; Stedman-Jones 2012). Yet Carr-Saunders' fingerprints were also on the social science that underpinned the policies and ideas that did find favour among policy makers after 1945. As director of the LSE during the 1940s

and 1950s, Carr-Saunders kept the college going when war brought a dramatic decline in student numbers, a forced relocation to Cambridge, and the secondment of faculty and then helped rebuild its staff afterwards (Dahrendorf 1995, Chap. 6). In so doing, he made sure a group of social researchers who were crucial for the development of sociology in the UK during the two decades after the Second World War remained in London. David Glass (1911–78), for example, had been employed at the LSE before 1939, first as Beveridge's research assistant and then in the department of social biology (Renwick 2014, 2016a). When he returned from government service, Glass quickly became one of the standard bearers for the quantitative methods and approaches that many commentators have taken as characteristic of British sociology, in particular through *Social Mobility in Britain* (1954b), the first substantial investigation of the subject in the UK. Of the debts he owed Hogben, his former boss, whom he thanked in the preface of *Social Mobility in Britain*, the population perspective was the most obvious. Glass and his 11 collaborators presented a detailed picture of social structure and stratification, tracing trends and movement across several generations. As he explained in his introduction, this approach was motivated by a belief that a better understanding of social structure and the role of institutions, in particular educational institutions, in facilitating movement through it were a prerequisite for policy makers who claimed to be interested in constructing a society in which promises of democratic participation meant something for everyone (Glass 1954b, 27-28).

Also among Glass' contemporaries at the LSE was Richard Titmuss, who rose to the position of professor in the department of social administration—one of the leading examples of the distinctively British field of social policy—and became one of Britain's leading socialist intellectuals during Carr-Saunders' directorship (Reisman 2001; Oakley 2014). A former actuary who left school at 14 with no formal academic qualifications, Titmuss made his name with *Poverty and Population: A Factual Study of Contemporary Social Waste* (1938) in which he turned the skills he had learned in the insurance industry to social, economic, and health problems. Linking his findings with studies of intelligence levels across different social classes, Titmuss argued that high mortality among the working classes was not a natural occurrence but a product of environmental differences. British social and economic organisation was incredibly inefficient and the consequence was not only suffering for those at the bottom but also squandered human capital. Something better than institutionalised economic selfishness and protectionism had to be possible, Titmuss argued—a point he picked up in his most famous book, *The Gift*

Relationship: From Human Blood to Social Policy (1970), in which he demonstrated how Britain's system of anonymous blood donation by volunteers was more efficient than the American insistence on payment.

Conclusion

The ideas associated with Hogben, Carr-Saunders, Glass, and Titmuss during the middle decades of the twentieth century were in essence social democratic engineering. The contrast between them and the likes of the neoliberal Hayek was not to do with flattening out society and eliminating differences; rather, it was down to Hayek's refusal to accept state intervention that aimed to produce specific outcomes, such as the movement of people through a hierarchical but dynamic social machine. It was for this reason that Hogben (1937), Titmuss (1938), and others frequently used the word "wastage" to describe what was wrong with societies where people were trapped in classes with wildly different basic outcomes and expectations. They believed that individuals were not continually reproduced according to a class archetype and that it was important that society did not squander talent and ability that was available to it. To be sure, these social investigators were inspired by ideas about social justice that had become popular in left and centre-left politics over the course of the previous 100 years, particularly since the rise of the Labour Party during the first two decades of the twentieth century. But, as we have seen, politics of this kind was also entwined with new ways of connecting biology and social science, with "population" as the bridging concept between the two.

The biosocial roots of these ways of understanding society are important for a number of different reasons. As Mayr argued, conceptual shifts often pass under the historical radar but are frequently more significant than simple discoveries, primarily because changes in the way people think can transform not only what they know about particular things but also how they come to know them in the first place. In this respect, whilst population thinking facilitated new ideas and practices in the biological and social sciences, it also opened up a range of new intellectual frontiers and possibilities. Indeed, as Osborne and Rose (2008) argued in their study of Carr-Saunders, the emergence of the population frame is crucial when it comes to the political implications and entanglements of those disciplines—as scholars inspired by Foucault's (2008) analysis of biopolitics have frequently suggested. Yet as the eventual settlement of thinkers like Carr-Saunders and Glass in British social, rather than biological, science

after the Second World War suggests, the dual roots of the population idea can often be obscured, giving a false impression of the relationship between biology and social science. It is sometimes tempting to see similar developments in the biological and social sciences as either parallel or evidence of superficial intellectual exchange—something that can be boiled down to the occasional shared metaphor or idea. As we have seen, however, there was a much stronger relationship between biological and social science in late nineteenth- and early twentieth-century Britain. This relationship included not only common questions, topics, and assumptions but also, as population research during the 1930s showed, a genuine shared space of biosocial investigation in which there was no clear ownership of problems, issues, or methods, let alone the results. That short-lived sphere may not have become quite what Hogben and others may have hoped, but it was a hugely productive site of research that bequeathed ideas and personnel of great import to both the biological and social sciences.

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5

The Concept of Plasticity in the History of the Nature-Nurture Debate in the Early Twentieth Century

Antonine Nicoglou

Introduction

At the beginning of his book *Émile, ou de l'éducation* (*Émile, or Treatise on Education*), Jean-Jacques Rousseau claimed "we shape plants through cultivation, and people through education," illustrating the two meanings of the word "culture" (Rousseau 1762). In a general sense, culture means either "to develop," for instance, a field, but it applies also to the mind. Therefore, "culture" has come to designate a set of collective norms but it is used to denote also the particular refinement that distinguishes one individual from his fellows. In English, the idea of moral training and rearing (or bringing up) is given by the word "nurture," which is itself borrowed from the old French (late eleventh century) words "*nurture or nurtoure*" ("*nourriture*" in modern French) meaning food. The alliterative expression "nature and nurture" in English has been famously in use since the Elizabethan period (sixteenth century). *The Tempest* by Shakespeare is often quoted to be the first reference to the nature versus nurture debate and its use, which may have influenced Francis Galton's understanding of the terms in his work.

Today, the phrase "nature and nurture" has become associated with the relative importance of innate qualities as compared to personal experiences in causing individual differences, initially in behavioral traits, even though it has

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now extended to all kinds of traits including morphological and/or phenotypic traits (to use the common biological terminology) of an individual of a species. From a general point of view, two types of issues are now linked to the nature nurture debate: what matters for defining the universal aspects of human nature or characteristics of living beings (depending on whether one focuses on the human or on living organisms in general) and what matters for understanding the causes of individual differences (note that most of the discussions have focused in biology on this second type of issue).

In the first section, I detail the origins of the opposition in Galton's work and the "naturalization" of what had been before, mainly seen as a lyrical-philosophical figure of speech. In a second section, I show how the emergence of genetics led to a reformulation of the nature-nurture opposition itself linked to a redefinition of inheritance. In the last section, I examine how the concept of plasticity has been used in psychology, philosophy, and biological sciences to depict the interplay between nature and nurture. I show that despite the fact that even though some early geneticists started to use the concept of plasticity as a way to bring together the two parties of the opposition in their understanding of traits formation, claiming an "interdependence of nature and nurture" (Hogben 1933, 91), the concept of plasticity remained, for most of them, associated to speculative ideas condemned to disappear because of their lack of scientific evidence such as those of the theory of Organic Selection from the end of the nineteenth century (Osborn, Baldwin, Lloyd Morgan). However, despite the criticisms of this theory and its continued rejection, the concept of plasticity has remained in the biological literature thanks to its renewed use in the field of population genetics, and especially in the mid-1960s with the work of Anthony Bradshaw (Nicoglou 2015).

The Origin of the Nature-Nurture Opposition in Biology

In this section, I emphasize the role Francis Galton played in the popularization and "naturalization" (i.e., the use of scientific methods to assert the importance of a philosophical or literary issue) of the nature-nurture opposition at the end of the nineteenth century. Because it led to the introduction of the debate in the field of genetics at the beginning of the twentieth century, and especially because the concept of plasticity was used in genetics to go beyond such an opposition, I see this episode as a major one in the history of biology in relation to social sciences. Before coming back to the appearance of the concept of plasticity within the debate, I first examine Galton's influences

concerning the phrase, nature and nurture, in order to understand the way in which he used it and eventually modified previous conceptions of the phrase.

Shakespeare's Influence on Galton

It is well known that Galton was a great admirer of William Shakespeare since he didn't hesitate to quote him in many of his books and papers. Various references to nature and nurture are made throughout Shakespeare's work. The most famous one appears in *The Tempest*, where the wealth of human feelings and behaviors are symbolized. In a few words, the story is the following one: the Duke of Milan, Prospero, having been deposed and exiled by his brother, finds himself with his daughter Miranda on an island. Prospero has magical powers, which allow him to control the natural elements and minds including Ariel, described as a positive spirit of the air and of the breath of life, and Caliban, a negative savage symbolizing the earth, violence and death. Both of them symbolize primitive peoples of the colonies, slaves and toys of the colonial powers, caught up in quarrels that they do not understand. They are seemingly both on the side of "nature" (even if Shakespeare already seems to play with the ambiguity of such a duality/dichotomy between nature and nurture as shown through this quotation of Prospero concerning Caliban):

> A devil, a born devil, on whose nature
> Nurture can never stick! on whom my pains,
> Humanely taken, all, all lost, quite lost!
> And as with age his body uglier grows,
> So his mind cankers. I will plague them all,
> Even to roaring. (Shakespeare *The Tempest* 1610–1611)

In Shakespeare's mind, neither nature nor nurture really seems to have a role to play in Caliban's behavior. Furthermore, Shakespeare seems to consider that noble characters too, like Miranda, adopt behaviors he describes as "natural." However, despite Shakespeare's ambiguous use of the two terms, Galton did not hesitate to see a strict dichotomy between these two agents in the role they play in the establishment of human behaviors, as we will now see.

Hereditary Genius, the book that Francis Galton (1822–1911) published in 1869, was the first scientific attempt to study the human genius and greatness. The book starts with a strong rejection by Galton of the inheritance of acquired characteristics. At that time, his cousin Charles Darwin had already published *On the Origin of Species by Means of Natural Selection, or the Preservation of*

Favoured Races in the Struggle for Life (Darwin 1859), but naturalists were still arguing over the validity of Darwin's hypothesis concerning the laws of variation. To argue against the inheritance of acquired characteristics, Galton referred to the example of the development of the jaw. Some scientists of his time had attributed its increased development in some human populations to specific and repeated masticatory practices generation after generation. He rejected the argument by presenting several cases of tribes with massive jawbones without major masticatory practice. He concluded that "the diminishing size of the human jaw in highly civilized people must be ascribed to other causes, such as those, whatever they may be, that reduce the weight of the whole skeleton in delicate *nurtured* animals" [my emphasis] (Galton 1869, xvi). At this moment of the text, Galton did not offer any hypothesis concerning these other causes.

While in *Hereditary Genius*, Galton was not yet referring to the opposition between nature and nurture per se (between heredity and environmental circumstances), his position concerning the meaning of the concept of nurture appeared clearly. It came after and was thus second compared to nature, in the course of things.

Darwin's Influence on Galton

The publication of *The Origin of Species* by Darwin was a striking event in the life and work of Galton. The first chapter of the book ("Variation under domestication"), which dealt with breeding and intraspecies reproduction, fascinated him. Indeed, Galton was mainly interested in the problem of variation in human populations and its implications for human evolution. He set up a research program that captured many aspects of human variation (from mental characteristics to size; from facial expressions to fingerprints). This also required the invention of new measurements of traits that would allow him to analyze his considerable data collections. Therefore, he established new statistical techniques for describing and interpreting these data. For instance, in *Hereditary Genius* he used historiometry as a method. The principle had been first invented by the mathematician Quetelet. In Galton's case, historiometry can be defined as the "historical study of human progress or individual personal characteristics, using statistics to analyze references to geniuses, their statements, behavior and discoveries in relatively neutral texts."[1] Following these first studies, he prepared a survey that he sent to 190 members of the Royal Society in order to see if and how human capacities were inherited. He tabulated characteristics of their families, such as birth order, the race, or

occupation of their parents. The results were published in 1874 in a new book entitled *English men of science: their nature and nurture*. The purpose of the book was clear: "[it] was to assert the claims of one of what may be called the "pre-efficients" [note: or, "all that has gone to the making of"] of eminent men, the importance of which had been previously overlooked […]" (Galton 1874, vi).

The underlying idea that Galton was following was obvious, as it appears here: "[…] I am confident that one effect of the evidence here collected will be to strengthen the utmost claims I ever made for the recognition of the importance of *hereditary influence*" [my emphasis] (Galton 1874, vii).

One of the first questions of interest for Galton was whether human ability was hereditary. The inquiry was a way for him to see if the qualities of eminent men were further spread among their relatives than among the general population. For instance, he attempted to discover whether their interest in science was 'innate' or due to encouragements of others: "What then are the conditions of nature, and the various circumstances and conditions of life,— which I include under the general name of nurture,—which have selected that one and left the remainder? The object of this book is to answer this question" (Galton 1874, 10) (Fig. 5.1 shows the results Galton gathered concerning this question and with which he attempted to demonstrate that interest in science is rather "innate").

This finally led him to promote the nature/nurture opposition:

SUMMARY OF RESULTS AS TO INNATE TASTES.

	Total cases	Decidedly innate.	Decidedly not innate.	Doubtful.
Physics and Mathematics	20	12	1	7
Chemistry and Mineralogy	11	5	1	5
Geology	8	7	0	1
Biology-Zoology	24	17	3	4
Botany	10	8	1	1
Medical Science	7	2	4	1
Geography (not discussed separately)	0	0	0	0
Statistical Science	6	3	1	2
Mechanical Science	5	2	0	3
	91	56	11	24

Fig. 5.1 Table from Galton's book *English men of science: their nature and nurture*, showing the innate tastes for sciences (Galton 1874, 192)

> The phrase "nature and nurture" is a convenient jingle of words, for it separates under two distinct heads the innumerable elements of which personality is composed. Nature is all that a man brings with himself into the world; nurture is every influence from without that affects him after birth. The distinction is clear: the one produces the infant such as it actually is, including its latent faculties of growth of body and mind; the other affords the environment amid which the growth takes place, by which natural tendencies may be strengthened or thwarted, or wholly new ones implanted. (Galton 1874, 12)

Very often when one quotes Galton concerning the nature/nurture opposition, it is precisely and almost exclusively this quotation that appears (when it is not truncated). However, in the following lines of his text, Galton gives more details about his position. Galton goes on: "Neither of the terms implies any theory; natural gifts may or may not be hereditary; nurture does not especially consist of food, clothing, education or tradition, but it includes all these and similar influences whether known or unknown." (Galton 1874, 12) Contrary to what is often argued, Galton did not claim (at least at the time) that nature equals heredity. However, he claimed "When nature and nurture compete for supremacy on equal terms in the sense to be explained, the former proves the stronger […] neither is self-sufficient" (Galton 1874, 12). Here, Shakespeare's influence on Galton's idea is straightforward.

From Literature to Biometry

However, his method of historiometry, while extensively developed in his work, remained in Galton's own opinion insufficient to prove the inheritance of human abilities. This observation drew him to study twins. He planned on testing if identical twins at birth would differ when submitted to dissimilar environments and if dissimilar twins at birth would converge in similarity when reared in similar environments. He used questionnaires to gather data that he then tabulated. He described his results in 1876 in a paper entitled "The history of twins, as a criterion of the relative powers of nature and nurture." This was effectively the first field study on twins (many others will follow in the field of behavioral studies). Thanks to his results, he concluded that evidence was in favor of nature rather than nurture for the transmission of most of human abilities.[2] This led to the general idea that "nature" meant "inheritance."

Galton defended a "strong conception of inheritance" based solely on natural selection. With his own experiments on the size and weight of seeds, he almost rediscovered the particle theory of inheritance of Mendel (before the

independent rediscovery of Mendel's laws in 1900 by de Vries, Correns and Tschermak) but he did not succeed because his focus was primarily on continuous traits (size and height etc.) instead of discrete traits (such as color or shape etc.). However, he initiated the first studies in biometry to understand inheritance by referring to statistics in order to analyze continuous traits and their inheritance on the scale of populations.

Karl Pearson and Walter Frank Raphael Weldon would subsequently take up this approach enthusiastically. Together they founded in 1901 the journal *Biometrika*. Consequently, it has been argued that the statistical methods Galton contributed towards inventing (i.e., regression etc.) and some phenomena he established (such as the regression to the mean) were at the basis of the biometric approach. They are, today, also central tools of social science.

Galton, in opposition to his predecessors (e.g., Maupertuis, Buffon, Kant), no longer thought about inheritance in terms of "parent-child" similarity at the individual scale, but he introduced a population perspective and a new scientific method to examine variation. Furthermore, as Dale Goldhaber has argued, Galton, more than his cousin Darwin, "saw in a theory of evolution a way to differentiate nature from nurture and then to ascribe what for him was the rightful importance of each. It is really Galton who was the first to see the roles of nature and nurture as distinguishable and perhaps of greater importance to the debate, as existing as oppositional forces, each competing to influence development. For Galton, nature was clearly the winner" (Goldhaber 2012, 15–16). While Galton clearly promoted nature over nurture, he also contributed to the idea that the inherited factor was more influential than any other factor in trait determination.

The Emergence of Genetics and the Redefinition of the Nature-Nurture Debate

How to Understand Inheritance with Genetics?

The rediscovery of Mendel's laws in 1900, and their recombination with the discovery of chromosomes, considered as the physical substrate of heredity (by Morgan in 1909), are at the origin of formal genetics in the early twentieth century.

The Mendelian theory of inheritance is different from biometric inheritance (although Sir Ronald Fisher were to make Mendelian inheritance consistent with the biometric conception of inheritance in 1918). For instance, in

the Mendelian theory the origin of traits is irrelevant for understanding the nature of heredity. Contrary to previous views, heredity is not seen as an accumulation of influences. What counts is the structure of parental generation: "heredity depends only on the make-up of the parental generation (not that of the more remote ancestors), and Mendel's laws."[3]

Therefore, when Mendel's laws were rediscovered—and even if they gave a solid explanation for discontinuous variation—they continued to receive strong opposition for many years. Opponents did not claim that Mendelian inheritance did not exist; they only thought that *all* inheritance was not necessarily Mendelian (Mayr 1982, 119).

At that time, Darwinian zoologists and botanists had already looked into the question of inheritance but mainly in connection with evolution (like Galton as we saw in the previous section). Therefore, when they started to take notice of the new discipline of Mendelian genetics, they did so mainly through Mendelian geneticists who also talked about evolution (e.g., Hugo De Vries, William Bateson). But in some respects, their views seemed unacceptable to Darwinian zoologists because both De Vries and Bateson claimed that the discontinuous nature of heredity was another proof for the saltationist view, which claimed that the appearance of species was discontinuous. Yet, such a view was in strict opposition with Darwin's evolution since he had constantly highlighted the gradual and continuous nature of evolution.

In addition to this theoretical opposition, new definitions and new terms accompanied the nascent disciplines of genetics increasing at times the confusion between supporters of Mendelism. For instance, it was becoming particularly difficult to think about the behavior of genetic material by referring to the sole phenotypic (apparent) characters. Hugo de Vries had suggested replacing the old notion of mixture (which referred to the appearance of intermediate traits in species breeding) by the notion of mutation. However, it was not clear if he considered "mutation" as something that could be described at the phenotypic level or at the underlying germplasm level. For animal breeders and botanists, such assimilation was inconceivable because they knew that many characteristics they selected were also influenced by environmental factors in addition to genetic factors (i.e., that nurture was influencing nature).

In addition to the way both sides—Mendelians and Darwinians—saw evolution in relation to inheritance, another problem arose: how could hereditary information be described? If both Darwin and Galton had a granular conception of inheritance—they thought that hereditary information was stored in particles—Mendelians were more divided concerning this issue.

For instance, the plant physiologist Wilhelm Johannsen who introduced in 1909 the notion of "gene," and in 1910[4] those of "genotype" and "phenotype"

interpreted the gene—the abstract substrate of inheritance—as a *statistical unit* but not as a *granular unit*. On the other side, Thomas Morgan, who after being a critic became a fervent advocate of Mendelism, developed a physical conception of inheritance by showing how the hereditary information was located in the chromosomes.

While Morgan is known to have played a major role in the foundation of modern genetics, before him, Johannsen had also offered a significant contribution to the nature-nurture distinction. Let me now explain why.

Johannsen's Contribution to the Nature-Nurture Debate: Genes Versus Environment

One of Johannsen's major scientific contributions concerned the common bean. He showed that in populations without genetic variation (homozygous for all traits, in Mendelian terms), seed size followed a statistical normal distribution. Indeed, he observed that the average weight of these beans was submitted to a "fluctuating variability" linked, in his opinion, to microenvironmental differences. He suggested that these results showed that homozygous beans did not change their inherited constitution (that he called "genotypical constitution") except by mutation (see Burian 2000).

In order to clarify his position, Johannsen distinguished his conception of inheritance from previous ones. He argued that most past conceptions were based on the meaning of the terms "heredity" and "inheritance" in everyday language (i.e., the idea of transmission of money or things, rights or duties or even ideas and knowledge from one person to another or to some others: the "heirs" or "inheritors"). According to Johannsen, this common or "naïve" conception of inheritance retained the idea that personal qualities of the parents (or ancestors) were transmitted to the progeny. He called this view of inheritance the "transmission conception of heredity" and qualified it as an "apparent heredity" since he considered that only superficial insights could be gained by working on this basis.

He conceded that medical and biological statisticians (starting with Galton) had been able to make statements of great interest, for the "eugenics-movement" for instance, with such a view. But he maintained that no "profound insight into the biological problem of heredity [could] be gained on this basis" since he thought that "the transmission-conception of heredity represents exactly the reverse of the real facts" (Johannsen 1911, 130).

For Johannsen, "the *personal qualities* of any individual organism do not cause the qualities of its offspring; but the qualities of both ancestor and

descendant are in quite the same manner determined by the nature of "sexual substances"—i.e., the gametes—from which they have developed" (Johannsen 1911, 130). The identification of "innate" factors should not be achieved through an analysis of the apparent personal qualities but through a precise analysis of the behavior of sexual substances. Johannsen suggested then that personal qualities rely on the "*the reactions of the gametes* joining to form a zygote" (Johannsen 1911, 130). Therefore "the nature of the gametes is not determined by the personal qualities of the parents or ancestors in question" (Johannsen 1911, 130). With this new view concerning inheritance, Johannsen forged the modern conception of heredity that he called the "genotype-conception" of heredity, based on the idea that it was not directly the qualities, which were transmitted, but *something that determined* those qualities. This new conception was also the result of the recent genetic research on "pure line" breeding and hybridization following Mendel's model.

Johannsen also thought that his conception differed from Galton's view because Galton continued referring to an outdated terminology despite what Johannsen considered to be his admirable ideas. Johannsen believed that Galton's insights had been essential for later understanding of qualities transmission.

In his "theory of heredity," Galton had indeed assumed that the newly fertilized ovum contained the sum total of the germs, gemmules and so on, that he called the "stirp" (Galton 1876, 330). Among the stirp only a few "are patent, developing into particular cell types, while the rest remain latent; the latent elements can be transmitted to the next generation, while the patent elements, with rare exceptions, cannot since they have developed into cells" (see Bulmer 1999, 263). While Galton's theory remained quite close to those of pangenesis of his cousin Darwin, Galton nevertheless distanced himself from Darwin's view[5]:

> We cannot now fail to be impressed with the fallacy of reckoning inheritance in the usual way, from parents to offspring, using those words [heredity and inheritance] in their popular sense of visible personalities. The span of the true hereditary link connects, as I have already insisted upon, not the parent with the offspring, but the primary elements of the two, such as they existed in the newly impregnated ova, whence they were respectively developed. (Galton 1872, 400)

For Johannsen, "those special ideas may have some interest as expressions of the searching mind […], [but they] have no support in experience". By contrast, his genotype-conception of heredity (although "initiated by Galton, but now revised as an expression of the insight won by pure line breeding and

Mendelism") was "in the least possible degree a speculative conception." (Johannsen 1911, 132)

From this moment, Johannsen proposed a new terminology and made three major lexical contributions to genetics with the terms of "genes," "genotype" and "phenotype" that he defined in the following way:

> The 'gene' is nothing but a very applicable little word, easily combined with others, and hence it may be useful as an expression for the 'unit-factors', 'elements' or 'allelomorphs' in the gametes, demonstrated by modern Mendelian researches. (Johannsen 1911, 132)

In Johannsen's mind, the gene does not necessarily have a physical reality. At least, he thought that, based on the knowledge of his time, it was not possible to propose any hypothesis concerning its nature, even though Mendelism had allowed biologists to claim that the gene did cover a material reality. Therefore, he was cautious in his use of the word as much as in his understanding of the "genotype" that he defined as "the sum total of all the 'genes' in a gamete or in a zygote." (Johannsen 1911, 132–133) Johannsen recommended using the adjectival term "genotypical" instead of the noun "genotype" since "we do not know a "genotype," but we are able to demonstrate "genotypical" differences or "accordances" (Johannsen 1911, 133). Finally, the phenotype is the statistical average of a sample while the genotype is the genetic makeup of the zygote, resulting from the union of two gametes.

While Johannsen's distinction between the genotype and the phenotype would later lead to a strong genetic determinism, allowing some advocates of the supremacy of nature (genes) over nurture (environment) to consider, for instance, that the sole action of a specific gene could determine a personal quality, at the time Johannsen claimed that his own definitions depended greatly on another zoologist of that time: Richard Woltereck. Yet Woltereck's view was located on the opposite side of an "innate," genotypical conception (understood as "innate") of trait determination.

Woltereck Offers Interplay Between Nature and Nurture: The "Reaktionsnorm"

Indeed, a few years before, during a meeting of the German Society of zoology, in June 1909, Woltereck had presented his work, which was the result of years of studies of a particular species: Daphnia, a small aquatic crustacean living in freshwaters. The purpose of his presentation was to support the

Darwinian view of evolution by showing that evolution occurs through natural selection acting on small continuous variations. He observed that pure lines of Daphnia kept their specific morphological shape through many successive generations of parthenogenesis. Based on this stability, he tried to see the influence of various environmental factors on a number of traits. For instance, he compared the variation of the "relative head-height" (a continuous trait) together and in relation to different nutrient levels the animals had access to (see Fig. 5.2).

He found that it varies between different pure lines, that it is affected by environmental factors (such as nutrient levels), that it varies almost independently of other environmental factors (such as temperature). Furthermore, he observed that the response to a particular environmental variation (e.g., nutrient level from level 1 to 2) was not the same between the different lines. He drew "phenotypic curves" to describe the phenomenon. Phenotypic curves can change with each new environmental variable considered (e.g., time of

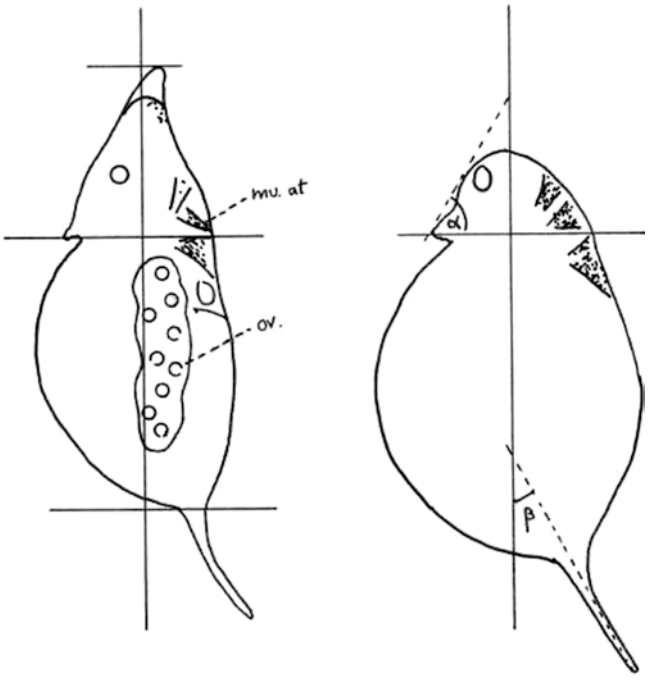

Fig. 5.2 Side views of two pure lines of Daphnia by Woltereck (1909, 114). The head-height is measured along the vertical axis between the uppermost horizontal line and the horizontal centerline. The "relative head-height" is the height of the head divided by the distance between the bottom horizontal line and the top one (and multiplied by 100 to be expressed as a percentage)

the year). Therefore, there were potentially almost an infinite number of them. He coined the term "*Reaktionsnorm*" to describe the totality of the *relations* that associate the curves with each other (see Fig. 5.3).

Finally, he thought that it was the Reaktionsnorm that was transmitted, and thus inherited.[6] With his interpretation, Woltereck could save Darwinism from saltationism since he had shown how selection would act on small gradual changes. However, with Woltereck's view, we are far away from a static view of the "innate" component, which would determine the phenotype. In contrast, the "inherited factors" appear as a dynamic component, which could not be analyzed without taking into account the environment (nature and nurture could not possibly be opposed) and Johannsen was well aware of this:

> The 'Reaktionsnorm' [a term proposed by Woltereck] emphasizes the diversity and still the unity in the behavior of the individual organism; certainly, the particular organism is a whole, and its multiple, varying reactions are determined by its 'genotype' interfering with the totality of all incident factors, be they external or internal. Thence the notion 'Reaktionsnorm' is fully compatible with the genotype-conception. (Johannsen 1911, 133)

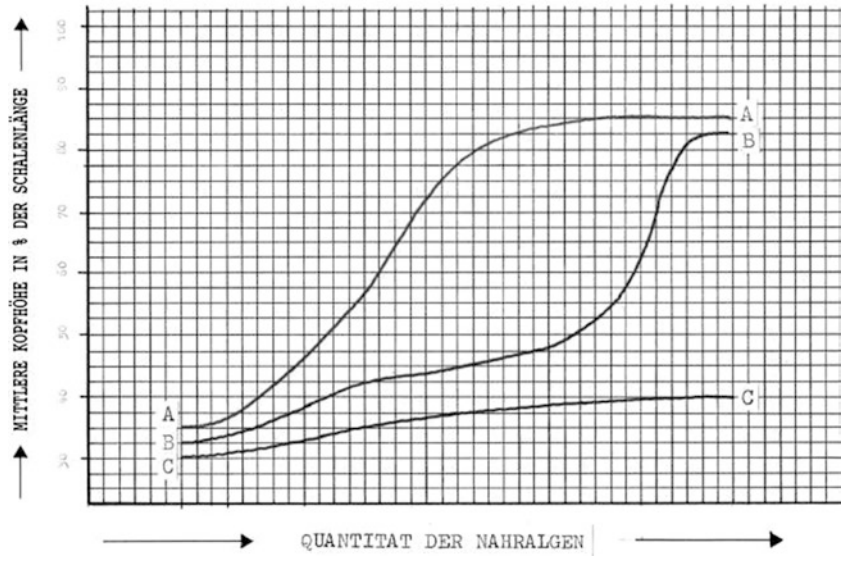

Fig. 5.3 Phenotypic curves of three females of pure lines of Daphnia by Woltereck (1909, Fig 12, 139) X-axis: nutrient levels; Y-axis: relative head-height. The curves show a non-uniform variation between pure lines. The "Reaktionsnorm" or the "genotypus," for Woltereck, is the sum of all possible curves for a given line (e.g., A+A'+A"+A"'…)

Through his representation, Woltereck tried to explain the *permissive*, though constrained, role of environmental factors on phenotypic variation. Woltereck thought of the inherited mutations as the modification of the Reaktionsnorm and "the environment was a constructive constituent of the individual phenotype" (Falk 2001, 123). His interpretation of the norm of reaction was slightly different from those of Johannsen, who mainly saw it as synonymous of his "genotype," which he described as a *determinant* agent of phenogenesis. Therefore, after Johannsen it became possible to distinguish not only nature from nurture but also to consider that the biological equivalent of nature was the inherited factor—the gene, or at least the genotype—and that the biological equivalent of nurture was the environment and that both of them contributed to the trait determination, even though (since Galton) we have known that the first one came first. Johannsen, on his side of the debate, would deny that the continuous traits were inherited and he agreed with the Mendelians, who adopted a saltationist conception of evolution (Provine [1971] 2002, 95).

Finally, the two conceptions—Johannsen and Woltereck's views—concerning the norm of reaction were eventually partially integrated in the 1950s by the American geneticist and evolutionary biologist Theodosius Dobzhansky[7] (more faithful to Johannsen's conception). The notion of norm of reaction would, then, be used to qualify each individual phenotypic curve and not the sum of the relations between these curves.

Nilsson-Ehle's Concept of Plasticity in Genetics

Despite Johannsen's success and his popularity until today in the field of genetics, his work was subject to much criticism in the early 1910s. Pearson and Weldon, the biometrician successors of Galton, were among his critics but also Nilsson-Ehle, a Swedish geneticist who carried out experiments at the plant breeding station in Svelöf from 1900. Known for the resolution of one of the many apparent exceptions to the Mendelian rules—the inheritance of continuously varying or fluctuating traits—he demonstrated that:

> By cross breeding two pure lines [homozygous combination, or the "genotype" of Johannsen] [one] can produce an amazing variety of new constant forms; Only one cross breeding of this type is enough to produce all the variety of constant forms or lines of an indigenous line. In other words, a whole 'population' of new forms or lines is obtained by cross breeding only two [genotypical] constant forms. (Nilsson-Ehle 1914, 862)

Basically, he discovered what we now call polygenetic inheritance: the fact that an organism's phenotype is due to two or more genes and conversely the fact that one unit of genotypical information can lead to several phenotypes (polyphenism).

Furthermore, Nilsson-Ehle was the first geneticist to use the word "plasticity" to describe specifically *the effect of the environment on the phenotype of an organism*. In an article published in 1914, Nilsson-Ehle used the term "plasticity" [*plasticitet*] to describe the acclimation of alpine plants to their environment (Nilsson-Ehle 1914, 542). He was also the first biologist to consider the plant *Polygonum amphibium* as "particularly plastic" because it could develop both terrestrial and aquatic characteristics according to the environmental signal it received (see Fig. 5.4 showing the different phenotypes of the plant).

Nilsson-Ehle mainly understood plasticity as part of self-regulation mechanisms (mechanisms by which an organism responds to environmental changes) as opposed to "evolutionary adaptive.[8]" Therefore, Nilsson-Ehle did not define plasticity as "a property of a single genotype" in the same way Johannsen had understood the notion of "norm of reaction" (i.e., as the "reaction range" of a single genotype to changing environments).

One question remains: if critics of Johannsen's view led a geneticist such as Nilsson-Ehle to refer to the notion of plasticity to argue for multiple factor determinism in trait determination and against a strong genetic determinism, what are the origins of such a term as "plasticity"?

"Plasticity": What Interplay Between Nature and Nurture?

Plasticity in Psychology and the Origins of the Concept in the Nature-Nurture Debate

When Galton was still offering a general review of his work on heredity at the end of the nineteenth century (*Natural Inheritance* 1889), on the other side of the Atlantic, the American philosopher and psychologist William James was publishing his *Principles of Psychology* (1890), which would have a lasting influence on the whole field of psychology. In this book, James explains, among other things, that the instincts are to be considered as "innate ideas." Moreover, when talking about habits, James offers a fairly original theory in which he tries to explain the modification and formation of new habits:

Fig. 5.4 Polygonum amphibium. (a) Water form and (b) Terrestrial form. By Johann Georg Sturm (painter: Jacob Sturm) from book *Deutschlands Flora in Abbildungen* at (http://www.biolib.de, public domain, https://commons.wikimedia.org/w/index.php?curid=750700.jpg)

> In the organic world […] the habits are more variable […] Even instincts vary from one individual to another of a kind; and are modified in the same individual […] to suit the exigencies of the case. The habits of an elementary particle of matter cannot change […], because the particle is itself an unchangeable thing; but those of a compound mass of matter can change, because they are in the last instance due

> to the structure of the compound, and either outward forces or inward tensions can, from one hour to another, turn that structure into something different from what it was. That is, they can do so if the body be plastic enough to maintain its integrity, and be not disrupted when its structure yields. (James 1890, 104)

James argues for the *plasticity* of the organic matter when submitted either to outward or inward forces. He compares it to the physical reactions that the inert matter can encounter when submitted to such tensions. From a quite precise "physical" description of the modification of habits through time, and under external and internal inputs (James 1890, 104), James comes to a definition of what he calls "plasticity":

> *Plasticity*, [author's emphasis] then, in the wide sense of the word, means the possession of a structure weak enough to yield to an influence, but strong enough not to yield all at once. Each relatively stable phase of equilibrium in such a structure is marked by what we may call a new set of habits. *Organic matter* [my emphasis], especially nervous tissue, seems endowed with a very extraordinary degree of plasticity of this sort; so that we may without hesitation lay down […], that *the phenomena of habit in living beings are due to the plasticity of the organic materials of which their bodies are composed* [author's emphasis]. (James 1890, 105)

In a way, James was claiming a certain sensitivity of organic matter (actually especially the brain tissue) to both internal and external influences. This sensitivity, that he called "plasticity," was, in his opinion, quite specific to the organic matter organized into structures.[9]

One year after James's publication, Conwy Lloyd Morgan, a British ethologist and zoologist, who became quickly interested in "mental evolution" and turned to psychology, wrote a book entitled *Animal, Life and Intelligence* (1891). In this book, and like James, he wrote about "Habit and Instinct." However, as a zoologist (he became a professor of psychology and education only in 1901) his primary influence was not James but rather the German evolutionary biologist August Weismann.[10] And his biological training permeated his view on animal intelligence as it appears here:

> The consideration of Animal Intelligence, from the scientific and philosophical standpoint, has been my primary aim. But so inextricably intertwined is the subject of Intelligence with the subject of Life, the subject of organic evolution with the subject of mental evolution, so closely are questions of Heredity and Natural Selection interwoven with questions of Habit and Instinct, that I have devoted the first part of this volume to a consideration of Organic Evolution. (Lloyd Morgan 1891, v)

However, Lloyd Morgan questioned how much we could rely on Galton's twin studies to come to any conclusions about the laws of variation:

> We require, however, further and fuller observations to render the evidence of such hereditary summation [i.e., that it would require persistent and long-continued influence to modify the individual, its structure inherited or given by nature] to any extent convincing. (Lloyd Morgan 1891, 170)

He, thus, had in mind an alternative view concerning evolution and the sensitivity of individuals to their environment. The specific influence that the environment can exert, in his opinion, on species will lead Lloyd Morgan to adopt an organic conception of evolution in which "the relation of an organism to its circumstances or environment is itself subject to change [and in which] the environment itself may alter, or the organism may be brought into relation with a new environment" (Lloyd Morgan 1891, 182). Therefore, Lloyd Morgan thought that one should pay attention not only to "the changes in an organism in the direction of more or less perfect adaptation to its environment, but also [to the] changes in the environment" (Lloyd Morgan 1891, 182) (see Fig. 5.5 for an instance of such organism).

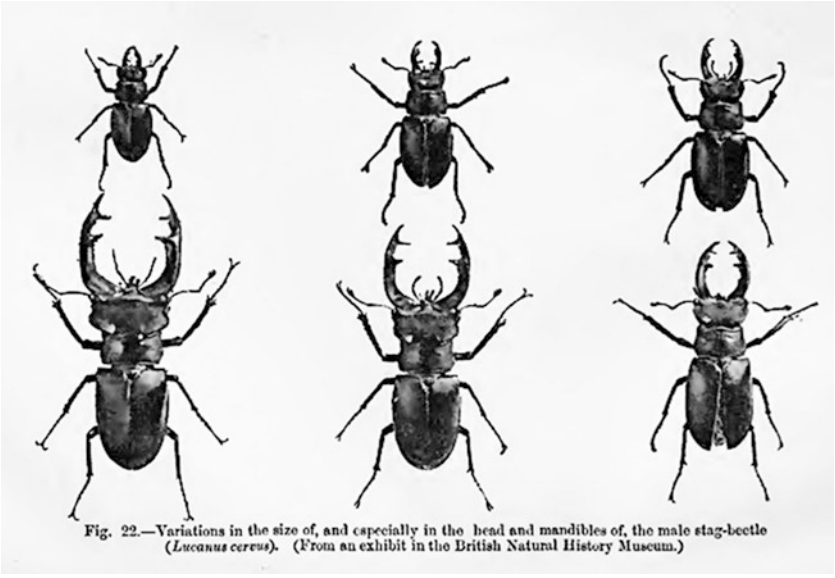

Fig. 5.5 Representation of different male stag-beetles, illustrating how the variation in the environment of the insect (linked to the need to defend) can lead to various forms within the same species (From Lloyd Morgan 1891, 180)

The Concept of Plasticity Within the Theory of Organic Selection

A few years later in 1897, Lloyd Morgan, Mark Baldwin and Henry Osborn published a series of papers in the journal *Sciences* entitled "Organic Selection," which were the result of their joint discussion on the topic. In the opening article, Professor Osborn explains that: "Independently, Professors Baldwin and Morgan and [himself] put together the facts of individual adaptation with those of determinate variation into an hypothesis which is in some degree new" (Osborn 1897a, 584). However, what Baldwin called "organic selection" was first described by Osborn as "a mode of evolution requiring neither natural selection nor the inheritance of acquired character" (Osborn 1896) and in a way, the three authors differed quite significantly in their respective points of view.

Osborn, for instance, disagreed with the idea of Lloyd Morgan and Baldwin that "[…] the power of plastic modification to new circumstances [i.e., self-adaptation] [was] in itself a result of natural selection" (Osborn 1897b, 950). In his view, natural selection was responsible, in organisms, for a "power of plastic modification," which was insensitive to the new conditions and, in most of the cases, substantially adaptive. Osborn saw the "plastic modification" as an *inherent power* constitutive of the protoplasm. Such a view was rooted in the embryological and zoological tradition of Hans Driesch, Edmond Wilson, and Thomas Morgan.

On the other hand, the American philosopher and psychologist Baldwin promoted the idea (inspired by his studies of children's learning) that behavior could affect the effect of natural selection, and even facilitate it. In his opinion, thanks to their learning abilities, individuals survive by adapting to adverse environmental conditions. For Baldwin, if the environment does not change too abruptly, the most adaptive mutations will change into congenital characteristics that were first brought about through learning. Consequently, learning (i.e., through education) "guides" evolution because it introduces a bias in perpetuated mutations. The ability to learn increases genetic variance of population. During a sudden environmental change, only very different individuals (who exist because of learning ability) may survive. Learning "accelerates" evolution and leads to evolutionary leaps.

Baldwin agreed with Lloyd Morgan that the term "organic selection" should replace the term "modification" for all ontogenetic variations. In other words, the term "organic selection" makes it possible to express the process by which individual adaptations guide evolution. Unlike Osborn, the two authors did not see "organic selection" as an inherent property of the individual.

Few zoologists will refer to this view, and particularly in order to reject a strong dichotomy between nature and nurture (i.e., between gene and environment). It is the case of the American zoologist Maynard Metcalf who published in 1904 a book entitled *An Outline of the Theory of Organic Evolution* intended for biologists but also for a wider audience. In this book, he assessed the importance and influence of individual plasticity upon evolution and referred to "plasticity" as a synonym of "educability" (Metcalf 1904, 177). In an article published in 1906 in *Science*, entitled "The influence of the plasticity of organisms upon evolution," Metcalf highlighted that while he agreed with Morgan, Osborn, Baldwin and others concerning the role of organic selection on evolution, he thought that the extent and the precise characteristics of its influence were still under discussion. He argued that there was another possible influence of plasticity, which had been underestimated: namely that it is linked to specific conditions of organisms rather than to environmental factors. For instance, he claimed, based on his own experiments, that "the appearance, generation after generation of the same mutants of *Oenothera lamarckiana*, in numbers far greater than could be explained by purely fortuitous variation, [was] a further indication of some internal control over variation, making it somewhat determinate instead of purely indeterminate" (Metcalf 1906, 787).

However, after the rediscovery of Mendel's laws in the 1900s and even more with the development of modern genetics in the 1930s (i.e., Mendel's laws combined with Morgan's chromosomes theory), the theory of organic selection will almost entirely disappear from the discussions. And it will mainly be Fisher (1930), Haldane (between 1924 and 1934, he published a series of papers titled "A Mathematical Theory of Natural and Artificial Selection") and Wright (1931) who will bring major contributions concerning this issue of "trends in evolution," based on mathematical models. By putting forward population genetics, to the detriment of embryology, these biologists (also supporters of a "synthetic" conception of evolution) will put aside the notion of plasticity (mainly associated with embryology and the theory of Organic Evolution).

Therefore, among geneticists also interested in the rejection of the nature/nurture dichotomy, the notion of plasticity rarely appears (e.g., Herbert Eugene Walter rejects the dichotomy but does not refer to the notion of plasticity, 1913). Some of them maintain more or less a reference to the notion of "genetic plasticity" (for the British scientists), as Gavin de Beer will define it in 1930, and focus on the "norm of reaction," that Theodosius Dobzhansky will later popularize through the notion of plasticity (Dobzhansky 1955). Therefore, Nilsson-Ehle's use of the notion of plasticity in relation to laws of variation and heredity is the one exception in the genetics of the 1910s.

Hogben's Conceptual Clarifications Concerning the Nature/Nurture Interplay

Despite the progressive disappearance of the theory of Organic Selection after the emergence of modern genetics, the rejection of a strict opposition between nature and nurture will be pursued. Scientists will particularly question the so-called determinate action of the genetic factor on the phenotype. It will be the case of the British experimental zoologist and medical statistician Lancelot Hogben who, in a little book published in 1933, throws light on the interdependence of nature and nurture and the gene-environment interplay. For instance, he claims that "no statement about genetic difference is clear, *unless it includes or implies a specification of the environment in which it manifests itself in a particular manner*" [author's emphasis] (Hogben 1933, 14). Therefore, in his opinion, "no gene can be supposed to have a single absolutely specific effect" (Hogben 1933, 16). Consequently, he thought that:

> […] when we speak of heredity or environment as more or less *important* in connexion with any differences between human beings, *our criterion of importance is relative to the historic environment in which the differences themselves are measured* [author's emphasis]. (Hogben 1933, 18)

Hogben was one of the first scientists to reject not only Fisher's view concerning the partition of nature and nurture (i.e., the partition of heredity and environment) but even more to reject his underlying objective in relation to his mathematical formalism: "[Fisher's objective] was 'to ascribe to the constituent causes' (heredity and environment) 'fractions or percentages of the total variance which they together produce'" (Hogben 1933, 92). Hogben's conclusions were clear and definitive: "the biometrical treatment of variability inherited from Galton a tradition of discourse in which the ambiguity of the concept of causation completely obscured the basic relativity of nature and nurture" (Hogben 1933, 95).

The dichotomy between nature and nurture (between heredity and environment and later between genes and environment) and their respective role on trait determination were only, for Hogben, the caricature of a conceptual reading of the question "what causes a human ability?" While Hogben's arguments did not receive much attention, algebraic analyses of John Burdon Sanderson Haldane would become the dominant arguments in the understanding of genotype-environment interactions (Sapp 1987; Sarkar 1999). Haldane's view was more nuanced than Hogben since Haldane quickly attempted to encompass the complexity of genotype environment interaction within the structure of Mendelian genetics (Haldane 1946).

Therefore, today the opposition between nature and nurture remains because it could partially be integrated into the Fisher's model, even though opponents of the nature-nurture dichotomy had attempted to reject Fisher's formalism. Concerning plasticity itself (i.e., genetic plasticity), any kind of formalism is still lacking (in the sense that the nature/nurture dichotomy was formalized by Fisher). One of the reasons can be the particular ambiguity of the term, since it appears in different fields, often with different meanings, or at least associated with different connotations (e.g., in developmental biology, the concept of plasticity is used to characterize cells' potential to divide and differentiate; in ecology, it is used to describe the extraordinary adaptability of organisms to varying environments; in genetics, it refers not only to molecular interactions' complexity, but also to the diversity of their corresponding phenotypic signals).

Conclusion

In this chapter, I have analyzed how Galton forged the nature/nurture opposition as it is commonly seen in biology. His purpose was to understand how human abilities were inherited. In order to do so, he separated innate qualities from personal experiences. His work based on data recording and statistics finally led to the idea that inheritance was not the combination of both nature and nurture but rather to the idea that "nature" was "inheritance" in a strong sense and should be opposed to "nurture." Yet, one of the first influences of Galton concerning this issue was Shakespeare. The literary and poetic figures showed more complexity in the relationships between the two instances of nature and nurture than Galton's own interpretation. However, the emergence of Mendelian genetics and the study of hereditary factors led biologists to question the usual understanding of inheritance and to argue for a "strong inheritance": inheritance was not solely the transfer of physical, material entities to the next generation; it was a potentiality factor, which, if transmitted, could be expressed through phenotypes. The progressive assimilation of these potentiality factors to the "genes" led to the idea that the nature/nurture opposition could be reformulated as the opposition between genes and environment—genes being seen as the most determinant factors among the causal factors for the establishment of organisms' phenotypes. The idea of a greater complexity between "nature" and "nurture" was left mainly to literary works and to speculative discussions. The few scientific opponents of such a dichotomy in the twentieth century have sometimes referred to the notion of plasticity to characterize the interplay between nature and nurture. Thus, the

concept of plasticity finally appeared as a key-concept to overcome the nature-nurture opposition.

In this chapter, I have tried to show that the ambiguity inherent in the causal status of the nature/nurture dichotomy is reflected in the ambiguity of the meaning of the concept of plasticity in biology in the twentieth century. In other words, while underlying the nature/nurture dichotomy, one can actually identify several causal factors for the establishment of organisms' phenotypes (i.e., inherited factors vs. environmental factors, genes vs. environment, innate vs. education, etc.) that are often intertwined rather than opposed, one can also identify many types of interplay between nature and nurture depicted by the concept of plasticity (i.e., adaptability of organisms to varying environments, molecular interactions' complexity, diversity of the phenotypic signals, etc.). For these reasons, while the concept of plasticity can first appear as a useful concept to overcome the nature/nurture dichotomy, the indetermination of its specific scientific focus has contributed to the maintenance of the nature/nurture opposition.

Indeed, despite many recent attempts to overcome the nature-nurture dichotomy (e.g., Gottlieb [1997] 2014; Pigliucci 2001; McKinnon and Silverman 2005; Keller 2010; Jones 2011; Bateson and Gluckman 2011)—thanks to advances in biology, in epigenetics, in genetics on spicing, post-transcription and post-translation, as well as through theoretical discussions concerning the role of genes—references to the opposition seem to persist in many public discussions. The reasons for such persistence have yet to be elucidated.

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Notes

1. *E-Study Guide for: Political and Civic Leadership: A Reference Handbook: Political Science, Political Science*. Cram101 Textbook Reviews, 2012. https://store.kobobooks.com/fr-fr/ebook/political-and-civic-leadership-a-reference-handbook
2. Galton also referred to adoption studies (including studies on inter-racial adoption) in order to distinguish the effects of inheritance from the effects of the environment. Note that Plomin et al. (ed.) mentioned that the first adop-

tion study, which investigated IQ, was reported in 1924 by Theis (Plomin et al. 2008, 76).

3. From the conference of Gayon, J., "Beyond genetics or beyond heredity? A retrospective look at 20th Cy biology", Workshop "How can we redefine inheritance beyond the gene-centered approach?", Paris, Oct. 2–3, 2014, Org. F. Merlin & G. Pontarotti.
4. First in a conference in 1910 in front of the American Society of Naturalists and then published in 1911 in *The American Naturalist*.
5. Darwin had developed what he called "a provisional hypothesis"—the theory of pangenesis—in the *Variation of Animals and Plants under Domestication*, which was quickly rejected. This theory implied that the whole of parental organisms participate in heredity. He speculated that inheritance relied on tiny particles he called gemmules that could be transmitted from parent to offspring. He thought that cells formed atomic sized gemmules that would diffuse and aggregate in the reproductive organs.
6. See Sarkar 1999, for details concerning this historical episode.
7. Theodosius Dobzhansky was a central figure in the field of evolutionary biology for his work in shaping the unifying modern evolutionary synthesis.
8. Nilsson's Ehle's view about plasticity differed from his successor, the geneticist Anthony Bradshaw, who will popularize the notion of phenotypic plasticity in the mid-1960s (for more concerning Bradshaw, see Nicoglou 2015).
9. Note the influence of Herbert Spencer, *The Principles of Biology* vol. 2 about the nerve and his doctrine of "physiological units."
10. August Weismann's main contribution is the germ plasm theory, according to which (in a multicellular organism) inheritance only takes place by means of the germ cells—the gametes. Other cells of the body—the somatic cells—do not function as agents of heredity.

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6

An Evolving, Evolutionary Science of Human Differences

Jonathan Marks

Introduction

The earliest discovery of anthropology was that nobody contemplates their own ancestors and relatives in objective, dispassionate, genetical terms. This generalization applies as well to scientists. Here, I track the growth of the scientific study of human diversity by examining its two major fallacies: first, that the human species is composed of zoologically meaningful taxonomic entities and, second, that human groups think differently in ways that are significantly innate. Both of these propositions have been falsified about as thoroughly as young-earth creationism, but their political value is sufficient to continually resurrect them. This in turn creates a moral dimension for contemporary scholars engaged in this science.

European Roots of Anthropology

Physical and behavioral contrasts among ancient peoples had been made by Herodotus, but by the seventeenth century new peoples, new economic relations, and new standards of rigor promoted a growing interest in systematizing human diversity. The colonial engagement with diverse non-European peoples introduced a rich source of data where previously

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scholarly knowledge of the human condition could be gained entirely by introspection. In the eighteenth century, the Swedish biologist Carl Linnaeus imposed a rigorous taxonomic structure upon the human species as a part of his classificatory enterprise, while the Prussian philosopher Immanuel Kant considered how human nations and peoples retained their distinctive features.

Catalyzed by the vexatious slavery question, early nineteenth-century scholars grappled with the nature of human differences in a largely biblical framework. If one imagined Adam and Eve as European-looking, then to account for African-looking peoples necessitated developing a theory of microevolution, by which a human lineage could change its appearance dramatically over the course of a few thousand years. Alternatively, one might imagine the human races as having been distinct since their respective creations, taking some liberties with Scripture—but in an age where the geologists had already shown that Scripture was scientifically untrustworthy, that might not be so bad (Greene 1954).

This question of monogenism or polygenism—a single origin for the races or multiple origins—lay at a crucial juncture between biological and political questions. Darwinism largely mooted the descent argument, but opened other avenues for naturalistically dehumanizing other peoples. To the influential German evolutionist Ernst Haeckel (1868), in the absence of a human fossil record, the non-European peoples constituted the connecting links between Europeans and apes; indeed, they comprised distinct species who possessed varying, but less fully human, natures.

Many of Haeckel's contemporaries were unenthusiastic about his casual dehumanizing of different peoples. Founded in 1869, the Berlin Society for Anthropology, Ethnology, and Prehistory was founded instead on the premise of "the psychic unity of mankind"—that all people were in fact fully human, and thus fully capable of reason and culture (Köpping 1983). Indeed, the thorough entanglement of evolution and racism in Germany left many empirical students of human diversity, such as Rudolf Virchow and Adolf Bastian, ambivalent about Darwinism itself (Zimmerman 2001).

Haeckel, of course, went on to wield enormous intellectual influence, and his works were widely translated. His view of evolution led essentially from the amoeba, by constant competitive struggle, to the Nordic state. Biologist Vernon Kellogg (1917), visiting the German military before the United States entered World War I, was struck by how the well-educated officers seemed to be able to rationalize their militarism by recourse to this version of evolution. Haeckel himself argued for the natural good of the Great War in *The New York Times* (Haeckel 1916).

In the Anglophone world, the biopolitical landscape was somewhat different, and the English translation of Haeckel's *Natural History of Creation* omitted the repugnant racial illustrations of the earliest German editions. The Ethnological Society of London was founded in 1843, but in 1863 its polygenist members seceded and formed the Anthropological Society. The early Darwinians generally remained monogenist "Ethnologicals" and the two societies re-merged in 1871, under the leadership of Thomas Huxley (Stocking 1971). Thus, where the German anthropologists found themselves across a political-ideological aisle from the leading Darwinians, the early English anthropologists and Darwinians were generally on the same side of the aisle.

The undeniable coexistence of ancient people with extinct animals seemed to beggar any possibility of reconciling Genesis with prehistory, which in turn promoted scholarly interest in archaeology. What were people like before fire, agriculture, or metals—a condition hardly even acknowledged in the Bible (Van Riper 1993; Livingstone 2008)? As biology was codifying descent by the early 1870s, so too was archaeology codifying prehistory, beginning with an 'Old Stone Age' (Lubbock 1865).

Edward B. Tylor's *Primitive Culture* (1871) formalized the knowledge of the day on prehistory and ethnology as a "science of culture." "Culture" was in turn borrowed from the German, and denoted the diverse aspects of human social life that were acquired, not genetically inherited. Like Lamarckism (Meloni 2016), culture provided a powerful scientific force of social progress distinct from biologically inherited properties. The question of exactly what was biologically or culturally inherited could nevertheless be contested.

While British biopolitics differed from its German counterpart, a strain of British biology nevertheless quickly emerged that could casually rationalize genocidal colonialism. Leading evolutionary geneticist Karl Pearson explained, "a capable and stalwart race of white men should replace a dark-skinned tribe which can neither utilize its land for the full benefit of mankind, nor contribute its quota to the common stock of human knowledge" (1892, 438). Culture, on the other hand, elevated that "dark-skinned tribe" to full humanity; the solution to its backwardness would thus lie in education, not in extirpation. The first generation of anthropology—in Tylor's words, "a reformer's science" (1871, vol. 2, 453)—could consequently be seen as undermining the biologist's call to genocide by inventing and deploying ethnocentrism.

There was even a third possibility to explain the savage's backwardness. When Charles Darwin and Alfred Russel Wallace parted intellectual ways, it was indeed over this very question. If the savage has a brain equal in capabilities to that of the civilized European, then why does he not use it? Natural Selection could not create an organ that would go unused by its bearer, reasoned

Wallace—and so he opted for a supernatural explanation (Wallace 1869). "I hope you have not murdered too completely your own and my child," wrote Darwin to Wallace in March of 1869.[1] An obvious way to rescue the theory of evolution by natural selection would be to locate the savage's limitations not in the organic structure of his brain, nor in the caprice of the Creator, but in the historical circumstance of his culture.

With an alternative scientific explanation for the savage's state, anthropology at the end of the nineteenth century became the science that studied the way that humans can progress and improve, regardless of their biological ancestry or limitations. From its inception, anthropology became a science through which reform politics was possible.

American Anthropology

In the United States, the earliest American anthropology was largely a scientific polygenist rationalization for slavery (Nott and Gliddon 1854), which was mooted by the Civil War. But late nineteenth-century science turned the Native American, now "pacified," into a scientific object, in imminent danger of disappearing before being adequately studied. As a scientific program for studying Native Americans scientifically, American scholars adopted the German model—which combined [physical] anthropology, prehistory, and ethnography—and eventually appreciated the value in documenting indigenous languages as well.

When Franz Boas began teaching at Columbia University in 1897, the "four-field approach" was already well-established. The maturation of anthropology in the twentieth century began with Boas's introduction of three critical intellectual innovations emanating from the German tradition within which he had been trained. The first involved centralizing "culture" and using it in a subtly different sense than Tylor's. Rather than referring to a degree of achievement—with, say, native Australians being the least cultured and the French being the most cultured—culture instead became the diverse thoughts and lifeways themselves, and became a plural noun (Hegeman 1998). This would now refer to the intellectually integrated, locally specific knowledge transmitted from parents to offspring non-genetically, which makes each group of people cognitively distinct. All human societies were now 100% cultural, and the task of the ethnographer was to document and analyze them, not to judge or rank them.

Second, Boas institutionalized this newer usage of "culture" as an organizing principle of anthropological museum collections. The alternative involved

keeping different arrowheads together, for example, so as to illustrate the "evolution" of the arrowhead, and was useless to the ethnographer. The ethnographer needed to see all the aspects of the material culture of a specific group of people, to try and make sense of their meanings and inter-relationships (Stocking 1994).

Boas's third critical innovation was actually the most ambitious, and centered on physical anthropology. In Germany, while Ernst Haeckel was idealizing human variations and reifying them as distinct species, Rudolf Virchow—cellular pathologist, social epidemiologist, anthropologist, and political liberal—organized a massive empirical project to measure German schoolchildren. The results of that study began the eventual disentanglement of race, nation, and physical form (Zimmerman 1999). Boas, however, went further, and set out to measure the heads and bodies of immigrants, whose physical features were indeed responsive to their new environs (Gravlee et al. 2003). Boas thus came to highlight human adaptability, that is to say, the effect of the conditions of life upon the growth and development of a human body.

Anthropology in the early twentieth century thus provided culture and human adaptability as two important alternatives to hard-line genetic determinism or hereditarianism (Nelkin and Lindee 1995). Human genetics often seemed indistinguishable from hereditarianism: the first English text of Mendelism concluded with the biopolitical inference that "Permanent progress is a question of breeding rather than of pedagogics; a matter of gametes, not of training … [T] the creature is not made but born" (Punnett 1905, 60). The first American textbook on the subject argued for allelic feebleminded-ness as the cause of poverty, crime, and backwardness (Davenport 1911).

Since the field of human genetics was virtually indistinguishable from its application as eugenics up until the early 1930s—particularly human genetics that called for sterilizing the poor and restricting immigration on account of bad genes—a bitter ideological battle for control of anthropology arose. The eugenicists Charles Davenport and Madison Grant (1916) were committed to the proposition that civilization inhered in the gene pools, while Franz Boas (1911) identified civilization in historical relations, not the genes.

The Science of Race

The field that sat uneasily between them was physical anthropology, at the interface of biology and anthropology, led by Aleš Hrdlička at the Smithsonian and Earnest Hooton at Harvard. While maintaining cordial and respectful relations with Boas, Hrdlička and Hooton also sat on the large "Advisory

Board" of the American Eugenics Society in the 1920s, along with nearly every biologist and geneticist of note. The scientific status of physical anthropology was appropriately precarious. When founding *The American Journal of Physical Anthropology* in 1918, Hrdlička was willing to put the proto-Nazi Madison Grant on its inaugural editorial board, if Grant would subsidize its publication (Spiro 2009). Grant reneged, Hrdlička replaced him with Franz Boas, and then invited Boas to review Grant's notorious *The Passing of the Great Race* in the new journal.[2] Boas obliged by calling Grant's book "hardly a subject for a review in a scientific journal," and lacking "a claim to consideration as a scientific contribution," which may accidentally leave an "impression upon the minds of uninformed readers that the book has merit as a work of science" (Boas 1918, 363). The right to speak for science was a crucial rhetorical property.

Ales Hrdlička and Earnest Hooton, as the leading physical anthropologists in America, were consequently also the leading experts on race, and specifically on how to diagnose one group of people from another from their bodies or heads. Hrdlička trained in medicine, and subsequently in the French school of craniometry, while Hooton trained in classics, and subsequently in British bio-archaeology. While respectful and cordial to Boas, neither had a strong interest in Boasian human adaptability, and Hooton in particular was committed to the idea that there is an intimate and causal relationship between one's looks and one's thoughts or acts. Consequently, he struggled largely in vain to differentiate good American physical anthropology from its evil German counterpart (Hooton 1936). Long after Charles Davenport's racist and eugenical ideas had fallen out of favor in the American genetics community, Earnest Hooton nominated him for the presidency of American Association of Physical Anthropologists, a position Davenport held when he died in 1944 (Little and Kennedy 2010).

Physical anthropology had begun as the science that naturalistically explained European political and economic global dominance. That dominance became in a sense inevitable, because Europeans possessed better brains, in better skulls; and physical anthropology was the science that documented that distinction. The generation of physical anthropologists after World War II, however, constructed a very different science than they inherited. Earnest Hooton's former graduate students practiced a "new physical anthropology," christened by Sherwood Washburn (1951); indeed by the end of the century, most practitioners would call themselves "biological" rather than "physical" anthropologists (Fuentes 2010). This anthropology would be more closely aligned with intellectual trends in evolutionary biology, and would see active intellectual engagements and collaborations with Theodosius Dobzhansky, George Gaylord Simpson, and Ernst Mayr.

The field that began the century as the science of race now became the science of human microevolutionary variation. The distinction is crucial, for empirical studies in the 1950s and 1960s increasingly demonstrated that without a priori racial lenses, patterns of human diversity do not easily sort themselves out in the taxonomic fashion that race is supposed to summarize. The British physical anthropologist Joseph Weiner could describe the human species "as constituting a widespread network of more-or-less interrelated, ecologically adapted and functional entities" (1957, 80). In 1962, genetic anthropologist Frank Livingstone noted that most human features vary gradually across geography; thus to summarize human diversity, "There are no races, there are only clines" (Livingstone 1962, 279). A decade later, Richard Lewontin (1972) would demonstrate that even the clinal variation constituted a small part of human genetic diversity, the great bulk of which was actually cosmopolitan or polymorphic, with the same genetic variants being found nearly everywhere.

There were probably three reasons that the ontology of race came to be interrogated at this time. First, race had already morphed once over the first few decades of the twentieth century and was consequently not necessarily a stable concept. At the beginning of the century, race was understood as an ancient inheritance—an essential, undiluted property. Here, race inheres in the person, who embodies the race. But by mid-century, race had been reconceptualized as a population, the critical difference being that now the person was in a race, rather than vice-versa (Huxley and Haddon 1935; Montagu 1942; Boyd 1950). Having already been biologically "real" in two entirely different ways, its biological reality was actually a bit less obvious than it had previously seemed.

The second reason involved the maturation of the generation of anthropologists who had served in World War II alongside black soldiers, and who were now tenured professors, but with life experiences quite different from those of their professors (Frederick S. Hulse, personal communication). And the third was the confrontation between the "old" and "new" physical anthropologies. Carleton Coon (1962), a classical physical anthropologist, prominent public intellectual, and sitting President of the American Association of Physical Anthropologists, developed a theory of human microevolution in which whites evolved from *Homo erectus* into *Homo sapiens* 200,000 years before blacks did. His theory resonated with his friends in the segregationist community (Jackson 2005), and in the face of harsh criticism, he postured as an objective scientist being silenced by political enemies—in this case virtually the entire membership of the American Association of Physical Anthropologists (Coon 1981; Lasker 1999).

By the 1970s, "race" had been largely abandoned as a scientific topic by physical/biological anthropology, although of course the elaboration of geographic patterns in the human species remained a major research program. Racial studies were studies of politics or history or law; the scientific study of patterns of biological diversity in the human species was "human microevolution" or "human variation." The study of human populations thus became the study of how they adapt, and the study of human differences was now complemented by a focus on the common foundations of human life—a remote ape ancestry and a more recent hunter-gatherer ancestry (Haraway 1989).

Addressing the Hereditarian Fallacy

Differentiating the study of race as a humanistic endeavor from the study of human microevolution as a scientific endeavor was a major advance for biological anthropology in the twentieth century. It identified the program of establishing a natural human taxonomy as fundamentally misguided, for there is no underlying biological taxonomic structure to the human species.

The biopolitics of hereditarianism has posed a more vexing and persistent problem. Once again, the fundamental issue is the relevance of science to equality. If people are entitled to equal rights and opportunities, then their DNA doesn't really matter. The hereditarian position is that people are not entitled to such equalities if they are by nature incapable of utilizing their opportunities effectively. This was an argument of the eugenicists in the 1920s (Allen 1983), the segregationists in the 1950s (Jackson 2005), and later in *The Bell Curve* (Herrnstein and Murray 1994), by a psychologist and a political scientist.

The Bell Curve's argument was consequently familiar to those knowledgeable about twentieth-century biopolitics. The book made unreasonable assumptions about intelligence (as a significantly innate, scalar quantity, accurately assessable by paper-and-pencil tests), about genetics (that the geneticist's statistic called "heritability" is a measurement of a trait's innateness), and about the properties of human groups (which are quite malleable). The conclusion was that the poor are irremediably unintelligent, and thus social programs were doomed to failure, and consequently not worth pursuing.

The impact of *The Bell Curve* was enormous, in large measure because mainstream scholars thought that those assumptions had been put to rest decades earlier, and they were caught unprepared for the media blitz that called into question decades of scholarship about human variation (Kincheloe et al. 1996).

The connection between hereditarianism and racism is critical to recognize. If one's fate is largely set by one's genes, and human groups are naturalistically constituted units, then one could argue that the fates of human groups are set by their genes. In a sense, then, hereditarianism could afford a larger umbrella under which to subsume classic scientific racism. Consequently, *The Bell Curve* advanced the argument that about 6 points of the average 15-point differential in IQ between US whites and blacks was attributable to genetic differences between the European and African gene pools. And since the African gene pool was now the natural intellectual inferior of the European gene pool, it followed that social programs mistakenly presuming their equivalence would be a waste of money. This argument had been advanced earlier by psychologist Arthur Jensen (1969) in the backlash that followed the Civil Rights movement. Jensen and physicist William Shockley (Shurkin 2006) had nursed old arguments from the 1920s about inherent inferiorities, intelligence, and breeding programs, and repackaged them for the 1970s.

Now, in the 1990s, the arguments resurfaced again. *The Bell Curve* paid its intellectual debt to Jensen explicitly, citing over 20 of his papers. And it also cited over 20 papers by a lesser-known Canadian psychologist, Philippe Rushton—and went on to defend his work pre-emptively in an Appendix, as "not that of a crackpot or bigot" and "plainly science" (Herrnstein and Murray 1994, 667). But once Rushton's work became more widely known, the crude and archaic folk ideologies that propped up *The Bell Curve* became clearer.

Philippe Rushton (e.g., 1995) believed that Europeans, Asians, and Africans had undergone separate evolutionary histories. Africans had evolved to be prolific, Asians to be intelligent, and Europeans to be the happy medium. He assessed these differences with surrogate measures, including crime rate, IQ, brain size, penis size, and self-reported measures of libido. He believed that the average IQ of indigenous Africans was about 70, equivalent to a handicapped European, and set by evolutionary genetics. The scholarly journal *Animal Behaviour* reviewed his work in uncompromising terms, concluding: "Bad science and virulent racial prejudice drip like pus from nearly every page of this despicable book" (Barash 1995, 1133).

The connection between *The Bell Curve* and Philippe Rushton led to the revelation that there existed philanthropies quietly dedicated to the promotion of these archaic ideas within the scientific community (Lane 1995). The most prominent of these is known as the Pioneer Fund (Tucker 2002; Lombardo 2002). Its top three beneficiaries included Arthur Jensen and Philippe Rushton (2002). When Rushton died, he was its sitting president.

Perhaps the oddest work of all that the Pioneer Fund supported involved studies of identical twins reared apart, whose behavioral convergences might

be interpreted as evidence for cryptic underlying genetic control—since they presumably shared the same genomes but different environments. While this seems like an interesting natural experiment in principle, in reality the mythology and politics make the scientific value of these studies very dubious. An influential British study of the genetic influence on IQ in twins in the early twentieth century turned out to have been largely imaginary (Judson 2004).

The new centerpiece is the 1979 story of two identical twins separated at birth and given the same name by their adoptive parents—Jim. They reunited at age 39, the story goes, and discovered that they shared amazing biographical details. They had both married women named Linda, gotten divorced, and remarried women named Betty. They gave their sons the same name. They each had a dog named Toy, smoked Camels, and even drove similar cars.

While possibly interesting as the opening of a campfire story, it transparently has no value for the science of human genetics. No self-respecting geneticist has ever believed that there is cryptic genetic control over the name you give your dog, or the name of the woman you marry. Nevertheless, with the goal of proving that general mental properties are largely innate, psychologist Thomas Bouchard followed up "the Jim Twins" and established the Minnesota Study of Twins Reared Apart over the course of 1979, collecting amazing stories of separated twins, with the financial support of the Pioneer Fund (Segal 2012).

Despite its preposterousness as science—after all, even if every detail of the story is absolutely true, it is still no more a story of genetics than of Extra-Sensory Perception (ESP)—the Jim twins and their psychologists were represented with utter credulity by the scientific media. They were written up no less than three times—in 1980, 1987, and 2009—by the news department of *Science*, the leading science journal in America (Holden 1980, 1987, 2009). The Pioneer Fund managed to gain temporary respectability for these stories of identical twins reared apart as ostensible evidence for the genetics of personality, but the conclusions do not stand up to the merest epistemological scrutiny (Joseph 2014). Whatever the stories about the similarities between separated twins may be, they are not genetic data.

The Bell Curve inadvertently succeeded in exposing a trail of scientific racism that most scientists were unfamiliar with and forced the scientific community to confront again the moral question of evil purveyed as science. Coincidentally, historians toward the end of the twentieth century were also revisiting the moral issues associated with the American eugenics movement, decades earlier (Kevles 1985).

Any scientific discourse on human origins and diversity is simultaneously a moral discourse, for we inhabit a universe of both ape ancestors and of social

inequality. Clarence Darrow came to this realization during the famous Monkey Trial of 1925, when Tennessee convicted schoolteacher John T. Scopes of the crime of teaching evolution to high school students. Darrow, attorney for the defense, was the hero of the scientific community for defending Darwinism. But upon reading the biology textbook out of which Scopes was accused of teaching (Hunter 1914), Darrow realized that it presented ideas about white supremacy, and sterilizing the poor on account of their bad genes, with the same authority that it presented photosynthesis and Darwinism. Worse, the biologists themselves saw no problem; the textbook accurately represented the normative biology of the day. Shortly after the end of the trial, Darrow began attacking the very same biologists whose views of human evolution he had just been defending, for their views on living peoples (Darrow 1925, 1926).

In the intervening decades, serious scholars of human variation have recognized that the science has never, does not, and cannot take place in a cultural, political, or moral vacuum. The suggestion that science is amoral and can only be used or abused is fundamentally undermined in the study of scientific racism. Generations of students of human diversity have inscribed their social values into their science—into the framing of the research and collection and analysis of the data (Fabian 2010; Keevak 2011; Morning 2011; Roberts 2011; Nelson 2016). We can never free the science from the politics, for we ourselves are cultural actors performing the science. We study the history of this science in order to understand the politics of the science, so that we can improve the science by inscribing more benign social values into it than our predecessors did.

Human Diversity as a Twenty-First-Century Moral Science

The study of human diversity became divorced from the study of race over the course of the twentieth century. Engaging with that history entails confronting its two central fallacies: the reification of races and the naturalization of inequality. While these ought to be as thoroughly repudiated as young-earth creationism in science—the products of antiquated ideologies—nevertheless, the unique biopolitics of race consign these particular fallacies to be continually probed or reinvented.

The reification of races lies in the background of "pharmacogenomics" as ready-made niche markets for drug companies. The path was cleared by BiDil,

which was approved by the US Federal Drug Administration (FDA) in 2005 to treat cardiovascular disease in specifically African-Americans, despite the absence of any valid epidemiological evidence that it worked better or differently in African-Americans than in anybody else.

The theory behind racial pharmacogenomics is highly essentialist: that members of different races would be physiologically distinct, thus necessitating different medical interventions. But basic population genetics dictates that any differential intervention based on genetic differences ought to be determined by the individual patient's genotype, for which race is a very poor surrogate, since human populations are highly polymorphic. That is, human populations tend to have the same alleles, but at different frequencies; consequently, most genotypes can be found in most populations. To base a dosage or an intervention on a patient's census assignment, rather than on the patient's actual genotype, is to risk introducing a high proportion of misdiagnosis and mistreatment.

Racializing BiDil served to extend its patent protection. Yet BiDil was a failure—although not because of the racialized pseudoscience behind it but rather because of its over-pricing. BiDil was never a public health measure, but a profit-generating venture from the very outset (Kahn 2012).

The other fallacy, naturalizing inequality, also makes a surprising appearance in twenty-first-century science. The leading science journal in America published two papers in 2005 which claimed to have identified brain mutations in genes known as MCPH-1 and ASPM, which differentiated Africans from Eurasians, arose several thousand years ago, and might even have been responsible for the rise of civilization, although as the scientists coyly added, "the significance of this [temporal] correlation is not yet clear" (Mekel-Bobrov et al. 2005, 1722). Less subtly, the senior investigator told *The Wall Street Journal* that he "favors the idea that the advantage conferred by the mutations was a bigger and smarter brain" (Regalado 2006).

Scanning the genome to explain the cranial shortcomings of Africans seems a hardly worthwhile endeavor for twenty-first-century science, except toward a radically biopolitical end (Richardson 2011). Crucially, the issue is not whether human genetic diversity should be explored (contra Lahn and Ebenstein 2009), but rather whether it should be explored by scientists who work within a racist intellectual paradigm. One would hardly expect the scientific community to support a study of bipedalism by creationists.

Sadly, while twentieth-century science successfully undermined race as a naturalistic category of people, it was largely impotent or irrelevant in the face of racism, an immoral political ideology (Sussman 2014). Unlike race, to study racism critically involves the introduction of moral discourses. Racism is about human rights, not human diversity.

The case of Philippe Rushton (above) is instructive. Once you become acquainted with his work in depth—misapplying evolutionary ideas to explain the presumptive intellectual inferiority and sexual superiority of Africans—it is hard to judge his work as anything but odious archaic ideologies relabeled for modern scientific consumption. And not simply racist, but egregiously, even ludicrously so. And yet, as a scientific racist, he was able to have a successful—if somewhat notorious—career as an academician and philanthropist. He continued to publish in psychology journals; psychologists took his work seriously. The journal *Personality and Individual Differences* even devoted its issue of July 2013 to a memorial in Rushton's honor.

The toleration of scientific racism thus presents a moral dilemma for modern science, which certainly has no room for creationism, astral projection, or the transmutation of base metals into gold—ideas that we reject as archaic or inane. Racism must ultimately come to occupy a spot external to science, like alchemy and ESP, but until it does, it is far more harmful than other ideologies. The only value of race in contemporary bio-medicine is to aid in the amelioration of racial differences in health care. To the extent that ancestry may be an individual genetic risk factor, it is only confounded by race.

Race is not the only moral issue with which modern human population genetics is forced to grapple, however. Population geneticists sought funding in the 1990s for a Human Genome Diversity Project, but were denied it, for their failure to grapple with the ethical issues they raised (Reardon 2004). The most important legacy of the Human Genome Diversity Project was its unwitting development of ancestry as a genomic commodity, as genomics was becoming absorbed into corporate biotechnology. Genomes can generate a probabilistic quantitative relationship between an unknown DNA sample and a panel of indigenous DNA samples. For the sake of simplicity, we can call that pattern of genomic similarity "ancestry," and there is indeed a market for it.

Yet while the classical goal of science is the production of knowledge, corporate science introduces an additional goal: the production of profits (Bolnick et al. 2007). These goals may of course happily coincide, but they nevertheless introduce a potential conflict of interests evocative of the one identified by Jesus in the Sermon on the Mount (Matthew 6:24): If you cannot serve both God and money, can you serve both genomics and money? Indeed, these genomic ancestry tests have no legal standing and are obliged to identify themselves as "recreational." Consequently, although there are tests available to tell whether you have the Y-chromosome of Moses or Genghis Khan, or are descended from a European Pleistocene clan mother or Zulu warrior, or you are 8% Native American and 2.6% Neanderthal, you cannot necessarily take

them at face value merely because they are science (Thomas 2013). The truth value claims of this science are compromised by their source.

Here once again we probe the boundaries of our traditional ideas about science, for we have entered another portal into a domain that scientists are generally not trained to work with or to think about, namely, the domain of moral discourses. We have already noted the merging of scientific and corporate moralities. Yet any corporate venture that sells kinship with indigenes to Americans as its product is literally fueled by the blood of native peoples. However, the political and economic environments that make it possible are no longer invisible, and the power relations that made it possible in earlier times no longer exist (Marks 2010). There is no international legislation covering the collection, storage, or repatriation of biological remains, but certainly moral attitudes are evolving, and we are presently experiencing the development of normative post-colonial scientific practices (TallBear 2013).

Finally, we are increasingly compelled to engage with the moral issue of repeatedly trying to explain human history in terms of nature, rather than of history. Reactionary scholars in the nineteenth and twentieth centuries attempted to rationalize global political and economic inequality by recourse to imaginary naturalistic differences between the dominant and the poorer classes or nations (Gobineau 1853). The social sciences were founded on a repudiation of this idea; and saw rather that the domains of the social and biological are phenomenologically distinct, and thus social facts can only be explained by prior social facts (Durkheim 1895). Whether the sociopolitical present is explicable by skulls (Coon 1962), genes (Darlington 1969; Cochran and Harpending 2009; Wade 2014), or geography (Diamond 1997), this removes contingency and agency from the analysis of human history and replaces it with crude teleological reasoning. That is, history becomes the unfolding or playing out of deeper differences between groups of people—rather than the consequence of decisions made and actions taken.

Attempting to reduce human social history to biology is an intensely political and moral exercise, absolving actors of moral judgments, relegating history itself to insignificance, and even negating the possibility of learning from history, if it is just an unfolding of nature. Early sociobiologists (Wilson 1975) were quite naive about this, and their successors (e.g., Ridley 2015) navigate this moral landscape uneasily.

Science has opposed "nature" to "nurture" at least since Galton (1874). We would like to transcend this facile dichotomy in the twenty-first century, and study the relationship between the facts of biology and those of political and economic history—for example, in the ways that racism is inscribed upon the physical body (Krieger 2005), or that human evolution is a fundamentally

biocultural history (Fuentes 2015). Unfortunately, however, the constant engagement with scientific racism and other reductive, naturalistic ideologies of the human condition impedes the progress of this evolutionary science. We are constrained to work within the biology-culture dichotomy to perpetually engage the scientific racists and hereditarians, whose ideas exploit the authority of science, toward an end that most citizens of the modern world find disreputable—the maintenance of social inequality (Fields and Fields 2012). Most sciences only have to engage with scholarship that is false, but this one must continually engage with scholarship that is both false and evil.

Notes

1. Darwin Correspondence Project, "Letter no. 6684," accessed on 4 October 2016, http://www.darwinproject.ac.uk/DCP-LETT-6684
2. Aleš Hrdlička to Franz Boas, 6 May 1918. National Anthropological Archives, Washington, DC.

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7

Experimenting in the Biosocial: The Strange Case of Twin Research

William Viney

Twins can be viewed as pivotal instruments in the articulation of modern scientific reason. Twin methodologies—using bodies individual, paired, or aggregated into statistical populations—and their derived datasets constitute a collective asset to twin researchers and others who have claimed an understanding of human development, health, and wellbeing through the use of twin-born people.[1] Both historical and contemporary uses of human twins in the life and human sciences provide fascinating instances of a human group instrumentalised in the advancement of competing forms of scientific inquiry, and their cultural identities partly formed through their participation in those competing visions of human life. Especially though not exclusively in the case of monozygotic ('identical') twins, the appeal of twin bodies may simply be optical: higher-order, developmental, and evolutionary processes thought to shape human health and behaviour, can be seen to take phenotypic expression in twin bodies. As 'living laboratories' championed for their capacity to isolate interior 'biological' mechanisms from external 'environmental' influences, twin studies have become synonymous with 'gene-centric' models of human behaviour and wellbeing. But because twins are presented by twin researchers as having an embodied and mediatory capacity within the process of research design and dissemination, twins are now being used as the living proof of new, complex settlements found in postgenomics—such as epigenetic, microbial, or metabolomic research—to complicate the partition between nature and nurture or dissolve it altogether (van Dongen et al. 2012). It is important to

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stress, therefore, that twins and twin data in the twentieth and twenty-first centuries have played an important and appropriately duplicitous role in defining, defending, and disrupting the polarities between what is biological and what is social.

This chapter describes the 'biosocial life' of twins as it has emerged in the sciences; the forms of life that emerge for twins through their participation in biomedical research science. In particular, it describes them as flexible figures taken to be the vitally epistemic things of experimental use and evidence, caught in the processes of research design, description, publication, and public dissemination. Twins who participate in and become used in such research are human brokers, elected go-betweens in the emergent assignations of 'the biological' and 'the social'. Though the use of twins in biomedical research has led to atrocity, debate, and controversy, the critical position adopted in this chapter seeks to understand why twins have been used and continue to be used despite critical attempts to disavow twin research methods (Davis 2014; de Nooy 2005, 90–93; Joseph 2003). Although the historical and political consequences of twin research cannot be expunged from any discussion of its past, present, or future ambitions, this chapter avoids the binary of advocacy or condemnation to explain the methodological persistence of twin models and the socialites they engender, from early to mid-twentieth-century transmission genetics to late twentieth- and twenty-first-century transition genomics, small-scale cohort studies to large-scale, computationally complex systems biology (Barnes and Dupré 2008). The remarkable longevity of twin research—used to stress the relative importance of biology or society in human life, to prove and disprove divisions between biology and environment—have repercussions for how we understand how human groups socialised into and through scientific endeavours. It is not simply the case that human twins are made to be the passive bearers of laboratory results formed within the life sciences, neither is it simply a matter of recognising how twins are the active constituents of novel 'biosocial' formations; rather, twins actively volunteer for, have their identities transformed by, and seek to adapt and inform the discrete workings of scientific knowledge production because the scientific *and* social basis of their twinship preexists their inclusion in experimental science. Finally, a focus on how the meaning of twins, as informed by their scientific utility, facilitates a reappraisal of social scientific approaches that define modern and contemporary twinning according to its dyadic dynamicism (Hoctor 2015; Davis 2014; Piontelli 2008) and 'supraindividuality' (Stewart 2000, 169). While twinning has been linked to the specific uterine quality of their co-development or to generalised patterns of psychological development said to be unique to twins (Joseph 2003; Leonard 1961; Burlingham 1945),

what has yet to be fully realised is how these conceptions of twin sociality are enhanced, diminished, or negated by being clinical labourers within scientific enterprise.

Born into Experiment

To recognise the historical circumstances by which human twins have entered the life sciences is to recognise how the division between 'biology' and 'society', as well as the subsequent attempts to renegotiate or even dissolve this division in postgenomic laboratory sciences, is marked by its historical attachment to the hereditarian twin research of British polymath, Francis Galton (1822–1911) (Meloni 2016). Galton should not only be credited as the first scientist to use twins in the formal, qualitative and quantitative biometric study of human development, ageing, and disease (Waller 2012), his studies of twins are simultaneously noted for advancing the polar distinction between 'nature' and 'nurture' into the scientific and popular imaginary.[2] In his 'The History of Twins, as a Criterion of the Relative Powers of Nature and Nurture' (1875), Galton observed the phenotypic diversity of twins—'strongly alike, moderately alike, and extremely dissimilar'. Though Galton lacked an embryological or genetic understanding of twin difference—categories such as monozygotic twins (abbreviated to 'MZ' or colloquially known as 'identical') or dizygotic twins (abbreviated to 'DZ' or colloquially known as 'non-identical') would not become firmly established and incorporated into experimental designs until the 1910s and 1920s—he made them equal in scientific utility. He compiled biographical information for 94 sets of twins by postal correspondence (Galton 1875b, 566). Studying these twins offered Galton a means to respond to complaints that earlier measures of heredity overlooked the role of chance, so that 'some new method [was required] by which it would be possible to weigh in just scales the effects of Nature and Nurture, and to ascertain their respective shares in framing the disposition and intellectual ability of men. The life-history of twins supplies what I wanted' (Galton 1875b, 566). As a consequence, narratives of twin lives, when reported to a trained researcher, became a methodology that could confirm and also measure the influence of biology or society, nature or nurture.

The wider aims of Galton's early use of twins also shaped the epistemological imperatives and research objectives of subsequent twin research using these people. First, Galton aimed to know whether twins regarded as highly similar at birth could develop physical and behavioural differences. Second, he wanted to know whether twins who were considered to be dissimilar at birth

could grow more alike in later life, meaning that the logic of between-pair concordance and discordance determined a scalar approach to trait analysis. Galton concluded that twins 'either grow unlike through the development of natural characteristics which had lain dormant at first, or else they continue their lives, keeping time like two watches, hardly to be thrown out of accord except for some physical jar' (Galton 1875b, 574). He had a strong conviction that twins of varying types made legible an interior component that guided physiological development. This interior 'mechanism' was immune and indifferent to external interference, pervious only to the most extreme circumstances, so that any evidence of between-twin concordance or discordance reflected the strength of heritable, biological characteristics.

Twin research has changed dramatically in scale and levels of sophistication since the end of the nineteenth century, yet two further aspects of Galton's use of twins are important to underline for his influence on subsequent research designs and findings. One concerns the exclusivity and significance placed upon twins as the methodological means by which to achieve certain kinds of scientific observation. The other concerns how that methodological importance, next to the evidence Galton drew from twins, extends throughout their 'life-histories'. As a consequence, cases of mistaken identity, attempted and achieved suicide, toothaches, malformed fingers, even the slow movement down a flight of stairs, were all felt by Galton to connect to the 'inner clockwork' whose influence he sought to expose. The twin body had become an abundant source of evidence, a place where traits could be calculated and placed within a scale of difference, not as the testing ground for new hypotheses about human life but as the living, embodied, and unmediated site of experimental observation.

There are now numerous methods of using twins in research, extending across the life and social sciences. Separate, discipline-specific histories can be written about how twin research has helped to form priorities in epidemiology, psychiatry, genetics, behaviour genetics, molecular genetics, and more recent -omics research. I wish to outline the most significant twentieth-century designs and their historic uses before discussing current applications. Each design approaches the 'nature and nurture' divide that Galton claimed twins exemplify from a different point of view, but each accepts that a division between the two is either actual or necessary to impose by statistical means.[3] The principal use of twins in biomedical research in the twentieth century was to calculate heritability scores. These measure the quantity of variance in a given trait that can be attributed to either genetics or to the environment. In the 1920s Herman Weiner Siemens is said to have invented the 'classic twin method' in order to calculate the trait resemblance between reared-together

monozygotic ('identical') and same sex-dizygotic ('fraternal') twins (Siemens 1924). It is a method of research that already assumes a quantitative genetic resemblance between those two twin types—its effectiveness depends on how monozygotic twins, who are born from the same zygote, share 100 per cent of their genes, while dizygotic twins, born from two separate eggs fertilised by different sperm, are said to share 50 per cent (Plomin, et al. 2008, 79; Barnes and Dupré 2008, 98–99). Based on this creation of two different types of twin based on zygosity, each individual in each zygotic group is measured for a trait and a numeric index of heritability ranging from 0.0 (no genetic contribution) to 1.0 (complete heritability) can be generated to express a ratio of between-pair variation to total variation for that given trait, otherwise known as an intraclass correlation (Ball and Teo 2008, 473). On this comparative basis, the classical twin method can help to estimate the heritability of a trait: the proportion of the variance in a given population that can be attributed to genetic variance. Following Galton's recognition that twins could be used to study *any* phenotype studies in the twentieth and twenty-first centuries have focussed on diverse and complex behaviours such as nail-biting (Ooki 2005), loneliness (Boomsma et al. 2005), mobile telephone use (Miller et al. 2012), intelligence (Haworth et al. 2010), sexual dysfunction (Burri et al. 2012), and happiness (Bartels and Boomsma 2009). Twins research has made these diverse behaviours not only more visible but visible through the lens of genetic variation.

After or in conjunction with the classical twin method, the second way of using twins in biomedical research employs twins reared apart. This methodology includes twins of both zygosities who have been separated, through adoption or by other means, at or near the time of their birth, and then studied in later life in order to model how shared genes and different environments have affected them. Early pioneers of this method include Horatio Newman, Frank Freeman, and Karl Holzinger in Chicago in 1937, British psychologist James Shields in 1962, and Danish psychiatrist Niels Juel-Nielsen in 1965, all of whom compiled studies of twins reared apart. None compare in scale (137 reared-apart pairs), longevity (20 years), or number of published outputs (more than 150 papers and chapters) to the Minnesota Study of Twins Reared Apart (MISTRA) (Segal 2012). As a logistically complicated and expensive form of twin research, the size of the MISTRA study almost certainly flourished thanks, in part, to the long-standing and wonder-struck narratives of visually alike twins being reunited; narratives that privilege what are now known as monozygotic twins but which historically intersect modern zygotic categories (Kooper 1994). Harnessing extra-clinical fascination about twinning, as well as the commercial opportunities that may arise in their

reunion, television and print media acted as a recruitment tool; through their appearance in the media, twins became an influential part of the study's methods of research dissemination (Segal 2012, 104). Working in union with classical twin methods using reared-together twin data, and also using dizygotic twins reared apart for added insight, the MISTRA group argued that their data showed how monozygotic reared-apart twins were viewed as broadly sharing intraclass correlations with reared together twins for a range of personality traits, including controversial measures of general 'intelligence' (Segal 2012, 100). They took this as proof of the marginal influence of non-shared environmental effects compared to genetics, and used their twin data to conclude 'about 70% of the variance in IQ was found to be associated with genetic variation' (Bouchard et al. 1990). While certainly not the sole finding from the 15,000 questions asked of each twin pair during a week-long assessment process, MISTRA's emphasis on the genetic component of complex traits left many concerned about the study's methodological weaknesses and potential policy implications: the relatively small sample size; the amount of time twins had spent together either before or after their separation; the way in which confidential protocols made data inaccessible to other researchers; more deep-seated concerns about how the stress upon 'genetic' influence over, say, intelligence, implies the actual or potential irrelevance of policies aiming to improve educational attainment and, by proxy, to tackling social, economic, and health inequalities (Lewontin et al. 1985; Segal 2012; Ashbury and Plomin 2014).

The final method of using twins in biomedical research is by using monozygotic twins who are discordant for a particular trait or disease. It also assumes that they 'share' almost all their genes. Phenotypically discordant monozygotic twins, however, are useful to researchers who want to understand how those deemed genetically alike can develop divergent health experiences. Comparable to the classical twin methods for its development within a context of twentieth-century eugenics, the discordant monozygotic technique was pioneered in the 1910s and 1920s by German researchers such as Heinrich Wilhelm Poll (1877–1939), Hermann Werner Siemens (1891–1969), and Otmar Freiherr von Verschuer (1896–1969) (Joseph 2003; Teo and Ball 2009; Roelcke 2013). Heinrich Wilhelm Poll, an advocate for and victim of scientifically driven policies of racial hygiene, promoted the importance of discordant twin research: 'the well-planned and critical investigation of each suspected inherited character for its modification in MZ [monozygotic] twins must be conducted as an essential first step in all human genetics investigations' (Poll 1914). Such methodological priorities had a direct influence over Horatio Newman (1875–1957) and colleagues at the University of Chicago,

who helped to establish twin studies in North America after World War II and inspired more extensive uses of the design at the University of Minnesota (Newman et al. 1937). In the years that followed the purpose of twin research moved away from proving the scientific basis for state-organised eugenics, yet the celebratory attitude towards the experimental possibility of monozygotic twins has scarcely changed in over 100 years of twin research. Robert Plomin, a leading behavioural geneticist and twin researcher, has argued that MZs differ for a given phenotype are 'a sharp scalpel for dissecting non-shared environmental effects from genetic effects' (Plomin 2011, 584). This is because the lives of monozygotic twins can be studied in order to disentangle how their behaviour and lifestyle interact with and act upon what is understood as a common genome, the cause of a considerable resurgence of discordant monozygotic twin designs in an era of postgenomic science (Castillo-Fernandez and Spector 2014; van Dongen et al. 2012; Bell and Spector 2011). These are the hopes and expectations, the version of twin research most eagerly reported by popular print, television and film media, since it appears amenable to dyadic narratives of nature vs. nurture, nature-nurture, nature-through-nurture (e.g. Mukherjee 2016; Spector 2012; Miller 2012). What is particularly interesting for the sake of this volume is that monozygotic twins are felt to harbour within them a 'nature' that comes into contact with and dwells within an 'environment'. If postgenomic research has sought the 'molecularization of biography and milieu' (Niewöhner 2011, 279) to understand the malleability of the human body, then the severance between, or imagined unification of, nature and nurture is internalised within the vital materiality of experimental twin designs: researchers can use twins to present their experiments and demonstrate the underlying dichotomy guiding human life without then having to translate findings into another kind of living body. The spheres of biology and society, like twins themselves, are then viewed as component parts acting within a wider ecosystem. Without the burden of analogy or translation, these twin studies promise to isolate genetic regions, life events, or behavioural patterns that can account for phenotypic discordance between pairs, leaving twins to operate as rhetorical and experimental vehicles for lived conceptions of the biological and social.

It is not my principal ambition to evaluate, as many others have done, the faults levelled against the methods and assumptions that guide twin studies: criticisms that range from accusations of genetic determinism; reductionism, the simplified genetic comparison between mono- and dizygotic twins, or the confusion over what a 'non-shared' environment means. Perhaps the most frequently cited criticism of classic twin models is the assumption that twins of different zygosities share environments in the same way (the so-called

equal-environment assumption); studies suggest that monozygotic twins are far more likely to be treated alike and exposed to similar environments than their dizygotic counterparts (Stevens and Richardson 2015; Joseph 2013; Ball and Teo 2008). If we doubt the equal-environment assumption, then not only do mono- and dizygotic twins fail to operate as a control population for the other, they no longer stand as a representative group for singletons, since they experience an environment that is specific to their twinship. Twin research has caused particular controversy when applied in psychology, psychiatry, and allied fields when false equivalences can be implied between complex, time-specific behaviours such as 'criminality' or 'fingernail-biting'. These, once made into heritability scores, can be and are easily mistaken by those not familiar with twin research for universal indices of genetic determination rather than as a measure of trait variation attributed to genes within a specific population, within a particular time and place (Burt and Simons 2014). When misunderstood in this way, measures of heritability for psychosis, schizophrenia and other expressions of mental ill health have led to a focus on the genetic determinants that may underlie these conditions to the relative neglect of other causes (Bentall 2009, 123–127). Elsewhere, more detailed levels of genomic analysis, combined with the hybrid effects of mosacism and chimericism, have led some to argue that the idea of monozygotic twins 'sharing' a genome is misleading (Dupré 2015; Barnes and Dupré 2008). Taken together, these doubts about twin research have led its most vociferous opponents to demand it be 'relegated to its proper place alongside the discarded pseudosciences of bygone eras, such as phrenology, alchemy, and craniometry' (Joseph 2003, 244).

Critics of classical and other twin methods have either perpetuated distinctions between nature and nurture—focusing on methodological weaknesses and neglecting to engage with the underlying dichotomies guiding this research—or they have sought to reverse the genetic tendencies of twin research by stressing the ways that environments and the independent agency of individuals have a greater effect on the expression of behavioural traits (Davis 2014; Piontelli 2008; Joseph 2003; Stewart 2000). They do this at the cost of understanding how the biosocial status of twins is at stake when participating in biomedical research. Focusing on the results that twin research produces, and levelling criticism towards the universal descriptions of human health and behaviour resulting from twin data, has meant that the status and position of twins within research science is considered of secondary or derivative importance. With the exception of Davis (2014), whose negative view of twin research stems from an assumption that all research scientists treat all twin participants as 'zombies or performing monkeys' (37), simpler questions

about the relationship between twins and twin research are not being asked: why should twins continue to volunteer their time, energy, emotions, and bodily matter to the advancement of a publically contested science? In later stages of this chapter, the development and transformation of twin research will be shown to depend on the recruitment and retention of a particular variety of clinical labourer, whose participation in research presupposes, informs, and legitimises the social status of twins, even in a postgenomic era that appears set to unravel the Galtonian nature/nurture distinction that brought twins into scientific studies in the first place.

Experimental Bodies

Having outlined some of the multiple ways by which diverse kinds of twins have been drawn into life and human science, largely to assert the division between 'genes' and 'environment', it is important to stress the terms by which twins have been promoted by twin researchers as experimentally significant. Doing so means that we can turn to why twin research and its associated controversies have persisted from an era of gene-centric, transition biology to more complex, contingent and entangled models of human life (Stevens and Richardson 2015; Barnes and Dupré 2008). Doing so means we can understand why the use of twins, while instrumental in bringing the division of 'nature' and 'nurture' into public discourse, remains a favoured method by which to conduct biomedical science even while the desired research findings seek to move beyond polarities of nature, nurture, and interactionalist metaphors of their correspondence (Keller 2015).

Natural experiments are prized as a gold standard among health and evolutionary scientists for their scale and variety of time, place, and observed specimen, since they allow for experimental conditions that cannot be generated in field or laboratory conditions and can reveal end results of ecological and evolutionary processes over long durations (Diamond 1983). The language that has validated the use of twins—whether using classical, discordant, reared apart, or combined methods—has been dominated by naturalism; the research achieved through twin bodies is neither confined to the artificial environments of laboratory-reared organisms or to the analogous caesuras that attend the use of model organisms. It also means that twin researchers can present their methods and findings as grounded in 'natural' phenomena. Twin research attains an abundant autonomy for those researchers who have come to depend upon and present twin studies as a 'naturally occurring experiment' (Smith et al. 2012, 12), and 'experiment[s] of nature' (Plomin et al. 2008, 38). Nancy

Segal, researcher for, and historian of MISTRA, claims that twins are 'an experiment of nature' (Segal 2012, 62)—a powerful investigatory community who are capable of generating 'unique insights […] simply by acting naturally' (Segal 2010, 317). The presence of twins continues to energise a powerful experimental licence. Thomas Bouchard, leader of MISTRA, whose extensive, costly, and politically contested studies into human intelligence were noted earlier, claims that 'molecular genetics looks at genes, not whole, live human beings', so for him the advantage of twin studies is that they 'add a very necessary human element to genetics' (quoted in Panofsky 2015, 164). In addition to the sense of wild, standardised abundance the twin models permit its users, the notion that twin research forms a 'natural experiment' connects it to John Snow's epidemiological studies of the 1850s, when Snow observed the randomised effects of differing water quality in two London boroughs. As is well known, his observations allowed him to deduce the source and spread of cholera through contaminated water. The claim that twin research constitutes a form of 'natural experiment' emboldens these designs and situates them within a canon of triumphant, life-saving and preserving discoveries, rendering the randomised distribution of genetic difference between monozygotic and dizygotic twins akin to the randomised distribution of contaminated water in Victorian London.

Trafficking between the molar and molecular, easing the uncertain 'coulds' and 'mays' of scientific commentaries and review articles, twins relieve twin researchers of some of the burden of scientific abstraction or the traditional, metaphors of scripts, codes, and copies that accompany efforts to make genetic findings understood by non-scientists (Pickersgill et al. 2013, 434, 443 n. 6; Nerlich 2016). By claiming that twin research is based upon a 'natural' kind of experiment twin models help to equivocate the technical, computational, social, and historical interventions made by expert analysts such as Bouchard. As if the expertise and technological infrastructures employed to articulate genetic findings can be substituted, or even elided, the presence of twin bodies means that the public understanding of DNA, RNA, single-nucleotide polymorphisms, bacteria and more, can be made known to wider populations through and between twin bodies. The spatial imaginary afforded to twin research through the bodies that they work on and work with is never simply limited to the discrete laboratories which elicit, recruit, and analyse the data attributed to them, but, instead, it is energised through the entire 'life-histories' that Galton claimed were the reason why twins were scientifically significant. When bodies are treated as a method, a global population of research subjects and a gateway to a standardised order of scientific utility, contemporary twin researchers such as Tim Spector can claim he and his col-

leagues have '11 million natural identical-twins experiments to choose from' (Spector 2012, 21). The experiments are already 'out there', waiting for Spector to capture. Elsewhere, Spector argues that his twin research is 'the closest we can get to doing animal experiments on humans' (quoted in Jolin 2013). Taken together, these statements might seem to appeal to opposed notions of experimental availability, intervention, and manipulation, yet it is claimed that twins give all the power of animal research without the temporal and spatial confinement of a laboratory setting nor the problem of translating between model and target organism.

A recent meta-analysis of twin research over the last 50 years has revealed the extent to which twins are being used in health research—over 17,800 traits have been assessed in 2748 scientific publications, including data drawn from more than 14.5 million twin pairs across 39 different countries (Polderman et al. 2015). Currently, researchers estimate that there are 1.5 million twins and their family members currently participating in ongoing cohort studies around the world, gathered into twin registries that contain anything up to 200,000 participants (Hur and Craig 2013). In this respect, twins are not ordinary experimental bodies; in an era of international research collaboration, their experimental capacities are corporeally global. Defined by Ilana Löwy, an experimental body is one 'which can be substituted for patients' bodies in order to investigate diseases and look for treatments' (Löwy 2000, 435). Biomedical modelling stresses translation and substitution, a relation that 'presupposes representation, not identity' between model and target organism (Löwy 2000, 447). In twin studies, however, model and target simultaneously occupy the same experimental location, even while that experiment is distributed not simply *within* a body but also *between* bodies; they can be both representative of their target and presented as 'identical' to it. Furthermore, the ways in which twins occupy an intermediate position between model and target makes translating laboratory findings and forging collaborations between different disciplines more likely. Löwy notes that 'an "experimental body" may allow the bridging of differences through the development of open-ended, "boundary concepts" which may have one meaning in their common use by several professional groups, and another when used by each specific group' (Löwy 2000, 447). Twins, then, are used to produce forms of evidence such as heritability estimates that translate complex behaviours into manageable data and can be shared across disciplinary communities. For Alison Cool these estimates are 'compact and comprehensible representations of less tangible social phenomena' (Cool 2011). The important point is that corporeal standardisation and the numerical representations of complex traits and behaviours have secured the reproductive success of

twin research. Heritability estimates may bring mobility and tangibility to less easily perceived social phenomena, but the social phenomena of twin research as a process of standardisation passes into obscurity. Twin bodies become, rhetorically at least, 'immutable mobiles' (Latour 1987) that are, like the heritability scores that spring from them, presentable, readable, and combinable beings that convey information without undergoing transformation.

Human manipulation, emotion, volunteerism, or coercion can be written out of the accounts of twin research that stress the spontaneous efficiency of the twin body in generating numerically meaningful 'scientific' evidence. As a consequence, twins serve as an experimental and explanatory resource that redistributes epistemological authority away from the analyst. The overall effect is to go beyond a power that Isabelle Stengers has claimed is typical of modern scientific objectivity, 'the invention of the power to confer on things the power of conferring on the experimenter the power to speak in their name' (Stengers 1997, 88). Twin studies does not confer upon the scientist a power to speak on behalf of mute phenomena, it confers on some scientists the power to indicate how phenomena speak themselves. In this case, the power of scientific discourse renders twin bodies the experimental and explanatory vectors of scientific fact, with the devolution of explanatory powers to twins in general, rather than to twin individuals. Twin research, in its efforts to displace the responsibility of its findings on the bodies that it analyses, fundamentally transforms the ways in which twins are viewed as a community of clinical labourers.

Making Up Twins, Fast and Slow

As we have seen, the presentation of twins as experimental bodies, experimentally necessary for the study of basic biology, development, health, disease, and studies of ageing, frequently points to the 'natural' availability of twins as a population, bodies that permit an explanatory power to pass from the scientific observer into the minutiae of twin lives. This control can be communicated between twin researchers and other expert practitioners in the life and human sciences through the immutable 'givenness' of twins. And yet that givenness, especially to longitudinal research programmes, capitalises on the preexisting relationship shared between twin pairs that cannot be considered to be the straightforward product of their embryological, gestational, genetic, or even epigenetic relations. The paradox is that though the practice of biomedical research provides twins an arena in which to situate their relations, that practice is extraneous to the experimental lives twins come to contribute to biomedicine.

What remains of this chapter examines the 'biosocial' constitution of twin identities in light of how biomedical research can or cannot inform, shape, or even determine how twinship is understood. Such an undertaking demands a more robust understanding of what twin participants experience in research contexts. I discuss concepts of the biosocial, theorised by Paul Rabinow, Ian Hacking, Carlos Novas, Nikolas Rose, and others and, in separate but related scholarship, I also consider what some call the 'biomedicalisation' of human identity—the increasing influence of biomedicine to affect the 'unprecedented and historically transformative differentiation of human bodies and futures clearly visible in contemporary struggles over pharmacological access, care, legal redemption, and therapeutic sovereignty' (Moyer and Nguyen 2016). Biomedicalisation, and hence the emergent biosocial identities it may forge, is used here to describe technological and economic processes that express the capture and control by, and imposition of, medical knowledge upon individuals (Clarke et al. 2010).

Theories of biosociality acknowledge a debt to Michel Foucault's interests in the historical constitution of subjects as they 'are gradually, progressively, really and materially constituted through a multiplicity of organisms, forces, energies, materials, desires, thoughts, etc.' (Foucault 1980, 97), while showing that Foucault did not anticipate the medical technologies or the kinds of subjectivity permitted by those technologies in the twenty-first century (Campbell and Sitze 2013). Paul Rabinow's early efforts to define 'biosociality' stressed acts of collective identification that emerge as a result of novel and often genetic findings; while Foucault stresses the multiple agencies that inform who and what different people are and do, Rabinow's intervention argues that technologically advanced biomedicine should be viewed as a dominant influence (Rabinow 1996). One outcome of the biomedical recreation of identity, claims Rabinow, would be the dissolution of the traditional division between nature and culture: 'nature will be modeled on culture understood as practice. Nature will be known and remade through technique and will finally become artificial, just as culture becomes natural' (Rabinow 1996, 99). The social and biological converge when 'new group and individual identities and practices aris[e] out of these new truths […] there will be groups formed around the chromosome 17, locus 16,256, site 654,376 allele variant with a guanine substitution. These groups will have medical specialists, laboratories, narratives, and traditions and a heavy panoply of pastoral keepers to help them experience, share, intervene in, and "understand" their fate' (Rabinow 1996, 102). Sociological analysis, coupled with and made possible through the emergence of new biomedical truths about individual health conditions, has encouraged subsequent theorists 'to name the kinds of socialities

and identities that are forming around new sites of knowledge (genetics, molecular biology, genomics) and power (industrial, academic, medical)' (Gibbon and Novas 2008, 3). Ian Hacking also notes that those collectivities are fundamentally shaped by institutional classifications that are managed by experts, experts whose classifications identify people 'that would not have existed, *as a kind of people*, until they had been so classified, organised and taxed' (Hacking 2007, 288). Following Rabinow, Hacking also gives a special role to the biomedical sciences in galvanising the various numeric, normative, correlative, medicalised, biologicalised, and genetic 'engines' that govern attempts to form human groupings: 'There has been making up of people', concedes Hacking, 'in all times and places, but only in the past two hundred years have the sciences been so central to the human understanding of who we are' (Hacking 2007, 305). The important point to underline here is how these theories of biosociality stress the novel contribution of biomedical science in identifying and grouping individuals around 'disease, disfigurement or disability' (Rose 2007, 137) in order to identify the vital and increasingly molecular components of individual and collective identity.

In the clamour to understand the novel contributions made by emergent information about the structures and contingencies of life, scholars of the biosocial risk overlooking an important and somewhat opaque qualification made by Rabinow in his early theorisation of biosociality: 'older cultural classifications will be joined by a vast array of new ones', he cautions, 'which will cross-cut, partially supersede and eventually redefine the older categories' (Rabinow 1996, 103). For Rabinow, the precise extent to which older cultural classifications enter into, cross-cut, and redefine newer identities is uncertain, nor is it entirely clear whether those older classifications are already 'biosocial' in the enriched biomedical sense that Rabinow describes. Furthermore, Rabinow assumes that the new will always topple the old and seems not to have considered the potential for established classifications to become resurgent, or to redefine the meanings attributed to biomedical innovations. Twin research may provide an important corrective to how theories of the biosocial explored above can be freighted with subtle yet hierarchical distinctions, ones that privilege the emergent over the established, the richly new over porously old, the dynamically fast before the superfluously old. The biosocial does not affect all subjects of biomedical research at the same speed, intensity or with the same emergent sense of dynamic novelty. This seems especially important when, as in the case of twins used in biomedical research, their status as 'natural experiments' runs contrary to sense of novelty that is being placed over the material that is being discovered within, through, and with them.

Though twins have been used in the life and human sciences since the end of the nineteenth century, they have been recruited on the assumption that they provide methodological immunity from the effects of 'biologicalisation'. What might first appear to be a divergence between the logic of twin models and concepts of biosociality may, in fact, reveal a more subtle interaction between the clinical and extra-clinical attributes of human groups that serve to ensure the continuation of twin research. To help explore the temporal and historical formation of 'twinship' I refer to the United Kingdom's largest twin studies project, TwinsUK, hosted by the Department of Twin Research, Kings College London.[4] This research group studies the aetiology of age-related diseases and the genetic pathways that inform those diseases, but it can only do so by carefully managing the clinical and extra-clinical identities of twin volunteers.[5] The twins in the TwinsUK cohort are not, however, clinical labourers of the kind documented in the work of Melinda Cooper and Catherine Waldby; they do not sell their cell tissue or reproductive services, nor are they financially remunerated for their participation in clinical tests and measurements (Cooper and Waldby 2014). The cohort of 12,000 twins each complete an annual questionnaire that gathers detailed information about birth and health histories, daily and monthly dietary intake, and exercise. The twins also attend a four-yearly clinical assessment that involves an intensive, daylong series of testing, measurement, and tissue sample collection. Clinical examinations can gather multiple samples of the blood, saliva, hair, skin, urine, and faeces, while a series of tests include blood pressure and glucose, renal function, liver function, cardiogram, bone mineral density, grip, and lung capacity tests, and cognitive and memory examinations. Results for each individual twin are returned to him or her by post and copied to the volunteer's general practitioner. With the exception of specific discordant monozygotic twin designs, the twins that participate in this research do not receive a clinical diagnosis before participation in twin studies but are used for the 'randomised control' that they provide. Nor is TwinsUK providing information that is exclusive to twin health or experience, their findings are frequently extrapolated to the general population. While some twins have outstanding health conditions, these are not the determining factor for their participation; these individuals are not grouped by biomedical practice according to a specific 'disease, disfigurement, or disability'. Instead adult twinship is the principal requirement for participation—conceived, gestated, and born in unison.

Twins who enter genomic studies of the kind conducted by TwinsUK experience a different process of biomedicalisation from that which is prevalent in sociological and science studies literature on biosociality. Next to the routine tests, clinical visits mark an occasion for twins to spend time with one another;

the tests and trials offer a combination of interpersonal interaction and personal discomfort, as well as occasions for playful, between-pair competition and comparison.[6] What is especially important to bring to light is how biomedical experts work to encourage and validate these extra-clinical benefits, respecting and often affirming 'older classifications' dear to twin siblings. Rather than simply making them redundant, twin researchers managing large cohorts utilise far older and in some cases ancient valuations of twin exceptionalism, especially those valuations that, rather than stressing clinical conditions affecting an individual, stress extra-clinical and collective effects of twinship such as the longevity and intimacy of the sibling relation or being the objects of curiosity and wonder of others. Celebrating the birthdays of older cohort members in newsletters and social media posts is one example of how engagement reinforces a set of values about how longevity, simultaneity, and continuity are shared by twins. Another example of how the sociality of twinship interacts with TwinsUK's research imperatives can be found in the following radio advertisement, aired in 2012 to recruit new twin pairs, it may be taken as exemplary for its celebration of a certain kind of prior, extra-clinical twin identity at the precise moment when twins are engaged as research participants:

> [*A male voice; background music*] You've lived your whole life as a twin, it's something that's really special to you and you're always keen to celebrate your uniqueness. We'd like to invite you to join TwinsUK, the biggest registry in the UK especially for twins. Our team of experts carry out innovative and research in the areas of genetics and aging. You'll be able to meet other twins and be part of the latest discoveries in science. For more info simply text 'TWIN' to 8400 […]. (DTR 2012)

To TwinsUK and numerous other research programmes that use data generated from tissue extracted from twin bodies, twin-born people are not simply passive assets: their experiences, beliefs, and behaviours fundamentally inform the particular quality of latent biosociality that initiates contact with medical researchers employed at TwinsUK.

Through social media accounts on outlets such as Facebook and Twitter, TwinsUK offers a wide range of items to engage their twin volunteers. Only a few posts disseminate research findings. Twins have been asked if 'you and your twin share a telepathic connection' (DTR 2013), shown pictures of twin volunteers on holiday in a regular 'Twin Travels' section, and are linked to stories about unusual or celebrity twin pairs. Perhaps, most striking was the promotion of a study—not conducted by TwinsUK—that 'shows that twins

enjoy better health and live longer thanks to their close bond' (DTR 2016) The post, popular among readers, in terms of comments, shared reposts, and 'likes', introduced research that compared the life expectancies of twins from a Danish birth registry to suggest that monozygotic twins live longer than dizygotic twins. The authors of the research paper claimed that this is a 'consequence of the social bond between twins buffering against risky behaviors, providing emotional or material assistance during times of stress exposure, and promoting health-enhancing behaviors' (Sparrow and Anderson 2016). It is a striking piece of research to disseminate through TwinsUK media channels, not only because it reinforces a deep-seated, cross-cultural belief that twins enjoy particular forms of intimate relation (Hoctor 2015; Davis 2014; Peek 2011; Piontelli 2008; Joseph 2003), but also for the evidence it gives to the long-standing criticism of twin models employed by TwinsUK. If different twin zygosities can be consistently associated with different biosocial ecologies (contradicting the 'equal-environment assumption'), then the controls classic twin studies designs provide for non-shared environmental effects are distorted (Joseph 2013). The biosocial identities of twins are neither aligned with nor straightforwardly attuned to the research in which they participate.

Conclusions

Twin cohorts may celebrate, indulge in, or build upon prior, extra-clinical exceptionalism felt for and by twins, forming a community of twin volunteers around their work that both reenacts and performs existing twin identities while co-emerging with novel research findings. Returning to Rabinow, the assumed dominance of biomedical practice and its capacity to create novel biomedicalised identities overlooks how twin researchers interact with the lives of twins, as well as how those lives have been the subject of focused medical discussion for thousands of years, not least because the circumstances of twin birth have grounded philosophical and theological interpretations of twin lives (Aristotle 1991; Dasen 2005; Hippocrates 2012). What is made apparent through the example of twin participation in TwinsUK research is the way that the biosocial expectations of the past facilitate those of the new. Moreover, while promising a renegotiation of nature and nurture, concepts of biosociality have depended upon an asymmetry between what is past and present, working through the assumption that 'traditional' collectivities are either 'non-biomedicalised' or less biosocially sophisticated. Nikolas Rose has argued that what is distinct to twentieth- and twenty-first-century biosociality is that 'making up biological citizens also involves the creation of persons with

a certain kind of relation to themselves' (Rose 2007, 140). Twins enrolled into scientific study, however, repeat at an inter-corporeal scale what has been identified at the historical scale: comparison, relation, and the dispersal of individual conditions across pairs and populations. Their ability to establish relations with themselves continues to be informed and mediated by their prior sibling relations; indeed, the forms of twin biosociality explored here, which produces scientific significance from rich mixture of somatic materialisations, descriptions, and communications that circulate both within and far beyond the laboratory, within bodies and between bodies, may also stand in contrast to the individualism and genetic individuation described in sociological studies of the contemporary life sciences.

While twins have been made to matter in the attempt to identify molecular entities, processes, and locations that have emerged through intense, rapid, technical, and financially complex developments in the life sciences, twins also constitute the molar and embodied evidence of such research, recruited as rhetorical devices and narrative protagonists by which to prove that those entities, processes, and locations can become publically understood. And yet, as a group of people that have long been distinguished for the gestational circumstances of their birth and attracted debate over their complex biological and social significance, twins are not a modern biosocial grouping in the sense that they, as a human kind, have been discovered through biomedical innovation. Nor do they necessarily participate as biosocial bodies thanks to the genetic identification of a given pathology or disease; even monozygotic twins, discordant for a given trait and included within a cohort of others for a study may recognise the trait analysed as a significant but not a sufficient condition for their twinning. First-person narratives and ethnographic studies of twins who have experienced divergent health experiences tend to stress the transcendence of twin relations over and above the influence of an acquired or congenital condition on one or the other (Davis 2014; Lewis and Lewis 2013; Stenzel Byrnes and Stenzel 2007; Spiro and Spiro 2006). There is far more to be discovered about how biomedical advances form and fail to form identities, and why theories of the biosocial are so quick to follow novelties rather than acknowledge classificatory resonance, dissonance, or ambivalence with long-standing patterns of knowing and being. Such an inquiry would not isolate the biological from the social or cultural still further, but provide a more nuanced understanding of how the biological and social are not only compelled by time's arrow but are concepts that are historically entwined.

Notes

1. Here I follow Nancy Segal's definition of 'twin-born people' as those who share 'simultaneous conception, shared prenatal environments, and common birth' (Segal 2000, 225).
2. Thought to have entered the English language in Shakespeare's *The Tempest* ('a born devil, on whose nature nurture can never stick' (4.1.188–189)), Galton's first use of the phrase 'nature and nurture' occurred in 1874, a year before he published his work on twins in *Fraser's Magazine* in 1875, when expressing interest in the 'energy, intellect, and the like' of other fellows of the Royal Institution (Galton 1875b).
3. For a succinct overview of twin research methods and its various attempts to identify numerical values for 'genes' or 'environment', see Ball and Teo (2008). That twin methods are inextricably tied to efforts to separate, even provisionally, different domains of so-called 'natural' or 'environmental' influence, may be observed in how behaviour geneticists such as Nancy Segal now regret using the expression 'nature vs nurture' to evoke an imagined battle between two opposing entities. But, testimony to how models of conflict between genes and environment have given way to more interactionist models, the old protagonists remain distinct entities, Segal now prefers either 'Nature-Nurture or Nature and Nurture, because it is widely appreciated that the two effects work together and are separable only in a statistical sense'. Her studies on twins concede interaction on the basis that their statistical separation is (and should be) achieved through twin research (Segal 2012, 96).
4. Described as 'the biggest UK adult twin registry of 12,000 twins used to study the genetic and environmental aetiology of age related complex traits and diseases'. See http://www.twinsuk.ac.uk/ Accessed 17 May 2016. Information on the activities of TwinsUK has been gathered by my participation (2012–present) and as a Volunteer Advisory Panel member (2014–2015). I conducted clinical visits in March 2012 and March 2016.
5. The TwinsUK website claims that its 'genome-wide association studies have identified over 400 novel gene loci in over 30 disease areas including osteoporosis, osteoarthritis, melanoma, baldness, and telomere length from TwinsUK data. Current research covers the genetics of metabolic syndrome and cardiovascular disease, musculoskeletal system, ageing and sight.' http://www.twinsuk.ac.uk/about-us/ Accessed 2 May 2016.
6. For documentary footage of such competition, see Alexander and Christoffer van Tulleken visit to TwinsUK in the BBC documentary, *The Secret Life of Twins* (van Tulleken and van Tullekan 2009).

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8

Histories and Meanings of Epigenetics

Tatjana Buklijas

We are living through a revolution in our understandings of heredity, or so we are told by the media, buzzing with suggestions that our health and our personalities are determined not just by the genes passed across generations but also by the experiences of our parents and grandparents: the wars and famines they suffered, the psychological traumas they experienced, the foods they ate (Anonymous 2012; Blech 2010; Costandi 2011; Shulevitz 2012; Knapton 2014). These experiences are inscribed and, arguably, inherited through a network of mechanisms that act as 'the molecular memory of past stimuli', modifying gene expression to supplement the slower-changing information encoded in the DNA sequence (Bonasio et al. 2010). The best studied mechanisms are DNA methylation (binding of a small chemical group, CH_3, onto the cytosine base of DNA) and the modifications of histones, proteins that package DNA into nucleosomes and in turn change the spatial conformation of chromatin. But other, less studied mechanisms, in the first place the activity of RNAs of different types, may play equal or even more important roles (Heard and Martienssen 2014).

Epigenetic control of gene expression may have profound implications for biology, medicine and wider society. It may, for example, open up new avenues to explain, predict and treat disease. Furthermore, because phenomena such as pollution, nutrition, stress, deprivation and even parenting are understood to leave marks on our genomes, social scientists have taken great interest in epigenetics (Landecker 2011; Landecker and Panofsky 2013; Meloni

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2016), arguing that epigenetics is 'the missing link between the social and the life sciences' (Meloni 2015) that has 'reignited the nature/nurture discussion' (Lock 2013). More than a hundred years ago, a particular set of historical circumstances separated the 'social' and 'biological' disciplinary epistemological domains, and they remained disconnected—though never entirely (Renwick 2016). Now new ways of thinking in science, in novel social circumstances, may be bringing them together (Meloni et al. 2016).

Yet these high expectations should be treated with caution. The science underpinning this comprehensive epistemological shift is changing daily. Even the supposedly best researched and fundamental epigenetic mechanisms—such as methylation of CpG islands in promoter regions silencing the expression of the relevant gene—appear uncertain (Ngo and Sheppard 2015). And even if epigenetics does turn out to be the unifier bringing together two separate domains of knowledge, it is by no means obvious how the new epistemological space should look, which methods of inquiry should be used or what shape research hypotheses should take (Niewoehner 2015; Newton 2016). A *Nature* editorial extended an invitation to social scientists to join the shared project of bringing the two knowledge domains together, but on biologists' terms and under their leadership (Nature 2012). Epigeneticists, by and large, do not read social science research. Indeed, in their attempts to make sense of their forever shifting research object—the response of the genome to continuous and diverse influences—they draw on cybernetics and physics, rather than social sciences, as sources of models.

The most controversial aspect of this new field is the one that makes epigenetics exciting beyond the laboratory: the possibility that environmental influences, captured in epigenetic marks, may be transferred not just mitotically (cell-to-cell 'epigenetic inheritance') but also, by way of the gametes, to the offspring ('transgenerational epigenetic inheritance'). This proposal challenges many of the fundamental concepts upon which modern biology rests. Firstly, the fertilized egg (zygote) begins its life as a 'blank slate', a union of parental genomes wiped clean of the records of their past lives in the form of epigenetic marks, so that the new organism is in a pluripotent state ready to acquire cellular specialization through development (Reik and Kelsey 2014). Secondly and perhaps more importantly, the proposal of transgenerational epigenetic inheritance contravenes the idea central since the nineteenth century: that there is such a thing as 'heredity' that is by and large stable and is transmitted across generations with a high level of fidelity, regardless of the experiences each generation has or conditions in which it lives (Müller-Wille and Rheinberger 2012). Instead, a heritable epigenome reflects the constantly fluctuating environment: the new organism is seen not as a random combination

of genes making or breaking it in the potentially hostile world, but, rather, a carefully curated collection, an ensemble well prepared for what might await outside.

With all these controversies and open questions, it is no wonder that different actors in the field view the role, significance and history of epigenetics differently. For those who are critical of the existing model of heredity and indeed of the entire framework established by the Modern Synthesis in the 1940s—let us call them 'dissenters'—epigenetics offers an important solution to the question: what can replace the existing 'genetic' model (Jablonka and Lamb 2005; Laland et al. 2014)? For others, epigenetic phenomena are part and parcel of genomics and genetics, important and potentially useful but by no means paradigm-changing. We could call this group 'conformists'. They may accept the transgenerational transmission of epigenetic marks, but even if they do, they will generally argue that it is of limited significance and that the current model of heredity is still valid.[1] 'Dissenters' are more likely to have backgrounds in animal behaviour, evolutionary and developmental biology, ecology and philosophy of biology compared to 'conformists', who by and large come from genetics (Dawkins 2004; Haig 2004; Bird 2013).

While there has been no attempt to write a detailed history of epigenetics, there is no shortage of narratives in circulation. Different views on the significance and role of epigenetics, and in particular of transgenerational epigenetic inheritance, are reflected in the kinds of stories different actors tell. 'Dissenters' tend to view epigenetics as the latest chapter in an alternative and highly provocative history of heredity that may stretch back as far as Jean-Baptiste Lamarck—or even earlier (Gissis and Jablonka 2011; Ho 2014). This narrative reasserts the importance of the 'Neolamarckists' of the early twentieth century, with some proponents going so far as to explain in epigenetic terms those historical experiments that claimed to prove the inheritance of acquired characteristics (Vargas 2009; Vargas et al. 2016). By contrast, 'conformists'—and those who stand in the middle ground—do not go far back. They may look back to the mid-twentieth century and the work of geneticists whose research was considered controversial, for instance, Barbara McClintock and Alexander Brink (see below), to show how genetics withstood and then incorporated knowledge that challenged contemporary dogma (Riggs et al. 1996). But by and large their histories are short: the story of epigenetics is, in their view, contained within the history of genetics.[2] The only points where these two groups meet is the history of the term 'epigenetics' (introduced by the British biologist and polymath Conrad Waddington around 1940, yet, as I will discuss later, in a meaning that does not correspond with today's). Also, both groups tend to agree that epigenetics as understood today begins with

two essays published independently in 1975 that suggested that DNA methylation is involved in the regulation of gene activity (Holliday 1996; Jablonka and Lamb 2005, 128).

So, how should one write the history of epigenetics? In this, preliminary, account of the history of epigenetics, I want to look at both long and short accounts of heredity. I suggest that a long history is useful because it forces us to rethink the standard narrative of heredity, one that privileges the gene. It can also help illuminate certain historical episodes (Graham 2016) or answer larger questions about the relationship between the biological and the political (Meloni 2016). At the same time, such a broad perspective cannot provide a finely grained analysis of the particular conditions—in science but also in society—in which modern epigenetics emerged and, especially, became famous. So this chapter combines both. After an overview of the long history of heredity, I will provide a preliminary overview of modern epigenetics: its early, 'genetic' era, from 1975 to the late 1990s, and its second, 'developmental' or 'human' period, from ca. 2000 onwards. Together, I hope to sketch how epigenetics came to high public prominence, and what kind of larger developments in science and society this prominence reveals.

An Alternative History of Heredity?

Genealogy has always been at the heart of human social relations, yet before the 1800s the making of life was generally understood in terms of generation, a creative process malleable by influences ranging from divine wrath and earthly politics to the diet and emotions of the mother (Shildrick 2000; Buklijas and Hopwood 2008). These influences could change the shape of the child at any point between conception and birth. Similarity between parents and offspring was explained not by shared hereditary traits, but by similar influences acting upon each generation (Hopwood 2009). Although it is today associated with the name of Jean-Baptiste Lamarck, the idea that properties of organisms change under direct environmental influence was common knowledge (Burkhardt 1995). It was only in the mid-nineteenth century that the idea of heredity as a material property similar to the concept of inheritance in law began to gain currency (López-Beltrán 2007). Heredity came to be seen as separated from the circumstances of conception and development, transmitted unchanged across generations and distributed in a predictable manner. But heredity only became a general biological problem when organisms acquired (evolutionary) history and 'the forms of life ceased to be fixed by assumed species boundaries' (Müller-Wille and Rheinberger 2012, 75).

Charles Darwin offered no convincing theory of heredity, although he knew he needed one (Olby 2013). Although Darwin's own tentative concept of 'pangenesis' had Lamarckian undertones—he suggested that a change to one's body simultaneously changed heritable particles, 'gemmules'—a 'hard' concept of heredity insulated from environmental influence increased in popularity during Darwin's lifetime. 'Hard' heredity was central to his cousin Francis Galton's *Hereditary Genius* (1869) argument that humans inherited their characteristics from their ancestors and that their mentality could be improved through 'good breeding' (Kevles 1985). It underpinned pessimistic views of degenerating humankind, first in the 'degeneration theory' popularized by asylum psychiatrists, and then in eugenics, a widely ranging programme for social improvement through the control of reproduction (Pick 1989; Levine and Bashford 2010). Experimental scientists provided biological explanations for these social theories by linking heredity with cell theory. From the 1880s the German zoologist August Weismann persuaded many that the hereditary material contained in the germ cells is insulated from changes taking place in somatic cells (Churchill 2015). In the early 1900s, cell research was brought together with the recently rediscovered laws of inheritance, established by the Bohemian plant breeder Gregor Mendel, in a discipline called genetics. Transmission and distribution of hereditary properties became the core concern of this discipline in its early decades.

Yet this genetic view—one that privileged nucleus and genes, emphasized the constancy of transmitted properties and sidelined development—was not universally accepted. The reasons why 'hard' heredity and genetics were pioneered in the United Kingdom and United States and not in Continental Europe are diverse, to do with institutional organization but also with intellectual traditions and socio-economic structures. The rapid social and demographic changes caused by the industrial revolution, initially in the United Kingdom, inspired not only Friedrich Engels and Karl Marx but also Darwin and Galton, as well as the turn-of-the-century geneticists. In France, 'Mendelism' held little appeal because it went against the ideals of biological research set by physiology, microbiology and embryology (Burian et al. 1988). There, genetics only gained a firm foothold when, around 1940, geneticists internationally were no longer content to study transmission of visible differences between organisms capable of being cross-bred, but rather began to inquire how genes exercise control over the physiological and biochemical properties of the organism (Sapp 1987). In German-speaking countries, institutional, disciplinary and social traditions that favoured a holistic view of biology account for the continuing inclusion of development within genetics as well as interest in the study of cytoplasmic hereditary particles alongside

nuclear genes; there, 'Neo-Lamarckism' persisted at least into the 1920s (Sapp 1987; Harwood 1993). In the newly formed Soviet Union, the debate about heredity played out alongside discussions about the history and future of the working class. While initially the orthodox genetic view won, by the 1930s the balance had swung towards 'soft' inheritance (Graham 2016). This consensus came with the repudiation of any kind of eugenics: humans could only be explained in Marxist terms, not biological ones.

Early twentieth-century Vienna provides perhaps the richest story about the science and politics of 'soft' heredity. In the early 1900s, in Austria-Hungary, many tried to marry Darwin's evolution by natural selection with the inheritance of acquired characteristics (Logan 2013, 52). At the Institute of Experimental Biology ('Vivarium'), Paul Kammerer studied not fruit flies in a highly controlled laboratory but slowly reproducing amphibians in environments of varying temperature and humidity. Kammerer claimed to have permanently changed hereditary properties through environmental modification, but his results were difficult to reproduce and his leftist politics, Jewish origin, complicated personal and social life, as well as lack of institutional backing made him vulnerable. Accusations of scientific fraud were published in *Nature* in August 1926, and Kammerer died, allegedly by his own hand, in September. His death was long understood as an admission of guilt, but recent research indicates that—as suggested by the Soviet media in the late 1920s and then by Arthur Koestler (1971) though without concrete evidence—he might have fallen victim to a right-wing, anti-Semitic conspiracy (Taschwer 2016).

In Vienna, the inheritance of acquired characteristics had an impact that extended beyond the walls of the 'Vivarium' and underpinned the connection between biology and society. Rudolf Goldscheid, the founder of the Sociological Society (1907), agreed with Darwin's theory of evolution by natural selection but disagreed with the Malthusian argument that all organisms have a tendency to reproduce until limited by resources. Instead he proposed that reproductive ability varied in response to environment. A well-adapted variety did not necessarily produce many individuals, but they were of 'high quality'; 'high quality' here referred to parental investment and developmental condition rather than 'good stock' (Exner 2013, 52–6). In 1913, the Society established a Section for Social Biology and Eugenics with Kammerer as the secretary and Julius Tandler, anatomist and Social Democrat, as the chair. In 1919 Tandler became the municipal councillor in charge of health and welfare for the newly elected Social Democratic government of Vienna. The widespread reforms of 'Red' Vienna to improve education, housing, nutrition and health, of all inhabitants but especially children and mothers, were based

in Goldscheid's theories and ultimately in 'Neo-Lamarckism' (Baader 2007; Weindling 2009; Logan 2013). Many famous scholars who lived and worked in early twentieth-century Vienna, such as Sigmund Freud and Karl Popper, remained sympathetic towards a 'Lamarckian' view of inheritance long after it had fallen out of fashion (Slavet 2008; Aronova 2007).

But by the early 1930s, the position of 'soft' inheritance had grown weak nearly everywhere. Countries from Germany to the United States and Sweden used 'hard' heredity as the scientific legitimation for their eugenic programmes (Lombardo 2010; Levine and Bashford 2010; Broberg and Roll-Hansen 2005). Transmission genetics reached its peak in the late 1920s. The 1930s and 1940s are generally regarded as the era when genetics, building on its new interest in natural populations and use of mathematical models, brought in evolutionary theory—changed little since the days of Darwin—as its theoretical foundation. The union between the two fields in the form of the Modern Synthesis refreshed both and gave them unprecedented power. The only exception was in the Soviet Union where political and economic circumstances propelled Trofim Lysenko, a provincial agronomist advocating an outdated concept of heredity, to the position of most powerful scientist in the country (Graham 2016). Although Lysenko's version of 'soft' heredity had very little to do with contemporary science, the association of the inheritance of acquired characteristics with Stalinism and politically directed science influenced the reception of the inheritance of acquired characteristics in the West for decades (Sapp 1987).

And yet, many established scientists at US universities and other publicly funded institutions pursued research programmes that involved changing hereditary properties through environmental modulation. Between the late 1930s and early 1970s, Tracy Sonneborn, a highly respected American geneticist who studied under 'Lamarckist' Herbert Spencer Jennings, investigated the unicellular protozoan *Paramecium*, which exhibits functionally relevant and heritable variations in cell surface configuration yet without genetic difference (Sapp 1987). It was a Sonneborn student, David Nanney, who first defined 'epigenetic control systems' as 'auxiliary mechanisms' (i.e. not in the sequence) 'involved in determining which specificities are to be expressed in any particular cell' (Nanney 1958, 712). He chose the term 'epigenetic' to underline their involvement in development (see below). At the US Army Biological Warfare Laboratories in Fort Detrick, the German émigré Otto Landman forced bacteria to stop building cellular walls by changing the growth medium. He wondered whether the 'environmental modulation of inheritance that we have observed is confined to this rather pathological system in microorganisms or whether other inheritance systems display similar

properties' (Landman and Halle 1963). Others showed that the genome was a reactive, dynamic organ rather than a fixed set of instructions. These, most famously, included plant geneticists working with maize: Barbara McClintock, who observed the effects of transposons, small pieces of DNA that could change their position in the genome, and Alexander Brink, who described paramutation where one allele heritably changed the expression of the other allele on the same locus (Brink 1968; Comfort 2003).

The name most closely associated with epigenetics is that of the British developmental geneticist and experimental embryologist Conrad Hal Waddington (1905–1977), a polymath who supported radical left-wing politics in the 1930s (Peterson 2016). He argued that the heritable capacity to respond to an external stimulus could, after multiple generations, result in individuals capable of response even without the stimulus (Waddington 1942; Gilbert 2000). Waddington introduced the term *epigenetics* to describe mechanisms and processes by which, during development (under its historical name, *epigenesis*), genes bring about phenotypic effects (Waddington 1940). Although he is today credited as the 'father of epigenetics', the current understanding of the term has departed from the original definition.

Waddington chaired the successful and large Edinburgh Department of Genetics and persuaded the Medical Research Council (MRC) to establish, in 1965, a laboratory for the causal study of development—or in his words, epigenetics (Robertson 1977). Yet very little research in the (otherwise productive) MRC Epigenetics Research Group was about development, arguably because the contemporary science was all about restriction enzymes, cloning and sequencing of DNA (Holliday 2012). More research needs to be done to elucidate the link between the work of Conrad Waddington—in theoretical and experimental biology but also his broader intellectual and political interests—and contemporary epigenetics. A cursory follow-up of institutional and personal connections reveals that Waddington's deputy, Max Birnstiel (1933–2014), mentored Adrian Bird, whose 1970s work would prove crucial to establishing methylation as the key mechanism for setting gene expression patterns. Also, Birnstiel later established the Institute of Molecular Pathology in Vienna, an institution that would play a central role in the nascent field of epigenetics through the 1990s (Jenuwein 2006; Anonymous 2015; Grunstein and Bird 2015).[3]

All of these stories show that a past in which belief in the gene—and the DNA sequence—as the sole and ultimate source of biological information never really existed. Of course, 'soft' inheritance, in the form that existed in the early twentieth century when it opposed genetics, was not part of scientific canon. Yet, with the consolidation of genetics around 1930, genetic

orthodoxy came to encompass much more than the information contained in the sequence: it was also about interactions between genes, regulation of gene expression, the role of carriers of heredity in the cytoplasm and the actions of the associated enzymes. For all the language of 'breaks' and 'revolutions', with regard to programmes of scientific research, there is much continuity between epigenetics today and twentieth-century genetics. The next section will explain this in detail.

1975 and the Origins of Epigenetics

The 1970s were the heyday of the Modern Synthesis and genetics. This was the decade of the 'selfish gene' and socio-biology, and also the decade of recombinant DNA, typified by the use of bacterial enzymes to cut and stitch together bits of the sequence and express them in experimental organisms to produce clinically and commercially useful protein in bulk. Nothing appears more emblematic of gene-centred biology than recombinant genetics; yet it was from recombinant DNA research that the first observations of phenomena were made that later came to be understood as epigenetic.

These first observations are contained in two papers that, as mentioned in the introduction, feature in most accounts of the history of epigenetics. Both were published in 1975, both by established geneticists, and both engaged with the central question of genetics of the era: how are patterns of gene expression established and maintained? Both reviews proposed, though using different models, that methylation changes gene expression. But both were highly speculative, and neither had much impact at the time of publication; so in both cases their significance was established retroactively.

The first paper was written by the prominent British geneticist Robin Holliday and his PhD student John Pugh (Holliday and Pugh 1975). Holliday, a former Cambridge student, was at that time the head of genetics at the National Institute for Medical Research at Mill Hill, London. Working on the fungus *Ustilago maydis*, Holliday had produced an influential model of genetic recombination ('Holliday junction') before embarking on DNA repair studies. In particular, in this period, Holliday became interested in DNA modification and restriction in bacteria: how enzymes can distinguish between short DNA sequences that are methylated and the same sequences that are unmethylated. It was Pugh who, while working on isolating mutants of *U. maydis* with increased recombination frequency, developed an interest in the possible function of the methylation of cytosine in DNA. This phenomenon, Holliday wrote years later, had been observed a few years earlier in bacteria (where

methylation occurs on both adenine and cytosine), but its function was not understood (Holliday 2011).

The puzzle that Holliday and Pugh attempted to explain was the existence of 'developmental clocks' or how, during development, certain genes are turned on (and then perhaps off too) at specific moments. Their proposal was that (1) methylation had a role in the control of gene expression, (2) de novo methylation was sequence and tissue specific and required a specific DNA methylase enzyme and (3) maintaining a pattern of methylation depended on the existence of an enzyme that recognized hemimethylated sequence and methylated the other strand at the replication fork.

The very same year, another scientist proposed a key role for methylation in gene expression. In terms of disciplinary affiliation, Arthur Riggs described himself as a physical chemist. He began his career by studying *lac* repressor, a protein binding to DNA to repress genes involved in lactose metabolism: this model of how genes are turned on and off had earned François Jacob, Jacques Monod and André Lwoff a Nobel Prize in 1965. Riggs accepted a position at the City of Hope in Duarte, California, a former tuberculosis sanatorium turned biomedical research centre, which entailed establishing a laboratory, but as he later wrote, he had 'no useful ideas' how to proceed with his research. It was the meeting with another City of Hope scientist, Susumu Ohno, which proved a 'light bulb moment'.[4] Ohno, a Japanese-born pioneer of what would become evolutionary cytogenetics, worked on the evolution of sex chromosomes: in 1956 he had proposed that the dense area of chromatin found only in females, called the 'Barr body', was an inactivated X-chromosome (Beutler 2002). But none of the several theories on how inactivation could occur met all the criteria for X inactivation: randomness in some animals and preferential maternal/paternal inactivation in others, reversibility in the next generation and permanence across mitosis (Riggs 1975). Riggs took inspiration from his own earlier work on enzymes and the way that *lac* repressor binds on the outside of DNA and then reads the bases. He combined this research with reports on methylation in bacteria to argue that known properties of bacterial DNA methylation enzymes are ideally suited to explain how inactivation of X occurs (Riggs 1975).

The immediate reception of both Holliday and Pugh's and Riggs' articles was modest. While their arguments were plausible, the texts were highly speculative. Riggs's paper had been rejected by several journals before it found home in a not very prestigious journal. Soon afterwards Riggs struck gold when his collaboration with Keiichi Itakura on the chemical synthesis of short

DNA sequences attracted the attention of Herbert Boyer, pioneer of recombinant genetic technology (Smith Hughes 2011). Their work famously resulted in the commercial production of synthetic insulin and the world's first biotech company, Genentech; this success also took Riggs temporarily away from further work on methylation. By contrast, Holliday, an established scientist, managed to place the paper in *Science*, but his attempts to interest leading developmental biologists in his hypothesis failed (Holliday 2011).

Yet by the early 1980s, experimental support for Riggs' and Holliday's hypotheses accumulated, most prominently through the work of Adrian Bird—whose interest in methylation began during a postdoc with Max Birnstiel—and Edwin Southern in Edinburgh (Bird and Southern 1978; Gitschier 2009). Various phenomena in which the activity of genes was altered came to be explained using methylation: from the expression of retroviruses inserted into DNA genomes to the phenomenon of 'imprinting', where alleles that come from one parent are expressed, and from the other silenced (Jaehner et al. 1982; Reik et al. 1987). By the end of the decade, gene expression control through methylation was no longer a tentative hypothesis, but, rather, an established fact. And it was not just about methylation: the 1980s saw a rise in interest in 'chromatin biology' and recognition that gene expression can be regulated in multiple ways, of which methylation could either be the most important or just the most easily recognizable readout of more comprehensive changes in chromatin shape and density (Lappé and Landecker 2015).

In 1985, Holliday, in a short conference summary, introduced the term 'epimutation' to describe 'heritable changes in gene expression' (Holliday 1985). Holliday was an innovative thinker who viewed the contemporary molecular biology as conceptually impoverished. He echoed Conrad Waddington when he wrote about the need to focus on the 'strategy of genes' in the control of gene expression (Holliday 1989, 16). Yet when he wrote about inheritance and heritability, he referred to the cellular level: does the information inherited as cells divide entail more than DNA sequence? Do outside signals change the pattern of gene expression, is this a rejection of the 'central dogma' and could we speak of 'Lamarckism at the cellular level' (Holliday 1988, 259)? While he did consider the possibility of the inheritance of methylation patterns and/or chromatin conformation in the germ line, this was never his key concern (Holliday 1987). The first strong argument in favour of 'transgenerational epigenetic inheritance' was put forward around the same time by the Israeli geneticist-turned-historian and philosopher of science, Eva Jablonka, together with the British geneticist Marion Lamb. They

argued, for the first time, that 'the inherited epigenetic changes in the structure of chromatin can influence neo-Darwinian evolution as well as cause a type of 'Lamarckian' inheritance' (Jablonka and Lamb 1989). Lamb and, especially, Jablonka would go on to become the staunchest proponents of epigenetic inheritance, 'dissenters' against the extant paradigm of heredity and evolution (Jablonka and Lamb 2005).

Throughout this period, the term *epigenetic*(*s*) was still used—if at all—in Waddington's sense, to denote causal mechanisms at work in development. Holliday defined 'epigenetic' as 'changes in gene activity *during development*'. But Jablonka and Lamb expanded and updated this definition, saying that 'in addition to the instructions coded in the base sequence of DNA, genes can carry and transmit information embedded in the structure and conformation of chromatin. Such information is epigenetic information (…) it will reflect the developmental and functional history of the genes, and it will be involved in their present and future activity' (Jablonka and Lamb 1989). The new meaning of *epigenetic*, as a catch-all term to describe anything around and on, but not within, the sequence began to gain popularity soon afterwards.

Could, then, 1975, and these two articles, be regarded as the beginnings of epigenetics? They were the first papers to suggest methylation as a mechanism for regulation of gene expression in vertebrates. Methylation would then go on to become the best studied, and best known, epigenetic mechanism. The story of papers rejected by journals and ignored by peers until much later also fits into a narrative of innovation ahead of its time. With his suggestions of epigenetic inheritance, Holliday's work was of interest to 'dissenters'. In that sense, 1975 appeals across the board.

But if we read these papers closely, then a different picture emerges. The mid-1970s genetics was all about the possibilities opened up by new technologies that used enzymes to cut out and then stick together pieces of DNA. An enzyme, methylase, plays a central role in the two 'methylation' papers too: indeed, we could easily read them not as papers about a mechanism for gene regulation, but rather about the activity of an enzyme acting upon DNA. The abstract of Riggs' article says that 'a key feature of the model is the proposal of sequence-specific DNA methylases that methylate unmethylated sites with great difficulty but easily methylate half-methylated sites'. Pugh and Holliday's paper calls the modification of bases 'enzymic', not 'epigenetic'; the most prominent section of the paper is dedicated to 'modification enzymes'. And in that sense these papers, rather than breaking up with the genetic tradition, make epigenetics firmly part of it.

Of Famines and Ancestors: How Epigenetics Became Famous

By 1996, the field had grown enough to require a book-sized overview of epigenetic research across the range of mechanisms, problems and experimental models: plants, mammals and microbes (Riggs et al. 1996). Riggs and Holliday occupied prominent positions as co-editors (Riggs) and/or authors of several chapters (both). Reprints of their 1975 articles cemented their status as the founding fathers of the field. Though much larger than just ten years previously, epigenetics was still a field practised by geneticists, within departments of genetics, and solving questions that had troubled geneticists for decades. Genetic imprinting, for instance, may be considered the main epigenetic research problem through the late 1980s and 1990s, pursued by multiple research groups. It was also the question that had puzzled geneticists for decades: a non-random inheritance process defying the rules of classical Mendelian inheritance. But in the early 2000s, several groups studying problems directly relevant to human health, located in or closely connected to medical schools and using mammalian experimental models, entered the field. Their appearance changed the key questions in the field and its public perception. Through the work of these groups and the publicity that they received, epigenetics both became a household name and attracted much controversy.

Of these, three would become the best known: Michael Skinner's laboratory at Washington State University, Michael Meaney and Moshe Szyf's group at McGill in Montreal and the Southampton group (Skinner and Anway 2005; Weaver et al. 2002; Lillycrop et al. 2005). Michael Skinner came into epigenetics from reproductive toxicology, where he studied how exposure to certain chemicals, in particular those acting on the endocrine system, changes the reproductive function of affected animals and their offspring. Michael Meaney's long-term interest in how early life events—and in particular parental care—influence later-life response to stress was in the early 2000s turned into an epigenetic problem. A crucial component was the collaboration with Moshe Szyf, a geneticist with a long-term interest in the reversibility of epigenetic marks and its clinical applications: the development of 'epigenetic drugs' that would reverse pathological chromatin modifications (Szyf 2009). Meaney's research, with its focus on psychological stress, emotions and parent-child relationships, later extended to intergenerational trauma, attracted the most attention both by media and social scientists—and most controversy, for its focus on maternal care (Richardson et al. 2014). Finally, the Southampton group was originally a foetal physiology laboratory that in the early 1990s was central to the establishment of the field of 'developmental origins of health

and disease' (DOHaD). The field originated in observations of correlations between conditions of early-life and later-life health in historical cohorts in British public records and turned them into clinical and experimental physiological problems (Gluckman et al. 2015). DOHaD hugely expanded through the 1990s, yet it was also plagued by accusations that it found correlations rather than causations. Epigenetics provided a plausible mechanism to show how events present in early life exerted influences later on. In the process, parental and infant nutrition became recognized as part of the 'molecular environment' of the organism (Landecker 2011).

Disentangling the multiple influences that made epigenetics the buzzword that it is today is a demanding task. Epigenetics is often pitched against genetics—'soft' versus 'hard' heredity—and, certainly, relations between 'new' epigeneticists, in particular those bold enough to advocate transgenerational genetic inheritance, and 'orthodox' geneticists have not always been harmonious.[5] Yet, as this chapter has shown, epigenetics arose from genetics and, largely, remains part of it. The field emerged out of attempts to solve the pressing problem of post-war genetics: the control of gene expression. Although the research of the 'new' epigeneticists applies epigenetic tools to questions intractable to clinical and experimental physiological methods, the rise of epigenetics is perhaps better explained by the limitations of biological knowledge acquired by DNA sequence alone, as exemplified by the Human Genome Project (HGP). As predicted by Evelyn Fox-Keller more than 15 years ago, the completion of the HGP, instead of supporting, undermined the very concept of the gene (Fox-Keller 2000, 5–6). The realization that knowledge of the DNA sequence is just the start of understanding the phenotype fuelled the rise of genomics and epigenetics. The failure of expensive genome-wide association studies, projects focusing on correlating sequence variation with phenotypic (often disease-related) outcomes, has further increased interest in other approaches (Maher 2008).

These are the narrow reasons for the success of epigenetics: but how should we explain the broader change in our outlook? A biological perspective that acknowledges complexity but continues to look inward, into the cell, is easily imaginable. Instead, epigenetics has turned outward to study how our changing environment—food, relationships with people, chemicals—increases the risk of common illnesses and affects reproductive function. This outlook, of course, speaks to the main concern of our times: how we (and what better symbol for us than our genomes?) interact with our environment. Epigenetics is a facet of a larger transformation in biological science towards characterizing the organism as interconnected, plastic, permeable and responsive to changes in its surroundings: a symbiotic community of micro- and macroscopic life. Meloni, Williams et al. (2016) summarize this shift as:

> (1) An unprecedented temporalization, spatialization, permeability to material surroundings, and plasticity of genomic functioning, with profound implications for the notion of heredity; (2) a shift in evolutionary thinking from individualism utilitarianism to the current view of evolution as favouring prosocial behaviours; (3) the increasing understanding that the brain is a multiply connected device profoundly shaped by social influences (…) (4) an increasing emphasis on symbiotic processes (5) a new attention to microbial life and its conceptual implications in terms of networks of ecological interaction.

This is why epigenetics has become so popular among those evolutionary scientists, developmental biologists and philosophers of biology who view the evolutionary model built on the Modern Synthesis as an overly reductionist and unsatisfactory explanation for observed change in the organic world (e.g. Laland et al. 2014). I characterized this very diverse group as 'dissenters' in the introduction to this chapter. Their view of epigenetics does not necessarily correspond with the prevailing position in the field. And yet it holds much appeal. This, I propose, could be seen as an expression of a large shift in biological science, privileging 'connectivity', 'crosstalk' and 'exchange' over one of 'control' that characterized earlier decades.

Notes

1. For example, Richard Dawkins described the inheritance of epigenetic effects as 'a flash in the pan, both in its evolutionary significance and the "15 minutes of fame" which he declares it is enjoying undeservedly' (Webb 2016).
2. Robin Holliday, who can be regarded the founder of modern epigenetics, devotes a short paragraph to the era before Waddington, and that paragraph is mostly about genetics, Morgan and Mendel—and then another short paragraph to Waddington (to say that 'not many scientists were influenced by him') and genetics in the 1960s. See Holliday(2012).
3. 'To Waddington, epigenetics was the study of the way the phenotype was determined by the genotype, and he felt that the only way to get at this was to understand how genes work at the molecular level.' So Birnstiel focused on separating out genes—later moving onto histone genes. See in Grunstein and Bird (2015).
4. An overview of Riggs' early research career at the City of Hope may be found here http://breakthroughs.cityofhope.org/art-riggs-epigenetics
5. So Michael Skinner has been cited to say that one of the forces working against him were 'genetic determinists clinging to an old paradigm' (Interlandi 2013).

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Part II

Genomics, Postgenomics, Epigenetics and Society

9

Scrutinizing the Epigenetics Revolution

Maurizio Meloni and Giuseppe Testa

Who you are is written in both pen and pencil: things written in pen you can't change: that's DNA; but things written in pencil, you can: that's epigenetics.
(Reliv International, promoting the soy peptide extract, LunaRich X™)

Succeeding by Blurring: The Irresistible Rise of Molecular Epigenetics

Molecular epigenetics, the "next big thing" in the world of bioscience (Ebrahim 2012), is a scientific success story that thrives in the ambiguity of its own definition. As to success, there can be little doubt about it: it is enough to look at the tenfold increase, over the last decade, in the number of publications carrying "epigenetic" in their title (Haig 2012). Only in 2011 the figure

Both the authors contributed equally. Article originally appeared in *BioSocieties* (2014) 9, 431–456. doi:10.1057/biosoc.2014.22; published online 4 August 2014. This article was originally published under a CC-BY License (https://creativecommons.org/licenses/by/3.0). It is re-printed here under the same license.

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of publications in the field had reached the astonishing amount of several thousands, possibly up to 20,000 depending on the search criteria (Jirtle 2012), and at any rate has continued to increase since then. Similar efforts aimed at computing the rise of epigenetics in terms of new networks, institutes, conferences, curricula and journals confirm the vertical growth of the field across the full range of academic indicators.

Within a few years, ambitious large-scale projects, such as the International Human Epigenome Consortium (IHEC) or the NIH Roadmap Epigenomics Mapping Consortium, aiming at mapping human epigenomes for a variety of cell types and/or disease states, have been launched worldwide. New journals (*Epigenetics, Epigenetics and Chromatin, Clinical Epigenetics*), professional bodies (the Epigenetic Society, the Clinical Epigenetics Society) and research centers have also appeared in just a decade. In sum, epigenetics has provided "a banner under which a new scientific movement has advanced" (Haig 2012, 5). Even beyond the boundaries of biomedicine, various other disciplines have started to signal the impact of epigenetics on some of their fundamental tenets: from bioethics (Dupras et al. 2012) to human geography (Guthman and Mansfield 2013), from political (Hedlund 2012) to legal theory (Rothstein et al. 2009), from epidemiology (Relton and Davey Smith 2012) to the philosophy of identity (Boniolo and Testa 2011).

Unsurprisingly, even a cursory glimpse into popular media reveals the increasing stronghold of epigenetics also on public imaginary. Epigenetics has gone pop (Davey Smith 2012) occupying the cover of global magazines under sensationalist claims such as "victory over the gene" (Der Spiegel 2010) or "your DNA isn't your destiny" (*Time Magazine*; see Cloud 2010). Holistic medicine and various spiritual advices are being reframed in epigenetic terms (Church 2007). Perhaps unsurprisingly, a new market niche has also started to emerge with companies spinning the business potential of the epigenetic idiom, as exemplified in the case of Reliv International, a producer of nutritional supplements, that launched its latest soy extract under the banner "You to SuperYou: Direct Your DNA Naturally Through Nutritional Epigenetics" (reliv.com/lunasin-and-epigenetics).

An Epistemology of the Imprecise

Precisely as a field, however, epigenetics seems to flourish in the remarkable ambiguity of its defining term, with its apparent ability to accommodate—and productively align—a rather diverse range of biological questions and epistemic stances. Echoing Rheinberger's (2003) endorsement for an "epistemology of the imprecise", we argue that the ability to entertain multiple understandings of what constitute epigenetic phenomena, and hence multiple ways to secure epi-

genetic evidence, is foundational to epigenetics' rise, both as a scientific discipline and as a popular phenomenon. Expanding on the notion of "boundary object" (Star and Griesemer 1989), Rheinberger (2003) framed the gene as a boundary object that molecular biology has been gradually encasing within an eminently flexible boundary concept, thus supporting the claim that "boundary objects require boundary concepts" because, "as long as the objects of research are in flux, the corresponding concepts must remain in flux, too". The same we believe applies today to epigenetics, with its elusiveness (Dupré 2012), polysemantic nature (Morange 2002, 56) and coexistence of multiple accepted meanings for some of its basic features (Haig 2004; see also Bird 2007 and Ptashne 2007).

In what follows, we thus start out not with the aim to provide a full disambiguation of epigenetics (including its more recent -omic descendant *epigenomics*, in which epigenetic regulation is studied at the level of the entire genome), as this would be at this stage largely futile and indeed counterproductive. Rather, we find it useful to trace the contours of this eminently flexible concept (epigenetic) and of the versatile fields that its flexibility propels. Specifically, our first goal is to highlight some key junctures at which the diverse streams of epigenetic research collide as well as the main knots through which they become entangled or conflated. The reason is that these instances of epistemic blurring open for social theory unique entry points to engage with the potentially transforming aspects of this burgeoning field.

Sources and Boundaries of Epigenetics

Epigenetics has a long history in biology, and its current molecular reconfiguration is the result of a series of conceptual and experimental shifts. The notion of epigenetics was first coined by embryologist and developmental biologist C. H. Waddington (1905–1975) in the 1940s to define in a broader *non-molecular sense* the "whole complex of developmental processes" that connects genotype and phenotype. "It is convenient to have a name for this complex" Waddington writes, and "'epigenotype' seems suitable" (reprinted in Waddington 2012). Note as an aside that the neologism *epigenetics* was coined by Waddington as a derivative of epigenesis (Van Speybroeck 2002), that is in a developmental sense, and was not meant in the current popular sense of what goes beyond/upon (*epi*—in Greek) the gene.

A second parallel origin of the concept seems to have had a stronger influence on the present understanding. This second tradition originates with Nanney's (1958) paper, *Epigenetic Control Systems*, and refers more specifically to the expression of genetic sequences (Haig 2012; Griffiths and Stotz 2013). As Haig explains, in Nanney epigenetic control refers to "which volume in the

library of genetic specificities was to be expressed in a particular cell". It is this second, more squarely molecular meaning that resonates to a greater extent with contemporary practices and that we refer to as "molecular epigenetics" to differentiate it from the original, developmentally centered and broader Waddingtonian sense (see for a distinction, Griffiths and Stotz 2013). In turn this "library-scanning" view is itself broad enough to accommodate two only partially overlapping meanings of molecular epigenetics.

On the one hand, in fact, the library analogy forms the backbone for today's broader—and in some respects more shallow—understanding of molecular epigenetic, where the "epi" has come to refer to virtually all levels of cellular function that overlay genes while representing the result, or indeed the cause, of their differential expression in different cells and/or in different conditions. This operational definition includes the full complement of chromatin (i.e. the three-dimensional mesh of structural and regulatory proteins within which most DNA metabolism takes place) but also the transcriptome, the proteome and the various omic-slices into which life's complexity has come to be parsed along the biochemical classification of its constituent molecules. In this sense, Nanney's definition, at its broadest, translates epigenetics into a problem, or rather into *the* problem of gene expression, and depending on the level at which one chooses to analyze the latter, the former becomes more or less distant from its original physical link to the genome.

The second, more precise and demanding meaning in molecular epigenetics involves operational definitions that are mostly negative, as in the study of "any long-term change in gene function that persists even when the initial trigger is long gone that *does not involve* a change in gene sequence or structure" (McGowan and Szyf 2010, 67 our italics), or of a "phenotypic variation that is not attributable to genetic variation" (Champagne 2010, 300), or of that portion of phenotypes that is transmitted though cell division or organismal reproduction but that is not encoded in DNA. In all evidence, we are still fully within the library analogy, except that now the only volumes that count are those that remain open long after the first reader is done with them.

It is apparent that both meanings of epigenetic deflate the role of genes as causally privileged determinants of phenotypes, the former by emphasizing the regulatory context that extracts diverse functional outputs from the same genome, the latter by highlighting those instances in which non-genetic changes persist, either in time or in space or in both. Viewed from this angle, both strands of epigenetic thinking and experimenting are contributing to a style of thought that, following in particular Griffiths and Stotz (2013), we can define as postgenomic. In the

> postgenomic era, when complete genome sequences are available for an increasing range of organisms, the range of molecular actors has expanded greatly. The genome is not merely a collection of genes, but houses diverse other functional elements. Genes no longer have a single function closely related to their structure, but respond in a flexible manner to signals from a massive regulatory architecture that is, increasingly, the real focus of research in 'genetics'. (Griffiths and Stotz 2013, 2)

Importantly, here postgenomic and postgenomics are meant not only *chronologically* (i.e. what has happened after/*post* the deciphering of the Human Genome in 2003) but also *epistemologically*, as the recognition of those gaps in knowledge and unforeseen complexities surrounding the gene (Maher 2008) that have made our understanding of its function cautiously provisional and perennially contingent.

Increasingly, it is under the overarching umbrella of epigenetics (in the first, more shallow meaning that we have sketched above) that the disentanglement of these new complexities is expected to take place, promoting a conflation of the *epigenetic* with the *postgenomic* around the context-dependent view of the gene (Keller 2000; Oyama et al. 2001; Moss 2003; Robert 2004; Mameli 2005; Morange 2006; Stotz 2006, 2008; Stotz et al. 2006; Griffiths and Stotz 2013; Nowotny and Testa 2011). In this contextual view, genes are addressed as "catalysts" more than "codes" (Elman et al. 1996), "followers" rather than "leaders" (West-Eberhard 2003), "embedded inside cells and their complex chemical environments" that are, in turn, embedded in organs, systems and societies (Lewkowicz 2011). As Meaney emblematically writes:

> the function of the gene can only be fully understood in terms of the cellular environment in which it operates. And the cellular environment, of course, is dynamic, changing constantly as a result of signals from other cells, including those that derive from events occurring in the external environment. Ultimately, function can only be understood in terms of the interaction between environmental signals and the genome. (2010, 48)

Expectedly, this way of thinking about biological processes has major consequences for established dichotomies of twentieth-century biosciences and in particular for the genotype/phenotype distinction (coined by Johannsen in the 1910s). In the context of the gene-centrism of the modern evolutionary synthesis, the relationship between genotype and phenotype was typically thought of as a relationship between a cause and its visible and mechanistically deduced effects, "between a plan and a product" (Jablonka and Lamb

2005, 33). In that theoretical framework, the chain of causal links moved unidirectionally from the active genotype to the "dead-end" phenotype. In the postgenomic era, instead, the relationship between genotype and phenotype is more often represented, rather than as a linear causal chain, in terms of a "rope" (Griesemer 2002), a term that wishes to capture the profound intertwinement of the actual genetic material with the various layers of its phenotypic "appearance" (Oyama et al. 2001). Surfing over this rope, epigenetics resumes its original Waddingtonian emphasis, becoming a convenient heading for the multiple strands and complex apparatus of "developmental transformations intervening *between* genotype and phenotype" (Pigliucci and Muller 2010, 308, our italics; see also Schlichting and Pigliucci 1998; Robert 2004; Hallgrímsson and Hall 2011).

Pulling together the threads of these imbricated, blurred, or at times frankly competing understandings of epigenetics, we can thus posit that its current and unifying thrust is, in a nutshell, the *promise to capture the analogical vastness of the* "*environmental signals*" recounted above through the digital representation of their molecular responses. If what seemed irreducibly analogic (the social, the environmental, the biographical, the idiosyncratically human) needs to be overlaid onto the digital genome of the informationally ripe age in a dyadic flow of reciprocal reactivity, then it seems that this overlay can succeed only once the analogic is interrogated, parsed and cast into *genome-friendly, code-compatible digital representations* (RNA, DNA found associated to specific chromatin modifications as in chromatin immunoprecipitation or ChIP, methylated DNAs, etc.). In this respect, epigenomic profiles (transcriptomes, chromatin maps and the further bits of living matter that technology is progressively digitizing, from proteomes to metabolomes, etc.) are increasingly fulfilling, in today's biology, the role that cellular lineages took on in what Morange refers to as the "crisis of molecular biology" in the 1970s and 1980s. Following the spectacular dissection of the genetic code, the challenge to explain development in equally molecular and code-compatible terms proved rapidly a major one. As Morange notes,

> The roots of the crisis should be sought at the epistemological level: what molecular biologists cruelly lacked, what led them to a feeling of decadence, was the total absence of a definition of … what would be an explanation of development, [… for this …] required that another level of description of the biological facts not be discovered, but valorized. This level was the cellular level, and this explains the dramatic development of cell biology during these years. Cell biology provided what Harold Kincaid called the 'place holders', the terms

> which are introduced to designate an entity, a process for which we have good evidence, but whose precise nature is unknown. (Morange 1997, 390)

Similarly, we argue that epigenomic profiles, in their expanding variety, provide the new place holders to anchor the environment to the genome and enable the attending analogic–digital translations, conceptually as much as experimentally.

Thus, having briefly mapped *the blurred and thereby productive* boundaries of today's epigenetics, we move now to explore three research pathways for an emerging field of "epigenetics and society": (1) epigenetic vistas across controversies, hypes and sociotechnical imaginaries; (2) epigenetics between facts and concerns; and (3) the emergence of a new molecular materialism mediated by the instruments and classifications of epigenetic research. Recent studies have begun to chart the contours of the new social studies of epigenetics, from an inquiry into the attitudes of epigenetics researchers (Tolwinski 2013) to an articulate endorsement of the possible types of engagement between epigenetics and the social sciences, ranging from the more "interventionist" to the more "self-reflexive" streams of Science and Technology Studies (Pickersgill et al. 2013).

Here we advance this agenda further by providing three critical elements: (1) a methodological anchor to the epistemology of the imprecise, which positions epigenetics *vis-a-vis* both its scientific antecedents (chiefly molecular genetics) and its prospective partner disciplines within the social sciences; (2) a focus on the digital feature of current epigenetics as a key resource to trace its explanatory success, again *vis-a-vis* its antecedents and prospective partners; and (3) a tripartite research program that should hopefully foster the exercise of a rigorous "political epistemology", for which the focus on epigenetics provides a paradigm of the inherently sociopolitical nature of biological discourse.

Pathway 1: Epigenetic Vistas Across Controversies, Hypes and Sociotechnical Imaginaries

The very notions of a "decade of the epigenome" (Martens et al. 2011) or even of an "era of epigenetics" (Hurd 2010) reveal how rapidly epigenetics has been rising to that level of salience, in both scientific and societal imaginary, that warrants the dedication of defined timescales in public attention and investment. And despite the fact that this "decade" has just begun, it is not too early to reflect on the societal impact of epigenetics. As we have already seen in the

past for genetics, neuroscience or stem cells, often pioneering but preliminary findings are construed as providing evidence upon which to draw consequences for human health and well-being, especially by policymakers, media commentators, lifestyle advisers and sometimes natural and social scientists themselves.

Indeed, there is the feeling that is already happening for epigenetics: in popular books epigenetics has already been employed to make claims about human talent (Shenk 2010), and in scientific articles epigenetic markers have been used to make claims about social inequalities and race differences in health (Kuzawa and Sweet 2009; Wells 2010; McGuinness et al. 2012). In triangulation with findings from the Developmental Origin of Health and Disease (DOHaD) literature, epigenetic claims have been used to target mothers as a new center of responsibility (Paul 2010), recasting the maternal body into a sort of "epigenetic vector" (Richardson 2016). Other expectations, as we will review later, exist with regard to epigenetics providing a possible new ground for legal claims and extended notions of responsibility.

A social study of epigenetics therefore should start from a reflexive analysis of the way in which epigenetic knowledge is becoming "a social phenomenon in itself" (Landecker and Panofsky 2013), including the imaginaries and visions that are catalyzing this transition, and that we will refer to here as *epigenetic imagination*. Its analysis will provide social scientists with a vast repertoire of empirical sources where to observe the full thickness of science and society interactions through three of the most flourishing streams of research in Sociology of Scientific Knowledge (SSK) and STS: (1a) the sociology of scientific controversies; (1b) the sociology of hypes and expectations; (1c) the emergence of sociotechnical imaginaries.

Controversial Knowledge

The study of scientific controversies has been a key heuristic methodology in SSK and STS for more than three decades now, prompting analysts to focus on the way in which scientific disagreement is handled, on the resources and practices that allow disputes to arise and persist, and finally on the decisional mechanisms by which consensus is reached (Nelkin 1979, 1992; Engelhardt and Caplan 1987; Brante et al. 1993; Martin and Richards 1995; Roosth and Silbey 2009; Martin 2008). Precisely through the blurred and at times frankly competing epistemologies that underpin the classification of its phenomena, epigenetics offers no shortage of controversies, especially around the following themes: (1) the relevance of intergenerational inheritance of epigenetic traits

especially in higher organisms; (2) the reappraisal of the concept of gene, and of the assessment of its functional significance, in the light of the unforeseen extent of several *epi*-layers of regulation (as most vividly captured in the heated controversies over the universe of non-coding RNAs unearthed by the ENCODE Project (Doolittle 2013; Graur et al. 2013); (3) the tension between the Modern Evolutionary Synthesis as a settled canon and the renewed interest, much more vocal than in the past, in epigenetic, neo-Lamarckian mechanisms of inheritance (Jablonka and Lamb 2005); and (4) the epigenetic underpinnings of human behaviors.

Here we briefly focus on two such controversies whose implications appear particularly far-reaching for the strands of sociological inquiry that we pursue in this work.

The first is a display of semantic tension more than an actual controversy, but illustrates nicely the contentious potential of the blurring that we have previously hailed as a key factor in epigenetics' success and the ambiguity of the epistemic space in which epigenetics prospers today. In a recent popular publication Eric Nestler, Director of the Friedman Brain Institute at the Mount Sinai Medical Center in New York and co-author of a very much quoted study on the epigenetics of psychiatric disorders (Tsankova et al. 2007), expresses caution on the potential of epigenetics by claiming that "much more work is therefore needed before we will know the extent to which epigenetic mechanisms represent *a third factor—beyond nature and nurture*—in controlling an individual's traits in health and disease" (Nestler 2013).

For Sweatt (2013) instead, one of the leaders of the emerging field of neuroepigenetics, "it is now clear that there is a dynamic interplay between genes and experience, a clearly delineated and biochemically driven mechanistic interface *between nature and nurture*. That mechanistic interface is epigenetics" (p. 624). The point here, however, is not so much about caution versus optimism. Rather, what counts for us is the radically distinct epistemic space in which epigenetics is recruited as an explanatory resource, by two authors who are both authorities in their field and have recently co-edited an important publication on the epigenetics of regulation of the nervous system (Sweatt et al. 2013).

In what Keller (2010) has defined as the mirage of a space between nature and nurture, Nestler posits epigenetic mechanisms as a third factor that reaches beyond both, whereas Sweatt sees them as the interface that obliterates the space and dispels the mirage. "Beyond" versus "between": there lies the difference it would seem, and indeed it will be interesting for the analyst to see whether such semantic tensions end up propelling fundamental theoretical or experimental distinctions, or whether they will remain as just the

rather innocuous legacy of that systematic blurring we have outlined above. In other words, it is conceivable that if epigenetic mechanisms are framed as distinct from both nature (a proxy for genes in such discourses) and nurture (a proxy for environmental triggers), rather than as the lens that illuminates the former through the latter (and vice versa) the very questions that end up being asked *may well differ significantly*, and with them also the host of attending experimental systems.

The second controversy concerns the difficulty in establishing the existence and relevance of transgenerational epigenetic inheritance in humans. Predisposition to colorectal cancer can be inherited through genetic mutations in several genes, including *MutL homolog 1, colon cancer, nonpolyposis type 2* (MLH1) and *MutS protein homolog 2* (MSH2). Some cases, however, were found to be inherited through epimutations in these two genes, that is to say in the abnormal methylation that ablated their function despite the integrity of their sequence, and they initially constituted the most striking example, documented in molecular detail, of transgenerational epigenetic inheritance in humans (Chan et al. 2006). Subsequent scrutiny, however, revealed that MSH2 methylation (the epimutation) was due to a genetic mutation in a neighboring gene (Ligtenberg et al. 2009). And also for MLH1, while the jury is still out, it is proving difficult to rule out upstream genetic causes and to unequivocally establish that the epimutation is itself inherited through the gametes rather than being simply triggered right after fertilization (Daxinger and Whitelaw 2012).

Beyond the details of these fascinating cases, what emerges is the problem associated with the depth and breadth of the molecular gaze that informs current biology, with its skyrocketing ability to reveal more and more minute details but also with the attending challenge to define thresholds of epistemic significance for each of them (Nowotny and Testa 2011). For in the age of so-called next-generation sequencing (the term itself a testimony to the open-endedness of the whole pursuit), with genomes and epigenomes stretching like acres of naked nucleotides ready to be read and re-read with ever greater accuracy, proving a transgenerational epigenetic effect in the outbred human population, requires de facto that all possible genomic causes are excluded. And yet, the vaster the genomic space we wish to sample to that end and the more certain we wish to be of that exclusion, the digger we have to deep. Thus, these controversies are paradigmatic because they set the stage for probing, in the many similar cases that will undoubtedly follow, what comes to constitute epigenetic evidence in the first place, through which work of purification and through which "trial of strength" (Latour 1999), be it material or statistical.

Orthogonally to this peer-to-peer debate among scientists, a second source of friction is already well-identifiable in the tension between the supposed consensus around epigenetic knowledge, as it is propagated in society, in the front-page headlines and also in some social science literature, and its uncertain, speculative status within the scientific community itself. It is in this mismatch between what is established and what is at present a source of heated scientific dispute that speculative assumptions, inflated discourses and enthusiastic media promotion, in a word all that create hypes around the epigenetic imaginary, are likely to find fertile ground. This brings us to the second point, the visions and expectations generated by epigenetic knowledge as it circulates through society.

Cycles of Hypes and Expectations from the Genome to the Epigenome and Back

As a test case for the sociology of expectations, epigenetic knowledge is also very well-positioned. A growing body of research over the last years has investigated the forward-looking dynamics of science and technologies and the "generative" role of expectations in "guiding activities, providing structure and legitimation, attracting interest and fostering investment" (Borup et al. 2006, 286; see also Brown et al. 2000; Brown and Michael 2003; Van Lente 1993, 2012). Although expectations have always been important in the modern history of science and technology, this stream of research has emphasized how "hyperbolic expectations about the future have become more significant or intense in late and advanced industrial modernity" (Borup et al. 2006). This saturation with anticipations, visions and promises has already accompanied the rise of genomics (Hedgecoe and Martin 2003; Fortun 2005; Sunder Rajan 2006; Martin et al. 2008; Tutton 2011) and it is against this backdrop that we wish to situate the current climax of expectations surrounding epigenomics. Specifically, we find that the hypes accompanying epigenomics, mainly at the level of popular science but also in sections of the scientific community, rest on a bivalent understanding of its relationship with genomics: on the one hand *as a missing link that can succeed where genomics purportedly failed*, on the other *as a quantum leap enabled by the very success of genomics*. This is because epigenomics, as we briefly summarize below, is exploding at a specific and highly interesting phase in the cycle of expectations and promises of genomics itself (for the literature on hype cycles, Van Lente et al. 2013).

Following the relative disappointment for the slow pace of translation of genomic knowledge into clinical practice, genomics is in fact experiencing

now a major come back driven largely by the unprecedented leap in our ability to sequence individual genomes. In a nutshell we can say that the newly found confidence in the genome as an explanatory resource for human traits (especially diseases) marks precisely the transition from the slightly abstract notion of the genome writ large coming out of the Human Genome Project (HGP) to the eminently concrete sequences of multitudes of individual genomes, individual not only in the sense that they come from individual beings but indeed, and increasingly so, from individual cells of the same being. From cancer (Burrell et al. 2013) to neurodevelopmental disorders (Poduri et al. 2013) to, indeed, healthy development (De 2011), next-generation sequencing has brought the genetic heterogeneity of our cells back to the fore, thereby beginning to illuminate the truly unprecedented extent of our somatic mosaicism (i.e. of the genetic differences found among cells of the same organism) and to propose for it an important role in a variety of conditions. Indeed, in an almost ironic twist, the very technology of epigenetic reprogramming (which allows to reset the epigenome of individual somatic cells and derive from them unlimited amounts of pluripotent stem cells which, among other things, greatly facilitates genome sequencing) is one of the most powerful approaches to probe the depth of our genomic diversity, both within and among individuals (Takahashi and Yamanaka 2006; Abyzov et al. 2012). Against the backdrop of these developments, which together re-emphasize the importance of genomes as explanatory resource, we can then observe how it is an intersection of two discourses that upholds the bivalent relationship between genomics and epigenomics that we have recounted above.

On the one hand, to the extent that the admittedly naïve expectations over the immediate impact of the HGP have not been fully realized, epigenomics has progressed within a new promissory discourse where its findings are conceptualized as the "key 'missing piece' of the etiological puzzle" and what will make justice of the promises of the now discredited "genocentric focus in our approach to human disease" (Szyf 2011). Examples of such a discourse abound and inform much of the excitement over epigenetics in biomedicine (Feinberg 2008; Choi and Friso 2010; Petronis 2010; Chadwick and O'Connor 2013; Mill and Heijmans 2013).

On the other hand, the ability to study both genomes *and* epigenomes *together* at unprecedented resolution has been inviting a different discourse where the former regains primacy in shaping the latter, from the emphasis on genetic mutations in epigenetic regulators that underlie an increasing number of diseases (Ronan et al. 2013) to the notion that somatic genetic mosaicism is not only widespread during development and aging but that it can itself

affect "the epigenetic patterns and levels of gene expression, and ultimately the phenotypes of cells" (De 2011). Clearly, depending on how far the pendulum swings toward the poles of these two discourses, one encounters a range of epistemic nuances, from the mutually exclusive attempts to replace the genome with the epigenome (or indeed vice versa) as explanatory resources, to the mutually reinforcing attempts to probe them in the increasingly visible circularity of their interconnections.

In this respect, and unsurprisingly, twin studies are proving to be an especially informative domain in which to flesh out the mutual reconfigurations of these two discourses. A source of permanent wonder throughout human history, twins have come to be a unique challenge and an equally unique opportunity once some of them "became" monozygotic, that is, once embryology and genetics led us to trace their identity to the sameness of cellular and genetic constituents, thus setting them apart from their "lesser" siblings that happened to share only a womb at a given time (i.e. the same *context of epigenetic triggers*, in today's language; see Nowotny and Testa 2011). The genetic identity of monozygotic twins, cast against the range of their phenotypic diversity, has thus become the most visible manifestation of the genome's insufficiency as sole or even main determinant/predictor for several human traits, offering for this very reason a unique entry point into the dissection of non-genetic contributions. In its proposed role of critical intermediate between genotype and phenotype or genotype and environment (along the many shifts we have encountered above), epigenetics has thus acquired increasing prominence in twin studies, as witnessed by what is arguably its most visionary and cogent pursuit, namely the Peri/Postnatal Epigenetics Twin Study with its systematic and prospective scrutiny of individual epigenetic variation in twin cohorts starting from birth (Loke 2013), that in turn builds on the first systematic scrutiny of the epigenetic changes that accrue over the lifetime of monozygotic twins (Fraga et al. 2005).

Against this backdrop, the recent popular science book by Tim Spector, *Identically Different* (2012) becomes a powerful example precisely because Spector is both a leading scientist in twin studies (Professor of Genetic Epidemiology at Kings College and founder of the UK Twins Registry, one of the largest world collections on twins) and, in this case, a popularizer of epigenetic findings. The book opens with Spector's confession, "Until three years ago, I was one of the many scientists who took the gene-centric view of the universe for granted", and proceeds to translate into popular culture the epistemic tensions of epigenetic research in its quest for the new paradigm that fills in *where classic gene-centrism has failed*. If twin studies ground in the genome only 35 percent of the variance that accounts for a whole range of

psychological and medical traits (Spector 2012,147), where should one look for the remaining unexplained variance, Spector's key argument goes, if not in epigenetics? The point, however, lies precisely in how that unexplained variance, the analogical vastness of environmental signals we have recounted above, is *being cast within the same digitally friendly language of maps, codes or blueprints* that enabled the gene-centric paradigm to rise in the first place. Just to quote an example from Spector (2012), the "religious susceptibility gene" remains steady, in this narrative, in the ambition to ground culturally sophisticated phenomena onto molecular codes, with the difference that these codes now take the form of flexible and hence reversible switches rather than fixed circuits (p. 107).

In sum, our conclusion is mixed. If one were to look in epigenetics for a radical disavowal of the digital primacy of the genetic language, she would be disappointed and might well conclude, following the famous dictum from the Italian twentieth-century masterpiece *The Leopard* that "everything needs to change, so everything can stay the same" (Tomasi di Lampedusa [1958]1960). If instead one looked in epigenetics for a defiance of genetic determinism that succeeds precisely by applying the same digital language but to include rather than to exclude context (environment, biography, lifestyle and so on), then she may more likely perceive the innovative thrust of the field.

Epigenetic Imaginaries

A growing interest in the broader landscape where scientists operate as "cultural producers" or "sociocultural entrepreneurs" has characterized recent work in the social studies of science (Fujimura 2003). The notion of imagination and imaginaries has been employed by several authors to emphasize the "historically inflected and socioculturally sedimented" context where scientific knowledge takes shape and "interpolates technical, biomaterial, political-economic, social, cultural, and ethical elements" (Fortun and Fortun 2005). The way scientific discourses are embedded with other cultural discourses and contribute to trigger the imagination of scientists and society has been analyzed especially in genetics and genomics. In a slightly different meaning, Jasanoff and Kim have introduced the notion of sociotechnical imaginaries to emphasize, in the context of a study on nuclear power, the "promotion and reception of science and technology by non-scientific actors and institutions" and national differences in "collectively imagined forms of social life and social order reflected in (…) scientific and/or technological projects" (2009, 120). It is therefore in this context of renewed interest toward the imagina-

tive/imaginary context of science that it is possible to suggest a third line of reflexive investigation on epigenetic knowledge, what we can name here the "epigenetic imaginaries'.

Epigenetics has shown in just few years to be a powerful imaginative tool. The profound impact of epigenetics on society and its symbolic landscape is exemplified by the rapid diffusion within the popular press, in pop science books (Francis 2011; Carey 2012) and documentaries (such as the BBC "Ghost in Your Gene" program or the more recent "The hidden life of our genes") of a whole series of new foundational stories that seem to play the same function as Dora's case did for Freud, Little Albert for behaviorism and Phineas Gage has been doing recently for moral neuroscience. These truly "dramatic epigenetic pin ups" (Davey Smith 2012) are constantly retold among the wider public to illustrate the social/historical relevance of epigenetics: from the "thrifty phenotype" of the DOHaD hypothesis to the impact of the Dutch Hunger Winter (1944–1945) on the lifespan, decades later, of people prenatally exposed to it (among which, we are told, Audrey Hepburn), from the consequences of the siege of Leningrad to the transgenerational effects of famine in the remote village of Overkalix, in North Sweden.

Also the more squarely experimental stories are shaping intensely contemporary imaginary, becoming true *topoi* in the genre: it is the case, for instance, of the switching on and off of the *agouti* gene in mice (through a methyl-rich maternal diet in gestation) that makes genetically identical offspring look phenotypically different, in coat color but, more importantly, in weight and susceptibility to disease (Waterland and Jirtle 2003, 2004). The passage on to the second generation of such an effect also has become emblematic of the idea that not only a mother's but a grandmother's diet can have a profound impact on the health of the grandchildren, an idea popularized in a classic epigenetic slogan such as: "you are what your grandmother ate" (Pray 2004). A similar iconic status, especially for its possible implications for social research, has been reached by Meaney's (2001b) study on how variations in maternal behavior of rats alter methylation patterns in the offspring and how these epigenetic alterations affect the next generation, but can be reversed by cross-fostering the pups to more "affective mothers". Along with the study on glucocorticoid receptor and child abuse (McGowan et al. 2009), this study has been hailed as evidence of how social experience gets under the skin (Hyman 2009), and this metaphor has traveled widely in the social science context and is today reinforced by a parallel notion of epigenetic effects going "into the mind" (Toyokawa et al. 2012).

Finally, the epigenetic imagination is also about novel metaphorical resources (Nerlich and Stelmach 2013). These metaphors are sensibly differ-

ent from the language that characterized the genetic landscape. The metaphors of epigenetics are meant to show reversibility where before there was stability (the "pencil's trait" that can be erased versus the pen, the epigenetic software versus the genomic hardware), a variation on the genetic script (epigenetics as the German umlaut that can change meaning to a word without changing its material succession of letters (Urnov and Wolffe 2001), or epigenetics as a removable post-it, a mere annotation on the genetic script), the persistence of past experiences through generations ("a ghost in the genes", a "cellular memory of past events", a "nuclear time bomb in our genes", a poison, a curse, a scar, a mark in the genes) or holistic view of biological processes (epigenetics as a "symphony" of elements, replacing the absolutist role of the gene as "the director of the play"; see Noble 2006; Qiu 2006; Francis 2011), but also to reinforce a new language of programming (fetal programming, environmental and social programming and so on).

Pathway 2: Epigenetics Between Fact and Concern

A second crucial aspect in the emergence of an "epigenetics and society" research program concerns the possible political, legal and ethical implications of epigenetic research. Following in the footsteps of its HGP antecedents, also epigenetics has started to trigger its own share of studies on Ethical, Legal and Social Implications (ELSI).

There can be little doubt as to the relevance of the ELSI studies that were spurred by and within the HGP, in terms both of what they accomplished directly and of what they set in motion more broadly for a sociologically minded approach to developments in the life sciences. In this work, however, we set out a task for ourselves that is clearly distinct from a discussion of the ELSI of epigenetics and that we hope in fact will be helpful in steering it along innovative directions. The reason is that, even at its most sophisticated, in its very wording the ELSI idiom reveals deep-seated assumptions, often unintentional or at any rate unscrutinized, about the flow of innovation in knowledge-intensive societies. After all, when discussing the ELSI of something, the very emphasis on the *implications of* this or that betrays the underlying model in which technoscientific ingenuity precedes (in the softer version) or frankly drives (in the harder flavor) social innovation. The analytical task is thus parsed from the outset into a neat demarcation of objects: on the one hand, science (whose epistemic nitty gritty is more often than not black-boxed) and, on the other, society (or its many proxies, from laws to publics, from regulations to markets and so on).

This, however, bears little resemblance to what by now four decades of empirical work in STS have been consistently showing, namely that in technologically complex, knowledge-intensive societies the actions of epistemic and normative ordering, and their results, are not only interconnected but indeed mutually constitutive of each other. The idiom of co-production (Jasanoff 2004) has captured this symmetrical constitution with particular cogency, highlighting how, when such settlements are eventually reached, they end up establishing not only an epistemic but also a normative order. In Latourian terms (Latour 2004), we thus propose that the second pathway for an emerging social study of epigenetics is the following: to define how matters of epigenetic fact have already become mobilized as matters of social concern and, vice versa, how matters of social concern are becoming matters of epigenetic fact, all the while keeping alert to how, by the same token, also matters of epigenetic concern can become matters of social fact.

Specifically, we anticipate two prominent directions of this mobilization: (1) the digitization of the environment, with its attending discourse of collective and individual responsibility, including the notion of transgenerational accountability; and (2) the identification of epigenomically distinct subgroups/subpopulations aiming at objectifying in molecular terms disadvantageous conditions and/or unequal social structures.

Digitizing the Environment: Plasticity, Responsibility and Purity

The digitization of the environment, and its impact on responsibility, cuts across the main line of tension in molecular epigenetics, that between stability and reversibility. On the one hand, molecular epigenetics is what promises to unravel genome's openness to environmental influences, social factors and the biographical marks of personal experience, making visible in molecular detail its essence of "reactive genome", following Keller (2011) and more recently Griffiths and Stotz (2013). Almost by definition, this openness to the environment, in its broadest sense, invites the expectation of change, the notion that once the genome has been downgraded from the high citadel of causal primacy to the messy roundabouts of reactive developmental resources, biological fates become inherently reversible and porous to intervention. From the massive investment in epigenetic modifiers within drug discovery to the rising prominence of environmental epigenetics (in the flavor of either blessing or curse), much of current molecular epigenetics revolves around the promise of change. On the other hand, however, the more stringent epigenetic phenomena, and those that are

triggering more widespread fascination, are those that typically resist change, those states that defy in their stability the inherent disruption of genome regulation associated to the cycles of reproduction in cells or organisms.

Here, the same factors (environmental or else experiential) that promise change are also those that can leave permanent imprints or even scars. This ambivalence (or more appropriately: dialectic) is evident in Meaney and colleagues' groundbreaking studies on the effects of maternal care on gene expression and neural development in rat pups (Meaney 2001b; Weaver et al. 2004) that have acquired almost iconic status in the present exploration of the biosocial link, including a recent expansion of their work to the human brain (McGowan et al. 2009). These studies reflect this profound line of tension in epigenetic research, implicit in the very notion of plasticity (Malabou 2008). The plastic brain and the plastic genome are those that can give form but also receive form from the outside: you can change your genes, but also your genes (i.e. the way in which they operate) can be changed, insulted, permanently damaged (or improved) by environmental exposures. It is thus against the backdrop of this tension between passivity and activity that we can most productively situate epigenetics' intellectual program of molecularizing the environment in digital terms, thus making its impact on living beings measurable, archivable and comparable.

Unsurprisingly, this digitizing epistemology, along with the technological frontiers that it discloses and stimulates, is entering as a powerful resource in a reconfiguration of individual and collective responsibilities. The increasingly visible plasticity of the epigenome supports the new postgenomic discourse in which the genome is understood as something malleable that can be trained and modified through an "extended practice" (Spector 2012). "Practice" is key here, as it captures how the potential reversibility of epigenetic marks grounds the rationale for continuous intervention and/or maintenance that may safeguard their plastic and hence vulnerable states. Responsibility ensues thus in response to both implications of epigenomic plasticity: (1) on the one hand frailty and danger, with the call to protect one's own epigenome from external insults (be they related to lifestyle, occupational hazards, environmental pollutants and so on); (2) on the other opportunity and resource, with the promise to change and improve upon one's endowment.

This dialectic spans both scholarly and popular literature, as well-illustrated in a recent *Time* article where epigenetics is presented to the broad public as bringing "both good news and bad", the bad news being the vulnerability of the epigenome to wrong lifestyles ("eating too much can change the epigenetic marks atop your DNA in ways that cause the genes for obesity to express themselves too strongly and the genes for longevity to express themselves too

weakly" (Cloud 2010) and the good news being the newly recognized capacity "to manipulate epigenetic marks in the lab", which means that scientists "are developing drugs that treat illness simply by silencing bad genes and jump-starting good ones". In all evidence, what lies ahead, and is already starting to unfold, is a major expansion in the care of the self along Foucauldian lines (Foucault 1988) and as concrete examples of this digitizing thrust begin to emerge, they will constitute a rich palimpsest of options for STS scrutiny. In particular, the co-productionist framework will allow to unpack how the processes of gathering, standardizing and certifying epigenetic evidence will align with political, legal and economic rationalities in bringing about new settlements (or possibly reinforcing existing ones) across some of the most persistent dichotomies that structure our reflection on the human experience: normal versus pathological (or enhanced), safe versus dangerous, natural versus artificial, individual versus collective.

But if the epigenetic digitization of the environment functions in the spatial reconfiguration of the body *vis-a-vis* various sources of environmental exposure (along with the power structures within which they materialize), no less momentous is the temporal dimension of its impact. Indeed, inherent to the very same intellectual project is the notion that the epigenetic body is at once inhabited by the traces of its past and seeded with traces of its future. And these traces can stretch not only over one's own lifetime or over one's own offspring's lifetime, but possibly over the lifetime of several following generations. Indeed, as we saw above, the transgenerational resilience of epigenetic states, especially when it comes to humans, remains at once a topic of intense research (including heated controversies) and the magnet of greatest public fascination through the emphasis on an epigenetically haunted body, as most iconically captured in the very title of the BBC documentary on epigenetics "Ghost in Your Genes" and its bold announcement that "The lives of your grandparents—the air they breathed, the food they ate, even the things they saw, can directly affect you, decades later, despite you never experiencing these things yourself".

We see at play, in principle but increasingly also in practice, an *expansion of the concept of responsibility* that reaches well beyond the individual and her direct offspring, fostering *the materialization of new bonds among generations*. Indeed, precisely this aspect has already triggered the attention of bioethicists and legal scholars in reassessing the intergenerational impact of traumatic social events and forecasting how "Epigenetic effects caused by chemicals and other environmental agents may provide a new source of litigation and liability under the common law. Such litigation, especially when it involves second- and third-generation effects, would raise a number of novel challenges and issues" (Rothstein et al. 2009). What is interesting here is how the ideas

of natural, normal and pure that have shaped the discourse on the genome as a collective resource in need of protection (as "heritage of humanity" characterized by a natural state, in UNESCO's wording) will map upon the epigenome when it comes to so-called *intergenerational equity*. We see already glimpses of such a one to one translation, as in the recommendation that "each generation should maintain the quality of the human genome and epigenome and pass it on in no worse condition than the present generation received it" (ibid.).

Yet, it is precisely the very notion of a "quality" of an epigenome that will likely become the terrain of both scientific and social controversies as we move from the already-challenging task of defining reference epigenomes as standards for the advancement of the field (i.e. the core mandate of IHEC) to the even greater challenge of accommodating and indeed interpreting those standards in terms of collective political intervention (Dupras et al. 2012; Hedlund 2012). "Each of us has far greater responsibility than we ever imagined!" claims a popular medical American website (www.drfranklipman.com/faqs-on-epigenetics/). Indeed, the most visible effect so far of this narrative of hyper-responsibilization is probably what emerges from the intense moralization of the maternal body and behaviors in the triangulation of epigenetic and DOHaD writings. Epidemiological studies linking the lifestyle (diet, smoking) of boys during puberty with the disease risk of their grandsons and in general the male line (Pembrey 2002; Pembrey et al. 2005) may possibly relieve the pressure on mothers, it has been claimed (Shulevitz 2012), but the maternal body and her lifestyle remain so far overwhelmingly central (Richardson 2016) as a target of responsibility for harmful epigenetic consequences on the child's health.[1]

Epigenetics in Social Policy and Public Health Discourses

The second axis of investigation regards the huge expectations placed on epigenetics in terms of social policy and public health. Biological arguments in social policy have a well-deserved history of being discredited as ad hoc justifications for natural inequalities, social hierarchies and the immutability of social structures. These arguments endemically reappear in the public arena as the recent polemics in the United Kingdom by a government policy advisor on "Genetics outweighs teaching" illustrates (Wintour 2013). Biology keeps being seen as a form of destiny but clearly epigenetics may introduce a strong discontinuity with this stereotypical thinking. By pointing to a new relationship between biological and social events, in which the social assumes a causative

role in shaping human biology to a degree unseen before (Landecker and Panofsky 2013), molecular epigenetics may produce significant conceptual changes in the applications of biological findings to social policy strategies.

Indeed, epigenetics is already being used in the service of explaining the persistence, within specific groups, of long-lasting social/health issues, such as obesity, cardiovascular disease, mental health, but also poverty, inequalities, neglect and their dysfunctional perpetuation generation after generation. Kuzawa's and Sweet's (2009) study on race is a very interesting example of this reconfiguration in epigenetic terms of racial disparities in cardiovascular health in the United States. Here an epigenetic developmental model of black–white disparities is said to provide "a more parsimonious explanation than genetics for the persistence of cardiovascular disease disparities between members of socially imposed racial categories". For the authors, epigenetics offers "an important set of mechanisms by which social influences can become embodied, having durable and even transgenerational influences on the most pressing US health disparities"(ibid.).

A second key example of a reconfiguration of social disparities in epigenetic terms comes from the empirical study of McGuinness et al. (2012) on the correlation between socio-economic status and levels of DNA methylation in Glasgow. This study, based on blood samples of 239 people from Glasgow's poorest and most affluent areas, found that global DNA hypomethylation was associated with the most deprived group of participants. The association between social deprivation and lower levels of methylation (in turn associated with enhanced inflammatory status and associated disease risk) enabled to posit for aberrant methylation, and by implication for other epigenetic signatures as well, the potential as new biomarker of social adversities, neglect and poverty. Local newspapers greeted this study as "the beginning of an explanation as to why Scotland's biggest city has the unwanted title of 'the sick man of Europe'" . Furthermore, charities celebrated the research as "'startling evidence' of the impact poverty can have on children before they have even left the womb, and warned that cutbacks to welfare provision would only worsen the damage" (Mclaughlin 2012).

It may be too early to say but, in a near future, it is foreseeable that epigenetic findings will become increasingly relevant in social policy strategies, and are likely to be positioned at the crossroads of three axes: (1) first, the use of epigenetic findings to offer an ultimate bastion of *biological evidence* for social deprivations and inequalities (Miller 2010) and influence specific political agendas (in this reproducing the impact and allure of fMRI studies in social policy in the last decade: Wastell and White 2012). (2) Second, to the extent that in epigenetic research social adversities, class inequalities and other soci-

etal factors operate *through the modification of biological endowments*, the deep-seated distinction between natural and social inequalities that has structured much political science as well as much policy work will become so visibly blurred so as to be open for a potentially thorough reframing (Loi et al. 2013). To catch this intellectual novelty a new hybrid terminology, beyond the nature/nurture divide (Singh 2012; Nature Editorial 2012), has already started to appear, from notions of "metabolic ghetto" and "maternal capital" (Wells 2010) to "molecular biology of the social position" (Niewöhner 2011). (3) Third, epigenetics may drive the emergence of a discourse that identifies, at the local level, subgroups/subpopulations with different epigenetic marks (reflecting for instance their disadvantageous conditions). These potentially vulnerable/risky subpopulations and "permanently undermined" groups may thus become the target of a new epigenetic biopolitics. A possible and updated revival of soft or Lamarckian inheritance in social policy discourses, in which local contexts decisively affect the quality of the epigenome and traumas travel intergenerationally to become ingrained within a specific population, should not make us forget that in the past these Lamarckian views of inheritance have become a fertile terrain for intensely racist and eugenic discourses on, for instance, the *irremediable degeneration* of the germ plasm in unfavorable environments (as in the case of the anti-Irish writings of British Lamarckian eugenicist E.W. MacBride, see Bowler 1984). Without implying that this is likely to happen today, social and political scientists need to be aware of the complex and often subtle nature of the implications that different notions of biological heredity may have when transferred to policy contexts.

Pathway 3: Paradoxes of Somatic Materialism

In the last two decades, the expansive success of the life sciences, from neuroscience to epigenetics, has extended its reach over much of what had been once reserved to the perimeter of "nurture", the vaguely defined but prestigious space where social and cultural influences were sovereign. Nurture has become today increasingly subject to the techniques of measurement, digitization and storage that are part of that molecularization of environmental and societal factors that is foundational to epigenetics' intellectual program. It is as digital representations of the environmental, social or biographical aspects of "nurture" that epigenomic profiles enable the molecular, and at times also experimentally tractable, understanding of living beings. Yet, while molecularization has already sparked an important debate in the social sciences over the last decade (Shostak 2005; Beck and Niewöhner 2006; Rose

2007; Nowotny and Testa 2011), epigenetics seems to herald a new stage that entails "a highly selective scanning of the sociomaterial environment in order to make snippets of it available for experimental work at the molecular level. The sociomaterial environment and increasingly everyday life itself is framed and ordered in terms of its effect on molecular processes in the body" (Landecker 2011; see also Niewöhner 2011).

The same emergence and contemporary diffusion of a term like "exposome",[2] coined by epidemiologist Wild (2012) to define "every exposure to which an individual is subjected from conception to death" although important in rebalancing the focus of medicine toward environmental factors, is symptomatic of a "certain ontological flattening" as Landecker and Panofsky (2013) claim, "by which different categories of things in the world are made equivalent by recasting them as different forms of exposure". The suffix -ome in exposome reflects such a digitization of all forms of environmental exposure, from motherly love to toxins, from food to class inequalities, into a single unifying category and syntax. It is at this level, we agree with Lock (2012), that "epigenetic findings may well set off a new round of somatic reductionism because research is largely confined to the molecular level". It would be inappropriate, however, to read this novel somatic reductionism as the next episode in a genealogically linear saga of the rise of modern scientific reductionism. Things are much more complex and in a way interesting in epigenetic research and the reductionism and materialism that we are witnessing today may be qualitatively very different from the one driven by genetics in the last decade of twentieth century.

Specifically, the paradox on which we want to call the attention here is that, differently from gene-centered twentieth-century biology, it is precisely the current unprecedented deflation and openness to environmental factors of the postgenomic gene, with the subsequent collapse of the nature/nurture border, to produce this new stage of materialism and somatic reductionism with its singular profile. Here, there is a two-way movement that is worth exploring in detail.

On the one side, the more scientists explore the molecular meanderings of the genome, the more they meet "the many ties that link the individual body and its molecules to the spatio-temporal contexts within which it dwells", as Niewöhner (2011) has aptly commented. Unsurprisingly, this notion that the line between the biological and the social has been erased to an unprecedented extent has been greeted in the social science and humanities. Representative of this attitude is an important recent article by Guthman and Mansfield that celebrates *environmental epigenetics* as fundamentally undermining "the boundaries [that are] often taken for granted between what is internal and

what is external to the body, between nature and nurture, and between time and space". "There is nothing about the body that forms a solid boundary—or threshold—between it and the external environment" they claim and "this interchange of environmental and bodily molecules suggests a transformation in what we mean by 'nature' and 'nurture' such that the lines between them are being erased" (Guthman and Mansfield 2013, 12–14). This extreme openness of the epigenetic body to the world's signals is certainly a major shift away from the mainstream lesson of twentieth-century biology. The most common view of the body in twentieth-century biology was derived from a Weismannian understanding of it as "a causal dead end" (Griesemer 2002) that saw causation going unidirectionally from genotype to phenotype (otherwise referred to as the hard distinction between soma and germ line). The Weismannian body found an isomorphic reconstruction in Crick's (1958) central dogma of molecular biology, which "states that once 'information' has passed into protein it cannot get out again". This made the body (i.e. the phenotypic level) a mere passive receiver of genetic information via the protein chain (or a "vehicle" of the genes, as in Dawkins' (1976) later speculations that followed the same tradition). Much more in the spirit of ecological traditions, or (if one is allowed) of the early twentieth-century phenomenological notion of the body as embedded in its vital contexts, the epigenetic body brings the Weismannian body to an end.

On the other side, however, epigenetics' materialization of novel links between the genetic and the social, its making the body porous and permeable to the world *is exactly the channel* by which the capture of the body in molecular terms is made possible. The openness of the genome to the social is thus always on the verge of collapsing the social onto a mere source of differential genetic expression. This dialectic within postgenomic research is implicitly recognized by philosophers of biology, Griffiths and Stotz (2013) (two unambiguous critics of reductionism) when they write how, in the current postgenomic and epigenetic landscape, the study of nurture is becoming "potentially as 'reductionist'—that is to say, mechanistic—as research in any other areas of the molecular biosciences" (p. 5). What we want to emphasize here is the fact that, again with Griffiths and Stotz (2013), "a more epigenetic understanding of nature" goes together with "a more mechanistic understanding of nurture" and both these phenomena are a direct consequence of the fact that genes are today postgenomically defined "by their broader context" (p. 228).

This is the reason why we do not believe that in this context it would make sense simply to read epigenetics as a climax of the themes of twentieth-century genetics. It is possible as Sarah Richardson claims that in epigenetic research *genes remain very much at the center* and very likely, as Richardson and Lock

have argued, that a novel wave of reductionism is very much an effect of contemporary epigenetics, but the epistemic sources of this reductionism are *very different from* those of late twentieth-century reductionism. Whereas in late twentieth-century gene-centrism, from sociobiology onwards, we found an increasing attempt to expand the reach of nature into the field of nurture, here the somatic reductionism of epigenetics is the effect of an opposite epistemic claim: that neither nature nor nurture makes sense anymore, and everything is part of an integrated and blurred nature–nurture ontogenetic system (Meloni 2013, 2014). The same notion of mechanism employed by Griffiths and Stotz has to be understood, following Bechtel (2008), in an integrative, *quasi*-holistic way as something that "recognizes the importance of the organization in which the parts are embedded and the context in which the whole mechanism is functioning" (2008, 21).

Similar paradoxes appear when dealing with epigenetics as "the agent of resolution" (Keller 2010) of the nature/nurture debate. On the one side, molecular epigenetics is certainly a welcome challenge to the biologically untenable dualism of nature and nurture (Meaney 2001a, 2010). In Galton's own terminology, the opposition of nature and nurture was supposed to distinguish "what one brings into the world at birth" versus "influences that act after birth" (Logan and Johnston 2007). It is clear just from this simple definition how fallacious this dualism appears today, when we know, for instance, how several forms of prenatal exposures have a profound impact on phenotypes in adult life.

On the other side, however, if epigenetics certainly undermines the naïve separation of nature and nurture, at the same time, in breaking this fragile boundary around which much of the twentieth-century episteme of the social and human sciences was constructed, it brings to light an entire new set of conceptual problems. To see this more clearly, we need to contextualize epigenetic research in a broader transition in the life sciences that increasingly incorporates the space of culture into an evolutionary framework.

The collapse of the boundaries between the cultural and the biological was strictly avoided in a post-Weismannian division of labor between the "nature fortress" and the "nurture fortress", but is instead very much part of new intellectual trends in biology, from Developmental System Theory to Niche Construction that have extended biological inheritance so much to include extragenetic resources such as culture or the symbolic system. In these trends, culture is not a biological adaptation in neo-Darwinian sense such as evolutionary psychology, or a meme to be studied on the fashion of a (narrowly defined) gene-centrism, but something that is taken much more seriously, as one of the four dimensions of evolution, itself structured as an inheritance

system (Jablonka and Lamb 2005). We sympathize with this theoretical approach but would just like to recall that precisely in response to this integrative neo-Lamarckian language the social sciences reacted in early twentieth century and constructed their autonomous episteme based on a hard separation of biology and culture (Stocking 1968; Kroeber 1917). The novel epigenetic language of extended extragenetic inheritance is likely therefore to be as provocative for neo-Darwinism as it will be for the social sciences and the humanities (Meloni 2014).

Conclusions

That epigenetics heralds a revolution, what we alluded to in the title echoing a recent popular book (Carey 2012), has become such a tacitly accepted notion that it has escaped scrutiny almost entirely. Here we set out to scrutinize the key claims harnessed in support of this revolutionary narrative, in scientific and lay discourses alike, starting from a brief historical and epistemological reappraisal of the various strands of epigenetic thinking, often productively blurred in their distinctions or at times frankly competing with each other.

More than the hyped upheaval promised by popular literature, what emerges from our analysis is more akin to what Italian political theorist Antonio Gramsci (see Gramsci and Forgacs 1988) famously referred to in the 1930s as "passive revolution". According to his definition, a revolution is passive when, far from being a radical break, it unfolds as a long-term process in which progressive and backward-looking forces coexist and overlap. It is passive (as in the case of the Italian Risorgimento) because it does not have the strength for (or may not even aim at) changing "the essential" and ends up thereby proceeding in a sort of limping way. And yet, despite an uncertain route in which vocal gestures end up often void or usher into bombastic but sterile statements, its impact can nonetheless prove revolutionary.

Without overdoing the analogy between political theory and science, we think that the Gramscian framework captures well the ways in which the ambition of molecular epigenetics innovates the current discourse on life while remaining loyal to the molecular gaze that has made it so productive and hence prominent in our society. In a nutshell, we have argued that that ambition is to tie the regulation of the genome to the digitization of the environment, bringing into relief the temporal dimension that this link invites (including its most far-reaching transgenerational instances). We have then proceeded to analyze how the pursuit of this ambition exposes the most salient lines of tensions of molecular epigenetics (from the epistemic to the normative), opening entry

points for a sociologically minded scrutiny for which we have proposed three paths of inquiry that will hopefully help structure an early engagement of social scientists with this still-emergent field of the life sciences.

Notes

1. As Richardson (2016) acutely notes in three of the most classic experimental studies of epigenetic mechanisms (agouti gene in mice; season's influence in voles; licking/grooming in rats) the epigenetic modification is always introduced via the behavior or physiology of the mother.
2. As a National Institute of Environmental Health Science document (http://www.niehs.nih.gov/about/strategicplan/visionary-ideas/health-status/index.cfm) explains the notion of the exposome "replaces the chemical-by-chemical approach to finding causes of disease and includes endogenous and exogenous exposures". The emphasis on this new concept is evident from the following lines: "Characterizing the human exposome represents a challenge similar to the HGP, which began when DNA sequencing was in its infancy". See also, The Human Exposome Project at humanexposomeproject.com/. Two major grants on the exposome have been awarded by the EU in 2012.

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10

Social and Behavioral Epigenetics: Evolving Perspectives on Nature-Nurture Interplay, Plasticity, and Inheritance

Frances A. Champagne

Introduction

Though there has been long-standing division between considerations of the role of nature vs. nurture in determining the origins of variation in personality, behavior, health, and well-being, this traditional view has been revised in light of demonstrated gene-environment interactions (GxE) and their influence on these outcomes. A classic example of this interaction is in the prediction of depression based on stress and genotype: individuals with a specific polymorphism within the gene encoding the serotonin transporter (SLC6A4) are at higher risk for depression *only* when they have experienced elevated lifetime stress (Caspi et al. 2003). Under conditions of low stress, no effects of genotype are observed. Thus, the impact of genes/nature on the traits of an individual is tempered by the environmental experiences of that individual. This shift in understanding of nature and nurture has important implications for how we think about genes and their influences. In particular, gene-environment interactions provide evidence of plasticity and an ability to overcome the constraints of genetic determinism. However, the occurrence of a gene-environment interaction is derived primarily from statistical relationships—the presence of a statistical interaction between genotype and environmental exposure. These interactions suggest a phenomenon but do not provide a mechanism.

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In the past decade there has been a rapidly growing literature focused on the biological mechanisms through which interactions between genes and the environment occur (Champagne 2012; Meaney 2010). At the core of these mechanistic studies is epigenetics. The term "epigenetics" was coined by Conrad Waddington, a developmental biologist, in the 1940s to refer to the interplay between genes and their products that account for variation in phenotype. From this perspective, genes were viewed as being "organized" or "induced" in their activity with resulting consequences for development (Waddington 1940). By the 1980s, biologists had identified possible molecular processes to account for variation in gene regulation through studies of DNA methylation (Razin and Riggs 1980). DNA methylation is the chemical modification of a cytosine within the DNA sequence, resulting in 5-methylcytosine (Culp et al. 1970). Early studies of DNA methylation indicated that the activity of genes can be altered in this way and that this alteration is fundamental to driving diversity of phenotype—albeit at a cellular/molecular level accounting for cellular differentiation (Jones et al. 1983). However, the notion that these molecular epigenetic mechanisms could be modified by the environment to account for the phenomenon of nature-nurture interactions has only been the focus of epigenetic research in the past decade (Weaver et al. 2004; Dolinoy et al. 2006).

The field of social and behavioral epigenetics explores the relationship between the quality of the social environment, epigenetic variation, and behavioral variation and is part of the broader study of how environments (i.e. nutritional, toxicological, social) come to induce phenotypic variation at the level of the organism (i.e. growth, metabolism, health, behavior) via epigenetic mechanisms. Though the initial studies linking social experiences to epigenetic changes in the brain with consequences for behavior were conducted in model organisms, such as rats, there is growing support for the relevance of these mechanisms for humans. Both individual-level social experiences, such as psychosocial stress (Monk et al. 2016), trauma (Yehuda et al. 2014), and exposure to adverse parent-offspring interactions (McGowan et al. 2009), and group-level experiences, such as poverty (Lam et al. 2012) and racial discrimination (Brody et al. 2016a), may exert lasting biological influences through epigenetic variation. Epigenetic studies illustrate the integration of biology and the social world in unprecedented ways by demonstrating the direct effect on DNA function of the social environment. Moreover, there is increasing focus on the transmission of environmentally induced molecular changes across generations. This multigenerational perspective has forced a reconsideration of the narrowness with which we view the biology of inheritance (Danchin et al. 2011) and suggests a broader and more dynamic process of

evolution (Laland et al. 2015). Given the scientific revolution that this body of work has triggered, social and behavioral epigenetics raises many issues of societal relevance, including the biology of social adversity, the relationship between DNA and identity, and intervention as a strategy to target epigenetic plasticity (Brody et al. 2016a, b; Swartz et al. 2016). This chapter will highlight studies within the field of social and behavioral epigenetics, discuss the changing scientific and societal views contributed to by these studies, and speculate about the future implications of this field of study for our evolving understanding of the gene, individuals, and society.

A Primer of Modern Epigenetics

Advances in the methodological tools available to interrogate biology at a molecular level have enabled rapid scientific discovery within the field of epigenetics. In particular, these advances have revealed the dynamic process of gene regulation—involving multiple types of epigenetic modifications occurring within a temporal-spatial context. DNA methylation is perhaps the most fully explored modification of cytosines within the DNA sequence (Razin and Riggs 1980). The addition of a methyl-group to cytosines within DNA is generally an epigenetic mechanism of gene silencing when occurring within the promoter—the regulatory region of a gene (Razin 1998). This chemical modification of DNA does not alter the DNA sequence. The gene silencing occurring as a consequence of DNA methylation is contributed to by the accumulation of methyl-binding proteins within the methylated genomic region which serves to limit accessibility to the DNA (Fan and Hutnick 2005). DNA methylation patterns are mitotically heritable such that when cells divide they transmit this pattern to daughter cells (Jones et al. 1983). This transmission process is critical to the phenomenon of cellular differentiation, where all cells descend from an omnipotent stem cell that generates more lineage-specific cell types.

In addition to direct chemical modifications to DNA, there are two other main classes of epigenetic mechanisms: post-translational histone modifications and non-coding RNA. Within the cell nucleus, DNA is physically wrapped around a cluster of proteins called histones (e.g. H3, H4). Histone proteins can, like DNA, be modified through the addition of a variety of chemicals, leading, for example, to acetylation, methylation, and ubiquitination (Cheung et al. 2000). Histone chemical modifications serve to either create a more densely packed chromatin structure associated with gene silencing or loosen interactions between DNA and histones to promote gene

activation. The type of chemical added, the location within the histone where the chemical has been added, and the genomic location where the modified histone interacts with the DNA are collectively predictive of the impact of post-translational histone modifications on gene expression (Jenuwein and Allis 2001). Finally, there is increasing understanding of the role of non-coding RNAs—RNA that does not produce a protein product—in gene regulation (Sato et al. 2011). The product of "junk DNA" (Ohno 1972), non-coding RNA molecules can alter the function of a gene by interacting with proteins and mRNA produced from coding regions of the genome (i.e. genes) and may also interact directly with DNA. The function of non-coding RNA molecules in gene regulation has challenged the way in which we define "functional" with regard to the genome—producing a protein may be one of many functions that a genome can have (Tragante et al. 2014; Graur et al. 2015).

Overall, though epigenetics is often described as a molecular "on/off" switch, the complexity of these biological processes is immense. Each of type of epigenetic modification operates within a genomic context and has spatial and temporal features that contribute to their predicted effects. Beyond that initial complexity, there is interaction between different types of epigenetic modifications in the prediction of gene expression (Molina-Serrano et al. 2013). Thus, increasing understanding of epigenetics reveals how highly complex, multilayered, and contextually sensitive these biological mechanisms are, as a first step in the process of generating phenotype from genotype. Though developing simple analogies to communicate the basic principle of epigenetics is important for transmitting emerging scientific ideas, the complexity involved in epigenetics should not be lost. Organisms are complex and epigenetics builds an infinitely complex and dynamic layer of biological information within the genome.

Mothering the Epigenome

The role of epigenetics in gene regulation and cellular differentiation has been accepted for decades; however, a relatively novel concept to emerge is that these mechanisms can be shaped or "induced" by the environment. Certainly, the cellular environment is important in setting epigenetic state of DNA as it is through cell-signaling and cell-cell interactions that cellular differentiation occurs. However, the question that has moved the study of epigenetics into the realm of the social world is whether the experiences of an individual can shape epigenetic variation within the genome (see Fig. 10.1). Theoretical discussions regarding epigenetic plasticity have existed within the literature for

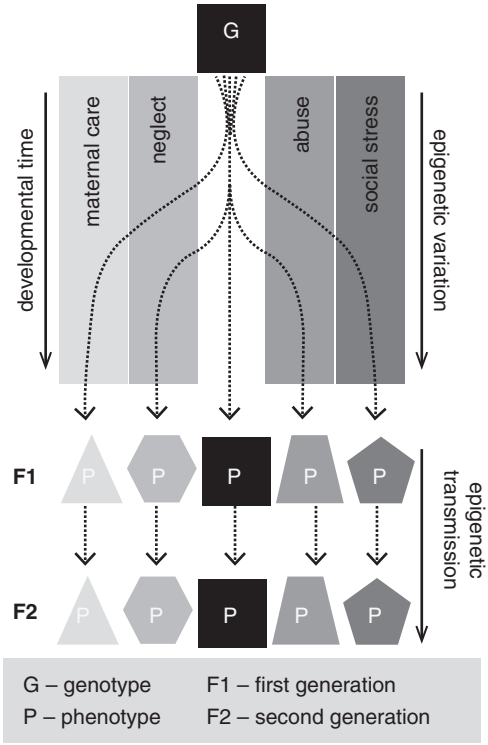

Fig. 10.1 Illustration of the complex interplay between genotype and social environment in predicting phenotype within and across generations. Epigenetic variation is a mechanism through which divergent phenotypes can arise through interactions of genotype with different environmental conditions across development. This epigenetic variation can be transmitted across generations leading to the inheritance of phenotypic variation

decades—such as the idea that memories might be encoded or "ticketed" within DNA through cytosine modifications (Griffith and Mahler 1969). However, a theoretical stumbling block to a wider appreciation of epigenetic plasticity was present in the hypothesized role of stable epigenetic patterns in defining cell types. How can a mechanism confer both stability of phenotype (i.e. maintenance of a muscle cell type vs. a neuron) and plasticity in response to a lifetime of environmental signals? Though the solution to this dilemma has yet to be elucidated, evidence of epigenetic plasticity and stability exists and is demonstrated by the impact of early life mother-infant interactions.

Mammalian development is characterized by a high level of investment in the care of offspring from the prenatal period through to young adulthood. Though biparental care is present in some species, including humans, mothers are the primary caregivers in most reproductive contexts and invest significant

energetic resources through placentation, lactation, and offspring-directed behaviors (Fowden and Moore 2012; Jenkins et al. 2016). Maternal reproductive investment is essential to offspring growth and development. However, there is significant within-species variation in maternal behavior (Hane et al. 2010; Maestripieri et al. 1997; Champagne et al. 2003). Studies of natural variations in maternal behavior reveal the critical role of mothers in shaping epigenetic outcomes. Offspring of female laboratory rats that engage in low vs. high levels of postpartum maternal licking/grooming (LG) during the first week of life differ significantly on physiological, neurobiological, and behavioral outcomes (Meaney 2001). These effects persist into adulthood. Adult offspring that have experienced low levels of LG during infancy have heightened stress reactivity, behavioral inhibition within novel environments, increased aggressiveness in social interactions, impaired learning/memory capacity, and altered reproductive behavior (Meaney 2001; Cameron et al. 2005). These functional outcomes are associated with altered gene expression within specific neural systems associated with the hypothalamic-pituitary-adrenal (HPA) response to stress, fear, cognition, and maternal/sexual behavior. What is particularly notable about the observed association between early life mother-infant interactions and gene expression is its persistence. The activity of genes within the brain is stably altered by the quality of the social environment occurring early in development. Analyses of DNA methylation levels and post-translational histone modifications within the brain of offspring that differ in their experience of postnatal maternal care reveal the role of maternal behavior in shaping these epigenetic mechanisms (Weaver et al. 2004; Suderman et al. 2012; McGowan et al. 2011). The epigenetic effects of maternal care occur at a broad range of genomic locations, including specific gene promoters involved in stress reactivity. The regulatory region of the gene encoding the glucocorticoid receptor (NR3C1) is hypermethylated in the hippocampus of offspring of low-LG compared to high-LG mothers (Weaver et al. 2004). In concert with increased DNA methylation are decreased levels of histone acetylation which collectively accounts for the decreased NR3C1 gene expression and protein observed in the hippocampus of low-LG offspring (Liu et al. 1997; Francis et al. 1999). The result of this gene regulatory state is to reduce the capacity of low-LG offspring to adapt to stress.

The determination of an epigenetic consequence of maternal behavior has been the launching point for studies of social and behavioral epigenetics. Moreover, further exploration of the dynamics of epigenetic change within the NR3C1 gene has revealed important principles of environmental interplay within the genome. First, epigenetic variation emerges in response to the cues in the environment. At birth, there are no epigenetic differences in DNA

methylation of NR3C1 in the hippocampus of low- compared to high-LG rat offspring (Weaver et al. 2004). After several days of differential maternal care, group differences in DNA methylation are observed and persist into adulthood. Second, though the epigenetic effects of maternal care persist into adulthood, there is continued epigenetic plasticity in the adult brain whereby NR3C1 gene activity can be "reset" resulting in a shift in the stress reactivity of offspring. Pharmacological manipulations in adulthood that decrease DNA methylation or increase histone acetylation can be used to shift the phenotype of a low-LG rat toward that of a high-LG rat, and the converse can be achieved by increasing DNA methylation (Weaver et al. 2004; Weaver et al. 2005). Thus, reversibility of both epigenetic variation and the phenotype associated with this variation is possible, even when stability has been maintained throughout infancy and adolescence. Finally, studies exploring the link between maternal behavior and NR3C1 DNA methylation have revealed the cascade of sensory, neural circuit, hormonal, and transcriptional events that link this particular aspect of the social environment to a change in DNA methylation (Hellstrom et al. 2012). Somatosensory stimulation features prominently in this cascade as a way through which an organism senses the quality of caregiving (Ferber et al. 2008; Hellstrom et al. 2012).

Though natural variations in maternal behavior have served as the starting point for studies examining epigenetic interplay with the social environment, subsequent studies have examined a broad range of "nurture" cues, including the experience of abuse and neglect. In rodents, disruptions to the postnatal environment result in an increased incidence of abusive caregiving, resulting in altered DNA methylation, histone acetylation, and gene expression within the brain of offspring (Roth et al. 2009; Blaze et al. 2015; Doherty et al. 2016). In humans, a history of childhood abuse is predictive of increased hippocampal DNA methylation within the NR3C1 gene and similar overall patterns of epigenetic variation to what has been observed in the rodent model comparing low- and high-LG offspring (Suderman et al. 2012). Global increases in DNA methylation have been observed in blood samples from institution-reared orphans (Naumova et al. 2012) and analyses of buccal cells indicate hypomethylation in the SLC6A4 gene as a function of increased exposure to institutional care (Non et al. 2016). The ability to detect epigenetic signatures of early life adversity in tissues outside the brain is an important methodological step in translating laboratory-based findings into the real-world analyses of human biobehavioral processes and to field studies of animals exposed to ecological pressures meaningful in discussions of fitness and evolution. Though there is ongoing debate about the relevance of these "peripheral" epigenetic changes in understanding the brain and behavior,

there is increasing evidence of epigenetic concordance across different tissue types in response to environmental cues (Nemoda et al. 2015; Farré et al. 2015; Kundakovic et al. 2015).

Evidence for the profound impact of maternal care on offspring development that extends to epigenetic outcomes has placed increased emphasis on the development of parenting interventions. Despite a recognized need to provide additional support and education to parents (Shuman and Masterpasqua 1981), these interventions have not typically been implemented at a global or national level. However, family-based programs that focus on developing attachment security, managing stress, and treating parental and child psychiatric illness have promise in reducing mental illness and improving child and parent well-being (Cicchetti et al. 2006; Lowell et al. 2011). Though parental neglect or abuse can exert significant "wear and tear" on the biology and behavior of children, it may be possible to shift developmental trajectories through intervention. Moreover, this plasticity may manifest at the level of epigenetic variation. One epigenetic metric that delves into the biological "wear and tear" experienced by an individual is referred to as "epigenetic age" (Horvath 2013). Analyses of DNA methylation from virtually any cell in the body can give an approximate estimate of our chronological age. Thus, our cells have a memory of time. However, in some cases, the epigenetic estimate of chronological age suggests we may be biologically "older" than our chronological age. This phenomenon is referred to as "age acceleration" and is thought to reflect a process of "wear and tear" (Horvath 2013). Epigenetic age acceleration has been observed in response to disease (Horvath and Levine 2015), prenatal adversity (Simpkin et al. 2016), and exposure to parental depression (Brody et al. 2016b). Within intervention studies, programs that reduce harsh parenting can reduce epigenetic age acceleration with potential for improved physical and mental health outcomes (Brody et al. 2016b). Intervention studies have significant potential to "reset" epigenetic outcomes. However, it is important within the context of intervention studies to not lose sight of the cascade of events within the social environment that influence parent-offspring interactions. Studies of maternal behavior in laboratory rodents and in primates provide empirical support for the influence of social stress and social support on the quality of mother-infant interactions (Ruppenthal et al. 1976; Curley et al. 2009; Champagne and Meaney 2007; Champagne and Meaney 2006). Similarly, human parenting occurs within a broader context of socioeconomic pressures, family dynamics, community well-being, and exposures to nutritional and toxicological factors that may alter reproductive systems and stress physiology. Integrating context into the discourse of the impact of mother-infant interactions on the epigenome will

be particularly important for identifying the distal predictors of parenting, identifying society/community level targets for intervention, and lessening the "blame the mother" sentiment that may arise from the focus on the more proximal influences of child development (Winett et al. 2016).

Psychosocial Stress and Epigenetic Plasticity

Stress is a highly conserved process of coordinating the biology of an organism in response to threat. Psychosocial stress and mood during pregnancy can have a lasting impact on offspring development with consequences for psychiatric risk (Koubovec et al. 2005; Weinstock 2008). These psychological states are associated with heightened HPA activation, resulting in increased glucocorticoid levels within the mother—a classic physiological response to threat (Kane et al. 2014; O'Connor et al. 2014). In humans, objective stress exposure, maternal perceived stress, anxiety, and depression can epigenetically alter offspring via three distinct yet interactive routes (Monk et al. 2012). The first pathway is through epigenetic variation within the placenta. During pregnancy, the placenta acts as a critical interface between the mother and the fetus (Burton and Jauniaux 2015). Gene expression and epigenetic profiles within the placenta change during the course of pregnancy (Novakovic et al. 2010; Sitras et al. 2012), and variation in these profiles is predictive of fetal growth restriction (Jensen et al. 2014; Roifman et al. 2016). Among mothers that report elevated perceived stress during pregnancy, there is increased placental DNA methylation within the 11HSD2B gene—a gene encoding an enzyme that buffers the fetus from maternal stress hormone (Monk et al. 2016). Moreover, increased 11HSD2B DNA methylation within the placenta is predictive of impaired neurodevelopment in the fetus. Variation in 11HSD2B DNA methylation is also observed as a consequence of socioeconomic status (SES)—though this association suggests decreased DNA methylation of 11HSD2B in response to stress (Appleton et al. 2013). Epigenetic variation in several other placental gene targets is predictive of stress responsivity, self-regulation, and sensory development (Paquette et al. 2014; Conradt et al. 2015). A second route of prenatal epigenetic influence is the direct impact of maternal psychosocial stress on fetal tissues—including the brain. In humans, analyses of epigenetic effects in offspring who have experienced prenatal stress have primarily relied on blood, buccal cells, or saliva. Altered DNA methylation within stress-related genes such as NR3C1 and neural plasticity-related genes such as brain-derived neurotrophic factor (BDNF) has been detected in these tissues associated with prenatal exposure to maternal

stress (Radtke et al. 2011; Hompes et al. 2013; Braithwaite et al. 2015; Unternaehrer et al. 2016). Moreover, studies in laboratory rodents provide experimental support for the presence of these epigenetic effects within the brain (Mueller and Bale 2008; Peña et al. 2012). A third route through which prenatal epigenetic effects may be mediated is via alterations in the quality of postnatal mother-infant interactions. Stress during pregnancy may alter mental health of the mother during the postpartum period, and there is a heightened risk of impaired mother-infant interactions associated with postpartum depression (Brummelte and Galea 2016; Dollberg et al. 2016). Influence of prenatal stress on the quality of the postnatal environment highlights the interplay between experiences occurring at different developmental time points.

Epigenetic plasticity in response to stress continues during postnatal development and persists into adulthood. The deprivation of maternal care during infancy can be perceived as a threat and activate the HPA response to stress with epigenetic consequences. In laboratory rodents, prolonged postnatal maternal separation, often referred to as early life stress, leads to increased activity of stress-related genes (Murgatroyd et al. 2009; Chen et al. 2012) and epigenetic silencing of genes involved in moderating stress responses (Kember et al. 2012; Kundakovic et al. 2013) within the hypothalamus and hippocampus. Moreover, the effects of maternal separation occurring during infancy can be ameliorated if offspring are placed on a diet that alters DNA methylation in adulthood (Paternain et al. 2016). Histone modifications and non-coding RNA expression are also altered by early life stress. For example, activity of the BDNF gene is decreased by maternal separation, and this effect coincides with decreased histone acetylation within the hippocampus (Seo et al. 2016). Altered expression of microRNA—a small non-coding RNA—is observed in the frontal cortex of offspring exposed to maternal separation (Uchida et al. 2010). Early life stress-associated epigenetic variation may account for increased stress vulnerability in response to subsequent stressors as both behavioral and epigenetic variations are exacerbated when maternal separation is combined with adult chronic stress exposure (Seo et al. 2016). Finally, studies in primates illustrate the integration of environment, genetics, and epigenetics in the study of stress vulnerability. Among rhesus macaques that possess the risk SLC6A4 gene variant, DNA methylation of the SLC6A4 gene rather than SLC6A4 gene sequence predicts heightened effects of maternal separation on behavioral stress reactivity in infants (Kinnally et al. 2010). Putative risk genotypes may thus mediate their effects via altered epigenetic variation, suggesting that the phenotypic effects of genes may be shifted through targeting of the epigenome.

Plasticity is typically a phenomenon associated with being young. However, it is apparent that, despite the potential stability of epigenetic effects of early life experiences, epigenetic plasticity can persist across the life span in response to social stress. In humans, adult trauma exposure is associated with epigenetic age acceleration (Boks et al. 2015), and altered DNA methylation is associated with adult SES (Subramanyam et al. 2013). Studies of SES have typically focused on the link between childhood SES and health outcomes; however, given the plasticity of the epigenome, a lifecourse perspective may be more informative in predicting, for example, indices of biological weathering such as epigenetic age acceleration (Simons et al. 2016). Studies in laboratory rodents indicate that a variety of social stressors in adulthood, including social exclusion (Krause et al. 2015) and exposure to aggressive social interactions (Jung et al. 2015; Kenworthy et al. 2014), can impact the epigenome, and pharmacological targeting of histones may ameliorate the effects of social stress (Covington et al. 2015). Moreover, resilience to stress can be described from an epigenetic perspective. Among adult mice exposed to social stress, there is decreased DNA methylation within the corticotropin-releasing factor (CRF) gene—a key player within the HPA response to stress (Elliott et al. 2010). However, among individual mice that are resilient to social stress (i.e. do not display social avoidance or depressive-like behaviors following social stress exposure), there is no alteration in DNA methylation of CRF—the gene remains epigenetically silent. Overall, increasing evidence for epigenetic plasticity in adulthood suggests that intervention and reversal of both genetic and environmentally mediated effects may be possible long after the sensitive period of early development. Further, it may be possible to "re-open" plasticity beyond classic critical periods occurring prenatally or in childhood, leading to improved biobehavioral functioning (Takesian and Hensch 2013).

Revisiting the Inheritance of Acquired Characteristics

The discovery of DNA canalized the gene-centric view of inheritance. This view was inconsistent with the notion of the inheritance of acquired characteristics that was historically integrated into theories of inheritance (Zirkle 1935) and was developed further by Jean Baptiste Lamarck into a theory of evolution (Lamarck 1809). Lamarck posited that the characteristics of an organism were driven by the "habits of life"—a statement describing the dynamic developmental process whereby environments shape the individual. Lamarck also described a process whereby if the environmental exposures that

are driving the "habits of life" were to be sustained over chronological time and repeated across several generations, the phenotypes that emerged would be passed to descents and preserved by heritability. The notion of the inheritance of acquired characteristics, within the context of Lamarckian theory, rests heavily on the idea that the phenotypic adaptations that emerge within an individual are important for the development and survival of that individual. To lose those adaptations from one generation to the next was to compromise the development and survival of generations to come. As Paul Kammerer, a biologist and proponent of Lamarckian theory, once wrote: "If acquired characteristics cannot be passed on … then no true organic progress is possible. Man lives and suffers in vain. Whatever he might have acquired in the course of his lifetime dies with him. His children and his children's children must ever and again start from the bottom" (Kammerer 1914). However, without mechanistic support, the idea of the inheritance of acquired characteristics failed to flourish and was supplanted by the more rapidly developing ideas within quantitative and subsequently molecular genetics.

Within epigenetics, the inheritance of acquired characteristics has gained new momentum, primarily due to experimental studies illustrating the transmission of environmentally induced phenotypes from one generation to the next (see Fig. 10.1).

Critical examples of epigenetic inheritance come from studies within social and behavioral epigenetics. For example, male mice exposed to social instability (i.e. changing social groups repeatedly to prevent the establishment of stable social groups) are altered in their phenotype—this manipulation leads to increased indices of stress (Saavedra-Rodríguez and Feig 2013). Grand-offspring and great-grand-offspring of stressed males exhibit increased indices of anxiety. This transmission is remarkable given that laboratory male mice have no postnatal contact with their offspring and that the transmission to great-grand-offspring occurs exclusively through the patriline (i.e. via male descendants). Though this transmission does not reveal a biological mechanism, it strongly suggests a germline inheritance of an environmentally induced effect. Analyses of sperm from males exposed to stress in early life or in adulthood indicate epigenetic variation, including altered DNA methylation and increased microRNA expression (Franklin et al. 2010; Gapp et al. 2014; Rodgers et al. 2013). Further, these epigenetic changes are also observed in the offspring of exposed males, and the phenotypes observed in offspring can be generated by manipulating epigenetic variation in the developing embryo (Rodgers et al. 2015; Gapp et al. 2014). Though the issue of how these epigenetic marks survive the epigenetic reprogramming that is occurring post-fertilization remains, there is increasing support for the hypothesis that

epigenetic inheritance is possible and may have adaptive consequences for offspring (Zeybel et al. 2012).

Though developmental studies of social and behavioral epigenetics have focused primarily on mothers, it is notable that studies of epigenetic inheritance focus almost exclusively on fathers. The rational for this parental divide is in the relative role of mothers vs. fathers in mammalian reproduction. While mothers create the context of development during prenatal and postnatal life, the role of fathers is limited to fertilization. Thus, for an epigenetic inheritance to occur via the patriline, it is assumed that the only route possible is via sperm/seminal fluid (Curley et al. 2011). However, mothers are also capable of transmitting traits across generations via epigenetic mechanisms. In contrast to fathers, mothers achieve this transmission through their interactions with offspring (Champagne 2011). For example, variation in maternal LG is transmitted across generations via the matriline, such that offspring and grand-offspring of low-LG mothers also engage in low levels of LG. This transmission occurs in response to the effects of postnatal LG on epigenetic regulation of the estrogen receptor alpha gene (ESR1) within the developing hypothalamus. The experience of low levels of LG results in epigenetic silencing of ESR1, and this effect persists into adulthood, rendering female offspring less sensitive to estrogens and less primed to engage in maternal behavior (Champagne et al. 2006; Peña et al. 2013). As a consequence, the LG phenotype persists in the next generation. Similar cycles have been observed in laboratory rats in response to abusive maternal care mediated through epigenetic regulation of BDNF (Roth et al. 2009). Maternal transmission of epigenetic effects across generations is entirely experience-dependent and can be modified by stress or social support (Champagne and Meaney 2007; Champagne and Meaney 2006) allowing for heightened responsiveness to intervention and changing environmental conditions. Finally, though paternal and maternal inheritance systems are often dissociated—either experimentally or theoretically—it is important to take an integrative perspective when considering how parents can epigenetically influence their offspring. Much like genes and environments, parents interact to produce phenotypic outcomes.

Epigenetics and the Gene

Given changing views of development and inheritance contributed to by advances in the study of epigenetics—how should we think about the gene? Genetics is certainly thriving and is central to many new health initiatives

including the Precision Medicine Initiative in the United States (Goodman et al. 2016) and the global Human Variome Project (Burn and Watson 2016). These initiatives focus on genome-wide sequencing of DNA as a strategy for improved diagnosis and treatment of disease. There is also increased availability of direct-to-consumer genetic sequencing resources aimed at defining the origins of individual traits or characterizing an individual's ancestry (Niemiec and Howard 2016; Phillips 2016). Thus, "identity" is still largely linked to DNA despite growing acceptance of the role of gene regulatory processes in shaping development and inheritance. A significant barrier to a better integration of epigenetics and genetics is likely methodological. DNA is stable and identical across tissues. Epigenetic variation is tissue specific and can vary within and across days. The divergent properties of these two molecular features within our cells create challenges when trying to generate a cohesive predictive model of phenotypic outcomes. Overcoming these challenges will be essential to better understand how knowledge of DNA and knowledge of epigenetic profiles can be better used in the design of interventions and to shape public views on the plasticity vs. stability of our biology in response to the social environment.

Future Directions in Social and Behavioral Epigenetics

Research within the field of social and behavioral epigenetics is rapidly evolving through incorporation of novel methods in the analyses of epigenetic variation and broader application of these analyses to humans. Though DNA is still the primary focus of much of the diagnostic work in the biomedical sciences, within the social and behavioral sciences, there has been more substantial integration of epigenetics. Behavior is complex and dynamic—much like the epigenome—and it is perhaps this complexity that has motivated biological explanations to span beyond the constraints of DNA sequence. Epigenetic variation provides a molecular context to DNA, and there is increasing evidence that the phenomenon of GxE interactions is accounted for by epigenetic mechanisms. One of the many challenges ahead for social and behavioral epigenetics is in the integration of multiple levels of the social environment. The tactile interactions between a human mother and infant that trigger epigenetic effects are the consequence of a cascade of individual- and group-level factors that characterize the environment of families, communities, institutions, and nations. Though animal studies can be used to strip away that context to examine the proximal influences on development,

translating these studies to humans requires a better understanding of the relationship between proximal and distal influences. A second challenge to the field involves the integration of genetics and epigenetics. The goal of studies within the field of epigenetics is not to replace the study of DNA. Rather, the goal is to integrate these molecular factors into a more comprehensive theory of the origins and inheritance of phenotype. This integrative approach will be necessary to avoid perpetuating nature vs. nurture dichotomies and to create a framework for understanding the coexistence of stability and plasticity of phenotypic variation.

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11

Molecular Multicultures

Amy Hinterberger

In the last two decades, the gene, as an object in which heredity was thought to reside, has been decentred, giving way to the language of postgenomics and epigenetics, which emphasise the reactive and interacting molecular, environmental, cultural and social aspects of health and disease. Such understandings of the genome offer new possibilities for the relationship between biology and society. Yet, these new understandings of biological life have arisen alongside the global proliferation of race, ancestry and nationalisms in bioscientific research. National heritage, along with continental heritage, has become reinvigorated territory for postgenomic exploration. There are now projects on the African genome, the Asian genome, the Mexican genome, the Iranian genome, the Indian genome and many others.[1] Some nations in the global south have claimed sovereignty over hereditary materials, including human genetic samples (Schwartz-Marín and Méndez 2012). Such claims to sovereignty 'tether' biological materials and data to both nation-states and continents (Benjamin 2009; Hinterberger and Porter 2015).

At the turn of the century, the promises of genomics were twofold: the biological basis of disease would be unlocked and intervened upon, and the idea that there existed relevant biological differences relating to race would be obliterated (National Human Genome Research Initiative 2000). The subsequent years of post-genomic research have done neither of these things. What has emerged is a highly complex biology of disease that has both undermined and revolutionised previous understandings of human genetics and molecular

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biology. And, rather than a focus on human similarity, it is the study of differences between people and groups that is seen to hold the key for turning genomic science into genomic medicine. Given these paradoxical developments this chapter asks, what has happened to multicultural politics, in light of the molecularisation of biology? I suggest that we might approach this question through the frame of molecular multicultures.

I introduce the term *molecular multicultures* to characterise the emerging cultural politics of heredity in the post-genomic era. Molecular multiculture situates genome science as a multifaceted continuation of, rather than radical departure from the vital colonial legacies that have shaped the concept of heredity in biology. In doing so, it prompts an expansion of the origin stories that undergird expressions of our contemporary predicaments in the post-genomic era: namely, how to approach the undoing of the gene on the one hand and the remaking of human difference on other. Such an expansion means locating heredity in a series of global processes that link the rise in population thinking, statistics and now, 'big data' biology with colonialism, imperialism and racism.

Second, molecular multiculture foregrounds the relationship between the nation and the populations, groups and collectivities, variously delineated, which are central to developing genomics-based biomedicine. Science and technology and biomedicine have become increasingly central to the modern constitution of citizenship and public identity (Epstein 2007; Clarke 2010; Jasanoff 2011). Molecular multiculture extends the focus from the individual towards the ways pre-existing groups and collectivities are increasingly becoming sites of ethical power and knowledge in genome science. The legitimation of group-specific research is often done by invoking the promise of personalised or individualised medicines and cures. In this regard, a consistent interplay between the level of the population or group and the individual or the personal is a fundamental aspect of molecular multicultures.

Heredity and Multiculture in the Post-genomic Era

One significant consensus about genomics has emerged in the twenty-first century. Evelyn Fox Keller (2015, 9) puts it this way: 'the genome is not the organism'. Such a sentiment is also expressed by Russ Altman (2015, ix): 'genome sequencing projects were neither unmitigated successes nor failures, but rather the start of a newly enabled era in which determining the sequence

of four DNA bases was easy, but understanding its role in biological systems is incredibly challenging'. In other words, sequence information alone does not tell us everything we need to know about being a living creature. Reflecting on these developments, Sarah Richardson and Hallam Stevens (2015, 8) explain that in our post-genomic era (or the era after the completion of the human genome project in 2002), long-standing concepts are currently up for grabs, including 'genetic determinism, reductionism, the role of the social and the environmental in human health and disease, and even the notion of the genome itself'.

It is this changing and uncertain post-genomic space which has ushered in a new politics of heredity and multiculture. The molecular scale, as many social scientists have argued, is an increasingly significant site for constituting human identities in the twenty-first century (Duster 2006; Rose 2007; Lock and Nguyen 2010; Whitmarsh and Jones 2010; Wailoo et al. 2012; Nash 2015; Nelson 2016). However, the ways in which the molecular scale is drawn on to negotiate, invoke, affirm or refute forms of human difference is as varied as the study of genomics itself. The relationship between biology and society is mediated differently across time and place. One of the ways that social scientists have begun to deal with this is to extend the study of technoscience and genomics beyond Europe and the United States, to places such as South Africa (De Vries and Pepper 2012; Tamarkin 2014; Foster 2016), Columbia (Schwartz-Marín et al. 2015), Mexico (García-Deister and López-Beltrán 2015), Singapore and Japan (Sun 2016) and Argentina (Adams Smith 2016)—to name a few. Social scientists have also opened up analysis within settler colonial nations where DNA becomes enrolled in contestations over sovereignty and indigenous rights to self-government (Kowal et al. 2013; Tallbear 2013). They have also done this historically, bringing together 'world histories, national styles, and international networks' to examine how the study of living human populations has been conducted historically and differently across time and place (Lindee and Santos 2012).

Taken together, such literature demonstrates how the nation-state continues to hold a powerful position in the biopolitical management of its populations, and this continues with genomic technologies where knowledges of medicine and natural history play a central role in the mediation between populations and publics, and between politics and the life sciences. Represented as both the objects of study (populations) and the deciding subjects (publics), human groups in biomedical and genomic research are increasingly disaggregated through forms of group standardisation along the lines of cultural, ethnic, racial and other forms of difference (Whitmarsh 2011; Fujimura and Rajagopalan 2011). The diverse organisa-

tions and institutions highlighted in this chapter serve as divisive expressions of power which naturalise group standardisations and define the means by which debates can be carried out about who is to be included, in what way and why.

In referring to multiculture, I draw on the distinction made by Stuart Hall between multiculturalism and multiculture (Hall 2000). Multiculturalism refers to the strategies and policies adopted to govern or manage problems of diversity and multiplicity that multicultural societies throw up. It is multiculturalism that is used as a strategy of governing which underpins the political logics of monitoring and sorting populations (Hall 2000, 210). Many of the examples in this chapter fall into this frame of multiculturalism, where nations have explicitly adopted multiculturalism as a formal policy, such as Canada (Hinterberger) and Columbia (Wade 2013), and these are fed into the design and conduct of genome science. However, nations do not need to have formal governing multicultural policies in place. For example, while generally not known for an official policy of multiculturalism, Steven Epstein (2007, 279) has charted how a series of legal reforms in the USA created a new set of meanings about medical research, the result being 'the invention of a sort of biomulticulturalism' that 'went hand in hand with institutional change, reflected in the creation of laws, policies, practices, and state bureaucratic office'.

Epstein describes the institutionalisation in the United States of what he calls a 'biopolitical paradigm' of medical research that considers group categories (such as sex, gender, race and ethnicity) biomedically significant. He traces out how a loose coalition of reformers during the 1980s, driven by the desire to counter the under-representation of women and minorities in medical research, lobbied the government for inclusion in medical research and policies. These desires became official policy in the 1993 National Institutes of Health Revitalisation Act, and in their subsequent adoption by the National Institutes of Health (NIH) and the Food and Drug Administration (FDA) which mandated the enrolment of women and minorities in medical and health-related research. Underpinning the biopolitical paradigm are processes of what Epstein (2007, 107) calls 'categorical alignment', where state administration and the political mobilisation of groups come to provide legitimate scientific taxonomies for biomedical research.

The logics of inclusion that Epstein identifies in clinical medicine have been extended to biomedical and genomics research more broadly. Contemporary large-scale genomics research on human health is not unreflective or unresponsive to concerns about the stigmatisation and discrimination of groups. Rather, these concerns are often consciously and deliberately

countered through a range of inclusionary techniques: incorporating vulnerable groups or stakeholders, public dialogue and engagement as well as community and group consultation strategies. As Jennifer Reardon (2007, 239) notes, 'these days, many in the arena of human genomic variation research require little convincing that the ideas and practices of this domain of research raise complex and vexed questions about how to order and value human beings in society'. In social and political contexts where diversity can be rendered a strategic resource, it is no surprise that large-scale public projects incorporate forms of multicultural inclusion as a way of gaining public legitimacy. After all, the recent successes and failures of large-scale population genomics projects (such as the Human Genome Diversity project) demonstrate that these projects require not only funds but also public faith and conviction.

Molecular multiculture highlights how multiple modes of group-making are present in today's genome science. For example, the incorporation of census classification is increasingly common in the design, conduct and regulation of human genomics research. An example of this can be found in an article recently published in the journal *Public Understanding of Science*. Entitled 'Ethnocultural Community Leaders' Views and Perceptions on Biobanks and Population Specific Genomic Research: A Qualitative Study', the article argues that ethnocultural community members are both sponsors and beneficiaries of biobanks, and that therefore their views and perceptions should be included (Godard et al. 2009). Noting the substantial investments nations, such as Canada, are making in population-based biobanks, and that the success of biobanks relies on 'community support and participation', the article draws on interviews with 'ethnocultural community leaders' about their perspectives on population research (2009, 1). The identification of the groups who made up 'ethnocultural communities' included individuals who self-identified 'with one of nine census populations in the greater Montreal area: Aboriginal, Chinese, Greek, Haitian, Hispanic-Canadian, Indo-Pakistani, Italian, Jewish and Moroccan' (2009, 3). Framing research through 'ethnocultural communities' keeps political logics of multiculturalism intact by viewing culture and ethnicity as something that 'other' groups have and that needs to be brought into the design and conduct of biobanks, not only for ethical reasons but also for their biomedical significance.

Such a moment of molecular multiculture brings into view how, as an approach to the study of human DNA, genome science requires the involvement not only of geneticists and molecular biologists but also of statisticians, computational biologists, project managers and—for many publicly funded projects—ethicists and communications experts. It also requires sophisticated

equipment designed to visualise and represent genetic information at the molecular level, such as sequencers, along with the computational power required to analyse biological information, and the capacity to store and retrieve these large amounts of data.

For genomic researchers, the genome sequences of humans represent foundational information for biology and biomedicine. Over the last 10 to 20 years, advances in molecular biology and genetics have made it faster and cheaper to detect and sequence genetic variation in individuals and human groups. These developments stem from a long history that began with the recognition of DNA as hereditary material, the determination of its structure, the elucidation of the genetic code, and the development of recombinant DNA technologies and ever-faster methods for sequencing DNA. In this regard, the emergence of genomics has diverse and complex intersections with the genetics that preceded it. Barry Barnes and John Dupre usefully emphasise that the central difference between genetics and genomics is one of increasing powers and capacities (2008, 3).

Large-scale genomics projects have to some extent internalised former critiques (such as those of the Human Genome Diversity Project; see M'charek 2005 and Reardon 2012) and have developed sophisticated ethical techniques to address charges of discrimination and stigmatisation. National as well as international bioethical guidelines increasingly stress cultural plurality, community and group diversity as an ethical resource which must be monitored and addressed in research practices. In performing accountability and transparency, large-scale publicly funded genomics projects increasingly draw on discourses of groups and cultures as well as the health benefits they will receive. In such scenarios, forms of difference can become reinscribed at the biological level, resulting in what Duana Fullwiley (2007) has called the molecularisation of race. Alongside these processes, genome science is itself in a state of flux, even undoing its own foundational premises about the workings of hereditary materials such as genes. Our post-genomic moment is thus characterised by a reinscription of molecular difference, along with changes in the concept of heredity.

Heredity Redux: Population and Its Discontents

A focus on heredity highlights how genome science is embedded in much wider social and political preoccupation with reproduction and propagation. The rise of heredity as a biological concept in the nineteenth and twentieth centuries is entangled in the emergence of 'population thinking' and statistics

which developed in conjunction with colonialism, imperialism and racism. Locating heredity within such global processes is integral to understanding how processes of racialisation, for example, the ones that characterised the settlement of the 'New World' in North America, surface in contemporary genome science.

Heredity, however, was not always a biological concept. Its origins are juridical, found in inheritance law, where systems of rules and distinctions regulated how goods were passed on to other persons when another died (Müller-Wille and Rheinberger 2012). It was only around 1800 that the notion of heredity in law worked its way into that of biological reproduction: 'The now dominant biological sense of the term—"heredity" resulted from a metaphorical transfer of a juridical concept to a description of the generation and propagation of living beings' (Müller-Wille and Rheinberger 2012, 5). Descriptions of species propagation thus drew heavily on the forms of classification and rules of distribution found in the juridical roots of the heredity concept.

Evelyn Fox Keller (2010) has demonstrated how heredity was interiorised within the body. Keller argues that it was through the works of Francis Galton (via Charles Darwin) and others that heredity became simultaneously internalised and turned into a substance—and the concept of heredity began to take on its modern meaning. What we have come to know as 'nature versus nature' (as that formulation has been traced through genetics) was to a large degree invented in Anglo-American culture in the late nineteenth century. The effect of this, Keller (2010, 21) argues, was that 'it was not the law, nor civil or church code, nor custom, or theological prescription, but the body that became the vehicle of inheritance'.

The becoming of heredity as biological, and its consequent interiorisation in the body, was also made possible through a series of global processes. Fundamental to heredity becoming a key concept in biology was the global movement of people, plants, animals and ideas: 'The knowledge regime of heredity…started to unfold as people, goods, and the relationships that mediated began to move and change on a global scale' (Müller-Wille and Rheinberger 2012, 3). Feminist and post-colonial approaches have shown how scientific research 'done off-shore, in the European empires and colonies, was central to the development of European sciences and technologies' (Harding 2011, 36–37).

One of the most elusive, yet enduring concepts that accompanied heredity's rise was that of population. The concept of population is simultaneously scientific and political—population is, as Michel Foucault has said, 'power's problem' (2003, 245). Ian Hacking has argued that 'the most famous piece of

biopolitics is the Malthusian debate' (1990, 22). In his essay *Principles of Population* (1888: reproduced in Spiegal 1991), Thomas Malthus problematised population in a manner that left an indelible mark not only on political economy but also biological approaches to population. Malthus, countered Adam Smith's moderate optimism on individual economic self-reliance by arguing that Smith's laissez-faire approach in economic thought would face severe obstacles—the ultimate being the pressure from the population (Spiegel 1991, 266).

According to historians of economic and scientific thought, Charles Darwin developed his ideas about evolution and natural selection through the influence of Malthus (Spiegel 1991, 400). Commenting on Darwin's theory of evolution as it is presented in the *Origin of Species*, Richard Lewontin argues that while no scientist doubts evolution, Darwin's explanation for evolution is certainly open to debate. This is because 'Darwin's whole theory of evolution by natural selection bears an uncanny resemblance to the political economic theory of early capitalism as developed by the Scottish economists' (Lewontin 1993, 10).

Other philosophers and historians of biology argue that Darwin's theories of evolution and natural selection revolutionised thinking on population, leading to a whole new kind of thinking: *population thinking*. Ernst Mayr argues that it was Darwin who unhinged more than 2000 years of Western thought dominated by typological thinking—a kind of metaphysical essentialism based on the assumption that there is an essence (or form) common to all the things within one population or group (Mayr 1970, 1988). Philosopher of biology Elliot Sober extends this idea to argue that Darwin emancipated population from the idea that groups are defined in terms of shared properties (1980, 353). Darwin demonstrated, for example, that one cannot draw a line where one species ends and another begins. As a result, typological thinking, which was rooted in essentialism, 'lost its grip when populations came to be thought of as real' (Sober 1980, 381). Sober goes on to argue that the approach to population founded by Darwin (and extended by many other thinkers and scientists thereafter)[2] can be summarised in the following manner: 'population thinking is essentially statistical thinking' (1980, 350).

Hacking (1990) has shown how in the late nineteenth century it became possible to think of statistical patterns as explanatory in themselves and hence as 'real'. During this time, philosophers, physical scientists, mathematicians and those working in social institutions displaced static ideas of human nature and developed models of 'normal people', such as Quetelet's 'average man' (for Quetelet's 'average man' see Hacking 1990, 104–114; Epstein 2007, 45; Lock and Nguyen 2010, 348). This argument is also developed by Sober, who dem-

onstrates that scientists such as Francis Galton sought to 'transcend the blooming, buzzing confusion of individual variation', and thus worked to develop the population as a unit of organisation, subject to its own forces (1980, 370).

Galton's significant contribution to the development of the autonomy of statistical laws is highlighted by both Hacking and Sober as signalling a significant shift, from the essentialisms of typological thinking to statistical calculations of variation and change. Galton's reputation as a challenger (like Darwin) to more than 2000 years of typological thought rooted in the theories of Plato and Aristotle is overshadowed, however, by his deep commitment to the idea of racial hierarchical types measurable through anthropometry. While Galton may have loosened the grip of essentialism on the idea that species have essences, he also founded one of the most insidious forms of hierarchal racial classification. Both Hacking and Sober concede this point in their writings, with Hacking noting that Galton's 'optimistic anthropometry' has become 'better known for its vices than its virtues' (1990, 180–183), and Sober acknowledging Galton's strong commitment to racial types (1980, 368).

The story of Galton demonstrates that while some forms of population thinking can unhinge essentialisms, others can, in the same moment, be brought to organise human population groups along hierarchal lines, and these movements can happen together with seemingly no contradiction (at least in the mind of statisticians). These paradoxical aspects of the study of population continue to characterise the cultural politics of heredity emerging in molecular multicultures. What we are seeing in the contemporary era are *multiple* biological conceptions of race, which draw variously on forms of statistical and 'population thinking' (see: Morning 2014). Over the last two decades, many nations have developed their capacities for biomedical genomics and in doing so are articulating national populations as unique, special and vital for the development of health and medicine. To this end, recent revolutions in molecular biology and the consequent disaggregation of biology have gone hand in hand with the nationalisation of genome research and the conflation of group identities within the state as biological. While it may be the case that we are no longer able to approach heredity through older forms of genetic determinism, without some consideration of its continuing significance, pressing questions about the proliferation of race and ancestry in the context of genome science cannot be asked at all. As I will show below, human genome science has galvanised new forms of multicultural inclusion that influence democratic politics and the marketplace.

Heredity and Heritage in Context: Governing Populations in the 'New World'

The problem of population requires continued consideration because it is often made invisible in the post-genomic era. For example, proponents of personalised medicine suggest that contemporary genomic research on disease had freed the population as a level of analysis from the spectre of eugenics. The idea here is that a whole new system of genomic medicine based on individual consumer choice (as opposed to population control) and the advancement of public/private research partnerships (as opposed to state-led health interventions) means that population is no longer a problem. Indeed, terms such as personalised or individualised medicine, often used in conjunction with genomic medicine, seem to eschew the very idea of population as a problem, contributing to the representation of population as a relic of old-style medicine and systems of state control. However, human genomics takes population squarely as its object of analysis through its group-specific approach of delineating 'subpopulations' in both ethical and scientific techniques. While it is true that individual genotyping costs continue to fall, the study of the human genome in relation to health and disease is fundamentally directed at population groups and the comparison of data gleaned in multiple ways from the study of such groups. In this regard, contemporary biomedicine links the objects of analysis in genomics research (populations), in sophisticated and complex ways, with the subjects it seeks to intervene upon (individuals and people).[3] This is population politics, and it is at the heart of human genomics research.

In Canada, for example, stories of national heritage and heredity have a close relationship. In previous work, I have shown how governmental strategies and rationales that guided state formation in Canada in the late nineteenth and twentieth century form part of twenty-first-century biomedicine (Hinterberger 2010, 2012). Canada required populations for its aims of state formation and land settlement; however, the wrong kinds of populations were seen as potentially detrimental to the success and settlement of Canada. The concept of population within Canada's borders was at once seen as a source of danger (in terms of filling the nation with less desirable populations of questionable heritage) but also as a source of social and state progress (since populating the land enclosed under the name Canada with the right kinds of populations was central to its success as a legitimate state). This suggests a need to understand how different nations draw on and reject, often simultaneously, aspects of their colonial histories in forming a contemporary approach to health and difference

in the life sciences. Large-scale genome projects on human health require many kinds of populations including sample populations, reference populations and diseased populations, but also populations that can give ethical consent to national investment in genomics technologies.

Despite the undoing of the gene and the decentring of heredity, there continues to be a proliferation of categories of race, ancestry and multiculture in genome science. For example, conventional interpretations of Canada's role in North America draw primarily on its reputation as an exemplar of multicultural governance as well as Canada's provision of public healthcare. Canada's multicultural policy, an approach to governing captured in the words of the Canadian Supreme Court: 'accommodation of difference is the essence of true equality', has been the subject of significant debate both inside and outside the country. Conversely, the rise of genome science in Canada has been narrated through the lens of bioethics with a focus on individual autonomy, consent and privacy. These dominant interpretations of multiculturalism and genomics in Canada have led to the depoliticisation of both state investment in genome science and its attendant social and cultural politics. This has prevented the development of an analytical lens which can provide a far richer genealogy of biopolitical transformation. Indeed, a conventional interpretation would be that the political model of multiculturalism, seemingly so aware of group difference, has somehow ameliorated the thorny and vexed questions raised in biomedical genomics about how to order and value human groups.

Such interpretations are unsatisfactory for several reasons and are being challenged by work that links population genealogies, colonial histories and genetics together in Canada (Leroux 2015). Powerful national myths operate in the public imagination that Canada is free from racism because of its institutionalised multiculturalism. These myths remain intact when multiculturalism is separated from a wider genealogy of settler colonialism which preserves the assumption that state formation in Canada involved placid forms of mutual coalitions, as opposed to more lively, fleshy and often bloody politics. Finally, by keeping multiculturalism within a narrow political genealogy rooted in the liberal political tradition, other domains of politics with which multiculturalism intersects, namely, the life sciences, remain distinct and separate.

Some of the most pressing and significant political issues residing in the genomics of difference in a settler society such as Canada revolve around questions of sovereignty and rights with regard to the indigenous peoples who live in Canada. These issues become acute where processes of inclusion seek to involve previously under-represented groups along cultural lines. Forms of inclusion which stress incorporating diverse cultural perspectives can gloss

over the assertions of nationhood and sovereignty of indigenous peoples, because such claims are at odds with current practices, such as data sharing. A central contention of Aboriginal scholars in Canada is that Aboriginal peoples do not belong to cultures but rather to nations, of which diverse cultural practices and beliefs are a component (Andersen 2014). In this respect, processes of inclusion risk 'culturalising' Aboriginal groups in political processes at the expense of seriously engaging with issues of sovereignty, property, land and treaties. These are some of the significant tensions that characterise the emerging molecular multicultures of the twenty-first century, for example, over questions of ownership, identity and representation that can be easily glossed over in demonstrative acts of cultural competence and consultation.

There is now a growing literature on the nation, race and genome science which explores these challenges in a number of local, national and global contexts (e.g. Tallbear 2013; Wade et al. 2014; Nash 2015; Nelson 2016). These studies provide the foundation for a relational approach that stresses the reproduction of social ties and historical legacies, along with how the colonial has shaped the contemporary. And while the state and nation figure heavily in these approaches, these authors do not take these categories as givens. Such approaches have been instrumental to showing what David Theo Goldberg (2015, 254) argues for in the study of race and racism more generally, namely, that 'ideas and practices emanating from elsewhere are made local; local practices that appear home grown more often than not have a genealogy at least in part not simply limited to the local'. What is distinctive about these accounts is how they foreground the multiple ways in which the molecular scale relates to state policies, multicultural governance and citizenship. For example, M'charek et al. (2014) have showed how in many European countries, the explicit discussion of race as a biological phenomenon has long been avoided in public discourse. The result, they argue, is that race in Europe is best viewed as an 'absent presence, something that oscillates between reality and nonreality, which appears on the surface and then hides underground' (2014, 459). Such findings provide a different way of considering race than those in the United States where Catherine Bliss (2012) has chronicled how race becomes an explicit object of discussion and provides grounds for legitimising research. In the UK context, Ros Williams (2017) has argued that there is no stable scientific conceptualisation of race in the molecular politics of blood cord banking, but rather it is a concept doing different things in different moments: 'it is useful to think of race's meanings and enactments not simply as rearticulating historical and problematic divisions, but as plural and complex in its various invocations and absences'. These different investigations which move across national boundaries illuminate how ideas of heredity and national

belonging, previously anchored in the gene, are being reshaped by forces of culture, governance, biomedicine, technology and markets.

When combined with the promises of genomic medicine, support for large-scale genomics research forms a powerful narrative: by curing the ills of certain populations, genomics research will also cure the ills of an unjust society (Hinterberger 2012). State-funded genome science goes beyond marshalling populations as objects for research, but it is equally invested in creating the kinds of subjects and citizens able to benefit from genomics in the name of the public interest. This rise of public power in the governing of the life sciences is characterised by a fundamental tension between the population and the public. There is a widespread rationale that genomics will provide a kind of individualism and personalised medicine which will transcend old models of state population management in public health. This, however, sits in contrast to the increasing need for populations to be reassembled under the control of the state for the creation and analysis of samples for large-scale genome science. These two goals, while seemingly in contradiction, are immanent to the contemporary political narration of the promises of genome science. Constructions of populations and publics thus animate each other through their polyvalent mobility in the political legitimisation of genome science.

Conclusion: Vital Legacies and the Futures of Genomic Difference

Emerging molecular multicultures, ushered in by the decentring of the primacy of the gene in biology, are a complex continuation of, rather than radical departure from, the vital post-colonial legacies that shaped the concept of heredity in biology. These are vital legacies in both senses of the word—regarding their continuing relevance for analysing the relationships between identity, health, and large-scale genomics—and in the vital properties bodies are seen to possess for the future of genomics-based biomedicine.

Human genomics research has taken on a new urgency and force in public discussion because of its promises for unlocking the biological basis of illness and disease. In this regard, the systems of population classification used in genome science may not only have impacts, at the level of recruitment, on clinical and genetic studies, but they may also have an impact on who gets to count as a legitimate group in the design, conduct and regulation of research. This chapter has shown that specific legacies of classification and the standardisation of difference in, for example, settler colonies like Canada, have

shaped contemporary politics in relation to biomedicine and health research as they are expressed in human genomics research.

While genome science is in a state of flux, even undoing its own foundational premises about the workings of hereditary materials, such as genes, critical analysis requires a more expansive understanding of newly emerging molecular multicultures, or in other words, the cultural politics of heredity. In light of the paradoxical effects of a post-genome world, this chapter contributes towards establishing a knowledge base for understanding the new kinds of social arrangements that have arisen in the place of genomic cures. Studies on human genomic variation are anchored in already established social categories and orders. Thus, rather than transcending the politics of social categories and identities, genome research mobilises many different publics and populations. Such registers of difference extend from national census categories and political doctrines of multiculturalism, to the development of biobanks and the establishment of ethical regulations for specific populations. Along each of these registers, negotiating, invoking and managing group difference is a central aspect to the governance of genome science. In this regard, the emerging molecular multicultures we see across the world, with all their twists and turns, show how group categories and collectivities are an integral site of ethical power and knowledge in contemporary science.

Notes

1. These include the Human Heredity and Health in African project (H3Africa), the GenomeAsia 100 K Initiative, the Mexican Genome Diversity Project, the Iranian Genome Project and the Indian Genome Variation initiative.
2. For example, the concept of population extends far beyond biology to the social sciences. See Osborne and Rose (2008).
3. See Prainsack (2015) for a vivid and erudite discussion of the relationship between the individual and population in relation to the goals of precision medicine.

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12

The First Thousand Days: Epigenetics in the Age of Global Health

Michelle Pentecost

The tagline of the ninth World Congress on the Developmental Origins of Health and Disease in Cape Town, South Africa, was displayed on a large screen in the Cape Town Convention Centre in November 2015: 'Combating the transgenerational risk of non-communicable diseases in transitioning societies'. An eminent South African scientist introduced the proceedings, highlighting that nowhere was this research more relevant than to the local context: 'In terms of Africa and in particular South Africa, we are experiencing transition in a very rapid time'. He explained that the complexity of the South African situation, given the concurrent epidemics of HIV and obesity and the lack of knowledge about the potential epigenetic effects of antiretroviral treatment during pregnancy, was particularly important for DOHaD researchers in this context and others like it. I was seated in the audience in the Centre's plush conference hall, laid out cinema style with burgundy curtains and red velvet seats, one of 600 delegates in attendance. The proceedings continued with a talk on the emergence of the field by one of its most prominent scholars. 'The developmental origins of DOHaD', the speaker quipped. He paid homage to David Barker, founding father of DOHaD, and to the first conference in 1990 in Mumbai. He ended by calling up the DOHaD logo: a foetus nestled in a womb that represents the earth. After pointing out

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that it looked like a breech presentation, he made the somewhat provocative joke that, as usual, 'the brain and the placenta are in the North, and the South can take care of the nether regions'. Africa, he suggested, is 'the omphalos': the source of nourishment.

The global imaginary evoked here—of the belly of Africa as the primordial womb—has long authorised intervention on the continent (Bayart 1993) and most notably for the figure of the mother and child (Vaughan 1991).

I attended the three-day conference as both a clinician with special interests in perinatology and the epidemiology of metabolic disease, and as an anthropologist of science and policy. The conference offered insights into the ongoing formation of the DOHaD research field and confirmed the strong presence of DOHaD research on the South African science front. The DOHaD focus on the global South was evident in the range of large-scale studies presented, taking place in the Gambia, South Africa, Tanzania, Chile, Brazil, India and the West Indies. As the symposium's tagline states, these 'transitioning societies' are the core target of DOHaD interventions. Throughout the conference, speakers and sponsors made reference to 'the first thousand days': a DOHaD-inspired campaign to focus nutrition interventions on the period between conception and the age of two years. Interactive displays in the conference hall, sponsored by Danone and Nestle, showcased large logos: 'What you do in the first thousand days will matter for the rest of your life'.

This chapter is about the first thousand days of life. The 1000 days between conception and a child's second birthday is considered to be a crucial period for determining future health and potential, shaped by knowledge in the fields of DOHaD, neuroscience and epigenetics. As of the late 2000s, the 'first thousand days' slogan has grown into a global movement endorsed by 50 nation-states under the aegis of the World Health Organization and the United Nations and 80 non-governmental organisations (NGO), donor and private sector partners, the largest of which is the Bill & Melinda Gates Foundation. As a large-scale transnational initiative led by a diverse group of public and private actors, the first thousand days project might be placed under the banner of 'global health'. As such, it provides a vehicle to consider how new understandings of health and heredity, as discussed in this volume, produce global imaginaries with material impacts on policy, publics and concepts of life.

Drawing on a case study of the global first thousand days initiative in the South African context, I examine how DOHaD and epigenetic knowledge, as 'biosocial' objects of enquiry, are embedded in global discourses that come to bear on the every day. From July 2014 to September 2015, I tracked the roll-

out of the 'thousand days' intervention in Khayelitsha, South Africa, where this initiative has informed national nutrition policy since 2013. The empirical material in this chapter derives from fieldwork in the policy context where new DOHaD and epigenetic imaginaries animate the formulation of antenatal and nutrition policy; the clinic, where these ideas are translated into practice; and the lives of pregnant women who are the recipients of these interventions. In this chapter, I draw on experiences and conversations with four of my 15 close interlocutors (Lindiwe, Inam, Bathandwa and Nonyameko).

Building on scholarship that queries the implications of DOHaD and epigenetic science for policy and concepts of life and health (Pickersgill et al. 2013; Meloni and Testa 2014; Richardson and Stevens 2015; Meloni 2016), and the emergence of new forms of governance (Merry 2011; Sunder Rajan 2012), this case study goes further to interrogate *epigenetics in the everyday*, tracking the ways in which global notions of risk and potential inflect in the local in uneven ways. I argue that we need to pay careful attention to how new notions of heredity are deployed under the rubric of 'global health', particularly where such interventions have their antecedents in development projects and colonial medicine. What becomes apparent is that the application of DOHaD and epigenetic concepts in global policy and development spheres reflects characteristic features of the global health epoch: economic applicability, humanitarian ethos and anticipation (Lock and Nguyen 2010). Building on postgenomic interrogations that adopt a postcolonial technoscience lens (Hinterberger, this volume; Bolnick and Smith, this volume; see also Anderson 2014), the broader implication of this chapter is that these new scientific discourses not only contain internal tensions that create 'a new sort of radicalised, governable object' (Mansfield and Guthman 2015), but that the ways in which DOHaD and epigenetic imaginaries are harnessed for policy aims across different settings reveal larger disparities about 'who' such policies are directed at, with distinct historical continuities.

The First Thousand Days: Epigenetics and Perinatal Nutrition Policy

The period from conception until the age of two years is recognised globally as the 'window of opportunity' for nutrition interventions to prevent childhood undernutrition and stunting, decrease the risk of adult noncommunicable disease and promote what economists term 'future human capital'. The 'first thousand days' concept arose from the 2008 *Lancet Series on*

Maternal and Child Undernutrition, which suggested that future human capital is best predicted by height for age at two years (Victora et al. 2008). The use of the concept of human capital, which includes adult height, educational achievement, income and offspring's birth weight, expresses the economic logics that underpin the intervention: good nutrition is a 'prerequisite for economic development' (Bryce et al. 2008). The Lancet's 2013 follow-up series, *Maternal and Child Nutrition*, adds that interventions in the early life period also impact on potential future burdens of overnutrition and chronic disease (Black et al. 2013). These recommendations informed the *1000 Days: Change a Life, Change the Future* programme and the United Nations' Scaling Up Nutrition (SUN) initiative and are an explicit focus of the post-2015 development goals. Fifty countries, including South Africa, have joined the SUN initiative.

The 'first thousand days' focus is based on life course epidemiological models that incorporate scientific concepts under the rubrics of *developmental programming theory*, *nutrition transition theory* and *epigenetics*. The notion that interventions in 'the first thousand days' could affect health outcomes in later life, and even in subsequent generations, has its rationale in DOHaD frameworks. DOHaD's foundations lie in the developmental programming work of David Barker and his colleagues (Barker and Osmond 1986; Hales and Barker 1992). DOHaD theory contends that adult obesity, cardiovascular disease and metabolic dysfunction result from a mismatch between early life and adult nutritional environments: 'environmental mismatch' is thought to occur in developing settings that have undergone a 'nutrition transition', defined as a population shift in dietary pattern from relative food scarcity to high availability of energy-dense food as a result of industrialisation (Popkin 1993). While mechanisms remain poorly understood, the emergent epigenetics paradigm has provided DOHaD scientists with potentially substantive methods of measuring correlations between exposure and outcome to add to the evidence for developmental programming (Waterland and Michels 2007). Life course theory's appropriation of epigenetic models thus formalises theories of the effects of early life factors on adult disease within frameworks acceptable to current scientific standards and evidence hierarchies, which privilege measurable components. Twenty-first-century 'epigenetic epidemiology' (Waterland and Michels 2007), incorporating DOHaD and epigenetics, operates in an expanded temporality that includes the potential for the transgenerational transmission of disease risk. DOHaD and epigenetic science underpinning the 'first thousand days' concept thus posits a different relationship between 'exposure' and 'outcome' and a revised definition of 'environment' (Pickersgill et al. 2013; Shostak and Moinester 2015), which is often

equated with the pregnant body (Landecker 2011; Richardson 2015; Warin this volume).

The first thousand days' campaign might be viewed as the latest in a long series of interventions in countries labelled as part of the 'global South'. The project is one example of a number of transnational programmes orchestrated by a diverse set of actors within the remit of 'global health'—the successor of the post-World War II 'international health' movement, which employed a nation-state-centred approach and focused on interventions in countries that have been historically labelled as 'developing' (Escobar 1995). The WHO-driven international health era has been replaced by a diffuse set of actors that include nation-states, NGOs, philanthropists and others, to constitute what Mark Nichter has called a new 'biopolitical project of "empire"', in keeping with previous development critiques (2008, 152). The boundaries and functions of this 'global health system' are difficult to delineate, but cluster around a central imperative to act for the sake of global biosecurity, economic development or 'humanitarian reason' (Lock and Nguyen 2010; Fassin 2012a).

The 1000 days project illustrates both continuities and departures from older ideas shaping public health interventions that find new expression in the era of 'global health'. Although the full history of maternal and child health policies cannot be rehearsed here, there are enduring features that require brief elaboration. The maternal-child dyad has been a prominent site of intervention for public health since the discipline's inception in the late nineteenth century (Kuh and Smith 1993). While the scientific reasoning for this focus has shifted according to socio-political milieu (see Meloni 2016), it is worth noting the historical continuities in the naturalisation of mothers as primary caregivers who should bear responsibility (Wheeler 1985; Baird 2008; Sridhar 2008) and the usefulness of the figuration of the child as a malleable entity whose value is found in its innocence and potentiality (Castañeda 2002). In addition, the linking of early life circumstances with adult disease risk is not new: the first epidemiological studies pointing to these associations were published in the 1930s (Kermack et al. 1934). However, in the same way that epigenetic theories (Waddington 1942) found no foothold in the gene-centrism of the mid-twentieth century (Müller-Wille and Rheinberger 2012), life course approaches to explain adult chronic disease were not favoured by the post-World War II paradigm of chronic disease epidemiology, which focused on new statistical methods that could assess correlations between adult environmental exposures and disease outcomes (Saracci 2007). Developmental programming theory later contested the genetic determinism and risk factor focus of the previous model, and chronic disease epidemiology shifted focus at the end of the twentieth century from lifestyle to life course (Kuh and Smith 1993).

Studying the First Thousand Days: The South African Example

As of 1994, the South African Nutrition Directorate has prioritised maternal and child nutrition, as part of the country's *National Development Plan*. The most recent South African Nutrition and Health Survey showed that the country's nutrition profile is characteristic of that described by life course epidemiologists for developing countries undergoing what they refer to as nutrition transition (Shisana et al. 2013; Vorster et al. 1999). Public health nutrition experts in South Africa thus recommend interventions 'during and even before pregnancy, as well as during the important "window of opportunity" up to around two years of age' (Shisana et al. 2013, 212). The country's Integrated Nutrition Programme was subsequently supplemented by the *Roadmap to Nutrition in South Africa 2013–2017*, which explicitly emphasises 'the first thousand days':

> **Rationale for Nutrition Roadmap**
> There is now a need to focus on priority target groups and interventions that can have the biggest impact, namely in the life-cycle stages before and during pregnancy, and in the first two years of life. Optimal nutrition during this period lays the foundation for a long and healthy life and reduces the risk of developing diet-related chronic diseases. The first 1000 days is therefore internationally recognized as the 'window of opportunity' for direct nutrition interventions.
>
> South African Department of Health (2013a, 15)

As is the case in other countries that have adopted this initiative, this policy is modelled on the UN's *Road Map to Scale Up Nutrition* and is the outcome of collaboration between the Nutrition Directorate of the National Department of Health, UNICEF, WHO regional affiliates and the Global Alliance for Improved Nutrition (GAIN) (DOH 2013, 9). This policy package of interventions comprises a micronutrient and deworming programme, a therapeutic feeding scheme for the treatment of moderate-to-severe child undernutrition and six behaviour change interventions. Of the six behaviour change targets, four specifically target pregnant and lactating women. Two derive directly from the 2008 *Lancet* Series' recommendations: to promote exclusive breastfeeding and to educate on complementary feeds (Bryce et al. 2008). The other two interventions target antenatal nutritional status: educate on 'healthy eating for optimal weight management' during pregnancy and postpartum and offer therapeutic intervention for malnutrition in pregnancy based on body mass index and mid-upper arm circumference measure-

ments (DOH 2013, 19). The authors suggest that this set of interventions targets both the immediate sequelae of maternal undernutrition, such as intrauterine growth restriction and increased risk of infection in infancy, and the potential future outcomes of obesity and chronic disease (ibid, 17). 'The priority target groups' are clear: nutrition policy is focused on pregnant and lactating women and young children, which raises the question of who might be inadvertently overlooked in the policy's formulation. That a focus on these groups might prevent 'diet-related chronic diseases' points to a new DOHaD logic for the prevention of 'non-communicable diseases' (NCDs).

DOHaD and NCDs: Shifting Categories of Disease in South African Policy Discourse

The formalisation of DOHaD as a research field has renewed interest in the mother-child dyad as a key target for public health interventions, on the premise that optimal early life nutrition will not only have immediate benefits but will alleviate the growing burden of NCDs in developing settings. These taxonomies of disease directly influence the framing of population health and disease burden in South Africa. NCDs represent a discrete arm of South Africa's 'quadruple burden of disease'. In a seminar delivered in Stellenbosch in late 2014, the Chief Director of Metro District Health in the Western Cape outlined these four disease burden categories for the province's *Healthcare 2030 Road to Wellness* framework: (1) 'HIV and tuberculosis', (2) 'trauma', (3) 'NCDs' and (4) 'communicable, maternal, perinatal and nutritional conditions'. Of interest is the shift in language in the formulation of 'the quadruple burden'. What have often been previously referred to as 'chronic diseases of lifestyle' (obesity, cardiovascular disease, diabetes mellitus, hypertension) (Steyn et al. 2006) now fall within the 'non-communicable diseases' category. 'Infectious' diseases are now framed as 'communicable'. 'Communicable, maternal, perinatal and nutritional conditions' are considered as a discrete category. While the implicit linkage between infection, nutrition, and maternal and child health has been a long-standing theme in public health, this shift to labels that denote communicability adds a new component to this nexus. In DOHaD frameworks, nutritional maternal and perinatal conditions predict for the potential future risk of NCDs: the notion of communicability expands to include what was previously non-communicable.

I discussed the *Healthcare 2030 Road to Wellness* framework with a researcher on the Western Cape Department of Health's epidemiological surveillance team a few weeks after the Chief Director's presentation. We spoke about the recently published Western Cape Mortality Profile (Groenewald et al. 2014),

which used the classification presented by the Chief Director to categorise cause of mortality in the Western Cape into four 'broad cause' groups: *Group I*—HIV/AIDS and TB; *Other Group I*—communicable, maternal, perinatal and nutritional; *Group II*—NCDs; and *Group III*—injuries. The production of a provincial mortality profile using cause of death data is a useful exercise to present a picture of the province's most pressing health issues and inform funding allocations to address these. However, what constitutes 'nutritional' causes of mortality in this picture is not clear. 'Nutritional conditions' are ill defined in the Western Cape Mortality Profile report and the *Healthcare 2030 Road to Wellness* framework. The epidemiologist explained the grouping of 'nutrition' with communicable disease and maternal and child health on the basis that together these constitute 'diseases of poverty'. The basis on which these mortality rates are grouped together may reflect the new scientific evidence that might link these conditions but might also reflect underlying ideologies of disease classification—in this instance, an implicit judgement of what constitutes 'a disease of poverty'. Just as pregnant women and children constitute 'priority target groups' for the present nutrition policy, the underlying ideology of disease classifications is that it is this group that is afflicted by 'diseases of poverty', which reflects long-standing assumptions in public health about vulnerability (Wheeler 1985; Zarowsky et al. 2013).

As such, shifts in the biological framework for NCDs to incorporate DOHaD and epigenetics recalibrate policy categories and targets with clear outcomes for resource allocation, but these ideas are simultaneously received within pre-existing frameworks. Ultimately the new 'quadruple burden' articulated in the report and the provincial government's 2030 plan reflects shifting categories of disease importance informed by the wider shift to a focus on NCDs, the (re)emergence of maternal and child health as a prominent focus of health policy, and the implicit linkage of the perinatal period with conditions associated with nutrition.

Practical Implications for Provincial Policy

The national reconfiguration of policy priorities filters down to influence healthcare delivery on the ground via provincial mechanisms of policy directives, new protocols, revised funding allocations and staff training. I had a better sense of the actualities of this process after interviewing an official at the Western Cape Department of Health. The policymaker explained that national policy informs the development of provincial protocols across South Africa's nine provinces, which involves all of the district stakeholders who are

involved in implementation. In the Western Cape, both the nutrition directorate and the women's health directorate had been involved in the formulation of the new basic antenatal care protocols, given that the new nutrition policy is largely directed at the antenatal and early postnatal period. 'The women's health, nutrition and child health sub-directorates now sit together around this issue', the official explained to me. 'At our Annual Review meeting, the emphases were on maternal and child health and on non-communicable diseases—a great emphasis was placed on getting it right in the first thousand days, because if we can get the infant off on the right start, we can prepare him for a healthy lifestyle later…The new policy places a lot of emphasis on the importance of maternal nutrition'. He underlined the role of intensive counselling in the new protocol for all women. 'We felt that with normal pregnancy, you know, when a woman is of normal weight, she also needs counseling about maintaining her weight…we feel that obesity is where we should be focusing'. The implicit logic here—that to intensify the focus on obesity requires closer attention to maternal nutrition—reflects a responsibilisation of mothers for obesity rates that has been well documented elsewhere (Maher et al. 2010; Zivkovic et al. 2010; McNaughton 2011; Warin et al. 2012).

The discourse of maternal responsibility and 'the first thousand days' also extended to other government offices, notably the Department of Education. It is important to note that apart from healthcare policy, the 1000 days concept also has huge currency in child development research. In the case of South Africa, this has informed the 2015 '0–4 years' curriculum, which was presented by the Superintendent General of the Western Cape Department of Education at a workshop in Stellenbosch on 'Overcoming Poverty and Inequality' in late 2014. 'There are things you can do that cost no money', she explained, 'like educating the mothers'. A clear link was made between maternal education and the capacity to parent effectively: 'Just keep getting the girls to finish school because they are more likely to look after their children if they are educated'.

The words of the health official and the superintendent for education reveal a logic that traverses the health and education sectors, rooted in maternal responsibility. Policy's naturalisation of mothers as primary responsible caregivers is long-standing (Wheeler 1985), and in the epigenetic era, responsibility is extended to span generations. Mothers become blameworthy targets of a moralising discourse that has special valence in obesity debates (Maher et al. 2010; McNaughton 2011; Warin et al. 2012). In addition, epigenetics has widened the scope of policy attention to include all women capable of conception (Richardson 2015), which resonates with a contemporary focus in development circles on young girls (Adams et al. 2009).

The renewed interest in the 'maternal body' as a key site of intervention in global health policy is thus constituted by a shift to focus on non-communicable disease, a simultaneous formalisation of DOHaD and epigenetics as research fields and a widening of this body to include the preconceptional period. This collective global knowledge now shapes national and provincial nutrition policy in South Africa, of which the key point of implementation is the clinic.

The Clinic as Mediating Site

The clinic is a key 'biosocial border'—the quintessential site where, to borrow from Hannah Landecker, 'social things' are reconstituted as 'biological things' (2016, 81). I spent four months (September to December 2014) in two small clinics in Khayelitsha, during which I interviewed clinic staff members, observed health promotion sessions and recruited 50 pregnant women for the community arm of my study. In documenting the roll-out of the first thousand days campaign in this distinct location, I found that the clinic became a window into the continuities of new policy with pre-existing perinatal protocols. What set the 'thousand days' apart, however, is its explicit link to adult health outcomes for the infant subjects of the intervention. In this section, I consider several ethnographic encounters which illustrate the clinic as a mediating site.

The Clinic as Catalogue

The clinic can be considered a kind of catalogue of the various international strategies directed towards malnutrition. It is a catalogue in the archival sense that its mundane materialities reflect a discursive continuity (Foucault 1972), and in the dynamic sense that, to borrow from Samuel Taylor-Alexander, it is a site of 'adjacent temporalities'. Taylor-Alexander uses this concept to foreground the role of time and temporality in the stabilisation of emerging technoscientific practice (2015). In the clinic, practice is informed unevenly by the selective primary healthcare strategies of the 1980s, approaches during 'the protein era' and the micronutrient focus of later interventions. Thus, the concept of 'the first thousand days' as a new formulation of a long-standing focus on the perinatal period travels alongside older pre-existing frameworks for perinatal care in the clinic, reflected in the everyday interactions of clinic staff and the materiality of the space in which they conduct their work.

The fact that maternal and child health and nutrition has long been a primary healthcare focus in the two clinics where I conducted my study was evident in the visual catalogue of campaigns on the clinic's walls. In the recep-

tion area of one clinic, a large mural depicted a child's first five years of life—on the left a baby on all fours, then a standing toddler and on the far right a small child playing with blocks. A rudimentary clinic card was painted in the top right-hand corner, with the list 'birth, 6 weeks, 10 weeks, 14 weeks, 9 months, 18 months, 5 years'. At the top of the mural was the Xhosa message: '*Abantwana bam basem—pilweni kuba ndibagonyisile ukuba kwiminyaka emihlanu*' ['All of our children are healthy, because they received all of their immunisations until five years']. The mural was painted by local medical students after the clinic's opening in 2005. Other familiar public health messages appeared in posters around the clinic:

> 'Your child needs vitamin A. Take your child to the clinic every 6 months until the age of 5 years.'
>
> 'Introduce solids from 6 months. Fruit and vegetables are important.'
>
> 'Exclusive breastfeeding for 6 months is the best nutrition for your baby.'
>
> 'Adequate nutrition during infancy and early childhood is important in the development of each child's potential.'

A few months after I had arrived, a stack of new booklets appeared in the observations room where pregnant women have their weight and blood pressure recorded. The nurse in charge confirmed that the pamphlets were new and would be dispensed to every pregnant woman attending the clinic. The pocket-sized, peach coloured booklets unfolded into a large poster that could be refolded into a neat square for easy transport. The brochure's title, 'Feeding Smart from the Start', was accompanied by a stylised image of an African woman feeding an infant. One side of the poster explained:

> The first 1000 days of a child's life (from when a woman falls pregnant to when her child turns 2) is a very important time for shaping a child's ability to grow and develop. Pregnant moms should eat a variety of healthy foods that are rich in vitamins and minerals. When moms eat well, so do their babies.

On the reverse of the pamphlet was the byline: 'These messages are brought to you by the South African Department of Health: Nutrition Directorate and GAIN (Global Alliance for Improved Nutrition)'.

In addition to the clinic's official information for mothers on the importance of the early life period, NGOs displayed posters in the clinic space. A notice in the waiting room read as follows:

> *Molweni BooMama*! [Hello mothers!] We are the team from Nonophela Centre and would like to talk to you about your babies…We are taking the first 3 years of life very seriously because it is during this time that the foundation is laid down for the future development of the child…. If you are feeling well then

> your baby will feel well and develop in the way you want it to. Just as you pay attention to your baby's physical needs, like food and immunisation, so you must pay attention to your own and your baby's emotional needs.

The changing visual material landscape of the clinic might thus be viewed as one aspect of the clinic as a dynamic catalogue of interventions in early life, and one way in which new concepts were communicated to the clinic 'clients', as the nursing staff were trained to call them. The catalogue was also evident in staff techniques and practices and in their language and comportment.

Health promotion sessions, for example, provided a forum for discussing the first thousand days. I sat in on the weekly breastfeeding support group. These sessions took place in a tiny office, which was a tight squeeze for mothers, infants, baby bags, a nurse, a health promoter, the ethnographer and her research assistant. During these sessions, the staff were at pains to impress on the young women the very serious consequences of not breastfeeding exclusively for six months, regardless of HIV status. They made an explicit link between 'mixed feeding' and the possibility of disease later in life, based on an understanding that the infant gut is vulnerable and that 'chemicals' in food can enter the gut and have long-lasting effects on health. 'If you mix feed', the health promoter warned mothers, 'you are introducing the baby to diabetes, to hypertension. You can see your child: maybe he gets diabetes or hypertension, because you as a parent, you introduced her to food at an early age'. Again, the language used denoted maternal responsibility and blame for future adverse outcomes. The health promoter's stern warning concluded with trite encouragement: 'Start bit by bit. A journey of a thousand miles starts with only one step. Only one step'. Rather than attempting to allay anxiety, this statement merely reinforced the mothers' responsibility for each step in a long and arduous 'journey' to intergenerational health.

Theory from the Waiting Room

As João Biehl has argued, 'ethnographic subjects allow us to return to the places where thought is born' (2013, 577). In the way of theory then, my informants' words in the waiting room can attune our understanding of epigenetics and the everyday. My informants' experiences in the clinic confirmed that pregnant women were highly aware of the concepts illustrated by clinic staff, health promoters, posters and pamphlets, and that these concepts circulated among

the women as well, particularly during waiting time. Here I draw on conversations with four of my informants: Lindiwe, Inam, Bathandwa and Nonyameko. I had met each of them in the clinic early on in their pregnancies, and we had spent many hours together in their homes and at their clinic visits, during which I acted as companion.

Lindiwe was a 25-year-old woman pregnant with her second child. She was studying for her final school exams when I met her, and she requested my help with her exam preparation. Mitosis, meiosis, genes and DNA were part of our common language as we pored over her biology textbooks. Lindiwe was knowledgeable and not shy to share her knowledge with others. She told me the story of her long queue at her two-month clinic visit. She had admonished the woman in the queue next to her for not breastfeeding her infant, based on the impression that the child was overweight. 'It's too much!' she exclaimed to me, 'You will go to the hospital for the rest of your life with that bottle feeding!' Lindiwe was concerned that the infant was already overweight, and that bottle feeding might increase the risk of childhood illnesses, such as asthma and adult diseases like diabetes.

Inam, a 23-year-old woman also pregnant with her second child, similarly linked early complementary feeding to later obesity: 'There is nothing wrong with being on a schedule for the baby to eat. Not 1-2-3 every time they cry you make a cereal and then feed them. Because then they just want bigger portions, they get bigger and bigger, and you have an obese child at age five. No: I like proper food, prepared in a certain way. If you get fat on junk, you're not fit. Then you get high blood, diabetes. And all these things could have been avoided'. Inam had a tertiary education and would 'google' any questions she had about pregnancy and nutrition.

Bathandwa had completed school and was hoping to open a crèche. She was 29 years old at the time of her second pregnancy and was conflicted about the nutritional advice doled out in the clinic. 'It must be something that I long for, for myself. Because I come to [the clinic]. But I don't like it when I am told what to do, because we are not the same. But some people would take the advice, because they see the danger ahead'. I offered that not everyone agreed that dietary advice should be part of medical care, but Bathandwa, despite her reluctance to accept advice, insisted. 'It *is* medical. It's like the old people—they have few times that they are ill. Maybe colds or a stomach bug or something, but not really sick. It is because of what they eat. They were eating veggies, more veggies, *imifino* [spinach], you know, green stuff! Beans, original beans. Now we are ill. *We are ill!* [raised voice]. Really. Everyone is ill. Sugar, cancer, TB, HIV, you name it'.

Epigenetics and the Everyday

Bathandwa's insistence that nutrition was 'a medical thing' was echoed by all of my informants. The casting of food as a medical concern, and eating as medicating, cannot, however, be solely attributed to information dispensed in the clinics. As significant work in medical anthropology has made clear, the clinic as an idea or a practice is enacted across multiple sites (Chatterji et al. 1998; Das and Das 2006; Goodfellow 2014; Carney 2015), and healthcare and health surveillance in Khayelitsha were not confined to the clinic facilities. Global health forms and actors are increasingly part of daily life in Khayelitsha. The households I came to be a part of during fieldwork were visited by trial recruitment teams, NGO outreach services, 'mentor mothers', community healthcare workers and even shady salesmen hoping to sell products with perceived health benefits. Trial participation, community surveillance and ideas of self-management are part of everyday life. This 'decentralisation of the clinic', as Megan Carney has observed, delivers clinical authority to previously intimate spaces (2015, 198).

For example, during one of the afternoons we spent with Nonyameko shortly after her baby was born in March 2015, our conversation was interrupted by the arrival of two women wearing light blue golf shirts with a small logo of mother and child over the left breast. They introduced themselves as 'mentor mothers' and stated that they had come to assess the baby as part of the programme they run with pregnant women that they meet in the clinic or in the community. The organisation's website stresses that 'If you get the first 1000 days (including gestation) right your impact over the life course of a child is much easier; you get it wrong and you're playing catch up'. The mentor mothers visit antenatally, three days after birth, at one week, at one month and then monthly. During pregnancy, they monitor the mother's weight of the mother, and then after birth, the infant's weight and wellbeing. 'The scale is an important tool in getting entry into a household', the website states. 'Mothers are keen to weigh their children and the scale becomes the central point around which a discussion about child nutrition and health takes place'. If the baby is not gaining weight, the mother is referred to the programme's nutritional adviser, who can, according to the women, 'teach the mother how to eat and how to feed the baby'.

Postgenomics and Global Health

This chapter provides an example of the ways in which global ideas about nutrition during the perinatal period, newly informed by DOHaD and epigenetic science, make their way through policy channels to bear on ordinary

life in settings in the global South. The 'thousand days' initiative offers an example of how biological concepts that are not necessarily new (Pickersgill et al. 2013; Meloni 2016) are harnessed for political and social projects. These may reflect familiar trends rather than truly novel interventions. Developments in the natural sciences are themselves a product of their social milieu, and their influence is predicated on socio-political stakes distinctive to their era (Meloni 2016). DOHaD and epigenetics have emerged as research fields in the time of 'global health'. 'Global health' is itself a knowledge field of the early twenty-first century, characterised by the logics of humanitarianism, anticipatory action and economic incentive (Lock and Nguyen 2010). The remainder of this discussion uses the 'first thousand days' case study to consider what this example might reveal about the dominant values that shape the uptake and application of scientific knowledge.

DOHaD Geographies

Anthropologists have questioned why 'some places, people, and health inequalities fall under the purview of "global health" while others do not' (Brada 2011, 286; see also Fassin 2012b; Pigg 2013; Crane 2013; Biehl and Petryna 2013). Similarly, we might ask why some places and people fall under the purview of 'DOHaD', while others do not. The DOHaD conference illustrated how scientists, clinicians and public health experts translate the language of DOHaD and epigenetics into applied policy, with a sharp focus on 'transitioning societies'. Their translation illuminates the legitimacy of interventions focused on developing countries and the mother-child dyad, the economic priorities of these interventions and the shifting categorisation of disease as adult non-communicable diseases become linked to early life.

The usefulness of transition theories for understanding disease distribution has been closely questioned by demographers and social scientists, who argue that local health and nutrition transitions are not unilateral, that patterns overlap and that local, historical and political-economic factors account for unique configurations of transition (Frenk et al. 1989; Chen et al. 1993; Ginsburg and Rapp 1991; Nichter and Kendall 1991; Ulijaszek et al. 2012). Yet transition theories continue to shape public health discourse in the global health era (see Yates-Doerr 2015). When articulated together, DOHaD and nutrition transition discourses rationalise interventions in the early life period in settings in the global South. The globalisation of the food system in these settings has radically increased the availability of cheap and energy-dense foods, to produce the so-called mismatch between early nutritional and adult nutritional environments that contributes to the emergence of non-

communicable disease via programming effects (Popkin et al. 2011). These discourses thus demarcate geographical regions of concern for DOHaD science. These regions are often glossed as 'the developing world', and are characterised by cyclical patterns of intergenerational metabolic disease, where there is a dual burden of child undernutrition and adult obesity (Vorster et al. 1999; Popkin et al. 2011). Whether this suggests the unreflexive application of 'transition' models (Carolina and Gustavo 2003) or a postcolonial agenda (King 2002; Brown and Bell 2008) is subject to debate.

The Global (Re)Turn to the Maternal: Economic Precepts and Concepts of 'Life'

Contemporary global health interventions depart from their predecessors in their use of explicitly economic frameworks that privilege measurable outcomes, and 'the thousand days' campaign is no exception. DOHaD 2015 encapsulated the 'global' view of the developmental origins of health and disease, epigenetics and the first thousand days: as a cost-effective project to decrease the transgenerational risk of non-communicable disease. That such interventions are underpinned by an economic logic was evident: the conference devoted an entire panel to 'life course economics', which included presentations on 'Childhood exposures and adult human capital' and the 'Best buys in the First 1000 days for South Africa'. A key message of the conference was that evidence with an economic rationale is more easily translated into policy, and a number of the satellite events featured sessions on methodology that could address this need, such as better longitudinal cohort data analysis, and how to conduct robust pre-conception studies. The perceived cost-effectiveness of early life interventions, and the currency of the mother-child dyad in the humanitarian imagination, has thus revived interest in, and funding for, maternal and child health interventions. As one epidemiologist told me, the first thousand days is 'where you get bang for your buck'.

That focusing on pregnant women and children 'gets you somewhere' with both state structures and private donors is likely a function of the perceived economic payoffs of this investment, and of the currency of the mother-child dyad in contemporary global health on the African continent. Interventions like the first thousand days campaign appeal to the logics of both saving 'lives' and valuing 'life'. The global (re)turn to the maternal reflects a contemporary value placed on 'life', a concept most fully embodied, as Barbara Duden has argued, by the unborn child. Analysing the evolution of the abortion debate in Germany in the twentieth century, she outlines how 'in the course of one

generation, technology along with a new discourse, has transformed pregnancy into a process to be managed, the expected child into a foetus, the mother into an ecosystem, the unborn into a life, and life into a supreme value' (1993, 2). Didier Fassin, drawing on his work on the South African HIV pandemic, argues that 'it is possible—and indeed crucial—to differentiate evaluations having for their object the worth of lives, and judgments predicated on the value of life. The grammatical number (lives versus life) is as important here as the lexical variation (worth versus value)' (2012b, 111).

Underpinning the first thousand days' intervention is a judgement of life's value, which is constituted in economic terms by the concept of 'human capital', newly shaped as an epigenetic outcome of nutrition in early life. Human capital formation is captured in indices of adult height, educational achievement, income and offspring's birth weight. These indices, following Didier Fassin, might be viewed as 'qualitative data offering political insights as to how societies produce and reproduce themselves' (2012b, 109; Canguilhem 1978). Siddiq Osmani and Amartya Sen view these interventions in the early life period as part of a 'hard-nosed economic calculus' (2003), a logic that appeals in an era that demands measurable economic outcomes and capital creation. Health and nutrition in this formulation become variables in predictive calculations of physical and cognitive development and work capacity (Yates-Doerr 2011). In addition, pregnant and newborn populations are easy to count. In the 'systematic triage' (Nguyen 2010, 178) that allocates funding and resources, a population that is easily constituted, easily singled out for intervention and easily counted—a governable population—appeals to the measurability, reproducibility and 'scaling up' that global health projects require (Adams et al. 2014). The 'thousand days' global health project is thus one instantiation of how the epistemology of market logics informs the life sciences and their application to public health concerns—what Kaushik Sunder Rajan refers to as 'the capitalisation of life' (2012, 1). That this capitalisation relies on DOHaD and epigenetic ideas supports Fassin's summation that there has been 'a profound change in the recognition of the value of life, which has shifted from the political to the biological' (2012b, 112).

Humanitarian Logic: The Travelling Technology of Mother and Child

If the image of the foetus is the paradigmatic emblem of 'life', the image of the African mother and child is arguably its other—the paradigmatic emblem of 'lives' (Fassin 2012b). Despite its global remit, the websites, posters, pamphlets

and media of the 1000 Days' initiative commonly feature images of African women and children, many of which are reminiscent of those used to depict 'Third World' famine in the 1980s and 1990s (Burman 1994), and later, AIDS orphans in Africa (Fassin 2013). The deployment of the African mother-child dyad and the moral sentiment it seeks to elicit exemplifies the logic of 'humanitarian government' that Didier Fassin describes for the present era (2012a). The contemporary circulation of this assemblage, itself closely tied to the invention of Africa (Mudimbé 1988), acts as a 'travelling technology' (Petryna 2009; Von Schnitzler 2013) that configures global health policy. In the pamphlets that now circulate through the waiting rooms in the antenatal clinics of Khayelitsha, that trope reappears in the form of a stylised image of mother and child, with the message that 'The first 1000 days of a child's life is a very important time for shaping a child's ability to grow and develop". Across this iconography, it is the image of the child which carries 'affective authority', as Liisa Malkki puts it, to constitute a transnational sphere of exchanges that can be thought of as ritual acts, given that they appear and are conceived of as 'apolitical, even suprapolitical', despite having clearly political outcomes (2015, 79). In Johanna Tayloe Crane's assessment (2013), global health projects in Africa today reproduce the colonial motivations of 'extraction' and 'salvation' that Nancy Rose Hunt has described as the main historical concerns of empire in Africa (1999). 'Salvation', in this formulation, has no better illustration than the repetitive images of African women and their infants that accompany promotional material, donor websites, editorials, media campaigns and the like. The 1000 Days campaign is no exception in this regard.

Anticipatory Global Health

The political currency of DOHaD and epigenetic concepts rests in their anticipatory nature. In 2014, *Lancet* Editor-in-Chief Richard Horton hailed early childhood nutrition as the 'origins of sustainable health' in a commentary titled: 'a new path towards anticipatory global health' (2014). The first thousand days project is the exemplar of the new anticipatory regime to which he is referring, in which the logics of biosecurity extend further into the everyday. Anticipation has been a central theme in the study of 'global health' phenomena, focused on biosecurity and preparedness (Lakoff and Collier 2008; Caduff 2014). Less attention has been paid to the opposite orientation that many global health projects espouse: that of potential, the utopian counterpart to the dystopic visions of war and pandemics. The 'unknowns'—killer

viruses, natural disasters, apocalyptic possibilities—that characterise and legitimise regimes of preparedness find their other in the 'unknown' of potentiality, defined by Taussig and colleagues to have three meanings: 'The first denotes a hidden force determined to manifest itself—something that with or without intervention has its future built into it. The second refers to genuine plasticity—the capacity to transmute into something completely different. The third suggests a latent possibility imagined as open to choice, a quality perceived as available to human modification and direction through which people can work to propel an object or subject to become something other than it is' (2013, S4). In the logic of biosecurity, the 'gap that cannot be known' is the space between the present and the time when the next pandemic or disaster arrives (Caduff 2014, 302). DOHaD and epigenetic logic, however, while it creates new issues for biosecurity (Guthman and Mansfield 2013), is also characterised by potentiality, which 'indexes a gap between what is and what might, could or even should be' (Taussig et al. 2013).

Following Alex Nading then, we might argue that the seamless uptake of DOHaD and epigenetic discourses into global policy is one reflection of a 'chimeric globalism', constituted by Nading as the 'speculative, spectral, and hybrid aspects of global health' (2015, 357). The notion of investing in future human capital appeals to the speculative logic that governs markets (Guyer 2007). Paradoxically, risk calculation in the epigenetic era, which is also the age of evidence-based medicine, operates on 'probabilistic information', a 'divination of the future' in Margaret Lock's estimation (2005, 52). Adams and colleagues use the word 'anticipation' to describe this new orientation, which 'authorises speculative modes of engagement' and entreats future-oriented thought and action (2009, 246). They argue that girlhood has become a central focus of this discourse as a crucial investment period for the formation of human capital. This 'preoccupation with the global future' is a central tenet of development ideologies (Quarles van Ufford and Giri 2003, 13), but this is newly formalised by projects with an explicit lifecycle focus that target measurable outcomes. DOHaD and epigenetic discourses are also spectral in nature: as Meloni has highlighted, DOHaD scientists appeal to notions of 'fate' and 'destiny' (2016, 208, referencing Gluckman and Hanson 2012). These imaginaries of future potential access 'an imaginative space of magic and mystery' where life is plastic and can be formed and reformed (Taussig et al. 2013). Hybrid notions of how 'bodies everywhere and always are being remade by their environments' (Guthman and Mansfield 2013, 499) have appeal in developmentalist discourse, and yet are paradoxically harnessed to reinscribe responsibility into the individual (often maternal) body (Mansfield 2012).

Conclusion

The 1000 days construct offers one lens with which to interrogate how new biosocial concepts of life arrive in policy and public spheres. The postgenomic age has been characterised as inherently global (Thacker 2005) and distinctly neoliberal (Meloni 2016) and founded on medical sciences that are 'fundamentally statistical in nature', and 'invested in probabilities and risks' (Abu El-Haj 2007, 289). Postgenomic medicine—its economic rationality and its focus on risk and potential—is one part of a socio-political milieu we might call global health, which has as its characteristic features a focus on economic applicability, an appeal to humanitarian imagination and an affective anticipation. The 1000 days campaign offers one example of the extension of these logics into ordinary domains of life. Further empiric work across different sites is needed to reveal the uneven ways in which new biosocial discourses come to bear on governance for different populations. As João Biehl contends, it is necessary to ask 'when and under what conditions marginalized people [are] accounted for as population-subjects in new biomedical regimes' (2011, 106). Ethnography will continue to be a key tool for rooting such investigations in historical and material context to persistently trouble emergent biosocial borders.

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13

Genetics, Epigenetics and Social Justice in Education: Learning as a Complex Biosocial Phenomenon

Deborah Youdell

Introduction

In this chapter I explore the possibilities offered to social justice-orientated education by bringing insights from sociology and biology together. I locate this in key education debates over inequality and social justice, debates that traditionally hinge on 'nature versus nurture' positions. This nature versus nurture bifurcation suggests that the biological and the social cannot be reconciled, let alone integrated, in education studies. Yet this, I argue, misrepresents the problem and is an oversimplification of a complex and moving terrain of policy, media, research and education practice that might be better characterized in the contemporary moment in terms of 'nature versus institution' and 'institution versus family'. In response to and in an attempt to shift the limitations of these debates, I introduce epigenetics—a whole range of work in new biological sciences that examines the interaction of environment and biological processes at the level of the gene and its functions. This is not a rush to replace sociological analysis with claims to biological evidence and 'solutions'. Rather it is to emphasize the entanglement of the social and the biological, and to advocate engagement with the breadth and nuance of sociological understanding of environment (Meloni 2016).

The chapter suggests that prior sociological analyses of institutions, politics, discourses and subjectivities remain crucial (but are not necessarily core)

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and—building on Foucault's account of productive power and subjectivation; Bulter's account of subjectivation, recognition and identification; Deleuze's work on assemblage and Barad's work on phenomena and intra-action—I suggest a move to thinking about socially just education *biosocially*. I posit the biosocial phenomenon of learning and outline some of the possible productive forces that in intra-action might make this, identifying some of the potential social, psychic, affective and biological processes and mechanisms at work. I then show how this approach opens up and extends lines of analysis by using it to think again about a pair of ethnographic school encounters (Youdell 2010). Through this engagement, I show the intra-action of multiple social and biological productive forces and make a case for a new biosocial orientation in education.

Socially Just Education: Educational Outcomes, Everyday Practice and Learning

One of the central premises of sociology of education is that schooling and the educational outcomes it produces are unfair. What precisely counts as fairness/unfairness remains unresolved, but the debate hinges on the presence or absence of equitable treatment, experience, opportunity (to participate and to learn) and outcomes. There is much international evidence that material, social and symbolic advantages and disadvantages correlate with educational outcomes. Population-level quantitative data on education outcomes, such as Programme for International Student Assessment (PISA) scores, show strong associations between test scores and socio-economic status, race, ethnicity, religion and nationality (OECD 2013). Poor and minoritized populations generally perform least well in such tests, and countries with the greatest material inequalities also have the greatest educational inequalities (Wilkinson and Pickett 2009). Test performance as a measure of educational inequalities is a site of substantial debate, but it is important to keep in mind that a rich account of social justice in education means more than outcomes alone.

A major stumbling block in media accounts, policy, research and educational practice is whether fairness is compatible with, or indeed to be achieved through, differentiation—treating different groups of students differently—and the relationship this should have with resource allocation. Key here are the terms of this differentiation and allocation. Critical sociology of education and progressive education have advocated socially

mixed schools and classrooms and 'mixed ability' teaching, while simultaneously advocating a redistributive approach to resource allocation favouring the most economically disadvantaged and socially marginalized (Gillborn and Youdell 2000; Lupton and Fuller 2009). In principle redistributive efforts are targeted at those doing 'least well' against the range of measures used by formal education in order to 'close the gap' between them and those doing 'most well' (a closure that Gillborn demonstrates is all but impossible in the current education system (Gillborn 2008)). In the UK a degree of redistribution through education is widely accepted, and at the macro level, this has been embedded in school financing in the form of additional payments to schools based on their student profiles. At the same time, neo-liberal and neo-conservative politicians, policymakers and lobbyists as well as traditionalist educationists and the popular media advocate differentiation based on 'ability' and extensive freedom for schools, including over resource allocation. Indeed, in the UK policy mandates 'setting by ability', which commonly includes 'gifted and talented' as well as 'remedial' and 'special' provision, and school freedoms mean that at a micro level resource allocation may well not neatly reflect a redistributive model.

This brings us to the nub of the debate over education and social justice—whether socially just education means we want all children to get the same outcomes. And whether in an ideal learning situation all children should and could get the same results. This is an ambition that remains legitimate in some systems, such as Finland (Butler 2016), but which has been rejected in many nations, including the UK. In the absence of all children getting the same results, the issues become whether socially just education means differential results that are evenly distributed across race, ethnic, gender, social class, religious and disabled groups. Of course in practice this is not what differential educational outcomes look like.

While learning is not the same thing as test performance and tests may capture only a part of children's learning, one of the things that children are taught is how to perform in tests, and it can be difficult to disentangle these tests and outcomes from the everyday processes of schooling. This is emphasized by analyses of the differential impact of testing regimes on types of school and on differently advantaged and marginalized groups of students and of the damage done to learning when it is reduced to test performance (Au 2007; Booher-Jennings 2005). Attending to social justice and injustice in educational outcomes, then, means simultaneously attending to everyday life in schools and classrooms.

Nature Versus Nurture in Education: Nature vs Institution, Institution vs Nurture (aka the Family)

The question of the shape and possibility of socially just and even equitable education for all students (learners?) is often cast as one of nature versus nurture—the learner is understood to be who s/he is *either* due to her/his pre-existing nature *or* due to the nurture s/he receives. This 'either/or' continues to resonate in the popular imagination and remains the cornerstone of much research that pursues one or the other of these lines of causation/influence. Yet this apparently clear distinction is not so clear at all.

Threaded through this enduring debate, there are more subtle distinctions. First of these is that of *nature* vs *institution*, in which the learner is understood as *either* encumbered by her/his genetically inherited intelligence and capacities for learning, as is proposed by evolutionary genetics (see, e.g. Ashbury and Plomin 2014) *or* by an education system and profession that creates these limits through its own constitutive practices, as proposed by critical sociology of education (see, e.g. Gillborn 2008, 2010). In this binary construction, education is either already socially just, producing the outcomes that differentially distributed genetic intelligence predicts, or socially just education is blocked by the very institutions that we charge with its delivery. The second distinction is that of *institution* vs *nurture*, where the institutions of education and social care understand (certain) children as encumbered by the particularities of nurture from their families (read as mothers), as suggested by popular and policy accounts of families (e.g. 'troubled families' in UK policy, Cameron 2015), and by epigenetic animal models (Champagne 2009) and their extension to humans (van Izjendoorn et al. 2011), *or* these accounts are critiqued as normative and marginalizing and resting on deficit accounts that are themselves constitutive (Edwards et al. 2015). In this binary construction, *either* education and social care intervenes into the problematic nurture of the family in pursuit of greater social justice *or* these interventions, despite having socially just intent, in fact reproduce the negative accounts of marginalized children and communities, and alienate them from schooling (Edwards et al. 2015). This is well illustrated by a recent article, 'Ten tips for being a better parent' written by a headteacher and published by the UK's *Guardian* newspaper in which the headteacher offers advice to and makes a series of requests of the imagined parent. The 'tips' cover interpersonal behaviour, conversations, homework, device usage, TV, social media, conflict at school, food and sleep. The tenor and substance of each of these does not suggest mutuality or collaboration; rather it suggests a parent body that is ill-informed, ill-behaved

and in need or correction: for instance, 'If you act aggressively in everyday life, in the street or car park, that could be reflected in how your children behave'; 'When your 11-year-old gets into the car after school, please get off your phone—she wants to tell you about her day'; and 'A bag of Doritos is not a meal' (Anonymous 2016). This sort of address from the headteacher to the parent body encapsulates the '*institution vs nurture*' (aka the family) orientation, and illustrates how in practice particular accounts of nature and nurture are entangled. It is not simply the case, then, that recourse to nature leads to discriminatory explanations, while recourse to nurture leads to socially just explanations. In the mobile terrain of education, nature, nurture and institutions do a variety of productive work, with potentially fluid relations to social justice.

Biosocial Education

A biosocial orientation to education offers the possibility of avoiding nature versus nurture debates and deadlocks and better understanding the entanglement of institutions, social forms and practices, relationships and bodies. Thinking about education biosocially suggests that neither innate ability nor nurture (nor institutions) is sufficient to explain educational differences or injustices.

In an attempt to sidestep the limits implicit in these dominant framings, I foreground learning, reconceptualized as a complex biosocial phenomenon produced through multiple influences and processes that are trans-scalar, contextualized and moving. This framing retains insights from Foucault (1988, 1990) on productive power, discourse and subjectivation and from Butler (1990, 1997) on performativity, recognition and identification. Its engagement with an expansive array of productive forces, including those that are molecular and those that are non-human, owes much to Deleuze's work on assemblage (Deleuze and Guattari 2008; Deleuze and Parnet 1983) and subsequent work by Barad (2007) on the intra-action of factors and forces within phenomena. In this sense it resonates with work in sociology of education exploring trans-disciplinary concept studies and using theories of complexity to think about complex causality (Evan 2014; Ivinson 2012). The approach I explore offers the possibility of including in analysis of educational injustice and justice new sets of evidence from biosciences at the same time as it remains engaged with the range of evidence from critical education research and maintains in analysis the complex intra-actions of multiple productive forces.

However, a move to a focus on learning is not straightforward. In the multidisciplinary field of education, approaches to learning reflect the concerns and research traditions of particular fields, proceeding with distinct problems, conceptual tools and units of analysis. Indeed, I want to argue that the field of education's limited capacity to respond to educational injustice is in part due to its partial and fragmented understanding of what constitutes learning, what learning is influenced by and what the mechanisms of learning might be. For instance, in curriculum, pedagogy and assessment research, the focus is on what is learnt, through what processes and with what outcomes (James et al. 2006; Pollard et al. 2005). Pedagogical research suggests diverse orientations to learning and shows how different pedagogical approaches and styles of teacher-student relationships influence learning (Bibby 2011; James et al. 2006). In educational psychology research, the focus is on developing cognition (Cohen Kadosh and Dowker 2015), although this focus is challenged by contemporary theories of learning that model the social and dynamic nature of learning (Engerstrom 2009; Wenger 2009). In critical policy and sociology research, the focus is on the structures, systems and everyday practices that form the contexts in which learning takes place (Youdell 2011; Ball 2013). This research insists on a distinction between performance in tests and learning itself (Gillborn and Youdell 2000) and asserts the influence of institutional and social structures, processes and practices on learning (Gillborn and Youdell 2000; Youdell 2006a, 2011) as well as the place of the body in pedagogy and learning (Evans et al. 2011; Ivinson 2012, Youdell 2011). Yet all of this education research remains a step removed from learning, and any physiological mechanisms and effects of learning remain well out of reach. A turn to bioscience does not simply resolve this. Much is still unknown about the molecular mechanisms that drive the particular functioning of particular cells. And new biosciences are themselves diverse and proceed with different orientations to the 'problem'. Genetic research continues to look for candidate genes for particular learning 'disorders' (see, e.g. Hofmeister et al. 2015), thereby resting on an acceptance of the sorts of diagnoses that sociology of education robustly resists and critiques as constitutive (Harwood and Allan 2014). Where no single gene can be identified (which is often the case (Rose and Rose 2013)), genome-wide association studies (GWAS) seek out the possibility of small influences across many genes (cf. Plomin 2014). While GWAS has not yet been interrogated in sociology of education, its basis in correlational studies that look into a body divorced from social forces is likely to attract similar critique.

The work in biosciences that seems the most interesting for educationalist is that in epigenetics and neuroscience, where emerging knowledge about the

influence of the environment on the regulation of genes and on brain function, combined with the mutability of these effects, suggest the potential for collaborative trans-disciplinary work investigating *biosocial* influences and mechanisms in education.

Research in neuroscience is showing the influence of the environment on brain development, structures and functions and is generating new understandings of the interplay of daily life experience, brain functioning and well-being (Rose and Abi-Rached 2013, Fitzgerald et al. 2016). Indeed, research in neuroscience identifies windows of particular brain plasticity—the period in-utero, the early years, adolescence and into early adulthood—'the window for experience-dependent plasticity is long' (Noble 2015, 773). In relation to learning, albeit at some distance from education, neuroscience research is showing how attention works (Johnston et al. 2011), how different types of memory are formed (Jackson et al. 2011) and what the brain does during particular learning tasks (Macintosh et al. 2007). Educational neuroscience builds on these insights, and while the field is still being developed (Fischer et al. 2010), patterns of brain activity associated with particular aspects of learning and learning difficulties are being shown (Kuppen et al. 2014; Goswami 2015).

Epigenetics identifies the effects of environment at a molecular level, specifically on how genes are regulated and expressed. This work moves away from hard heredity and emphasizes the interplay between the events, experiences and exposures of a life and the way the body's genetic code is put to work (Moore 2015). These epigenetic effects do not change the genetic code (genome) and are not fixed—they can be further changed through environmental interventions (Champagne 2012). Epigenetic regulation of gene expression is not inherently good or bad 'it is an environmentally primed adaptation that may or may not be adaptive to future environments (van Ijzendoorn et al. 2011, 307). Epigenetic influences include nutrition and its metabolic effects: work in nutrigenomics shows the interaction between diet and gene expression, and work in metabolomics shows the molecular influences of nutrition and physical activity that result from the intermediate chemical processes involved in metabolism (Mickleborough and Lindley 2013). As with educational neuroscience, epigenetic findings, often from model animals, are only beginning to be translated to human development or learning. Furthermore, the complexity of the sort of phenomenon of concern to education and social welfare researchers, the limits of associational epigenetic studies which (necessarily) predominate human studies and the need to rely often on blood or buccal cells as proxies for human cells of interest (e.g. brain tissue) mean it is difficult to claim definitively epigenetic mechanisms or

disentangle proposed epigenetic mechanisms from the range of causes of adversity that social science ordinarily studies. With these important provisos for critical social scientists, epigenetic research into early-life experiences and educational trajectories shows associations between: sustained impaired learning and adverse experiences early in life (Bick and Nelson 2016; Kok et al. 2014), quality early-years education environments and improved developmental outcomes (Luijk et al. 2015) and modest uplift in income and improved developmental and learning outcomes (Noble et al. 2015).

Intra-actions

This multi-disciplinary evidence leads me to want to experiment with analyses of multiple intra-acting productive forces that might be influences on, or mechanisms of, or indeed be understood as the *phenomenon of learning*. These forces might include institutions, social milieu, politics, policy, discourse, subjectivity, pedagogy, relationships, feelings, food, physical activity, sleep, objects, fauna, flora or architectures, each of which may be productive at a range of scales of granularity—from the macro to the molecular. It is not possible to explore all of these here, but it is worth considering a selection in which the social and the biological are both suggested as influences or mechanisms and through which we might begin to think about the intra-actions of forces in phenomena.

Ethnographic research into student *subjectivity* demonstrates how students are 'subjectivated' (Foucault 1990) as engrained and overlapping discourses about race, religion, gender, social class, culture and intelligence inform and constrain teachers' sense of the possible (Youdell 2006a, 2011). This means that the knowledge about students that circulates in schools is informed by teachers' explicitly and implicitly held beliefs about them, as well as their wider values and perspectives (Bradbury 2014). At the same time, how children act, speak, play, what they value, the skills they can show and how they see themselves all inform teachers' implicit and explicit beliefs about students and the sort of recognition they offer to them. These interacting processes of recognition are constitutive—performatively producing the person that they recognize (Butler 1990; Youdell 2006b) and setting limits on the identification available to the person recognized—the child recognized as bright understands herself as bright and manifests her brightness in the classroom, while the child recognized as struggling understands himself as struggling and manifests his struggles in the classroom (Harwood 2006; Youdell 2006a). In neuroscience, research on memory and brain networks also suggests a 'self' that is

repeatedly remade, with imaging research suggesting a self-made over and over again as renewing neuronal and glial tissues take up endlessly renewed places in shifting networks of activity (Heatherton 2011; LeDoux 2003).

It is possible that, in the production of learners and learning, these ongoing social and discursive processes of subjectivation intra-act with ongoing neuronal processes of self-making and that these intra-act with *pedagogic relations*. The relationship between the teacher and the learner is central in pedagogy. Ethnographic and case study research in schools shows the intra-subjective relations of recognition and identification at play in classrooms (Butler 1997; Youdell 2006a) as well as the psychic dimensions of learning, in particular the ruptures experienced by learners as they learn and the need for learners to be 'held in mind' (Bibby 2011 after Winnicott 1960). Epigenetic research using animal models also shows the potential significance of nurturing relationships for learning. Studies of the epigenetic effects of maternal care in rats suggest that diminished care impacts stress responses which in turn effect cognition and sociality, but that these effects can be reversed through cross-fostering (Champagne 2009, 2013a). While it may not be immediately evident to social scientists how this can relate to human children, the relational and environmental stressors that are introduced to rats under controlled laboratory conditions are argued to be reflective of the sort of amalgam of factors that coalesce to create conditions of profound disadvantage for children (van Ijzendoorn et al. 2011). The care relations that produce epigenetic effects in rats, then, might be thought about in humans, as it is in some of the literature (c.f van Ijzendoorn et al. 2011), through the lens of attachment and sensitive-responsive care (Ainsworth et al. 1971; Meins 2013). Developmental epigenetics currently takes the mother-child dyad as its focus; however, it is beginning to investigate the potential effects of broader care relations, for example, with fathers (Gudsnuk and Champagne 2011; van Ijzendoorn et al. 2011), and plasticity suggests that potentially epigenetic responses may also be susceptible to relationships in educational settings (Luijk et al. 2015; Tognini et al. 2015).

It is possible that social, psychic and epigenetic forces of relationality in pedagogic encounters intra-act with subjectivities as well as with *feelings*. Children's capacity for emotional self-regulation in the classroom has been associated with effective learning (Bomber and Hughes 2013). Yet theoretical work suggests that feelings do not simply exist inside a person who is variably able to regulate these, but are social phenomena (Ahmed 2004; Boler 1999). Ethnographic research seeks to show through observation how feelings circulate as affective intensities, including in learning situations and in classrooms, and the social and educational effects that these have (Kenway and Youdell

2011; Youdell and Armstrong 2011; Mayes 2013). Research in educational psychology shows that feelings are integral to cognition (Cromby 2015), and behavioural psychology shows the stressfulness of schooling, demonstrating a significant rise in stress, for instance, as measured through levels of the stress hormone cortisol in hair samples, as children move from nursery into school (Groeneveld et al. 2013). Experimental animal work that exposes rats to foot shock in novel environments shows that in rats trauma has epigenetic effects that enhance memory, a common neuroscience proxy for learning (Molfese 2011). Yet experimental work that introduces rats to novel objects shows that inducing stress in these encounters impedes memory (Bevins and Besheer 2006; Leger et al. 2013), and studies of the epigenetic effects of early care in rats similarly show the negative impact of stress on learning and sociality (Champagne 2013b; Gudsnuk and Champagne 2012). In metabolomics, which considers the body's metabolic responses to stimuli, analysis of volatile organic compounds (VOCs) in exhaled breath (Turner et al. 2013; Heaney et al. 2016) is emerging as an important approach. Turner et al. (2013) demonstrates the impact of stress on VOC profiles, and VOC analysis of the atmosphere in a cinema auditorium during multiple film screenings has suggested 'emotional signaling molecules' in audiences responding to different types of film content (Williams et al. 2016, 1).

It is possible that the social, psychic, neurological, epigenetic and metabolomic forces of feeling intra-act with pedagogic relations and subjectivities and that these productive forces also intra-act with *food*. The health impacts of children's diets, in particular fat, sugar and highly processed foods, are a policy concern internationally (World Health Organisation (WHO) 2013). Education research in the experimental model is exploring the potential for nutritional involvement in a range of socially diagnosed 'disorders' and 'learning difficulties'. In this body of research, the existence of such disorders, so vigorously contested in disability studies and inclusive education (Harwood 2006, Youdell 2011), is taken as given. The focus is on the potential of diet, in particular Omega-3 supplementation, to ameliorate the experiences associated with the diagnosis of Attention Deficit Hyperactivity Disorder (ADHD), general 'anti-social' behaviour as well as reading and language learning difficulties and general cognitive development, without engaging the subjectivating force of these diagnoses.

Omega-3 supplementation studies in education are so far equivocal (Kirby et al. 2010b; Tammam et al. 2015), and while specific findings are associational and cannot offer mechanisms or causal pathways, they do suggest lines of influence. Omega-3 deficiency has been associated with reading and language difficulties (Kirby et al. 2010a), and supplementation has been

associated with higher literacy performance (Kirby et al. 2010b) and a high Omega-6/Omega-3 ratio in the central nervous system has been shown in negative association with neurotransmission in children (Tammam et al. 2015). In some children diagnoses of ADHD have been associated with deficiency in Omega-3, and supplementation resulting in higher Omega-3 levels has been associated with improved parent and teacher assessments of attention, hyperactivity and anti-social behaviour (Kirby et al. 2010b). Omega-3 supplementation has also been associated with reduced 'behavioural transgression' (Tammam et al. 2015). In health, physiology and sports medicine, findings of the positive effects of Omega-3 supplementation are more compelling, with tissue-specific studies pursuing molecular mechanisms of action (Kumar et al. 2016; Shei et al. 2014).

Social, discursive, psychic, affective, neuronal, and metabolomic processes and mechanisms intra-act as forces of subjectivation, relationality, feeling and food themselves intra-act to produce the *biosocial phenomenon of learning*. This brief consideration of some of the potentially intra-acting productive forces identified from emerging research across domains demonstrates the importance of pursuing empirically the analytic potential of biosocial analyses in education. In the final section of this paper, I draw on the analytic potentialities of these intra-active forces to revisit a piece of ethnographic data and analysis in order to illustrate what this approach might offer.

Reading Everyday Life in School Biosocially

In a previous paper exploring the subjectivation of learners in the everyday life of schools (Youdell 2010), I presented ethnographic observation of two encounters between a student and his teachers. It was a school for boys diagnosed (through the usual education processes, not using any form of biomarker) as having 'Social, Emotional and Behavioural Difficulties' (SEBD); the student was Graeme, a 14-year-old boy, and the teachers were Miss Groves, the ICT teacher and a senior male teacher. The two encounters were just minutes apart but were striking in the very different ways in which these encounters subjectivated Graeme. In the first encounter that took place in an informal ICT 'club' at morning break time, I offer an account of Miss Groves advising Graeme on aspects of an upgrade to a defunct school laptop. It appears that this is an independent extracurricular project that Graeme has chosen for himself. He appears curious, committed and self-motivated. In the chapter, I argue that through the encounter and the prior and future practices it is entangled with, Graeme is subjectivated as a good learner, even in the 'SEBD'

school, with this recognition extended by Miss Groves and allowing this identification to be taken up by Graeme. In the second encounter, I offer an account of Graeme, as he makes his way to his next lesson, the laptop under his arm, pursued across the school quadrangle by a male teacher who, hand bearing down on Greame's upper arm, demands to know the provenance of the laptop. Just moments and metres beyond Miss Groves' ICT classroom, the possibility of recognition as a good learner disintegrates, in this place and this moment Graeme is an impossible learner, there can be no legitimate educational reason for Graeme to be in possession of the laptop, he must be a thief.

Through these encounters, we can glimpse how everyday practices are forces of subjectivation that render 'good learner', as well as 'impossible learner' and 'thief', and trace the institutional forces that push children out of mainstream education into SEBD schools. These are embodied encounters of moving bodies; student and teacher bodies orientated towards each other, in pursuit and flight, hand to arm, eye to eye. How does Graeme feel as he discusses his project with Miss Groves? How does he feel as the male teacher rushes behind him? How does that hand feel on his arm?

In the Deleuzian mode that is popular in education studies at present, we might ask about the affective intensities that flow through these encounters, through this school, through schooling (see, e.g. Hickey-Moody 2009; Kenway and Youdell 2011). Approaching these encounters as biosocial and with attention to emerging epigenetic research creates another orientation, made possible by a prior Deleuzian encounter with assemblage, which opens analysis up to the interplay of multiple productive forces spanning orders and scales. It suggests intra-action (Barad 2007) between social, psychic, affective and biological processes that are not reducible to causal relationships, much less divisible from each other. Thinking in this way protects against switching sociological for biological or neuroscientific accounts, but instead, in Baradian style, suggests that we consider phenomena as the complex interplay of inseparable productive forces in intra-action. This then draws attention to possible spaces and omissions in the analysis I offered in the previous chapter (Youdell 2010).

Thinking about this encounter biosocially asks us to consider how intra-acting subjectivating, relational and affective forces intra-act with epigenetic and metabolomics processes and mechanisms. It enables me to consider how these subjectivating encounters might be entangled with hormones and neurotransmitters and their pathways and functions. It allows me to consider how oxytocin (a neuropeptide with a role in stress and social behaviours), the dopamine system (neural pathways in the brain that transmit dopamines

which have roles in motor function, cognition and pleasure and motivation) and the 'HPA' or 'stress axis' (the confluence of hypothalamic, pituitary and adrenal activity) might be implicated in these encounters that subjectivate a 'good' and 'impossible learner' as well as 'teacher'. It suggests that the capacities of cells to downregulate adrenocorticotropin (ACTH) (a pituitary hormone acting on the adrenal cortex) and corticosterone (an adrenal steroid hormone provoked by ACTH) are significant as one body pursues and another body flies across the quadrangle, constituting 'thief' and 'authority' (policeman). Thinking biosocially pushes to the fore the distinct temporal movements of the biochemical, the metabolomics, the social and the relational, and how the social and relational come to be instantiated in the body (Frost 2016). This leads me to consider how these neurotransmitters and molecular capacities trace lines in space and time to other encounters, passing and significant, to prior school and other spaces, to prior teachers, friends, foes, carers and mothers. It leads me to think about how these mechanisms might orientate these bodies in a particular way (as opposed to myriad other ways) in the ICT classroom and across the quad. And it leads me to question how these biological processes might relate to any answer that Graeme or the male teacher could give to a question of how they felt, how they felt in relation to the other or what their body could/or could not do in the moments of the subtle flight and obvious pursuit.

Thinking biosocially also enables me to consider the possible metabolomic and epigenetic influence of these bodies sleeping last night, the night before, the night before that and their histories and habitual patterns of sleep. And it enables me to consider the possible metabolomic and epigenetic influence of the food eaten (or not eaten) this morning, yesterday, the day before and histories and habits of eating. It demands the consideration of how these might relate to what these bodies felt and could do in these encounters. Thinking biosocially also allows me to consider the volatile organic compounds (VOCs) that might be found in the exhaled breath of Graeme and Mrs Groves as they discuss the laptop project, or in the atmosphere of this ICT classroom at break time, and what this tells us about what bodies do and which students can be here. Likewise it allows me to consider the VOCs that might be exhaled as one body seizes another in the empty quadrangle. In so doing it opens up the possibility of exploring what volatile organic compounds can tell us about the metabolic processes of these bodies, in these moments and in the minutes and hours before and afterwards, and what they can tell us about these encounters.

Critical sociology of education is not undercut by these considerations; its analysis of fields, institutions, discourses, structures, subjectivation, and

affective, temporal and spatial flows still stand. While an engagement with questions, findings and methods from biosciences may feel risky, and for good reason, education scholarship, and potentially practice, in which social justice is central is emboldened by an engagement with intra-active social and biological forces.

Conclusion

Biosocial education research brings into view the trans-scalar and trans-temporal—epigenetic, neuronal, metabolomic, social, cultural, affective, psychic and relational processes operate across multiple temporal frames and scales, from the micro and the momentary, to the macro and the enduring, even as they intra-act as productive forces (Frost 2016; Moore 2015). It also demands that we address difference (and its deployment in hierarchical ordering) as more than social and/or discursive, taking seriously the possibility that some differences are instantiated in bodies in ways that are both opaque and potentially difficult to shift (Frost 2016, this collection). Furthermore, it underscores how the experiences and exposures of a life; social, culture and discursive flows; and biological processes are enfolded together in the making of subjects.

Across disparate social and biological fields of research, what constitutes learning remains unsettled, with learning meaning a range of things, from performance in standardized tests (OECD 2013) to animals' trained responses to environment (Molfese 2011). Knowledge about influences on learning remains similarly diffuse, from teachers' pedagogic practices and relationships with children (Bibby 2011; Youdell 2011) to modes of early care (Noble et al. 2015; Kok et al. 2014) and nutritional metabolites (Kirby et al. 2010a; Tammam et al. 2015). Likewise the mechanisms of learning, which range from teaching and learning approaches (James et al. 2006; Pollard et al. 2005) to associations between patterns of brain activation and learning tasks (Jackson et al. 2011; Bhide et al. 2013) and the methylation of particular histones in animal models (van Ijzendoorn et al. 2011; Molfese 2011). What is clear is that much remains unknown—we are a long way from understanding biosocial mechanisms of learning and the sorts of economic, social, institutional, pedagogic or even pharmaceutical interventions that such mechanisms may suggest in time. In the present, novel collaborations between new biological sciences and sociology of education have the potential to provide not only new or enriched insights but also to change the nature of the questions we ask

and the possibilities for pedagogic and political action we pursue (Fischer et al. 2010; Youdell 2017).

This biosocial understanding of the folding together of multiple intra-acting forces in the making of learners and learning that I have begun to explore here may have the potential to open up new lines of social justice-oriented critique, for example, of 'intelligence', of diagnosed 'disorders' and even of an education system based on competitive individualism under a meritocratic veneer. Caution is needed, however, as the potential 'plasticity' of the subject may well simply shift the locus of governance from the self-realizing subject to the machines of control societies (McGimpsey et al. 2016; Allan and Youdell 2017).

Attending to learning as assemblage of productive forces enables us to better see possibilities for and limits to a social justice agenda for education. It exposes, and perhaps offers ways around, some of the limits of existing research and policy agendas that posit singular and competing accounts of the causes (sometimes cast as primary or originary) of and therefore proper responses to social and educational injustices. Through biosocial analyses, the possibilities for social justice are clarified, even if they are also rendered more complex. It becomes clear that accounts of single-solution approaches will have (do have) limited effectiveness—analyses must attend to a range of productive forces, including some of those set out in this chapter, as well as their intra-actions. Indeed, we can begin to understand these as *intra-actions of productive forces in the complex biosocial phenomena of learning*. Once we understand learning in this way, our ambitions for socially just education, and education for social justice, must attend to this complexity and its biosocial character.

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14

Assembling Biomedical Big Data

Sabina Leonelli

Introduction

Big Data—the opportunity to assemble and analyze vast datasets produced at high speed and volume from several sources at once, thus promising to document in detail all aspects of phenomena under investigation—is hailed by many governments and funding agencies as a revolutionary tool for biomedicine (e.g. Hey et al. 2009; Kitchin 2013; Cambrosio et al. 2014; Tailor 2016). Its potential is thought to encompass a new relationship between patients and healthcare providers, and faster, more reliable and earlier diagnosis of disease. It is believed that it will offer a better understanding of the treatments likely to have a therapeutic effect based on the availability of larger bodies of evidence that can be statistically analyzed (e.g. Solomon 2015), and more precise understandings of the underlying causes of disease based on the integration of data from human subjects and non-human models (e.g. Clarke et al. 2014). In this chapter, I focus on the promise of big biomedical data to improve current understandings of disease. I examine the infrastructure needed to assemble, integrate and analyze the relevant big data and focus particularly on the ways in which online databases collecting human and non-human data are developed and maintained, with the aim of illustrating the opportunities and the challenges involved in such an exercise of data stewardship.

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This work builds on long-standing efforts within STS scholarship to note and investigate the role played by online databases within science, including its impact on the organization, order and communication of research outputs (e.g. Hilgartner 1995; Leigh Star and Rhleder 1996; Bowker 2001; Wouters and Schröder 2003; Hine 2006 and more recently Edwards et al. 2011; Stevens 2013; Borgman 2015 and Leonelli 2016). The dissemination of data through electronic means is now an essential complement to the publication of research papers, and the consultation of online, widely accessible databases has become part and parcel of everyday routines within experimental research (Lenoir 1999). The heightened need for specialist skills in computer programming has also affected the division of research labor and the ways in which scientists are trained. While university curricula in natural science are giving new prominence to information technologies and data science, database "curators" have emerged as a professional figure whose responsibilities lie in developing databases that satisfy the needs of prospective user communities (Baker and Millerand 2010; Chow-White and Garcia-Sanchos 2011; Leonelli 2016). The impact of online resources is particularly evident within the biological and biomedical sciences, where research communities dedicated to the study of popular model organisms have developed sophisticated databases for the organization, dissemination and comparative analysis of genomic data coming from different species (Leonelli and Ankeny 2012). These tools are often treated as a model for how cross-species data mining should be organized. This has special relevance for the development of information infrastructures for post-genomic, molecular-based medicine, which requires digital platforms through which data on human and non-human organisms can be integrated and compared. Indeed, the databases developed within model organism biology have been hailed as critical to "unlocking the very essence of biologic life and enabling a new generation of medicine" (Buetow 2005). Databases are expected to facilitate the achievement of these ambitious goals by fostering the integration of biological and biomedical knowledge, thus supporting translational research toward new forms of diagnosis and treatment.

This chapter investigates the role played by databases in facilitating the collection and use of big data in biomedicine through an examination of the practical difficulties encountered by database curators in fulfilling this task. The hype attached to database development as an easy solution to the deluge of data of biomedical relevance, coming from clinical trials and laboratory experimentations, as well as patient records, observational studies and health apps, has taken attention away from the problems involved in actually using data found online toward further research. In particular, matching in silico

representations of the world with experimentation in vivo and in clinical intervention, and aligning the experimental practices characterizing research on humans with the ones used to research non-human organisms present significant challenges. I view database development not primarily as a means toward the solution of those problems, but rather as a site where diverging stakes, values and epistemologies characterizing experimental cultures in biomedicine can be identified and discussed, thus highlighting the opportunities and challenges associated with the assemblage and integration of vast datasets derived from very different sources and methods.

The use of cross-species databases is an excellent instance of "biomedicine", defined as the set of practices which brings biological and clinical knowledge and techniques to bear on each other (Keating and Cambrosio 2003). Historians and sociologists of medicine have pointed to the extensive fragmentation characterizing the epistemic communities involved in biomedical research and have analyzed the complex relations and intersections between them (e.g. Loewy 1986; Quirke and Gaudillière 2008). Science and technology studies scholarship has documented how scientists attempt to overcome this pluralism in order to achieve common standards and procedures, for instance, in the case of clinical trials (Kohli-Laven et al. 2011), the trading of biological data and materials (Parry 2004) and the standardization of microarray experiments (Rogers and Cambrosio 2007). Database curation is another area where the introduction of standards, norms and specific technologies clashes with a highly fragmented and localized landscape of research habits and practices. This holds especially when considering the materials—the organisms—on which data are produced and disseminated. While some clinical research is conducted on humans, much clinical work involves the collection and use of data acquired on rats, mice and other, more distant relatives of *Homo sapiens* (Spradling et al. 2006). Similarly, biological research largely revolves around few key model organisms, such as the nematode *Caenorhabditis elegans*, the fruit fly *Drosophila melanogaster*, the thale cress *Arabidopsis thaliana* and the zebrafish *Danio rerio* (Davies 2004). Database curators can contribute different skillsets depending on the history and aims of the specific database at hand, and indeed their backgrounds can range from information engineering to bioinformatics, experimental biology or medical training. Their familiarity with the organisms whose data they are stewarding can thus vary substantially. Curators are typically aware of their important role in facilitating the comparison and integration of data on humans with data on model organisms. They are also aware that the success of their products depends on how useful they prove to be to experimenters, as this determines the levels of funding and community support that they will receive. Thus, the career of

curators depends at least in part on their ability to identify, embrace and constructively engage as many epistemic cultures in biomedicine as possible, which, in practice, means making their digital representation of data at least compatible with, and at best conducive to, widely diverse forms of intervention on actual organisms (Leonelli 2016). This inescapable commitment makes curators' insights into the interface between biology and medicine and the experimental cultures that characterize these two realms uniquely informative and valuable.

Model Organism Databases and the Incorporation of Human Data

Within model organism biology, huge efforts have been invested in database development over the last two decades (Bult 2006). These investments were fueled by the growing recognition, across biomedicine as a whole, that collection and dissemination practices affect whether and how data are reused toward new discoveries (Buetow 2005). Moreover, the extent to which model organism communities have engaged in database development is linked to their unique historical role in fostering a collaborative ethos within the notoriously competitive culture of biomedical research. Many of the most popular models, including the fruit fly, the thale cress, the nematode and both baker and fission yeast, owe some of their success as laboratory organisms to the collaborative ethos and interdisciplinary ambitions fostered by the scientists who pioneered their use in biology (e.g. Kohler 1994; Rader 2004).[1] Their explicit long-term goal was understanding organisms in all of their complexity through an interdisciplinary approach that would include genetics as well as cell biology, physiology, immunology, morphology and ecology. Their strategy to achieve this was to accumulate and integrate knowledge on the biology of individual species, which would then provide a blueprint and reference point for comparative, cross-species research. Over the last three decades, this attitude has been incorporated into the building of model organism databases, which are often referred to as "community databases" to stress their role in serving researchers by gathering and integrating all the information available on a specific organism of interest to them (Vize and Westerfield 2015). These databases are freely accessible online, thanks to public funding from national and international agencies, and have become an important component of the very identity and status of model organisms in research, on par with other characteristic features such as their capacity to represent other spe-

cies, their tractability in the lab and the extent to which they embody biological processes of interest (Ankeny and Leonelli 2011).

Examples of well-curated and widely used community databases include FlyBase, WormBase, The Arabidopsis Information Resource, PomBase, Mouse Genome Informatics and the Zebrafish Model Organism Database.[2] These databases were initially funded by public agencies to disseminate data coming out of sequencing projects. The curators of these databases took advantage of the funding to build tools to potentially incorporate other types of data on the same organism, and they have aimed to increase the diversity of the data that they host ever since. As a result of these efforts, the curators of these databases acquired a sophisticated understanding of the factors that influence the future adoption and use of data collected on model organisms across research contexts. These factors include the need to integrate different data types produced through various kinds of instruments and techniques, ranging from sequence data to photographs or tissue samples as well as the need to collect meta-data documenting the provenance of data. Further, database users want to be able to retrieve data through familiar keywords that they employ in their own research, and to be able to visualize data in a variety of ways, which helps with spotting significant patterns and correlations. Finally, users are typically interested in being able to access the organic materials on which data were originally acquired, such as specimens of a given mutant or a particular kind of tissue. Curators have proposed themselves as possessing the right skills to fulfil these complex requirements, and regular Biocurator meetings are now held across the globe to facilitate cooperation and interoperability across different databases (Howe et al. 2008). On the basis of their growing expertise and increasing need for comparative analyses, the curators of community databases have also engaged in the development of cross-species databases, where existing data on different organisms can be searched, viewed and compared. A well-known initiative of this kind is the Gene Ontology (GO), a bio-ontology developed jointly by the curators of several community databases for the cross-species annotation of gene products (Gene Ontology Consortium 2000). All the curators involved in GO hold a PhD in a branch of experimental biology and use that expertise to inform their curatorial activities. The GO currently includes data from dozens of species, including several grains, yeast, slime mold, rat, several microbes and *Homo sapiens*, and is coordinated by a central office at the European Bioinformatics Institute near Cambridge, UK.[3] Another important initiative is the Generic Model Organism Database project (GMOD), also referred to as the "myriads" database because of the sheer number of species that it incorporates. The GMOD project is the result of an extensive cooperation involving over 100 participating databases, including

repositories of human data such as Human 2q33, Chromosome 7 Annotation project, Xmap, Ymap and HapMap. Its main goal is to help species-specific databases to coordinate their efforts, so as to guarantee interoperability across databases and thus facilitate cross-species analyses. To this aim, the GMOD encourages database curators to use a common set of software packages, such as tools for browsing and annotating genomes, and to take account of the standards already employed by the main model organism databases when setting up new tools and resources (GMOD 2016).

Thanks to initiatives such as GO and GMOD, several features of the community databases developed within model organism research have been proposed as standards for the online gathering and distribution of biological data, including data on humans. The technical expertise accumulated by biologists in disseminating data obtained from model organisms is viewed as immediately relevant to human data. Yet, it is not obvious that such expertise is sufficient to manage and coordinate the dissemination of data obtained through clinical research. The paradox of proposing to treat humans as a model organism, while acknowledging that this effort is not typically carried out in cooperation with human geneticists and clinicians, is captured by the following quote from a paper summarizing discussions held at the 2006 meeting of the Genetics Society of America:

> A critical need is better cross-organism databases that enable one to compare the genes, expression patterns, gene functions, cell types, tissue organization, and biological subprocesses across organisms, including humans. Maintaining and expanding our community resources, such as mutant collection and siRNA libraries for many organisms, including those not amenable to standard genetic techniques, is crucial. They provide access to the genetic power of the different model organisms and enable investigators to take full advantage of whole-genome sequence information. Finally, we must look for ways to interact with clinician scientists and human geneticists and bring their knowledge and perspectives to the modeling efforts. (Spradling et al. 2006)

This quote indicates that clinicians have not been involved with developing cross-species databases, and that this lack of involvement needs to be remedied. This situation is puzzling, especially since one of the key purposes of cross-species comparisons is to achieve a better understanding of humans, leading to improvements in medical knowledge, diagnosis and treatments. In the words of a curator interviewed in 2008,

> model organism databases also included human because obviously people are interested in what goes on in human, so that gets included even though there isn't an organism database.

The curator is referring to the fact that, while there are hundreds of disease-/system-/organ-specific human databases, there is no unique "model organism database" for *Homo sapiens*. There are practical reasons for this: the sheer diversity and scale of data-collecting practices on human beings; the multiplicity of sites where such collections are taking place, and the impossibility of coordinating and standardizing the formats of collection; and the restriction to interoperability and access to human data, motivated by ethical concerns with privacy as well as by intellectual property, security and confidentiality issues in clinical research. If we stick to the above characterization of model organisms as ones on which all types of data can be collected and exchanged without restrictions, it is clear that *Homo sapiens* is not a model organism, nor could it become one in the future. Yet, in the context of cross-species databases, human data are often treated in the same way as data coming from model organisms.

This observation opens a host of ethical questions about privacy concerns and the status of individuals and populations in biomedical research, which are being examined by other chapters in this volume. In what follows, I wish to explore the differences in research practices that are brought to the fore by attempts to develop cross-species databases, and the resulting concerns for big biomedical data integration and analysis. I focus on four sets of issues that curators perceive to be emerging when human data are added to model organism databases. My analysis is based on a cross-examination of the content and guidelines of the GO and GMOD websites; and multi-sited ethnographic research on curation practices and database building carried out between 2004 and 2014, which included attendance at scientific meetings concerning biocuration in both model organism biology and medicine; visits to laboratories engaged in extensive bioinformatic work, including the development of cross-species databases; and extensive interviews with curators of cross-species databases based in the UK, Germany and the USA.

Issue 1: Data

The first issue concerns a divergence in the criteria used to determine what counts as reliable evidence. The problem is exemplified by the unclear status of microarray data as a source of evidence about gene expression. A great deal of standardization of terminology, experimental protocols and instruments is required to describe a microarray experiment—and, at the same time, to make sure that the procedures and techniques used within such an experiment are intelligible and replicable across different laboratories. This is mainly because

of the strong influence that even minute variations in a laboratory environment, such as a difference in temperature or lighting, can have on the results of a microarray experiment. However, it can also be due to the specific samples used, the ways in which instruments are calibrated and the ways in which the original model organism was nurtured in the lab. The MIAME project, which stands for Minimal Information About a Microarray Experiment, has been set up precisely to address this need and streamline the process of agreeing upon, and implementing, such standards. Still, the development of standards such as MIAME has been fraught with difficulties and controversies (Rogers and Cambrosio 2007), and MIAME standards are still not universally applied. This means that the quality and reliability of microarray data remains contested, and their replicability is sometimes questioned (McCarthy et al. 2009, 149). Indeed, several model organism databases do not accept microarray data as a valuable source of information. The following quote exemplifies the feelings of several curators whom I interviewed on this subject:

> I'm doubtful that we would include any micro-array results at the moment.[…] You get very variable results from micro-array and you get lots of indications that genes are involved in certain processes when they may not be. They're up-regulated because of various different reasons which may not be related to the experimental conditions that are used. So, yeah, they're a bit doubtful.

In clinical settings, the variability and lack of experimental "validation" of microarray data do not seem to raise the same amount of skepticism. Despite ongoing debates around the evidential value of the correlations emerging from genome-wide association studies (e.g. Fujimura 2015), there is widespread agreement that microarray experiments play an important role as data sources, especially since microarray results are being used in conjunction with other sources of evidence on the same genes/processes. The idea of experimental replication as a way to validate results is not as strong in clinical research as it is in biological research, for the simple reason that replicating experiments on the same tissues in humans is expensive, and often impossible, since even the samples that are stored in biobanks are unique and depletable. Further, clinicians see microarray experiments as exploratory tools: they can point to interesting correlations and patterns that might, upon further research, turn out to have biological meaning, even if they provide no clear evidence that those patterns exist, and would certainly not be trusted in isolation from other types of data. There is no reason why this approach should not be equally powerful in the biological realm. Nevertheless, curators perceive many biologists as showing a low level of trust in microarray results acquired by other

researchers and ideally wanting to be able to assess the reliability of microarray data on a case-to-case basis.

Another potential discrepancy between clinical and biological contexts concerns the ways in which data are mined from publications. Several model organism databases rely on text-mining, or in some cases even manual annotation, to extract published data from available literature on a specific organism. This arduous task is made easier by the existence of a coherent corpus of literature on each popular organism. The situation is perceived to be different in clinical research on humans, as discussed by one curator as follows:

> the literature[…]is probably the biggest single thing that model organisms have because of model organism databases and that is missing from the systems that deal with the human genome sequence or human gene expression data.

The large amount of literature available on the human body makes it hard for curators to find references for their annotations and spot uncontroversial, consistent sources for their work.

Issue 2: Meta-Data

A second challenge for database curators is the lack of agreement on what information needs to be included about the experimental circumstances in which data are originally obtained—in other words, information about the provenance of data, the processes through which data was produced and formatted for dissemination (Bowker 2001, 664). This information, technically referred to as meta-data, is crucial to assessing the evidential value and reliability of data found in a database. By accessing meta-data, users get to know who gathered the data of interest, the methods employed to do so and the research interests that motivated data production in the first place. These are all elements that help researchers to evaluate whether data are trustworthy, how they compare to other datasets available on the same phenomena and, as a result, what biological interpretation they could credibly support.

The gathering of meta-data is complicated by the fact that different labs disagree on what elements are crucial in describing the provenance of data (Edwards et al. 2011). Further, experimental protocols and procedures are constantly shifting, making it difficult to settle on fixed types of information as meta-data. Still, the curators of model organism databases argue for the importance of settling at least minimal standards for what counts as important information about an experiment (e.g. Taylor et al. 2008). The funda-

mental piece of information that needs to accompany each dataset is, unsurprisingly, the specific organism on which the data was obtained. The very idea of comparing data obtained on different organisms depends on clearly identifying the species, and sometimes even the individual specimen, on which the data were originally collected. And yet, precisely on this crucial point, curators find that clinical and biological researchers differ in how they conduct and describe experiments. Clinical researchers are perceived as frequently mixing organic materials coming from different types of organisms. According to the curators I interviewed, they often contaminate human samples with materials coming from other species—RNA probes coming from bacteria, for instance—and do not care to specify this when writing up their results. They sometimes even fail to specify whether they are working on human cells or mouse cells, on the grounds that they are convinced that this will not matter for their conclusions. This attitude clashes with the strict standards for annotating experimental materials and procedures adopted within model organism biology. This sometimes results in curators refusing to include data in a cross-species database, because they cannot classify them according to the organism on which they were originally acquired.

Further, curators are committed to distinguishing results acquired through experimental procedures (referred in the quote below as "primary annotations") from the interpretation of those results given by experimenters ("author statement", typically acquired by curators via direct queries to data producers). One of the worries underlying the contamination of samples is that experimenters tend to decide, on the basis of their own experience and of the specific circumstances in which data are produced, whether contamination is relevant or not to interpreting the results.[4] The reasons for this important decision are thus kept tacit and inaccessible to the users of databases that report those data, who are left with the only option of trusting the scientific judgment (and thus the beliefs and expertise) of the original data producers. This situation generates uneasiness among curators, since efficient data re-use is understood to involve the possibility to scrutinize (and if necessary, challenge) the beliefs and context in which data were originally produced. A consequence of such uneasiness is that human data on gene products are often annotated as author statements, because experiments are not carried out entirely on human tissue—which can be interpreted as indicating that these data are intrinsically less reliable and trustworthy.

One way to explain the perceived difference in the ways in which clinicians and biologists annotate their experimental results is to think of the different priorities and commitments involved in their daily activities. It is often said that while clinicians aim to cure, biologists aim to explain. This distinction

cannot be applied too neatly to experimental cultures in the two realms, since they both attempt to understand biological processes (whether general processes like metabolism and development or specific syndromes such as breast cancer) and to successfully manipulate organisms. However, curators' perceptions of how experimenters annotate and assess their data point to some interesting differences in the ways in which biological and clinical experimental results are valued and used. These differences might be partly explained by the ways in which experimentation in the two realms is evaluated by funding bodies. While biologists are increasingly asked to produce results of social and economic relevance, the quality of biological research remains primarily assessed through peer review of papers resulting from research efforts. As a result, enhancing the quality and credibility of experimental research in biology involves documenting and validating the sources of the evidence used to back specific claims, so that peer reviewers reading the resulting documents are satisfied. By contrast, clinicians' experimental results are valued primarily for their fruitfulness in supporting effective treatment of patients, and thus there is less incentive to carefully document every step of their experimental procedures.

Issue 3: Materials

The third issue I wish to examine is the handling of materials relating to the data generated and disseminated in biological and clinical settings. In addition to the discussion above, another possible explanation for the difference in experimental annotations concerns the relationship built by researchers with the organisms that they study. This raises questions about the experimental procedures used to select, manipulate and standardize organisms, both individual specimens and parts such as tissues, cell cultures, blood, and organs. Within model organism biology, the standardization of organisms is of paramount importance: being able to access specimens that are genetically and/or phenotypically identical to the ones on which experiments are carried out is seen as crucial to validating experimental results and pursuing research that builds on previous efforts (Rosenthal and Ashburner 2002). Model organisms are standardized through two types of processes. The first consists of the processes of transformation from organisms found in the field to laboratory specimens that are easy to keep in a laboratory environment and use for experimental interventions (a set of features typically referred by researchers as the "tractability" of an organism or species). The very act of transporting an organism into a laboratory environment occasions several changes to its biol-

ogy (ranging from its physiology to its genome), due to the need to live in an environment where the basic rules of survival in the wild are subverted. Organisms are also often genetically modified to exhibit features suited to the research goals at hand (e.g. the oncomouse). The second type of standardization is the one involved in the dissemination of specimens and related findings across research communities. For organisms to become favored scientific materials, it is not sufficient that they are tractable in a laboratory environment and useful for the research that is carried out. The organisms themselves need to be able to travel across different labs, so that researchers can verify those results and/or further them through more experiments. This contributes to defining the characteristics of the organism selected for research: bigger organisms fare worse than smaller organisms and organisms that easily survive displacement are favored.[5]

As illustrated by these procedures, the need to standardize guides and conditions all stages of researchers' interactions with model organisms. This situation is obviously different from the ways in which researchers interact with human subjects, and indeed neither of the two processes of standardization described above maps neatly onto the treatment of humans in clinical research. Let us consider the process of transformation first. It is true that human subjects are selected as subjects for research according to their biological characteristics, including at times their genetic make-up or their ethnic background.[6] Some clinical studies look for "adequate" populations across the globe—where adequate means representative of the traits that researchers wish to study, and/or amenable to the kind of treatment and sampling required for clinical research purposes. However, this latter interpretation of what constitutes adequate populations is under heavy ethical scrutiny. This is because the specific characteristics of the population being examined matter a great deal when attempting to establish the efficacy of a treatment, and yet many groups who may benefit from targeted medication have been historically excluded from acting as an adequate sample (e.g. children and pregnant women). Further, the notion that human beings might be used as instruments for research, to the point of infringing on their basic rights (among which the right to privacy), is extremely controversial (e.g. Waldby and Mitchell 2006; Sunder Rajan 2017). Another possible parallel to the process of attempting to transform humans into a model organism is the way in which patients are "prepared" for participation in a clinical study, for instance, through a specific diet and/or by imposing a set of appropriate behaviors and habits as a condition for participation (e.g. stopping to smoke or drink alcohol). Even when taking this into account, however, human subjects cannot be viewed as undergoing

physical modifications comparable to the transformation of model organism specimens so as to fit research needs.

Turning now to the process of dissemination, the parallels with the treatment of model organisms are more striking, even if still limited and controversial. While individual subjects are not routinely shipped around the world as a research commodity, samples of their tissues, cells or blood are disseminated through biobanks and thus selected on the basis of clear standards for what constitutes an acceptable donation (for instance, in terms of its integrity, characteristic features, provenance and means through which it was collected). Still, such dissemination of samples is subject to stringent regulation that vary across national borders (Gere and Parry 2006; Kaye and Stranger 2009). Also, variability across human individuals plays an important role in clinical research—each sample is unique and not easily cloned or reproduced, which turns samples into a precious commodity whose dissemination is only agreed upon under specific circumstances (Parry 2004). Overall, ethical, practical and regulatory constraints make it impossible to think of human subjects in the same way as we think of specimens in non-human research. Indeed, this is the very reason why, despite the well-known ambiguities in inferring medical insights from research on model organisms and the controversy surrounding the use of animals in research, experimentation on non-human remains a stepping stone for clinical research.

This set of considerations adds another layer to curators' worries about extensive differences in how researchers treat organisms. Clinicians working on mice are much more likely to adhere to the practices recommended by database curators to describe their specimens, while researchers carrying out experiments on human subjects and their parts operate in quite a different experimental culture. It is then not surprising that, while clinicians working on humans were not involved in the initial effort to develop the GO, prominent representatives of the mouse community were among its founders.

Issue 4: Terminology

The last issue is the choice of terminology used to classify and retrieve data across organisms. Already within model organism communities, the problem of choosing terms that different groups will recognize and understand is one of the most urgent issues confronted by curators. Achieving terminological compatibility across the human/non-human boundary and across biological and clinical practice is even more daunting, especially given the efforts already invested by the medical community (e.g. the Medical Subject Heading cre-

ated by the National Library of Medicine to index medical literature). Attempts to integrate the terminologies used in medicine with the ones used in biology have been under way for decades, and scientists are making headway especially when focusing on specific areas or diseases. To exemplify the issues that might emerge when merging vocabularies coming from model organism research and research on humans, I consider the recent merger of GO terms with the Unified Medical Language System (UMLS), a metathesaurus of 900,000 medical terms developed by the National Library of Medicine which is recognized as one of the most authoritative references for standard medical terminology (Nelson et al. 2002; Bodenreider 2004). Despite curators' published claims to the effect that the merger had been relatively smooth (Lomax and McCray 2004), my interviews with the curators involved reveal that this attempt toward integration generated some interesting paradoxes and led to revisions of GO. For example, a key organizing principle within GO is to distinguish terms that describe a molecular function from terms that describe a biological process. Within medical discourse, such partition is hard to apply, since the molecular function of gene products is automatically equated with a characterization of the biological process in which that gene is involved; thus, the UMLS nomenclature does not classify its terms in this way. Even where terms overlap between the two nomenclatures, the meanings assigned to those terms might differ. Indeed, nomenclatures such as ULMS and GO are organized hierarchically through a series of relationships. Basic relationships in GO are mereological ("part_of") relationships and functional relationships such as "regulates". In contrast, ULMS uses a broader range of relationships including "physically related to", "spatially related to", "temporally related to", "functionally related to" and "conceptually related to". Given these differences in semantic structure, terms shift their meaning depending on where they are situated in the network—in much the same way as the interpretation of single words in everyday communication depends on the linguistic and social context in which they are used.

Another issue emerges in relation to the process through which curators select which terms should be used to classify given sets of data. In biology, annotations tend to be based on peer-reviewed publications relating datasets to specific processes, functions or entities. In clinical research, it might be hard to find a direct, well-established link between a dataset and a term of interest—for instance, a disease. Still, there might be good reasons to suspect that such a link exists, and thus to annotate those data under the term referring to the disease in question. Trying to accommodate these different criteria is puzzling to curators trying to work on both realms. This brings us back to

the divergence in priorities discussed with reference to data assessment and meta-data annotation, and enables me to add a further layer to it. Clinical researchers can use incomplete information because, in their worldview, this is better than having no information at all. Biologists are typically more cautious in claiming causal links between biological processes, while clinicians are more used to take account of information deemed to be relevant to a given disease without fully understanding the mechanisms causing it. Thus, in biomedical research, any hint that points to the etiology or treatment of abnormal human phenotypes merits mention, whereas experimental research using similar data would be characterized as largely exploratory.

A more fundamental, conceptual problem with integrating nomenclatures across the human/non-human boundary concerns assumptions about the normal and the pathological. Databases such as GO have been built to focus on non-pathogenic entities and processes—which are referred to as "normal" (Gene Ontology Consortium 2000). The reason for this is that model organisms' datasets are supposed to be representative for the biology of a wide set of organisms and are thus conceptualized as documenting "typical" of "normal" gene functions found in a given species. Clinical research on humans has almost the opposite connotation: because the main interest is in understanding and treating specific pathogenic conditions, cross-species research is centered on diseases and the vast majority of available human data document so-called pathological states. This situation causes problems when it comes to incorporate data on diseased organisms into GO, with the consequence of making cross-species databases potentially less interesting to clinical researchers. It also raises the philosophical question of what constitutes "pathogenesis" and "normality" in the biological and clinical realms. This goes well beyond what I can tackle here, but it is important to mention since the way in which researchers answer this question deeply affects their conceptualization of how data should be collected, disseminated and interpreted.

Conclusion: Big Data Integration and Diverging Research Cultures in Biomedicine

I have singled out four challenges in the development of cross-species databases, which curators view as evidencing potential discrepancies between clinical and biological research practices: (1) the criteria for what counts as reliable evidence, (2) the selection of meta-data, (3) the standardization and description of research materials and (4) the choice of nomenclature used to classify

data. The controversies surrounding these aspects of database development reveal their significance in demarcating, and possibly reinforcing, epistemic differences between the lab and the clinic and between human and non-human research. Both sides aim to understand and change the world. Yet, biologists use data collections as a way to extend and test their understanding of organisms, while clinicians view the accumulation of data as essentially aimed to treat patients. This difference in emphasis is amplified by the evaluative cultures within these two realms. Clinicians are working in an environment where research is evaluated both for its contributions to medical knowledge and for its impact on treating patients. Despite the increasing push toward applied research, this is not the same for biologists, whose outputs are evaluated mainly through their published outputs. This in turn reinforces differences in how biologists and clinicians generate, evaluate and disseminate data. Clinicians tend to use data that biologists consider to be potentially unreliable and to value causal information that biologists do not see as conclusive. For them, inserting such information in databases means increasing the chance of gathering useful clues toward understanding phenomena of interest. By contrast, biologists fear that lowering standards for what counts as evidence will weaken the overall reliability of data found in databases, which will in turn encourage misleading or even wrong interpretations. At the same time, many of the challenges listed above stem not from cultural divergences between clinicians and biologists, but rather from differences in the research practices of experimenters who work with non-human organisms and experimenters who work with humans. Clearly, experimenting on humans brings ethical, financial and material constraints that are not present in model organism research, which generate differences in the ways in which researchers communicate and process data.

Databases have become crucial sites for the encounter of those diverging cultures, the identification of differences and the expression of conflict (which may or may not pave the way to its resolution; Leonelli 2016). The recent deluge of data is making it ever more difficult for biologists and clinicians to interpret the wealth of information found online in ways that help understanding the material bodies they work with—whether they are insects, plants, fungi, animals or humans. This process of aligning the informational with the material is specific to big data assemblages and analysis, and constitutes one of the foremost scientific challenges of the twenty-first century. The divides between biologists and clinicians, on the one hand, and human and non-human research, on the other, make this alignment evermore complex to achieve; and the work done by database curators is key to confronting this

challenge. How curators deal with pluralism in data production and interpretation is likely to have a huge effect on how different constituents of biomedical research relate to each other. The ways in which databases are structured, and the choice of which data get included and how, can dilute or reinforce the differences in research cultures noted above and provide a platform for critical and constructive discussion of how underlying disagreements and diversity in methods and materials can be handled when using big data assemblages as evidence for new medical claims and/or interventions.

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Notes

1. The importance of model organisms as tools for interdisciplinary collaboration has been stressed by pioneering STS studies, including most famously the notions of model organisms as "right tools for the job" (Clarke and Fujimura 1992) and "boundary objects" (Leigh Star and Griesemer 1989).
2. See the list of the main community databases funded by the National Institute of Health (http: www.genome.gov/10001837).
3. Details on the history, personnel and characteristics of GO can be found in Leonelli (2009, 2016).
4. This underlines an important tension between standards used in data infrastructures and the experience/context-specific knowledge of researchers, which I cannot discuss at length here for reasons of space, but is analyzed at length in (Leonelli 2016).
5. Plants, whose specimens can be sent around in the form of seeds, are among the best stocked and standardized organisms (Leonelli 2007), while mice and rat researchers rely both on whole specimen collections and tissue cultures (Davies 2011).
6. As discussed by other chapters in this volume, these are anyhow complex and criticized proxies for homogeneity.

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Part III

Neuroscience: Brain, Culture and Social Relations

15

Proposal for a Critical Neuroscience

Jan Slaby and Suparna Choudhury

Critical Neuroscience: Towards an Undisciplined Ethos for Critique

We outline the perspective of 'critical neuroscience': a stance of critique pertaining to neuroscientific methods, practices, concepts, discursive effects, formative backstories and societal impacts. Critical neuroscience brings together work from various disciplines with the aim to engage neuroscience practitioners as well as decision-makers, stakeholders and the public, bringing them to adopt a critical stance towards the entirety of the 'Neuro complex' in its present guise, including its broader impacts on scholarship, academia and wider society. This text is a programmatic outline which traces major lines of influence and theoretical backgrounds. It is an invitation to neuroscientists and critical scholars from different fields to engage in collaborative reflection on the present and future of human neuroscience in its dynamic socio-cultural surroundings. The chapter is a moderately revised and updated version of the foundational chapter 1 of our volume *Critical Neuroscience: A Handbook of the Social and Cultural Contexts of Neuroscience* (Chichester: Wiley-Blackwell 2012).[1]

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The label 'critical neuroscience' captures a productive tension. The title represents the need to respond to the impressive and at times troublesome surge of the neurosciences, without either celebrating them uncritically or condemning them wholesale. 'Critical' alludes, on the one hand, to the notion of 'crisis', understood—in the classical Greek, predominantly medical sense of the term—as an important juncture and point of intervention, and relatedly, to a task similar to that proposed by Kant in *The Conflict of the Faculties* (rather than in his more famous 'Critiques'), where he defends a space of unconstrained inquiry freed from the continual pressures put on scientific knowing by the vagaries of government and the political sphere at large (Kant 1992). In Kant's perspective, this opens up a space for inquiry that is itself inherently and self-consciously political, insofar as no allegiances to any pre-established orders of power will detract from reason's—and by the same token, free citizen's—self-determination. Fending off the multifarious forces that threaten to shrink or impede this unconstrained public sphere remains a perpetual task, one that gets specifically vexing when some of the toxic constraints imposed upon it are issued by institutional science itself. That is, when scientists—or, more often, their institutional advocates, sponsors, pundits—resort to dogmatism, positional authority or blatant self-promotion. On the other hand, the concept of 'critique' raises important associations with Frankfurt School critical theory. While critical neuroscience does not directly adhere to a 'Frankfurt School programme', nor to the rather swift identification of most work in natural science with positivism espoused by early critical theory, it does share with it a spirit of historico-political mission. This mission chiefly revolves around the persuasion that scientific inquiry into human reality tends to mobilise specific values and often works in the service of interests that often come to shape construals of nature or 'naturalness'. These notions of nature or of what counts as natural, whether referring to constructs of gender, emotion, mental disorder, normal brain development or other key human traits or capacities, require unpacking. Without reflective scrutiny, they can appear as inevitable givens, universal and below history, and are often seen as a form of 'normative facticity', presumably imposing specific demands on us and our conduct in everyday life (Hartmann 2012).

In the following, we will spell out how our proposal for a critical neuroscience is not motivated by the aim to undermine the epistemological validity of neuroscience or debunk its motives, nor is it simply an opportunity to establish yet another neuro-prefixed discipline. Situated between neuroscience and the human sciences, critical neuroscience uses an historical sensibility to analyse the claim that we are in the throes of a 'neuro-revolution' since the beginning of the Decade of the Brain in 1990. It investigates sociologically the

motivations and the implications of the turn to the *neuro-* in disciplines and practices ranging from psychiatry and anthropology to educational policy, and it examines ethnographically the operationalisation of various categories in the laboratory. Investigating the historical and cultural contingencies of these neuroscientific categories, critical neuroscience aims to analyse the ways in which, and conditions through which, behaviours and categories of people are 'neuro-naturalised'. It also traces how putative 'brain facts' are appropriated in various domains in society, starting with medicalised contexts of the West, but also with an eye to cross-national comparative analysis to understand the production and circulation of neuroscientific knowledge globally. Maintaining close engagement with neuroscience is on the one hand crucial for building accurately informed analyses of the societal implications of neuroscience, whilst on the other hand, providing a connection, a reflexive interface, through which historical, anthropological, philosophical and sociological analysis can feed back into research practice and provide critical as well as creative potential for experimental research in the laboratory.[2]

In attempting to build up a picture of what critique might look like for this project, we avail ourselves of a number of disciplines and sensibilities that can contribute as resources for critique. Our goal is to render critique amenable to a number of diverse disciplines and scholarly outlooks. This versatile set of tools can contribute to reviving a critical spirit while also broadening the neuroscientist's gaze. That being said, we do not intend to outline a fully fledged programme or recipe for critique. Instead, we will sketch building blocks for a mode of engagement, an ethos, that aims to raise awareness of the factors that come together to stabilise scientific worldviews that create the impression of their inevitability.

To bear relevance outside the narrow scholarly sphere, such an endeavour requires a self-reflexive hermeneutics that is necessarily multi-dimensional, even 'undisciplined'. The result, we envisage, will not so much be an unpacking of the black boxes of the neurosciences as an assemblage of resources that ultimately widens the ontological landscape of the diverse and varied behavioural and social phenomena under study. It is the plurality—reflecting the complexity of behaviour as well as the many contingencies of neuroscience—of elements of this landscape that gives rise to the solidity of a claim, the 'realness' of a fact. Contextualising neuroscientific objects of inquiry—whether the 'neural basis' of addiction, depression, sociality, lying or adolescent behaviours—can, in this way, demonstrate how such findings, whilst capturing an aspect of behaviour in the world, are also held in place by a vast number of contributing factors, co-produced by a collection of circumstances, social interests and institutions (Hacking 1999; Young 1995). These circumstances

and interests are often quite systematically ignored in neuro-discourse (see, e.g. Heinemann and Heinemann 2010; Weisberg et al. 2008).

However, we propose that critical neuroscience should not stop at description and complexification. Indeed, we share a sense of uneasiness, repeatedly voiced within the field of Science and Technology Studies (STS) in particular (Anderson 2009; Cooter and Stein 2010; Forman 2010; Mirowski 2011) about depoliticalisation of scholarship in the face of the increasing commercialisation of academia. In line with a broader cultural tendency favouring voluntarist conceptions of the 'entrepreneurial self', centred around ideas of 'resources' and personal 'capital' (social, emotional, 'mental'), there is a notable correspondence, sometimes quite open, sometimes in the form of less obvious complicities, between scholarly discourse and economic imperatives and normative schemas.[3] We share the conviction that a more radical and openly political positioning is needed in face of these trends. In the first instance, it is important to reinvigorate a sense of the impact that broader social, political and economic dynamics have on the very shape of academic and scientific culture.

Assemblage: The Thickening of Brain-Based Phenomena

Bruno Latour, in his essay about critique and its effect of *weakening* scientific facts, appeals to his critically oriented readers to 'suspend the blow of the [critical] hammer' and calls for a renewal of a realist attitude oriented to matters of concern, rather than matters of fact (Latour 2004). Matters of concern are those around which the human world revolves: they enthral, involve, challenge us to embrace or oppose them—matters of concern are focal points in practices, discourses, disputes. Critical neuroscience shares this constructive spirit, the 'stubbornly realist attitude' and the focus on what matters in relation to scientific practices (Rouse 2002). Critical neuroscience embraces the added dimension that enters the scene with the focus on matters of concern: values, conflicting moral and political outlooks and evaluative perspectives, changes in the attribution of relevance pertaining to a given phenomenon or scientific result, often contested among affected parties. There is no layer of scientifically accessible reality that is beyond or beneath this messy entanglement with concerns, values, interests and the conflicts between such rivalling evaluative outlooks. With this orientation, critical neuroscience specifically emphasises the politics implicit in scientific practices (see Rouse 1996; Haraway 1997).

However, while Latour and other champions of the by now fashionable anti-critique stance deem themselves non-dogmatic and quasi-democratic in giving a voice to participants in practices—both human and non-human—in the process of assembling their formations and collectives (instead of silencing the actors behind grand-scale theoretical assumptions), they relinquish too much. An overly celebratory or idly metaphysical gabbling too easily results from the sidelining of any non-local invocation of the social, the economic or the political. By contrast, our proposal for critical neuroscience calls for a less detached attitude on the part of the critical investigator, a more active engagement and, at times, a more confrontational response in cases of violation of scientific standards (Fine 2010; Jordan-Young 2010), strategies of ignorance (McGoey 2009), imperialistic export of Western assumptions to non-Western contexts (Watters 2010) or the political use of preliminary data (Choudhury et al. 2010). Such responses need to be supported by attempts to identify and render explicit more subtle biases and frames of evaluation: the specific organisation of public attention, patterns of distribution of affective energies, collectively sustained valuations and schemes of judgement that are instituted in subtle but pervasive ways in both scientific and popular discourses, in representations of scientific results, but also in spheres of public understanding at some distance from the practice of research. Notions such as the neural basis of adolescent risk taking, hard-wired sex differences, molecularised understandings of mental illnesses, or narratives about behavioural and emotive tendencies universally present in humans and 'set in stone' by evolution are cases in point. Some of these narrative patterns solidify to form what Judith Butler has called 'frames'—powerful yet unnoticed ways in which perception, knowledge and normative judgement are tacitly pre-organised so that some conceptualisations and evaluations are made likely while others are ruled out a priori (Butler 2009). Critique here has the task of working against deeply engrained habits of perception, thought and judgement, against pervasive orders of the sensual and the sensible, in order to enable and actively promote alternative framings of matters of concern.

What we envisage as the practice of critique, therefore, may well start with the critical yet constructive activity of assembling (Latour 2004, 246). Building on Latour's understanding of the term, 'assembling' in our usage refers to the collection of material from multiple sources and perspectives in order to enrich scientific conceptualisation as well as the broader intellectual horizon in which problems and issues are framed for empirical investigation and interpretation. Objects of neuroscientific investigation can, as a result, be situated in the full fabric of meaningful relations—while this very fabric is itself placed under scrutiny and has to be kept open for contestation. The

social situatedness, cultural meanings and various interests of affected groups all package the ontological landscape of neurocognitive phenomena. This view holds that what we see in the brain is at any time held in place by a rich web of factors within the epistemic culture (Knorr-Cetina 1999; Young 1995), and in the ambient society, which in turn mobilises these findings beyond the laboratory. Insights from multiple disciplines can bring to light the internalised scientific ideals, or 'epistemic virtues' (Daston and Galison 2007) that direct the formulation of neuroscientific findings—the filtering of information, the criteria for, and goal of, objectivity and the operationalisation of chosen aspects of the lifeworld. This encompassing embeddedness of neuroscience's objects of study is now increasingly acknowledged in various so-called *biosocial* research perspectives, which treat what formerly were thought of as separate biological and social dimensions as inextricable (see Meloni et al. 2016; Meloni 2014).

To illustrate this by way of an example, let us briefly venture into the case of addiction and addiction research. Addiction is increasingly understood as a disease of the brain, in which addictive substances cause malfunction of the frontal regulation of the limbic system, thus 'hijack[ing] the brain's reward system' (Leshner 2001) and potentially even altering gene expression (Kuhar 2010). The goal of these brain-centred approaches to addiction is to locate candidate molecular mechanisms that can lead to effective new treatments (Hyman and Malenka 2001). While these studies have yielded some notable findings, addiction is far more than (and different from) a mere change in brain chemistry. 'Addiction' denotes a family of conditions that are inextricably tied up with social environments, drug markets and cultural triggers (Campbell 2010), and depend on collectively developed and sustained habits and also upon institutional practices that emerge in response, as a feedback, to the original phenomenon—through classificatory looping as described by Ian Hacking (1999).

Approaching addiction using an ecological systems view, through multiple epistemic cultures, would mean to re-inscribe and integrate these multiple causal factors. Such an approach examines the linkages across levels of description using various methodologies and includes recording the cultural phenomenology of addictive behaviours. Additionally, it attends to the political economy of addiction and the effects of industry on concepts of addiction. And it strives to include the perspectives of those most directly concerned so as to work towards emancipatory and empowering strategies in facing the challenges that addiction poses. Taken together, this integrative approach will yield an explanandum much richer than any of the narrow construals developed exclusively from a single scientific or medical

perspective. Clearly both registers—social and biological—are necessary to assemble a richer and also sufficiently dynamic understanding of addiction. The more relevant questions for a critical neuroscience to work out will be how to overcome the gap between social and neural, how to develop conceptual vocabularies and frameworks that overcome this distinction and how to empirically study phenomena like addiction with a view of the *situated* brain and nervous system and including the personal perspective and experience of those concerned.[4]

How Does the Social Get Under the Skin?

Ethnographic work by Margaret Lock has provided powerful evidence for the need to collapse conventional dichotomies between the 'inside' and 'outside' of the human body. Her seminal study of the experience and physiological characteristics of menopause among Japanese and American women led her to the concept of 'local biologies', a useful way to denote her finding that social context and culture can refashion human biology (Lock 1993; Lock and Kaufert 2001; Lock and Nguyen 2010, ch. 4). Lock found that the cultural differences in menopause/konenki ran deep, manifesting on biological, psychological and social levels. She argued that the different experiences of hot flushes were not simply due to differences in cultural expectations in relation to the body, but down to the biological effects of culturally determined behaviours such as diet. This finding challenges the tendency in biological science to draw boundaries at the skin, and demonstrates instead the ongoing dialectic between biology and culture. Laurence Kirmayer has extended these ideas to the brain and behaviour through his concept of 'cultural biology', which understands culture as a biological category in the sense that human beings have evolved a 'biological preparedness to acquire culture … through various forms of learning and … neural machinery' (Kirmayer 2006, 130). Lock and Kirmayer's concepts of 'local biologies' and 'cultural biologies', respectively, capture a notion of central importance to critical neuroscience: biology and culture are mutually constraining and dynamically co-constitutive, such that they are each conditions of the other's determination and development.

Explanations that situate brain and cognitive function within the social and cultural environment of the person are, in fact, increasingly encouraged within psychiatry and neuroscience. Calls for interdisciplinary research that lead to integrative explanations are certainly heard within psychiatry as a route to developing multi-level theories of disease and their aetiologies (Kendler 2008). Advances in epigenetics have been especially influential in fuelling

major shifts in scientific thinking about the linkages between the body and its environment, between soma and society. Research on epigenetics has begun to reveal how interactions between the genome and the environment over the course of development lead to structural changes in the methylation patterns of DNA that regulate cellular function. There is compelling evidence, for example, that early parenting experiences and social adversity alter the regulation of stress response systems for the life of the organism (Meaney and Szyf 2005). Such studies provide biological evidence that lived experience, developmental histories, dynamic interactions and cultural contexts are all fundamentally bound up with biological processes as 'low level' as gene expression.

In parallel to these developments in genomics, social and cultural neuroscience have become the most rapidly developing areas of cognitive neuroscience. These research fields posit that the human brain is fundamentally a social brain, adapted for social learning, interaction and the transmission of culture (Frith and Frith 2010; Rizzolatti and Craighero 2004). Moreover, its structural malleability is understood to be experience dependent and long lasting. Evidence of genomic and neural plasticity thus forces scientists to rethink the primacy given to biophysical levels of explanations and challenges us to destabilise the dichotomy of nature/culture and instead address the fundamental interaction of mind, body and society.

This concept of the situated brain brings up a number of possibilities and challenges for critical neuroscience. First of all, it requires the critic to act as a *bricoleur*, collecting data at a number of different levels, layering phenomena, such as menopause or addiction, with these different strands of inquiry that ultimately serve to enrich one another in their explanatory value. Secondly, the emerging discourses of 'interaction' require critical analysis by sociologists and anthropologists of science. How exactly are aspects of social life, culture and individual difference incorporated into scientific observations and methodologies? Furthermore, when the environment and biology are each assigned roles in the development of pathologies such as schizophrenia or antisocial behaviour, how are the social and cultural realms made concretely relevant or rendered visible in medical explanations? How might the more complex ontologies of mental disorders that result from these integrative explanations bring about new ethical and political challenges by opening up new spaces of intervention or creating new 'at risk' populations (Pickersgill 2009; Rose 2010; Singh and Rose 2009)?

Situating the brain and behaviour in social and cultural contexts also underscores the importance of examining recursive loops between neurobiological and social/cultural processes such as the way in which explanatory

theories of illness and behaviour interact with the physiological processes involved. This 'bio-looping' as discussed by Ian Hacking and others refers to the ways that both culture and local biologies can transform one another, exerting their influence on the way we understand ourselves, the way we experience mental and bodily phenomena and the way that this in turn shapes the corresponding biological processes (see Choudhury and Slaby 2012; Seligman, *this volume*). We return to these issues later in a discussion of what critical neuroscience can do for neuroscience itself.

Re-invoking the Social in Studies of Neuroscience

Openly politicised forms of critique are no longer much in evidence, be it in STS or more broadly, and may not currently seem very workable. Prevalent, for example, in science studies and cultural studies are approaches that appear to trade in critical engagement for an aestheticisation of scientific practices, stopping short of penetrating into manifestly pathological developments. One reason for this may be the increasing professionalisation and differentiation of various metascientific approaches over the past 40 or so years: are practitioners no longer 'allowed' to operate on a broader, holistic level of social understanding that transcends clearly circumscribed local expertise?[5] It is likely that certain intellectual as well as political and economic developments support some of this academic quietism (Forman 2010).

In opposition to these tendencies, critical neuroscience strives to regain room for scrutiny, in reckoning with perspective-bound and interest-specific constraints that belie, in some contexts at least, objectivist aspirations of neuroscience and of those enthusiastic about its applicability in everyday life. Certainly, the gathering of context in many cases may end up laying bare the economic and political imperatives that sustain particular styles of thought from 'screening and intervening' to 'essential differences' (Abi-Rached and Rose 2010; Fine 2010; Jordan-Young 2010). It may also end up shedding light on the ways in which the very concepts and categories that produce new kinds of responsibility towards the 'natural' make-up of our minds are—knowingly or unknowingly—themselves shot through with our projections, and give rise to 'facts', worldviews and policies that may collude with social and political orders (Hartmann 2012; Malabou 2008). This is well illustrated by Cordelia Fine's study *Delusions of Gender*. Fine, trained both as a cognitive neuroscientist and a science journalist, rigorously analyses neuroscience experiments, their results and their interpretations among media exegetes, that purport to show hard-wired differences in behaviour between men and

women. She demonstrates with much technical insight how biases creep into the assumptions involved in experimental paradigms, and how cultural stereotypes are reified by 'brain facts', amounting to a form of neurosexism (Fine 2010; cf. Jordan-Young 2010).

As variously indicated above, critical neuroscience puts particular emphasis on the social. It is important not to take 'the social' as a static, homogenous formation, but rather to work with this notion as a variable proxy for the complex associations between scientists, laboratories, media, agencies, governments and other constituencies. Scientific knowledge as such can be viewed as embodied in material alliances or what Rouse, alluding to Wartenburg's conception of socially distributed power, has called 'epistemic alignments' (Rouse 1996; Wartenburg 1990). Effective knowledge only exists in concrete material-practical interactions between people, things, instruments, agencies and policies, and thus cannot be understood in abstraction from 'the various kinds of resistance posed by anomalies, inconsistencies, disagreements and inadequacies of skill, technique, and resources' (Rouse 1996, 194).

While no grand-scale invocations of 'social factors' can substitute for precise analyses of particular interactions and alignments between social actors and material actants, it is important to keep the bigger picture in view. It is here that we diverge from the localism of actor-network theory and the STS mainstream: epistemic and political alliances, as well as cognitive and affective frames and interpretive schemes instituted by them, often operative through media representations or discursive practices that begin in local settings and are subsequently broadened, all contribute to a structure of secondary objectivity or 'second nature'. These processes of solidification can easily escape the purview of science and its commentators because of the incremental nature and slow timescales of change and because of the authoritative nature of the finished product: established, official, institutional knowledge—that which gets variously coded in prevailing discursive formations and is disseminated via official channels of institutional PR and leading media.

The 'social' needs to be viewed not as an assumed explanatory factor but as the result of various micro- and meso-level operations and alignments between a wealth of actors, tools, quasi-objects and agencies. In turn, the social re-emerges as a potential explanatory resource, for example, in the mobilisation and distribution of attention, of concern and relevance and in the workings of tacit schemes of interpretation and normative judgement (Butler 2009). In light of this, it is not enough to merely point to ontological hybridisation or celebrate one's having superseded modernist dualisms (Latour 2005). Neither does it suffice, for our purpose, to merely neutrally chart cartographies of 'emergent forms of life'—such as biological citizenship and neurochemical

selfhood—nor simply to leave it upon others to 'judge' these developments (Rose 2007, 259).[6]

While such descriptive endeavours provide important staging for subsequent analysis, it is crucial to penetrate beneath the surface of emerging practices, relations and styles into the dynamics of power that may shape or stabilise surface phenomena, facilitate or hinder effective alliances or actions. It is important to reckon with pathological developments and render explicit interest-driven biases, hegemonic schemes of judging, templates of knowing and classifying, dangerous blind spots in interpretations, unquestioned narrative patterns and various unholy material alliances.[7] For example, the neoliberal mobilisation of human resources in the name of employability, flexibility and 'soft skills' has found a new space to take shape among neuroscientists performing the naturalisation of social/economic categories, and increasingly biologised notions of personhood, human experience and the good life (cf. Malabou 2008). Subjectivity is parsed from the outset into economic categories and becomes a type of bio-economic 'capital' that is in turn used to sort people into kinds, construct risk profiles and suggest enrolment in enhancement programmes (Fricke and Choudhury 2011).[8]

In light of this, we argue that critical neuroscience must ask hard questions about conceptual and normative assumptions and strategic alliances and work towards re-opening contestations and restaging alternative interpretations and evaluations.[9]

Structural Pathologies in Science and Society

The activity of assemblage, in our sense of the term, is thus an inherently political one. It allows the critic to identify what Axel Honneth has called 'social pathologies of reason' (Honneth 2009, ch. 2)[10]: defects or malfunctions in social systems, practices and institutions—malfunctions that come into view against the background of a normative understanding of society and the purposes of functioning institutions. In the case example of addiction, described earlier, one might come to reckon with diverging perspectives from medical professionals, pharmaceutical companies, health administrators, social workers, governments and political parties, the education sector, newly constituted 'risk populations' and certainly 'the addicts' themselves. However, 'addict'—and similarly, other kind terms in use in neuroscientific research—must be seen as a category that is co-produced through dominant classifications, styles of thought and cultural practices. Incisive analysis of the interactions which make possible these neurological categories give ground

for active assertions about what is at stake, in the case of 'brain overclaim' or tangible corporate influences on scientific practice.

For example, as Laurence Kirmayer and Ian Gold (2012) argue, there is a trend in mainstream Western psychiatry to employ increasingly narrow construals of mental suffering that neglect the situatedness of patients in distorted social environments and direct the focus away from cultural embeddedness—including politically problematic societal arrangements—towards assumed 'neurological underpinnings' of illness, agency and personhood. Ignoring the social and cultural contexts of phenomena under investigation can render neuroscientific research complicit with problematic developments in the medical sector, despite the best intentions of many individual practitioners. Scientists are not usually trained to be very sensitive to the subtleties of, and social conflicts within, political and institutional environments. This can lead to distorted interpretations of experimental results—with very real consequences in the lives and treatment choices of patients. Continuing the above example of addiction research, a narrowly neuroscientific understanding of substance addiction might lead to the neglect of the conditions that stabilise addictive behaviour and thus encourage forms of practice and treatment less conducive to the well-being of those affected than those that become available through a more complex understanding of the condition. Moreover, such narrow explanations fail to acknowledge the role of politics, social engineering and economic pressures in addiction and other forms of human suffering.

Intensified media representation coupled with audiences increasingly trained, through continuous exposure, to be receptive to easy-to-digest narratives of self-objectification contributes to the distorted images of the person—as lacking in free will, possessing skewed decision-making powers, being driven instead by automatised emotions and thus as not genuinely responsible for their acts (while simultaneously making them responsible for 'managing' their brains). Media reporting in this manner can lead to a climate of opinion that singles out sensationalistic themes, often ideologically laden, and pushes towards simplified, technocratic solutions to social problems. Critical neuroscience aims to function as an informed voice opposing those distorted images. Importantly, Fine's critique of neurosexism mentioned earlier is made particularly strong by her close engagement with the experimental design and statistics as well as her skill to write compellingly for a broader audience. Given that the flawed findings she critiques have travelled into the popular cultural script of male/female differences, critical writing for a public audience is a vital skill of immense value within the toolbox of critical neuroscience.

Whose Norms? Expertise, Participation and Contestation

The goal to scrutinise and lay bare scientific conventions that are taken for granted, tacit knowledge, vested interests at work in neuroscience research or their impacts on people, opens up complex questions about norms. In order to identify social pathologies or general 'system malfunctions', any critical endeavour inevitably operates in a normative space, reflecting particular assumptions about the conditions for both social organisation and individual well-being. Theorists cannot remain neutral but have to stake their particular political orientation—but it is crucial to see that there is no alternative to a necessarily partial, situated, specifically committed stance in the midst of the practices and developments under study. There is no neutral vantage point, no 'god's eye view' (Rouse 1996; Haraway 1997). What we deem 'pathological' at a given juncture depends on a contrast with nontrivial ideas of a non-pathological alternative. Where individual subjects are concerned, this calls for the articulation of a situated, conceptually thick orientation towards an image of the good life. Accordingly, no version of a critical neuroscience can simply impose a set of normative standards or values. The critic's task rather is to render the implicit norms of a given social domain or lifeworld segment explicit, point to possible tensions between different normative outlooks and, where necessary, measure institutional realities against the normative assumptions that legitimate them, yet without recourse to a fictional vantage point of neutrality above the fray of situated practice. This raises questions of power, the constructions of expertise, the social distribution of knowledge, and the possibilities for participation in decision-making processes—questions that have to be confronted from within the practical domains in which they arise, and by all those whose well-being, flourishing and political agency is concretely at stake.

The last few years have seen a steep increase in numerous forms of popularisation of neuroscience. Driven by various parties, including neuroscientists, funding agencies and the media, public engagement in neuroscience has emerged in the form of outreach projects, popular science writing and—not least—as interactive neuroscience exhibitions geared towards a range of audiences, with the aim of informing and engaging the lay citizen. If critical neuroscience advocates informed participation in the scientific process, then it will need to confront questions about representation, expertise and agency of citizens, particularly in information societies characterised by a more demanding and active citizenry (Beck 1997). There is no doubt that efforts to

'democratise' scientific processes this way pose difficulties. With hindsight, earlier optimism about the potential of a renewed politicisation of society around issues of science and technology seems to have been premature (Kerr and Cunningham-Burley 2000).[11] Rather than an emerging 'sub politics' (Beck 1997)—grass root political engagement that responds to hazards of scientific and technological development—we increasingly witness restricted expert circles monopolising the negotiation and regulation of relevant issues (cf. Mirowski 2011).

One way for critical neuroscience to attempt to establish (or challenge) normative conceptions—themselves always necessarily under reflexive scrutiny—is by creating a discursive space for debate both in professional and practical domains about the categories and applications of neuroscience, and about related social issues such as the organisation of labour, conception of health and disease, goals and practices in parenting and education, issues about law and punishment, technological self-optimisation and much more. In order to make this move however, it needs to probe critically at ways in which the choices and views of the public are regulated, particularly amidst the growing clamour for 'neurotalk' in public spheres (Illes et al. 2010). Expert counselling and state-run programmes of screening and risk assessment (Rose 2010) and the instant professional take-up of ethical concerns into an institutionalised 'neuroethics' (de Vries 2007) increasingly occupy the space for public engagement. In what ways might the space for 'science in society' or 'neuroethics' experts, as well as the domains of psychiatrists, doctors and educators (connected to government, funders or companies), act as intermediaries in aligning public opinions with scientific agendas, ratifying or legitimating neuroscientific research programmes? Who can legitimately make knowable what the 'public' wants or thinks about neuroscience and its applications? How can participatory approaches avoid opening up new forms of stratification?

With such problems in mind, critical neuroscience aspires to open up discursive spaces that facilitate debate among practitioners, 'stakeholders' and lay citizens about the goals, concerns, and normative standards that society wants its science to pursue or live up to: where the work of the critic involves not merely encouraging the accessible promotion of new ideas from neuroscience, but invites plural viewpoints and promulgates a degree of critical rigour through provocation—that is, by illuminating blind spots and by questioning assumptions. It is vital that public neuroscientists conceive of audiences not as listeners or viewers but as potential speakers. It is at these sites of contestation that specific normative issues surrounding scientific matters of concern can emerge and take shape. This process pushes science beyond reliable knowl-

edge—subject only to validation within its own disciplinary context—to the production of 'socially robust knowledge'; that is, knowledge tested for validity both outside and inside the lab, developed through the involvement of socially distributed experts including those from different disciplinary and experiential backgrounds within and outside of academia (Nowotny 2003). While the embeddedness in society and the iterative process of open contestation may render this knowledge more robust, the means of such forms of polycentric knowledge production in neuroscience must be carefully worked out (Jasanoff 2003).

A model of 'public' neuroscience such as this faces challenges within the changing structure of the university and changes in the organisation and funding of professional research. Both are increasingly oriented towards a corporate, neoliberal management model (Giroux 2007; Mirowski 2011). How can critical neuroscience reach its goals in a system that places its values on outcomes and efficiency, that increasingly fosters commercialisable or applied research and encourages corporate influences in the form of sponsorship, company spin-offs, profitable patents and institutional joint ventures?

There are trends pulling neuroscience in different directions, certainly not all negative. The ambivalence of the situation can be illustrated by reference to 'interdisciplinarity' (a term that has become a powerful buzzword, notably in neuroscience). Successful integration of distinct perspectives and methodological approaches can lead to unforeseen benefits and novel insights. However, genuine inter-, trans- and post-disciplinary research is constantly forced to acknowledge, and to work with, tensions between ontological and epistemological frameworks, and is thus necessarily slow, compared to conventional single-discipline research processes.[12] In order to enable a reflexive ethos, and to keep open a space for critical inquiry in a context that favours 'outcomes' in terms of revenues and commodities, critical neuroscience will need to continue discussing structural transformations, and challenging the increasing dominance of market orientation in academia.

What Difference Can Critique Make to Neuroscience?

The metaphor of the *looping journey*—of that which is taken to be a 'brain fact'—can help to operationalise critique, opening up possibilities for thickening, or assembling, a given, brain-based phenomenon. Whether we focus on the neural basis of addiction, depression, adolescence, culture, gender,

morality or violence, the journey can be traced using multiple methodologies, from the point of a theme's entry into—and treatment in—the lab, through various technical and knowledge practices, to the interaction with the media and policy, to its reception by the public. What we mean by a 'brain fact' is not a thing-in-itself, but a specifically conceptualised phenomenon or 'local resistance' that emerges from the collective practices and directed cognition of neuroscientists working in a community at a given time and in a given context (see Choudhury et al. 2009).[13]

With this in mind, it is important to ask what difference do second-order observations of laboratory conditions, communities of scientists and historical and cultural contingencies make to neuroscientists themselves, whose goal is to develop and test paradigms that ultimately contribute to mapping social, cultural or perceptual processes on particular brain regions. Critical neuroscience renews the possibility for critical commentators to be engaged with, rather than estranged from, laboratory science. Functioning through the collaboration of work from multiple methodologies, it aims to find entry points for social theory, ethnography, philosophy and history of science, in the laboratory. In the following, we put forward ways in which the latter fields can play a contributory role in both the *practice* of neuroscience in the lab and in the ways in which neuroscience is constellated into broader socio-cultural formations and practices beyond the lab.

From educational initiatives for junior-level researchers to the development of collaborative working groups investigating behavioural phenomena from different disciplinary perspectives, critical neuroscience explores whether an ethos of reflexivity can, through interdisciplinary training, be inscribed into experimental practice. The aim here is not to conduct a purer or 'better' neuroscience. Instead, reflective practice includes social and historical contextualisation and cross-cultural comparison of behavioural phenomena, within neuroscience. Examining these contingencies will generate alternative possibilities for findings in neuroscience, which on the one hand opens up interesting empirical questions for neuroscientists, and on the other hand, functions as a form of critique from within.

How should we conceive of the relationship between first-order (descriptions of brain and behaviour) and second-order (descriptions of neuroscientists observing behaviour) observations (Choudhury et al. 2009; Roepstorff 2004; Fitzgerald and Callard 2015)? We believe engagement between these socio-cultural and historical studies and experimental neuroscience can be constructive in a number of ways:

(1) Demonstration of alternative possibilities of results of neuroscience experiments by modifying technical parameters or comparing and re(de) fining categories[14];
(2) Exploring routes to empirically investigate social and cultural phenomena without assuming universal neural mechanisms from the outset;
(3) Enriching behavioural theories by allowing for pluralistic viewpoints and methodologies to result in layered explanations of complex phenomena;
(4) Examining the subtle relationship and feedback loops between popular opinion or ideologies about the brain and findings in neuroscience.

Such goals can only be realistically achieved through collaborative work. Working groups, as initiated since the emergence of critical neuroscience, consist of the following.

Sociologists of science who observe communities of scientists and capture the thought styles that govern their cognition in studying the particular phenomenon in the lab (Fleck 1935/1979). Fleck described the 'tenacity' of systems of thought that govern scientific practices and explanatory styles, and that ultimately give rise to what from then on will count as fact. What solidifies a local resistance into a recognised 'fact'? By studying the journey of a phenomenon in and around the neuroscience lab, we can study how the methods, concepts and theories involved in the development of a fact of neuroscience may be culturally conditioned; in addition we can identify the refractory effects of the thought collective that sustain it and the wider culture in which it functions (cf. Dumit 2004, 2012). Neuroscientists are working at a time of unprecedented politicisation through the commercialisation of research (Wise 2006), and sociological analysis can highlight the pressures that commercial, pharmaceutical, and military interests place on neuroscience (Healy 2004; Moreno 2006). Moreover, sociologists can begin to draw cross-national comparisons of the social structures of neuroscience. Comparing the international contexts of trends in neuroscience research and its representation will help to spell out the logic of the neuro-industry, that is, the institutional, historical, political and ideological planes in which the rapid developments, the allure and the influence on cultural formulations and other academic disciplines take place, over and above the events within neuroscience per se (cf. Rose and Abi-Rached 2013).

Philosophy contributes the analysis of central phenomena under investigation (and their different, often competing, conceptualisations), for example, emotions or moral decisions. It also serves to clarify the content and status of notions such as determinism, reductionism, specificity and consilience—concepts that have been floated in neuroscience and its critiques for a while, and

require sharpening. Often, these and other concepts play key roles in what Hartmann (2012) calls the 'hidden hermeneutics' of the neurosciences: structural narratives that practitioners routinely employ as they describe their objects of investigation and construct interpretations of data, but that are rarely reflected upon explicitly. Ideas about 'cerebral subjectivity' (Vidal 2009) or the ubiquitous but often vague appeals to evolutionary theory are good examples (Richardson 2007); similarly the hype around the notion of cerebral plasticity (Malabou 2008), or heart-warming yet factually shallow stories about empathy, affective contagion and the social brain (Young 2012).

The task here is to elucidate a specific meta level: ascending from the manifest contents of theories, explanatory frameworks and core concepts in current neuroscience to the analysis of latent assumptions and formative backgrounds, such as the implicit construal of the brain as the stable ontological foundation of both personal traits and social and cultural phenomena (Slaby and Gallagher 2015), or the complicity of neuroscience-backed construals of human subjectivity with capital-driven appropriations of health, self-care or the 'neo-social' optimisation of human conduct (Slaby 2015). Philosophy might also contribute to enriching the description of phenomena under study through phenomenological investigations, for instance, by performing what has been called 'front-loaded phenomenology' (Gallagher 2003; Gallagher and Zahavi 2008; Ratcliffe 2008).[15]

Cognitive neuroscientists contribute to technical and conceptual analysis of research processes, including methodological assessments. What are the potentials and limits of specific methodologies or tools, such as fMRI and the associated statistical methods, and to what extent are these clear or made clear in different venues (Logothetis 2008; Vul et al. 2009; Gonzalez-Castillo et al. 2012; Miller et al. 2012; Stelzer et al. 2014)? Here, we have obviously seen a massive increase in the level of critical awareness and readiness to confront these issues within neuroscience over recent years.[16] Much work remains to be done, however. For instance, how are cultural, psychological, functional and genetic models of cognitive phenomena mapped onto each other (Turner 2012)? Which principles shall guide the determination of significance in neuroanatomy, that is, what standards to apply in parsing the brain into 'regions' (Haueis 2014)? Once a phenomenon enters the neuroscience lab, how do scientists break down the phenomenon into constituents that they are able to study within the constraints of their methodology (Dumit 2004, 2012)? What efforts are involved in setting up experimental apparatuses and stabilising the phenomena under study? How are the results analysed and evaluated in comparison to other data from different experiments? How can data—

quantitative and qualitative—from social science and humanities disciplines be brought to bear on the neurobiological results?

Cultural or medical anthropologists will draw on ethnographic field work to develop cross-cultural comparisons of behavioural phenomena or symptoms and experimental paradigms (tasks, questionnaires) that have largely been studied on—or standardised using—particular groups of subjects deemed to represent the 'norm' (Henrich et al. 2010).[17] Critical neuroscience draws on medical anthropology to supplement findings of neural correlates with phenomenological insights, biographical accounts of the person and the *meaning*—that is, the social, cultural, moral or spiritual significances—of behavioural phenomena, including mental illness and interventions (Cohn 2012). Critical neuroscience resonates with cultural psychiatry, in emphasising that the allegedly most 'fundamental' level, using neuroscience in its current form, is not necessarily the most appropriate either for explaining or intervening in psychopathology. While neuroscientists and medical practitioners increasingly invoke the use of neuroscience in psychiatric nosology and clinical practice (Hyman 2007; Insel and Quirion 2005), critical neuroscience works towards ways to understand how 'meaning and mechanism' intersect via the brain (Choudhury and Kirmayer 2009; Seligman and Kirmayer 2008).

The biosocial subfields of social and cultural neuroscience have indeed begun to investigate how aspects of cultural background may influence cognition, such as the expression and regulation of emotions and understanding of others (Chiao et al. 2008; Zhu et al. 2007). As this area of neuroscience burgeons, critical neuroscience aspires to contribute to the conceptualisation of culture in experimental design and interpretation, to explore how environmental factors, including cultural practices, habits and understandings, interact with the development of structure and function of the healthy nervous system in such a way that several vocabularies of description—social, cultural, psychological and biological—can coexist (Kirmayer 2006; Lock and Nguyen 2010).

Historians of science trace historical trajectories of the conceptual construals, models and modelling practices, interpretive contexts and experimental set-ups common to contemporary neuroscience (Foucault 1973; Hacking 2002; Young 1995; Borck 2012). Historical analysis can thus show how particular problems such as the criminal brain, post-traumatic stress disorder, the risky teen or the empathic female become questions for the neurosciences, and how particular methodologies are valued over others as allegedly more objective. Critical neuroscience will yield important insights from the history of concepts, modelling practices and the various trajectories of objects of scientific inquiry, to understand how technologies, political and moral contexts converge to give rise to

diagnostic categories, how aspects of the self have come to be objectified and considered in certain contexts as clearly reducible to the brain (Vidal 2009) and how scientific objectivity itself developed—and changed significantly—as an epistemic virtue (Daston and Galison 2007). *Longue durée* analysis can additionally serve to interrogate the air of radical departure that surrounds much of the rhetoric around neuroscience (Borck and Hagner 2001). Unpacking these histories will help scholars and researchers gain distance from the inflated, spectacular and brain-centric rhetoric which parts of the neuro-industry seem to dictate (Casper 2014; Stadler 2012, 2014); at the same time, historians of science are well equipped to also come to critical terms with the broader significance of the neuro-turn, including its more indirect but no less pervasive impacts on scholarship and academic work at large (Cooter and Stein 2013).

Conclusion

We have sketched a broad picture of a critical neuroscience that probes the extent to which claims *about* neuroscience do in fact match neuroscience's real world effects. Our approach sets out to analyse the allure and functions of the *neuro* in the broader scheme of intellectual and political contexts including the rise in recent years, of a new (neuro) 'biologism' in many academic disciplines and popular culture at large. Our aim is to contribute these observations from the human sciences to neuroscience so as to demonstrate the contingencies of neuroscientific findings and to open up new experimental and interpretive possibilities.

Assembling and broadening ontological landscapes of behavioural phenomena requires researchers to move beyond the nature-nurture distinction when conceptualising phenomena such as addiction, adolescence, autism or depression. Instead, critical neuroscience will work with concepts such as 'cultural biology' and 'local biology' which bring to the fore the co-constitutive relationship between the brain and its context.[18] The 'endorphin-challenged alcoholic', the 'neurological adolescent' or the 'female brain' is richly situated and sustained in habitats made up of interactions between institutional, cultural and neuronal infrastructures. Such a framework poses intellectual challenges to cognitive and clinical neuroscience—challenges that must be taken up, especially as the notion of neuroplasticity or the field of cultural neuroscience open up potential to investigate brain-environment interactions. We emphasise the need to rethink the conception and location of these borderlines at the skull or the skin in a way that troubles the arbitrary distinctions and moves beyond biological determinism and social constructionism

(cf. Fitzgerald and Callard 2015; Pitts-Taylor 2016). If fMRI can show that cultural upbringing modulates brain activity or new biotechnologies permit us to tinker with the brain and cognition, it is apt for neuroscience to acknowledge that human brains are represented in terms of cultural categories and that brains also do 'cultural work' in distinguishing what is natural, who is healthy, different, normal, rational (Lock and Nguyen 2010).

In general, then, work collected under the umbrella of 'critical neuroscience' undertakes explorations of the discursive space that is opened up once the outworn distinctions and dualisms are surpassed, and once constructive interaction between practitioners from different methodological universes is enabled. The critical ethos we invoke, therefore, is not one that rejects but one that aims to elicit change: both in how significant phenomena are explored within neuroscience, and in how the social implications of neuroscience are analysed. The conceptual changes involved in studying the situated brain in its context, the pedagogical initiatives that bring multiple traditions of scholarship into contact and the calls for contestation in neuroscience funding and application, all disturb boundaries—between the brain and its environment, between disciplinary vocabularies and methodologies, and between science and society. These interruptions will provoke us to imagine the brain in different terms and to probe its functions in alternative ways. Such changes—towards which we sense an increasing openness among neuroscientists and social scientists alike—will open up potential for a more realistic picture of the function of neuroscience in society while simultaneously commenting on the broader socio-political changes in contemporary societies that impact its developments, for better or for worse.

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Notes

1. Updated and extended version of Chapter 1 in *Critical Neuroscience: A Handbook of the Social and Cultural Contexts of Neuroscience*, 2011, Wiley. We reproduce the material here with kind permission of the publisher, Wiley-Blackwell.
2. The more descriptive portions of this agenda overlap in part with the careful and competent work that Nikolas Rose and Joelle Abi-Rached have done in their seminal study *Neuro: The New Brain Sciences and the Management of the Mind* (Princeton UP 2013).

3. How these postmodernist tendencies might have rendered explanations that invoke 'social influences' less common and less valued in STS is helpfully discussed by Forman (2010).
4. This goal would take as a premise that the brain and nervous system are nested in the body and environment from the outset and that their functions can only be understood in terms of the social and cultural environment (Choudhury and Gold 2011). For the more general background to this perspective, see Protevi (2009).
5. This might be one reason why critique of scientific and medical malpractice and corporate influence has recently been more a business of journalists, popular writers and non-academic intellectuals than of professional STS practitioners (recent examples: Fine 2010; Greenberg 2010; Watters 2010).
6. We refer here to the puzzlingly moderate final remarks in Nikolas Rose's *The Politics of Life Itself* (and echoed again throughout Rose and Abi-Rached 2013). Rose's proclamation of neutrality at the end of that work is surprising in face of the many blatantly critique-worthy developments he had charted so rigorously throughout his book.
7. Here critical neuroscience preserves what could be called historical solidarity with the project of critical theory: the similarity lies in the attempt to move beyond sporadic interventions towards a theoretically integrated account of an assumed system of normative assumptions, interpretive patterns and material conditions that jointly stabilise, on the scale of society or significant segments of it, a tacitly pathological status quo. The term 'theory' in critical theory is no accident (Geuss 1981; Honneth 2009).
8. Take for example the UK Foresight Project's definition of 'well-being': 'Mental well-being, […], is a dynamic state that refers to an individual's ability to develop their potential, work productively and creatively, build strong and positive relationships with others and contribute to their community' (Beddington et al. 2008, 1057).
9. Besides Cooter and Stein's (2010) refreshingly explicit political positioning, we have been inspired by the rigorous critical and scholarly stance of historical of economics Philip Mirowski. Especially his paradigm historiography of cybernetic's influence on contemporary economics (2002) would deserve a separate discussion, as there is much overlap with the formative developments that have led to the present-day shape and impacts of the neuro-cognitive sciences.
10. We take up Honneth's notion in a rather loose manner, divorcing it from the specific context of a theory of rationality implicit in approaches to 'critique' from a Frankfurt School perspective.
11. The most optimistic voice in this area has been German sociologist Ulrich Beck (see, e.g. Beck 1997).

12. Since we first wrote this chapter, Des Fitzgerald and Felicity Callard have done excellent work on the prospects and pitfalls as well as conceptual and practical backgrounds of interdisciplinary cooperation between neuroscience and the humanities and social sciences. They also helpfully focus on the issue of experimentation (see Fitzgerald and Callard 2015).
13. We use the notion of a 'brain fact' analogously to Ludwik Fleck's conceptualisation of a scientific fact in his seminal study *Genesis and Development of a Scientific Fact* (see Fleck 1935/1979). On the looping journeys of scientific facts in the context of neuroscience see also Dumit (2004).
14. This is an example of how neuroscience itself can be used to subvert its own assumptions and demonstrate the contingencies of categories and methodologies it employs, a move we have called 'experimental irony'. Margulies (2012) illustrates the power of this strategy of critique 'from inside' through a review of the famous study by Bennett et al. (2009) that used a dead Atlantic salmon in an fMRI scanner to highlight the high possibility of red herrings in brain imaging research.
15. Of course, philosophy—as a specialised domain of *philosophy of science*—also contributes directly to the methodological reflection, analysis and critique of neuroscientific research practice (see, e.g. Klein 2010; Haueis 2014).
16. This is one of the areas where the situation has changed to a notable extent since we first articulated the programme of critical neuroscience, and since the first version of this chapter was published in 2012. In this respect, then, we have seen a considerable gain in self-reflective awareness as part of neuroscience's professional outlook. It is much harder to get methodologically suspect studies published these days then it was, say, 10 or 15 years ago, although many problems still remain. Stelzer et al. (2014) review a lot of the work that has been published around and after the time our first critical neuroscience publications were written.
17. In their landmark comparative article, Henrich et al. (2010) use the acronym WEIRD to denote the White, Educated, Industrialised, Rich and Democratic societies that behavioural science researchers take to be 'standard subjects', in spite of the considerable heterogeneity across populations taken to be groups, and in spite of the fact that so called WEIRD populations are frequently *unusual* or *outliers.*
18. Besides the increasingly prevalent understanding of 'biosociality' as a fertile perspective in various fields (Meloni et al. 2016), we also consider the more critical perspective of 'biocapital' as highly relevant here. Where the concept of biosociality operates on a fairly broad and neutral plane, 'biocapital' hints at the quite direct—and often problematic—*economic* appropriation of biological materials, biological knowledge and biology-informed ethical outlooks (see Sunder Rajan 2006; Cooper 2008).

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16

On the Neurodisciplines of Culture

Fernando Vidal and Francisco Ortega

The phenomenon variously known as "neural turn," "neuro-turn" or "neuroscientific turn" consists of the use of neuroscientific theories, methods, practices or idioms in a wide array of non-medical fields, in particular in the humanities, the social sciences and the sciences of culture (Littlefield and Johnson 2012). In these fields, the neuroscientific turn seems at first sight to partake in a broader late twentieth-century development where biology "becomes social" and the social, biological (Meloni 2013, 2014). In evolutionary theory and molecular epigenetics, the distinction of nature and nurture has dissolved, and environments and cultures have moved center stage. The neurosciences do not treat the brain as an isolated data processor but as an organ largely shaped by the external world it helps create. In practice, "neurocollaborations" between the human sciences and the brain sciences have been dominated by the latter and have inspired calls for new configurations

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that would be neither *inter* nor *trans*, but rather "entanglements" no longer inspired by the "fantasy of parity" among the disciplines (Fitzgerald and Callard 2014, 16; see also Callard and Fitzgerald 2015; Fitzgerald et al. 2014; Littlefield et al. 2014). Overall, however, the ideal of two-way collaboration remains the rule and inspires the "neurodisciplines" that, from neuroaesthetics to neurotheology, have emerged since the Decade of the Brain within a broader universe of "neurocultures" (Ortega and Vidal 2011).

The common goal of these new "neurodisciplines" is to understand the neurobiological processes that "underlie" the behaviors they study (Littlefield and Johnson 2012; Vidal and Ortega 2017). While also postulating that those processes are universal, yet "modulated" by contextual factors, some of those disciplines, such as neuroanthropology and cultural neuroscience, have focused on difference and on how culture is cerebrally "inscribed." They emphasize, as we shall see, the *interactions* of culture and brain and especially the brain's "enculturation." They therefore seem to attribute a crucial role to culture and to differentiate themselves from the position, which prevails in other neurodisciplines, of giving a primary ontological and causative role to biological factors and in particular to the brain as proximal source of all behavior. We shall discuss the differences between the two main neurodisciplines of culture—neuroanthropology and cultural neuroscience—but shall explore in particular the fundamental commonalities that make it possible to envisage a broader category of "neurodisciplines of culture" and to ask whether their research practices enact their claims about enculturation and about the interactions of brain and culture.

In 2012, the editors of *The Encultured Brain*, a volume presented as "an introduction to neuroanthropology," explained that:

> forms of enculturation, social norms, training regimens, ritual, language and patterns of experience shape how our brains work and are structured. But the predominant reasons that culture becomes embodied … is that neuroanatomy inherently makes experience material. Without material change in the brain, learning, memory, maturation, and even trauma could not happen…. Through systematic change in the nervous system, the human body learns to orchestrate itself. Cultural concepts and meanings become neurological anatomy. (Downey and Lende 2012, 37)

Such considerations state very general facts about the nexus of brain and world. But since one of the chief purposes of the neurodisciplines of culture is to understand *culture* (and not only the brain), it is apposite to ask whether, or in what sense, examining changes in the brain contributes to that purpose

beyond reiterating that those changes happen and may be necessary; and it is also appropriate to ask how the notion of *culture* operates within a conceptual and methodological framework designed to investigate neurobiological processes across cultures.

On the one hand, we may examine how the postulate of brain-culture "bidirectionality" within the neurodisciplines of culture translates into concrete investigative strategies and empirical results. On the other hand, we may interrogate these disciplines for their implicit values and epistemic hierarchy. For in spite of an emphasis on the two-way processes that turn brain into culture and culture into brain, a common feature of the neurodisciplines of culture is their belief in the ontological primacy of the brain and therefore in the plausibility of characterizing human groups as "communit[ies] of brains" (Domínguez Duque 2015, 292). Such ontological primacy reduces culture to an external factor that "shapes," "influences" and "impacts on" neural activity, function and processes. What does that perspective mean for both the neurodisciplines of culture and for the very concept of culture?

A Cluster of Disciplines

Like several other neurodisciplines, those that concern culture passed in a few years from being an informal group of scholars with common interests to having their own name and Wikipedia article, professionals, institutions, journals, societies, colloquia, educational events, blogs and websites, programs and graduate students. *Psychological Inquiry* (2013), *Social Cognitive and Affective Neuroscience* (2010), the *Asian Journal of Social Psychology* (2010) and *Progress in Brain Research* (2009) have devoted special issues to *cultural neuroscience*, the *Handbook of Social Neuroscience* offers an overview (Chiao 2011), and the collective volume *Cultural and Neural Frames of Cognition and Communication* (Han and Pöppel 2011) includes several contributions from the discipline. As for *neuroanthropology*, the term was used in the early 1990s, and by the middle of that decade, presentations of the discipline had durably entered anthropology reference works (Marcus 1997; Downey 2012a). In 2012, the same year of *The Encultured Brain*, special issues of *Anthropological Theory* and *Annals of Anthropological Practice* were devoted to neuroanthropology.

The term "cultural neuroscience" seems to have been first used in print in 2007, in a chapter for the *Handbook of Cultural Psychology*. It was then defined as "an area of research that investigates cultural variation in psychological, neural, and genomic processes as a means of articulating the interrelationship

of these processes and their emergent properties" (Chiao and Ambady 2007, 238). Cultural neuroscientists hold that values, practices and beliefs both "shape and are shaped by the mind, brain and genes" and claim that the study of "cultural variation in mental, neural and genomic processes" articulates "the bidirectional relationship of these processes and their emergent properties" (Chiao and Cheon 2012, 288; see also Chiao et al. 2013; Kim and Sasaki 2014).

While the notion that complex behavior "results from the dynamic interaction of genes and cultural environment" is no revelation, cultural neuroscience offers itself as "a novel empirical approach to demonstrating bidirectional interactions between culture and biology by integrating theory and methods from cultural psychology, neuroscience and neurogenetics" (Chiao and Cheon 2012, 289). It seeks "to explain a given mental phenomenon in terms of a synergistic product of mental, neural and genetic events" (Chiao and Cheon 2012, 289) and claims to have "potential implications" not only for psychiatry, business and technology but also for global public policy issues in health, globalization, immigration and interethnic ideology (Chiao 2009b; Denkhaus and Bös 2012). Cultural neuroscientists describe themselves as driven by two "still unanswered" questions: How do cultural traits "shape" neurobiology and behavior? And how do neurobiological mechanisms "facilitate" the emergence and transmission of cultural traits (Chiao et al. 2010, 356)?

We have just quoted from texts labeled "cultural neuroscience." However, the plural in "neurodisciplines of culture" is important. The fields known as *social neuroscience*, *affective neuroscience* and *cultural neuroscience* overlap with each other as well as with *neuroanthropology* and *transcultural neuroimaging* (Domínguez Duque et al. 2009, 2010; Lende and Downey 2012a; Han and Northoff 2008); names such as *sociocultural neuroscience* underline interconnection (Wajman et al. 2015). Intersections and synergies, however, go hand in hand with differentiation dynamics. Neuroanthropologists have been especially keen to stress the distance that separates them from cultural neuroscience, highlighting their preference for fieldwork, expressing concern about cultural biases in experimental research and calling for an increased awareness of the historical, social and political circumstances under which experiments are conducted (Domínguez Duque et al. 2010; Domínguez Duque 2012; Lende and Downey 2012a). In spite of such individualization strategies and the different methodological ideals they convey, all those disciplines share the aim of understanding how the brain "mediates" social interactions and culture.

Despite recognizing the existence of such a cluster, this chapter focuses on cultural neuroscience. The reason is not that we take this field as a synecdoche

for the neurodisciplines of culture or ignore what differentiates it from others but that, because of its chosen method, it is the one that has remained closest to brain research. Indeed, cultural neuroscientists assume that neuroimaging demonstrates "how 'deep' culture can go into the human brain" (Kitayama and Park 2010, 124) and have therefore adopted it as a chief method for studying the "encultured" brain. We discuss below the methodological and ideological dimensions of such choice.

Neuroanthropology, in contrast, has emphasized fieldwork. Until now, however, it has juxtaposed, rather than integrated neuroscientific results with data drawn from the direct observation of cultural settings and situations, and has remained a program for studying the "interplay" of culture, brains and experience. An investigation on the anthropology of opioid maintenance treatments for addiction may be labeled "neuroanthropology" and redescribed as "the neuroeconomics and neuroracial politics of opioid pharmaceuticals," yet merely acknowledging that a treatment for addiction necessarily involves the brain should not suffice to make it neuroanthropological (Hansen and Skinner 2012).[1] Similarly, it is simply by attaching the label "neurocognitive" to the skills involved that ethnographies of rugby and capoeira become "neuroanthropology" (Downey 2012b, c).[2] In short, as a prominent advocate of the field has written, so far "neuroanthropology argues that neurologically plausible accounts" of cultural skills and experiences "are both possible and theoretically productive" (Downey 2016, 41). In contrast (and independently of the quality of the investigations or the relevance of their results), by directly undertaking neuroimaging research, cultural neuroscience has gone beyond the plausible. The question we address in common to the neurodisciplines of culture, regardless of the nature of their results and the background of their practitioners (mainly psychology for cultural neuroscience, mainly anthropological for neuroanthropology), is whether they have the methodological and conceptual means to go beyond statements such as "Cultural practices adapt to neural constraints, and the brain adapts to cultural practice" (Ambady and Bharucha 2009, 342), which simply reiterate the creed they share.

Neuroimaging Culture

Cultural neuroscience uses neuroimaging so systematically that it is often associated with terms such as "trans-cultural neuroimaging" (e.g. Zhang et al. 2011) and "cross-cultural neuroimaging" (e.g. Kitayama and Huff 2015). What are its assumptions and how does it proceed? Cultural neuroscience assumes that "understanding cultural and genetic *influences* on brain function

likely holds the key to articulating better psychological theory" (Chiao 2009b, 290). The quest for "influences" is reinforced by the premise that "human behavior *results* from neural activity" and by the further inference that behavioral variation among cultures "likely *emerges* from cultural variation in neural mechanisms underlying these behaviors" (Chiao 2009b, 290; our emphasis; see also Chiao and Cheon 2012, 289). Though "likely" provides a varnish of caution, the reasoning moves from genetics and the brain toward mind and culture. Neuroimaging and genomic methods for "mapping" neural processes and genes onto neural, mental and cultural processes produce correlations, but the belief that cultural traits constitute evolutionary adaptations reinforces the causal dimension (from brain to culture) (Chiao and Blizinsky 2010).

The tension between correlational results and causal claims undermines cultural neuroscience's calls for bidirectionality. We later examine the relevant research, but now let us take as example the assertion that cultural values, practices and beliefs "impact human behavior" or that the "cultural dimension" of *individualism-collectivism* "affect[s] a wide variety of human mental processes at a behavioral level" and "modulate[s] neural and electrophysiological responses" (Chiao 2009b, 291, 295). Yet, on the one hand, a cultural "dimension" includes by definition mental and behavioral processes, and these necessarily correlate with some features of brain functioning. On the other hand, the cultural dimension is itself defined, at least in part, on the basis of the mental and behavioral processes it is supposed to "affect."

The way out of such a circle is to assume that culture is a product of the prefrontal cortex. This area, it is said, "stands first to be modified or constituted by cultural experience as it is the structure that *lays* culture's foundations" (Domínguez Duque et al. 2009, 60, 61, our emphasis). Such an assertion embodies a significant asymmetry: The claim that culture (including forms of learning) "modifies" the brain is substantiated by empirical observation. In contrast, except in its most diluted interpretation, the claim that the prefrontal cortex "lays" the foundations of culture formulates an ontological belief. But it is precisely this belief which translates into how research is performed.

Let us take a frequently quoted article in the field, "Neural basis of individualistic and collectivistic views of the self," published in 2009 in *Human Brain Mapping*. Its goal was to understand how individualism and collectivism "modulate neural representations underlying social cognition" (Chiao et al. 2009, 2813). According to earlier studies, people who support individualistic values think of themselves and others as independent and as having stable personal traits, whereas those who endorse collectivistic ideals see people as interconnected and describe themselves as immersed in a social

context. The authors drew on the notion of self-construal style (SCS), which has been used to differentiate Western and East Asian views of the self, without reference to research questioning that self-construal reflects individual-level cultural orientation or mediates and explains cross-cultural differences (Levine et al. 2003).

Based on previous work suggesting that activity in the medial prefrontal cortex (MPFC) "reflect[s] the neural basis of self-knowledge" (Chiao et al. 2009, 2814; Kelley et al. 2002), the authors hypothesized that individualists would show greater response for general self-descriptions, and collectivists, for contextual self-descriptions in the anterior rostral portion of the MPFC. Twenty-four right-handed university students were recruited for the study, half native Japanese from Nagoya and half "Caucasian-Americans" from Chicago. They were shown 72 stimuli (in Japanese or English, respectively): 24 general self-descriptions, 24 contextual self-descriptions and 24 self-descriptions in italicized or non-italicized font.

The results seemed to demonstrate that "self-relevant processing within MPFC varies as a function of SCS." People who endorse individualistic values show greater MPFC activation during general self-descriptions, while those who endorse collectivist values display greater MPFC activation during contextual self-descriptions. In both cases, increased MPFC activity "reflects the role SCS plays in how knowledge about the self is formed, and possibly also stored and retrieved." The researchers concluded that "knowledge self-representations of one's self … are culturally specific at the neural level." A meta-analysis of relevant research published between 2003 and 2014 confirmed that result: "East Asian cultures are associated with increased neural activity in the brain regions related to inference of others' mind and emotion regulation whereas Western cultures are associated with enhanced neural activity in the brain areas related to self-relevance encoding and emotional responses during social cognitive/affective processes" (Han and Ma 2014, 293).

Such a study of the neural "bases" of individualism and collectivism is characteristic of the neurodisciplines in at least two ways. First, it illustrates the slippage, typical of this kind of research (Schleim and Roiser 2009), between establishing statistical correlations (here, with culture as predictor) and inferring anatomo-functional "bases" or "underpinnings." Second, the outcomes are predictable without neuroscience or neuroimaging. The authors point to "an intriguing aspect" of their findings, namely, that participants' cultural values (individualism or collectivism), rather than cultural affiliation (being white American or native Japanese), "modulated" neural response during self-judgments (Chiao et al. 2009, 2819). In the Western and East Asian contexts from which the study drew its subjects, people adjust to various environmental

demands, so that culture, as defined by ethnic or national affiliation, does not always match individual behavior. The findings, therefore, are not at all "intriguing," and the excitement they generate manifests the conviction that a phenomenon becomes more real and is more objectively known after its supposed neural correlate has been identified. Only such conviction justifies costly neuroscientific research as a means to apprehend the "dynamic nature of cultural values across individuals and cultural groups" (ib., 2819).

Cultural neuroscientists may retort that neuroimaging allows them to show "how such dynamic cultural values shape neural representations" (ib., 2819). However, in the same way that they cannot specify the neural "bases" of values or attitudes, they cannot demonstrate how *particular* values or attitudes shape the brain. Obviously, "cultural values, beliefs, and practices must be important for social brain functioning" (ib., 2819). This, however, is so by definition. First, because anything brained organisms do is related to brain function. Second, because given that "social brain" refers to the brain regions involved in understanding others (Blakemore 2008) and that social cognition is, in humans at least, inseparable from culturally determined ways of interacting with others, culture is necessarily "important" for the social brain.

And Cultural Diversity?

In short, with respect to their significance for understanding culture, imaging experiments recover at the end what they put in at the beginning, namely, the notion that culture has neural "bases" and correlates. This is publicized with a rhetoric of wonder that recurrently describes findings as "intriguing" or "extraordinary" (Domínguez Duque et al. 2009, 60). Yet, as has been noted even from inside the discipline, "it should not be surprising per se that there exists a neural difference underlying a psychological difference"—in fact, the existence of such a difference is "an axiomatic assumption" of cultural neuroscience, not an "empirical question" (Freeman 2013, 26).

The cultural neuroscientists whose study we just sketched reported on the "influence of cultural values on neural responses within MPFC during self-judgments, despite the absence of differences at the behavioral level," and concluded that their results "reveal an advantage of examining cultural values such as SCS at the neural level" (Chiao et al. 2009, 2819). The "advantage" seems to consist in the capacity of discovering cultural affiliation in the absence of overt behavior. Now, such inscription of cultural values "at the neural level" could mean two things. One is that culture, including beliefs, norms and meanings, is somehow embodied in individuals and specifically in their brains,

pre-reflexively shaping their actions (Choudhury and Slaby 2012a; Gallagher and Zahavi 2008; Noë 2009). Another is that the neural level displays a truth about humans as cultural beings that is not knowable by examining social and cultural practices. Although cultural neuroscience's programmatic statements seem to favor the former interpretation, its practice embodies the latter.

A frequently cited study on the "neural basis of cultural influence on self representation" provides another illustration of such a perspective (Zhu et al. 2007; see also the replications: Ng et al. 2010; Ray et al. 2010). The authors used fMRI to analyze brain activity of "Western" and "Chinese" subjects as they judged personal trait adjectives regarding self, the mother or a public person. Like other investigations in the field, they started with the observation that North Americans and Europeans tend to view the self as independent, autonomous and separate from others, while East Asians emphasize interdependence and interconnectedness. The experimental design was standard: 13 Chinese and 13 Western college students were scanned while judging if an adjective was adequate to describe the self, the mother and the other and also (in a control task) judging the font of the words.

The findings were said to provide evidence of a neural distinction between self and intimate persons for Westerners but not for Chinese. Thus,

> in Chinese individuals, mother-judgments generated enhanced MPFC activity compared with other-judgments and the null condition. Consequently, the representation of Chinese mother cannot be distinguished from the representation of their selves, in terms of the MPFC activity, indicating that Chinese individuals use MPFC to represent both mother and the self. In contrast, MPFC activity corresponds to a representation of only the individual self in Western subjects. (Zhu et al. 2007, 1314)

The study gives the impression of striking a balance between a social constructivism that downplays the role of biology and a naturalistic reductionism according to which interpersonal and cultural relations arise in the brain. However, statements such as "culture influences the functional neuroanatomy of self-representation" or "habitual cognitive processes are accompanied by detectible [sic] parallel neural processes" (ib., 1315, 1314) only serve to mask the obliteration of cultural difference as a *cultural* phenomenon.

As the authors explain, social psychology demonstrated behavioral and cognitive differences between the Western and the East Asian self. But since it did not tell "whether cultures influence the relevant neural mechanisms," it remained necessary to look for neuroimaging evidence that Western and Chinese selves effectively differ "at a neural level" (ib., 1313, 1315). Two poles

were thereby joined: Culture both "affects the psychological structure of self" and "shapes the functional anatomy of self-representation" (ib., 1310). On the one hand, however, correlations do not reveal relations that can be captured by verbs such as "affect" and "shape." On the other hand, the use of those verbs manifests a peculiarly abstract and mechanical view of culture. Contrary to the way they are conceptualized here, notions, attitudes and practices connected to the self are integral parts of culture; they are among the key features that contribute to its enactment, not something that a free agent called "culture" shapes from the outside.

Insofar as cultural diversity is conceptualized essentially in terms of the brain, the experimental setups and results of cultural neuroscience may become part of identity politics (Roepstorff 2011, 40). At the same time, by positing the existence of difference between selves "at a neural level," cultural neuroscience contributes to downplay diversity within the group. In both scenarios, interethnic difference and intra-group identity, the brain is endowed with ontological primacy: the mind is what the brain does, and culture is included in the process. Cultural neuroscience perhaps suggests that universal values do not exist (Begley 2010), but it still naturalizes cultural stereotypes in the laboratory (Choudhury 2010; Choudhury and Kirmayer 2009). Although there have been calls for a more nuanced consideration of socioecological factors (Cheon et al. 2013), they are difficult to realize in experimental work, and cultural neuroscience has not even drawn consequences from the complex intellectual and political histories of sampling categories such as the usual "Caucasian-American" (see Painter 2010 for an overview). Indeed, as critics have pointed out, cultural neuroscientific research tends to classify subjects on the basis of outer appearance at the expense of behavior or sociological or cultural dimensions and has "an understanding of 'culture' and 'race' which still appeals to biology, blood and ancestry" (Martinez Mateo et al. 2012, 160). Whether or not cultural neuroscience really reinforces "Western dominance in a postcolonial situation" (Martinez Mateo et al. 2013, 3), its notion of "culture" clearly functions as a proxy of "race" (Heinz et al. 2014).

Culture as Brain Activity

One could object that individualism/collectivism and self-representation are particularly problematic topics or that we confined ourselves to investigations that explicitly claim to be about "neural basis" (for a synthesis, see Zhu and Han 2008). The studies we chose are nonetheless representative.

Several neurocultural anthropologists seem aware that the notion of culture involves complexities that cannot be studied via the usual experimental designs. They recognize, for example, that there is no such thing as a homogeneous "Western" or "East Asian" culture (Han and Northoff 2008). It has been remarked that cultural psychology, the parent discipline, may give the impression that "there is a very small number of cultural identities (North American vs. East or Southeast Asian), that vary principally on the dimensions of individualism-collectivism or independent-interdependent self-construal" (Cohen 2009, 194). The same applies to cultural neuroscience, whose preferred methods and experimental designs homogenize and factorize culture. More importantly, cultural neuroscience does not take culture as its object of study but as an independent variable on which a dependent one, such as the individualist-collectivist disposition, rests.

We have already noted that some contributors to the neurodisciplines of culture think of their object in a more nuanced way. Anthropologists have suggested an experimental approach that would take into account both the anthropology of experimentation and research subjects' lived experiences (Roepstorff and Frith 2012; Roepstorff and Vogeley 2009). Some neuroanthropologists have criticized cultural neuroscience's "primarily psychological" concept of culture, understood as a set of variables affecting the brain and therefore setting aside "the actual social processes by which cultural knowledge is constituted," and advocate a discipline in which "research and analysis techniques from cultural (and more broadly, social) neuroscience are integrated into and embedded in ethnographic research" (Domínguez Duque et al. 2010, 143, 144; Domínguez Duque 2012, 25). Others demand more attention to the ways culture is conceptualized in the design and interpretation of experiments and underline the need to take into account the historical contexts of the phenomenon under scrutiny, to consider the meanings experimental categories may have in different cultures and to identify cultural biases and beliefs (Choudhury 2010; see also Choudhury et al. 2009; Choudhury and Slaby 2012b).

As for the concept of culture itself, neuroanthropologists counter the psychologism of cultural neuroscience by emphasizing that culture is socially created and transmitted as "shared structures of meaning" through which people interact with each other (Domínguez Duque et al. 2010, 139; Domínguez Duque 2012). Such criticism of the notion of culture implicitly used by the "first generation" of cultural neuroscientists, as well as the emphasis on the contested and evolving nature of the concept, is accompanied by proposals to incorporate an anthropological understanding of culture into experimental settings. Those goals, it is proposed, could be achieved by com-

bining qualitative and quantitative methods with critical theory and reflexive ethnography and by "historically, socially and politically contextualizing the circumstances under which enquiry takes place" (Domínguez Duque et al. 2010, 144).

In a similar perspective, two German scholars have proposed to replace the "entity conception of culture" underlying the homogenizing and essentializing tendencies of cultural neuroscience by a notion of culture as "patterns of representations, actions and artifacts that are distributed or spread by social interaction" (Denkhaus and Bös 2012, 445). The reference to "actions and artifacts" implies that culture is not in people's head but is simultaneously in the individuals, their brains and minds and the world they inhabit (ib., 450). The authors thus criticize the assumption that culture is "stored in people's brains" (Ames and Fiskes 2010, 72). Han et al. (2013) have also underlined the constitutive rather than merely modulatory role of context. While in the modulatory context-dependence model, neuronal and cultural influence interact but remain separate and independent from each other, the notion of constitutive context dependence implies no clear-cut separation between the biological domain of the brain and the social domain of culture. In this model, brains are "biosocial" and culture is "sociobiological" (ib., 353). Thus, some cultural neuroscientists have proposed to redefine culture as that which is manifest in "the direct dependence of the brain's neural activity" on context (Northoff 2013, 95), and others wish to integrate factors such as socioeconomic status, unemployment rate, residential mobility or population density in their definition of cultural influences as a way to address within-nation variation (Ng et al. 2013).

Although such critical perspectives may help give cultural neuroscience a more solid foundation, they do not seem to alter its basic assumption, which is—as a neuroanthropologist put it—that neuroscience provides "the most fundamental perspective yet available" on how people appropriate culture (Domínguez Duque et al. 2010, 140). Indeed, so far the declarations of intention about the co-construction of brain and culture have not altered how experimental work and fieldwork are conducted nor have they prevented neuroanthropologists from claiming that "the shared webs of signification that make up culture are primarily the product of the activity of the PFC [prefrontal cortex]" (Domínguez Duque et al. 2009, 60). The PFC is surely necessary to enable culture; asserting that it *produces* culture goes against the spirit of the neurosciences of culture's own claims about bidirectionality (while also manifesting their ideological foundation).

A review published in 2015 claims that, from a functional connectivity analysis showing that functional neural connections between MPFC and

bilateral temporoparietal function (which is said to be "implicated" in perspective taking) "were much stronger for Chinese than for Danes during the judgment of social attributes of the self," it can be inferred "that the Chinese self is constituted by a more integrated, or holistic, representation of both direct and indirect appraisals. In comparison, the Western self appears more one-dimensional in the sense that it is defined largely on the basis of the first-person perspective alone" (Kitayama and Huff 2015, 6). Such an inference, however, is fallacious. This is not because it implies the questionable existence of a homogeneous, perfectly self-consistent Western or a Chinese self; in fact, the review itself reports that in Asian-American individuals, who have multiple cultural identities, brain response patterns depend on which "cultural frame" is made salient (ib., 10), but it does not establish that this cannot happen in allegedly monocultural persons. The conclusion is fallacious because the nature of a self cannot be inferred or even hypothesized from the existence of certain "neural connections." However, such an inference exhibits the ultimate implicit goal of much neurodisciplinary research: to diagnose and classify on the basis of neural correlates. A similar ambition can be detected in some areas of psychiatric neuroimaging (Vidal and Ortega 2012).

Getting Rid of Culture

What, then, does the *culture* that the neurodisciplines of culture locate in the brain consist of? Even leaving aside its meanings when used outside professional anthropology, such as when Hannah Arendt spoke in the early 1960s of "the crisis in culture" or when half a century later Zygmunt Bauman wrote of "culture in a liquid modern world," the concept is notoriously malleable. In *Primitive Culture*, Edward Tylor (1871, 1) defined "Culture or Civilization" as "that complex whole which includes knowledge, belief, art, morals, law, custom, and any other capabilities and habits acquired by man as a member of society." Since then, many others followed more or less his lead, seeing in culture "the complex of values, customs, beliefs and practices which constitute the way of life of a specific group" (Eagleton 2000, 34). Different emphases are also to be found, with a range and overlap of meanings, as illustrated in Raymond Williams' (1985, 91) observation that "in archaeology and in *cultural anthropology* the reference to *culture* or *a culture* is primarily to *material* production, while in history and *cultural studies* the reference is primarily to *signifying* or *symbolic* systems."

Anthropologists Alfred Kroeber and Clyde Kluckhohn (1952) enumerated over 150 definitions, which they classified into different types:

descriptive, historical, normative, psychological, structural and genetic (in the sense of developmental). In addition, they identified the numerous elements that went into them, from acts and activities to feelings, languages and traditions (see also Shweder 1991). Two things emerge from that variety. One is that students of culture tend to characterize their object as "the organization of human experience and action by symbolic means" (Sahlins 2000, 158). The other is that those organizations and means are neither static nor form systematic and homogenous totalities. Early twentieth-century anthropologists sometimes regarded culture in that way, producing what Marshall Sahlins (ib., 159) critically called "anthropology-cultures." In that framework, it was always possible to identify the authentic native who perfectly embodied the culture. Indeed, as James Clifford (1988, 338) noted, the very idea of culture "carries with it an expectation of roots, of a stable, territorialized existence."

Such integrated coherent totalities probably never existed, and if they did, they certainly no longer do in the context of "locally lived lives in a globally interconnected world" (Gupta and Ferguson 1992, 11). Cultures are rather characterized by internal contradiction. The debate around Margaret Mead's 1928 *Coming of Age in Samoa* is a good example. Mead offered the image of a harmonious society with a liberal attitude toward sexuality. Then in 1983, Derek Freeman published *Margaret Mead and Samoa: The Making and Unmaking of an Anthropological Myth*, where he argued that Mead was misled by native informants and ignored evidence contrary to her depiction of Samoan life. Freeman's work has in turn been questioned. For the present discussion, the controversy highlights the extent to which Samoan culture contained paradoxes and contradictions, which were, as Nancy Scheper-Hughes (1984, 90) put it, "culturally structured but never actually resolved." Mead captured only *one* Samoan truth, and anthropologists have since abandoned the idea "that everything in a society must adhere to a single configuration or pattern," no longer thinking of culture as a "single integrated reality" (ib., 90, 91).

Nevertheless, when cultural neuroscience draws on tools such as the Self-Construal Scale, it invokes exactly such a view of culture, according to which any one factor (being "independent" or "interdependent") must necessarily correlate with some basic principle or attitude (such as individualism or collectivism) considered definitory of the culture. Like neuroaesthetics trying to establish the neural correlates of beauty, but incapable of taking into account the fact that one same stimulus can be judged both ugly and beautiful (Vidal 2011), cultural neuroscience can only identify the supposed neural correlates

of isolated factors. That is because it assumes that culture consists of "factors that affect the biological and psychological processes that shape beliefs and norms shared by groups of individuals" (Hyde et al. 2015, 76).

Moreover, cultural difference is not a basic given which correlates with belonging to some form of "people" (Western, Asian), but rather "a product of a shared historical process that differentiates the world as it connects it" (Gupta and Ferguson 1992, 16). Cultural neuroscientific practice assumes separate and discrete cultures, which it juxtaposes in its experimental designs. It thereby participates in the processes whereby differences are constructed. This is in itself unproblematic and perhaps inevitable. The difficulty and the challenge lie deeper, and they apply to all the neurodisciplines of culture: their assumption that culture is causally and ontologically a product of the brain does not equip them well to deal with cultural phenomena. At the same time, thanks to the global prestige of the neuro, it gives them a powerful tool for shaping culture itself.

Notes

1. Hanser and Skinner (2012) was published in the special issue "Neuroanthropology and Its Applications" of the *Annals of Anthropological Practice*, 36, 2012, where other examples can be found.
2. Downey (2012c) was published in *The Encultured Brain* (Lende and Downey 2012a) where other examples can be found.

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17

Affective Neuroscience as Sociological Inquiry?

Christian von Scheve

Introduction

Recent decades have seen a notable influence of research in the neurosciences on other disciplines, for instance, economics, philosophy, and psychology, as the number of publications, workshops, and funded research projects that are probing possible avenues of cooperation shows. In sociology, the label "neurosociology" reflects this influence and denotes efforts at understanding social action and interaction in terms of human neurobiology (Kalkhoff et al. 2016; Franks and Turner 2013; Tibbetts 2016). Neurosociology, as understood here, is a relatively recent development that originates primarily from the field of North American sociological social psychology, in particular symbolic interactionism, as well as from evolutionary sociology, but is also partly reflected in the views of sociologists working on social epidemiology and physical and mental health, genetics, and social stratification (e.g., Falk et al. 2013; Keyes and Galea 2016, 197f; Freese 2008). This conception of neurosociology is much closer to what is commonly called "biosociology" (e.g., Hopcroft 2016) than those sociological approaches to the neurosciences that are more firmly rooted in the tradition of science and technology studies and the sociology of science and medicine (e.g., Pickersgill 2013; Fitzgerald and Callard 2015).

Neurosociology in this understanding conceives of neural processes and mechanisms as a specific level of *social* reality that can be investigated through

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radically micro-sociological forms of social inquiry (TenHouten 1997). One of the basic tenets of neurosociology is that the social sciences should take into account research in the neurosciences to advance and refine primarily micro-sociological concepts such as self, experience, mind, thinking, and feeling. In this view, insights from the neurosciences are not so much interesting because of their medical and diagnostic findings, but because they claim to provide insights into the foundations of human social behavior and mental processes.

Hence, neurosociologists argue that many neuroscience studies and paradigms as well as their hypotheses and results are directly adaptable to and relevant for the processes and mechanisms traditionally studied by sociologists (TenHouten 1999; Franks 2010). Although this particular neurosociological perspective on the potential contributions of the neurosciences to understanding social reality seems fruitful and promising, in particular for traditionally micro-sociological questions and challenges but also for macro-social, population neuroscience approaches (e.g., Falk et al. 2013), it also bears a number of problems and pitfalls—in theoretical, methodological, and epistemological respects—that often remain unacknowledged by the proponents of neurosociology (Callard and Fitzgerald 2015; von Scheve 2011).

In this chapter, however, I will set aside these concerns for a moment and broadly follow the paradigmatic assumptions of neurosociology to outline and discuss the ways in which a particular area of neuroscience research, *affective neuroscience*, may inform sociological analyses of affect and emotion and sociological inquiry more generally. Affective neuroscience is a nascent field closely related to social and cognitive neuroscience that aims at understanding the neurobiological basis of (human) emotion, primarily in relation to other mental processes like language, vision, or memory (Armony and Vuilleumier 2013). Although the field is much broader in scope, the research discussed in this chapter will focus on studies that are mainly concerned with identifying brain systems implicated in the processing of emotion, that is, their generation, experience, recognition, and regulation (Dalgleish et al. 2009). This chapter will therefore not account for research on non-human emotion and will mostly review studies that capitalize on functional neuroimaging techniques, leaving out scholarship that highlights the role of genes, neurotransmitters, or hormones in the processing of emotion.

The Sociology of Emotion and the Body

The sociology of emotion is classically concerned with what Hochschild (1979) identified as its two main tasks, that is, either to "study the social factors that induce or stimulate primary (i.e., nonreflective, though by definition

conscious) emotions" or to investigate the "secondary acts performed upon the ongoing nonreflective stream of primary emotive experience" (552). Ever since, sociologists have been struggling not to "over-culturalize" and "over-cognitivize" emotions, to not exclusively pay attention to these secondary acts informed by norms and values associated with emotion, or to the cultural meanings of emotion, or to the various strategies to purposefully communicate, negotiate, and regulate emotions.

Instead, sociologists are also supposed to investigate the "nonreflective" and "primary" (Hochschild) emotive experience, how it is formed by culture and social structure, and how it in turn affects and shapes the social. This task capitalizes on what most approaches tell us is part of the essence of emotions, namely that they are not only discursive, linguistic, and symbolic but also *bodily* phenomena, entailing both distinctive physiological reactions and characteristic bodily feelings.

In most strands of the sociology of emotion—as well as in sociology more generally—the body is accounted for predominantly as a site of practices and performativity, as an object that the feeling subject purposefully *works upon*, for example, in rituals, expressive movements, gestures, or through impression management. However, this is of course not what Hochschild had in mind when she was talking about the "ongoing nonreflective stream of primary emotive experience" and its bodily dimension. This "performative" or "dramaturgical" view of the body is also evident in theories that expressly emphasize the *embodied* nature of emotion (e.g., Shilling 1997). Although this line of inquiry highlights that emotions are bodily grounded and that any understanding of emotions that fails to account for this bodily grounding remains incomplete, they hardly provide any models or theories of how, exactly, the body is implicated in emotion. This embodiment perspective therefore largely remains at the level of the body *as such*, hardly ever digging deeper into the more precise biological underpinnings of emotion and how they can inform sociological research.

In some sense, this emphasis on the body and on non-linguistic or non-conceptual processes has been taken up by *affect theories* in cultural studies that have largely remained unrelated to the sociology of emotion. Affect studies can be understood as a theoretical and at times also political movement that emphasizes the bodily and affective constitution of the social world. Most affect theories converge on the idea that affect is a pre-linguistic, pre-discursive, and pre-conceptual force or intensity that impinges on a body's capacity "to act and be acted upon" (Seigworth and Gregg 2010, 1). Although many take this as a very general premise for a revisionary social ontology and attribute this capacity to bodies of varied sorts (i.e., not only to human or animal

bodies), others have considered affect to be closely related to emotion and human physiology, arguing that affect is something like a bodily "substratum" of emotion. Many of these body-centered approaches to affect frequently make reference to findings from the neurosciences (see Leys 2011; Papoulias and Callard 2010, for critical overviews). However, these rapprochements hardly ever serve to build more precise or refine existing theories of affect and emotion, but are rather considered "conversations" in which (affective) neuroscience, "through its focus on affect at a molecular level", is taken to be "what makes critical-theoretical practice possible" (Papoulias and Callard 2010, 38).

Hence, there is an expressed interest in the sociology of emotion as well as in parts of affect studies to account for the bodily aspects of affect and emotion in order to advance understanding of the social and cultural dimension of the human body and/or to further develop existing sociological theories of emotion. Understanding emotions (and affect) more comprehensively in sociological terms therefore, to some extent, may require an understanding of human physiology and of how the body processes, constitutes, and represents emotions.

For early sociologists of emotion, like Kemper (1987), but also for more recent proponents, for example Turner (2000) or Hammond (2007), Darwinian and other evolutionary theories have been an important point of reference in coming to terms with the physiology of human emotion. These theories more or less emphatically rely on models of "basic emotions" or "affect programs" and assume that there are a limited number of cross-culturally universal emotions that can be defined by specific constellations of cognitive, physiological, expressive, and phenomenal components. These models, however, have been criticized on various grounds (see Scarantino and Griffiths 2011, for a discussion and defense of basic emotions accounts) and studies in affective neuroscience have both supported (Panksepp and Watt 2011) as well as severely questioned them (Lindquist et al. 2012; see also Sander 2013).

Given this interest in the bodily dimension of affect and emotion, there seems to be a need for a more in-depth understanding of the biological and physiological processes and mechanisms that are implicated in human emotion. This need can not only be satisfied by insights into human neurobiology but equally well by other domains of biology, such as genetics or biochemistry (e.g., Leknes and Tracey 2008). Scholarship addressing these needs is of course well-advised to take into account the long-standing reservations of sociological inquiry regarding biological explanations and the manifold concerns over biological reductionism (see the overview in Meloni 2014), but can equally rely on works that have developed suggestions on how at least the more

well-known fallacies may be avoided, not least in view of neuroscientific findings (e.g., Pitts-Taylor 2016; Cerulo 2010; Walsh 2014). In this chapter, I will follow this latter strand of work when engaging with research from the field of affective neuroscience, asking whether this field provides insights into the bodily dimension of affect and emotion that may advance current sociological theorizing.

The Generation of Affect and Emotion

To better understand the black box between some kind of stimulus or situation and an affective or emotional response, and to provide some explanation for the mechanisms involved in producing these responses, early neuroscientific research relied on lesion studies to examine the role of certain areas of the brain for human and animal behavior. By looking at changes in behavior and cognitive capabilities, researchers made inferences about the functions of those parts of the brain that were damaged or lesioned.[1] Results of some of these early studies showed that even animals with extensive lesions of the cerebral cortex, the outer layer of the brain that realizes sensory and motor processing as well as capacities such as memory and attention, still exhibited emotional behavior with characteristic reactions of the autonomous nervous and motor systems (Bard and Rioch 1937; Cannon 1931). However, the more the cortex receded, the more clearly changes in behavior began to appear. Animals were much more easily provoked and emotional displays were often inappropriate to the situation, extraordinarily intense, and increasingly undirected or undifferentiated (for overviews, see LeDoux 1996; Koelsch et al. 2015; Dalgleish 2004).

If, in addition to certain cortical areas, deeper and phylogenetically older brain structures were damaged or lesioned, these deficiencies often lead to the complete loss of affective reactions. These structures include, for example, the hippocampus, which plays an essential role in memory, the hypothalamus, which controls basic biological functions and is responsible for the body's hormonal state, and the amygdala, which seems to plays an important role in evaluating the affective salience of a stimulus (LeDoux 1996, 92ff). These insights have led to the development of theoretical models of the neurophysiological basis of affect and emotion that concur in assuming two different kinds of neural systems (or pathways) through which affect and emotion are generated. It is now widely believed that the complexity of human emotion is driven, on the one hand, by a relatively rudimentary "affect system" or "network" encompassing subcortical structures that produce affective responses to

certain stimuli and fulfill important evolutionarily functions. Neural processing in these circuits is generally assumed to be rapid, bottom-up, and automatic with little conscious or higher cognitive involvement (Lindquist et al. 2012; Sander 2013, 17–22; Dalgleish et al. 2009). On the other hand, paralimbic and cortical brain areas are considered to play an important role in the differentiation, categorization, and top-down control of these basic responses. These neural circuits are also critical to the cognitive representation of affect, its influence on executive functions, and the integration of affective and cognitive information, which also includes conceptual knowledge that couples rudimentary affective reactions with culturally derived concepts of discrete emotions such as anger, fear, or joy (Barrett and Bliss-Moreau 2009; Dalgleish et al. 2009).

Affect

The idea of a rudimentary affect system that operates largely independently from conceptual thought and conscious control is part of many affect theories in cultural studies, as outlined above. In many of these theories, the bodily dimensions and at times also the neural architecture of affect play a central role (e.g., Sedgwick 2003). One of the main references used by affect theorists is Tomkins's (1963) work on *Affect, Imagery, and Consciousness*, in which Tomkins, an experimental psychologist, proposes a number of (evolutionary stable) basic "affect programs". It is interesting to note, however, that affect theory has, for the most part, not cared to take into account the developments in the neurosciences that have developed from Tomkins's and related works and today paint a picture of affect that notably deviates from what Tomkins had suggested.

For many contemporary researchers, the affect system, much like other perceptual systems, is primarily tasked with transforming stimuli into a meaningful "motivational metric" and to rapidly initiate behavioral responses (Cacioppo et al. 2004). The structures implicated in this system are thought to "represent crucial components of a network that bind sensory stimulation from inside the body to that coming from outside the body, and in so doing each gives the other informational value" (Barrett and Bliss-Moreau 2009, 173). An influential model of how affect is produced has been developed by LeDoux (1996), who was interested in the question of which brain areas imbue a stimulus with affective significance. He capitalized on the processing of auditory information in non-human animals and investigated the information processing pathways of conditioned fear responses. His "dual path" model

of affective processing postulates, on the one hand, the rapid but coarse processing of information in subcortical areas of the brain, in particular the amygdala, a collection of neurons in the medial temporal lobes that plays a central role in attributing affective significance to sensory stimuli. On the other hand, the model suggests a slower but more differentiated route through cortical networks of the brain. Subcortical processing relies on simple and fuzzy representations of a stimulus and its principal characteristic is the speed of processing that comes at the cost of error-proneness. Basic and bodily affective responses are produced in these subcortical areas before a stimulus can even be consciously perceived. More recently, LeDoux (2012) has integrated these findings into a "survival circuit" model in which affective reactions occur as parts of a more general architecture for how organisms detect and behaviorally respond to various environmental challenges and opportunities.

Although dual path models of affect and emotion are widely referred to in the literature, they have not remained without criticism and indeed there is little direct evidence for this kind of neural processing in humans (see Sander 2013, 20; Vuilleumier 2005). Nevertheless, the dual path model as well as the survival circuit account can provide some insights into the rapid elicitation of basic affective responses and associated action tendencies that are much more in line with the available evidence than, for example, Tomkins's widely referred to affect programs. These models may explain, for example, why we often experience certain feelings without being consciously aware of what has triggered these feelings in the first place. Also, it can be linked to understanding recurring patterns of social action that are driven by affective responses of which actors are not necessarily aware. Some sociologists have tried to explain the emergence of social practices through these affective processes (e.g., Wetherell 2012), and LeDoux's account may provide some support for this conjecture. Importantly, this account is hardly reductionist in any sense. On the contrary, learning and conditioning play a central role in the model that links certain "innate" stimulus-response couplings (whose phenotypes are assumed to be adjustable and flexible) to a broad variety of prototypical—though historically and culturally contingent—social events and situations through experience and learning.

A comparable neuroscientific model with a slightly different emphasis has been proposed by Rolls (2004). He defines emotions as produced by instrumentally rewarding or punishing stimuli. Rewarding stimuli are those for which an organism is willing to invest energy and punishing stimuli are those on whose avoidance an organism is willing to spend energy. Both kinds act as reinforcers since they influence the *probability* with which certain behaviors will occur. The actual reinforcing qualities of a stimulus are its somatosensory

consequences, such as taste, smell, or pain (Rolls 2004, 18), and emotions can be differentiated by means of various stimulus-reinforcer associations.

For the sociology of emotion, the distinction between primary and secondary reinforcers seems of particular interest. Whereas primary reinforcers are *innately* rewarding or punishing stimuli such as certain tastes, touches, or smells, secondary reinforcers are acquired through learning and association with primary reinforcers and may represent a wide array of different kinds of information, such as certain visual patterns (e.g., architecture, landscapes), symbols (e.g., religious and political symbols), or abstract social concepts, for example, money (Knutson and Bossaerts 2007). Secondary reinforcers are generated from associations between previously neutral stimuli and primary reinforcers, a learning process that seems to take place mainly in the amygdala and the orbitofrontal cortex (OFC) (Rolls 1999). Stimuli initially need to be categorized in the sensory cortices and subsequently their reward value is determined in the OFC and the amygdala that attributes aversive or appetitive significance to sensory information (Rolls 1999, 102; Leknes and Tracey 2008).

At first glance, the concept of secondary reinforcers might resemble the mechanisms of classical conditioning. However, their function is different from that of stimulus-response learning, because conditioned stimuli do not elicit specific behaviors (the much-feared stimulus-response scheme that sociological behaviorism propagated in the 1950s), but rather affective reactions that allow for flexible and adaptive behavioral responses (Rolls 1999, 62). Because of the potential diversity and complexity of secondary reinforcers, Rolls's account provides insights into how basic affective responses can be socially shaped and structured at a bodily, that is, neurophysiological level and in a way that is not necessarily linked to language and conceptual thought.

Aside from these two models that strongly rely on social learning and conditioning, a third and more recent approach has focused more specifically on the neural basis of affect. This approach is also more theory-driven in that it originates from dimensional models of emotion instead of basic emotion or affect program theories. Dimensional theories broadly assume that emotion is not a "natural kind" (Barrett 2006) and that discrete emotions such as anger, fear, sadness, or joy are not characterized by unique (neuro-)physiological signatures. Instead, these theories propose that "core affect" is a common denominator of different emotions and other psychological processes and can best be described in terms of different dimensions, usually valence (pleasant or unpleasant) and arousal (arousing or calming). Core affect is assumed to be "a basic, universal, and psychologically irreducible property of the mind" (Barrett and Bliss-Moreau 2009, 168) and "grounded in the somatovisceral, kines-

thetic, proprioceptive, and neurochemical fluctuations that take place within the core of body" (ibid., 171). Core affect is realized in what Barrett and Bliss-Moreau (2009) call a "neural reference space" divided into two main functional networks (see Lindquist et al. 2012, 2016, for comprehensive reviews). The first, a sensory integration network, establishes experience- and value-based representations of objects that include their "external" sensory features as well as their impact on bodily homeostasis. The second, visceromotor network guides a body's autonomic, endocrine, and behavioral responses to an object. This network is, among other things, implicated in altering stimulus-reinforcer associations and also contributes to decisions that are based on feelings and intuitions rather than on rules and principles (Barrett and Bliss-Moreau 2009).

This neuroscientific research contributes to the sociological understanding of affect and emotion in at least three ways: First, it provides a model of the neurophysiological aspects of emotion that is decidedly more in line with the available evidence[2] than sociological theories that assume some common bodily signature of specific discrete emotions. Second, these models offer a perspective on affect as a bodily substratum of emotion that can be operationalized and tested empirically, not only with neuroscientific but potentially also with sociological methods and research. This is of course not a trivial task, as discussed elsewhere (von Scheve 2016). Third, this perspective as well as the accounts provided by LeDoux and Rolls offer conceptions of how affect is implicated in producing specific action tendencies. Affect in many sociological and cultural studies theories is hailed for being a "potential to act," but hardly any of these theories provide accounts of how, precisely, this potential might be conceptualized. In the works reviewed so far, action tendencies are thought to be mediated by neural *approach* and *avoidance* systems that broadly correspond to Rolls's (1999) criteria of positive and negative reinforcers and their behavioral implications (see also Dalgleish et al. 2009). These two systems are supposed to imbue a wide range of stimuli with an evaluative and motivationally relevant meaning.

Although initial research on these processes has relied on animal studies and thus needs to be interpreted with caution, more recent studies have increasingly been able to replicate these findings also in humans (e.g., Lindquist et al. 2016). It should also be noted that, although the affective responses produced within milliseconds in certain neural networks are relatively undifferentiated, they still have far-reaching consequences for more complex emotional reactions, cognitive processing, and social action. Hence, this research suggests that affects are not "hard-wired" or "instinct-like" responses to specific situations, rather they are highly susceptible to social

learning and environmental (for instance, cultural and social structural) influences.

Emotion

Emotions in everyday situations arise not only in response to the perception of rudimentary sensory information but often are reactions to symbols, thoughts, and memories. In generating fully fledged emotions, affect networks are complemented by brain systems implicated in the processing of more complex cognitive information, for example, the sensory cortices, areas of the associative cortex, the anterior cingulate cortex, and prefrontal areas of the brain (Barrett et al. 2007; Kirkland and Cunningham 2011; Ochsner and Barrett 2001). Emotions, in contrast to basic or core affects, therefore require some associations between conceptual representations and the basic affective responses as well as some forms of representation of these responses.

These associations and representations are processed in different areas of the brain and involve a number of cortical networks. In a now classical account, Damasio (1994) suggested that these associations are established through "dispositional representations", that is, acquired representations that link certain classes of stimuli to certain patterns of affective and physiological responding. He suggested that these representations are realized in areas of the prefrontal cortex that transmit information to subcortical brain networks (e.g., the amygdala), which then initiate affect-specific physiological and motor reactions (Damasio 1994, 136ff). This role of the frontal lobes has been established in subsequent studies and is comparably well documented. Research has shown, for example, that an impairment of the OFC brings about notable changes in social and emotional behavior and goes hand in hand with deficiencies in decision-making (Bechara 2004). The OFC also plays a fundamental role in stimulus-reinforcement association learning since it is supposed to represent reinforcers' positive and negative contents. This area seems to allow for the flexible learning and relearning of stimulus-reinforcement associations, a function that is particularly important in complex social and cultural environments, in which objects, acts, and situations constantly change in view of their reward (or punishment) value. In contrast, subcortical affect networks seem to function much less efficiently in response to changes in these contingencies (Rolls 1999, 2004).

Regarding sociological understandings of emotion, these insights can be important to arrive at a better understanding of the rigidity of some of our emotional reactions in contrast to the flexibility with which actors learn novel

ways of emotionally relating to the social world. For example, social, spatial, and cultural mobility confronts actors with emotion cultures that are potentially different from those in which most of their emotional socialization took place. On the one hand, deeply ingrained and habituated affective responses to objects or behaviors may be difficult to alter and may therefore pose challenges in different environments. On the other hand, actors seem to be able to establish and learn the emotional significance of novel objects, acts, and situations with relatively little effort.

A closely related matter concerns the capacity to manage, regulate, and adapt actual emotional responses, a topic that has received broad attention in the sociology of work and organizations (e.g., Wharton 2009). Regarding the neural basis of the regulation of emotion, studies have shown that the connections between orbitofrontal areas and parts of the subcortical affect system grant the OFC a special role in the control of affective processing, for example, through modulating or inhibiting amygdala activity. Further support for this view is provided by studies on emotion regulation and the reappraisal of emotion (Buhle et al. 2014; Ochsner and Gross 2005; Silvers et al. 2015). Determining the necessity for the regulation seems to involve the anterior cingulate cortex (ACC), a structure that plays a role in monitoring and evaluating a person's internal state and events that prompt the need for changes in behavior (Ochsner and Barrett 2001; Barrett et al. 2007). The ACC and the OFC are thus supposed to be important in assessing social situations characterized by, for instance, contingency, uncertainty, or specific affective expectations (Dalgleish et al. 2009).

For the sociology of emotion, these findings are important for three reasons. First, they support long-held assumptions that emotions are generated through an interplay of basic affective responses on the one hand and conceptual representations (including thoughts, norms and rues, language, memories, ideas, etc.) on the other hand. Second, neuroscience research supports the view that the sociality (i.e., the social and cultural "construction") of emotion is not confined to these conceptual representations but extends significantly to the more basic affective responses and to the bodily substratum of emotion. Third, and probably most importantly, this research provides insights into the malleability and variability of emotion. In contrast to those sociological perspectives according to which emotion is wholly flexible and can be regulated and managed more or less at will, neuroscience research provides a more nuanced picture of whether, how and over which timeframes affect and emotion can adapt to changing social and cultural circumstances and normative expectations.

Decision-Making

Looking at instrumental decision-making as a form of social action, some fields in sociology have been relying—depending on national traditions more or less extensively—on rational explanations and approaches based on norms and values (see Miles 2015; Kroneberg and Kalter 2012, for discussions). In both domains, recent theoretical developments have increasingly turned to research in economics and psychology to complement and extend existing models of decision-making, for example, by referring to dual-process theories of information processing (Vaisey 2009) or by acknowledging the importance of heuristics (Goldstein 2009). Interestingly, in many of these current developments, affect and emotion play a central role (e.g., Manzo 2012).

Decision-making is one of the key fields in social cognitive and affective neuroscience research and therefore too vast to be summarized here (see Volz and Hertwig 2016; Inzlicht et al. 2015; Phelps et al. 2014). Hence, instead of providing a broad but necessarily coarse overview, I will focus on the well-known "somatic marker hypothesis" that emerged from a number of classical studies and has not only inspired a broad range of subsequent research on the role of emotion in decision-making but at the same time strongly emphasizes the social nature of the influence of emotion on decision-making at the neural level.

The somatic marker hypothesis draws on findings related to the neural basis of affective processing and the malleability and responsiveness of this basis to subjective experience and social learning. It proposes that decision-making essentially depends on and is guided by certain kinds of affective feelings (Bechara 2004; Dunn et al. 2006). The hypothesis, originally developed by Damasio (1994), is based on the previous studies of two prominent patients in medical history who had suffered comparable damage to certain cortical areas of the brain. These two as well as related cases showed that "reduced" emotionality (following brain injury) can under certain circumstances be associated with the inability to make instrumental-rational decisions and to implement corresponding actions. In view of decision-making in everyday situations and regarding personal future outcomes, the functioning of certain cortical brain areas which are central to decision-making seems to depend on affective processes, in particular those realized in higher cortical areas of the brain, such as the OFC (Bechara 2004; Bechara et al. 2000; Hornak et al. 2003).

The somatic marker hypothesis, which has successfully been tested empirically (Bechara et al. 2000), proposes that before any decision following ratio-

nal deliberation is made, the possible consequences of particular decision options are automatically paired with specific patterns of affective arousal, possibly with a discernable positive or negative valence (akin to the proverbial "gut feeling"). This autonomous physiological reaction or "bodily feeling" highlights certain consequences of a possible decision option and thus reduces the possible alternatives available to a subsequent deductive decision-making process.

According to this perspective, "somatic markers" facilitate more rapid and efficient decision-making, while their absence reduces its efficiency or even renders it impossible. However, somatic markers are not a substitute for deliberative decision-making. Rather, they act as weighting mechanisms for "calculating" different decision options, making available additional information that can be used to evaluate the broad spectrum of possible decision options and consequences, which in social and personal situations is wide-ranging and characterized by uncertainty (Hinson et al. 2002). Importantly, somatic markers are no hard-wired or innate decision-making mechanisms but rather internalized during socialization and regular interactions in stable social environments through the combination of certain categories of (social) stimuli with certain categories of somatic (affective) states (see Damasio 1994, 177).

The somatic marker hypothesis therefore challenges theoretical models of social action and decision-making that emphasize instrumental-rational deliberation and the abstract calculation and weighing of probabilities. Instead, the hypothesis is more in line with "embodied" accounts of decision-making that highlight routine and bodily practices well as the cultural and affective bases of value and valuation (e.g., Bourdieu 1990). Practice theories, for example, have been challenged by the question of how practices are transmitted between actors, given that they are supposedly based on tacit and implicit knowledge that is not easy to verbalize (Turner 1994). The concept of somatic markers might contribute to our understanding of how patterns and regularities not only of decision-making but also of social action more generally come into existence when no explicit rule learning is involved.

Also, the somatic marker account can be linked to theories of embodied action that emphasize the role the body plays over that of (propositional) "mental" or "cognitive" beliefs and representations (e.g., Strand and Lizardo 2015). Although most of these theories concur in that there is a need to more comprehensively account for this bodily dimension, it often remains limited in both theoretical and empirical terms, for instance, to observable behavior. Here, the malleability or "plasticity" of somatic markers opens up avenues for coming to terms with embodied action that take serious those bodily aspects that are not readily observable but still critical for social action and at the same

time accounts for individual and collective past experiences and socialization, thus avoiding recourse to any kind of biologically "predetermined" action.

Social Interaction

Social interaction is a key element of sociological theory in a number of ways but primarily as a locus for the exchange of significant symbols, the negotiation of meaning, the presentation of self, and, more generally, the constitution of intersubjectivity. The sociology of emotion has been keen to understand the importance of emotion for social interaction as well as to gain insights into the ways in which social interactions produce emotions (e.g., Katz 2001). An important facet of emotions in social interaction is their facial expression. In sociology, the concepts of "emotion work" and "feeling rules" (Hochschild 1979) have gained foothold when it comes to investigating the expression of emotion, both verbally and facially. The basic assumption of this perspective is that emotions are almost never expressed in some "natural" or "unregulated" way, because social norms governing the experience and expression of emotion dictate when and how emotions are to be expressed. We are socialized to adapt our expressions to different social situations and expectations (e.g., implied in gender roles of power hierarchies), and this adaptation does not necessarily have to happen as a conscious and purposeful effort but can well happen involuntarily and automatically (e.g., Vandekerckhove et al. 2008).

Emotions are a fruitful domain of sociological inquiry precisely because they are at the crossroads of what is, on the one side, evolutionary inherited, physiological and beyond volitional control, and socially and culturally constructed on the other side. For instance, it seems fair to assume that expressions of fear or anger that are considered prototypical in Western societies do have *some* communicative value also in non-Western societies (and vice versa). But precisely this association between emotions, characteristic facial displays, and the ability to recognize emotions from these displays are a matter of ongoing debate.

Whereas some argue for cross-cultural universality in the generation of characteristic facial expressions and the ability to recognize these expressions (e.g., Sauter et al. 2010), others have mounted evidence that facial expressions are deeply shaped by culture and society (e.g., Elfenbein and Ambady 2002; Gendron et al. 2014). These latter studies show—in line with most ideas in the sociology of emotion—that not only the physiology underlying facial expressions but also whole-body expressions of emotion are shaped or "cali-

brated" by cultural and social structural factors (see de Gelder and Huis in 't Veld 2016, for a recent review).

Studies in affective neuroscience may help shed some light on this controversy. Taking a closer look at the physiological aspects of emotion expressions might further clarify the interactions between their biological and socio-cultural foundations. Research speaking to these issues, however, has mainly concentrated on the decoding rather than the encoding of facial expressions (George 2013). Therefore, the basic principles of visual perception seem to be an adequate starting point. Addressing the issue of cross-cultural universality in the recognition of emotion expressions, Adolphs (2002) distinguishes (early) perception from (subsequent) recognition. Basic perceptual processes occur immediately after visual stimulus onset and are primarily based on activity in the sensory cortices that process the characteristics of a stimulus and their configurations, such as geometric features. At this stage, for example, two different faces can be distinguished from one another. In contrast to the mere perception of an expression, which is solely based on information inherent to the stimulus, its *recognition* requires additional information. This includes, for example, (historically contingent, normative, and biographical) knowledge about the contexts in which a facial expression has previously occurred, about other behaviors that accompany an expression, and behaviors of other individuals in the social situation.

Taking the neural basis of visual processing as a starting point, the question arises at which point the various kinds of information conveyed by facial expressions, such as age, sex, motives, or underlying feelings, become available. A number of models assume that some of these features are processed in early perceptual systems by specialized neural circuits (Haxby et al. 2000). In light of the debates on the universality of emotion recognition, some have suggested that perception of certain components of facial expressions may occur universally in early perceptual systems and are only later associated with different, culture-specific concepts and meanings (Adolphs 2002). This view is further supported by the fact that cross-cultural universality has mainly been observed for simple categorizations of expressions, while cultural differences have been shown in particular for their conceptualization and symbolic representation (ibid.). For sociological perspectives on the role of facial expressions in social interaction, this would mean that only some configurations of the facial muscles might indeed universally and meaningfully convey a limited spectrum of bodily and psychological states and that most expressions of emotions, such as fear or anger, require the involvement of culture and conceptual knowledge.

This perspective is backed by evidence on the neural basis of face perception and the interaction of basic perceptual processes (as the low-level processing of sensory input and the transformation of this input into meaningful information, for instance, the extraction of certain features from a visual stimulus) with visual recognition (involving conceptual knowledge and expectations). Recognition can be the result of the perceptual categorization of stimuli based on, for example, geometric features. In view of facial expressions, this would mean that no additional knowledge would be required to differentiate and categorize distinct expressive patterns and to identify discrete (basic) emotions from these patterns (see Adolphs 2002 for an overview).

Some empirical studies indeed suggest that, in principle, the visual characteristics of certain facial stimuli seem to be sufficient to activate the structures that are often employed to culturally categorize emotions. These studies also show that even the early perception of facial expressions proceeds largely categorical and that individuals are also able to reliably assign similar expressions to a specific emotion category (Adolphs 2002). These findings thus lend credit to the view that categories of facial expression may be isomorphic with the structure of culturally derived emotion concepts.

This neurophysiological view therefore suggests that emotion expression differs notably from sign-based and symbolic communication, in particular language, and could be described as a rapid and non-symbolic means of interaction that at the same time recruits and "activates" stocks of conceptual knowledge that are relevant to the ongoing interaction. The cultural specifics of emotion expression are likely to be rooted in neural structures that realize early visual perception and represent associations between basic stimulus features and cultural concepts of emotions and their facial expression. This perspective closely tallies with both, assumptions in affect studies of non-linguistic and pre-discursive affect that relates bodies of various sorts to one another, and accounts in the sociology of emotion that ascribes a special role to the expression of emotion in the coordination of social interaction and the establishing of intersubjectivity.

Further insights into the neural basis of face perception come from studies on patients with blindsight. Blindsight is a form of visual impairment in which not the eyes but rather parts of the visual cortex are affected. Under this condition, certain areas of the visual field cannot be consciously perceived, although visual information is still processed in other areas of the brain and can therefore, under certain conditions, trigger behavioral reactions. Studies on patients with blindsight have played an important role in clarifying how precisely the perception of visual stimuli takes place. Interestingly, blindsighted patients seem to be able to distinguish certain expressions of emotion

(presented in the blind area of their visual field) from each other despite the lack of conscious recognition of those stimuli, a capacity frequently referred to as "affective blindsight" (e.g., Tamietto and de Gelder 2008). Neuroimaging and lesion studies on blindsighted patients suggest that subcortical structures, especially the amygdala, play a critical role in the ability to distinguish facial expressions, in particular in cases of fear and other negative emotions (e.g., Celeghin et al. 2015).

Although these studies do not warrant the conclusion that the categorization and differentiation of emotion expressions can take place entirely in subcortical brain structures, this form of decoding probably contributes significantly to the rapid and reliable decoding of facial expressions and possibly also to the explanation of some degree of cross-cultural commonalities in emotion expression. This does not yet, however, explain how discrimination and categorization are linked to conceptual and highly culture-specific knowledge and thus the full *recognition* of certain facial expressions *as* emotions.

In sum, insights from affective neuroscience might make a contribution to our understanding of how emotions and facial expressions become an integral part of interaction situations. Whereas sociological accounts of the role of emotions in social interactions have mainly capitalized on the purposeful regulation of emotion expressions according to rules and norms, these studies point to a more fundamental role of emotions in the mutual attribution of feelings, mental states, and motivations for action and further contribute to our understanding of the bodily aspects of this role. Emotionally expressive signals, although significantly shaped by cultural practices, facilitate intersubjective understanding beyond symbolic interactions and language-based communication.

From the Sociology of Emotion to Broader Biosocial Matters

Over the past decade, the field of affective neuroscience has developed theoretical models and accumulated empirical evidence that can complement sociological understandings of affect and emotion and their bodily dimensions. Some of this research sheds light on the question of what emotions are and of how social factors impinge on what Hochschild (1979) called "the ongoing nonreflective stream of primary emotive experience". Importantly, and contrary to the concerns insistently voiced by some sociologists, affective neuroscience models of emotion are hardly ever "reductionist". Quite the

opposite, many theories and concepts emphasize the social plasticity of the neural mechanisms involved in generating both basic affects and the more complex emotions. Concepts such as secondary reinforcers or dual path models of the neural processing of affect and emotion can advance sociological theorizing and inform empirical research on how emotions are socially constructed not only in terms of norms, values, and practices but also down to the level of human physiology. They may also shed light on the social consequences of emotions, as in theories of social action and interaction. Some neuroscience models offer insights into how instrumental decision-making is systematically influenced by emotion and how cognitions are, as a matter of principle, infused with an affective valence that "biases" most cognitive operations, from perception and attention to categorization and memory formation. This view not only informs rational-deliberative approaches to social action but likewise cultural theories of action, in particular regarding the bodily aspects of social practices.

On a more general note, these insights are critical to better understand current political endeavors at social control and the reemergence of social engineering techniques. In particular, "nudging" as a form of behavioral governance has recently attracted increasing attention as an unobtrusive technique for dragging people's behaviors in a collectively (and politically) desirable direction (Leggett 2014). Nudging techniques mostly rely on insights from psychology, behavioral economics and neuroeconomics, and emotion and affect are presumed to play an important role in the implicit nudging of behaviors (Bovens 2009). Investigating these new forms of governance from a biosocial perspective requires an in-depth comprehension of the mechanisms underlying nudging and how they interact with social and cultural environments, and some of these insights might be provided by sociological encounters with affective neuroscience.

Finally, the research summarized above touches upon long-standing issues in social and sociological theory, in particular regarding social interaction and intersubjectivity. The neurobiology of facial expression and perception provides models of how inherent aspects of emotion expression interact with social and cultural concepts of emotion. These insights add to our understanding of social interaction in face-to-face encounters by going beyond purely symbolic and language-based modalities of interaction. From a more general sociological perspective, the affective neuroscience of facial expression may not only complement theories of affect and emotion but also—and some might say more directly—connects to broader biosocial debates that involve, for example, biotechnological arrays of face perception, classification, crowd control, and public security. The increasing prevalence of video surveillance in

public spaces has prompted debates on how biometric surveillance data are used and what readouts of this data are in principle possible (e.g., van der Ploeg 2012). Social sorting algorithms not only categorize and discriminate in terms of the facial recognition of identity, race, gender, or physical health but may—and do—also classify according to emotional states. This is in particular so in the domain of policing and "crowd control", where automated recognition of facial expressions is used to estimate future crowd behavior. A better understanding of what facial expressions actually represent and what social and cultural differences are implied in these expressions may contribute to a broader apprehension of the societal implications of automated recognition systems (see Gates 2011).

As promising as these insights from affective neuroscience might be for sociological inquiry, affective neuroscience in turn can and should pay attention to relevant sociological theory and research. In the very moment in which neuroscientists endeavor to utilize sociological concepts that are essential for a comprehensive understanding of culture and society, for instance, social inequality (along with categories such as age, gender, race, or ethnicity), status and status differences (e.g., in terms of social class or power), or culture and cultural differences (e.g., in terms of lifestyles, capital, or cultural consumption), they can profit from over a century of research in these fields. This pertains, on the one hand, to the many ways in which "the social" can be understood and conceptualized, for instance, in terms of networks and structural arrangements and in terms of social fields, institutions, or organizations. Also, neuroscientific studies interested in comparative research on different social groups and/or categories can profit from existing accounts of how these groups and categories are socially constructed and how they usually overlap, as is evident in research on intersectionality. On the other hand, this also pertains to sociological methods, in particular established measures that can be used to reliably assess, say, social class or status. However, this direction of collaboration seems much less common than vice versa. This might be one reason why research in affective neuroscience (and in other branches of the neurosciences) hardly finds its way into general sociology and requires substantial efforts at translation.

Although affective neuroscience concepts and theories are informative for sociological inquiry, their hypotheses and results are hardly ever *directly* adaptable to the processes and mechanisms studied by sociologists, as some have suggested (TenHouten 1999; Franks 2010). One of the main reasons is that sociologists usually ask different questions than affective neuroscientists, and hence the answers that affective neuroscience provides are answers to neuroscientific and not to sociological questions. Therefore, findings from the neu-

rosciences can hardly ever be used in a direct fashion, but rather need to be translated and closely scrutinized before making their way into sociological theory and research.

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Notes

1. See, for example, Volz and Hertwig (2016) for a recent discussion of the epistemological fallacies of this "reverse inference" account.
2. With this I do not mean, of course, that the research discussed can provide some kind of "ultimate" truth. On the contrary, many of the authors I discuss take great care in explicitly declaring that they provide sets of empirically testable hypotheses rather than a comprehensive theory that makes claims for some ultimate truth value.

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18

"Bio-looping" and the Psychophysiological in Religious Belief and Practice: Mechanisms of Embodiment in Candomblé Trance and Possession

Rebecca Seligman

Introduction

This chapter explores the processes that mediate the relationship between cultural and social experiences and bodily responses, in the context of intense religious devotion. Using ethnographic and psychophysiological data from a study of spirit possession among mediums of the Candomblé religion in Brazil, I offer a close examination of the interactions between enactments of roles and meanings, and the bodily states experienced by mediums. In doing so, I aim to offer insight into the mechanisms of what anthropologists call "embodiment." In particular, the chapter provides a novel and accessible account of how psychophysiological systems are implicated in embodiment, providing evidence that the embodied learning involved in trance and possession is reflected in distinct patterns of autonomic nervous system regulation among mediums. The concept of "bio-looping" is proposed as a way of modeling the circular and mutually reinforcing processes through which religious meanings and practices come to shape bodily experience and functioning.

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Trance and Possession in Candomblé

At 11 pm, the *festa* (public ritual) for the goddess Oxum at Pai João's Candomblé center was just getting under way. The Candomblé religion, practiced widely in Salvador, Brazil where my research was conducted, is an African-derived spirit possession religion brought to Brazil by slaves. Participants in the religion are predominantly Afro-Brazilian, and many of them are low income. Forms of devotion in Candomblé range from casual lay participation, to initiated ritual adherence, and devotees engage in both private rituals, and the kind of large public group ritual taking place at Pai João's place that evening. Candomblé cosmology holds that there is a pantheon of deities (*Orixás*) and lesser spirits who may possess the bodies of human beings and displace the human's consciousness for a period of time during which they occupy his/her body. Mediums, or *filhos de santo*, are those humans who have been chosen by the deities to enter into long-term relationships with them, becoming regular vehicles for their divine materialization in the human world after a rigorous process of initiation.

The sound of fireworks set off in the concrete yard outside Pai João's *terreiro* alerted people that the ritual was about to begin, and people who had been milling around began to wander inside. Some of the *ogãs* (male ritual assistants) began to pound out a slow, slightly disorganized rhythm on the tall drums arranged along the back wall of the large open room where the ritual would take place. The initiated mediums of the *terreiro*, dressed all in white, gathered in the center of the room.

As the *ogãs* at the drums began to pound out the songs of greeting to each of the Candomblé deities, the *filhos* danced and sang and members of the audience joined in, clapping their hands, swaying to the drums, and singing along. Once the songs of greeting had been sung to each of the deities, the *ogãs* began to drum in earnest, pounding with more intensity as they played the special rhythms designed to call the deities down to their earthly mediums. The *filhos* seemed to move with more intensity as well, performing the specific dances for each of the gods.

Soon, the first of the *filhos* went into trance. It was Jalita, a medium who had been initiated at the age of twenty-eight after becoming physically and mentally ill. Jalita had become ill soon after getting married and having three children in just four years. Overwhelmed by the sudden transition to motherhood, a lack of resources, and low levels of social support, she suffered psychological symptoms of anxiety, depression, and possibly psychosis that were unresponsive to medication. She had also been afflicted with undiagnosable

somatic symptoms including fever and cough. Now, some twenty-six years later, Jalita was a senior member of the *terreiro*, serving as its official second in command, and she had not suffered from such physical or psychological distress in the years since becoming a medium.

As she went into trance, Jalita's eyes suddenly squeezed tightly shut, indicating that she, the earthly medium, had become unconscious and a spiritual entity had descended into her body. Her head dropped forward precipitously on her neck and she began to tremble violently. In the midst of her trembling, Jalita let out a series of high-pitched cries. Then, suddenly, she swayed so dramatically to one side that she appeared as if she would fall over. She caught herself at the last second as she veered to one side, and before she could fall, hopped on one foot several times in a small circle until she stood fully upright again. Finally, she went still. As the drums pounded on and the rest of the *filhos* continued to dance around her, Jalita stood still, her body holding a distinctive posture with hands clasped behind her back, chin dropped to her chest, brow furrowed, and eyes squeezed tightly shut, rocking back and forth on her feet. This posture signaled that Jalita was now fully entranced—meaning that her own consciousness was entirely displaced or suppressed and her body occupied by the animating force of a deity—in this case, the goddess Oxum.

As the now possessed Jalita stood rocking in the middle of the room, two *ekédis*, or female ritual assistants, approached her. The *ekédis* gently stabilized Jalita and began to ritually prepare her. They removed her glasses, shoes, earrings and hairpins—for the *orixás* do not wear shoes, earrings, or glasses. They took a large piece of fabric and tied a sash around her middle, with a big bow in back, symbolizing the elaborate attire of the *orixás*. Once the *ekédis* had moved away, Jalita, now Oxum, began to dance once more. Soon other *filhos* followed suit, their entry into trance accompanied by the same distinctive pattern of bodily movements.

Since human consciousness is believed to be displaced while the individual is possessed, Jalita and the other mediums were totally unaware of what their bodies were doing while possessed, describing complete amnesia for the entire period of trance. Yet while *filhos* report amnesia for everything that occurs while they are possessed, many of them describe the sensations associated with the *onset* of trance in similar terms: they talk about a feeling of excitement, a tingling sensation, experiencing chills and *arrepios* (goosebumps), faintness or dizziness, and a sense of distance from their surroundings before consciousness is lost.

After all of the *filhos* had entered trance, they were led away to a back room of the *terriero*, where they were dressed in ritual attire: in this case, since most

of the *filhos* were possessed by the Oxum, they were dressed in white gowns with ornate head pieces and given mirrors to hold as symbols of the beautiful and vain goddess. Once all of the possessed *filhos* were dressed in their ritual garb, they returned, still in trance, to dance for the rest of the night. The movements and postures of the possessed mediums resembled the known bodily qualities of their possessing deity—powerful, aggressive movements for Ogum the warrior, a stooped posture for Oxalá the wise senior god, and so on. Thus despite her age and weight, the possessed Jalita danced with great intensity for hours while possessed by her youthful Oxum, feet moving lightly across the floor, arms gliding from side to side. The ecstatic energy of this performance contrasted with the calm, deliberate energy of the unpossessed Jalita, whose only knowledge of her performance that night would come from the reports she received from others.

Embodiment and Bio-looping

As the preceding description demonstrates, entry into trance evokes both the same behavioral enactments and the same bodily sensations across different Candomblé mediums. In addition, the psychophysiology measures I used to examine aspects of mediums' bodily functioning revealed that Jalita and other mediums also share a distinctive pattern of autonomic nervous system activity. I will explain the measures I used and what they mean in depth later in the chapter, but for now, the point I want to make is that taken together, these observations make evident the central role that bodies play in Candomblé trance and possession.

At the most basic level, the bodies of mediums are the vehicles through which they enact their roles, and through which trance and possession are performed. Mediums are able to use their bodies as a means to express their religious belief and commitment to other members of the community, and the community as a whole uses the bodies of mediums as objects of shared meaning and sites of cultural production. Collective beliefs and values concerning the spiritual and material, agency and moral responsibility, and even bodies and selves, are played out on and through the forms of mediums. But bodies are neither neutral nor stable objects through or upon which culture may be written (Scheper-Hughes and Lock 1987; Turner 1995). Bodies are dynamic. They absorb and integrate cultural and social information, literally *incorporating* experience. For instance, Jalita and the other mediums with whom I worked talked about emerging from their religious initiations already

possessing a great deal of new bodily knowledge, including how to invite possession and how to dance for, and as, the gods. In fact, mediums described a fundamental transformation of their bodily experience—not only during possession, but also through the broad process of religious formation, which for many included the experience of bodily healing.

How do we account for these experiential transformations and the accumulation of bodily knowledge associated with mediumship? And more broadly, how do we explain the ways in which practice and discourse, or peoples' socially and culturally informed behaviors and ideas, affect their bodily states? These are questions about what many scholars refer to as embodiment (Mascia-Lees 2011). Anthropologists use the term embodiment to refer to the way in which social and cultural knowledge are absorbed by and through the body. Embodied knowledge is understood to be largely unconscious, learned through our practices and lived interactions with our social and cultural environments. However, most anthropological approaches to embodiment fail to address the kinds of questions I am posing here—questions about how embodiment works, about the mechanisms that mediate the relationship between cultural and social experiences and bodily responses, between the roles and meanings mediums enact, and their bodily states.

One possibility that has rarely been given any real consideration within cultural anthropology is that biological processes play a role. Most studies of trance and possession within cultural anthropology have treated these phenomena primarily as social role performances rather than as bodily states with a biological component. The fact that different mediums describe a similar set of physical sensations at the onset of trance suggests, however, that these sensations may index a distinctive pattern of physiological activity associated with their ritual performance. The practices and meanings involved in possession rituals and trance induction techniques may, in other words, result in a measurable change in the bodily states of mediums. The kind of physiological process that may be involved in the experience of trance and possession is suggested by the findings from my psychophysiology measurements. As I will describe in further detail below, I used a technique called impedance cardiography to compare patterns of cardiovascular activity between mediums and non-medium initiates. My analysis of the data from these measures revealed that not only do mediums experience distinct physical symptoms at the onset of trance, but compared to non-medium initiates (*ogãs* and *ekédis*), they also show a distinct pattern of heart rate regulation. These findings suggest a link between the bodily transformations associated

with mediumship, and the psychophysiological functioning of mediums. I return to this idea later.

Consistency in the way that mediums describe their sensations at the onset of trance, and the near uniformity in the way that trance is performed also suggest an important role for learning (Halloy and Naumescu 2012). Mediums learn to talk about the onset of trance in terms of a specific set of sensations because those particular sensations are imbued with significance. They learn both from their own experience, and from the way that other mediums describe their feelings, to associate goosebumps, dizziness, and tingling with the nearness of the gods and the onset of possession. And because those sensations are particularly meaningful, mediums learn to recognize and attend to them (Halloy and Naumescu 2012). At the same time, irrelevant sensations—ones that are not linked to important cultural meanings—are ignored. Mediums do not talk about feeling hot when their deity is about to descend into their body in part because this is not a culturally meaningful symptom—it is not an element of the cultural model for trance that mediums learn when they become involved in Candomblé.

In addition, practices like the ritual change of clothing and the adoption of physical postures and actions characteristic of the occupying spirit or god also contribute to the embodied experience of possession. It is easy to imagine how wearing a big, heavy, ornate dress, and enacting a set of stereotyped movements could make a medium's body *feel* different than usual. Moreover, these behaviors and bodily symbols communicate to the social group that a shift has taken place in the consciousness of the possessed individual, and others then reinforce the performance of her change of state by treating the medium's body differently. Hence, meaning and practice are also crucial dimensions of embodiment. The question is: how do the bodily, practice, and meaning aspects of embodiment interact?

I propose that we think about such interactions in terms of a feed-forward or "looping" process in which the mechanics of bodies are affected by learning and experience, and the characteristics of particular bodies in turn shape the ways in which they come to learn from and embody experience. In other words, the qualities of particular bodies figure as both causes and effects of experience. Deconstructing the "loop" may help to clarify what this means. Bodies are not all the same—they differ in size, shape, strength, sensory and physical capacities, metabolism, microbial content, and so on. The characteristics of different bodies affect the way that they interact with the world. Being very tall shapes the way that an individual views his or her environment, both literally and figuratively.

Some of the ways in which bodies differ are the product of learning. Bodies of athletes have learned through rigorous physical training to do things that the bodies of other people cannot do, and this in turn affects the nature of the activities and practices in which athletes engage. The circularity or looping effect that I referred to is thus in the way that the learned characteristics of bodies—how they are shaped by experience—combines with the meanings attributed to those qualities, to influence subsequent experience and shape exposure to and embodiment of new knowledge.

The philosopher Ian Hacking proposed the metaphor of a loop, or "looping," in the context of a discussion of psychiatric illness, to refer to this kind of circular process. Hacking was theorizing the way in which the social meanings of particular expressions of distress come to reinforce the existence of particular disorders, and ultimately lead to the creation of different categories or "kinds" of people—for example, "schizophrenics" (Hacking 1995). For Hacking, the looping metaphor was a way of transcending debates about whether mental illnesses are "real" or "socially constructed"—he argued that social constructions of mental disorder *become* real through looping. Looping thus provides a model for thinking about how the effects of social and cultural experiences on peoples' bodily states become "real," and how the qualities of peoples' bodies come to "really" affect their psychological and social states. I borrow Hacking's (1999) term "bio-looping," which he uses to refer to the ways in which biological knowledge production helps to create and reinforce different categories of people, but I use it to draw attention to the ways in which embodied processes, including biological ones, are implicated in the continuous and mutually reinforcing relationships among meaning, practice, and experience (Seligman and Kirmayer 2008).

Embodiment can itself be thought of as a product of bio-looping, not reducible to any one of these interacting elements. A bio-looping model can thus help to answer the questions I posed earlier about the mechanisms of embodiment, and how the bodily, practice, and meaning aspects of Candomblé mediumship contribute to the transformations that many mediums experience. In bio-looping terms, we can understand the distinctive physiological profiles of mediums that I described earlier as part of the circular process through which bodily qualities feed-forward to influence the kinds of experiences that people have and the cultural knowledge and skills they develop. In other words, differences in psychophysiological functioning might be thought of as the kind of bodily quality, like being tall or strong, that shapes and is shaped by experience. These qualities may, for instance, enhance their capacity to learn and attend to the sensations, meanings, and enactments associated with trance and possession.

Learning, Motivation, and Capacity

The bio-looping model can thus help to explain persistent questions about why some people become mediums and others do not—why only certain individuals are both willing and able to enter trance states and become possessed. Becoming a medium is not simply a matter of motivation—even if an individual wants to take on the role, not every individual has what it takes to become a medium. For example, an *ogã*, named Edvaldo told me the story of how his repeated failure to enter trance during his *ensaio*—a ritual meant to help pre-initiation novices learn to succumb to possession (O'Connor 2013)—had led to the realization that he was really meant to be a ritual assistant, not a medium.

Thus, potential mediums must be able to successfully learn the beliefs, practices, and role requirements associated with mediumship, and in particular, they must be able, or must learn to be able, to become possessed. In the context of her work on prayer practices in a contemporary Evangelical church, Tanya Luhrmann argues that people differ in their "proclivity" for learning what she refers to as the "metakinetic" aspects of prayer—that is, the embodied dimensions of such practices, associated with things like emotional response and alterations in consciousness (Luhrmann 2004; Luhrmann et al. 2010). Luhrmann has shown that there are differences between individuals in their capacity for absorption, or deeply focused attention and imaginative experience, and differences in this capacity correspond with differences in the proclivity for achieving vivid, intimate experiences of god through prayer. To use Luhrmann's terms, then, what distinguishes those who become mediums is their proclivity for learning the embodied dimensions of their role. In other words, just as some Evangelicals have a proclivity for absorption, those who become mediums may have a proclivity for trance and possession. In bio-looping terms, we might understand this proclivity as a result of pre-initiation experiences of psychosocial stress and bodily affliction, like the ones that Jalita experienced before her initiation, which feed-forward to shape the particular ways in which prospective mediums engage with the meanings and practices of the religion.

Jalita and many other Candomblé mediums came to their role with histories of psychosocial and bodily distress, social marginalization, and a disrupted sense of selfhood. In fact, mediums in my study reported more unexplained bodily symptoms like back pain, headaches, and fatigue on a psychological questionnaire than any other group of religious participants.[1] Experience of unexplained symptoms, known as somatization, is often a consequence of

particular kinds of social and cultural experiences—in particular, it is a way of experiencing and expressing distress caused by experiences of social marginalization, inequality, and misfortune (Nichter 1981; Guarnaccia et al. 1996). But in the case of mediums, somatization also represents a *cause* of social and cultural experiences in the sense that, according to their own accounts, somatic symptoms served to motivate the religious initiations of many mediums. While the stories of most of the mediums I worked with described a history of hardship and loss, it was the experience of acute bodily afflictions and unexplained somatic symptoms that ultimately drove them to seek out Candomblé. Within the religious community, these symptoms were read as a message from the gods that the person needed to be initiated into mediumship.

As I have argued in depth elsewhere (Seligman 2005a, b, 2010, 2014), previous experiences of distress and affliction not only motivate participation, but also shape the way that mediums internalize and identify with the beliefs and meanings of Candomblé. For many mediums, the distressing experiences they have before their initiations undermine their self-understandings, creating conflict between goals, aspirations, and desired selves on the one hand, and life experiences on the other. Such conflict in turn affects the way that mediums take up the religious symbols and meanings of Candomblé. Having been overwhelmed by life transitions, uncontrolled emotions, and unexplained illnesses, Jalita for instance, was especially primed to learn to understand herself as the victim of spiritual affliction and to embrace the influence of her goddess, Oxum, over her experiences and behaviors. These religious meanings offered a way to understand her sense of deviance and distress as signs of a spiritual calling, and herself as spiritually empowered, rather than ill.

Those with a particular capacity to identify deeply with these religious meanings may also be more likely to become deeply *absorbed* by those meanings and the behavioral enactments that go along with them. For mediums, trance and possession are the ultimate enactments of their belief in the influence of spiritual forces in their lives. Since mediums like Jalita are primed to find this belief particularly personally meaningful, they are also primed to focus intensely on and become deeply absorbed by practices surrounding the enactment of this belief. In particular, this deep personal investment may predispose them to get the most out of the practices associated with trance induction—to focus intensely on the pounding drums, ritual prostrations, and stylized movements of the ritual dances, because these practices are meant to call down the deities. They are also primed to become deeply immersed in a set of expectations about the effects of those practices—effects like the

sensations of goosebumps, dizziness, and excitement, as well as the loss of self-awareness and memory known to accompany trance and possession.

Anthropologist Thomas Csordas has talked about such processes in terms of what he calls "somatic modes of attention" which he defines as "culturally elaborated ways of attending to and with one's body" (2002, 244). This term is a way of talking about how the body itself attends or acquires knowledge in ways that while preconscious are still inherently cultural and thus shaped by cultural expectations. Certain bodily sensations have particular cultural significance and individuals may therefore develop somatic modes of attention in which those sensations are focal. Dizziness is particularly meaningful in some cultures, heat in others (Hinton et al. 2008). For female refugees from El Salvador, for example, bodily heat holds significance as a symptom of fear, despair, and anger. Salvadoran women are thus likely to pay special attention to sensations of heat, and to attend to and perceive their social environments differently when experiencing such heat (i.e. to perceive the environment as more threatening or oppressive) (Jenkins and Valiente 1994).

The "somatic modes of attention" concept thus highlights the importance of non-discursive forms of bodily knowing and learning. It suggests that bodies may be entrained through their exposures to particular experiences and meanings, and that this entrainment has crucial implications for how we perceive self and world. In other words, somatic modes of attention may result from processes of bio-looping. Moreover, my research findings suggest that entrainment of psychophysiological systems may represent a crucial part of how these loops link together body, meaning, and experience.

Human Biology, Psychophysiology, and the Mechanics of Embodiment

What kinds of effects do social and cultural entrainment have on psychophysiological systems? And what are the mechanics of such a process? Theory and evidence from the fields of biological anthropology and psychophysiology can help us address these questions.

Differences between individuals in patterns of physiological arousal are a major focus of psychophysiology research. People differ in both the shape and magnitude of their physiological responses to the stimuli they encounter in their environments. Stimuli consist of almost anything encountered as individuals move through the world—from the sights, sounds, and smells of sensory input and the sensations of physical contact with objects, to social

interactions and symbolic transactions. Psychophysiology recognizes a suite of biological systems that tend to be associated with arousal to stimuli. These include the neuroendocrine system, particularly the hypothalamic-pituitary-adrenal axis (HPA) which regulates things like your stress hormones; the autonomic nervous system (ANS), which is in charge of things like the activation of the cardiovascular system, including your heart rate and blood pressure; and the feedback and control of various elements of the central nervous system (CNS), which plays a major role in whether you perceive stimuli as threatening, inviting, or neutral.

The activity of these physiological systems often varies widely across individuals, and may also vary independently within individuals—that is, the regulation of your heart rate and blood pressure may or may not follow the same pattern as the regulation of your stress hormone responses. The term reactivity has been used to describe characteristic individual patterns of physiological activity and arousability of these various systems in response to stimulation (Rothbart 1989). Such differences in reactivity are closely associated with individual differences in stress sensitivity (Boyce et al. 1995; Cacioppo et al. 2005).

The ability to effectively regulate one's state of physiological arousal is the other side of the reactivity coin; this capacity for "state regulation" affects an individual's control over his or her own level of arousal, including the ability to limit and recover from arousal through the use of behavioral and cognitive mechanisms (Rothbart 1989; Kagan 1994). Individuals who have high state regulatory capacity may still be highly reactive to stimulation, but able to modulate or recover from their arousal effectively. Those with low state regulatory capacities may be highly reactive to stimulation, and *unable* to modulate their arousal. Hence, variations in reactivity and state regulation are examples of ways in which different bodies bring different sets of characteristics to interactions with their social and cultural environments.

Psychologists and physiologists have developed the complementary concepts of "allostasis" and "allostatic load" to capture how different bodies develop patterns of reactivity and state regulation. The concept of allostasis highlights the important capacity of bodies to adjust to shifting contexts through flexibility in the regulation of physiological systems, since different kinds of activities and environments make different demands on these systems. The complementary concept of "allostatic load" refers to the negative health consequences (i.e. cardiovascular disease) of physiological reactivity when it outweighs environmental demands, and to the physical burden of appropriate, but continuous, reactivity in situations of chronic stress (Schulkin et al. 1998; Schulkin 2011). Allostasis and allostatic load thus both represent

processes through which bodies are tuned to their environments, and bodily functioning is entrained by experience.

These processes are mediated in important ways by culturally and socially informed beliefs and knowledge that affect how we interpret and make meaning of our experiences. These meanings in turn shape our learned bodily responses. In fact, individuals may develop consistent patterns of response to stimuli that are perceived to be the same—a tendency referred to as "response stereotypy" (Cohen et al. 1997; Strelau 1988). This suggests that the meaning of an experience is not necessarily filtered through conscious awareness, but may become a part of an individual's *physical* being through such patterned responses.

Physiological responses are also related to differences in how individuals *attend* to experience—to their "somatic modes of attention" (Csordas 2002). An individual may be more or less likely to focus attention on a particular social stimulus, meaning, or bodily sensation, based not only on its personal and cultural meaning but also on their past and present embodied responses. In fact, individuals make attributions about the meaning that particular experiences have for them, based on their bodily response. If flying on an airplane results in an intense physiological response, including a surge of stress hormones and an increase in heart rate and blood pressure, this will affect the associations an individual has with flying. This is because physiological systems send feedback to the brain that is designed to affect evaluative processing of experience. Thus, we learn to attend differently to experiences based on the way they affect our psychophysiological functioning. Changing the meaning or attributions that an individual makes about his or her bodily responses to an experience therefore has the potential to change the meaning of the experience. Changing the meaning of experience, in turn, has the potential to change patterns of bodily response. Hence, cognitive factors like belief and meaning play an important role in state regulation.

Knowledge of psychophysiological processes thus helps to further flesh out the bio-looping model, illustrating potential mechanisms through which the effects of experience on bodies, and bodies on experience, are manifest. This is a novel way of theorizing embodiment because it includes a concern with the ways in which biological systems are shaped by learning and experience. Thus, instead of talking in broad terms about "bodily ways of knowing," bio-looping offers a complex, multilevel understanding of the ways in which bodies are designed to respond dynamically to and integrate social and cultural experience into their functioning.

Psychophysiology of Mediumship

How can knowledge about reactivity and state regulation be applied to understanding the bodily learning and experiences of transformation that mediums undergo? And how might something like autonomic nervous system regulation of cardiovascular function affect the proclivity for trance and possession? In order to investigate these questions, I measured aspects of cardiovascular activity among a group of ten mediums and a comparable group of ten non-medium initiates (*ogās* and *ekédis*).[2] Measurements of cardiovascular activity, particularly heart rate (HR) and high-frequency (HF) heart rate variability, have a long history of use as indicators of how cardiovascular activity is regulated by an individual's autonomic nervous system (ANS), and can thus be used as markers of autonomic reactivity and self-regulatory capacity.

The ANS is divided into two branches and the heart and other organs are enervated by nerve fibers from both branches. Each branch is associated with different functions: the sympathetic nervous system (SNS) is associated with reactivity—it readies the body for action. SNS nerves act like a gas pedal, speeding up HR when activated, and slowing it down through withdrawal. The parasympathetic nervous system (PNS) is associated with state regulation, conservation, and repair. PNS control over the heart via the vagus nerve acts like a brake, slowing down HR when it is activated, and speeding it up through withdrawal. Past psychophysiology research has focused on the relative balance between the activities of the SNS versus the PNS. More SNS activity meant that an individual was more highly reactive, and this was generally thought to be a bad thing (Uchino et al. 1996). Greater PNS activity, on the other hand, meant that the individual had higher state regulatory capacity and this was generally considered to be a good thing (Porges 1992; Porges et al. 1994).

Mounting evidence suggests, however, that the total regulatory capacity of both sub-systems combined may be a particularly meaningful marker of adaptive physiological flexibility. Tight control over HR activity by both branches means the ability to activate and withdraw dynamically, representing both reactivity and state regulatory capacity. Thus, total regulatory capacity indexes the propensity for allostatic regulation, or the flexible adjustment of physiological function in response to environmental demand (Berntson et al. 2008b). Total regulatory capacity also indexes the ability to respond to stimuli in the environment through cognitive and behavioral channels like attention; in fact, regulation of arousal has been directly correlated with the ability to initiate, sustain, and terminate attention (Hansen et al. 2003; Porges 1992).

Individual differences in total regulatory capacity are thus relevant for understanding variability in the way that bodies respond to their environments, the way that individuals attend to and through their bodies, and the range of effects that experiences have on bodily characteristics.

Psychophysiologists have recently introduced a measure of total regulatory capacity called "Cardiac Autonomic Regulation" (CAR). CAR is calculated by adding together measures of SNS control (HR) and PNS control (HF). Higher scores are associated with a variety of positive outcomes (Berntson et al. 2008a). Individuals with higher CAR appear to be more capable of adjusting their patterns of cardiovascular response to meet the level of demand, and of appropriately matching psychophysiological arousal to circumstance. Individuals with low CAR are more likely to respond disproportionately to challenges or fail to mount adequate responses, leaving them either unable to respond sufficiently or responding excessively, thus damaging their bodies over the long term. However, such psychophysiological profiles are by no means fixed or static phenomena. Patterns of autonomic nervous system regulation may vary across the life-course, respond to shifts in context, and be moderated by social support, life events, and experience (Alkon et al. 2003; De Haan et al. 1998).

In my study, I measured CAR for mediums and non-medium initiates using data from a method called impedance cardiography.[3] Along with electrocardiogram (ECG) data, impedance can be used to derive measures of HR and HF that can be combined to calculate CAR. I used baseline measurements rather than measurements taken during trance and possession, in part because of methodological and ethical challenges associated with trying to gather such measurements in ritual contexts, but also because I was interested in the bodily capacities or tendencies individuals bring with them to mediumship, not simply the transient effects of induction techniques on physiological states.

What I found is that the baseline CAR scores of mediums were substantially higher than those of non-medium initiates. Seventy percent of mediums (seven out of ten) had CAR scores above the median for both groups, while 70 percent of the non-medium initiates had CAR scores that were below the median. Mediums' scores were also less variable, or more similar to one another, than their counterparts' scores.[4] These data indicate that compared to *ogãs* and *ekédis*, the mediums in my study tended to have higher levels of total regulatory control over their cardiovascular function—and we know that such control is associated with a greater capacity for dynamic reactivity and state regulation in response to contextual demands.

These findings have a number of implications for our understanding of mediumship, trance, and possession. Most notably, they suggest a link between the proclivity for Candomblé mediumship, and the ability to dynamically regulate arousal. In other words, these data show that the motivation and capacity for mediumship are reflected in the bodily functioning of mediums. This is most compelling in the sense that it documents a connection between a particular kind of religious participation, and particular kinds of bodily qualities among participants. However, because I was unable to collect longitudinal data on the CAR scores of mediums, which would have allowed me to compare their autonomic regulatory capacities before and after initiation, we cannot know with certainty at this time whether this pattern of autonomic control is a cause or effect of the religious embodiment associated with the mediumship role. That is, we cannot be sure whether mediums had higher CAR before becoming mediums, or if they developed higher CAR afterward. Both scenarios suggest interesting and important pathways for how bodily processes and embodied beliefs and practices influence one another and it is worth exploring each in depth.

One possibility is that the psychophysiological profiles of mediums have changed since they became mediums. Under this scenario, mediums would have had lower CAR before initiation, a pattern associated with a lower capacity for state regulation, and then developed higher state regulatory capacity through their experiences as mediums. This interpretation is consistent with the life history narratives of mediums, which describe escalating cycles of psychosocial stress and somatic suffering prior to initiation, followed by the transformation to a state of well-being after initiation.

The somatic suffering prior to initiation and the transformation afterward can both be understood as part of a bio-looping process in which the social and cultural experiences of mediums have contributed to shaping their bodily processes, and vice versa. Prior to initiation, experiences associated with poverty, social marginalization, and psychosocial distress may have set up negative patterns of perception, attention, and reactivity. In such high-stress environments, people's bodies may come to anticipate stress and arousal, adjusting their patterns of reactivity through the kind of feed-forward, allostatic processes described earlier. The chronic nature of such physiological up-regulation contributes to allostatic load, or bodily wear and tear that may cause somatic symptoms.

Physiological reactivity and bodily symptoms are both perceived negatively, and loop back to reinforce the negative meanings of experiences—making stressful experiences more stressful. Moreover, physiological reactivity and

somatic symptoms shape the way that individuals attend to and through their bodies. Such states call attention to themselves, and may promote even greater arousal and more distressing symptoms, through cycles of attention, negative attribution, and symptom amplification (cf. Kirmayer and Sartorius 2007). Bio-looping would thus be responsible for creating a stress-sensitive "mode of attention" (Csordas 2002) among prospective mediums, in which the reactive qualities of their bodies were a response to the negative qualities of their social worlds.

However, the relatively high CAR scores of these mediums at the time that I measured them suggest that the experience of initiation and the practices and meanings associated with mediumship may have played a role in disrupting or redirecting this looping process. Somatic suffering and physiological arousal have very different meanings within the context of Candomblé, as indicators of a spiritual calling and the material evidence of the work of the deities in one's life. As we discussed earlier, changing the meaning or attributions that individuals make about their bodily responses has the potential to change the meaning of their experience, and changing the meaning of experience, has the potential to change patterns of bodily response. Hence, patterns of negative reinforcement among stressful experiences, meanings, and bodily responses are interrupted by the introduction of a set of religious meanings that make these kinds of bodily experiences the basis for a set of positive expectations and attributions.

One of the most noteworthy aspects of this bio-looping model, then, is the idea that the same bodily qualities that fed into the negative loop prior to initiation, could also feed-forward into a positive loop established through religious involvement. In this scenario, the reactivity that mediums experienced prior to initiation is not extinguished after initiation, instead it is counter-balanced by the development of higher levels of PNS control established through religious participation, and thus becomes part of a high *total* regulatory capacity. In other words, engagement with Candomblé gives mediums the cognitive and behavioral tools to increase their state regulatory capacities. For example, deep internalization of the meanings associated with possession, combined with the behavioral training of trance induction, helps *filhos* learn to manipulate attention and perception in ways that allow them to enhance or inhibit physiological arousal—by focusing narrowly on stimulating sights, sounds, and internal sensations and blocking out non-relevant input. Such skills may transfer outside the religious context as well, helping mediums learn to use attention and perception as everyday tools for state regulation.

This interpretation of the data thus suggests that, in much the same way that social support can disrupt patterns of psychophysiological reactivity among people with histories of adversity and a supportive caregiving environment can help reactive children thrive (Nachmias et al. 1996; Uchino et al. 1996), participation in mediumship may reshape patterns of physiological response in a way that not only allows individuals to induce trance states in appropriate contexts, but may also carry over to help them establish a more general pattern of enhanced state regulation and reduced psychosocial stress.

On the other hand, we must also consider the possibility that mediums already had high CAR prior to initiation. Under this scenario, mediums would have already been both highly reactive to stress and challenge, and highly capable of state regulation, allowing them to recover effectively from their stress responses. This kind of autonomic flexibility is associated with positive health outcomes (Berntson et al. 2008b) but also with high levels of body awareness, and somatic symptom reporting (Zachariae et al. 2000). High CAR may thus have contributed to a high degree of body awareness among mediums—a somatic mode of attention that, in a high-stress environment, made them particularly aware of their own bodily responses to stress and particularly prone to experience and attend to somatic symptoms. This scenario is consistent with the pattern of somatic suffering reported by mediums prior to initiation, and the finding, discussed earlier, that mediums reported higher numbers of somatic symptoms on a psychological inventory. Such tendencies may have been partly responsible for attracting mediums to the behavioral and cognitive tools offered by mediumship, as a way to make meaning of their bodily responses and somatic symptoms.

Having high CAR might also have made it easier for these individuals to induce trance states right from the beginning. Dissociative states like trance and possession are characterized by a pattern of narrowly focused attention and selective awareness (Seligman and Kirmayer 2008), and the ability to regulate arousal is directly correlated with attention regulation (Hansen et al. 2003; Porges et al. 1994). In fact, previous research has specifically demonstrated that high autonomic regulatory capacity is associated with the capacity for absorption (Zachariae et al. 2000), or intensely focused attention. This suggests that high CAR among prospective mediums could have made them particularly suited to the techniques of trance induction. In this scenario, a higher capacity to regulate arousal helped make people like Jalita more capable of focusing intense and exclusive attention on the drumming and singing, ritual setting and ritualized actions, meaningful sensations and spiritual significance, of ritual enactments and experiences.

In either scenario, then, the bodily qualities represented by higher levels of CAR are likely to have interacted with meaning and practice to shape *filhos'* experiences both before and after their engagement with Candomblé. But while these physiological characteristics undoubtedly had important effects on mediums' own experiences, it is also worth considering the possibility that through the performances in which they enable mediums to engage, these qualities also affect other members of the Candomblé community. As I have discussed in depth elsewhere (Seligman 2014) shared practices are crucial to Candomblé. Mediums play a central role in the religious life of the group by bringing everyone closer to the spiritual power of the gods. At the same time, mediums depend on others to support the transformations they experience as a result of their initiations. Thus, there is a reciprocal relationship between mediums and the rest of the community, which may extend to the physiological dimensions of trance and possession as well.

Sociologist Randall Collins (2005) argues that certain kinds of rituals are characterized by "a process in which participants develop a mutual focus of attention and become entrained in each other's bodily micro-rhythms and emotions" (47). What Collins means is that ritual practices serve to synchronize the experiences of the individuals present—creating shared behaviors and emotions that may themselves become the focus of mutual attention. For example, a process of joint attention between mediums and audience members during ritual enactments of trance and possession may result in a kind of co-construction of the experiences of absorption, physiological arousal, and spiritual transcendence. I am suggesting the possibility that the processes of mutual entrainment that characterize many rituals may have a psychophysiological dimension. In this case, mutual entrainment, or the synchronization of body and emotion among participants, might include sharing of the autonomic nervous system responses of mediums by others during Candomblé possession rituals. A similar phenomenon was documented during a fire-walking ritual in Spain, in which the pattern of arousal of the ritual participants was mirrored by a similar pattern of arousal among related spectators (Konvalinka et al. 2011).

Thus, the qualities of mediums' bodies that allow them to embody the spirits and deities through possession, may also contribute an embodied dimension to the way in which they bring the rest of the community closer to the spiritual force of the gods—helping lay-people to really *feel* the presence of the deities. Such a phenomenon could contribute to a group level bio-looping process, in which joint attention and shared bodily experiences reinforce spiritual meanings for all involved.

Conclusion

Elsewhere, I elaborate on the ways in which the process of bodily transformation I have been describing comes together with a process of narrative transformation of the self, in order to create the experience of an encompassing self-transformation (Seligman 2010, 2014). Building on the model presented here, I make the argument that bio-looping effects can create mutually reinforcing, therapeutic changes in bodily and cognitive dimensions of selfhood. By analyzing how the meanings and practices of Candomblé specifically contribute to such an integrated process of self-healing for mediums like Jalita, I argue that bio-looping can illuminate processes of self-transformation and the role of meaning and practice in healing more broadly (Seligman 2014). A full presentation of this discussion is, however, beyond the scope of this chapter.

Here, I have focused on the elaboration of a novel model for thinking about the mechanisms of "embodiment." In particular, I provide evidence that the embodied learning involved in trance and possession is reflected in psychophysiology regulation among mediums. In doing so, I provide a way of understanding how biology is implicated in processes of embodiment, demonstrating how cultural and social meanings and practices shape bodily states and the qualities of individual bodies, which in turn shape processes of cultural and social learning and uptake.

Because the interactions of body, meaning, and practice are so highly visible in trance and possession, it in many ways represents an ideal site for examining bio-looping processes. But the bio-looping model also applies more broadly to many everyday processes of socialization and enculturation. To give just a few examples: the process through which children learn to experience and express socially appropriate emotions, learning embodied skills like playing a musical instrument or practicing a martial art, and the health effects of psychosocial stress, can all be understood in terms of bio-looping and the feed-forward interactions among experience, physiological response, and meaning. By calling attention to the way that particular embodied selves, shaped through experience, may in turn be predisposed to particular kinds of experience, bio-looping helps us to think about the developmental trajectory of embodiment. As such, it has the potential to illuminate enduring questions in psychology and anthropology about why, all other things being more or less equal, some people are more vulnerable to depression, alcoholism, or Post Traumatic Stress Disorder (PTSD). Moreover, since bio-looping is fundamentally based on a notion of biological plasticity, bringing this concept to

bear on such questions helps us to resist more reductionist approaches that would answer such questions in terms of genetics alone.

Notes

1. Mediums had a mean of 4 somatic symptoms on a psychological inventory, compared to a mean of 3.3 for the other participants. This difference was significant at the $p = 0.05$ level when non-religious control groups were included in the analysis.
2. *Ogās* and *ekédis* make an excellent comparison group because they are similar to mediums in their level of dedication to and responsibility within the religion. Their primary difference from mediums is that they do not become possessed or enter trance states.
3. In impedance cardiography measures resistance to electrical stimulation in the thorax caused by changes in blood volume as the heart ejects blood into the periphery. For methodological details, see Seligman (2014).
4. CAR scores were compared by group membership using ANOVA, and results indicate a trend in the differences between mediums and non-medium initiates: baseline CAR scores of mediums are a half a standard deviation higher than those of non-medium initiates.

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Experimental Entanglements: Social Science and Neuroscience Beyond Interdisciplinarity

Des Fitzgerald and Felicity Callard

Introduction

A spectre haunts social science: the spectre of the brain. We are, writes the historian Roger Cooter, in the midst of a pernicious 'neuro-turn', in which scholars assume that, among other things, '"the social," and "life" itself have… undergone a refashioning as a result of the new life sciences in general and neurobiology in particular' (2014, 146). With the advent of this turn, the anthropologist Emily Martin argues,

> we are seeing the effects of a form of reduction that is likely to impoverish the richness of human social life….Social practices involved in gift giving, child raising, courting, working, cohabiting, co-organizing and a myriad others—all situated in particular contexts, times and places—fall out of the picture and do not return. (2010, 369)

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Such laments—and they are not idiosyncratic (Ortega and Vidal 2007; Choudhury et al. 2009)—should be situated within a broader anxiety, evident in the humanities and social sciences in the last decade, about the increasing tendency for researchers, from several disciplines, to blur the boundaries between the traditional concerns of a social or humanistic interest, and the technologies and methods of the neurosciences. Many interpretive and humanistic scholars have thus begun to sense, within their once-secure intellectual domains, the soft, ominous tread of the new brain sciences (Cromby et al. 2011). If this intellectual development is truly a 'refashioning of our older disciplinary habits of the heart', Cooter continues, in an unusually dramatic intervention, then there can be 'no task…more vital and urgent' than its critique (2014, 154).

Perhaps we should not be too surprised by such talk: 'the materiality of the world', Helga Nowotny reminds us, has a tendency to 'upset the existing intellectual division of labour, and the cognitive and practical order upon which boundaries rest' (2005, 24). But if we are indeed living in a neurobiological age, what are we actually to do—and we use we here performatively, to gather together social theorists, humanists, and qualitative social scientists—when the webs of human social and cultural life that we had come to understand as *our* particular object of knowledge, seem more and more open to being figured neuroscientifically and experimentally?[1] Certainly, solutions have been offered—that we subject the new brain sciences to a refined sociocritique (Ortega and Vidal 2007); that we demand their political reform (Choudhury et al. 2009); that we welcome them into cultural theory (Wilson 2004a, 2011); that we use them to upset our taken-for-granted assumptions (Stafford 2008); that we embed them within our accounts of the political (Connolly 2002); that we regard their deconstruction of subjectivity as more effective than Derrida's own (Malabou in Johnston and Malabou 2013); that we join them (Roepstorff et al. 2010); that we analyse them (Dumit 2004; Cohn 2008); that we reject them (Martin 2004); that we accept them (Franks 2010); that, taking the longer view, we locate them within a much thicker braid of social and biological torsion (Rose 2013). So, on and on, go the debates.

We are in various states of agreement and disagreement with these proposals. Recent calls by Rose (2013) and by Rose and Abi-Rached (2013) for new ways of figuring the space between the social- and neurosciences have been particularly important for what follows. But we want also to expand that discussion into a new terrain: the terrain of the experimental. If there has been extensive discussion of what these developments entail conceptually and institutionally for the social sciences and humanities (Cromby 2007; Pickersgill

2013), there has been less critical attention given to what the rise of the neurobiological age might entail for the social sciences and humanities *methodologically* and *in practice.* If a wider social-science literature is taken up with expressions of straightforward gratitude for, or equally straightforward rejection of, findings from neuroscientific experiments, there has been little suggestion that *experimental labour itself* might be worthy of sustained attention from social scientists and humanists.[2]

What would happen if we changed the spatio-temporal dynamics of this scene? What if social scientists and humanists moved away from conceiving the domains of the neuroscientific and the experimental as the unchallenged province of the brain sciences—whose apparent territorial expansiveness they must welcome, ignore or repel? Could the neuroscientific *experiment*, as a rich and ambiguous way of producing different knowledges, help us to think some more creative and entangled ways of exploring these questions? In this chapter, we claim another intellectual space, cutting across the contemporary neurosciences and social sciences. There are three elements to our proposal: (1) If there is now much critical, conceptual discussion about the space 'between' the social- and neurosciences, there is strikingly little attention to how *methodological* novelty, serendipity and contingency might conjure a more constructive space of shared collaboration. (2) A turn to 'experiment' offers an entry point to this space. We fix on experiment because it captures both (i) the means by which cognitive neuroscience derives many of its epistemological claims from laboratory practices, and (ii) a wider ethos of openness to different procedures of action and investigation (Morawski 1988). And if we are preoccupied with cognitive neuroscientific experiments that employ magnetic resonance imaging (MRI) (Poldrack 2010; Bandettini 2012), our proposal extends an invitation to other histories and territories of 'experimental entanglement'—to correlational or observational studies, to clinical spaces, to behavioural research or indeed to any other site of the broadly conceived experimental repertoire of the new brain sciences. (3) Through our turn to methodological novelty, and to experiment in all its guises, we propose what we believe to be a more compelling platform for scholars who may have some urge, now, to think through the intersections of neurobiological and social life. In particular, we want to help such scholars circumvent a burgeoning, but bloodless and sterile, literature on 'interdisciplinarity' *between* the social sciences and the life sciences. 'Experimental entanglements' is our name both for a new way of addressing these questions *and* for the contingent, unstable, fleeting empirical commitments in which that argument is embedded.

In what follows, we do not follow these threads in order, but work them through a four-part argument: First, we consider recent developments in the

neurosciences, and we show how collaborative possibilities for the 'social' sciences have opened up around them. Second, we offer a sustained analysis of literatures that have already interpreted this space, which we group under three headings: *critique*, *ebullience* and *interaction*. Third, we argue that this entire discursive space is torqued by a series of epistemological and ontological commitments that limit the scope of collaboration between the neurosciences and social sciences. We name this limitation 'the regime of the inter-'. Fourth, we elaborate our own programme of 'experimental entanglements', and we argue that our interest in contingent, fleeting moments of methodological novelty may offer potent possibilities for inhabiting the space we have identified. At the heart of the chapter is an argument for rethinking the laboratory-based experimental domains of the cognitive neurosciences as both spaces and moments for firing strange alliances between neuroscientists and social scientists.

In a related publication (Callard and Fitzgerald 2015), we offer pragmatic advice on interdisciplinary interaction for collaborators from all disciplines. But this chapter has a narrower remit: here, we intervene in *internal* discussions, within the social sciences and humanities, about possibilities for, and encouragement towards, collaboration with the neurosciences. Our interest is in significantly expanding that conversation: the chapter is aimed at scholars within those disciplines who have some urge towards concrete engagement with the neurosciences, but who remain unmoved by today's arid rhetoric of 'interdisciplinarity'. The unabashedly programmatic aim of this chapter is to put pressure on the usual ways in which such possibilities between the social sciences and the neurosciences are understood (e.g. European Commission 2011). Our chapter sets out the core conceptual ground for the elaboration of an alternative programme, paying particular attention to the 'experiment' as a space of intervention, and using 'entanglement' explicitly to depart from logics of 'engagement' and 'dialogue'.[3]

Why the Neurosciences?

Today, cognitive neuroscience[4] is frequently held up as the greatest intellectual resource for the humanities and social sciences (Pinker 2013)—or the gravest intellectual threat (Tallis 2011). This prominence is inseparable from the neuroscientific claim on the 'space inside the skull' (Beaulieu 2000), that prized locus of so much interpretative scholarship. If there is still much research to be done on the uneven historical and geographical contours of

neuroscientific authority, it remains undeniable that many facets of human life that were, for much of the twentieth century, primarily understood through the abstractions of 'culture' or 'society'—commercial and economic life, governance, historical change, identity, distress and suffering—are increasingly understood as functions of the cerebral architecture of individuals or of groups of individuals (for examples, see Adolphs 2003; Camerer et al. 2005; Chiao 2009; for reflections, see Rose 2010; Vrecko 2010; Matusall 2012).

There are many ways to respond to this social fact. We start, here, from the realization that in a growing number of research areas, bioscientists, as Nikolas Rose maintains, increasingly characterize

> living organisms as dynamic and complex systems, located in a dimension of temporality and development, and constitutively open to their milieu—a milieu that ranges in scale from the intracellular to psychological, biographical, social and cultural. (2013, 5)

Indeed, and especially within the new brain sciences, it is clear that, just as technologies have emerged to measure the workings of the central nervous system in vivo, so is that system becoming conceptually inseparable from the social, cultural and familial contexts in which it developed: biology, Maurizio Meloni points out, 'has become porous to social and even cultural signals to an unprecedented extent' (2014, 2; cf. Bird 2007; Hyman 2009; Niewöhner 2011). On the one hand, of course, this presents a significant opportunity for social scientists. As a recent *Nature* editorial pointed out:

> Sociologists have been studying human environments for decades, and have tallied the social damage that stresses such as poverty or child abuse can cause. Biologists are now in a position to benefit from their insights. (*Nature* [editorial] 2012, 143)

If we are not bowled over by this description of what sociological labour might offer, it seems indisputable that there is something important that social scientists now 'offer' the life sciences. As Ilina Singh points out, the 'emerging disintegration of the nature-nurture divide' from within the biosciences offers a new collaborative space for social scientists (2012, 316–317). And the neurosciences, especially, Rose and Abi-Rached remind us, are currently 'struggling towards a way of thinking in which our corporeality is in constant transaction with its milieu' (2013, 3). We are in significant agreement with both the general claim that social science has something to offer, and that new

forms of collaboration should be risked in order to grasp these opportunities. But here we append two further remarks.

First, there is a risk of these careful arguments being (mis)interpreted as encouragement to leap faithfully into a newly socialized biology. But we are painfully aware that the 'social' of a 'social neuroscience' is often a rather mangy-looking beast—an animal quite alien to the rich and fat understanding of a century-old anthropology or sociology (Matusall 2012). We also worry about how 'culture' is commonly imagined as just another input within a straightforwardly bioscientific schema, and we know well that awkward questions remain about the epistemological politics at stake within these generous-looking invitations (Choudhury and Kirmayer 2009; Young 2012). We cannot ignore, as scholars trying to make a space for our interests within a shrinking, instrumentalizing academy, the shifts in scholarly prestige that surely guide, for example, the increasingly warm *rapprochement* between analytic philosophy and cognitive neuroscience (e.g. Smith 2012). We have squirmed our way through too many 'interdisciplinary' meetings to remain innocent of just how narrowly the world outside the skull sometimes gets figured within these 'biosocial' narratives.

Second, and this is where our chapter finds much of its impetus, it has not been easy to imagine or specify how these collaborations might be enacted in practice. This is an intrinsically vexed question, and we offer no simple solution here. As we will argue below, however, one way to move the discussion forward is to think more creatively about *experiments*. While we have been inspired by broad calls for social scientists to take up new possibilities for collaboration, we have often been dismayed by the narrow rhetorics and frameworks of interdisciplinarity that seem to govern actual, real collaborative spaces *beyond* those calls. And yet, at the same time, our collaborative imaginaries have consistently been fired by experimental moments—admittedly often short, contingent, serendipitous—that we have painstakingly sought, located and nurtured within such spaces. There *are*, now, real opportunities for collaboration between the social and neurosciences. But these opportunities are often occluded by the narrow discursive range of contemporary 'interdisciplinarity'. This chapter therefore draws attention to some more *experimental* modes for re-imagining that space. Before we elaborate on our own approach, we first distinguish it from the most prominent modes through which the relationship between the social sciences and the neurosciences has hitherto been understood.[5] In line with our programmatic aim, we have distilled the core features of heterogeneous and expansive endeavours. We trust that the benefits of clarity outweigh the risks of caricature.

Three Modes of Neuro-engagement

Critique[6]

Arguably the most common way of positioning the social sciences and humanities in relation to cognitive neuroscience is to interpret their task as the critique of neurobiological chauvinism. This mode uses the tools of historical, social and cultural analysis as external methods to either (1) uncover unconscious or hidden biases within the new brain sciences, and to locate nefarious social, political, economic and epistemic agendas within them (e.g. Ortega and Vidal 2007; Choudhury et al. 2009); or (2) deflate particular neuroscientific trends or claims that have found favour within the humanities or social sciences (e.g. Ashton 2011; Kramnick 2011).[7]

These engagements commonly lean on long-standing claims concerning the fundamentally *sociocultural* nature of scientific (including neuroscientific) knowledge (Pickering 1992). The most compelling articulations of this critique come from a trio of scholars and groups who have argued, trenchantly, for: the fundamentally sociocultural basis of the neuro-reductionist urge (Martin 2004, 2013); the political ill-effects of this urge on our senses of self (Ortega and Vidal 2007; Vidal 2009), and the need for the new brain sciences to be radically re-imagined (Choudhury et al. 2009; Slaby and Choudhury 2012). Emily Martin was perhaps the first to identify the emergence of a cultural figure whose levels 'begin with molecules, but go no farther than the central nervous system' (2000, 574). Thus, Martin argues, 'all of what anthropologists call culture has drained through the hole and dissolved in the realm of neural networks' (2000, 576). Martin locates the cultural and institutional desire for the 'restraining force' of this 'ahistorical concrete body' in manifestly social developments: for example, in the need for a reaction to the mania and wildness of *fin de siècle* capitalism (2000, 576, 581), in psychiatric-expert attempts to 'snare' the 'criteria of rationality' and the 'meaning of language' (2004, 194) and in 'contempt for anything that limits the kind of commensurability that our markets and systems of governance demand' (2013, s157).

But there is a deeper point embedded here, and this is Martin's argument for the ontological primacy of the sociocultural over the neurobiological, in order to 'detect the real prejudices hidden behind the appearance of objective statements' (Latour 2004, 227). Fernando Vidal, similarly, has argued that attempts to locate some organic and naturalized account of the self in fact long precede the emergence of the new brain sciences—that this is an ideology on to which neurobiology is mapped *post hoc*: 'the idea that "we are our

brains" is not a corollary of neuroscientific advances, but a prerequisite of neuroscientific investigation' (2009, 7). A related argument has been made by the exponents of 'critical neuroscience' (Choudhury et al. 2009; Choudhury and Slaby 2012). The essence of their account, which is inspired by the Frankfurt School,[8] is not to tear down neuroscience, but to inculcate among neuroscientists:

> self-critical practices, which aim to achieve reflective awareness of the standpoint-specific biases and constraints that enter into the production, interpretive framing and subsequent application of neuroscientific knowledge. (Choudhury et al. 2009, 65)

In other words, neuroscience *itself* should be reformed as a critical practice, and become aware of its own political and economic standpoints. But neuroscience must *also* harness the 'emancipatory potential' for neuroscientific workers to reflexively labour upon the biases embedded in their own practices (ibid., 65). Once again, the point is to understand 'neuroscience itself as a cultural activity'—to re-situate it within a 'social structure' and re-formulate it as a practice run through with economic drivers, political climates and cultural contexts (ibid., 62–64).

Such critiques are salutary reminders of the need to devote analytical attention to the 'logic of the neuroindustry'—and there are resonances between our proposal and some of the more pragmatic steps proposed by scholars in this tradition (Slaby and Choudhury 2012). But the stance of critique tends too readily to wield the master term 'reductionistic' to characterize both neuroscience's own knowledges *and* its effect on other disciplines (e.g. Kirmayer and Gold 2012). In fact, an insistence on 'reduction' renders much of what is most analytically interesting about neuroscience—including its relationship to other domains, and how those relationships might be re-imagined—invisible. One central example comprises the fascinating and novel ways in which 'culture' and 'neurobiology' are drawn *together* and how bodies and cultures have become experimentally legible *in* one another (e.g. Lende and Downey 2012, 23). In fact, relations between metabolic brain processes, sociocultural environments and 'mental processes' are being repeatedly experimentally *re-adjudicated* in cognitive neuroscience. And this is just one instance of the uneven and creative ways in which the dynamic relationship 'between'—though that is not quite the right adjective—the 'neurobiological' and the 'cultural' is kept in play (Callard and Margulies 2011).

Where we most significantly depart from colleagues in the critical tradition is in our refusal to cede ontological primacy to the *sociocultural* within this

terrain, certainly in light of the far-reaching theoretical challenges that have been launched at such a premise (Whitehead 1964; Haraway 1991; Braidotti 2006). Interestingly, the critical literature often perfectly well sees, but then usually scotomizes, the complexity and subtlety of the new brain sciences—missing, in particular, how they think through, and work on, the tangled imbrication of bodies, brains, minds, subjectivities, lives and machines. Kelly Joyce, for example, draws on a powerful image from Elizabeth Grosz (1994) to suggest that MRI images '"etch together" local decisions and priorities, technology, and aspects of the physical body to produce what is perceived as cutting-edge, authoritative knowledge' (Joyce 2008, 70). But what gets missed in Joyce's desire to show that 'there is nothing natural or inevitable' about MRI is precisely the intellectual force of a science that *can* 'etch together' local politics, de-oxygenated blood, sick bodies, nuclear physics and the clinical gaze, to produce what for many is a convincing image of a person, and a body (ibid., 20).

Ebullience

If much neuro-critique is built on a presumption of the ontological primacy of 'culture', then the 'ebullient' mode tends to take experimental results and theoretical statements from the neurosciences as more-or-less true—with little contest or context, and in the absence of a sense of the wider, often fierce, epistemological and ontological debates *within* those sciences. As Papoulias and Callard (2010) have argued, the emergence of what is commonly now known as 'affect theory' within cultural studies has often been the ground for such enthusiasm. Here, many social and cultural theorists rest accounts of the dynamic inter-relations between cultural theory and neuroscientific fact via skilled and lengthy attention to the former—and surprisingly thin, often naïve, summaries of the latter.

Strikingly, many ebullient engagements with the neurosciences from humanists and social scientists barely stray further than scientists' 'crossover' publications for lay audiences (here, Damasio's volumes [2000, 2004, 2006] are highly favoured)—evidence of the strangely credulous and limited reading practices of those who accrue intellectual capital *precisely for* the acuity and breadth of their reading. The philosopher Catherine Malabou, for example, has provided one of the most provocative and renowned accounts of how current research in the life sciences (and particularly the neurosciences) pushes beyond post-Husserlian conceptualizations of subjectivity in Continental philosophy (2008, 2012; Johnston and Malabou 2013). Central to Malabou's

argument is her conviction that current neurobiology effects wide-ranging transformations in understandings of affect, producing a more radical challenge to conceptualizations of subjectivity than those articulated by deconstruction and psychoanalysis: 'Current neurobiology is engaged in a deep redefinition of emotional life', Malabou argues:

> The brain, far from being a nonsensuous organ, devoted solely to logical and cognitive processes, now appears…to be the center of a new *libidinal economy*.… A new conception of affects is undoubtedly emerging. (Malabou, in Johnston and Malabou 2013, 3)

But this authoritative characterization concerning the huge and heterogeneous field of neurobiology is founded almost entirely on Malabou's enthusiastic reading of a very select number of scientists who have published for a general audience. And while Malabou's monograph *The New Wounded* (2012) is full of acute and contrapuntal readings of Freud, her engagements with the neurosciences are largely restricted to adulatory reiterations of sentences from Antonio Damasio, Joseph LeDoux and Oliver Sacks. In developing our own formulations about our relations with the neurosciences, we have gained much from the audacity of Malabou's forays. But what we miss in her publications is a strong sense of scientific nuance and breadth: Malabou's monographs demonstrate limited engagement with peer-reviewed scientific publications, with internal criticisms of Damasio, and with histories of science—any one of which might provide a thicker, more adhesive texture for claims regarding a field's 'deep redefinitions' and the challenges these pose to theorizations from the humanities.

Or consider Brian Massumi's influential essay, 'The autonomy of affect', which aimed to provincialize a reliance on signification and language in cultural theory by drawing attention to the 'dynamism' of the neurological sciences (1996, 100). As we have ourselves become more intimately involved with experimental spaces, it strikes us that the neuroscience that emerges through Massumi's account is, in contrast, not at all dynamic, or flexible, or even very interesting. Neuroscience is in fact figured by Massumi as lumpen, univocal and tediously certain. Moreover, the science on which Massumi's theoretical claims rest makes startlingly brief appearances—accurately characterized by Ruth Leys as a 'strategic' and 'fleeting' service for Massumi's 'rather opaque philosophical-speculative reflections' (Leys 2011).

'The manner in which "science" is often invoked in cultural theory texts', Papoulias and Callard point out, 'testifies to a desire for a certain kind of revelation that science will be able to satisfy' (2010, 36–37; see also Barnett

2008). Authors in the ebullient tradition, in their desire to designate generative spaces for the mingling of biology and culture, unintentionally *foreclose* the space for a dynamic and mutually constitutive traffic across them; they are much too willing to assign to the natural and experimental sciences the task of generating the findings that will confirm, verify and/or reveal the theoretical insights of cultural and social theory. If this mode of engagement with neuroscience is characterized by ebullience towards its desired objects and partners, it tends to remain demurely secluded from the hubbub of experimentation itself.

Interaction

A relatively small group of scholars has, in recent years, begun to undertake the rather thankless task of locating a conceptual space between the social sciences and the neurosciences—while resisting the attention-grabbing rhetorics of critique or ebullience. What we term the 'interactive mode' is characterized neither by a desire to provincialize the pretensions of the neurosciences nor by an uncritical acceptance of insights from those spaces. Instead, scholars focus on research on humans' neurological propensities but, crucially, they also maintain an epistemic parity between this research and the traditions and paradigms of the interpretive and social sciences. These works grant the same kind of sustained and critical attention to neurology and neurobiology as they do to the interpretative social sciences. They read, in the neurosciences, a complementary desire for mutuality, and a willingness to allow insights from sociocultural theory to fold back onto neuroscientific research; in so doing they strive for a neurobiology that might help to develop different kinds of theories about the contemporary figure of the human *as such*.

In *Neuro*, for example, Nikolas Rose and Joelle Abi-Rached argue that new styles of thought emergent in neuroscience

> offer the possibilities of a more positive role for the human and social sciences, an opportunity to seize on the new openness provided by conceptions of the neuromolecular, plastic, and social brain, and to move beyond critique and find some rapprochement. (2013, 24)

Such a rapprochement, they argue, may even contribute to a new kind of progressive thought—refusing an account of human societies as composed of maximizing individual organisms or of governmental modes designed to regulate such organisms (2013, 234). 'At their most sophisticated', Rose and

Abi-Rached suggest, '[the neurosciences] are struggling towards a way of thinking in which our corporeality is in constant transaction with its milieu, and the biological and the social are not distinct but inter-twined' (2013, 3). Other scholars in the interactive mode have tried to mobilize such transactions: Andreas Roepstorff (2001), for example, has used his dual identity as a brain imager and a cultural anthropologist to revive the animalistic, world-experiencing 'biophilosophy' of Jakob von Uexküll and has argued (Roepstorff et al. 2010) that rethinking forms of social interaction as 'patterned practices' might operationalize the entanglement of cultural and neural networks.[9]

Another sustained attempt to re-calibrate relations between the neural and the sociocultural has been made by Elizabeth Wilson (1998, 2004a, 2004b, 2010, 2011), whose broad project works the neurological into feminist accounts of the body, and to feminist theory more generally. In tandem with other accounts that have mobilized scientific literatures to explore and conceptualize affective relationality (Sedgwick 2003; Blackman 2008), Wilson's cultural-theoretical project pursues a mingling with neurology in terms of the 'potential in the neurosciences for reinvention and transformation' (2004a, 13). She argues that between psychology and neurology, 'forces of influence and determination are more mutually entangled than the critics of neurological determinism have hitherto acknowledged' (2004a, 16). In the circuit of body, psyche and environment, we do not find a relationship of simple causation but rather 'a system of mutual constitution from which no particular element emerges as the originary, predetermining term' (2004a, 19). Thus: 'neurological material is more confident, flexible, resilient, and assertive than many critics have yet acknowledged' (2004a, 22). This, on Wilson's account, is what socio-critique prevents us from seeing: 'by disconnecting biology from its constitutive relations with other ontological systems', she argues, 'biology becomes isolated and destitute' (2004b, 70).

Our project of experimental entanglement is indebted to this stance. Following Rose, we are in pursuit of 'an affirmative relationship' with an emerging 'new and non-reductive biology of human beings and other organism in their milieu, which can thus be brought into conversation with…the social and human sciences' (2013, 24). With Wilson, we seek a neuroscience that 'may…be a resource for theoretical endeavour, rather than the dangerous and inert substance against which criticism launches itself' (Wilson 2004a, 29; cf. Stafford in Turnbull 2007, 347). The work that remains, then, is to think about how such insights can be realized in empirical projects or how they can be more concretely situated within a more expansive research practice. While we are hardly the first to pursue this question, our experience is that when similar programmes *are* moved onto a more empirical terrain, the

core insights of the interactionist mode have been hard to maintain. Too frequently, soft boundaries between social and neural are maintained through a model of disciplinary partnership (e.g. Lende and Downey 2012); the biosocial nexus starts to look distinctly bio-centric (e.g. Chiao 2009); the empirical project distances itself from (and thus struggles to move) the core concerns of sociocultural knowledge (e.g. Roepstorff and Frith 2012); or the disciplinary 'role' that each intellectual party plays in the programme becomes solidified, such that the possibilities for folding insights *across* epistemological domains are reduced (e.g. Sambo et al. 2010). We see a gap, then, in which the final step is not yet enacted in practice or where there tends to be a limited working-through of the dynamic complexity of the ontological and epistemological reshufflings that might be enacted *through* such practice. It might, indeed, be such an absence that has allowed the more critical and ebullient voices to dominate the debate within the social sciences.

The Regime of the Inter-

The modes of 'critique' and of 'ebullience' seem to sit at opposite ends of the spectrum. But we suggest that they are animated by a shared commitment—namely, that the sociocultural and the neural are different domains of knowledge and that they address themselves to different kinds of objects or to different aspects of objects. For the critic, a commitment to this divide between the sociocultural and the neural means defending the boundary points and re-asserting the strict differences between the two areas.[10] For the enthusiast, the divide describes instead a hierarchized division of labour—and a willingness to render unto the neurosciences what is truly neuroscientific. But if the critic and the enthusiast are very different from one another, they share the most important commitment: namely, that there are things, and ways of knowing things, that are sociocultural; and there are things, and ways of knowing things, that are not. The only difference is that the critic insists that this is how it should be, whereas the enthusiast would rather redraw where the line falls, in acquiescence to new neuroscientific knowledge about (what were previously thought of as) sociocultural preoccupations. But this is a trivial distinction. The existence and salience of what is really important here—the dividing-line itself—is never in question. Slaby and Choudhury, for example, place 'particular emphasis on the social' in the face of a fashionable and shallow 'ontological hybridization' (2012, 36–37). Von Scheve, by contrast, calls on sociologists to attend to 'actual neuroscientific findings' (2012, 256). But for each of them, there is a thing called social science that

addresses itself to one kind of object; and there is a thing called neuroscience that addresses itself to another. The only controversy is about whether current flirtations between the two should be consummated. This debate thus operates entirely within an unquestioned, shared space, which we call *'the regime of the inter-'*.

The *'regime of the inter-'* refers all analysis about the space between the social sciences and the neurosciences to a guiding question: given that there is the possibility of overlapping interests and objects between these sciences, then how large should that space of overlap be, how should it be populated, what kinds of objects should be located within it, and what should count as a sufficiently ecumenical research programme to address those objects? But this regime excludes consideration of the history, topology and salience of that space *as such*; about the border-practices that bind it; and about how even the very preposition 'between' forecloses other ways of conceptualizing its characteristics, and the relationalities comprising it. Moreover, we contend that this regime governs most—if not all—of the institutional spaces that lay claim on what is seen as the growing need for *inter*disciplinary labour *between* the neurobiological sciences, and the social sciences and humanities.[11] Our intimacy over a number of years with a number of these explicitly designated 'interdisciplinary' spaces has strengthened our conviction that their governing ethic of epistemological seclusion (of the social sciences/humanities from the neurosciences and vice versa) is a recalcitrant fantasy—one premised on a sanitized history of disciplinary domains, of the frequent intimacies that have enjoined them, and of their respective objects of study (for alternative genealogies, see Donzelot 1988; Renwick 2012; Rose 2013). In this regime, certain visions of territory—along with the corollary concepts of borders, incursions and empire-building—tend to loom large. In contrast, our proposal takes for granted the conceptual, methodological and terminological crossings—admittedly often forgotten, often fugitive—that have long tacked back and forth between (and within) the domains of the sociocultural, the psychological and the neural, and that have been variously distributed within and across so-called disciplinary divides. We think, for example, of the genetic (and eugenic) history of early British social science (Osborne and Rose 2008), of the presence of non-human animals in a developing sociology (Shearmur 2013) or of the deeply uncanny biology bound within long strands of twentieth-century psychoanalysis (Laplanche 1989). Our interest, as both subjects and analysts of an emerging neurobiological age, lies in understanding how social scientists might best employ and re-energize that rich archive of crossings. We want to know how they—we!—might forge differ-

ent and unexpected relations, whether intellectual, methodological or affective, with the neurosciences.

Our proposal thus sets itself against the *'regime of the inter-'*. 'Experimental entanglements' start *in media res*, where there are neither neatly bordered disciplines nor any clear dispensation regarding which 'objects' of study are appropriate for each. Our gambit is that if a different sociocultural research practice—one that attempts to do epistemic and ontological justice to the fertile crossings between the so-called social and the biological—is to achieve any kind of epistemic force in the decades to come, then at least some of that force may come via recourse to a form of knowledge production that is, in fact, already aware of the potency of these exchanges: cognitive neuroscientific experiments.

Experimental Entanglements

Experiment: Entangled

At least since Ian Hacking's *Representing and Intervening* (1983), scholars have addressed experiment and experimentation as complex, knowledge-producing phenomena in their own right, rather than simple accomplices of scientific theory (cf. Galison 1987; Gooding et al. 1989; Davies 2010). Some of the most compelling research in the history of science has indicated that if we want to understand, or, indeed, help foment, the formation of new knowledge practices, we should not—as much discourse under the 'regime of the inter' does—focus our gaze at the scale of disciplines or paradigms. Rather, we should, as the historian of science Hans-Jörg Rheinberger has demonstrated in his work on modern experimental systems, be alert to:

> the digressions and transgressions of smaller research units below the level of disciplines, in which knowledge has not yet become labeled and classified, and in which new forms of knowledge can take shape at any time…novelties generated in one system can quickly spread and create effects at other places. (2011, 315)

With Rheinberger, we direct attention to spaces of experimentation in which the intersections between scientific 'objects', instruments, apparatuses and experimenters still quiver with uncertainty—where the liveliness of experimentation has not yet been stilled by epistemological resolution. A living experimental system, Rheinberger argues, has '*more stories* to tell than the

experimenter at a given moment is trying to tell with it' (1994, 77–78). Because such a system still holds 'excess' within itself, it 'contain[s] remnants of older narratives as well as fragments of narratives that have not yet been told' (ibid.).

This account of excess underpins our argument for turning to experiment in cognitive neuroscience. One of the distinguishing characteristics of the contemporary neurosciences is that, because of the still-recent emergence of novel methods and sub-disciplines affiliated to this area, as well as their ongoing shuffling and realignment, core methods and assumptions have still not been entirely ossified (Abi-Rached 2008). Certainly, this is subject to change, and some procedures and constructs—for example, the relation between the BOLD (blood oxygenation level dependent) signal, which fMRI picks up, and brain activity—have over time been 'black-boxed' in a Latourian (1999) sense. But our collaborations with neuroscientists have consistently thrown up instances in which our collaborators were already deeply preoccupied with which of many methods to employ, how best to instruct research subjects, how to understand the relation between subject and researcher, how to operationalize constructs (e.g. Filevich et al. 2013) and so on. Cognitive neuroscience is thus a field in which many experimental systems are (still) in motion (e.g. Le Bihan et al. 2001; *Neurocritic* 2012; Callard and Margulies 2011). It is not a desire for control that undergirds our positive turn to experiment. Quite the opposite: we are compelled by the promise of digressions, transgressions, mistakes and the subterranean existence of not-as-yet-played-out narratives.

A core goal of 'experimental entanglements' is to intensify the energies *already* within these experimental systems by seeding research projects and centres with researchers carrying heterogeneous modes of practice from the social sciences and humanities. We wish to do so because we want to *magnify* the productive untidiness, and temporal out-of-jointedness of those systems. An expansion in styles of taking measurements, using instruments, engaging with research subjects and tinkering with protocols might just help both to render and expose new biosocial stories (Rheinberger 2010, 218–219). Of course, we are not naïve about how unevenly epistemic and institutional authority is likely to be distributed across such entanglements, and we do not elide the unequal dynamics of power and prestige here. Nor do we pretend that the desire to rethink paradigms, and to tinker with protocols, is likely to be as strong for neuroscientists *en masse* as it might well be for collaborating social scientists. We have no fantasy of parity here—nor do we assume that the most congenial and democratic spaces are always the most interesting or productive (Fitzgerald et al. 2014). We remain sanguine—we have no choice

to act otherwise—about the likelihood of an experimental entanglement resulting in entropy, frustration or failure.

'Experimental entanglements' are modest, often awkward, typically unequal encounters that work to mobilize specific and often serendipitous moments of potential novelty in and outside the laboratory. These moments might reside in the methods chosen, the conduct of the experiment itself, the theoretical armature that surrounds it—or the roles that researchers play within the experiment, its analysis and in its dissemination. 'Experimental entanglements' refuse preliminary decisions about the shape or outcome of such an interaction: they denote an ad hoc process of shuffling histories, methods and assumptions from the social sciences and humanities *through* such partial moments and of picking through the scraps of knowledge and thought produced *by* the subsequent torsion. Our 'entanglements' are thus *never not* temporary, local assemblages of motivation, interest, people and machinery—in which we, and our collaborators, are able momentarily to think something exterior to both the conventions of experimental practice *and* the taken-for-granted dynamics of epistemic power that underwrite its conduct. This vision of being entangled is something very different from calls for neuroscientists to develop 'second-order observations of laboratory conditions, communities of scientists, and historical and cultural contingencies' (Slaby and Choudhury 2012, 42). Our model, through its attention to untidiness, excess and chance, strives to avoid such pre-determined demands for reflexive practice from either side. Instead, we seek the entanglement of researchers, instruments, writing practices, discourses, observations, archives, bodies, topologies and, in general, accounts of what that opaque object of neuroscientific research, around which all of these circle, just might be (Box 19.1).

On such a model, *our own* knowledge practices will also, of course, be bound up with specific entanglements of context, thought and affect. Attending to experiment demands attending to how the bodies, gestures and feelings of individual researchers are registers and generators of positive knowledge. Natasha Myers, in her ethnography of experimental manoeuvres within molecular biology, has described how scientists' bodily contortions can help to 'render' the objects of research; using the body, she argues, 'can generate both new forms of knowing, and the things known' (Myers 2012, 161, 172; cf. Fitzgerald 2013). We draw particular attention to this quality, because one of the most potentially fertile attributes of many cognitive neuroscientific experiments is the dynamism enabled by the fact that there are commonly *at least* two minds and bodies—that of the experimenter and that of the experimental subject—built into the experimental assemblage. What we might call 'the inter-subjective' is *always already* instantiated in both the practice and the data of cognitive

neuroscience—although this is rarely explicitly recognized in canonical texts (see, e.g. Frackowiak et al. 2004; cf. Schilbach et al. 2013). Such entanglements pose multiple trajectories for novel inquiry: who or what is the instrument? Who or what probes whom or what? Who or what yields data? How are relations of influence and connection between experimenter and experimental subject imagined, materialized, felt and traced out? Such combinatorial possibilities offer germs through which new forms of knowledge might emerge.

Box 19.1 The Neural Correlates of Deception: Imaging, History, Context and Feeling

In one entangled experiment, Fitzgerald and his colleagues (led by Melissa Littlefield) used neuroimaging to explore the deep and contingent intertwinements of truth, lie, situation and feeling (Littlefield et al. 2014; Littlefield et al. 2015; Fitzgerald et al. 2014). This group, made up of scholars from social science, literary studies, clinical psychology and neuroscience, took an historical and literary-critical commitment to the contingency of 'truth' and 'lie' *into* a novel neuroimaging design: through a highly artificial set-up (which induced participants to tell awkward truths), they played with, and expanded, the deeply situated nature of experimental design *and* an external sociocultural knowledge about the embodiment of deception, to produce brain-imaging data that troubled neurobiological distinctions between truth and lie. The researchers refused simply to exchange information between humanists, social scientists and neuroscientists but rather generated a loop in which the experiment was not only contextualized but in which context was also experimentalized. At the same time, the difficult compromise of the experimental mode, and the awkward, unarticulated feeling of being compromised, produced further reflection on the nature of truth and lie—as the experimenters wondered about their own small acts of equivocation and self-deception. Thus, and in another unexpected loop generated by the entanglement, just as the neuroimaging experiment worked to disrupt the biological legibility of deception, so did the experimenters' feeling of discomfort remind them of the deeply embodied nature of the same quality. The generation of these loops that work to resolve, but refusal to settle, the relationship between biological and cultural productions of truth, is precisely what we intend here with the notion of experimental entanglement.

Entanglement: Experimentalized

We have argued that it is increasingly difficult for the social sciences to maintain a potent hold on the expansive category of 'human life' while remaining indifferent to the complex neurogenetic textures of human capability. But while there is good reason, then, to cease the hygienic practices of many of the mainstream 'social' sciences (Goodman 2013), no new epistemic model has yet emerged to express this possibility. In promoting a return to experiment, we contend that the laboratory spaces of the new brain sciences offer hitherto

underused fora to draw out the tangled biological and sociocultural processes of human life. We situate the cognitive neuroscientific experiment—understood as a tumbling and uncertain mode of knowledge production—as one possible space in which both to register and to interpret these processes.

We draw inspiration from the work of feminist philosopher Karen Barad (2007), and her insight that sustainable and more-or-less bounded ways of producing knowledge might in fact come *after*—and not before—awkward mixtures of knowledge and material. Two features of Barad's recent work give energy to our proposal. First, her account of an 'agential realism' attempts to think a constitutive relationship between the mess and ambiguity of entanglement, *and* the confounding possibility of distinction or singularity—with the latter coming after entanglement, and not before. Thus Barad's approach:

> does not take separateness to be an inherent feature of how the world is. But neither does it denigrate separateness as mere illusion…relations do not follow *relata*, but the other way around. (2007, 136–137)

Barad argues instead for a metaphysics based on 'phenomena'—a term that designates *both* 'the ontological inseparability/entanglement of intra-acting agencies' and the 'primary ontological units' of the world (Barad 2007, 139–141; cf. Marres 2012). That the inseparability of agencies does not mitigate against 'determinate boundaries and properties of "entities" within phenomena' is crucial for our account of 'experimental entanglement' (Barad 2007, 148). Perhaps counter-intuitively, our approach wishes to similarly preserve *both* the fundamental inseparability of the biological and the sociocultural, *and* the possibility of a subsequent cut. If we refuse to position neuroscientific experiments as bounded or controlled spaces, we do not regard them as doomed to a morass of uncertainty. While we wish to affirm the ontological and methodological 'mess' of any neuroscientific experiment, we also contend that such experiments are able to produce meaningful knowledge about the biosocial complexities of human life.

Second, Barad refuses to separate the practice of science from the practice of studying science from the outside: 'the tradition in science studies', she points out, 'is to position oneself at some remove, to reflect on the nature of scientific practice as a spectator' (2007, 247). Barad invites us instead to think about the ways in which insights about the so-called 'social context' of science might also be intrinsic to the scientific practices in question (2007, 247). She posits a mode of engagement in which an 'understanding of the entangled co-emergence of "social" and "natural" (and other important co-constituted) factors' might best come from 'engaging in practices we call "science studies" together with practices we call "science"' (2011, 446).

With these two interventions, Barad proposes a radically different programme for sociocultural attention to, and 'engagement with', the natural sciences. In particular, she departs from modes of interdisciplinary engagement, which, as with all modes governed by the *'regime of the inter-'*, are premised on a recognition of the *solidity* of disciplinary borderlands (however deeply either envisages trade and exchange across those boundaries; see Galison 1997; Thompson Klein 2010). Our 'experimental entanglements' follow Barad in their insistence that neurobiological knowledge is a *product of*, and not a *precursor to*, disciplinary transaction—that the complex intersections of social and biological agencies come *prior* to, for example, the kind of agential cut that critical neuroscience insists on maintaining. Indeed, in a formal sense, introducing 'critique' and 'context' really does pollute the neuroscientific experiment—but precisely because this insistence *reduces* entangled complexity to a series of distinctive and competing perspectives.

There are costs to taking this position seriously—as we do. In particular, because Barad's conception of entanglement insists on the ontological *priority* of intersection, it becomes methodologically fruitless, in the kinds of experiment we envisage, to delineate distinct tasks, inputs and divisions of labour for 'social scientists' and 'neuroscientists' in advance. It is not a commitment to obscurantism that makes us resistant to clearly setting out, for example, 'who' might do 'what' within an 'experimental entanglement'. Rather, we maintain that ideas about 'who' and 'what' must remain in play when we proceed on the assumption that entanglements—of bodies, epistemologies, apparatuses, elements of experimental systems, operationalizations of terms—might produce something new in the world, even as the forms that that newness might take are undecided, and undecidable, prior to the moment of experimentation (for example, see Box 19.2).

We are insistent that this suggestion is not opposed to the ethic and ethos of experiment as such (e.g. see Donna Haraway's (1997) 'modest' modes of

Box 19.2 Experimenting with 'Rest' in fMRI Research

In another entangled experiment, Callard has been working with a neuroscientist (Daniel Margulies) to puzzle out how the nascent experimental paradigm of 'resting state' fMRI research emerged and how a narrative of this experimental emergence might be recounted to audiences 'inside' and 'outside' the neurosciences (Callard and Margulies 2014). (Resting state research evaluates regional interactions in the brain that occur when a subject is not engaged in an explicit task in the scanner; that there are strong correlations in BOLD fluctuations at rest is a recent finding—and one that was initially troublesome to the fMRI community; see Callard and Margulies 2011.) The collaborators made no prior determination about what kinds of knowledges might be required to understand

'rest': their contributing insights, which might, retrospectively, be distilled as physiological, anatomical, cultural, sociological, philosophical and theological, did not in fact adhere to each experimenter's supposed area of skill or expertise, nor did they divide neatly one from the other. This joint puzzling has also entailed both collaborators using heterogeneous methods (e.g. quantitative and hermeneutic) to interrogate 'rest' (e.g. Callard et al. 2013). By insisting on entwining their methods and epistemologies, the collaborators are developing novel genealogies of 'rest' that draw together different kinds of archives: they imagine stories about 'rest' that track, in unexpected ways, across the metabolic, the psychological and the cultural, while simultaneously planning experiments, in and outside the laboratory, that can bring these archives to light. This collaboration also pays particular attention to joined writing, working to disrupt a model in which the 'scientist' analyses and writes up the technical and empirical sections, and the social scientist pulls together the 'social and historical context' as well as the discursive analysis. Instead, the collaborators within this experimental entanglement specifically allow the experimental and narrative loops generated by this shared project to shift each collaborator into labouring within the prescribed space of the other collaborator.

engaging experimental spaces). Roepstorff and Frith, for example, in their reflections on neuroanthropology, direct attention to the experimental itself, as a productive object to think with: experimentalism, they point out, is 'a complicated practice, a *bricolage* tinkering with the possible elements (Pickering 1995) to make things work' (2012, 103). Thus, experiments do not—and are not supposed to—*settle matters*: an experiment in neuroimaging, no less than the much-analysed space of anthropological fieldwork, is variously intimate, awkward, lonely and boring; the generation of facts from data, in the neuroscientific laboratory, has never not been painful, messy, unsatisfying and contingent. With this in mind, Roepstorff and Frith encourage us to regard the 'experiment' not as a nitty-gritty, world-testing, fact-producing machine, but as a *performance*—and thus potentially as a risky, more avant-garde space. They describe this as an 'aesthetics of research practice', a mode of engagement in which the neuroimaging experiment becomes something akin to 'trying out new ways of writing, new ways of being in the field, or novel forms of intervention' (2012, 105). And they suggest that it is a form of aesthetic attention that allows the social scientist to take some kind of experimental rubric into her fieldwork. We linger on this description because we, too, are committed to using the experimental mode to rethink the ways in which relations across the social sciences and neurosciences are imagined and materialized. Our aesthetics of experiment fixes attention on the capacity of experimental intervention to unfold, to ally itself with, and then to elaborate upon, the determinedly entangled nature of human subjectivity.

Conclusion

We asked, at the start of this chapter, what might happen if we set aside our usual disciplinary allegiances and identifications to think more experimentally about the constitution and dynamics of the cognitive-neuroscientific-experimental domain. Our question was driven by our weariness with what we have described as the *'regime of the inter-'*: a regime which, we believe, has not only too frequently resulted in social scientists either clapping or barking at the neurosciences, but has commandeered both the imaginative and institutional space through which engagements 'between' the social sciences and the neurosciences might be envisaged. Our urge to disrupt this regime is motivated by our desire to move beyond the etiolated and benumbed visions of experiment and experimentation that, too commonly, are proffered under it.

The founding principle of an experimental entanglement is that it is 'discipline' that needs explanation, not promiscuity. What might be imagined as a securely 'cultural' or 'social' knowledge is a *product* of collaboration with the biological (and other) sciences: it is not a precursor to that collaboration. Our use of the term entanglement thus signals our growing suspicion that the central epistemological and institutional problem is not one of whether, or to what degree, disciplinary and epistemic boundaries might be crossed. The pressing question, it seems to us, is how, as human scientists, we are to produce knowledge amid a growing realization that those boundaries are pasted across objects which are quite indifferent to a bureaucratic division between disciplines; and that scholars and researchers of all stripes invariably attend to, and live among, objects whose emergence, growth, development, action and disappearance do not at all admit of neat cuts between the biological and the social, or between the cerebral and the cultural.

The labours of experimentation are frequently onerous and fruitless. And this has been as evident to some scholars in the humanities and social scientists—where there is also, of course, a rich legacy of experiment and experimentation (e.g. Clifford and Marcus 1986; Clough 2000)—as it doubtless is to many practising cognitive neuroscientists. But those labours can also yield unexpected harvests. We have argued that the cognitive neuroscientific experiment—understood as a kind of narrative excess, interpreted as an aesthetics, and approached with intellectual modesty—might be a space in which richer elaborations of human subjectivity might materialize than is commonly imagined. This is not a demand that the sociocultural and interpretive sciences 'reduce' themselves to the manipulation of laboratory apparatuses: ours is not a fantasy in which hordes of social scientists are re-directed from libraries and offices to the neuroimaging scanners in the basements. But it *is* a call for a

more expansive imaginary of what experiment—as practice and ethos—offers to practitioners within those disciplines. Our suggestion is that it might offer a moment in which some elements of the biosocial entanglement of human life are centrally at stake, and in which they might be brought into some kind of richer understanding. We have many more suggestions for what those moments might look like in practice (Callard and Fitzgerald 2015). This chapter establishes some of the core theoretical ground for our having made that move; it must end as an invitation to the interested reader to step outside the *'regime of the inter-*' and begin to trace her own trajectories of entanglement.

Notes

1. Note that 'webs of human social and cultural life' have been figured quantitatively and 'scientifically' in several social science disciplines for some time—not least in the archaeological and geographical sciences. Here, however, we address ourselves to those parts of the social sciences and humanities whose intellectual roots are in the emergence of the 'social' and/or 'cultural' as a distinct object of knowledge, and within which tentative, empirically focused turns towards biology have not been met with alacrity. See Donzelot (1988), Rose (1991), and Latour (2005).
2. Historians of science, sociologists and researchers in science and technology studies (STS) have taken 'experimental labour' as an object of study; we want here to explore how cognitive neuroscientific experimentation might be a methodological and epistemological resource for social scientists and humanists. We are indebted to (and expand upon) some recent exceptions to the general disregard for this question, such as Nikoleyczik's 'multidimensional' and 'integrative' approach (2012; see also Bluhm et al. 2012) and Roepstorff and Frith's (2012) ethic of conceptual 'front-loading.'
3. This chapter draws on our many years of separate and conjoined engagement with interdisciplinary neurobiological-sociocultural experimentation. What we here name as 'experimental entanglement' theorizes our long-standing frustration with the 'interdisciplinary' approaches that dominated these engagements. Here, we articulate the conceptual ground that lies beneath this frustration; more detailed case analyses of some of the 'entanglements' that we have helped to initiate are provided in Callard and Fitzgerald (2015).
4. In this chapter, we move between 'the neurosciences' and 'cognitive neuroscience'. The neurosciences incorporate a huge range of methods and foci that encompass molecular, cellular, developmental, structural, functional, evolutionary and computational studies of the brain in its 'normal' and 'abnormal' states (see Rees and Rose 2004, or Abi-Rached 2008). It is most commonly cognitive neuroscience that is the focus of much attention in the social sciences and humanities.

5. Some social scientific (and humanist) research, in approaching the neurosciences as an object of historical and/or sociological study, does not neatly fall into any of the three modes we delineate below. In this chapter, we are interested in social scientific scholarship that does not simply take the neurosciences as an object of study, but rather addresses how the growth in the neurosciences poses questions vis-à-vis how the social sciences might or should respond to this.
6. The concept of critique of course has great semantic density as well as a complex genealogy, as de Boer and Sonderegger (2012) demonstrate.
7. Some deflationary accounts leave open space for what they think might be more productive 'interdisciplinary ventures between the humanities and the sciences' (Kramnick 2011), but they tend, overall, not to be interested in the mechanics of such ventures.
8. Slaby and Choudhury argue, specifically, that:

> While critical neuroscience does not directly follow a Frankfurt School program … it does share with it a spirit of historico-political mission; that is, the persuasion that scientific inquiry into human reality tends to mobilize specific values and often works in the service of interests that can easily shape construals of nature or naturalness. These notions of nature or of what counts as natural … require unpacking. Without critical reflection, they appear as inevitable givens, universal and below history, and are often seen as a form of "normative facticity," making specific claims upon us in everyday life. (2012, 29)

9. Of course, the interactive mode, too, has a history—not least a history of transdisciplinary scholars, or those working in formative moments for their disciplines, who thought the experimental relationship between social life, psychological life and the brain. Particularly noteworthy here are the works of, for example, Kurt Lewin (1947) and Kurt Goldstein (2000 [1939]).
10. For example, see the concluding comments of Ashton, a literary theorist, in her critique of neuroaesthetics:

> This essay argues for why we should not just be delighted with the [neuroaesthetic] results, or rather, why we can't be delighted with the results and still maintain a coherent account of what we're doing when we're doing the interpretive work of literary or art history and criticism.….Neuroaesthetics is answering a set of questions about causes, while the interpretation of a work of art depends on having answers about its meaning.

11. See, for example, documents on the European Commission's unfolding 'Horizon 2020' research and innovation programme, which argues that:

> Radical breakthroughs with a transformative impact increasingly rely on intense collaboration across disciplines in science and technology (for instance, information and communication, biology, chemistry, earth system sciences, material sciences, neuro- and cognitive sciences, social sciences or economics) and with the arts and humanities. This requires not only excellence in science and technology but also new attitudes and novel interactions between a broad range of players in research. (European Commission 2011, 35)

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20

Developing Schizophrenia

John Cromby

Introduction

There are multiple, reciprocal and mobile relations between neuroscience, persons and their brains, and psychiatric diagnoses such as schizophrenia. Understandings of schizophrenia frequently implicate notions of the brain, its normal workings and its potentials to go functionally or structurally awry. Research in neuroscience is frequently justified with respect to its claimed potential to lead to new interventions for diagnoses such as schizophrenia. Significant research funding in the biosciences generally, including the neurosciences, has historically flowed from pharmaceutical companies seeking to develop and test new drug treatments for schizophrenia, and other related diagnoses (Rose and Rose 2012). These drug treatments have sometimes given rise to hypotheses about brain function in schizophrenia (and other psychiatric diagnoses), hypotheses which have sometimes proliferated throughout neuroscience more generally. Conversely, neuroscientific research into the structure and functioning of healthy brains continues to inform the development of new hypotheses about impaired structure or unhealthy function in relation to schizophrenia and related diagnoses. These developments are also

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being taken up within other biosciences, such as epigenetics, and within cognate disciplines including psychology.

In short, neuroscience and schizophrenia are closely linked in a dense, interdependent, evolving nexus of ideas, practices, technologies and knowledge. Conceptualizations of mental health and illness, concepts and images of brains, their parts and their functions, practices of treatment and intervention, and the somewhat disparate interests of multiple professions—most proximally, neuroscience, psychiatry, psychology, pharmacology—are continuously circulated and exchanged, and mutually, dynamically and contingently related. If they are not actually symbiotic, then, neuroscience and psychiatry are certainly densely inter-related and mutually informing, to such an extent that Insel and Quirion (2005) could describe psychiatry as "clinically applied neuroscience."

A critical examination of the concept of schizophrenia and of (some of) the research associated with it is, therefore, a way of shedding some light upon neuroscience more generally. Before presenting any such analysis, it is necessary to emphasize that, whilst schizophrenia remains a contested concept, the overwhelming majority of critics today recognize that the experiences associated with this diagnosis are typically traumatizing, debilitating, and associated with marked occupational and social dysfunction. Unlike some high-profile work associated with the so-called anti-psychiatry movement, contemporary critiques rarely try to normalize the experiences associated with this diagnosis (although they may note that voice-hearing, for example, is, in fact, quite prevalent in the general population—Beavan et al. 2011). Nor do they position schizophrenia as something heroic (cf. Deleuze and Guattari 1984), albeit that they do recognize that voice-hearing and related experiences can have positive aspects (Romme et al. 2009). Whatever the scientific status of schizophrenia, it is recognized that those given the diagnosis are frequently in urgent need of help and that its worldwide diagnostic prevalence of around 1% indexes significant social, economic and personal costs. Researchers, whether accepting of the schizophrenia concept or critical of it, are therefore mostly united in the goal of clarifying the causes, character and consequences of the experiences associated with a diagnosis, and so developing more effective remedial and preventative measures. It follows that the aim of critique is certainly not to negate the relevance of neuroscience; rather, it is to clarify and develop its contribution in order to maximize its potential benefits. With this aim in mind, we consider some implications for and connections to neuroscience of three aspects of schizophrenia: its initial development, its current status and its likely future development.

Initial Development

This section draws primarily upon Boyle's (2002) critical history of schizophrenia. This is appropriate for three reasons: first, Boyle presents a particularly close reading of original writings by Kraepelin, Bleuler and others who developed the concept; second, she is a clinical psychologist so her analysis (which is not solely historical) is informed by clinical practice; and third, whilst influential and highly regarded in UK clinical psychology, Boyle's work is less well known elsewhere. Like Heinrichs (2003), Boyle's history recognizes that schizophrenia emerged relatively suddenly early in the nineteenth century, and like Berrios, Luque and Villagran (2003), she rejects the "continuity hypothesis" that schizophrenia has always existed, and that work during the last century has identified an ontologically real, stable and unitary disease entity. Accordingly, like Berrios et al., Boyle engages with, rather than glosses over, differences between the early architects of this putative disease, and between these early works and the concept as it exists today.

Most historical accounts trace the modern concept of schizophrenia to the publication of Kraepelin's 1896 research on what he called dementia praecox. This term, meaning "early dementia," was occasionally used as early as the 1850s, and it is also sometimes said that the disease was first medically described (but not named) in case studies dating from 1797 to 1809 (Heinrichs 2003). Nonetheless, Kraepelin's work, which claimed to distinguish dementia praecox from other putative diseases of asylum inmates, is typically identified as the origin of the modern concept. Exemplifying the biologically oriented German psychiatric tradition, Kraepelin's account characterized dementia praecox as a whole-body disorder, which he hypothesized was produced by systemic, metabolic or "auto-intoxicating" disease processes that cascaded through the endocrine and peripheral nervous systems until, eventually, they reached the brain (Noll 2011).

It was Bleuler who deployed the term schizophrenia, although he was clear that this term described the same disorder that Kraepelin called dementia praecox. Bleuler's new term reflected his observation that—unlike in dementia—outcomes were sometimes positive and also mirrored his own conceptualization of the disease. Whereas Kraepelin largely emphasized biology, Bleuler (partly due to the influence of Freud, an influence often mediated by Bleuler's research assistant, Jung—Makari 2008) proposed a more diverse theorization—albeit one with biology always at its root. Bleuler proposed that an (unspecified) brain impairment produced a cognitive dysfunction which, in turn, disrupted the usual seamless integration of psychological functions such as memory, affect and self-awareness. This disruption interfered with typical

processes of association and inhibition, allowing excesses of affect to form and then become associated with memories and symbols, forming what Bleuler called "idea complexes." Whilst these idea complexes are the basis of the delusions and hallucinations commonly said to characterize schizophrenia, in Bleuler's concept these dramatic, disturbing experiences are merely the secondary effects of a brain disease which is first of all organic and then cognitive.

The concept of schizophrenia subsequently underwent further revision by Schneider in 1956, and it has since been modified less substantially in each revision of the main psychiatric diagnostic manuals, the International Classification of Diseases (ICD) and the Diagnostic and Statistical Manual of the American Psychiatric Association (DSM). Most revisions were made with the explicit goal of improving diagnostic reliability: for example, when DSM5 was released in 2013, it abolished the five sub-categories of schizophrenia (hebephrenic, catatonic, paranoid, disorganized and undifferentiated) that were introduced in DSM3. Nevertheless, for the most part psychiatry continues to trace the history of schizophrenia jointly back to Kraepelin and Bleuler, uniting their two concepts in a foundational origin story that posits the discovery and elucidation of a new disease (e.g. Fusar-Poli and Politi 2008; Burton 2006). Kraepelin and Bleuler are often described as the "founding fathers" who first supplied the notion that, whilst schizophrenia has many different manifestations, it is in essence "a disease of the mind that is characterised by a 'disturbance in thinking'—an abnormality in a fundamental cognitive process" (Andreasen 2000, p. 108). So despite modifications to diagnostic criteria, and notwithstanding occasional challenges such as those posed by the so-called anti-psychiatry movement, psychiatry since Kraepelin and Bleuler has primarily conceived of the core deficit in schizophrenia as an organic brain disease giving rise to a cognitive dysfunction that, in turn, fragments experience and thought: "Whilst the illness has detectable effects in virtually every sphere of mental activity, it is in the realm of higher mental function, including the experience of oneself as the source of one's own mental activity, that the most striking features of the illness are seen" (Liddle 2001, p. 181).

The historical analysis of schizophrenia by Boyle (2002) recognizes the foundational status that psychiatry affords to Kraepelin and Bleuler. Boyle presents a comprehensive analysis of their writings, framed by a history of some of the significant social changes of the eighteenth and nineteenth centuries, including the rise of the asylum, the birth and decline of moral management, and the relations between neurology and the nascent field of psychiatry as they appeared in different countries, particularly Germany (where these

two disciplines were especially closely related). Since Boyle's lengthy, detailed work defies summary within the confines of a single paper, the following précis will primarily emphasize her reading of Kraepelin. One of Boyle's key contentions, which she evidences by a close reading of Kraepelin's works, is that he:

> in fact appears to have been unaware of, or to have chosen to ignore, even the most basic principles of empirical enquiry—the need to present systematically gathered data, rather than to rely on personal experience and beliefs; the importance of clear description so that others can try to replicate the observations; the importance of reliability of observations and the dangers of question begging. (Boyle 2002, p. 59)

For Boyle, the significance of this contention cannot be overstated, precisely because of the foundational status of Kraepelin's work (including its uptake by Bleuler). If, as Boyle suggests, the initial processes whereby Kraepelin derived the concept of dementia praecox were inadequate or flawed, we must call into question the subsequent work elaborating it, refining it, and seeking causes and treatments for it: just as we might question the validity of any work refining personality structures based upon astrological star signs, since the existence of distinct personalities consistently linked to birth dates has never been convincingly demonstrated.

Boyle builds her case by first of all engaging with Kraepelin's early work, in which he claimed to be using observed similarities in onset, course and outcome to identify dementia praecox amongst asylum inmates. She observes that whilst each of these criteria is actually an umbrella term that covers a potentially large range of continuous changes and dynamic processes, they might nevertheless be deployed with some reliability *after* a coherent disease construct had already been identified. However, Kraepelin's use of them to initially identify dementia praecox inevitably encountered difficulties, since it was logically impossible for him to know in advance of their identification which of the (typically very many) observed similarities and differences in onset, course and outcome were significant and related. These difficulties were further compounded because his judgements of onset and (to some extent) course necessarily relied upon retrospective data, which may or may not have recorded changes whose putative significance would only later become apparent, in relation to the new construct. A somewhat different problem arose in respect of the criterion of outcome, since it was impossible for Kraepelin to conclude anything definitive under this heading until no more changes were actually able to occur.

Given the intransigence of these problems, it is perhaps not surprising that Kraepelin explicitly acknowledged difficulties using the criteria of onset and course:

> The whole upheaval can take so imperceptibly and with such indefinite indications that those around imagine that they are confronted simply with the outcome of an unhappy development, perhaps even of some character fault. In more than half the cases, the upheaval occurs so imperceptibly and with such indefinite indications that its actual beginning cannot be determined in retrospect. The course of this process of illness can take the most varied forms. The further course of the illness in these cases is a varied one insofar as the imbecility sometimes develops more rapidly, sometimes more slowly, and can in fact stop progressing at very different stages. Kraepelin, in Boyle (2002, pp. 47–49).

It is perhaps also unsurprising that, in relation to the criterion of outcome, Kraepelin provided only what Boyle calls "highly varied" descriptions with no indications of actual numbers of inmates to whom they applied. These descriptions were accompanied by what Boyle calls "vague statements" about psychological characteristics, statements that were neither linked systematically to observations nor supported by numerical analyses:

> although patients are more placid, it is only to reveal ever more clearly the indications of a fairly high-grade psychological weakness. The common outcome of all severer forms of dementia praecox is idiocy. Most frequently, however, the illness seems to lead to an insane confusion. Kraepelin, in Boyle (2002, pp. 50–51).

Consequently, Boyle questions the adequacy and reliability of the criteria which Kraepelin used to initially identify his new disease of dementia praecox. Vitally, she further observes that Kraepelin did not only need to be using reliable criteria: he also needed to present systematic analyses of the within-group similarities and between-group differences that these criteria produced. Boyle finds, however, that nowhere in his work did Kraepelin present any such systematic analyses. She suggests that his writings seem to obviate the need for them by frequently deploying phrases such as "'one often notices'; ' it is occasionally observed'; 'in some cases'" (Boyle 2002, p. 46): phrases which seemingly ground his work in thorough observation without ever reporting actual numbers of inmates who conformed—or did not conform—to the descriptions he presented.

In fact, Boyle suggests, it seems that Kraepelin must have effectively been working backwards: rather than present, enumerate and analyse evidence on

the basis of systematic observation, and then infer the existence of a new disease construct on that basis, Kraepelin seemingly *began* with some idea of the construct and then proceeded to describe what he said were cases of it:

> Kraepelin wrote as if by some independent and valid criteria, established by past research, dementia praecox had already been inferred in the sample and he was merely engaged in recording his impressions of the group. (Boyle 2002, p. 46)

Boyle therefore concludes that, despite the wealth of detailed observations Kraepelin presents, the process by which he sifted, sorted and analysed them in order to derive a putative new brain disease was inadequate. Rather than conduct the necessary systematic analyses of these (in any case problematic) observations, using within/between-group comparisons to derive robust differences, Kraepelin seems to have performed an intuitive, impressionistic reading that would, inevitably, have primarily confirmed whatever he already believed he was trying to find.

Bleuler then took Kraepelin's claims to have discovered a new disease largely on trust, just as, subsequently, Schneider accepted the work of both of his predecessors largely uncritically (and just as the DSM and ICD later did with the work of all three men). Boyle finds this especially remarkable given that the highly varied symptom profiles presented by both Kraepelin and Bleuler as characteristic of schizophrenia consistently included a range of striking neurological and physiological problems—tremors, tics, paralyses, gait disorders, oedemas, excessive sweating, and cyanosis of the hands and feet, as well as delusions and hallucinations—that are rarely, if ever, seen amongst today's schizophrenia patients. Boyle suggests that (whilst it is impossible now to be certain) this might be because many of Kraepelin and Bleuler's patients were actually suffering from undiagnosed encephalitis lethargica: a viral disease first identified by von Economo in 1917, the symptoms of which include all of those identified by Kraepelin and Bleuler as symptoms of schizophrenia. It is now known (but could not have been known by Kraepelin or Bleuler) that there were recurrent epidemics of encephalitis lethargica in Europe during the late 1800s and early 1900s, and so significant numbers of asylum inmates were quite likely to have been suffering with its effects. Whilst the extent to which Kraepelin and Bleuler took cases of this neurological disorder as cases of schizophrenia/dementia praecox is impossible now to decide, their doing so would account for the otherwise puzzling change that has occurred in the typical symptoms they identified, and those that typify schizophrenia today.

As a clinician, Boyle's aim is to neither dismiss nor normalize the experiences associated with this diagnosis. Instead, Boyle wants to clarify their nature and causes, in order that more effective interventions can be tailored. In the present context, her analysis speaks directly to what Cohn (2010) characterizes as a paradox in the intimate relationship between psychiatry and neuroscience. On the one hand, the reliability and validity of neuroscientific research into schizophrenia "is in effect dependent upon the more general assumption that current psychiatric diagnosis offers an approximation of specific conditions that unquestionably exist beneath" (p. 185). On the other hand, this dependency co-exists alongside "the more general view that neuroscience needs ultimately to divorce itself from its reliance on the mess of contemporary psychiatry, and find ways of establishing pathology independent of persons" (ibid.). Neuroscientific research in schizophrenia follows "the logic that biology is…the 'base' of illness and has to be the definitive, singular cause of disease" (p. 186). Simultaneously, however, many neuroscientists believe that their research may eventually "identify biological abnormalities which not only endorse the old psychiatric logic but potentially break free from it" (ibid.). If Kraepelin and Bleuler never did demonstrate the validity of schizophrenia as a disease concept, as Boyle claims to have shown, the neuroscientific paradox that Cohn (2010) identifies becomes considerably sharper, and the (sometimes covert) difficulties it generates for neuroscientists are potentially more widespread. Moreover, as consideration of the current status of schizophrenia shows, it is possible that neuroscience is already encountering such difficulties.

Current Status

So schizophrenia has from the earliest been framed as a brain disease or illness—albeit one with an unspecified pathology. This neural emphasis is demonstrated by a bibliometric analysis of the abstracts of nearly 10,000 papers presented at two major international conferences on schizophrenia between 1988 and 2004 (Calton et al. 2009), which found that 75% of these papers were primarily biological in their orientation, whereas less than 5% took a predominantly psychosocial perspective (and less than 2% gave explicit consideration to actual experiences). Consequently, with respect to neuroscience, schizophrenia is positioned as a highly fertile concept, one that seemingly yields unanswered questions, the possibility of fame and even fortune, and the opportunity to make a significant difference to the quantum of human suffering.

An important component of Rose and Abi-Rachid's (2013) extended dissection of contemporary neuroscience is their demonstration that psychiatry

has long struggled to define the boundaries of sanity, madness and normality, and that with the rise of community treatment in recent decades the boundaries between neurosis and psychosis have become more blurred. Hence, statements about the interpretation of diagnostic criteria increasingly emphasize heterogeneity within the various categories, even whilst at the same time promoting degrees of specificity in their (presumed) underlying biological pathologies (e.g. in the continuing idea of schizophrenia as an organic disease of higher cognitive processes). At the same time, the enduring problems of validity and reliability associated with all of the functional psychiatric diagnostic categories, schizophrenia included (Johnstone 2000), mean that the continuously evolving relationships between psychiatry, schizophrenia and contemporary neuroscience, whilst mutually imbricative, are complex and sometimes contradictory.

Rose and Abi-Rachid (2013) also observe that neuroscience has frequently mobilized and traded in the concept of schizophrenia, just as psychiatry has exploited the cachet and resources of neuroscience to warrant its conceptual frameworks and promote its own research agendas. Nevertheless, overall progress has been disappointing. Despite frequent claims to have found "the" neural or genetic basis of schizophrenia, and notwithstanding that efforts to do so have benefitted from very generous research funding and, in recent years, an explosion of powerful new technologies, the results of this massive research effort remain largely inconclusive. Cromby, Harper and Reavey (2013) observe that in recent decades schizophrenia has been associated with abnormalities of, or differential functioning within, dopamine, glutamate, serotonin, acetylcholine, gamma-butyric acid, prostaglandin and neuropeptide systems and pathways. At the same time, schizophrenia has also been associated with neuroanatomical features including enlarged ventricles, cerebral asymmetry, temporal lobe abnormalities, thickened corpus callosum, thinner corpus callosum, abnormalities of the basal ganglia and cerebellum, and reduced overall brain volume. So it is not that there is no evidence of neural variation in relation to the diagnosis of schizophrenia: the problem is that there is *no coherent pattern of evidence* consistent with psychiatric claims of a distinct neural pathology as the basis of this diagnosis.

These problems are compounded by others, notably that the results of many studies are confounded by the effects of psychiatric medication; also that both population norms and what Rose (1997), following Dobzhansky, calls "norms of reaction" (i.e. the range within which phenotypic gene expression can vary without functional failure and beyond which usual functioning breaks down) are largely unavailable for these various features: hence neither their pathological significance, nor their prevalence in the general population,

have typically been definitively established. Hence, some 17 years ago, an editorial in Nature Neuroscience (1999) noted that:

> Schizophrenia remains unexplained. None of the abnormalities reported in the brains of schizophrenics is clearly diagnostic for the disease in the way that (say) plaques and tangles are for Alzheimer's disease.

And this uncertainty continues unabated:

> the field of psychiatry has thus far failed to identify a single neurobiological phenotypic marker or gene that is useful in making a diagnosis of a major psychiatric disorder. (Charney et al. 2002, p. 33)
>
> Our understanding of the biological mechanisms of diseases such as mood disorders, schizophrenia and autism is frustratingly limited. There is also a lack of reliable biological markers for characterising these diseases. (Chou and Chouard 2008, p. 889)
>
> efforts to understand the neurobiological bases of the clinical heterogeneity that schizophrenia comprises, mainly by correlating neurobiological measures with specific symptoms, have been largely unsuccessful. Indeed, it is fair to say that "inconsistency" has been the most consistent finding to emerge from such efforts. (Mathalon and Ford 2012, p. 1)

Whilst genetic research is not the focus here, it should be noted that, to some extent, these acknowledgements reflect recent molecular genetic research into schizophrenia, which has not discovered any major genes of significant effect associated with this diagnosis (Crow 2008). Summarizing the current overall state of knowledge, Kendler (2005, pp. 434–436) says:

> We have hunted for big, simple, neuropathological explanations for psychiatric disorders and have not found them. We have hunted for big, simple, neurochemical explanations for psychiatric disorders and have not found them. We have hunted for big, simple genetic explanations for psychiatric disorders, and have not found them.

Consequently, although the notion that schizophrenia is a brain disease continues to predominate, in both neuroscience and psychiatry, current research is still developing in strikingly diverse ways. With respect to its neural, biological and genetic aspects, this diversity can be illustrated by considering a recent issue of the leading journal "Schizophrenia Bulletin." In November 2013 this journal published papers relating schizophrenia to endophenotypes identified by P50/P300 ERP events; variability in the ZNF804A gene; dis-

ruption of corollary discharge function for motor movements; temporal lobe volume abnormalities; aberrant salience and dopamine activity; low birth weight; connectivity between the default mode network and task-processing networks after ingesting psilocybin; and activation differences amongst people given the diagnosis of schizophrenia in the posterior cingulate, precuneus and other regions in self-other differentiation tasks. These various strands of investigation have obvious affinities with work in (predominantly) cognitive and affective neuroscience (and in genetics and epigenetics), and it remains hypothetically possible that one of them might ultimately identify a distinct neural pathology for schizophrenia. Nevertheless, whilst it remains highly prevalent, promissory discourse of this kind is now increasingly accompanied by other discourses associated with somewhat different research strategies.

Possible Futures

So there is widespread acknowledgement that the search for brain impairments specific to and consistently associated with schizophrenia has failed, and this is leading many researchers to adopt alternative strategies. As Cohn (2010) suggests, for some the problem is primarily the inadequacy of diagnosis: hence, anticipating publication of DSM5, the director of the American National Institute of Mental Health (NIMH), Thomas Insel, announced in April 2013 that the Institute's funding would in future be oriented away from psychiatric diagnostic criteria. Whilst this statement was later qualified, the NIMH strategy—of developing its own Research Domain Criteria as an alternative, more valid taxonomy of distress—remains. In this regard, it has been argued that philosophical phenomenology might contribute to neuroscientific research by "front-loading" studies with consistently established experiential distinctions which might, in turn, assist with the identification of related neural systems (Woods et al. 2014). One psychiatric strategy that has long been evident is to conceive of schizophrenia as a syndrome: to figure it within diagnostic manuals as a singular disease, whilst simultaneously describing it elsewhere as a diverse collection of closely related illnesses (e.g. Roberts 1990). Conversely, Frangou (2014) advocates a strategy of using imaging technologies within a "systems neuroscience" perspective with respect to both schizophrenia and bipolar disorder. Although here, as in other recent studies (e.g. Fillman et al. 2014), both of these diagnoses can be subsumed within the broader category of psychosis, other reconfigurations are also appearing: for example, a genome-wide association studies (GWAS) meta-analysis by the Cross Disorder Group of the Psychiatric Genetics Consortium (2013) identi-

fied possible genetic influences operating equally within schizophrenia, bipolar disorder, major depression, autism and attention-deficit/hyperactivity disorder (ADHD). An alternate strategy involves leaving the diagnosis largely unquestioned but adopting different bioscientific research strategies: Light and Swerdlow (2014) propose using biomarkers to identify *unimpaired* neural and cognitive function amongst people given a schizophrenia diagnosis, using drug challenges and practice effects to identify areas of continuing neuroplasticity; and monitoring therapeutic progress in these areas using neurophysiological measures such as prepulse inhibition of startle.

In recent years, there has also been a resurgence of interest in the social and relational factors associated with diagnoses of schizophrenia. There is good evidence showing, for example, that social inequality and low socioeconomic status (SES) are causally associated with a schizophrenia diagnosis (Harrison et al. 2001); that ethnic minority status, and the effects of discrimination and prejudice, make a schizophrenia diagnoses more likely (Boydell et al. 2001); and very robust evidence that child physical and sexual abuse, neglect, bullying and emotional abuse are all causal of the experiences associated with a schizophrenia diagnosis (Read et al. 2005). Relevant research in neuroscience (and other biosciences, notably epigenetics) consequently aims to identify the specific neural pathways or processes modified by these toxic combinations of adverse circumstances (e.g. Tyrka et al. 2013)

Another range of alternative strategies is also emerging from (predominantly British) clinical psychology. Inspired in part by Boyle's work, many researchers and practitioners largely disregard the concept of schizophrenia (Bentall 2003). It is not that they necessarily overtly reject the diagnosis (although their professional body has done so—DCP 2013), rather that they neither invoke it as a causal explanatory device nor as a necessary organizing paradigm. Research (and interventions) associated with this perspective focus on specific, relatively homogenous experiences—hearing abusive voices, holding unusual and distressing beliefs—rather than relatively heterogeneous diagnostic categories, and uses experimental techniques to identify cognitive, affective, neural and physiological processes associated with these more specific difficulties. For example, Bentall, Kinderman, and Kaney (1994) found that a combination of low self-esteem and external locus of control were associated with the presence of persecutory delusions; they argued that self-worth was being defended by (mis-) attributing negative occurrences to malevolent others. Conversely, Garety et al. (2005) used measures of reasoning, emotion, belief inflexibility and extreme responding to demonstrate that people with delusional beliefs displayed more evidence of the cognitive bias of "jumping to conclusions." Likewise, a meta-analysis of externalizing biases in people experiencing auditory hallucinations (Brookwell et al. 2013) found robust, moderate-to-large effects associating these

experiences with cognitive impairments in source monitoring (i.e. attributing self-generated "inner speech" to an external source).

Whilst there are extensive methodological parallels between neuroscience and this clinical psychological research, the substantive connections are sometimes less immediately apparent. Nevertheless, researchers accept that neural systems continuously enable (if not simply cause—Harre 2002) all psychological phenomena, and this understanding provides the basis for collaborations and, in some cases, models that combine psychological and neuroscientific influences (Bentall 2003). For example, Read et al.'s (2001, 2014) traumagenic neurodevelopmental model recasts the various discrepant brain features sometimes observed amongst people given psychotic-spectrum diagnoses as injuries, caused by (predominantly early) trauma. The model embraces current neuroscientific evidence regarding brain development and the functions of different regions and systems, drawing upon imaging and related studies to demonstrate the many similarities between the brains of abused and neglected children and the brains of (some) people given diagnoses of schizophrenia. At the same time, by treating these brain features as injuries (not illnesses), and by focusing on the broader notion of psychosis rather than the more specific concept of schizophrenia, the model negates the psychiatric conception of schizophrenia as a brain disease or illness.

Summary

Despite the foundational status accorded to Kraepelin and Bleuler's work, schizophrenia may not have been initially established as a coherent disease construct. If so, it would not be surprising that subsequent research, instead of clarifying the biological basis of this presumed disease, has reproduced and perhaps magnified this original confusion. Consequently, with respect to schizophrenia, a range of future neuroscience research strategies can currently be discerned:

- identifying biomarkers for unimpaired function
- identifying systemic neural deficits
- combining schizophrenia with other psychiatric diagnoses
- developing the NIMH Research Domain Criteria
- researching relatively homogenous experiences such as "hearing voices"
- re-interpreting neural differences as injuries rather than illnesses

Some of these neuroscientific research strategies are relatively orthodox, in that they retain a notion of diagnosis and posit distinct causal neural pathologies; others are less so, dispensing with diagnosis and focusing instead on

specific aspects of experience such as hearing voices. They differ in the extent to which they can readily incorporate the epidemiological evidence regarding trauma, abuse, SES and ethnicity (since illness-based strategies often import a hierarchy of evidence within which neuroscientific or biological evidence tends to predominate). They also differ in how they incorporate this epidemiological evidence—as the "trigger" that might release a neural vulnerability, or as the adverse circumstances that might cause brain injury. Consequently, there is also variation in the manner and extent to which these strategies open neuroscience to the social and cultural contexts within which it gets conducted (Choudhury and Slaby 2012). Nevertheless, to some degree, each strategy recognizes that the construct of schizophrenia may not provide a sufficiently sound basis for future neuroscientific research, and all offer viable alternatives that could generate rich empirical studies with considerable potential to expand knowledge and alleviate suffering.

The example of schizophrenia, therefore, shows that neuroscience need not confine itself to models of psychological functioning derived uncritically from biological psychiatry. Valid neuroscientific research programmes can be pursued without endorsing psychiatric diagnoses that lack reliability and validity, and indeed without presupposing medical notions of illness. Scholars who have recently suggested that social neuroscience could make a strong contribution to psychiatry (e.g. Cacioppo et al. 2014) might want to pay particular heed to such suggestions. At the same time, they are relevant for neuroscience generally, precisely because it is so densely entangled with psychiatric theories, evidence and practice.

Considering its implications more broadly, this example also illustrates how neuroscience can benefit from sustained engagement with psychology and social science. Such engagement will encounter methodological, conceptual, linguistic and ideological problems (Cromby 2007; Papoulias and Callard 2010), and will raise challenges for psychology at the same time as it overturns established certainties in psychiatry. We should welcome these challenges and the opportunities they will bring.

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21

Epigenetics and the Suicidal Brain: Reconsidering Context in an Emergent Style of Reasoning

Stephanie Lloyd and Eugene Raikhel

The rapidly growing area of biological research known as "epigenetics," or more specifically "environmental epigenetics," has been hailed by many scholars as a paradigmatic overturning of received wisdoms about evolution, heredity and distinctions between "nature" and "nurture." Seen by its proponents as a powerful form of inquiry for the study of a wide range of health conditions and social factors (Berntson et al. 2008; Collins et al. 2004; Dancause et al. 2011; Meaney 2008; Roseboom et al. 2001; Ryff and Singer 2008), epigenetic research on various health conditions, ranging from cancer to mental illness, has had a profound impact on the scientific community and has been the subject of much media attention (Canadian Newswire 2010; Dolgin

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2009; Paré 2003; Tonnietto 2003; Watters 2006). Some observers and participants in this epigenetic turn have characterized it as a revolution in understandings of how gene expression takes place in interaction with the environment and the ways in which brain development occurs by means of experience-dependent processes (Meaney and Szyf 2005; Weaver et al. 2004). Other researchers remain much more sceptical. Some describe epigenetics as simply another movement in the long-running tradition of studying gene expression (Niewöhner 2011; Pickersgill 2016; Tolwinski 2013). Yet others are intrigued by these claims and the preliminary findings emerging from environmental epigenetics, but remain highly sceptical of the evidence presented thus far (Weinhold 2012).

In this chapter, we examine the styles of reasoning and epistemic objects emerging from environmental epigenetics, focusing on research into suicidal behaviour being conducted at the McGill Group for Suicide Studies (MGSS), a multidisciplinary research group based at the Douglas Mental Health University Institute in Montreal. Drawing upon our own research with members of the MGSS, we build upon work by other social scientists working on epigenetics (Landecker and Panofsky 2013; Lock 2013a; Meloni and Testa 2014; Niewöhner 2011) and argue that this research represents an emergent style of reasoning in which a range of contextual and environmental factors are both molecularized and located in the brain. We argue that implicit in this research is a notion of a "suicidal brain": a brain that responds to adverse life experiences with an increase in risk of suicidal behaviour. In examining both this concept and its attendant styles of reasoning, we highlight some of the issues which research in environmental epigenetics raises for the study of suicide, as well as for the social sciences more broadly.

The concept of "styles of reasoning" used in this chapter was developed from the work of Ludwik Fleck (1979) by Ian Hacking (1992) and other historians and philosophers of science and employed productively by anthropologists to discuss the historical and social variability of scientific cultures. As Allan Young has put it, a style of reasoning "is composed of ideas, practices, raw materials, technologies and objects…It is a characteristically self-authenticating way of making facts, in that it generates its own truth conditions" (Young 2000). Importantly, this notion encompasses not only elements enacted by individuals or groups (e.g. tacit knowledge and embodied skill) (Polanyi 1966) but also those material objects, technologies and infrastructures without which certain kinds of knowledge production would be impossible.

We begin this chapter by analysing environmental epigenetics as an emergent style of reasoning and then examine its relevance to psychiatry and the

study of psychopathology at a time when attention is increasingly paid to issues of lifespan and development. We then discuss the research taking place at the MGSS and the way in which suicide risk is being conceptualized within this research unit, drawing upon our preliminary fieldwork. Of immediate interest are the ways in which context or environment is defined and conceptualized in this research on suicide. In particular, we ask which aspects of the environment have been operationalized into measures and thus are included in studies and which ones are left aside. We will draw attention to some implications of adopting this particular view of the role of environment in suicide with a brief discussion of how environment or context is understood in anthropology and sociology, focusing on the example of social science studies of suicide in Canadian Aboriginal communities. We conclude with a discussion of some of the general issues, risks and opportunities that emerge for social scientists through epigenetics research on suicide risk.

Epigenetics and Its Styles of Reasoning

Because the term "epigenetics" is used to designate a variety of concepts and domains of investigation, it is important to clarify some of these distinctions before describing the contemporary research which interests us. The term "epigenetics" dates at least to 1942, when it was first used by embryologist and geneticist Conrad Hal Waddington (Choudhuri 2011; Holliday 2006; Slack 2002). Working at a time when genetics and development were studied in parallel rather than as closely entangled phenomena (Choudhuri 2011), Waddington used the term to refer to the study of the unfolding of the genetic programme for development or the process that creates a phenotype from a genotype. Following Waddington's usage, "epigenetics" came to denote phenomena above and beyond genetics, for which genetics could not provide an adequate explanation: particularly the question of "cell fate specification"—or how a single fertilized egg cell differentiated into the multiple types of cells during the early development of an organism. In other words, since cells in skin, bone and brain tissue, for example, all share the same genetic material, some non-genetic mechanism was needed to explain how they developed into different, specialized cells and tissues.

While such theories long lacked any empirical evidence of specific mechanisms, in recent decades, scientists have identified several processes by which the regulation of gene expression may take place, with most research focused on three areas: (1) the methylation of DNA, (2) modification of the histone proteins that form the spool around which the DNA double helix winds and

(3) noncoding RNAs. All of these are mechanisms by which the genome may be modified without the alteration of DNA bases, and all of them are reversible. As one summary of research in this domain has it:

> Epigenetic mechanisms collectively act as an editorial hand that edits and modifies the language of DNA. What remains to be understood is… how signals trigger epigenetic changes that, in turn, edit and modify the language of DNA. (Choudhuri 2011, 270)

While these mechanisms are now widely accepted as central to cellular differentiation and other processes, a more specific set of theories—which we refer to here as "environmental epigenetics"—argues that they also mediate the effects of specific environmental conditions on gene expression. While this remains an emergent field and central questions about causality continue to be questioned and debated (Daxinger and Whitelaw 2012; Landecker and Panofsky 2013), another highly contentious argument being made by some researchers in this field is that epigenetic changes can, in some cases, lead to heritable changes in phenotype.

Two aspects of environmental epigenetics' emergent style of reasoning are particularly important to note here: its attendant conceptualizations of environment and temporality.[1]

In order to study the mechanisms by which environments shape gene expression, researchers engage in what has been called "pragmatic" (Beck and Niewöhner 2006) or "methodological reductionism" (Kirmayer and Gold 2012), conceptualizing environments as a set of molecular inputs. These inputs range from environmental toxins to nutrients to stressful experiences such as childhood abuse. This logic requires the abstraction and operationalization of these inputs; distinctions in content or derivation are flattened and rendered incidental. As Meloni and Testa have argued, "the analogical vastness of …'environmental signals'" is translated to "genome-friendly, code-compatible digital representations" (2014, 435). In addition, this style of reasoning effectively erases clear distinctions between environments "internal" and "external" to what Margaret Lock and Judith Farquhar have called "the body proper"—"a skin-bounded, rights-bearing, communicating, experience-collected, biomechanical entity" (Landecker and Panofsky 2013, 339; Lock and Farquhar 2007). As Michael Meaney, a pioneering researcher in the field, has written, "The relevant environmental event may be internal or external to the organism; e.g., a change in the availability of glucose, an electrical impulse, or a social interaction" (Meaney 2010, 50, quoted in Landecker and Panofsky 2013, 339). In other words, such theories suggest the literal interpenetration of bodies with material and social environments.

This "molecularization of milieu" thus shapes the particular conception of the body that emerges from epigenetics research, a conception which Jörg Niewöhner has described as "the embedded body" (2011). This is "a body that is heavily impregnated by its own past and by the social and material environment within which it dwells. It is a body that is imprinted by evolutionary and transgenerational time, by 'early-life' and a body that is highly susceptible to changes in its social and material environment" (Niewöhner 2011, 289–290).[2]

In addition to the molecularization of the environment, epigenetics entails a set of temporalities which are quite distinct from those of the genetic style of reasoning that was dominant from the post-WWII period until recently. First, environmental inputs or signals are understood to have a greater effect at certain periods in an individual's life than at others (Fagiolini et al. 2009). Such "critical periods" may vary depending on the type of environmental signal in question, but in human beings they range from the early in utero stages into early childhood. These periods are characterized by a higher level of cellular, neural and developmental plasticity, during which "life-long trajectories of metabolic and behavioral homeostasis" may be shaped (Szyf 2009, 882). Writing specifically about the potential effects of early life adversity on mental health, Moshe Szyf says: "The new balance of methylation emerging early in life in response to cues from the social environment would then be sustained through life and affect behavior and mental health but for a strong intervention that would reverse this epigenetic program later in life" (Szyf 2009, 881). Underlying this argument is the idea that such early responses may represent an adaptive process: "Adversity during early life would anticipate life-long harsh conditions and readjustment of epigenomic programs to the environment" (Szyf 2009, 881–882). Thus, the temporalities of epigenetics extend from small-scale reactions to environmental inputs in childhood to involve lifelong reactive processes (Lappe and Landecker 2015).

The temporal horizons raised by epigenetics research are also highly variable. While some epigenetic changes may last for very short periods of time—for example, epigenetic mechanisms have been linked to circadian rhythms (Bellet and Sassone-Corsi 2010)—others may last across the individual's life course, long after the initial signal or exposure which catalysed the changes has passed. Much discussion has taken place over the possibility that some epigenetic changes may even, in some cases, be heritable to subsequent generations, although research on the mechanisms through which such intergenerational heredity may take place remains emergent and highly contested. One central question centres on whether such changes may be inherited through the germ line (Holliday 2006). Some scholars working in a developmental tradition have argued that regardless of whether epigenetic markers are

inherited through the germ line or through particular modes of maternal care during a critical window in early development (see discussion of this theory below), this new research necessitates a broadening of our conceptualizations of heredity as well as a reappraisal of Lamarckian theories of the inheritance of acquired characteristics (Jablonka and Raz 2009; Landecker and Panofsky 2013, 348). In effect, while many researchers may reject the claims of transgenerational epigenetics, new languages are emerging to describe "parallel" or "soft" forms of inheritance that result from care, context and personal experience, ones they argue need to be taken as seriously as germ line inheritance (Bonduriansky 2012; Handel and Ramagopalan 2010). No matter what specific form the theories take—epigenetic or developmental—emerging arguments about the transgenerational transmission of acquired traits are positioned to have potentially profound consequences for theories of evolution and development across the disciplines (Meloni 2016).

For some natural and social scientists, these recent trends in epigenetics research have led to a new vision of the relationship between society and biology, while for others they have bolstered long-held ideas about biosocial complexity. Researchers have called for more sophisticated theories and experimental practices that overcome nature-nurture dichotomies and instead reflect the way our context—which includes our social, cultural, and environmental surroundings—is integrated into the body via epigenetic and neuroplastic changes throughout the lifespan (Choudhury 2010; Dominguez 2012; Neddens et al. 2003; Niewöhner 2011; Roth et al. 2009; Seligman and Kirmayer 2008; Wexler 2006). Of course, interest in the relationship between the environment or social structures and health is not new in itself. Social epidemiologists have been studying the social determinants of health and illness for many decades, and results from paradigmatic studies, such as the Whitehall Study (Brunner et al. 1997; Marmot 1993; Singh-Manoux et al. 2003) and the Dutch Famine Birth Cohort Study (Lumey et al. 1993; Phillips et al. 2012; Roseboom et al. 2001; Van Noord et al. 2002), in many ways inform key assumptions made in epigenetics. What is novel is a shift in emphasis from general *correlations* to specific hypothesized *mechanisms* through which social position, for example, may confer risk of sickness (Marmot 2008).

It is important to note that according to many researchers, the evidence for epigenetic marks mediating environmental events or conditions and phenotypic outcomes still remains largely correlative, that is, as associations between experiences and molecular profiles. Furthermore, as one of the researchers at the MGSS emphasized in our conversations, studies comparing individuals exposed to some stressor or adverse experience to controls have not shown a clear-cut distinction in epigenetic marks such as methylation—either "on" or "off"—but differences in their frequency at specific sites. So, although specific mechanisms

have been associated with specific outcomes, many researchers believe that hypothesized impact of these mechanisms must be proven in far more controlled settings—removing a lifespan of confounding life factors—before it can be accepted. Drawing what some researchers would characterize as a "causal link" under such conditions would require the technology and materials to introduce, manipulate, and modulate epigenetic marks directly.

Moreover, these hypothesized mechanisms are not causal in any straightforward way but distributed, systemic and deeply complex in their effects. As Hannah Landecker and Aaron Panofsky have argued, "epigenetic effects are drawing new ontologies of outcome: The mechanism being traced is one that acts through effects that might be seen systemically or multiply, simultaneously manifesting as physiology and behavior, or shifts that are simultaneously metabolic and mental" (Landecker and Panofsky 2013, 342). Here we are far from an earlier style of reasoning, demonstrated perhaps most vividly with the millennial excitement, hype and expectations associated with the decoding of the human genome, which sought to link particular diseases or behavioural phenotypes to individual genes or small variations in the genetic sequence (Rabinow 2008). This shift has profound consequences for the conceptualization of disease, and indeed the relationship between norm and pathology in the biosciences, and finds support in the resurgence of research on the level of systems.

Among researchers studying psychopathology, there is wide support for a shift away from both reductive biological explanations of mental disorder as well as from a categorical logic of discrete disease entities (Kendler 2008). Indeed, it is worth remembering that epigenetic work is one part of a growing body of research on the neurodevelopmental factors and mechanisms potentially implicated in mental illness and particularly the impact of stress and adversity on children's and adults' risk of developing a mental illness as well as interest in tracking early signs of psychopathology in the children of people diagnosed with mental illness (Bruffaerts et al. 2010; Kessler et al. 2010; McLaughlin et al. 2010; Turecki et al. 2012). These research findings, along with some paradigmatic shifts in the understanding of neural plasticity, have fed into an increasing reframing of mental illnesses as neurodevelopmental disorders (Rees 2015).

The Suicidal Brain and the Molecularization of Context

Since the nineteenth century, researchers in the human sciences have struggled to explain why suicide, an ostensibly individual action, often occurs in clusters or concentrated in specific subpopulations. And while it has also long

been clear that suicide rates are higher among the marginalized and socially excluded (Anderson and Smith 2003; Kirmayer et al. 2009; Krull and Trovato 1994; Silviken and Kvernmo 2007), as well as among groups of people suffering from certain psychiatric disorders (Black et al. 2004; Kessler et al. 1999; Mościcki 1997; Renaud et al. 2008; Turecki 2005), explanations for the clustering of suicide have developed on two largely distinct levels of explanation and styles of reasoning, which in turn suggest very different modes and sites of intervention. Social scientists' and epidemiologists' explanations on the level of environment and social structure (Dahl et al. 2000; Hicks 2007b; Hicks et al. 2007; Kirmayer 1994; Légaré 2009) are used to argue for the reduction of marginalization and the recovery of social, cultural and community practices (Kirmayer and Valaskakis 2009), while psychological and biological explanations (Garland and Zigler 1993; Gunnell and Frankel 1994; Isacsson 2000; Mann et al. 2005) authorize individual psychiatric and psychological interventions to manage risk and reduce patient symptomatology (Möller 2003).

Emergent research on suicide in environmental epigenetics and neuroscience attempts to bring together these styles of reasoning by asking, "Why, in response to relatively common human experiences of loss, hardship, or marginalization, do some people commit suicide while others do not?" This approach toward understanding suicide builds on existing research on genetic-social environment interactions that have been observed to lead to long-term changes in biological and behavioural reactivity to stress in animals, leading to ill health (Hernandez et al. 2006; Meaney 2001a, b; Meaney and Szyf 2005; Weaver et al. 2004). This research—including the work on suicide discussed in this chapter—is now being translated into human studies (Collins et al. 2004; Dancause et al. 2011; Hyman 2009; McGowan et al. 2009; Ryff and Singer 2008; Turecki 2001). Because epigenetic markers are tissue-specific, translating such studies to human beings requires access to well-preserved post-mortem brain tissue, making researchers dependent on expensive and relatively rare biological platforms including brain banks. The existence of a brain bank at the site of the MGSS provides its members with the raw materials they need for their research, even as numbers and types of brains constrain the kinds of questions they can ask. For example, recent findings suggesting that social and environmental experiences early in life can produce epigenetic effects that put people on a track for long-term psychopathology and suicide risk require the brains of "suicide completers" who, through the process of psychological autopsies, have been found to have experienced early childhood adversity (McGowan et al. 2009; Turecki et al. 2012). Opening up lines of research on resilience, for example, even if of keen interest to the scientists,

would require a new collection of the brains of people who have died of natural causes, but who experienced early childhood adversity. Evidently, the structural and biological platforms for this research are significant.

In a certain sense, research on the suicidal brain is a part of a long-term search for the personality traits and psychological risk factors implicated in suicide. However, this search is now being conducted on the molecular level, focusing on neural markers potentially associated with suicide risk. In this chapter, we are particularly interested in two complementary types of research, including the epigenetic work of Gustavo Turecki, director of the MGSS, and the imaging work of Fabrice Jollant. Both are clinician-scientists. Turecki's research attempts to identify the changes in the brains of "suicide completers" that might be considered epigenetic responses to early childhood adversity or trauma (Fiori et al. 2011; McGirr et al. 2008, 2009; McGowan et al. 2009; Turecki 2001, 2005; Turecki et al. 1999, 2001, 2012). Information on life events is collected via psychological autopsies with the kin of "suicide completers," in which they act as proxies for their lost loved ones, answering psychological questionnaires and recounting the major life events of people lost to suicide, to the best of their knowledge. Through this research, life stories of the dead are constructed as scientists attempt to correlate epigenetic markers in the donated brains of suicide completers with their negative life experiences. Jollant's research attempts to identify the regional neural specificity of suicide risk. This involves fMRI studies of the kin of "suicide completers" as well as suicide attempters. Participants respond to psychological tests meant to assess their decision-making skills and level of impulsivity while being scanned. The working hypothesis in these studies is that early adversity might make one more vulnerable to suicidal behaviour because of acquired bad decision-making and impulsive behaviour (Jollant et al. 2008, 2010, 2011; Olié et al. 2010). This fMRI research attempts to identify the regional specificity of these traits in the brains of the kin of suicide completers, a neural specificity they are presumed to share with their lost loved ones (e.g. through shared environment or transgenerational transmission of risk).

In this literature, early childhood adversity is defined as acts of a parent or caregiver that inflict harm on a child. Examples include sexual abuse, physical abuse, emotional abuse, and neglect (Turecki et al. 2012). These studies are interested not only in how these traits might develop over the course of individual lifespans but also in their transmission across generations and prevalence within particular families. Overall, these neuroscience and epigenetic models suggest that particular life experiences, especially early experiences, leave lasting traces in the brain, rendering an individual and potentially his or her descendants, through a variety of mechanisms, susceptible to suicidal behaviour. Put

differently, epigenetic models of suicide suggest that experiences such as early childhood adversity can lead one to develop a "suicidal brain."

In effect, these studies are attempting to lay bare the "suicidal brain," identifying which factors, at a neurological level, predispose some people to suicide. While this has the potential to offer interesting insights into suicidal behaviour and to provide eventual targets for intervention, it also raises many questions. For example, it is unclear whether and how a specific—and perhaps singular—life event can be causally linked to molecular epigenetic changes. Attempts to find clear-cut answers are further confounded by the marginal significance of correlative results thus far in this research. For instance, when the brains of suicide completers are compared to those of controls, such as people who did not commit suicide, the same epigenetic markers such as methylation might be present. At this point, the science of environmental epigenetics is working in a space of marginally significant correlative mechanisms. Given the structural burden of carrying out this research and its nascent character, this is not particularly surprising. It does not mean that the researchers will be unable to one day prove their claims to the satisfaction of the scientific community more broadly, but for the moment the explanatory potential attributed to this type of reasoning outpaces accepted scientific findings.

The Politics of Epigenetics and the Case of Aboriginal Suicide in Canada

This epigenetic research on suicide risk has reverberations outside the domain of science, with increasing impacts on society and politics more generally. Many researchers have taken note of this and as such much epigenetics research cited in this chapter is explicitly ambitious not only scientifically but also politically. It is progressive in its goals, presenting theories on how stress, social inequality, racism, social exclusion, marginalization and adverse life experiences have "real" (i.e. biological) effects on how people live (e.g. morbidity and mortality) and therefore deserve to be taken seriously (Berntson et al. 2008; Collins et al. 2004; Dancause et al. 2011; Meaney 2008; Roseboom et al. 2001; Ryff and Singer 2008).

By demonstrating the mechanisms by which negative life experiences impact the body, epigenetic theories have taken up a strong position within a hierarchy of explanatory power (Bell 2013). Neuropsychologist Vaughan Bell provides a correlate example of the increasing references to neuroscience research, in terms of the impact of context on the brain, by politicians as a means to argue for social reforms and interventions with "problem families."

He cites a British politician who stated: "unemployment was a problem as it has 'physical effects on the brain'." Bell responded to this statement, saying:

> As everything has a physical effect on the brain we are left none the wiser but it is interesting that not having a job was not considered problem enough. It's not that neuroscience isn't relevant to these concerns, but just that it has gained such rhetorical power that explaining your concerns in terms of fairness, success, pain or poverty no longer seems sufficient. (Bell 2013)

This draws attention to the significant impact that the neuroscience and epigenetic "revolution" stands to have on the way we understand and intervene on inequality and illness, raising the apparent moral stakes of engagement with and social responsibility for these issues while providing a new explanation of how the social world affects us and what we should do about it.

The indiscriminate adoption of such explanatory models has the potential for unintended consequences. Most clearly, epigenetic explanations of the impact of context on biology could potentially supplant other types of correlative studies on similar issues, as the search for mechanisms has become an idealized and prioritized form of research, highly regarded in scientific, medical, and research funding communities (Marmot 2008). Additionally, as epigenetic explanations of the impact of misfortune, negative life experiences, or unhealthy environments on the body become normalized, it threatens to change the type of proof that might be expected in claims for social justice, for example, and it directs people to different (e.g. often medicalized, molecularized, and individual) forms of interventions for their newly defined problems.

For instance, if suicide, by virtue of ongoing epigenetic research, becomes more fundamentally linked to trauma and early adversity—seen as leading to a psychological and biological predisposition to suicidal behaviour—it is imperative to ask what impact this shift might have on the broader meanings of suicide. While these factors have been highlighted in social science and public health studies, focusing on the suicidal behaviour of people who experienced structural violence or specific forms of childhood abuse (Dube et al. 2001; Fullilove et al. 1998; Kira 2001; Straus and Kantor 1994), the traumas focused on in these studies are often analysed in their particular contexts, in terms of the local meanings to be taken account of not only in understanding the effects of the trauma, but also in how to address its origins and outcomes. By contrast, in epigenetic studies, trauma is treated as a black-boxed and dichotomous (i.e. present or not present) category, with the effects of varying experiences in differing contexts generally left undifferentiated.

Much research has been conducted on the relationship of suicide to environmental factors, including social cohesion, social integration, politics, economics, and social change (Bollen and Hoyle 1990; Durkheim 1979; Kawachi and Kennedy 1997; Pierce 1967). These factors have been used to explain locally high rates of suicide (Almasi et al. 2009; CBC News 2009; Krull and Trovato 1994). These and other studies suggest that suicide is inscribed in highly specific social, political, and economic contexts, each of which provide intimate and local meanings to suicidal acts. In Canada, high suicide rates among Aboriginal populations have been linked to long-term structural violence in the form of racism, poverty, marginalization and forced attendance of residential schools, and tuberculosis evacuations, all of which served to destabilize individuals and communities in ways that impact not only the generation directly affected but also subsequent ones (Chansonneuve 2005; Kirmayer et al. 2007; Kral 1998; Stout and Kipling 2003). The endemic suicide in these communities takes on specific meanings. It becomes a powerful shared experience whose discussion and even enactment is a "way of belonging," (Kirmayer et al. 2009; Niezen 2009; Stevenson 2009). As a means of exploring an alternate vision of the relationship between context and suicide, we would like to briefly delve into this last example.

Rates of suicide in Canadian Aboriginal communities are up to ten times the national average (Eggertson 2013). Suicidal behaviour has been on the rise since the 1980s and is highest among young men (Hicks 2007a; Kral 2012; Suicide Prevention Strategy Working Group 2010). Researchers have documented the impact of loss of cultural reference points, loss of connect between elders and youth populations, and loss of family cohesion—a general untethering of individuals from their sources of identity. These and the factors cited above have contributed to rapid cultural change that, in some communities, is viewed as heavily implicated in increased suicidal behaviour.

The life experiences of those people affected for several generations now include what is defined in epigenetic research as early adversity. Specifically, the physical and sexual abuse, along with neglect, experienced by Aboriginal children in residential schools over the course of the twentieth century has led to lasting emotional wounds and changes in the behaviour of many of these people. The result has been that those abused are now, at times, perpetrators of the same forms of violence, leading to transgenerational effects of the schools (Suicide Prevention Strategy Working Group 2010). Researchers confirm the early childhood abuse often experienced by people who take their lives as teens (Niezen 2009). Thus, the immediate and secondary effects of these schools, among other factors, can be seen in the emotional and psychological problems in these communities and their rates of suicide, which have risen dramatically the in the past 40 years.

However, despite the early adversity experienced by many Aboriginal people who commit suicide, anthropologists are reluctant to place too much weight on adversity and insist on the limitations and dangers of a one-idea explanation, and the solutions this explanation might suggest. Suicide, they argue, is not a one-factor problem. They point to the highly variable suicide rates in Aboriginal communities who share forms of collective or individual trauma as evidence of the weakness of one-idea explanations. (Chandler and Lalonde 1998; Kirmayer et al. 2003)

One argument that has been put forward to explain local variable rates of suicide has focused on the integration of the idea of self-destruction as central to group belonging. In the absence of comfort, consolation, and security from their community due to weakened pathways for socialization, for instance, between older and younger generations, self-destruction can become a central value of social life. In these contexts, Ronald Niezen proposes, suicide may gain wider currency as youth abandon "expectations of a better future" and ultimately negate "their personal attachments to life itself" (Niezen 2009, 181). Though there are some understandings of the "social or cultural processes or 'routes of exposure' by which susceptible individuals come to seriously accept the idea of suicide" (Niezen 2009, 184), they nonetheless remain incompletely understood. However, it is clear that they are complex and that there is more going on than unresolved collective grief or individual identification in clusters of suicide.

This perspective is not incompatible with the idea that people who commit suicide in these communities often have suffered a great deal and that some of them might experience psychopathology or "perturbations." However, it does underline the claim that the social study of suicide is complex and entails taking into account documented and recounted historical and social conditions that contribute to the "perturbation" as well as the less tangible influence of local cultures and the very idea of suicide. As such, psychological disturbance does not naturally or predictably follow from adversity or trauma, such as has been seen in the colonial legacy in Canadian Aboriginal communities. What we see in this example is a substantially different view of the role of context in suicide.

Concluding Thoughts: Suicide, Epigenetics and the Social Sciences

While the environmental epigenetics of suicide risk remains a highly emergent area of research—and although it certainly may not have the explanatory power attributed to it by some—its novel style of reasoning has the potential to profoundly shape broader social conceptualizations and man-

agement of suicidal behaviour. Yet, as a number of social scientists have pointed out, there is a danger of what has been called a "molecularization of biography and milieu" in epigenetics research (Niewöhner 2011). Namely, there are worries that the discussions of the effects of environment and social context will be reduced to conversations about molecular mechanisms and that potential interventions may also be more likely to be conceived of on this scale. As Margaret Lock has put it, "Although the contribution of environments, social and physical, to human development, health, and illness, are now well recognized, there is a distinct danger that the molecular endpoints that these variables bring about, and very little else, will receive due attention" (Lock 2013b, 292). We see evidence of such "neoreductionism" (Lock 2015, 151), a potential constriction of attention to environments both in the widespread framing of contextual research as "exposomics" (a study of "environmental exposures") and in the particular focus of some discussions on the potential of epigenetics to produce new molecular targets for pharmaceuticals. Pointing to recent research on maternal epigenetic programming, Sarah Richardson has suggested that rather than being overturned, "traditional forms of genetic determinism and reductionism are [being] subtly reformulated" (2015, 211).

Even if we concede that epigenetics has the *potential* to draw attention to what has long been called "social determinants of health and illness," we should be mindful of the reasons for this rhetorical power. Arguably, this rhetorical power draws from the promise of mechanistic explanations that some attribute to it—a promise which runs deeply counter to other interpretations of epigenetics as contributing to a systems-based understanding of disease and distress as emergent phenomena (Kirmayer and Gold 2012). At a time when it has become, at least in certain settings, more effective to base social justice claims in biological rather than social or political terms (Fassin 2001), it is particularly important to consider the impact of such an epigenetic justification for social justice.

In our own work with epigenetics research on suicide risk, we have found that the understanding of context or environment, which is enacted in experimental work, is one that is molecularized and limited to a select number of factors that have been operationalized and validated in scientific studies. The result is the potential naturalization of, in the case of suicide, early adversity as a primary determinant of suicidal behaviour while the rest of people's lives (e.g. experiences both positive and negative) are bracketed off as confounding "noise." This approach leaves individual experiences of adversity undifferentiated, with little or no understanding of how the black-boxed notion of adversity interacts with the rest of people's life experiences.

It is important to emphasize that such a consequence is unlikely to arise from the particular ethical, political, or even ontological commitments of researchers themselves. Most researchers understand the reductionism they undertake as "pragmatic" (Niewöhner 2011) or "methodological" (Kirmayer and Gold 2012)—in other words, utterly necessary for the conduct of experimental science, but not necessarily entailing any ontological assumptions about the structure of the world itself or the phenomena under study. Our fieldwork suggests that epigenetics researchers are profoundly aware of the limitations which such reductionism entails and that there exists ample opportunity for engagement with social scientists around these important issues.

Equally promising is the fact that this shift toward an increasingly—or at least potentially—non-reductionist biology is being recognized by many in the social sciences. Indeed, one of the interesting and important effects of epigenetics and other work in biosciences is that it is being used by some social scientists to argue for a renewed biosocial agenda (Fitzgerald et al. 2015; Ingold and Palsson 2013). The aim of such cross-disciplinary engagement would be the production of more robust accounts of illness and distress—including suicide, understood in terms of "cultural biology" (Kirmayer 2006), a "neuroanthropology" of the "encultured brain" (Downey and Lende 2012), or "situated biologies" (Lock and Palsson 2016).

Yet, as others have pointed out, in order to attain such engagement, social scientists must do much more than simply celebrate widely publicized findings of environmental epigenetics as validating the significance of social and environmental factors in determining or shaping health outcomes. While there is certainly significant "common ground" between the arguments being made by many anthropologists and sociologists about suicide risk and those of researchers in epigenetics, we would caution against an overstatement of such agreements (even a "strategic" overstatement). A productive engagement between anthropologists, sociologists and researchers in epigenetics can only emerge from an analysis and discussion of the epistemological distinctions between these disciplines.

Notes

1. In this paper, we focus on environmental epigenetics, which focuses on drawing links between environmental changes and gene expression, as opposed to some other domains where epigenetics is also employed.
2. This embedded or porous body of epigenetics is arguably part of a broader environmental turn in the biosciences. An increasingly large number of

researchers engaged in cutting edge work are actively re-imagining the relationship between biology and society. Such changes in the way scientists study body-world and biology-environment relationships cut across the fields of genetics, neuroscience, immunology, endocrinology, and microbiology and have altered our understanding of the means by which factors such as trauma, stress or social exclusion lead to ill health (Chiao et al. 2008, 2010; Fish et al. 2004; Hertzman and Boyce 2010; Kuzawa and Sweet 2009; Slaby and Choudhury 2012; Thayer and Kuzawa 2011). Moreover, such work is increasingly taking place at the intersections of these domains of biological research. For social scientists, many of whom are also intent on destabilizing traditional conceptions of the skin-bounded body and the autonomous individual, the embedded body of epigenetics and other emergent biosciences offer both possibilities and challenges, which we discuss at greater length below.

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Part IV

Social Epidemiology

22

The Embodiment Dynamic over the Life Course: A Case for Examining Cancer Aetiology

Michelle Kelly-Irving and Cyrille Delpierre

The Concept of Embodiment in Social Epidemiology

The starting point for this chapter is Nancy Krieger's definition and use of the term "embodiment" within the field of epidemiology, and more specifically social epidemiology. Krieger can be credited with developing one of the only theoretical frameworks within epidemiology, the Ecosocial Theory, wherein she states that "embodiment as, an idea, refers to how we, like any living organism, literally incorporate, biologically, the world in which we live, including our societal and ecological circumstances"(Krieger 2005). We will briefly describe the concept of embodiment here and attempt to build on Krieger's concept in the light of further developments in research focused on how the social becomes biological. We will then argue that cancers should be examined as a set of pathologies exemplifying life-course embodiment processes from early life. Through the literature and our own empirical findings we will explore the hypothesis that adult cancers may originate in early life through the embodiment of adversity.

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Background on Embodiment

Most researchers in epidemiology employing the term embodiment do so either without specifying its meaning or by briefly referring to Krieger's definition. This may be because the idea behind embodiment is somehow intuitive. Our popular culture, novels and films are riddled with stories about how people embody their experiences. The premise of most "alternative" therapies is founded on the intuitive concepts of the lived-body (Leder 1984). When we attempt to deconstruct the concept, the crux of embodiment exists in the mundane. Within the layers of structured complexities characterizing human societies, embodiment is simply the outcome of everyday life.

The notion of embodiment is central to the only comprehensive "theory" of social epidemiology, formalized by Krieger (Krieger 1994; Krieger and Zierler 1996; Krieger 2001b). She described the Ecosocial Theory as broadly encompassing historical and contemporaneous literature on the social origins of disease and illness. Embodiment has been described by Krieger as "a concept referring to how we literally incorporate, biologically, the material and social world in which we live" (Krieger 2001a). The idea is synonymously referred to as biological embedding by Hertzman (1999), this occurs when experiences alter human development and biology. The way in which this happens is influenced by systematic differences in social environments that endure over time and have the capacity to affect individuals over their life course (Hertzman 2012; Hertzman 1999). Krieger and Davey Smith (2004) further explored and exemplified embodiment as a construct which "invites us to consider how our bodies, each and every day, accumulate and integrate experiences and exposures structured by diverse yet commingled aspects of social position and inequality" (Krieger and Davey Smith 2004). The life-course approach was another conceptual framework that entered epidemiology via sociology and social psychology (Giele and Elder 1998) around the same period as Ecosocial Theory, as described by Kuh and Ben Shlomo (1997). The life-course framework proposed conceptual mechanisms to facilitate the difficult move from theoretical processes towards empirical model testing (Ben-Shlomo and Kuh 2002).

The mechanisms include those typically referred to by life-course researchers in epidemiology: accumulation, describing how exposures tend to build-up cross-sectionally and over time in a snowball-like fashion, sensitive periods, referring to developmental time-windows when humans are biologically more sensitive to their environments and pathways, referring to social trajectories

over time leading towards health states (Ben-Shlomo and Kuh 2002). As an illustration, Galobardes et al. (2004) have shown that the mechanisms of sensitive periods and accumulation, underlined in the life-course epidemiology framework, are all likely to link early-life conditions to adult morbidity and mortality (Galobardes et al. 2008; Power et al. 2013). They are conceptually useful mechanisms; however, they usually cannot be disentangled and often operate simultaneously; The pathways mechanism being particularly difficult to distinguish from the other two. These classic life-course mechanisms vary according to when they occur along the life-course and the timing of mechanisms relative to one another. The definition of embodiment as a dynamic allows for this inherent complexity. A key component to a dynamic definition of embodiment is timing and time, allowing for pathways of embodiment and the cumulative interplay of exposure, susceptibility and resistance, key tenets in Krieger's Ecosocial Theory. But the challenge of understanding how this occurs, however, is still an obstacle in the study of health, disease and inequalities.

Embodiment, the Environment and Social Facts

Here, we refer to the environment in its broadest sense, as a set of concentric circles containing all facets characterizing human lives, moving from the most "proximal" and close to the individual, towards "distal" social and societal structures. Many academics from different disciplines have used this image of concentric circles (Bronfenbrenner 1977; Dahlgren and Whitehead 1991) akin to the idea of Russian Dolls or Chinese boxes (Susser and Susser 1996).

The structural nature of the societies within which humans interact has a strong influence over the way our environments are organized and how we interact with them. This is evocative of Durkheim's social facts, which he described as ways of acting, thinking and feeling, external to individuals, and upon whom they exert a coercive force (Durkheim 1937). The daily interactions between individuals and their surroundings occur via social, economic psychological and cultural processes, and ultimately to modifications of a biological nature. Humans become altered and changed by the environment they spend time interacting with. The nature of these alterations is complex and cyclical, affecting in turn physiological, psychological, social and cultural factors.

Embodiment is therefore a dynamic set of social and biological processes and interactions between individuals within a population and their environments over time. It

is a dynamic that is socially stratified representing the past environmental landscape and an ongoing response to the present environment. Humans thus come to physically represent their past environments in their present state through a constant process of change.

The social stratification of the embodiment dynamic is important to take into account. This strong influence of social structures prevails over the embodiment dynamic, and leads to the observation of the social gradient in health over time and across populations (Hertzman 2012).

Embodiment and Health

Embodiment is a latent variable. This means it cannot be directly measured, but is inferred. To understand the processes involved in producing socially graded health states over time (Hertzman 2012), researchers attempt to measure response states at any one time. They usually use a proxy measure as a snapshot view representing the embodiment dynamic. For example, body mass index (BMI) may be used to describe a person's health at one time point; however, it is measured as a relationship between height and weight, reflecting something of past biological processes responding to the environment, behavioural tendencies and genetics. BMI reflects something of one set of relationships within the embodiment dynamic. Depending on their motivation, researchers may use criteria from biomedical disciplines which typically define when a state is "normal" or "pathological" (Canguilhem 1991), so in the example of BMI, definitions of 'normal' versus 'abnormal' body size. Such interpretations of embodiment by researchers or clinicians as being expressed via disease states or poor developmental outcomes are driven by disciplinary preoccupations, trends in science and hypothesis testing. They should not be confused with the underlying neutral form that the embodiment dynamic takes, being neither good nor bad, diseased nor undiseased, adapted nor maladapted. In short, embodiment is a set of relationships and responses, whether those are deemed positive or not depends upon the research object or clinical question. In terms of health, public health and epidemiological research, this boils down to understanding developmental and health states as expressions of lives lived.

Unobservable by nature, the embodiment dynamic is predictive, in biomedical terms, of future morbidity and mortality. Embodiment can be approximated via physical and biological measurements ideally combined with measurements of perceived state (perceived health, wellbeing). However,

such measurements will always act as proxies for an unobservable latent state which is in the process of evolving. Thus, measurements made at one time point reflect something of a past state that no longer exists, while repeated measurements at multiple time points reflect something of a set of previous interrelated states. These measurements will provide approximations of the embodiment dynamic, and thus how a person or population has responded to their environment over the course of their life.

Encompassing multiple life-course mechanisms, it is via the embodiment dynamic that health inequalities are generated from early life. The dynamic explains how social and psychosocial elements structured into different layers within the environment are causally related to physiologically measureable states, morbidity and mortality. Therefore, the embodiment dynamic represents the complexity of interrelated processes and mechanisms leading to the social-structuring of human developmental states and health outcomes from early life. Embodiment may be viewed as the dynamic that leads to population patterns of health and illness. As such, understanding these covert interactions occurring from the early stages of the life-course before their emergence as health outcomes or health-care trajectories is fundamental to the success of any attempts to thwart the socioeconomic gradient in health. The nature and cadence of the embodiment dynamic varies over the life-course, and may be made up of many different processes; however, it always encompasses at least one biological mechanism.

Biological Mechanisms

Researchers from many disciplines may wish to breakdown the embodiment dynamic into plausible mechanisms and pathways. For example, the mechanism of attachment (Bowlby 1969) may be an important process for understanding the nature of interpersonal relationships in psychology, or language acquisition over childhood may be key to a cognitive scientist. An anthropologist may wish to examine cultural processes, and a medical researcher may take an interest in pathological mechanisms. As social epidemiologists, we are interested in understanding how social gradients in health are produced. The social epidemiology and medical sociology literature has highlighted associations between measures of the socioeconomic environment and subsequent morbidity and mortality. We aim to study the specific pathways along which these social-to-biological associations are likely to operate. A growing body of research hypotheses on the specific pathways that may operate between different environmental factors and embodiment can be identified and tested,

informing deductive methods and the rejection of hypotheses or formulation of new ones (Kelly-Irving et al. 2015). However, when taking an interest in how many of these different processes may affect the production of the social gradient in health, we suggest that one or several biological mechanisms are always ultimately implicated. Below, we outline the two major types of biological mechanisms.

Mechanisms of Exogenous Origin

Our biological systems may become modified by the introduction into our bodies of external entities. Blane et al. refer to these types of biological mechanisms as material, consisting of the "living (bacteria, viruses) and inert (asbestos fibres, folic acid) materials which have an impact on the body's structure and immune system. Impact can be beneficial (essential gut flora; folic acid-dependent embryonic neural tube development), harmful but contained (antibodies; scar tissue) or pathological (respiratory tuberculosis; mesothelioma)" (Blane et al. 2013). Social position, through its influence on the nature of the external environment we live in, such as the quality of housing or the type of occupation, may act on the probability of coming into contact with these exogenous factors that become incorporated. An example of this process is in Bartley et al.'s findings from the 1958 Birth cohort study, whereby financial hardship in early life in Great Britain was associated over the life-course with a lower lung function at the age of 45, taking into account many other social and behavioural factors (Bartley et al. 2011). The authors hypothesized that exposure to poor quality damp and overcrowded housing was likely to be the key plausible pathway between social position and lung function.

Mechanisms of Endogenous Origin

This set of mechanisms refers to biological and physiological responses occurring within our bodies in response to environmental changes. In Blane et al., these correspond to the Central Nervous System-mediated mechanisms (Blane et al. 2013) and are sometimes generically referred to as "psychosocial". Perceptions, emotions, personality, self-efficacy and many other mechanisms located in the mind can lead to a cascade of responses from the neuroendocrine system, to physiological stress responses in various biological systems (neurological, inflammatory, hormonal, etc.) (Lupien et al. 2009). During a phase of rapid development, a biological system is more sensitive to

exposures in the environment, and especially deviations from "normal" exposures expected during that particular phase of development for that particular system (Bruer 2001). Given the vast array of developmental processes occurring between conception and adolescence, no single sensitive period can be identified; rather, differing levels of sensitivity are constantly shifting for different systems, which in turn vary in their complexity. Every developmental window is in fact characterized by a different susceptibility depending on various environmental factors. Due to this developmental sensitivity that is more pronounced in children, the experience of acute or chronic stressors, which can induce several known biological responses (Shonkoff et al. 2012a, b), could have an impact on subsequent biological and behavioural functions depending on the timing of initial exposures and be mediated subsequently by later exposures. For this reason, childhood exposure to adversity is a possible source of both acute and chronic stressors, and can be examined as a potentially important initial exposure on a pathway towards adult ill health. We give a detailed example of this below taking the example of cancer.

Links Between Mechanisms of Exogenous and Endogenous Origin

Both broad types of biological mechanisms, exogenous or endogenous, may implicate molecular-level transformations, such as epigenetic or even genetic changes, which in turn may alter endogenous biological mechanisms. The two types of mechanisms may also interact and affect each other. Once an exogenous entity has become incorporated, it may affect endogenous biological systems positively, negatively or have a neutral effect. For example, humans became habituated to living in relative harmony with living "pathogens", which effectively infected us, but remained harmless. According to Rook et al. "the Old Friends mechanism states that mammals co-evolved with an array of organisms that, because they needed to be tolerated, took on a role as inducers of immunoregulatory circuits" (Rook et al. 2013). These organisms include bacteria, helminths (worms), chronic infections and environmental organisms from animals and water that humans evolved, and lived with until recently. Separating biological mechanisms into exogenous and endogenous types is of course merely a construct which might facilitate our understanding of the embodiment dynamic. In many pathological processes both are likely to be at play, however, identifying them may facilitate our understanding about how to prevent disease and improve health.

Embodiment, Life Course and Cancer

In this section, we introduce the idea that cancer should be studied as a set of pathologies which may represent life-course processes and the embodiment dynamic expressed as a disease. The life-course approach has been widely used to explore many chronic diseases, but it has often left out cancer. However, cancers are an interesting set of pathologies which simultaneously have a common root in the immune system, yet consist of a number of different aetiological processes and biological mechanisms. Cancer is also becoming one of the major causes of morbidity and mortality worldwide.

Why Cancer?

Cancer development has mainly been considered as a consequence of DNA mutations and consequently tends to be viewed through the lens of molecular biology. However, cancer may be a disease model particularly relevant to investigate how the social becomes biological, via a life-course approach. Many socially stratified biological mechanisms have been identified as being part of the causal chain of risk for cancers: material exposures (including inert ones such as asbestos, or living ones such as Human Papilloma viruses) and behavioural mechanisms (including tobacco consumption and fatty food intake).Furthermore, the life-course approach is of particular interest when studying cancer because of the long latent time period during which exposures occur, before the onset of disease. There is increasing evidence for the role of chronic stress in cancer development and progression (Antoni et al. 2006; Lutgendorf et al. 2010).

Upstream, cancer aetiologies differ, some being exogenous in origin (viruses, smoking) and others endogenous (hormonal cancers). However, here, we argue that all cancers are at some point rooted in the immune and inflammatory system, and thereby their initiation is susceptible to stress-related factors affecting biological mechanisms of endogenous origin at some point along the aetiological pathway. If the immune system is impaired from killing-off damaged cells, the risk of developing tumour cells is heightened (Stewart and Abrams 2008). A damaged immune system is also an important accelerator of cancer progression (Kim et al. 2007). This root in the immune system is currently the source of the most promising immunotherapy treatment for many cancers (Dustin 2016).

Disadvantage in early life has been considered an important determinant of morbidity and mortality for many years, but the variables used to characterize

childhood circumstances have often been non-specific, and the mechanisms involved remain to be clarified. Stressful events are likely to be experienced differently depending on an individual's hierarchical position on the social gradient. Individuals lower on the social gradient may be more vulnerable to the physiological or behavioural effects of stressful environmental exposures with fewer resources and coping strategies at their disposal compared to individuals with a higher social position (Baum et al. 1999). Social gradients may also confer stress to individuals via status anxiety, which has also been shown in non-human primates (Sapolsky 2005). Intra-familial conditions occurring from conception into adolescence may programme physiological responses during sensitive periods of development, altering an individual's biology, rendering them poorly or, conversely, well adapted to their environment and subsequent exposures later in life (Bailey et al. 2001). We hypothesize that taking into account early-life exposures to chronic stress is important in understanding the aetiology of cancers, which have a common root in the immune and inflammatory systems. These systems form part of the overall physiological stress response.

Stress and Cancer: The All-cancer Immune System Model

One of the main biological mechanisms used by organisms to adapt to their environment involves stress response systems, through the hypothalamic-pituitary-adrenal (HPA) axis and sympathetic-adrenal-medulla (SAM) system. These systems control the release of stress hormones such as glucocorticoids (cortisol), catecholamines (adrenaline and noradrenaline) and other hormones (prolactin, oxytocin). The association between stress and cancer development and progression has been shown in biological studies. In animals over the life course, modification of HPA and SAM activities have been shown to alter many biological mechanisms implicated in tumorigenesis, including tumour growth, cell migration and invasion, inflammatory and immune responses (Antoni et al. 2006). This is well described in vitro and in animal models of chronic restraint stress or of social isolation (Antoni et al. 2006; Gidron and Ronson 2008; Lutgendorf et al. 2010; Thaker et al. 2006). When exposed to chronic stress, "the body remains in a constant state of overdrive" (Lutgendorf et al. 2010) with adverse consequences on the regulation of systems implicated in cancer progression. In terms of stress exposures, maternal care defects in animals have been shown to increase HPA responsivity to stress. These effects seem to be derived from changes in forebrain corticosteroid receptor systems which determine glucocorticoid negative feedback sensitivity (Meaney

et al. 1994). As a key step in controlling the stress response, the glucocorticoid pathway has been dissected in animal (Weaver et al. 2004) and human (Vaiserman 2015) models of early-life stress and found to be associated with epigenetic modifications. These are global responses applicable to all cancers (Antoni et al. 2006).

In humans, evidence is more difficult to obtain; therefore, available studies are sparse and mainly based on correlations and not on causal associations. However, an increasing literature suggests links between psychosocial factors, like stress, depression or social isolation, and cancer progression through activation of HPA and SAM systems (Antoni et al. 2006). Accordingly, altered levels of stress hormones have been observed in human cancers (Antoni et al. 2006), and a number of correlations between psychosocial stress, biological pathways related to tumorigenesis and cancer have been reported in humans. For instance, psychosocially stressed patients have fewer leucocytes, decreased cytotoxic T-cell and natural killer cell activities, high levels of serum cortisol (basal), acute-phase proteins, increased plasma concentrations of inflammatory cytokines and more inflammatory responses including DNA damage, growth and angiogenic factors, and proteases (Gidron and Ronson 2008). Stress-related immunological changes bring about declines in natural killer cell activity by depressing their ability to respond to tumours or virally infected cells, and causing a reduction in the body's defences linked to the repair of damaged DNA (Kiecolt-Glaser and Glaser 1999). This *all-cancer immune system model* means that research questions assessing cancers together, as well as by aetiological group, deserve scientific consideration (Fig. 22.1).

The social environment and the stress that it creates, as well as individual perceptions of external events, have an effect on the level of activity of central and sympathetic nervous systems (hypothalamic-pituitary-adrenal axis; noradrenaline and adrenaline levels). Psychosocial stress modifies the activity of neuroendocrine system and glucocorticoids (cortisol), catecholamines (adrenaline and noradrenaline) and other hormones (prolactin, oxytocin). Over time, these modifications can alter physiological mechanisms implicated in tumorigenesis, including oxidative metabolism, DNA repair, oncogene expression, in the production of growth factors and other regulators of cell growth, and in immune function. It has been shown that endocrine factors regulate activity of some angiogenic (formation of new blood vessels) factors, inflammatory cytokines and molecules of adhesion implicated in the tumour progression. Several studies have shown a relationship between depression, the lack of social support and neuroendocrine activity. For example, the lack of social support is associated with a high level of VEGF (vascular endothelial

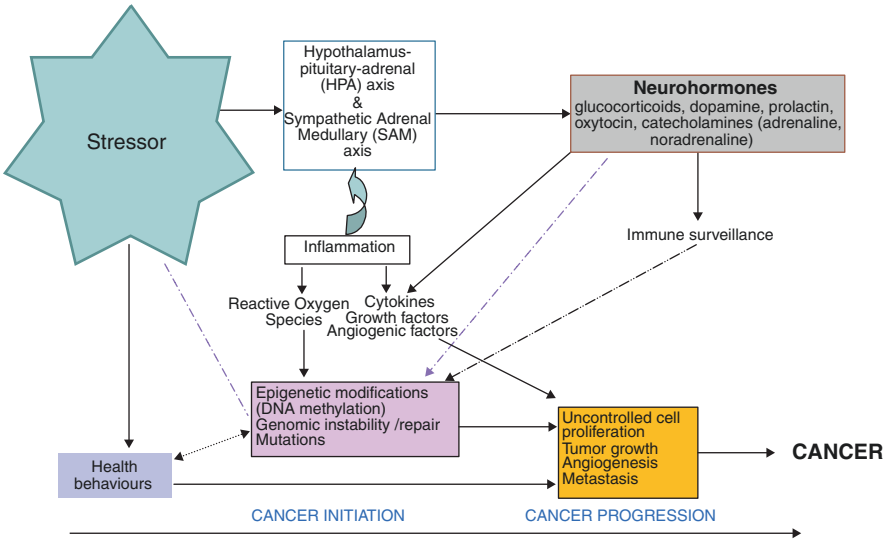

Fig. 22.1 Plausible biological pathways involved in exposure to stressors and cancer development (based on Kelly-Irving et al. 2013a)

growth factor), potentially mediated by an elevated level of noradrenaline which improves the production of this growth factor (Cohen et al. 2008).

Epigenetic modification—a molecular-level mechanism involved in the developmental origins of disease—has thus been used to explain a number of common pathologies such as cardiovascular diseases, psychopathologies and cancer (Hochberg et al. 2011; Szyf 2009). Furthermore, epigenetic modifications have been put forward as a plausible link between environmental factors, alterations in gene expression and disease susceptibility (Jirtle and Skinner 2007). In recent years, research has been carried out establishing the link between psychosocial and socioeconomic exposures in early-life and epigenetic modifications potentially leading to adverse health outcomes later in life (Borghol et al. 2011). In animals, this has been described through the modification of the glucocorticoid receptor (GR). The most commonly described epigenetic mechanism is methylation, which occurs when a methyl group (CH3) attaches itself to the cytosine base, one of the bases found in DNA and RNA. Usually, this results in the cytosine base not being transcribed into RNA, potentially affecting gene expression. Articles linking environment, DNA hypomethylation and cancer development suggest that the environment may modify DNA and gene expression (Nise et al. 2010). Methylation was observed in the hippocampus of rat pups in response to maternal care whereby the levels of methylation at the 5'-end of the GR gene promoter (seg-

ment of DNA that initiates transcription of a particular gene) in the hippocampus were inversely proportional to the extent to which rat pups were licked, groomed and nursed by their mothers. Furthermore, the increased level of methylation at the GR promoter was correlated with reduced GR transcription confirming that levels of gene expression were indeed affected by methylation (Weaver et al. 2004). Accordingly, in humans, hypermethylation and reduced expression of the GR gene was found in the post-mortem hippocampus of suicide victims with a history of abuse in childhood but not among suicide victims without a history of childhood abuse or controls who died suddenly from causes other than suicide (McGowan et al. 2009).

Given that the glucocorticoid pathway is strongly involved in stress modulation, and immune response, methylation of the GR supports the idea that psychosocial stress may have an impact on tumorigenesis, and that this may be triggered and sustained by epigenetic changes. Since methylation is a potentially reversible biological signal, DNA methylation patterns can be used as a plastic biological framework that might play a role in the adaptive responses to changing environments early in life and possibly throughout life (Szyf 2009). Patterns of methylation can be acquired during life or be inherited from the mother's behaviour affecting the DNA methylation patterns of the offspring (Weaver et al. 2004). Consequently, an individual's health status is the result of a remodelled epigenome which itself is the outcome of complex cumulative interactions between the genotype and the environment over time (Hochberg et al. 2011), wherein early-life exposures are so critical (Gluckman et al. 2011).

Another set of biological mechanisms potentially linking stress to cancer is via risky health behaviours. Previous studies on psychosocial stress have established links between adversity in childhood and an increased risk of smoking, alcoholism, early sexual activity and having multiple sexual partners (Anda et al. 1999, 2002, 2006; Chung et al. 2010; Dube et al. 2003, 2010), all of which are risk factors for cancer. Epigenetic mechanisms, which can be instigated by exposure to stressors, such as early adversities, have been identified as underlying addiction and neurobiological responses to addiction (Wong et al. 2011) as well as being linked to disruptive behaviour problems in children (Tremblay 2010).

Early-Life Stress and Cancer: Life-course Evidence

We have described some of the possible biological pathways and mechanisms linking early-life stress to cancer in adulthood, acting on physiological processes applicable to an all-cancer model (Figure 21.1). Both the accumulation

of environmental exposures over time, or exposure occurring during sensitive periods, may lead individuals along trajectories towards health outcomes. Stress can occur across the social gradient, and the way in which neurobiological and behavioural processes respond to these psychosocial exposures is likely to be different based on individuals' social positions (Seeman et al. 2010).

Over the last number of years, we have been testing the early-life stress and all-cancer model by developing hypotheses within a life-course framework. In our previous work, we defined Adverse Childhood Experiences (ACE) as intra-familial events or conditions in the child's immediate environment causing chronic stress responses (Kelly-Irving et al. 2013b). These include notions of maltreatment and deviation from societal norms, and need to be distinguished from events or conditions linked to the socioeconomic and material environment. Importantly, we based this definition on prospectively collected data. Our definition of ACE has been influenced by previous epidemiological studies of ACE, notably the San Diego study (Felitti et al. 1998), the Australian study (Rosenman and Rodgers 2004) as well as discussions on ACE by a WHO expert committee in 2009. We sought to disentangle elements within the material environment, such as poverty, from adversities caused by dysfunction or disruption amid the child's important relationships, such as those with family members. Adversity caused by poverty is no doubt a source of psychosocial stress; however, the child is more likely to cope with this type of stress if his/her close family relationships are positive and confident. ACEs are, therefore, likely to interact with socioeconomic disadvantage. Previous studies have described a strong graded relationship between ACE and cause of death, including from cancer (Felitti et al. 1998). Fuller-Thomson et al. found an association between childhood physical abuse and self-reported cancer after adjusting for mediators such as smoking and alcohol as well as other confounders, suggesting that a direct association between childhood adversity and cancer may exist (Fuller-Thomson and Brennenstuhl 2009). The main methodological flaw in these studies is that ACE was self-reported by adults who were asked questions about trauma and adversity they may have experienced during childhood. Such questions are inevitably vulnerable to recall bias, where adults with poor health may be more likely to report adversity during childhood, but often this is the only method available to researchers exploring the consequences of childhood adversity.

In epidemiological studies, evidence of a direct association between exposure to stress and cancer incidence is mixed and inconclusive (Schraub et al. 2009). These studies combine a number of definitions or forms of stress reported in adulthood that may be relevant to the chain of risk leading to cancer development, but do not relate to ACEs specifically. A Danish cohort

study on 8736 men and women found no direct association between cumulative stressful life events collected retrospectively and cancer incidence, though they did identify a relationship between stress and unhealthy lifestyles (Bergelt et al. 2006). Ollonen et al. (2005) found support for an overall association between stressful life events and breast cancer risk in their Finnish case-control study (Ollonen et al. 2005). Evidence from the West of Scotland collaborative study, a prospective cohort study of 5743 men and 991 women, found an association between medium levels of reported daily stress and breast/prostate cancer development after adjusting for prior confounders and mediating risk factors (Metcalfe et al. 2007). Conversely, Nielsen et al. (2005) found a significant reduction in the hazard ratio of women exposed to perceived stress after adjusting for confounders. The authors explain that chronic stress impairs oestrogen synthesis, which is a known risk factor for breast cancer (Nielsen et al. 2005). A meta-analysis of studies on the association between stress and breast cancer did not support an association between stressful life events and breast cancer risk (Duijts et al. 2003). Keinan-Boker et al. have observed in a large cohort (more than 4,900,000 person-years) a higher risk of all-site cancer among Israeli Jews who were potentially exposed to the Holocaust than those who were not. Age at exposure modified the strength of this association: the risk of cancer was the highest for those who were born between 1940 and 1945 and thus exposed to the Holocaust between 0 and 5 years (Keinan-Boker et al. 2009). This suggests a stronger effect of early-life stress.

An important difference between our work and other research on ACE is that the data used here are prospective. A second important difference is that this work conceptualizes the psychosocial consequences of ACE as a separate mechanism in relation to subsequent health outcomes versus other studies where early socioeconomic adversity has been studied (Tubeuf et al. 2012). We used data from the National Child Development Study (NCDS), a prospective birth cohort study with information collected over 50 years. At baseline, the NCDS included all live births during one week in 1958 (n = 17,416) in Great Britain. By the time the respondents turned 50 years, 9790 among them were still participating. Among this sample, some people answered questions about whether they had cancer. Self-reported cancer incidence was based on 444 participants reporting having had cancer at some point and 5694 reporting never having cancer. ACE was measured using reports of: (1) child in care, (2) physical neglect, (3) child's or family's contact with the prison service, (4) parental separation due to divorce, death or other, (5) family experience of mental illness and (6) family experience of substance abuse. The resulting ACE variable had three categories—no ACEs/one ACE/2+ACEs and was used to test for a relationship with cancer. We considered this infor-

mation on conditions and events as a proxy for chronic stress from early childhood up to the age of 16. Information on socioeconomic characteristics, pregnancy and birth were extracted as potential confounders. Information on adult health behaviours, socioeconomic environment, psychological state and age at first pregnancy were added to the models. Multivariate models were run using multiply-imputed data to account for missing data in the cohort. An accumulation of ACEs remained a strong predictor of cancer in women, after taking important potential mediating factors at age 23 years into account, including smoking, drinking, BMI, age at first pregnancy and socioeconomic factors. Women who experienced one ACE in childhood had a 30% increase in their risk of having a cancer before 50, and those who experienced two or more ACEs doubled their risk relative to women who had had no childhood adversities. The strength of the relationship between adversity and cancer was of the same magnitude as that observed between age at first pregnancy and cancer, a well-known risk factor for breast cancer. The findings also highlighted that smoking was significantly related to reporting a cancer before 50 years among women. In men, the models showed no association between early-life socioeconomic variables or perinatal variables and cancer. ACE was not significantly associated with cancer, and neither were social variables at age 23 years, or smoking and alcohol age 23 years. Rather than indicating a sex, or gender difference, the lack of association between virtually any of the variables and cancer among men may be due to a lack of power ($n = 93$). The distribution of cancer types observed in the cohort will continue to evolve over time, and begin to represent a greater proportion of men due to the increased occurrence of prostate and lung cancer among men >50 years.

Overall, these findings suggest that cancer risk may be determined, in part, by exposure to stressful conditions and events early in life. This is potentially important in furthering our understanding of cancer aetiology, and consequently in redirecting scientific research and developing appropriate prevention policies. The major limitation of this work was the self-reported nature of the cancer data. We hope to validate and further explore these initial findings by using up-to-date exhaustive data on cancer from the UK registries on both cohort members. Scientific and social investment in these research tools is key.

Conclusions and Future Research

In this chapter, our aim was to define and describe the notion of embodiment as a neutral adaptive dynamic occurring over the life-course. We put forward the idea that health states, however they are measured, are expressions of the

dynamic. The coercive nature of the social structures that make up the outer layer of our environments means that the embodiment dynamic occurs differentially across the social strata. One or several biological mechanisms, be they exogenous or endogenous in origin, are a necessary component of the embodiment dynamic, which may be preceded by other mechanisms defined by a disciplinary approach. To exemplify the embodiment process over the life-course, we take the example of cancers, as a set of chronic diseases which develop and progress over the life course with a shared inflammatory/ immunological root, but often different aetiological processes. We argue that exposure to early-life chronic stress may be an important set of biological mechanisms affecting the development of cancers. Even in the case of cancers where the aetiological pathway stems from an exogenous exposure, such as tobacco smoking for example, we hypothesize that understanding how chronic stress initiated early in life may adversely affect the inflammatory and immune systems' defence against tumour cell development may facilitate our understanding of why some smokers develop cancer, while others do not and move beyond genetic explanations.

We conclude on two important points that we think would move us forward in our understanding of how our social environments becomes embodied. First, a scientific shift is needed in our approach to understanding chronic diseases, notably cancer. The current landscape maintains its focus on proximal risk factors for these diseases, mainly individual behavioural ones. The public are constantly being fed information on what not to eat, drink or do in order to avoid developing cancer. However, behaviours are merely consequences of social and cultural factors that are structured into our societies and acquired from an early age. We could even go so far as to say that behaviours may be a form of adaptive response allowing us to cope with environmental challenges. The need for a scientific shift where we place biological mechanisms in their social contexts is fundamental. This would affect how we think about diseases and go about studying them allowing us to address real primary prevention. This shift needs to come from scientists, researchers, doctors—not the general public, which is already receptive to thinking about cancers as a product of life-course processes and an embodiment dynamic. Second, we need to continue our investment in longitudinal studies, such as cohorts and panel studies, collecting data of a social scientific and biomedical nature, but also biological measures of general processes and registry data for pathologies. With this type of data, plausible hypotheses on the pathways between the social and the biological can be tested—and, most importantly, links can be made between life-course research and complex real-life interventions to improve health and reduce inequalities.

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23

Epigenetic Signatures of Socioeconomic Status Across the Lifecourse

Silvia Stringhini and Paolo Vineis

Why Epigenetics Is Useful in Investigating the Effect of Socioeconomic Status

Epigenetics—defined as "the study of mitotically heritable and reversible molecular information outside of the DNA sequence" (Ladd-Acosta and Fallin 2016)—offers a conceptually sound paradigm to understand long-term acquired susceptibility to disease. As opposed to genetics (i.e. inherited predisposition to disease related to structural changes in DNA sequence) epigenetic changes are sensitive to environmental influences and are amenable to preventive interventions. Their reversibility (at least for some of the changes) makes preventive action possible and in fact realistic, as we see below in the example of smoking. In this chapter, we will discuss the role of epigenetics in ageing as well as the potential role of epigenetics in the biological embedding of socioeconomic factors. Further, we will take the example of smoking-induced epigenetic modifications to speculate on the reversibility of epigenetic changes triggered by environmental stimuli.

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Evidence for the strong influence of socioeconomic factors on health, morbidity and mortality dates back through most of recorded history (Townsend and Davidson 1982; Marmot et al. 1984; Mackenbach et al. 2008) and has been confirmed by recent reports, (Chetty et al. 2016; Mackenbach et al. 2016; Stringhini et al. 2011; Mayhew and Smith 2016) with studies showing a widening in relative inequalities in mortality (Chetty et al. 2016; Mayhew and Smith 2016) despite falling absolute inequalities in some countries (Mackenbach et al. 2016). This is particularly true for lifestyle-related diseases such as type 2 diabetes (T2D) and cardiovascular disease (CVD) (Kanjilal et al. 2006; Imkampe and Gulliford 2011; Espelt et al. 2011; Jemal et al. 2008; Bronnum-Hansen and Baadsgaard 2008; Charafeddine et al. 2009). With the burden of cardio-metabolic disorders expected to rise rapidly over the next decades in low and middle-income countries (Mathers and Loncar 2006), the understanding of the mechanisms driving these inequalities is a major public health priority (World Health Organisation 2011). First, health differences by socioeconomic status (SES) can occur because of "health-related selection", through which people with poorer health are selected out into lower social classes. Although health-related social mobility cannot be completely ruled out, decades of epidemiological research have established that it is mostly the SES of individuals which influences their health, through differential exposures to health hazards (Elovainio et al. 2011; Bartley and Plewis 1997; Fox et al. 1985; Power et al. 1996). Several processes of social causation have been postulated, including: (1) access to/use of medical care (van Doorslaer et al. 2006; Kelly-Irving et al. 2011); (2) access to health information (Abel 2008; Kickbusch 2001); (3) patterns of unhealthy behaviours (smoking, heavy drinking, unhealthy diet, physical inactivity, drug use) (James et al. 1997); (4) exposure to environmental hazards (Lynch et al. 1997b, 2000; Siegrist and Marmot 2004, Mitchell et al. 2002); (5) exposure to stressful situations (Lantz et al. 2005; Siegrist and Marmot 2004); (6) access to resources mediating the physiological consequences of stress (i.e. social relationships and support, cultural capital) (Abel 2008; Lantz et al. 1998); (7) early life experiences (Lynch et al. 1997a); (8) time preferences (van der Pol 2011). Because no single factor can entirely explain the SES-health association, and most of the underlying mechanisms are interrelated and differently relevant depending on the health outcome, the understanding of the determinants of social inequalities in health is particularly challenging, and the implementation of effective policies to reduce those remains problematic.

In the last years, research has expanded with the aim of identifying the biological mechanisms through which SES is embedded and eventually "gets

under the skin" (Krieger 2005; Hertzman and Boyce 2010; Crimmins and Seeman 2004). This new research area stems from the argument that if differences in the social environment are causally related to health, then differences in social dimensions must express themselves in terms of variations in biological factors that are linked to health. The identification of these factors might be important not only for clarifying the complex mechanisms involved in the social distribution of diseases, but also for better targeting public health interventions aimed at reducing these inequalities. Epigenetics offers a model for mediation of SES effects *via* risk factors (including behavioural, environmental and psychosocial risk factors), that are influenced by socioeconomic factors and are related to epigenetic modifications. Further, epigenetics can potentially be a direct target of social adversity, for example, through a direct effect of social adversity in early life on DNA methylation (DNAm). In fact, although a relatively large proportion of the effect of SES on health status is explained by common risk factors for chronic diseases—like smoking or poor diet (Stringhini et al. 2011, 2012)—part of the socioeconomic gradient in health still remains unexplained after accounting for these factors, suggesting that there is something additional in SES that still needs to be identified. Further, the actual biological mechanisms linking the social environment to known or unknown risk factors to health are not fully understood.

Human and animal studies have identified several interrelated processes through which the social environment could be embedded, including dysregulation of the hypothalamic-pituitary-adrenal axis (HPA), inflammatory processes, neural function and structure, and, ultimately, epigenetic mechanisms (Rutter 2012). In humans, low SES across the lifecourse has been associated with greater diurnal cortisol production (Cohen et al. 2006a, b; Miller et al. 2009; Chen et al. 2010), increased inflammatory activity (Ranjit et al. 2007; Loucks et al. 2010; Carroll et al. 2011), higher circulating antibodies for several pathogens (suggesting dampened cell-mediated immune response) (Steptoe et al. 2007; Dowd et al. 2009), reduction in prefrontal cortical grey matter (Gianaros et al. 2007), and greater amygdale reactivity to threat (Gianaros et al. 2008; Gianaros and Manuck 2010). Evidence is accumulating for a crucial role of epigenetic modifications induced by the experience of social adversity in initiating these physiological dysregulations (Miller et al. 2011), in particular those related to the immune function. More specifically, human and animal studies have shown that SES influences DNA methylation and gene expression, in particular across genomic regions regulating the immune function (Miller et al. 2009; Tung et al. 2012; Borghol et al. 2012; McGuinness et al. 2012; Stringhini et al. 2015). Indeed,

if we assume that the social environment has a causal impact on health, then it must leave a trace which is remembered and stored, probably as an epigenetic modification.

Epigenetic events, including DNA methylation and histone modifications, are increasingly recognized as key mechanisms involved in response to environmental stimuli, and in disease onset. In particular, we consider in this chapter examples concerning DNA methylation. DNA methylation typically occurs in a CpG dinucleotide context (i.e. where a cytosine is followed by a guanine). The addition of a methyl group to DNA cytosines at selected CpG sites usually leads to suppression of transcription and therefore to a reduced expression of the corresponding gene, especially if these sites are located at gene promoter regions (a region of DNA that initiates transcription of a particular gene).

Not only diseases but also the process of ageing is associated with widespread changes in methylation (mainly hypomethylation) in tissues, that is the proportion of cells bearing unmethylated CpGs increases compared to those with methylated CpGs with ageing (with site-specific occurrences of hypermethylation) (Giuliani et al. 2015). DNA hypomethylation can have an impact on the predisposition to pathological states and disease development, in particular of cancer, atherosclerosis, Alzheimer's disease and psychiatric disorders (Pogribny and Beland 2009). The link between ageing, external exposures and methylation may be provided by oxidative damage, though this is still speculative. Reactive oxygen species (ROS) are unstable molecules containing oxygen and they seem to be responsible for oxidative damage to macromolecules, such as DNA, during the lifetime of an organism and after external exposure to certain agents. The increase in DNA oxidative damage in turn leads not only to DNA base modification, DNA deletion and chromosomal breakage but also to changes in methylation. ROS have been shown to be responsible for epigenetic changes in several cancer models. 8-hydroxyguanine (8-OHdG) and hydroxymethylcytosine are common base lesions due to oxidative stress; the former is widely used as a measure of oxidative damage to DNA (Rang and Boonstra 2014). *In vitro* experiments showed that the presence of 8-OHdG negatively affected methylation of adjacent sites (Rang and Boonstra 2014). Molecular studies demonstrated that oxidative damage to methyl-CpG sequences inhibits the binding of the methyl-CpG binding domain (MBD) of methyl-CpG binding protein 2 (MeCP2) (Valinluck et al. 2004). The role of oxidation is also supported by a protective role of antioxidants against both disease and DNA hypomethylation. The process of ageing is strongly influenced by SES and there is initial evidence to suggest that low SES accelerates epigenetic ageing (see below).

A Conceptual Framework

One of the difficulties related to the elucidation of the role of epigenetic modifications as one of the biological mechanisms through which socioeconomic factors are biologically embedded is related to the fact that epigenetic modifications can both be an intermediate factor between exposure and disease as well as a consequence of the disease itself. Further, in certain instances epigenetic modifications can precede individual SES if they are a consequence of social experiences encountered by ancestors and are inherited transgenerationally. Key in understanding the role of epigenetic changes in leading to diseases or accelerated ageing is a correct reconstruction of the sequence between SES, exposures (e.g. environmental agents), epigenetic changes and health outcomes. A conceptual framework has been proposed by Ladd-Acosta and Fallin (2016), clarifying that epigenetic changes can play at least three distinct roles: they can be a genuine mechanistic mediator within the causal chain linking exposure and disease; they can act as effect modifiers of the causal association, without a direct involvement; and they can provide a biological mechanism to explain how genetic and environmental factors may, in combination, be involved in the disease process (GxE). Irrespective of the role played in causal chains, epigenetic markers can be used in several epidemiological contexts. In addition, epigenetic markers can be useful as both disease and environmental biomarkers, where one is the consequence of a disease process and has clinical utility, while the other is the consequence of exposure to environmental stressors/toxins without being necessarily related to a specific disease.

In addition to the conceptual clarification about the role of epigenetic changes in epidemiological associations, there are several important questions that still need to be addressed in this area of research:

(1) How durable are epigenetic changes, and are they reversible? This is particularly important for establishing the extent to which epigenetic changes that may occur early in life, for example, as a consequence of adverse socioeconomic circumstances starting *in utero*, may be modified by later life exposures.
(2) Is there evidence (direct and/or indirect) of a mediation role between exposure and disease? To date, although a mediating role of epigenetic changes in the association between socioeconomic status and health has been hypothesized theoretically, there is no empirical evidence confirming that. We refer later to examples coming from the study of tobacco

smoking, because these examples are the richest so far, partly because of the strength of association of smoking with disease and because of the low risk of misclassification with this exposure. Low level of misclassification—compared to many if not most environmental and social variables—makes tobacco smoking a relatively easy exposure to investigate and may explain the early successes of tobacco epidemiology.

(3) Are epigenetic changes related to the experience of social adversity transmitted across generations? Although intergenerational effects (such as maternal effects) certainly occur in humans, the extent to which epigenetic modifications can be transmitted to offsprings in the absence of the initial trigger remains highly debated (Heard and Martienssen 2014). Epigenetic inheritance of social (and other) exposures still needs to be demonstrated empirically.

Duration of Methylation Changes

Smoking is an exposure with broad and well-characterized health effects. In a series of studies on tobacco smoking we found that hypomethylation of several genes was associated with smoking in white blood cells (WBC) and also in human lung tissue (Shenker et al. 2013a, b) among healthy subjects. To investigate the dynamics of methylation in smoking, we conducted epigenome-wide analyses in a population sample of 1000 subjects (Guida et al. 2015). When we examined the distribution of methylation changes by time since smoking cessation, we observed that while for many CpGs methylation reverted back to levels of never smokers, for some CpGs hypomethylation was still present after 30–40 years since smoking cessation (Fig. 23.1).

The stability of the latter methylation changes is not compatible with the WBC half-life (death/replication rate), that is, it suggests that hematopoietic stem cells (i.e. stem cells that give rise to all the other blood cells through the process of haematopoiesis) of the bone marrow must be involved.

The gene that was most affected by methylation changes in the majority of studies was AHRR, the repressor of the Ah receptor (AhR), that in turn is involved in the interface between the cell and the external environment. In our study, we found that the list of CpGs with persistent tobacco-induced methylation changes included a number of CpGs associated with Aryl Hydrocarbon Receptor Repressor (AHRR). We also compared methylation levels of 49 AHRR probes in WBC with methylation in lung macrophages from the literature, finding that the effect of smoking was broadly similar in the two tissues despite the large difference in baseline expression levels (very low in WBC, high

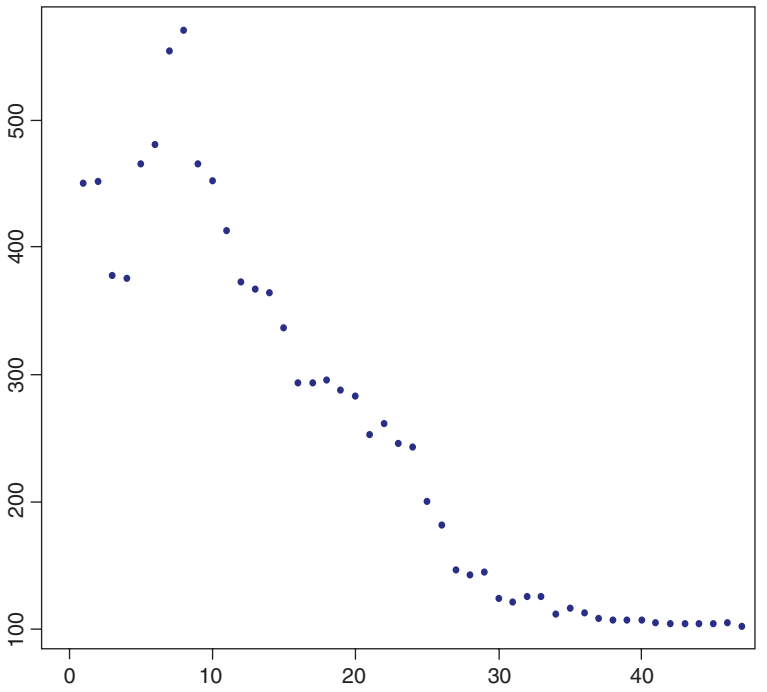

Fig. 23.1 Distribution of the methylation level according to time since smoking cessation (on *horizontal axis*). Stronger hypomethylation (*vertical axis*) is closer to smoking cessation. Increasing methylation levels approach those of never smokers several years after smoking cessation. After many years the number of signals levels-off, suggesting the existence of sites whose methylation status remains altered even more than 35 years after smoking cessation. Courtesy of Florence Guida

in lung macrophages). AHRR expression was upregulated by smoking in both tissues. In a mouse model of smoking, we observed an initial decrease in expression of AHRR at 3 days of exposure and a significant increased expression after longer-term exposure after 28 days (Shenker et al. 2013a).

The Ah Receptor (AhR) is well known for regulating responses to an array of environmental chemicals. A growing body of evidence suggests that the AhR also plays a key role in modulating critical aspects of cell function including cell growth, death and migration (Hahn et al. 2009). *AHRR* is highly conserved in evolution (Hahn et al. 2009). While its most studied function is the mediation of the effects of exogenous chemicals such as dioxin and Polycyclic Aromatic Hydrocarbons (PAHs) on the cell (Basham et al. 2014), *AHRR* is also involved in many other functions including the effects of tobacco smoking in pregnancy. AhR is antagonized by the preventive agent and antioxidant resveratrol (Papoutsis et al. 2015).

This provides a clear example of a gene pathway activated by the exposure and a gene-specific epigenetic hypomethylation that persists, retaining memory of the exposure. This example of persistent methylation memory of past smoking is particularly relevant for the study of social differences in DNA methylation, not only because smoking is probably the strongest socially patterned risk factor, but also because this finding provides evidence for a long-term biological memory of exposures occurred earlier in life, suggesting that epigenetics may well be one of the mechanisms through which lifelong socioeconomic status is biologically embedded.

The Mediation Role of Epigenetic Changes

The meet-in-the-middle approach (MITM) (Vineis et al. 2013) to understand the temporal relationships between agents, mechanistic changes and diseases is based on the construction of a multilayer causal framework. This approach is usually based, within a population study, on a combination of a prospective search for intermediate biomarkers—which are elevated in participants who eventually develop disease—and a retrospective search for links of such biomarkers to past environmental (including social) exposures. The reasoning behind this approach is in three steps. The first step consists in the investigation of the association between exposure and disease. The next addresses the relationship between (biomarkers of) exposure and intermediate omics biomarkers of early effects, "omics markers" generally referring to markers ending in -omics, such as genomics, proteomics or metabolomics (i.e. the high-throughput measurement of all molecules in a compartment). As a third step, the relation between the disease outcome and intermediate omics biomarkers is assessed. The MITM approach stipulates that the causal nature of an association is reinforced if evidence is found for all three steps. An example is the role of methylation of the AHRR gene in linking tobacco smoking and lung cancer in prospective cohorts (Fasanelli et al. 2015). In a series of epigenome-wide studies of DNA from pre-diagnostic blood samples, we observed that the most significant associations with lung cancer risk were for cg05575921 in AHRR and cg03636183 in F2RL3, previously shown to be strongly hypomethylated in smokers. These associations remained significant after adjustment for smoking and were confirmed in statistical mediation analyses suggesting that residual confounding is unlikely to explain the observed associations and that hypomethylation of these CpG sites may mediate the effect of tobacco on lung cancer risk, that is, be a genuine MITM marker (Fasanelli et al. 2015).

If it was relatively easy to assess the potential mediating role of methylation of AHRR in the link between tobacco smoking and lung cancer, things are more complicated with socioeconomic status, not only because socioeconomic factors are a more difficult exposure to measure but also because their impact on specific genes that are related to specific diseases is still unclear. In addition, there are several challenges in proving the existence of epigenetics mediating mechanisms, such as tissue specificity, difficulties in establishing causality, and the fact that there may not be a single pathway but complex multiple interconnected biological pathways (Richmond et al. 2015). Moreover, it is not only possible that socioeconomic factors specifically influence DNA methylation (or other epigenetic markers) of specific genes, but also that they generally impact the epigenome of certain regions, for example, through a hypomethylation effect (see later). In this latter case, proving a mediating effect of epigenetics in the link between SES and disease would be even more complex.

The Evidence on SES and DNA Methylation

As mentioned earlier, in the last few years evidence from human and animal studies has accumulated for a role of epigenetic modifications induced by the experience of social adversity in initiating physiological dysregulations of the immune function (Miller et al. 2009, 2011; Tung et al. 2012; Borghol et al. 2012; McGuinness et al. 2012). A pivotal study in macaques detected altered levels of expression and methylation in inflammatory genes (in particular *NFATC1*, *IL8RB* (CXCR2 *in humans*) and *PTGS2*) in relation to hierarchical status (dominance rank, a proxy for social status) (Tung et al. 2012). Few studies have considered the effect of SES on DNA methylation in humans. In one study, 40 adult males from the 1958 British Birth Cohort Study were selected from SES extremes. Methylation levels for 1252 gene promoters were associated with childhood SES, and 545 promoters with adulthood SES. Functionally, associations with childhood SES appeared in promoters of genes enriched in key cell signalling pathways (Borghol et al. 2012). In another study based on 239 subjects, global DNA hypomethylation was observed in the most socioeconomically deprived subjects. Occupational position demonstrated a similar relationship, with manual workers having 24% lower DNA methylation content than non-manual workers. Additionally, associations were found between global DNA methylation content and biomarkers of cardiovascular disease (CVD) and inflammation, including

fibrinogen and interleukin-6 (IL-6), after adjustment for socioeconomic factors (McGuinness et al. 2012).

In the largest study so far (Stringhini et al. 2015), we examined the association between lifecourse SES and DNA methylation of candidate genes, selected on the basis of their involvement in SES-related inflammation, in the context of a genome-wide methylation study. Participants were 857 healthy individuals sampled from the EPIC-Italy prospective cohort study. Indicators of SES were associated with DNA methylation of genes involved in inflammation. NFATC1, in particular, was consistently found to be less methylated in individuals with low vs. high SES, in a dose-dependent manner. IL1A, GPR132 and genes belonging to the mitogen-activated protein kinase (MAPK) family were also less methylated among individuals with low SES. Changes with upward/downward life trajectories were explored suggesting that the biggest changes in methylation were associated with a stable low SES, and that low SES in childhood followed by improvement in adulthood led to more moderate changes than the reverse. This observation is interesting because it suggests persistence of epigenetic changes in the course of life. Whether early life exposure to social stressors has a more profound effect than later exposures remains to be clarified, if possible with studies where information of epigenetic markers and socioeconomic stressors is collected from early life.

Although it is now accepted that low SES has an impact on the epigenome, it remains to be established whether low SES has a direct impact on DNA methylation, for example, through the stress pathway, or whether epigenetic changes are the product of exposures that are more common among disadvantaged populations. Indeed, in the framework proposed by Ladd-Acosta and Fallin described earlier, epigenetics may be causally involved in a disease pathway by socioeconomic risk; it could act as a modifier of socioeconomic risk or be a biological mechanism to explain how socioeconomic factors may be involved in the disease process. In our study, the association between lifecourse SES and DNA methylation was not modified after accounting for major lifestyle risk factors. However, to our knowledge no study has so far assessed the role of environmental factors, and it is virtually impossible for the majority of studies to account for exposures that occurred before birth. Further, the interplay between low SES, DNA methylation, other epigenetic markers and gene expression still needs to be clarified. Finally, current research in humans has generally only assessed DNA methylation in lymphocyte/blood-derived DNA, and the impact of social adversity on the epigenome of other cell types still needs to be elucidated.

The Epigenetic Clock and Age Acceleration

One aim of current research on SES is to explore whether low socioeconomic status has an impact on biological ageing. Epigenetics can be very useful in measuring age acceleration with the so called "epigenetic clock", a construct developed by Steve Horvath (2013). Using 82 Illumina DNA methylation array data sets involving 51 healthy tissues and cell types, Horvath at UCLA developed a multi-tissue predictor of age which allows one to estimate the DNA methylation (DNAm) age of most tissues and cell types. DNAm age has the following properties: (a) it is close to zero for embryonic and induced pluripotent stem cells; (b) it correlates with the number of cell replications; and (c) it gives rise to a highly heritable measure of *age acceleration* (Horvath 2013).

In two prospective cohorts (one in Italy and one in Australia), we have tested whether age acceleration is affected by SES. We observed that in both cohorts all coefficients linking SES and the methylation clock were negative (with very few exceptions), indicating that with increasing SES there is a tendency of the epigenetic clock to decrease, corresponding to a biological age lower than the chronological age. In other words, higher SES makes people biologically younger and low SES increases "age acceleration" (Fiorito et al. unpublished data).

Policy Implications

As discussed earlier, in our studies of the impact of SES on both DNA methylation and age acceleration, traditional risk factors such as smoking, alcohol, diet, BMI and physical activity did not seem to play an important role. This is in contrast with evidence from classic epidemiological studies where lifestyle-related diseases usually mediate a significant proportion of social differences in health. One possible interpretation of this is that in the case of epigenetics most of the effect of social factors is direct, meaning that epigenetics may be a specific and direct target of social factors. Another interpretation is that other pathways not explored in our study may be involved, such as the stress pathway. Our approach based on epigenetic measurements may contribute to the identification of SES-specific mechanisms that compromise ageing and health. Indeed, epigenetic modifications in specific regions of the genome may provide an indication of exposures that have occurred earlier in life or that are not easy to measure through questionnaires.

Recent research suggests that early life exposures (including those associated with SES) during sensitive periods may be stored in cells through epigenetic modifications that can be sustained for decades (Guida et al. 2015; Stringhini et al. 2015). As we argue elsewhere (Vineis et al. 2017), long-term effects of early life exposures can be explained as being due to impacts on somatic stem cell populations that persist as a form of cellular memory that is akin to immunological memory and includes changes in the patterns of DNA methylation. The implications of long-lasting impact of life experiences and particularly SES on modulation of DNA expression are vast, partly for their policy significance. Most policies targeted at poverty are in fact focused on adults or elderly people, for example, the unemployed or workers with low incomes (such as the EITC programme in the USA), although some of these policies also cover infancy, for example, Conditional Cash Transfer programmes that incentivize schooling and health programmes for children. From our biological studies suggesting that lifecourse socioeconomic position has an impact on biological ageing, we could conclude that earlier interventions are likely to pay greater dividends in the rest of life, compared to interventions in adult/old age. Finally, one of the still-open issues is whether and to what extent epigenetic changes are reversible, and which interventions could reverse them (e.g. improved diet, physical exercise and lower psychosocial stress). From our own research we found that the impact of low SES on methylation is particularly important in those whose socioeconomic position starts low and remains low in later life, compared to those whose position improves in the lifecourse (Stringhini et al. 2015). This suggests some degree of reversibility, as we noticed more prominently for smoking-associated methylation.

Conclusions

Recent evidence suggests that epigenetics may be implicated in the biological embedding of socioeconomic factors. Socioeconomic factors have been shown to have either a specific impact on the DNA methylation of specific genes, particularly in genome regions regulating the immune function, or a general unspecific impact, for example, in the form of a genome-wide hypomethylation. We hypothesize that epigenetics may be a mediating pathway through which socioeconomic factors (or their associated exposures) influence disease risk and ageing. As epigenetic modifications are potentially reversible, the impact of SES on health through epigenetics may be modifiable. However, mediation by epigenetics of the SES effects on health has still to be proven as

causal. Furthermore, research has so far only focused on one epigenetic mechanism, DNA methylation, and only on peripheral white blood cells DNA methylation. Furthermore, whether the specific epigenetic impact of SES is reversible is not firmly established yet. Further research should establish whether these conclusions hold for different epigenetic markers and cell types, and examine the reversibility of epigenetic changes triggered by social factors, preferably using longitudinal data with repeated measurements of social factors and DNA methylation.

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24

An Inter-generational Perspective on Social Inequality in Health and Life Opportunities: The Maternal Capital Model

Jonathan C.K. Wells and Akanksha A. Marphatia

Introduction

The twentieth century saw increasing bifurcation in the academic study of human variability and its association with social behaviour. On the one hand, biological approaches were profoundly influenced by progress in molecular biology, emphasising the genetic basis of phenotypic variability. This led to interest in the heritability of behavioural traits, including the specific mechanisms through which genes may influence behaviour. Partly in opposition to this approach, social scientists developed a very different perspective, focusing on the sensitivity of behaviour to living conditions in order to understand how the organisation of society impacts biological phenotype. When we come to the study of social inequality, whether we are interested in health outcomes, life opportunities or wealth, these two approaches offer starkly different perspectives. In this chapter, we aim to provide a holistic perspective shedding new light on how social inequality can propagate trans-generational effects that are not the product of genotype.

From the late nineteenth century, eugenicists had argued that societies would become healthier if the 'weaker' members (e.g. those judged to have

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mental or physical illness) were 'selected out' by a winnowing process, thus improving the 'genetic stock' of the remaining members (Pearson 1912; Pearson and Lee 1903). Eugenics itself became increasingly unacceptable following the Second World War, though sterilisation programmes to prevent reproduction on several grounds remained widespread in subsequent decades, and are still evident in a few countries (Dikötter 1998). Nonetheless, the notion that a substantial component of behavioural variability has a genetic basis remains supported by twin and family studies, and the new science of genomics has made possible the search for individual alleles associated with variability in behavioural traits such as addiction, aggression, intelligence and attention span. On this basis, those at the top or bottom of social hierarchies might in theory be there in part because of their genotype. Some argue in favour of this, claiming support for the hypothesis that social rank in animals may be shaped by genetic differences in personality traits such as 'anxiety, agonistic behaviour, motivational processes and… behavioural vigor' (van der Kooij and Sandi 2015). With respect to humans, a combination of environmentally and genetically transmitted personality traits has been proposed to underlie inter-generational correlations in economic position (Bowles and Gintis 2001). Others dispute this approach, arguing that the association of genes with personality traits is too weak to contribute substantially to the perpetuation of socio-economic status across generations (Holtzman 2002).

More generally, social scientists typically reject the notion that social gradients in health and life opportunities derive from genotype. Any notion that individuals are 'captives' of their genetic constitution contradicts hundreds of years of philosophical thought emphasising free will, human rights and democratic governance. Rather, if there is social inequality in health and capabilities, it is primarily because humans have generated it through the structure and functioning of their societies. Taking this view to its extreme, we are born equal, and inequality is imposed by social institutions and practices through the lifespan.

The idea that social rank is assigned within the life-course was already apparent in early medieval thought, as for example in this thirteenth-century chess allegory:

> The world resembles a chessboard which is checkered white and black, the colour showing the two conditions of life and death, or praise and blame. The chess-[pieces] are [people] of this world who have a common birth, occupy different stations, and hold different titles in this life, who contend together, and finally have a common fate which levels all ranks. The King often lies under the other pieces in the bag. (Murray 1913)

If humans are characterised by a 'common birth'—equal standing at the start of life—then social policies must be key to resolving inequalities in health and opportunity, and constraining the negative effects of social stratification. Many different social policies duly attempt to promote equality, targeting different aspects of development or living conditions. Such efforts are directed at public health to reduce inequalities in ill-health, at social institutions in order to equalise access to infrastructure and support, and at schooling in order to equalise the distribution of knowledge, skills and training. The overarching aim is to equalise access to society's benefits, to benefit society at large. If such policies were successful, then the penalties of inequality would diminish, and should ultimately be less evident in biological phenotype and capabilities (Wilkinson and Pickett 2009; Marmot 2000, 2005).

Undoubtedly, the effects of social hierarchy extend back to physiology and morphology. The auxologist (scientist of human growth) James Tanner explicitly expressed this through his argument that the physical growth of children could act as an objective mirror of the level of equality characterising any society:

> If you want to measure the classlessness of a society, and you are not interested in rhetoric but in actual conditions and facts, then looking at the growth of children … is perhaps the best way. (Tanner 1990)

More unequal societies show steeper social gradients in children's growth (Eveleth and Tanner 1976), and even in relatively wealthy countries such as the UK, these gradients are reducing very slowly over time (Kuh et al. 1991). Conversely, in more egalitarian Scandinavian countries, the social gradient in height has progressively narrowed across recent decades, though it has not vanished entirely (Meyer and Selmer 1999; Peck and Vagero 1987). The weight of Tanner's proposal lies in the fact that height is no 'neutral' outcome; rather it is a very powerful marker of health status and human capital, as discussed in more detail below. Poor growth in children and short adult stature are associated with poor educational attainment and increased morbidity and mortality (Victora et al. 2008). Secular trends in height among poorer groups are therefore indicative of underlying trends towards societal equality in health and opportunity.

Unfortunately, however, the analogy provided by the chess game is somewhat simplistic. Though the medieval author referred to the chess pieces being 'levelled in status' at the end of the game, no such levelling actually occurs. At the beginning of every new game of chess, pawns remain pawns, and kings remain kings. Each piece has its allotted social role, and however many times

the game is played, indicative of successive generations, high and low ranks propagate themselves over time. It is clear that social hierarchy also persists across generations in human societies, but the underlying reasons have remained poorly understood. Despite much discussion of social mobility, the inter-generational transmission of social rank is powerful, with recent analyses suggesting that the heritability of wealth is even stronger than that in height (Clark and Cummings 2014, 2015).

While rejecting 'genetic determinist' accounts of inequality, however, social scientists have themselves proposed theoretical models of its inter-generational transmission that remain deterministic despite not referencing genes. In the mid-twentieth century, for example, one school of thought considered that poverty might replicate itself through cultural transmission. Based on interviews with families from Mexico and Puerto Rico, Oscar Lewis argued that impoverished communities in Latin American societies were characterised by a unique culture that was transmitted across generations (Lewis 1959, 1966a, b). He considered some elements of this culture to be self-defeating, giving rise to disorganisation, resignation and apathy: these characteristics then contributed to the perpetuation of poverty.

> The culture of poverty is not only an adaptation to a set of objective conditions of the larger society. Once it comes into existence it tends to perpetuate itself from generation to generation because of its effect on the children. By the time slum children are age six or seven they have usually absorbed the basic values and attitudes of their subculture and are not psychologically geared to take full advantage of changing conditions or increased opportunities which may occur in their lifetime. (Lewis 1966b)

Later, responding to criticism of his approach, he downplayed the role of culture in perpetuating poverty (Lewis 1969). Nevertheless, the idea proved attractive to policymakers, particularly in the US, and stimulated heated debate over whether the poor were architects or victims of their misfortune (Valentine 1971). The 'culture of poverty' concept contributed to a 'war on poverty' where social programmes sought 'to correct the social, occupational and physical deficits of people born and raised to a life of poverty' (Gladwin 1967). When these programmes, which made little effort to change structural factors, did not succeed, the intractability of the 'culture of poverty' was duly invoked as explanation. Apparently, its inter-generational cultural basis had simply made poverty 'ineradicable' (Seligman 1968).

We need to move beyond such deterministic approaches, in order to improve understanding of how social inequality can arise through

environmental stresses and yet be persistent in the face of efforts to change it. As evidence from 'genome-wide association' studies accumulates, it is no longer possible to deny that genetic factors contribute to variability in key markers of inequality such as health and educational attainment. Nevertheless, these studies typically explain only a small minority of the variance, and there is abundant and compelling evidence that living conditions, lifestyle and the broader structural environment strongly shape health and schooling outcomes. The primary risk factors for chronic degenerative diseases such as stroke, hypertension, type II diabetes and cardiovascular disease are diet, obesity and physical inactivity level. Social gradients in the risk of these diseases can thus be attributed to underlying inequalities in living conditions and access to health care (Wells 2016). Likewise, the component of education that most powerfully expresses social inequality is whether a child is in school at all. Clearly, none of these outcomes can be said to have a primary genetic basis. But in that case, why do these inequalities persist across generations?

Recently, there has been increasing recognition that the inter-generational persistence of social inequality involves mechanisms of plasticity, rather than genomic transmission. This represents something of a 'middle ground' between gene-based and cultural models of variability, and it forms the focus of this chapter. Biological plasticity refers to the capacity of phenotypes to respond to diverse environmental stimuli and stresses. One approach to this middle ground is to focus on how genes are *expressed* according to prevailing ecological conditions, while further acknowledging that experience in early life generates long-lasting imprints on DNA expression (Petronis 2010; Borghol et al. 2012). This focus on 'epigenetic' mechanisms may however lose sight of more fundamental explanatory approaches, which are our priority here.

The aim of this chapter is to elucidate *how* social inequality perpetuates biological effects across generations, and to identify how we can benefit from this understanding with the aim of reducing social gradients in health, education and life opportunities. We will see that despite the role of plasticity, chronic exposure to adversity can induce a cumulative phenotypic condition that may take several generations fully to reverse. In other words, biological penalties may be hard to resolve and yet this does not mean that they are inevitable. This approach goes beyond previous consideration of how social stresses lead to biological 'embedding' or 'embodying' (Krieger 2001; Hertzman and Boyce 2010), by placing unique emphasis on the mediating role of maternal phenotype.

Developmental Plasticity

At a proximate level, the inter-generational propagation of social inequality arises through developmental plasticity, a mode of response to environmental stimuli that operates during early life and generates long-term impact on phenotypes. Although a number of different environmental stresses are important, nutrition merits particular attention.

In the 1960s, classic studies showed that the effect of under-nutrition on rats depended on the timing of the insult. If the animal was underfed directly after birth, it would never fully resolve the deficit in body size, and would remain small in adult life. If the insult occurred several weeks after birth, however, growth would only slow temporarily, and as soon the nutritional constraint was lifted, rapid weight gain ensued, restoring the animal to its original growth trajectory (McCance 1962). This indicated 'critical periods' in growth, and subsequent work has shown that this concept applies not only to body size, but also to a host of specific tissues and organs, including the brain with implications for behaviour (Smart 1986; Smart 1991; Petry et al. 1997; Davison and Dobbing 1968; Davison and Dobbing 1966).

Amongst the underlying reasons is that the nature of growth changes fundamentally through early development. Early growth comprises an increase in cell number through cell division, known as *hyperplasia*, whereas later growth comprises increases in cell size, known as *hypertrophy* (Bogin 1999). The great majority of hyperplasic growth occurs during foetal life and early infancy. In the rat, for example, organ and tissue growth is entirely due to cell proliferation until approximately 17 days after birth, with minimal change in cell size. Detailed studies on rats have found that if nutritional constraint is imposed during this period, the animals develop lighter organs with fewer cells in them. If the nutritional insult is delayed until later, the animals can regain their organ masses and cell numbers after re-feeding (Winick and Noble 1965, 1966; Enesco and LeBlond 1962).

This means that the mammalian body cannot reverse major structural 'decisions' already locked into physiology through foetal or infant growth patterns. Although not all of these studies have been conducted in humans, for obvious ethical reasons, the profile of human growth is essentially similar to that observed in rats. Consequently, exposure to adversity in early life, whether this derives from nutritional or psychosocial stress, induces long-term changes in physiological structure and function, impacting both the body and the brain. The other side of this coin is that long-term improvements in nutrition during early life potentially represent a key opportunity for improving health and human capital. The importance of such early 'critical windows' of devel-

opment is now recognised through the slogan 'the first thousand days of life', and is researched under the Developmental Origins of Health and Adult Disease (DOHaD) hypothesis (Gluckman et al. 2008).

As soon as we consider in more detail what environmental factors matter during the first 1000 days, however, it is impossible to ignore the importance of maternal phenotype (Wells 2010). Critical windows of physiological sensitivity occur primarily during the periods of gestation and lactation, during which all stimuli experienced by the developing organism are transduced by maternal phenotype. Although not all women breast-feed in contemporary populations, we have argued from an evolutionary perspective that windows of plasticity evolved in concert with maternal nutritional care, in other words gestation and lactation (Wells 2003, 2014). In turn, many components of maternal phenotype that impact offspring development are powerfully shaped by maternal social rank. On this basis, we can see something profound: an inter-generational process in which (a) maternal social rank influences development of the offspring, and (b) developmental experience of the offspring shapes its social rank in later life (Fig. 24.1).

To some extent, the influence of maternal metabolism on the developmental trajectory of the offspring represents a form of protection against external ecological insults (Wells 2003, 2016). For example, even when the

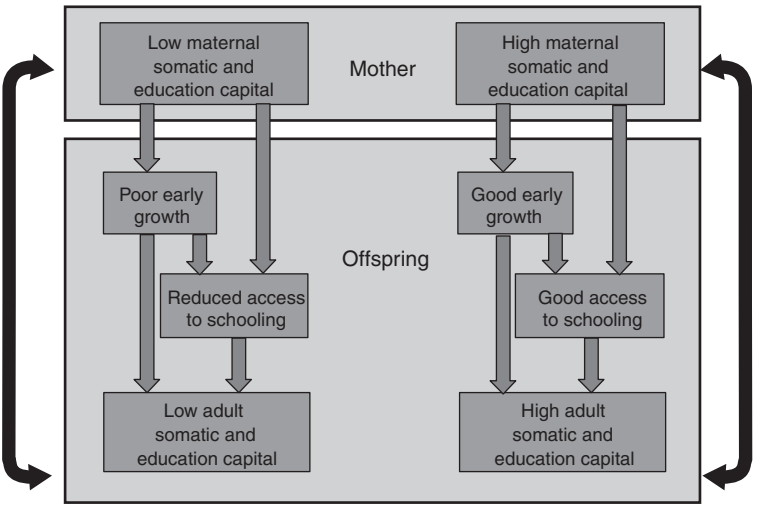

Fig. 24.1 Schematic diagram showing an inter-generational cycle between the level of maternal capital and the acquisition of somatic and educational capital in the offspring. Those receiving poor nutrition and less education in early life embody these traits in adulthood and transmit them to the next generation. Those receiving high investment can transfer more to their own offspring

mother herself is exposed to famine and experiences drastic reductions in energy supply, growth of the foetus is only relatively mildly affected (Stein et al. 2004). Maternal physiology functions to buffer the immature foetus from adversity, and this buffering is of particular value because it shields the period of hyperplasic growth, when the organs and tissues are most sensitive to disruptive ecological stresses (Wells 2003, 2014). However, the very protection that the mother can offer her offspring can become its own constraint when the mother herself has experienced chronic exposure to adversity. We have referred to this as a 'metabolic ghetto' (Wells 2010, 2016), where the developing foetus cannot escape exposure to the mother's long-term experience of deprivation. As in a castle that is besieged using its own defensive walls, layers of maternal physiological protection can lock in their own stresses so that they manifest to the foetus or infant as a developmental constraint (Wells 2016).

Maternal Effects

Small maternal size, poor nutritional status at the time of conception, poor dietary intake during pregnancy and exposure to infectious diseases have all been shown to impact development of the foetus and infant, in different ways (Ozaltin et al. 2010; Dominguez-Salas et al. 2012; Stein et al. 2004; Guyatt and Snow 2004; Ticconi et al. 2003). Maternal psychosocial stress can also generate adverse effects on foetal growth, through the medium of maternal levels of cortisol, a physiological marker of stress (Entringer et al. 2008a, b, 2009, 2011).

In turn, variability in foetal and infant patterns is now well established to represent a key component of variability risk of chronic diseases in later life (Leon et al. 1996; Hales et al. 1991; Barker et al. 1989; Li et al. 2015). These diseases now comprise the primary burden of ill-health in high-income countries, and despite a persistent burden of infectious disease in low- and middle-income countries, chronic diseases are also the main source of premature mortality and morbidity in these countries too (Lozano et al. 2012). In England during 2009–2011, for example, living in an area with the highest deprivation (measured in deciles) was associated with seven and nine years shorter life expectancy for women and men, respectively, compared with those in the least-deprived areas (Office for National Statistics 2014). Equivalent differences in healthy life expectancy were twice as large. Elevated burdens of long-term ill-health and premature mortality are thus key aspects of social inequality.

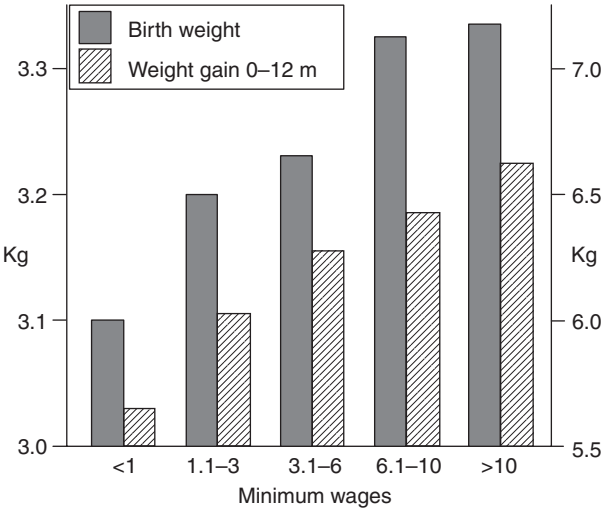

Fig. 24.2 Association of birth weight and infant weight gain with family income, assessed in 'minimum wages', in the 1982 Birth Cohort from Pelotas, Brazil. Data from Victora et al. (1987)

Early growth variability feeds directly into such health inequalities. In most populations, birth weight and infant weight gain show profound social gradients, such as those illustrated in Fig. 24.2 for the population of Pelotas in southern Brazil (Victora et al. 1987). Even where low birth weight is followed by compensatory catch-up growth (Ong et al. 2000), the recovery of deficits in size occurs at the expense of long-term health (Metcalfe and Monaghan 2001). Catch-up growth is an independent risk factor for adult chronic disease, and exacerbates the effects of low birth weight (Eriksson et al. 1999).

Such inter-generational associations help understand social gradients in adult chronic disease risk, but to fully explain patterns of variability we also need to take into account the effect of social inequality in later life. Just as the risk of low birth weight is greater in those of low social rank, in high-income populations the risk of obesity is also greater in those of lower socio-economic position (Giskes et al. 2008; Sobal and Stunkard 1989; McLaren 2007). This means that the poor, in affluent societies, have two independent risk factors for chronic disease: low birth weight imposed by their mother's deprivation, and an unhealthy lifestyle and physiology imposed by their own experience of deprivation in later life. However, in low- and middle-income countries, risk of obesity is greater in those of higher socio-economic position (Subramanian et al. 2011; Neuman et al. 2011). In these populations, therefore, chronic diseases remain clustered amongst more wealthy groups, whilst those of low

social economic position are most vulnerable to under-nutrition and infectious disease.

We have termed our model of developmental plasticity the 'maternal capital' hypothesis (Wells 2010, 2012a) to emphasise that the offspring initially calibrates its developmental trajectory to its allocation of maternal investment. Building on Kaplan's concept of 'embodied capital' (Kaplan et al. 2003), maternal capital is defined as 'any aspect of maternal phenotype, whether somatic or behavioural, which enables differential investment in offspring' (Wells 2010). As we have seen above, those receiving less 'capital transfer' in early life through the medium of maternal nutrition are more susceptible to ill-health, though the relevant diseases depend on the level of economic development.

When maternal physiology provides a stable metabolic signal for the offspring, that signal carries the imprint of maternal rank (Wells 2010). The offspring is exposed to the sum total of maternal capital, which has accumulated through the mother's life-course. That such signals elicit a response by the offspring is shown by correlations between many stable components of maternal phenotype and those of the offspring. For example, maternal birth weight, adult height and leg length, and body composition at the time of conception all show correlations with the offspring's birth weight (Wells 2016).

Energy stores are undoubtedly important, but there are many other components of maternal capital, including nutrients (vitamins, minerals, macronutrients), social capital, and indices of completed growth such as stature or pelvic dimensions. Beyond nutritional resources themselves, another crucial component of maternal capital comprises her capacity for metabolic homeostasis. In contemporary populations, mothers with hypertension or gestational diabetes expose their offspring to perturbed metabolism (Wells 2007). Such effects can continue in infancy, when the breast milk of diabetic mothers has high sugar levels (Plagemann et al. 2002). Just like maternal starvation, such metabolic dysfunction can be considered a depletion of maternal capital, and it can impose long-term metabolic penalties on the offspring. Now the 'walls' lock in too much fuel, rather than too little, and the offspring again experiences metabolic penalties (Wells 2016).

Each offspring continues to process ecological cues throughout development, resulting in a chain of 'decision nodes' that collectively constitute its life history strategy (Wells 2012b). These 'decisions' are generally not expected to involve conscious thought, and instead are generated through physiological or subconscious mechanisms shaped by natural selection. Even where behaviours (e.g. sexual activity) do involve conscious decision-making, it is possible

that such decisions may simply provide post-hoc rationalisation—more a consequence of behaviour than cause. For example, similar behaviours in other species would not be assumed to involve any conscious deliberation.

Each of these decisions reflects the expression of its genotype under the influence of the quality of the environment. The earliest decisions occur through calibration to maternal phenotype (Wells 2010), whereas later decisions are elicited directly by the environment. For example, studies of migrants from Bangladesh to the UK show that reproductive physiology retains plasticity throughout childhood and adolescence (Nunez-De La Mora et al. 2007, 2008). Since later decisions are shaped by earlier ones, however, the whole chain maintains the initial maternal imprint (Wells 2010).

Figure 24.3 shows how offspring tailor their life history strategy to the magnitude of investment they receive, using data from a study of young South Asian women living in the UK (Wells et al. 2016). A lower level of nutritional

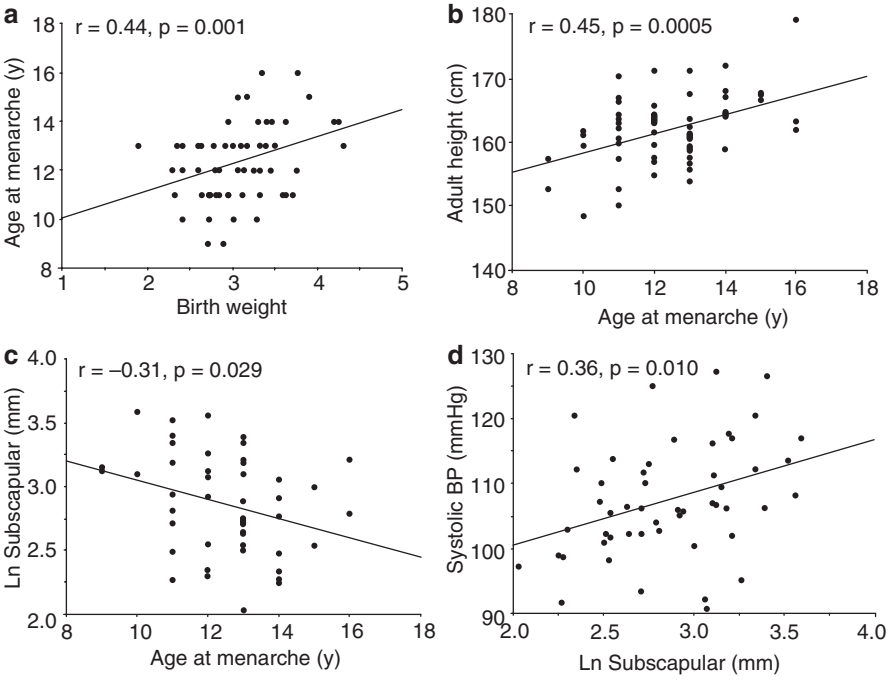

Fig. 24.3 Associations of maternal investment (proxied by birth size) with maturation rate and adult phenotype in young healthy South Asian women living in the UK. **(a)** Birth weight is inversely associated with age at menarche. **(b)** Earlier menarche is associated with lower adult stature. **(c)** Earlier menarche is associated with higher adult subscapular skinfold. **(d)** Subscapular skinfold is positively associated with systolic blood pressure. Reproduced with permission from Wells et al. (2016)

investment, indicated by lower birth weight, was associated with an accelerated pattern of development, as indicated by earlier puberty. However, this accelerated maturation reduced the total period of growth, and was associated with shorter adult stature and with elevated adiposity. Finally, high levels of body fat were associated with higher blood pressure. This study is important because it shows that daughters who received less investment from their mothers adopted a 'fast life history', which prioritised the acquisition of energy stores for reproduction at the cost of investment in somatic growth and health. Other studies show that adult women may propagate these traits to the next generation, perpetuating the cycle (Ong et al. 2007).

Maternal Capital and Education Outcomes

It is clear therefore that variability in growth patterns in early life contributes to health inequalities in later life, and recently the same approach has been used to explore variability in education outcomes. As with chronic disease risk, educational attainment has been associated with patterns of growth during the period before children are of school-going age.

In a large cohort study of 8362 children in Brazil, Guatemala, India, the Philippines and South Africa, for example, lower birth weight, slower linear growth and lower relative weight at 2 years were all independently associated with an increased risk of not completing secondary school (Martorell et al. 2010; Adair et al. 2013). Similar findings have been reported from other studies in Ghana and Tanzania (Beasley et al. 2000; Fentiman et al. 2001; The Partnership for Child Development 1999). Although faster growth at later points in the life-course may also benefit cognitive performance (Horta et al. 2009; Cheung 2006), there are substantial 'trade-offs', for faster weight gain from mid-childhood onwards is associated with increased risk of chronic diseases (Victora et al. 2008). In other words, growth is most beneficial for *both* health and education if it occurs during the early critical windows that are under the influence of maternal metabolism. Breast-feeding is an important part of this process, because in addition to providing optimal nutrition, including many nutrients critical for brain development (Isaacs et al. 2010; Anderson et al. 1999), it also reduces the likelihood of acquiring infectious diseases, which can stunt growth and cognitive development (Walker 2010).

While these observational studies link poor growth in early life with lower educational attainment, they do not provide robust evidence of the causal linkage. One exception however is a longitudinal study in Guatemala, which found that in comparison to children who received a different nutritional supplement, those who had received a high protein nutritional intervention

between birth and 36 months completed 1.2 more years of schooling and performed better on cognitive tests (Maluccio et al. 2009).

Collectively, these studies provide insights into the mechanisms through which early life nutrition is associated with educational outcomes. Growth retardation, indicating a lack of nutrients at the cellular level in early life, has been associated with poorer brain and neurological development (Martorell et al. 2010). For example, a study of Scottish school children broadly showed that the shorter the duration of foetal development, indicated by gestational age at birth, the higher their risk of cognitive impairment (MacKay et al. 2010). Whether and how this cognitive 'deficit' is then directly associated with educational outcomes require more research, including data on school-based factors. However, some studies imply such associations. In their first year of school, under-nourished children from Mexico performed poorly on cognitive tests and participated less in the classroom with 38% repeating a grade (Chavez et al. 2000). A longitudinal study in Guatemala also suggested that psycho-social factors such as lower levels of stimulation and social interaction may delay the age at school entry for stunted children, thereby increasing their risk of repeating or dropping out of school (Brown and Pollitt 1996).

Although growth in early life is associated with educational success, a key aspect missing from these studies is the influence of maternal phenotype during the period of children's early growth. Further research is also required on how maternal nutritional status directly predicts children's educational outcomes (Walker et al. 2011). An analysis of a longitudinal biomedical study of 838 children since birth in Dhanusha, Nepal, adopted a more comprehensive approach, testing the independent associations of different components of maternal somatic and educational capital and family economic capital with children's educational attainment (Marphatia et al. 2016b). The results showed that children aged 8.5 years were at a higher risk of completing fewer years of schooling if their mothers had lower levels of capital defined by lack of education and poor nutritional status, especially anaemia, and that these associations held after adjusting for broader components of family capital.

Figure 24.4 shows independent associations of different components of maternal phenotype with poor educational attainment at age 8.5 years, expressed as odds ratios for categories of maternal capital. In this model, the association of maternal low haemoglobin (anaemia) with children's educational attainment was mediated by poor rates of growth between birth and 2 years of age, adjusting for size at birth. Overall, maternal lack of education, a proxy of the social capital offered to children throughout their life-course, had the strongest magnitude of association, followed by different maternal biological markers for the two sexes, high land ownership for boys and rural location for girls.

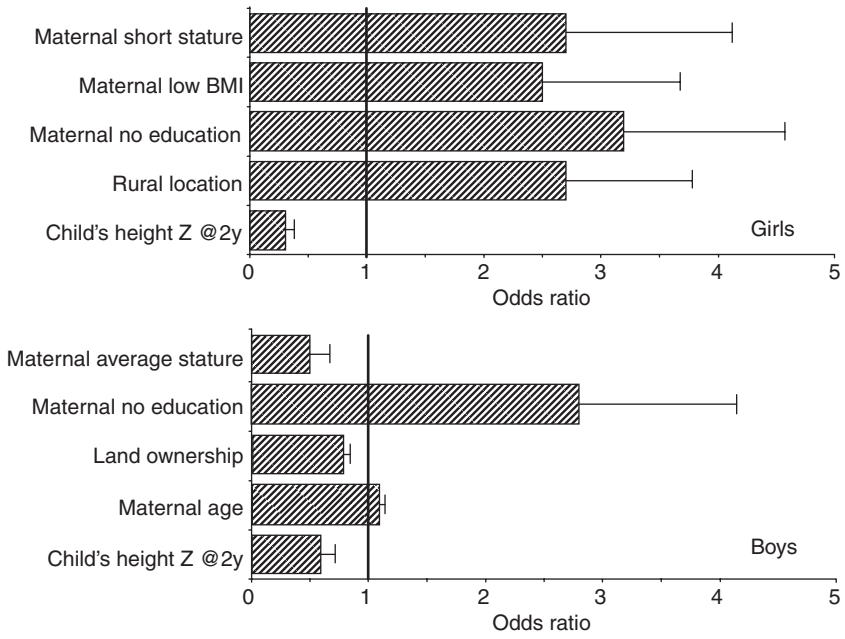

Fig. 24.4 Odds ratios for the risk of Nepali children completing less than 3 years education by ~8.5 years, for a variety of parental or family risk factors, or conditional growth of the children themselves. Based on data of Marphatia et al. (2016b)

These results suggest that, as with health inequalities, educational inequalities are shaped early in the life-course, and are strongly associated with maternal phenotype. Given such trans-generational perpetuation of disadvantage (Schell 1997), school-based efforts may arrive too late to support their participation in education.

Crucially, low birth weight is often followed by some form of catch-up growth, which as we saw above accelerates the pace of maturation. In turn, this may trigger a social response—identifying fast-maturing girls as ready for early marriage. This is important because studies find that adolescent girls who marry at an earlier age are more likely to drop out of school, experience early and repeated pregnancies and have children of low birth weight, who are under-nourished and also more likely to complete less schooling (Fall et al. 2015; Godha et al. 2013; Santhya 2011).

Here, therefore, we see the full trans-generational cycle of disadvantage, in which the pattern of development experience in one generation shapes that in the next generation. None of these associations need derive from genotype, indicating that interventions could aim to improve outcomes if conducted

over lengthy periods. Greater focus on building the educational and nutritional capital of girls and women is likely to be mutually beneficial for both mothers and children, as discussed further below.

Societal-Level Gender Inequality and Children's Outcomes

Although many components of maternal capital reflect the individual circumstances of particular women, we can also consider how the organisation of society influences maternal capital at the population level. One way in which this can be done is by assessing women's status in society relative to men, for example, using the Gender Inequality Index (GII), a measure of women's reproductive health and their participation in education, the labour market and political representation relative to men. Recent ecological studies have used this index to investigate associations between societal gender inequality and child malnutrition and survival.

For example, across 138 countries societal-level gender inequality was associated with neonatal, infant and childhood mortality rates (Brinda et al. 2015). Another ecological analysis investigated whether two countries with similar national wealth, but with different status of women, have different levels of low birth weight, child malnutrition and survival across 96 countries (Marphatia et al. 2016a). Figure 24.5 shows a linear, dose-response between these factors with an increase in gender inequality associated with increases in adverse child outcomes.

These associations were still present even after controlling for markers of economic growth, measured by gross domestic product (GDP). This suggests that the current paradigm of addressing child malnutrition and mortality through promoting GDP is unlikely to be successful unless and until women's status in society also improves (Marphatia et al. 2016a).

Conversely, simulations based on statistical models of these data suggest that reducing gender inequality would have major reductions in child survival and malnutrition globally. These analyses suggest that efforts to promote women's ability to participate, at an equal level, with men in society are likely to have substantial benefits for children's health and survival, especially in low- and middle-income countries. The value of such composite indices is that they identify specific capabilities and opportunities of women that interventions could target in order to improve both their well-being and the health of their children.

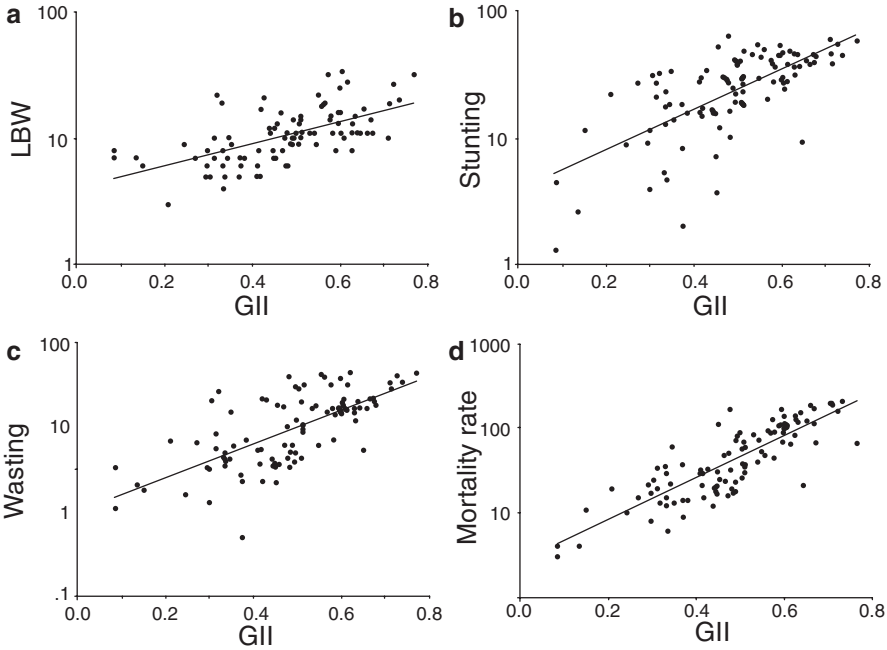

Fig. 24.5 Associations of the Gender Inequality Index (GII) and the prevalence of (**a**) low birth weight (LBW), (**b**) stunting, (**c**) wasting and (**d**) child mortality rate in 96 countries (2 missing data points for LBW). Reproduced with permission from Marphatia et al. (2016a)

Maternal Capital as an Opportunity for Intervention

If maternal capital generates such profound long-term impacts on the developmental trajectory of offspring, then it appears as a key opportunity for interventions intended to break the 'cycle of disadvantage'. Of particular interest, we have demonstrated that each of health and education is associated with growth patterns in early life, which in turn demonstrates the importance of both maternal nutrition and educational attainment.

Paradoxically, efforts to promote maternal nutrition during pregnancy and infancy have had mixed success. A number of supplementary nutrition programmes targeted at pregnant women achieved relatively modest increments in birth weight, averaging around 40 g, although larger increments occurred in particularly under-nourished groups (Kramer 1993). However, one challenge is that most of these programmes were only initiated midway through pregnancy, and thus missed the period during which the placenta develops,

along with the earliest periods of organogenesis. A unique study that supplemented women across two pregnancies achieved a substantially greater increment in birth weight of 150 g (Villar and Rivera 1988). Studies that have supplemented during infancy have also tended to have greater benefits (Conlisk et al. 2004; Kinra et al. 2008), though the challenge remains that the ideal route is to supplement the mother so that the infant benefits through breast-feeding.

These interventions also do not address the critical importance of simultaneously increasing maternal education, which studies find improves both maternal and child well-being. Incorporating training on literacy through women's groups often used in non-formal education programmes may thus enhance the success of these interventions. On their own, these initiatives also have their limitations, the trade-offs of which may be better understood by adopting a holistic understanding of both biological and social factors as highlighted in this chapter (Marphatia and Moussie 2013).

Using maternal phenotype as a medium through which to operationalise interventions targeting the offspring might be considered unethical, on account of treating the mother as a 'passive vehicle' without taking her own needs or identity into account. We believe this perspective is unhelpful, because as we have discussed above, the health of the offspring is fundamentally associated with the health of the mother. In the vast majority of situations, the interventions that are crucial for transmitting benefits to the next generation (e.g. promoting healthy maternal metabolism) will also benefit women in their own right. Our data on the harmful consequences of societal gender inequality highlights the importance of improving the circumstances of women.

How successful might this approach be? Returning to our analogy of the mediaeval chess game, how many games (generations) would it take for public health interventions to dissolve the inequalities in health that currently characterise pawn, queen and king? The available evidence suggests that each generation can accumulate phenotypic improvements, but that a number of generations are required in order to shift phenotype substantially (Wells 2012a). Whilst this may make progress seem hard to achieve, we should also look at the long-term benefits: when mothers have accumulated substantial capital, they are resistant to short-term stresses and can buffer their offspring during early critical windows. This is the 'natural advantage' already enjoyed by those at the top of hierarchies, and it could be shared across the population if societies organized themselves to achieve this aim, as the evidence from gender-equal and egalitarian societies attests.

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25

Quantifying Social Influences Throughout the Life Course: Action, Structure and 'Omics'

Michael P. Kelly and Rachel S. Kelly

Introduction

In recent years, developments in high-throughput sequencing technologies have demonstrated possible mechanisms whereby factors in the social and material environment may have a direct effect on biological processes. However, social science has not kept pace with these developments and has made little progress in examining the society-biology interface (Kelly et al. 2014; Meloni 2014; Meloni 2015a, b). Engaging with the new biology now represents an urgent priority for the social sciences. The human genome project was anticipated to lead to a much better understanding of the *biological* origins of disease; this promise has not really been fulfilled. But, paradoxically, developments arising from the sequencing of the human genome project and the subsequent developments in other 'omics' technologies have served to underscore the importance of the *social* (Kyrtopoulos 2013). And it is on the interaction with the *social* that the next generation of scientific biological developments will need to focus. It is imperative that the social sciences engage in this fully.

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Our argument draws upon a range of social scientific ideas, particularly structuration theory (Giddens 1979, 1982, 1984), social phenomenology (Schutz 1967, 1970) and critical realism (Bhaskar 2008, 21–24) to show how the human interactome, that is, the epigenetic marks, genes, RNA molecules, proteins and metabolites in a biological system, can be read as a dynamic timeline of a life that encompasses both its underlying biology and its interactions with its external social and biological worlds. We contend that the social and the biological realms should not be construed as separate analytic levels (driven by conventional disciplinary paradigms) (Rose 2005, 84–95). Rather, we conceptualise an empirical unity in which individual and population health outcomes, positive and negative, are conceived as emergent properties of the interaction between human consciousness, agency, social structures *and* dynamic biological processes.

Human life is both social and biological. Human consciousness and sociability interact with the biology of the species and with the biology of the external environment. The interaction is a dynamic process, not a deterministic one. Therefore, biological and social processes should be viewed as operating simultaneously and constantly impressing themselves upon each other with the emergent consequences being implicated in further continuing interactions (Rose 2005, 140–142).

Illness is ubiquitous to the human condition. We conceptualise morbidity as a set of inter-related processes which are the result of the unique interaction between an individual's biology and the external world across their life course. Humans exhibit wide individual biological variation, by virtue of genetics, nutritional status and other biological factors. One individual's response to an exogenous factor or curative intervention will vary from another's. At an individual level, a person's unique life course up to the present, and what we conceptualise in this chapter as their life world, interacts with their unique biology to produce their current state of health. At the social or population level, these unique interactions are patterned as a consequence of the interplay of the social practices in which people are engaged, producing health differences between social groups and between societies as a whole. At population level the patterning of diseases linked to social factors is well known (Commission on the Social Determinants of Health 2008; Marmot 2010; Marmot and Wilkinson 2006). The clustering of morbidities in individuals and in populations, along with the coalescence of the social behaviours and social circumstances that lead to them, reflects the overlapping nature of life worlds which humans inhabit (Buck and Frosini 2012). However, it is important not to conceptualise so-called lifestyle behaviours *or* the wider social factors like pov-

erty and disadvantage as separate from the biology and the individual level as separate from the population level. It is important also not to simply see these links between social factors and biology as mere statistical associations; they causally interact in ways that generate individual morbidity and the patterning of population health. Out of these interactions, properties such as the patterning of diseases linked to disadvantage emerge.

The stressors which precede disease, whether microbiological, biological, social-psychological, environmental or genetic, are not simple linear determinants of pathology. The stressors are mediated through a vast array of human behaviours as well as through biology—it is an interactive process (Kelly et al. 2014). These interactions in turn produce the interactome, which we define as the complete complement of 'omic' profiles (see below). There is a large literature describing some of these *social* processes but it does not for the most part engage with biology. It focuses on lifestyle behaviours (Marteau et al. 2015) or on the social, economic, material and political determinants of health (Kelly and Doohan 2012). Both approaches tend to separate behavioural and social circumstances from the biological processes occurring in the human body. In contrast, a properly sociological approach emphasises the need for contextual and temporal analyses emphasising the importance of the life course and the social factors which shape that life course in understanding health and disease and the interactome.

Social Life: Human Agency, Social Structures and Social Practices as Mechanisms Which Interact with the Human Interactome

In order to demonstrate the processes involved in the interactions between the social and the biological, we begin by describing three elements of social life—human agency or volition, social structures and social practices. As noted above, our account is based specifically upon structuration theory, social phenomenology and critical realism rather than other strands of contemporary theorising.

Social life is repetitive and recursive as well as continually changing and evolving for individuals and for collections of people. Both the change and the repetition are the products of the dynamic interaction between human agency and volition on the one hand and social structures on the other. Social practices are emergent dynamic properties of the interaction between human agency and social structure.

Human Agency

Volition or agency is expressed in the language idiom, 'I do, I can do, I will do, I did do, or I have done… X, Y or Z'. The 'I' of the language is a constituent of our sense of self. Our sense of self is of being a thinking subject who can reflect, ruminate and act on the external world, that can interpret external and internal information and stimuli, that conceives of itself as a single subject separate from the external world and from others and that is unified through time and situated in place and time as an 'I'. That self is the centre of its own life world and is in relationships with objects and people that are physically proximate or distant in space and exist in the present, past and future. This recognisably modern and western formulation of self was developed originally by Immanuel Kant in the *Critique of Pure Reason*, was brought into sociology by George Herbert Mead and is linked to modern social theory through Alfred Schutz's phenomenological writings (Kant 1781; Mead 1934, 1936; Schutz 1967, 1970).

The thinking 'I' responds in two ways to the external and internal environment—automatically and reflectively (Strack and Deutsch 2004; Kahneman 2011, 21–28). Automatic activity requires little cognitive engagement and insofar as thinking goes on at all, the world is just assumed or taken for granted (Schutz 1970, 72–80, 111–112, 236–237). Automatic responses are either completely unthinking or habitual (Marteau et al. 2012) or are based on simple heuristics (short cuts in thinking) (Kahneman 2011, 71–88, 118). Reflective activity on the other hand arises because the self also engages in cognitive reasoning (Marteau et al. 2011). Human actors are conscious, thinking and calculating beings who process information, who have stores of background knowledge and information derived from their own direct and vicarious experiences (Schutz 1967, 81–87; Mead 1934, 69–98, 136–186). They are able to make assessments of the immediate and past and future environments. They also imagine and anticipate the consequences of their actions on those immediately around them and into the longer run (Schutz 1967, 99–128; Blumer 1962, 180–184).

The environment to which they are responding has four elements—biological, physical, social and emotional/subjective (Kelly 2016). The environment is ubiquitously biological consisting of an external and internal cocktail of microbes, plants, insects, fish, animals and people. There is always a physical context for human activity, which provides the cues and sets the boundaries to human activity within this environment (Hollands et al. 2013). In addition, the environment is social. It consists of other humans with whom an individual is in social relationships. These humans are those in the immediate environment but also include imagined others who although not physi-

cally present may be very important—friends, family, colleagues, enemies and neighbours, for example (Schutz 1967, 141–150). Subjective emotions and feelings are the fourth element in the environment. People respond to their own emotions and feelings, as well as to physical things and other people. Emotions can be described as real cognitions (Damasio 1994, xvi) and people respond to those emotions just as they respond to objects and people. This mix of automatic and reflective activity, of taken-for-granted notions about the way the world is, is what all human life consists of, and out of these, billions and billions of actions social patterns emerge.

The fact of individual volition and agency and that abiding sense of self which is at the centre of people's life worlds easily conceal the fact that the individual is in an interactive relationship with the broader social patterns or social structures. It can also create the sense that the self is separate from its own biology and the broader biological reality in which it lives. Paradoxically, there are very strong social structural factors at work which reinforce the idea of a highly individuated self which is quite separate from the rest of the world. The notion that westerners have of being autonomous individuals is in part driven by their language (Harre 1987, 23) but also by cultural and legal systems. There are civil/legal artefacts, such as birth certificates, passports and driving licence numbers, which construct us as a unique individual quite separate both legally and personally from others. At a gross level we appear superficially to be bodily distinct from each other, empirically, however, we are intrinsically biologically and socially connected with others and with our environment.

The sense of the individuated self, the 'I', with duties and responsibilities, with a personal history and autobiography, related to, but separate from others, characterises contemporary western life. This is true even where people deliberately subvert the legal-bureaucratic identifiers and have, for example, false passports. The false one still constructs them as a unique individual. So dominant is this idea that we are unique individuals, it is quite difficult to imagine things otherwise. It is in a way so obvious that we are individuals what is there to explore further? In some disciplines, a reductionist focus on the individual constitutes a core epistemic assumption, economics and clinical medicine being typical in this regard. In contrast, economic and clinical individualism notwithstanding, the disciplines of biology and sociology operate with a different epistemic perspective which for both disciplines is *relational* (but see Rose 2005 who notes that much biology is also highly reductionist). Both see interaction between within discipline phenomena as central, one for biological processes and the other for social processes; but neither do much to consider the relationship, between the biological and social!

So to return to human volition and the idea of self, humans have varying degrees of ability by their own agency to control their biological, physical, social and emotional environments—their own life worlds. People are not free volitional agents; rather their actions are constrained by the social structures which make up human life and which are as ubiquitous and inescapable as biology or the air they breathe. The very old, children, the ill and the infirm have fewer resources and skills with which to control their life worlds and sometimes have much less capacity for self-control and self-direction than typical adult members of society do. But even within adult society there are marked variations in the possibilities that individuals and groups have to control their own life worlds; gender, social class, tribe, caste, nation and status—all impact on the ability to control resources, self and others. This ability to control personal life worlds is crucial to the degree of positive and negative exposures experienced across the life course. No human is ever completely free to do exactly as they please, but there are marked differences in people's ability to define and do things the way that they want to—access to power. This has considerable implications for the life world and hence the interactome.

Social Structure

The billions of individual actions in physical-biological, social and emotional environments produce patterning of human behaviour. These patterns are social structures. Social structures are the product of individual actions, but they also then constrain the possibilities of individual volition. Those constraints have further effects on behaviour and in turn these impact on the structures and so on in a continuous interactive, complex and continuous cycle. Structures are the patterning of interaction across time (Giddens 1979, 62) and the rules and resources embedded in broader social systems (Giddens 1982, 34–35).

The social structure is tangible; we become sensitised to structures as we mature and become aware of peer pressure, norms, values and folkways within sub-groups and sub-cultures in our personal life worlds. These life worlds are not isolated; they are nested within and overlap with other life worlds. Shared experiences in social structures produce patterning of health in social groups as a consequence of those shared experiences producing similar patterns at the biological level in the interactome. Social structures are not visible, unlike the physical architecture of a place or the biological ecology of an environment, but their constraining and boundary limiting qualities are just as real. And just as the individual and the social structure are in an interactive cycle, the

physical, biological and emotional are also part of the mix, inextricably interconnected in continuous and recurring patterns which play out, amongst other things, as the human interactome.

These social and biological realities are undoubtedly complex and have very numerous possible variations. But certain patterns do recur and some of the recurring phenomena drive population health inequalities. Social life has a highly repetitive quality both for individuals and for the social collectivities which give social systems their stability. The social structures with which the volitional self then interacts are the repetitive patterning of social life produced by the aggregation of individual human conduct (Giddens 1979, 4–5). But this is not just social; individual human activity interacts with the recursive patterning of social life and with the biological systems within individuals and externally in the wider ecology. Giddens has described duality of structure, by which he means that the structural properties of social systems are the medium *and* the outcome of the things people do (Giddens 1979, 69, 1982, 36–37). We want to add to this duality biological process as both a medium and an outcome.

Social Practices

Social actors are in relationships with each other. These relationships involve mutual interdependence. The mutual interdependencies consist of social practices which repeat across time and place (Giddens 1979, 2–5, 65–66). The concept of social practice helps move us away from thinking about individual behaviour and towards the idea of networks of people doing things in concert with others and of that network existing across space and time with different people involved as time goes by (Giddens 1982, 34–37). For example, the population of smokers today is different to the population of smokers in 1940, but the elements of smoking—the practice—remain (Blue et al. 2016). And smoking will continue as a practice in subsequent generations.

To understand the dynamic nature of practices, imagine that individual human beings, as active agents, think, act, move, communicate and engage in many different practices in the course of a single day and many thousands in the course of their lifetimes. These practices have a quality that is separate from the individuals that engage in them as they transit across time and place. The practices, the patterns of human conduct, are real and themselves constitute the social context in which individual activity occurs. So as practices set boundaries to human conduct, they constrain and limit behavioural possibilities; they also drive the interactome. Although the interactome is

physically located in the individual and is subject to the random and stochastic nature of biology, it is also the product of dynamic social processes. The *social* patterning of the interactome is not random, rather it is systematically structured by social practices.

The recursive nature of social life therefore has profound biological implications. The recursive nature of practices as diverse as eating, working, child rearing, travelling, loving and drinking alcohol, all in various ways write themselves on the 'ome'. So, social reality has a biological reality too. Social practices are the medium of our existence. Our personal sense of who and what we are—our self and the identities that others bestow upon us—are derived from the practices in which we engage. This is true of the most transient and fleeting engagements of our moment-to-moment existence, to the broader and more embedded things we do on a regular and continuous basis across time and space, but all of them have biological consequences which of course in turn impact on social life. Life worlds are made up of intersecting and networked social practices. The degree to which people are able to exert control over these practices is critical in the degree to which they are vulnerable to the intrinsic stressors of daily life (Kelly and Doohan 2012, 93–104). The life world affects all biological processes as context, as boundary and as an intrinsic part of the human interaction.

The Human Interactome and the Timeline of Life

The advent of the 'omics' era, made possible by the recent developments in high-throughput sequencing technologies (Ward and White 2002), provides a novel means of exploring how the social exposures of the life course have profound biological effects on the life world (Table 25.1). We are now able to measure almost the entire complement of genes, epigenetic marks, RNA transcripts, proteins and metabolites that comprise a biological system (Table 25.1) (Bonassi et al. 2013). Dunn and colleagues neatly describe the biological parameters of this 'new biology': 'The genome defines what may happen; the metabolome defines what has happened' (Dunn et al. 2011). To paraphrase Dunn, this can be expanded to say the epigenome, transcriptome and proteome define everything that has happened in between. Your 'omes' reflect your ancestry, history, environment and exposures at every stage of the life course through prenatal to very recent contemporary experiences. In other words, an integrated 'omic' profile can be read as a timeline of a life course or life world. In fact, this can be extended further to say that this profile is not just the timeline of your life but those of your mother, your father, and your

Table 25.1 Omic technologies comprising the 'Interactome'

Discipline	Definition	Measure
Genetics	The study of genes, genetic variation and heredity (*the genome*)	DNA sequence
Epigenetics	The study of heritable changes in gene expression that do not involve changes to the underlying DNA sequence (*the epigenome*)	DNA methylation, histone modification, chromatin Remodelling
Transcriptomics	The study of how genes are expressed in a biological system (*the transcriptome*)	Gene expression, RNA transcripts
Proteomics	The study of the proteins in a biological system (*the proteome*)	Proteins
Metabolomics	The study of the small molecules in a biological system (*the metabolome*)	Metabolites (small molecules <10 kilodaltons)

grandparents, and, in many important respects, of the whole society of which you are a product.

In this fashion, 'omics' technologies provide compelling evidence that social disadvantage can lead directly to negative health outcomes. The effect of such social disadvantage is an interactive process, and conceptualising it deterministically is not helpful. It casts the biology into a passive-dependent role, with the interesting biology only emerging once the social exposures have taken place. It is better to see the process as an interactive social *and* biological one, with social practices conceptualised as the vehicle of exchange. The human body is not empirically a physically isolated thing either biologically or socially but is simultaneously part of a social and biological universe. Individual life courses have chronological, biological and social trajectories. These trajectories interact with each other. The velocities of these trajectories will vary, again with considerable social and biological consequences. Technology now enables us to track these.

The biological component of the interaction has been well described. In the context of molecular biology, the interactome is defined as the complete complement of molecular interactions in a biological system (Vidal et al. 2011). It encompasses multiple interconnected and overlapping biological networks, including gene regulatory, protein-protein interaction, biochemical and metabolic networks, forming a complex, dynamic and interactive regulatory system (Tieri et al. 2011). Multiple complex levels of regulation driving core processes are required for such systems to interact with environmental changes. 'Omics' technologies allow us to visualise and explore how such environmental changes, exogenous exposures and life experiences affect the network

topology and dynamics to influence health and disease (Khoury and Wacholder 2009; Boccaletti et al. 2006). In this way, the interactome can also be conceptualised, as we have done here, as the interacting social and biological networks and life worlds that can now be measured with the full complement of 'omics' profiles. The study of this interactome allows us more fully to explore the complex networks regulating the state of our health taking social factors into account.

It is becoming increasingly clear that this 'new biology' of 'omics' can be exploited in multi-faceted ways in the study of human health and disease. In fact, the 'omic interactome' is the best model we have yet to encompass the factors and systems originally specified by George L. Engel in his biopsychosocial model of disease causation, from the biosphere and society, through culture, community, the family, the dyad and the individual, to the nervous system and organs, tissues, cells and molecules (Engel 1960, 1977, 1981). If biological theory enabled the development of the interactome, social theory provides a means of describing how it is itself part of a broader dynamic interacting system involving human agency and social structure. We next describe four specific periods over the life course where particular recursive practices occur and demonstrate how 'omic' technologies can be used to directly link the social to the biological.

History and Ancestry

An individual's ancestry forms a vital component of their life, socially and culturally as well as biologically. Interrogation of the 'omic interactome' provides a novel approach to understanding how one's ancestors and the lives they lived may be affecting your present. Here, the genome is particularly informative. Population stratification is the presence of systematic genetic differences within a population due to non-random mating between groups, often resulting from their physical separation (Yashin et al. 2014). The study of this phenomenon can provide information on the race and geographical origins of one's ancestors. Furthermore, large sequences of the genome can be conserved through generations and so may reflect the adaptive response of ancestors to the evolutionary pressures they faced including climate, altitude, diet and disease, for example, the variation in skin pigmentation across the globe (Sturm and Duffy 2012) or the persistence of sickle-cell anaemia in populations originating from malaria-endemic regions (Aidoo et al. 2002). Finally, it has been demonstrated that your epigenome, transcriptome, proteome and metabolome, all exhibit an inherited genetic component thereby

further inextricably linking you with your ancestors and their lived experiences (Ritchie et al. 2015).

Recently, the interest in ancestry has taken an important turn in respect to the transmission of disadvantage—epigenetics. It has been demonstrated that the environmental conditions of your more immediate ancestors, your parents and grandparents, can also affect your health status. The development of epigenetics gained considerable impetus from observing the descendants of individuals affected by the 'Dutch famine' (a period of starvation in the previously well-nourished Western Netherlands in late 1944 following a Second World War German blockade). Not only were those who went hungry affected by subsequent diabetes, obesity, coronary heart disease and neurological conditions, their children and grandchildren were also affected. These effects occurred too quickly to be evolutionary changes, rather they appeared to be operating through a different mechanism which was influencing the genome. Incidence of these conditions was subsequently reported to be linked to differential methylation in several biologically relevant genes (Heijmans et al. 2008). The term 'epigenetics' comes from the Greek word 'epi' meaning 'above' the genome (Waddington 2012). Heijmans' study demonstrated for the first time that epigenetic changes could be passed down through generations and that the lived experience of your recent ancestors could influence your genome.

The implications of this for the reproduction of health disadvantage across generations and the stubborn nature of health inequalities across time are profound. Till relatively recently, health inequalities have been seen to be either the consequence of the health behaviour of individuals in their own lifetimes or alternatively the consequence of the social determinants of health operating in the here and now or the relatively recent past. The scientific developments reported in this chapter strongly suggest that health disadvantage linked to social disadvantage is reproduced both biologically *and* socially across generations and, importantly, provide an idea of how this is happening (Relton and Davey Smith 2012).

In Utero

That is not to say contemporary and recent exposures do not play a critical role. The importance of *in utero* exposures have long been known and is perhaps best characterised by Barker's 'foetal origins hypothesis', which demonstrated causal linkages across generations between social circumstances, maternal health during pregnancy and subsequent adult health of the child

(Barker 1991; Barker and Martyn 1992; Barker and Osmond 1987; Barker and Thornburg 2013; Barker et al. 2013). Some impacts of *in utero* exposures such as foetal alcohol syndrome or the legacy of the thalidomide tragedy are tangible and obvious. Yet, increasing evidence shows that maternal experience as a consequence of the social practices embedded in the mother's life impacts the 'omes' of their offspring and that these effects can influence their child's future health. Consequently, the study of these 'omes' may point to previously unknown mechanisms explaining Barker's striking observations.

Developing organisms are known to demonstrate considerable plasticity in response to environmental influences (Bateson and Gluckman 2012). Therefore, the epigenome is thought to be particularly sensitive to environmental factors during the critical window of extensive reprogramming that immediately follows fertilisation and to *in utero* exposures. In particular, maternal smoking, diet, medication use and stress have been shown to impact on the health of the child, and an increasing body of evidence supports the notion that that these effects are manifested through epigenetic mechanisms (Lee 2015; Monk et al. 2012; Knopik et al. 2012; Soubry et al. 2011). The downstream 'omics' profiles including the transcriptome, proteome and metabolome of offspring have also been shown to be susceptible to maternal experiences. Differential expression of genes involved in immune function as a consequence of maternal exposure to polychlorinated biphenyls (PCBs—synthetic chlorinated hydrocarbons that are the products and incidental by-products of multiple industrial and agricultural processes) has been reported (Hochstenbach et al. 2012). Similarly, cord serum proteomic profiles have been shown to differ by the smoking status of the mothers during pregnancy (Colquhoun et al. 2009) and to be affected by maternal exposure to arsenic (Bailey et al. 2014). Although to date there is a dearth of literature from human studies exploring the effect of maternal exposures on the offspring metabolome, evidence from animal models, including primates has identified metabolomic changes in the offspring of mothers fed a high-fat diet, that had health consequences in their later life, suggesting the impact is likely to be considerable (Cox et al. 2009). The study of these -omes therefore allows us to view the findings of Barker on the importance of maternal health in a whole new light.

That the health of women in pregnancy has an effect on their baby has been known since the nineteenth century and probably long before then. Similarly, that the mothers' health is affected by her social circumstances has also been known for many decades. What was not known was the link between the two and the mechanisms driving them. Interrogation of 'omic' signatures enables the exploration of how maternal exposures translate into health effects in their offspring. The mechanisms revealed by this new biology enable the visualisation of

a direct link between noxious social circumstances during pregnancy produced as a consequence of social practices and their toxic effects on human biology.

Early Life and Puberty

In addition to the *in utero* period, early life and puberty also represent particularly important developmental periods in terms of susceptibility to the exogenous world. Consequently, they have formed an important focus of the 'omics' literature to date. Early-life socioeconomic status, as measured by a number of indices including parental education and income, and family structure, as well as early life nutritional status and obesity, have all been associated with differential methylation of genes both in children at the time of exposure and in the adults they grow into (Demetriou et al. 2015). The transcriptomic profiles of children exposed to PCBs, air pollution and other environmental pollutants have been interrogated to reveal functional links between differentially expressed genes and the associated health effects (Dutta et al. 2012; Mitra et al. 2012; van Leeuwen et al. 2006).

The fact that such adverse conditions in childhood, such as exposure to environmental pollutants, parental smoking and suboptimal nutritional status, are biased towards the poor and the disadvantaged underlines the social reasons for the patterning in the associated health effects. The fact that children are powerless to control these conditions demonstrates the inextricable links between the interactome and the social. It also points away from blaming mothers in particular but parents and families more generally for the state of the health of their children. Social disadvantage, lack of power and resources with its associated social practices create a noxious social and biological environment which will wreak its devastation on the health of the child, on their subsequent health in adulthood and on the health of their offspring over succeeding generations. The idea, long part of public health policy thinking in the UK and beyond, that the alleged knowledge deficit about risks to health of the poor and disadvantaged can be remedied through health education and advice from professionals to change behaviour and produce fewer smokers, drinkers and overweight people is risible in the face of the new biology (Kelly and Barker 2016).

Adulthood

Importantly, your interactome remains susceptible to external influences throughout your life. 'Omic' signatures at the level of the epigenome, transcriptome, proteome and metabolome associated with lifestyle factors such as smoking, obesity and stress have been identified (Vineis et al. 2014;

Bortner et al. 2011; Hsu et al. 2013; Meng et al. 2013). Further, -omic profiles at every hierarchical level have been shown to reflect disease status; profiles associated with diseases spanning cancer, diabetes, psychiatric disorders, multiple sclerosis and neuronal injury have all been reported (Achiron et al. 2004; Tang et al. 2003; Twine et al. 2003; Chen et al. 2011). Perhaps, most pertinently, 'omics' profiling does not only provide a picture of what has been and what is happening currently, it is also able to utilise this information to tell you what may be to come. Signals generated at the earliest stages of disease pathogenesis before the onset of clinical symptoms have been identified in 'omics' profiles, hence they have the potential to predict future disease (Widschwendter et al. 2008; Wang et al. 2011; Wheelock et al. 2013).

On the flip side, healthy and positive life exposures have also been shown to alter the interactome. In one fascinating study, volunteers performed knee-extension exercise training on only one of their legs. Skeletal muscle biopsies from the exercised and unexercised control leg demonstrated biologically relevant changes in both the epigenome and the transcriptome (Lindholm et al. 2014). Similarly, dietary interventions have been demonstrated to be linked to changes in the metabolome (Elizabeth et al. 2013). Such data raise a vital and as yet unanswered question 'Given the dynamic and fluctuating nature of the interactome, to what extent can the embedding of social disadvantage be reversed'. Here again, in the exploration of this question, 'omics' is likely to play a pivotal role (Thayer and Kuzawa 2011).

Conclusion

The integration and analysis of multiple 'omes' allows the identification of meaningful biological networks to achieve an increased understanding of the human body on a holistic and temporal level (Kyrtopoulos 2013). By considering many hundreds or thousands of variables simultaneously, a picture of health and disease evolution can be constructed (Kyrtopoulos 2013). It is a novel approach to bridging the gap between social and biological factors and to demonstrating the deep-rooted and intricate way these factors interact in disease pathogenesis. Such analyses may offer the best explanation yet of how social and behavioural factors can be active parts of the same disease mechanisms. By visualising the temporal progression of the disease pathway, and potentially the 'critical windows' of exposure, we can gain a better understanding of where and how these social factors are working. And crucially what we might do about it.

As such, the interactome represents an exciting new development for both biological and social theory and crucially the integration of the two. However, currently, we lack the technical and analytical methods required to fully maximise and exploit its potential. On the biological side, the methods for both generating and interpreting 'omics' data are underdeveloped. Standardised protocols are lacking in terms of study designs and laboratory procedures and uncertainty surrounds the biological interpretation of 'omic' markers and their role in causation (Kyrtopoulos 2013). Large changes in the philosophies and methods of statistical analyses are required to keep pace with the technological advances. Further, the generation of such data, on such a vast scale, raises novel and important ethical and legal issues (Tieri et al. 2011; Kyrtopoulos 2013; Bonassi et al. 2013).

On the sociological side, while structuration theory and particularly the concept of social practice provide an overarching and promising framework to articulate the way overlapping life worlds and life courses are likely to shape the 'ome', there are to our knowledge no empirical studies that have attempted to explore this. These ideas remain at the level of a hypothesis. And of course other social and social and biological theorising may also generate related important hypotheses (see e.g. Rose 2005). Further, the degree of granularity required to explore what are likely to be highly nuanced and variegated intersecting social patterns and social practices remains to be formulated sociologically. And, perhaps most importantly, the dismissiveness of biology by some sociologists and the dominance of the reductionist paradigm in biology (at the expense of considering the social) (Rose 2005) both need to be overcome so that the distance between the disciplines can lessen and interdisciplinary progress can be made. Unfortunately, the long shadows of socio-biology and eugenics (Rose 2005) has meant that this interface has been a no-go area for many social scientists for too long.

But there is progress; as we noted above, it was initially hoped that the characterisation of the human genome would enable us to unlock the mysteries of health and disease, yet despite high expectations and a number of notable successes, ultimately, this has not been the case. Large-scale GWAS (genome wide association studies) have been under way for over a decade but have yielded a relatively small number of gene-disease associations, which have enabled only marginal increases in the ability to predict disease risk and offered only limited insights into the biology. They tell us little about how gene function and the biological processes are altered (Kyrtopoulos 2013; Manolio et al. 2009). However, one of the key developments that has arisen from the study of the genome is the importance of gene-environment interactions (Ottman 1996). It has therefore, perhaps surprisingly, revealed the necessity of taking an interactive

approach integrating biological and sociological analyses as ways of understanding individual and population levels' health.

What we suggest is critical; desirable and necessary is the development of the explanations of the social dynamics whereby the human interactome can be read as a timeline of a life world. The life course—the timeline of life—is related to social exposures in the life world. The life world is the locale where the products of the interaction between structure and agency—social practices—have their impacts via the recursive nature of social life. This is more than saying our interactome is the product of the way we and our ancestors lived. It is to say that by taking a sociological approach to the understanding of the social practices in which our lives are embedded and the patterning of those social practices, that it is possible to articulate the ways that individual and population health are produced and reproduced.

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26

Health Inequalities and the Interplay of Socioeconomic Factors and Health in the Life Course

Rasmus Hoffmann, Hannes Kröger, and Eduwin Pakpahan

Introduction

Health as a Meeting Point for Culture and Nature and for Sociology and Medicine

The study of health is a prominent example of a field of science where sociology and biology can meet to explore interactions between culture and nature. Analogous to environmental issues where society and culture are faced with their outside nature and its rules, health confronts sociological issues with the biology within the human body.

Medicine as a biological and natural science has always been the dominant scientific approach to health, but social determinants of health (education, behaviour, welfare) have been increasingly recognized as important factors. This has brought about a more social view on biological processes. At the same time, sociology has kept some distance from the biological view of the world and the biology in the human body. However, the question of how social

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factors "get under the skin" to influence biological parameters, illness and eventually death has increasingly attracted interest in several interdisciplinary fields such as demography, social epidemiology or medical sociology (Blane et al. 2013). In the field of life course research, the need for precise suggestions to combine social and biological knowledge in research on health have been introduced with the terms "social and biological plausibility" (Kelly-Irving et al. 2015).

Many scientists acknowledge that medicine cannot ignore social determinants of health just like sociology cannot ignore that death and the processes leading to it are biological facts that are largely outside the field of sociology or any cultural, social or subjective interpretation.

Before discussing social factors that create social differences in health and mortality, it is worth looking at a broader picture of how the biological health of the population benefitted from social and cultural progress: the massive increase in human life expectancy in the past 175 years (Oeppen and Vaupel 2002), which does not only mean survival but also better health, can be understood as a large-scale interaction between nature and culture. The great cultural achievements in nutrition, medicine, sanitation and education have made this progress possible. But these factors could only lead to such changes because the genetic and biological framework in the human body was malleable enough to allow for an almost doubling of life expectancy, offering a whole new life stage to large segments of the population. Again referring to the analogy between outside and inside biology mentioned above it is noteworthy that the cultural progress of humans was advantageous for biological processes within the human body while it seems to be partly disastrous for the natural environment. The positive consequences for health affect almost all humans but to a very different extent, resulting in very different health status between social groups and regions of the world.

Social and Other Differences in Health and Life Expectancy

Health inequalities between social groups have been found in all periods and countries for which data is available. Morbidity and mortality rates are systematically higher among people with lower socioeconomic status (SES), as measured for example by education, occupational status, wealth or income. Health inequalities usually amount to between 5 and 10 years difference in life expectancy and between 10 and 20 years difference in disability-free life expectancy (Mackenbach 2006). While average health and life expectancy

improves over time in almost all countries, also relative health inequalities increase (Mackenbach et al. 2015). The economic costs of health inequalities in the European Union have been estimated at about 1000 billion € per year, which is 9.5 per cent of EU-GDP (Mackenbach et al. 2011). The social factors that contribute to these health differences can be grouped into material living conditions (e.g. income or wealth), psychosocial conditions (e.g. social participation, job demand control), lifestyle (e.g. tobacco, overweight, physical activity, food, alcohol) and access to essential services (e.g. education, health care)(Doblhammer et al. 2009; Graham 2009).

When the average level of life expectancy or the magnitude of health inequalities is compared between countries within a relatively homogenous cultural setting such as the European Union or the USA, it is often assumed that international differences can be attributed to social/cultural factors rather than genetic or environmental differences (Hoffmann 2011). This makes it possible to attribute differences in health and health inequalities to man-made factors that, in principle, could be changed. In the empirical part of this chapter, we adopt this approach and compare European regions in order to explore if there are systematic differences. This reasoning may not apply to health differences between individuals where genetic differences or just "bad luck" have to be considered next to social, structural or behavioural factors. If instead larger groups of individuals are concerned, we can assume that random individual differences cancel each other out, provided that these factors are not systematically associated with the variable used for social stratification.

Without discussing in detail sociological concepts of stratification, one can call health inequalities between social groups "class-specific health differences" and compare them for illustrative purposes to race- and gender-specific health differences. All three types of health differences are examples of the interplay between biological/genetic and social factors, but to different degrees.

Gender or sex, basically a genetic and biological variable, has a major impact on how an individual is influenced by social factors. The term "gender" includes biological sex and social roles. Men suffer higher mortality than women, and part of this increased risk is due to certain behaviours and roles. Different roles for men and women in society also imply that they face different mortality risks. On the other hand, gender differences in life expectancy are partly genetic: women have different diseases, die of different causes of death and have a higher life expectancy than men. But they also have higher prevalence of illness (Doblhammer and Hoffmann 2009). The puzzle why "women suffer, men die" has been investigated intensively. Some studies suggest that about half of the differences in life expectancy can be attributed to genetic and the other half to social factors (Luy 2003).

Racial mortality differences also include genetic differences. Estimates suggest that they can be explained to more than 60 per cent by social differences (Smith and Kington 1997). Racial mortality differences have been described such that being black in the USA means having the health status of a white person who is five years older (Menchik 1993).

After illustrating how social and genetic factors contribute to health differences between gender and races, we return to class differences in health. It is difficult and ethically problematic, and politically at least detracting to say that SES also has a genetic background. But it is plausible to assume that at least to some extent genes also contribute to an individual's SES. Height and beauty, which both not only have a genetic component but also a social meaning, may illustrate how in principle such a causal relationship from genes to SES may work. For example, physical beauty may facilitate social acceptance in upper class social circles and this in turn may enhance upward social mobility. Health is another factor in a possible causation from genes to SES. But even if such pathways cannot be excluded, there is clear evidence that mortality differences between social groups caused by social factors are much larger than those caused by genes. Moreover, they are large enough to be addressed by research and policy and also large enough to rule out the assumption that social health differences represent a "natural" difference that cannot be changed.

The Causal Direction between Socioeconomic Status and Health

Whether systematic health differences between social groups are mainly a result of SES or whether there is also causality in the direction from health to SES is relevant for our understanding of the interrelation between biology and society and for normative and political questions. Scientific results that can be interpreted as causal evidence and that can be understood as an explanation (an answer to a why-question) have great practical relevance. For example, the success of interventions to prevent or reduce health inequalities crucially depends on manipulating causal factors and not just correlated factors. If research on health inequalities is supposed to go beyond a description and identification of disadvantaged groups, both the identification of mechanisms and the separation of causes and effects are important. Mechanisms that create health inequalities are manifold and have been discussed extensively in the literature (Case and Deaton 2005; Galama and van Kippersluis 2010; Hoffmann 2008). A thorough investigation of these mechanisms reveals those

by which SES influences health (social causation) and those by which health affects SES (health selection). The first of these causal models (social causation) implies, for example, that education influences health through health knowledge and risk behaviours, occupational status influences health through prestige and occupational hazards, income and wealth influence health through the affordability of health care, environmental hazards, consumption and the psychological burden of being poor. The second causal model (health selection) may entail that health influences education (during childhood) through the ability to invest in education, health influences occupational status through the ability to invest in a career and health influences income/wealth again through the ability to invest in a career and medical care expenditures (see Fig. 26.1). A third causal model to explain health inequalities is that (unobserved) background factors influence both SES and health (indirect selection) (Goldman 2001b). These factors may be genes, family background or personal characteristics (genetic or acquired), such as body height, personality or preferences with regard to behaviour and lifestyle. This third causal model is more difficult to test empirically than social causation versus health selection. Hence, in our chapter, we concentrate on the question whether social causation or health selection is more important in the creation of health inequalities in the life course, but we also consider indirect selection.

There is a disagreement between involved disciplines such as health sociology and social epidemiology on the one hand, and health economy on the other hand, concerning the relative importance of social causation versus health selection. The disagreement is not only due to different underlying ideas of the relation between social structure and individual agency, but also to different research designs and methods as well as to divergent views on

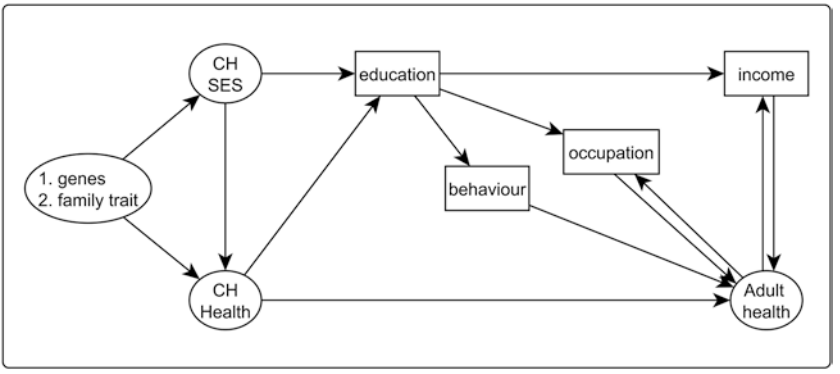

Fig. 26.1 Conceptional model for bi-directed relationships between SES and health in the life course. Notes: *CH* childhood, *SES* socioeconomic status

concepts of causality. Most studies in health sociology and social epidemiology use observational studies and interpret the social health gradient as causal influence of SES on health. The main strategies to explain health inequality are longitudinal designs in which the temporal sequence of SES, intermediate factors and health can be controlled and taken into account in statistical models. For example, Schrijvers et al. (1999) have compared the role of behavioural (e.g. alcohol intake, smoking or physical activity) and material factors as mediators and have attributed the main contribution to the material factors (e.g. income or housing conditions). Ross and Wu (1995) have explained health differences between educational groups with a direct effect of education and with the fact that higher educated people live under better economic conditions and have a healthier lifestyle. Estimates of the relative contribution of health behaviour and lifestyle to overall health inequality vary from 25 (Deaton and Paxson 2001) to 50 per cent (Adler 2001). There are quantifications of the contribution of specific risk factors (Eikemo et al. 2014; Hoffmann et al. 2015; Kulhánová et al. 2014; Kulik et al. 2013). Beside these empirical findings that make social causation plausible, there are theoretical arguments against health selection which address the timing of cause and effect in the life course. For example, it is claimed that although health changes in middle and old age cannot influence formal education any more, there are considerable health differences between educational groups (Haan et al. 1989). A central assumption of the selection hypothesis is social mobility as a result of health. While there are indications for a certain health-related social mobility at labour market entry (Power et al. 1998; Smith 1999), the amount of health-related social mobility is rather low (Smith et al. 1994; Kröger 2015). Moreover, the order of social mobility at younger ages and increasing health problems at higher ages seems to contradict the assumption of the health selection hypothesis (Hoffmann 2008) which postulates the reverse order: first there are health differences which then influence SES.

While these arguments and findings seem persuasive, it is often neglected that reverse causality from health to SES can bias the coefficients of conventional statistical models if the direction of causality is simply assumed (Hertzman et al. 1994). For example, it is often assumed that a social variable that is entered in a regression model as a predictor is really a cause and not a consequence of health. Normal regression results include no indication to what extent health also influences social status (health selection). Only few epidemiological studies have examined the possibility of selection (Blane et al. 1993; Chandola et al. 2003). The majority of authors in the fields of health sociology and social epidemiology believe that health selection is not very important (Chandola et al. 2003; Deaton and Paxson 2001; Goldman 2001a; House

et al. 1994; Manor et al. 2003). An agreement between health sociology/social epidemiology and health economy exists about the fact that education influences health (van Kippersluis 2010; Lleras-Muney 2005). But many economists think the influence of material resources on health is very low (Cutler et al. 2010; Michaud and van Soest 2008; Smith 2007) and that the influence of health on the material status is the strongest causality in the relationship between SES and health all together (Galama and van Kippersluis 2010).

In a recent systematic literature review on the relative importance of social causation versus health selection, we have selected 34 out of 2952 studies from the past 20 years, applying strict selection criteria. Then we have analysed them qualitatively and in a quantitative statistical meta-analysis (Kröger et al. 2015). The result is that, across disciplinary boundaries, there is no preference for one of the two causal directions. 12 studies supported causation and 10 support selection. The other studies supported both directions equally. Besides a partial lack of robustness, comparability and quality of the statistical results, we found a connection between the used indicators for SES and the preferred causal model: studies using an indicator that is closer to labour market performance (earned income, promotions, job market status, etc.) are undetermined, while studies using education or household income have a preference for social causation.

Our study offers an empirical starting point and perspective on the complex relationship and co-evolution of health and SES and thereby illustrates an important interplay between biology and society.

Data

We use the third wave (SHARELIFE) of the Survey of Health Aging and Retirement in Europe (SHARE) in which elderly persons were asked retrospectively about the development of their SES and their health since childhood. The data is representative for the population living in households and contains detailed information on events and periods of health and SES changes (Börsch-Supan et al. 2013). The data was collected with personal interviews at home using computerized questionnaires. We limit our analysis to ten countries (Austria, Belgium, Denmark, France, Germany, Italy, the Netherlands, Spain, Sweden and Switzerland) and to persons aged 50–90 at the time of the interview in 2008/2009 (n=19,549). For a description of the sample and the variables see Table 26.1.

We use three indicators for SES in childhood at age 10: the number of books in the household, the number of rooms per person and the occupational

Table 26.1 Description of the data (indicators, variables, categories, distributions)

Latent Construct	Variable	Category	*N* = 19,549	
	Country	Austria	819	4.19%
		Germany	1818	9.30%
		Sweden	1839	9.41%
		The Netherlands	2171	11.11%
		Spain	1971	10.08%
		Italy	2448	12.52%
		France	2408	12.32%
		Denmark	2070	10.59%
		Switzerland	1249	6.39%
		Belgium	2756	14.10%
	Age in 2009 (Wave 3)	Mean	67.07	
		Min	50	
		Max	90	
	Gender	Male	8832	45.18%
		Female	10,717	54.82%
	Education (number of years)	Mean	10.82	
		Min	0	
		Max	25	
		Missing	*2608*	
CSES(Childhood SES—Age 10)	Number of books	0–10 books	8127	42.43%
		11–25 books	4129	21.55%
		26–100 books	4166	21.75%
		101–200 books	1344	7.02%
		>200 books	1390	7.26%
		Missing	*393*	
	Father's occupation	ISCO Level 1	3771	20.64%
		ISCO Level 2	11,839	64.79%
		ISCO Level 3	884	4.84%
		ISCO Level 4	1780	9.74%
		Missing	*1275*	
	Rooms per capita	Mean	0.78	
		Min	0	
		Max	10	
		Missing	*466*	
CHEALTH(Childhood health—Age 15)	Self-rated health	Poor	466	2.42%
		Fair	1292	6.72%
		Good	4893	25.45%
		Very good	6009	31.26%
		Excellent	6565	34.15%
		Missing	*324*	
	Missed school	Yes	2271	11.78%
		No	17,010	88.22%
		Missing	*268*	
	Hospitalized	Yes	1211	6.27%
		No	18,097	93.73%
		Missing	*241*	

(*continued*)

Table 26.1 (continued)

Latent Construct	Variable	Category	*N* = 19,549	
ASES(Adult SES—Age 30–50)	Occupation	ISCO Level 1	2692	16.82%
	ISCO-Classification	ISCO Level 2	8780	54.86%
		ISCO Level 3	1756	10.97%
		ISCO Level 4	2775	17.34%
		Missing	*3546*	
	Average wages	Mean	1352.15	
		Standard deviation	1000.55	
		Min	7.49	
		Max	9124.65	
		Missing	*9913*	
AHEALTH(Adult health—Age 30–50)	Percentage of years of non-illness	Mean	0.97	
	Percentage of years of non-poor health	Mean	0.97	
	Percentage of years of non-stress	Mean	0.90	
OSES(Old SES—Age 50+)	Household income	Mean	19,344.24	
		Standard deviation	19,958.81	
		Min	0	
		Max	586,047.10	
		Missing	*2366*	
	Household wealth	Mean	157,892.50	
		Standard deviation	247,233.40	
		Min	–3,041,502	
		Max	6,932,346	
		Missing	*2606*	
OHEALTH(Old health—Age 50+)	Self-rated health	Poor	2279	11.79%
		Fair	5197	26.89%
		Good	7183	37.17%
		Very good	3097	16.03%
		Excellent	1568	8.11%
		Missing	*225*	
	Grip strength	Mean	33.94	
		Standard deviation	12.22	
		Min	1	
		Max	85	
		Missing	*1683*	

status of the father in four categories of the International Standard Classification of Occupations (ISCO). Education of the person was measured as number of years spent in education. As main working age, we define the age range 30–50 and use two social indicators, occupational status (ISCO) and estimates of average monthly wages in these 20 years, corrected for purchasing power and inflation by purchasing power parities (PPP) relative to German Euros 2006 (Weiss 2012). Because many persons are retired by the time of the interview, we measure SES in higher ages with the net-equivalent income and household net wealth per capita, both at the time of the interview (ages 50–90). Wealth includes, for example, real estate, cars, company shares and liquid funds and it subtracts debts.

Health in childhood is measured by three indicators referring to age 15: self-assessed health in five categories, the question whether school was ever missed because of health for one month or more, and the question whether one month or more was spent in hospital as a child. At ages 30–50, our health measure is based on three dichotomous self-assessed indicators, namely having bad health, illness and stress. We computed three continuous variables that measure the share of the 20 years spent in a particular status.

In old age, health was measured with the indicators self-rated health and grip strength. Grip strength has become a popular indicator of physical functioning in surveys. It is indicative of overall muscle and physical functioning (Cooper et al. 2011). Grip strength is objectively measured, avoiding biases that might arise in self-reports. It is further predictive of mortality, showing that it is related to health status more generally. Our health variables are coded such that higher values indicate more health problems. Age at interview, that also reflects the birth cohort, is a control variable in the statistical models, but shows no significant effect on the results.

Method

We chose a model-based approach to causal analysis using life-long retrospective data in order to study the interplay between SES and health. The advantage of a model-based approach compared to design-based approaches such as quasi-experiments is the possibility to simultaneously model two related processes (causation and selection) in which the outcome of one process is the predictor of the other. The assumption of this approach is that all relevant confounders are taken into account and a quasi-random distribution of the exposure to the risk factor (conditional independence) is achieved.

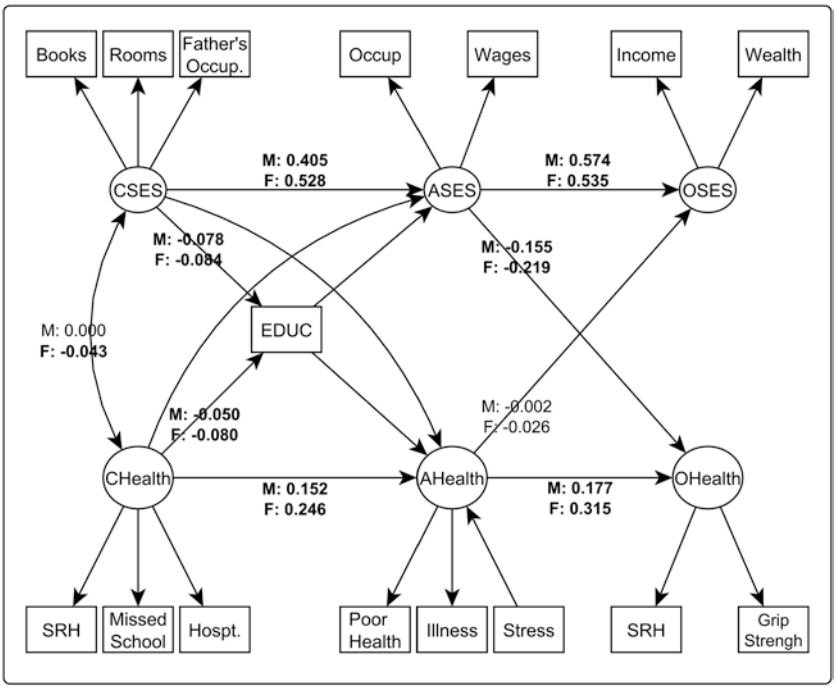

Fig. 26.2 Structural equation model for bi-directed relationships between SES and health in the life course with results for all countries combined. Notes: *countries* Austria, Germany, the Netherlands, France, Switzerland, Belgium, Sweden, Denmark, Spain, Italy, *C* childhood (0–15), *A* adulthood (30–50), *O* old age (50–90), *M*/*F* men/women; for indicators of the latent variables, see data section; *bold numbers* statistically significant ($p<0.05$)

We estimate the parameters of a structural equation model that represents the relative importance of causation and selection in different stages of the life course (see Fig. 26.2).

Structural equation models are a combination of path analysis of complex structural models and confirmatory factor analysis that describes latent variables using several observed indicators in a measurement model and minimizes measurement error (Bollen 1989; Pakpahan et al. 2015). We model SES and health in three different ages as latent variables. Our structural equation model computes the paths between these latent variables and education as observed variable. The models are estimated using the Full-Information-Maximum-Likelihood (FIML) method that includes persons with item nonresponse in the analysis. We show standardized coefficients in a uniform value range of −1-1 in order to make them comparable across paths and models.

Our model estimates the correlation between SES and health in childhood that can be jointly influenced by common unobserved background factors, for example, genetic factors or unobserved characteristic of the family. The actual path coefficients can be divided in two groups, first the autoregressive parameters showing the effect of SES at t_1 on SES at t_2 (the same for health), second the cross-lagged coefficients showing how SES at t_1 influences health at t_2 (causation) or health at t_1 influences SES at t_2 (selection). The cross-lagged coefficients in the first life course transition (from childhood to working ages) can be subdivided into direct and indirect effects, the latter being mediated by education. Direct and indirect effects can be summed up to the total effect.

We calculated the models on three levels, first for each country separately, second for countries grouped according to their region in Europe. These regions also roughly reflect different welfare state models: Austria, Germany, the Netherlands, France, Switzerland and Belgium represent west-European welfare states, Denmark and Sweden the Scandinavian welfare model and Spain and Italy Southern Europe. Thirdly, we combined all countries in one joint analysis. We consider this to be an explorative approach that illustrates the heterogeneity within Europe as a whole and within regions, but we do not claim to test particular welfare state-specific hypotheses. We do not show country-specific results below and we use country dummies in our aggregated models to control for unobserved country differences. All models are calculated separately for men and women.

Results

Results from the structural equation models are shown in Fig. 26.2 (as graphical illustration of the model and with results for all countries together), in Table 26.2 (all coefficients and standard errors) and in Fig. 26.3 (only results that are relevant for our main causal question).

As expected the correlation between SES in childhood (CSES) and health in childhood (CHEALTH) is mostly negative, that is, higher SES is correlated with less health problems. But only for women in all countries combined the coefficient (−0.043) is statistically significant, albeit small. This suggests that common background factors are not of major importance for the relation between SES and health. Except for the path from CHEALTH to AHEALTH among women in Southern countries, all autoregressive coefficients in both phases of the life course are statistically significant and range between 0.303 and 0.722 for SES and between 0.127 and 0.616 for health. A value of 0.616

Table 26.2 Results from structural equation models on the interplay between SES and health during the life course

			Parameter		Male				Female			
					West	North	South	All	West	North	South	All
Correlation			CSES ↔ CHEALTH	Coef.	0.015	−0.026	−0.013	0.000	−0.028	−0.081	−0.024	**−0.043**
				S.E.	*0.026*	*0.045*	*0.040*	*0.020*	*0.027*	*0.044*	*0.040*	*0.021*
Phase 1	Autoregression		CSES → ASES	Coef.	**0.303**	**0.336**	**0.495**	**0.405**	**0.632**	**0.314**	**0.529**	**0.528**
				S.E.	*0.034*	*0.040*	*0.069*	*0.027*	*0.038*	*0.044*	*0.067*	*0.027*
			CHEALTH → AHEALTH	Coef.	**0.144**	**0.127**	**0.164**	**0.152**	**0.244**	**0.413**	0.029	**0.246**
				S.E.	*0.035*	*0.050*	*0.064*	*0.026*	*0.050*	*0.131*	*0.116*	*0.042*
	Causation	Indirect 1	CSES → EDUC	Coef.	**0.488**	**0.456**	**0.617**	**0.500**	**0.486**	**0.515**	**0.592**	**0.503**
				S.E.	*0.017*	*0.025*	*0.025*	*0.013*	*0.016*	*0.023*	*0.023*	*0.012*
		Indirect 2	EDUC → AHEALTH	Coef.	−0.052	**−0.138**	−0.059	**−0.080**	**−0.072**	−0.059	−0.072	**−0.085**
				S.E.	*0.039*	*0.059*	*0.052*	*0.029*	*0.032*	*0.067*	*0.042*	*0.027*
		Indirect 1 × Indirect 2	(CSES → EDUC) × (EDUC → AHEALTH)	Coef.	−0.026	**−0.063**	−0.036	**−0.040**	**−0.035**	−0.031	−0.043	**−0.043**
				S.E.	*0.019*	*0.027*	*0.032*	*0.015*	*0.016*	*0.035*	*0.025*	*0.013*
		Direct	CSES → AHEALTH	Coef.	−0.038	−0.028	−0.040	−0.038	−0.035	−0.080	−0.034	−0.041
				S.E.	*0.040*	*0.052*	*0.050*	*0.029*	*0.036*	*0.073*	*0.039*	*0.030*
		Total	CSES → AHEALTH	Coef.	**−0.064**	**−0.091**	**−0.076**	**−0.078**	**−0.070**	−0.111	**−0.077**	**−0.084**
				S.E.	*0.030*	*0.044*	*0.033*	*0.023*	*0.028*	*0.069*	*0.039*	*0.024*
	Selection	Indirect 1	CHEALTH → EDUC	Coef.	−0.014	−0.015	**−0.063**	−0.014	−0.003	−0.027	−0.004	−0.004
				S.E.	*0.020*	*0.034*	*0.030*	*0.015*	*0.019*	*0.030*	*0.043*	*0.013*
		Indirect 2	EDUC → ASES	Coef.	**0.383**	**0.505**	**0.490**	**0.468**	**0.375**	**0.572**	**0.502**	**0.470**
				S.E.	*0.030*	*0.034*	*0.062*	*0.023*	*0.032*	*0.038*	*0.066*	*0.024*
		Indirect 1 × Indirect 2	(CHEALTH → EDUC) × (EDUC → ASES)	Coef.	−0.005	−0.008	**−0.031**	−0.007	−0.001	−0.015	−0.002	−0.002
				S.E.	*0.008*	*0.017*	*0.015*	*0.007*	*0.007*	*0.017*	*0.021*	*0.006*
		Direct	CHEALTH → ASES	Coef.	−0.013	**−0.104**	**−0.107**	**−0.044**	−0.044	**−0.116**	−0.109	**−0.078**
				S.E.	*0.023*	*0.041*	*0.045*	*0.020*	*0.031*	*0.047*	*0.059*	*0.023*
		Total	CHEALTH → ASES	Coef.	−0.018	**−0.112**	**−0.138**	**−0.050**	−0.045	**−0.131**	−0.111	**−0.080**
				S.E.	*0.025*	*0.044*	*0.049*	*0.021*	*0.033*	*0.052*	*0.069*	*0.024*

(*continued*)

Table 26.2 (continued)

				Male				Female			
		Parameter		West	North	South	All	West	North	South	All
Phase 2	Autoregression	ASES → OSES	Coef.	**0.722**	**0.537**	**0.596**	**0.574**	**0.536**	**0.560**	**0.518**	**0.535**
			S.E.	*0.062*	*0.057*	*0.064*	*0.041*	*0.033*	*0.073*	*0.051*	*0.029*
		AHEALTH → OHEALTH	Coef.	**0.151**	**0.157**	**0.184**	**0.177**	**0.260**	**0.616**	0.158	**0.315**
			S.E.	*0.041*	*0.069*	*0.051*	*0.026*	*0.056*	*0.156*	*0.106*	*0.045*
	Causation	ASES → OHEALTH	Coef.	**−0.118**	**−0.131**	**−0.193**	**−0.155**	**−0.162**	**−0.246**	**−0.220**	**−0.219**
			S.E.	*0.033*	*0.050*	*0.031*	*0.023*	*0.024*	*0.106*	*0.039*	*0.021*
	Selection	AHEALTH → OSES	Coef.	−0.009	−0.078	0.032	−0.002	−0.039	−0.102	**−0.085**	−0.026
			S.E.	*0.055*	*0.051*	*0.079*	*0.034*	*0.041*	*0.097*	*0.043*	*0.027*

Notes: Standardized regression coefficients; *S.E.* standard errors, *CH* childhood, *A* adulthood (30–50), *O* old age (50–90), *SES* socioeconomic status, *West* Austria, Germany, the Netherlands, France, Switzerland, Belgium, *North* Sweden, Denmark, *South* Spain, Italy, *Phase 1* transition from childhood to adulthood, *Phase 2* transition from adulthood to old age; for interpretation of the coefficients: for example, 0.5 means that one standard deviation change in the independent variable results in 0.5 standard deviation change in the dependent variable. Statistically significant coefficients ($p<0.05$) are printed in bold

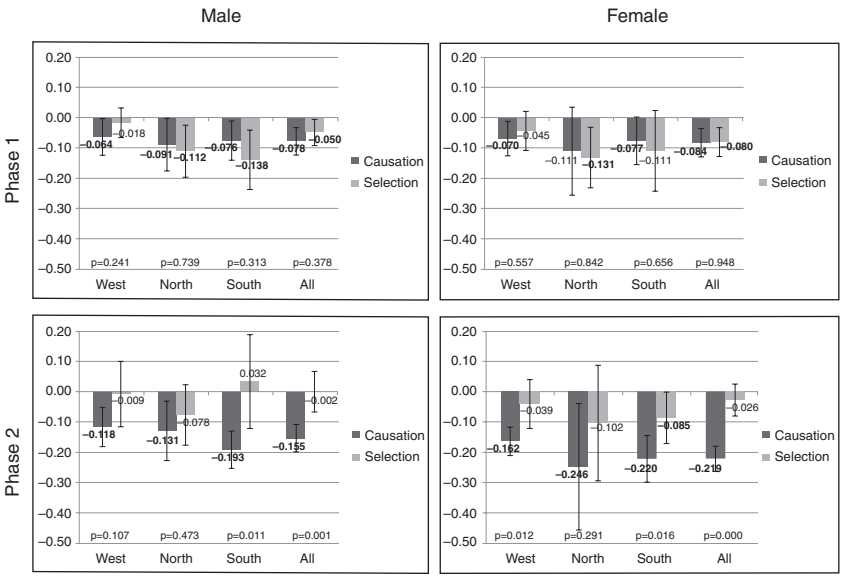

Fig. 26.3 Comparison of the strength of causation and selection, by life-course phase, gender and European region. Notes: *Causation* causality from SES to health, *Selection* causality from health to SES, *West* Austria, Germany, the Netherlands, France, Switzerland, Belgium, *North* Sweden, Denmark, *South* Spain, Italy, *Phase 1* transition from childhood to adult age, *Phase 2* transition from adulthood to old age, *bold numbers* statistically significant ($p<0.05$), *p*-values in the graph are from a direct Wald-test for difference between the coefficient for causation and the coefficient for selection

means that, for example, one standard deviation increase in CSES predicts a 0.616 standard deviations increase in ASES. These results show that SES and health substantially depend on their prior status, but this path dependency is stronger for SES than for health.

Our main causal question can be answered by comparing the coefficients that represent each of the two causal directions in the model (causation and selection). In the first phase, this comparison is more complex because our model estimates direct effects between SES and health and indirect effects that are mediated by education. Our results for the indirect effects show strong effects between education and SES in both directions: for all countries combined, the effect from CSES on education is 0.500 and 0.503 for men and women, respectively. The effect from education to ASES is 0.468 and 0.470, respectively. There is almost no association between education and health and no statistically significant effects from CHEALTH to education, except for men in Southern countries (−0.063), and only some small effects from education to AHEALTH (for all countries combined, −0.080 and −0.085 for men and women, respectively). This shows that education has some importance as

a mediator between SES and health in the transition from childhood to adult age, but the direct affects (all those not mediated by education) are equally strong. The two rows "Total" in Table 26.2 show the sum of the direct and indirect effect that is also displayed in Fig. 26.2 for all countries combined. As expected, all 16 coefficients in these two rows are negative, which means that higher SES leads to less health problems and more health problems lead to lower SES. Out of 16 coefficients, 12 are statistically significant and range between 0.050 and 0.138, which can be classified as relatively small effects that also do not show clear differences between countries or gender. To summarize phase 1 of the life course, we can say that among men causation seems to be slightly stronger than selection, while these two causal directions are similar among women.

For phase 2, our model is simpler because it does not consider indirect effects. As expected the coefficients for causation and selection are all negative, except for a non-statistically significant result for men in Southern countries (0.032). All coefficients for causation are statistically significant and range between −0.118 and −0.246, while only one out of eight coefficients for selection is statistically significant (−0.085 for women in Southern countries). In addition, the coefficients for selection are much smaller than for causation.

Comparing phases 1 and 2 we see that in phase 1 causation and selection have low values (causation being slightly higher than selection). Turning to phase 2, the amount of causation substantially increases while selection decreases which results in the finding that in phase 2 causation is much more important than selection, in terms of size of the coefficients and statistical significance. This overall result can also be seen in Fig. 26.3 where we only show the relevant coefficients for causation and selection, including a direct test for their difference that shows that in almost all regions causation plays a more important role than selection in phase 2.

Discussion

This study showed in a comprehensive life-course perspective that first, SES and health in childhood are not much correlated; second, SES and health during the life course substantially depend on their prior status; third, the mutual influence between SES and health increases with age; and fourth, in the transition from working age to old age the effects of SES on health (causation) are much stronger than the effects of health on SES (selection). These general findings apply to all country groups and men and women. We assume that

also a statistical test of gender differences would not have revealed statistically significant gender differences.

With regard to differences between European regions, to the extent that they also reflect different welfare systems, we adopt the plausible assumption that effects of the welfare state should be visible for men and women. In this sense we do not observe any clear differences between regions that would indicate clear evidence for country differences. This leads to the preliminary conclusion that welfare systems do not substantially influence the interplay between SES and health on the general level as we study it here. This includes the result that the more generous Nordic welfare model does not seem to perform better than other European regions. More detailed studies are needed to assess the role of different welfare systems for the relation between SES and health. From our study we can just conclude that both, ill health because of low SES (causation) and low SES because of ill health (selection), indicate a dysfunction of the social security system that should in principle try to avoid both mechanisms.

Our finding that causation and selection occur on a similar level in the first phase of the life course is in line with previous assumptions that there is a relatively high social mobility at labour market entry, where health matters for the career (Power et al. 1998; Smith 1999). On the other hand, our finding that subsequently causation increases and selection decreases is not in line with previous claims that selection is especially important in mid ages (Huisman et al. 2003) or older working ages where many health problems start to become more prevalent (Smith 2003).

To compare our results to existing similar studies, we refer to our own systematic literature review (Kröger et al. 2015) and discuss three selected studies that use similar age groups, indicators and methods. The first study uses prospective data from the USA, but with an age range from 41 to 88, that only covers our second phase. It shows that both causal directions are present, causation slightly more than selection, and with only small gender differences (Mulatu and Schooler 2002). Another study with mostly prospective data from the USA in the age range 18–65 also shows that health in childhood has no effect on the educational achievement. Interestingly, this study finds no evidence for selection (Warren 2009). Third, Finnish register data in the age range 17–66 shows causation being slightly more important than selection. However, their indicator for health is limited to sickness absence from work (Aittomäki et al. 2012). Other authors stress that evidence on the relative importance of causation and selection will always be a contingent result that depends on the social context, the method and the indicators used (Huurre et al. 2005). These indicators cannot be assessed on a simple gradient of more

or less validity, but particular dimensions of SES are probably also related through specific mechanisms to certain aspects of health. In this sense, it is possible that certain aspects of health are especially prone to contribute to selection while certain dimensions of SES foster causation. For example, many types of cancer are more equally distributed across social groups than other diseases, because their degree of genetic determination is relatively high. Therefore, these types of cancer would arguably contribute more to selection. In the realm of social variables, education probably contributes more to causation (from education to health) while social indicators that are related to labour market performance are affected by health selection (e.g., the effect of health on labour market participation). This complexity constitutes a theoretical and empirical problem that is not solved yet and our study is just a little step towards a critical reflection and some possible preliminary answers to a complex question.

In the quantitative assessment of the relative importance of causation and selection, the third causal model of indirect selection also needs to be discussed. It assumes that SES and health are determined by common background factors, such as innate cognitive or physical characteristics (O'Rand et al. 1999), that are genetically determined and that can lead to the development of specific personalities and even concrete preferences in lifestyle (Fuchs 1982). It is very difficult to empirically measure such common background factors and related mechanisms. We understand our result that health and SES in childhood are only weakly correlated as a modest hint that indirect selection is not of major importance. We would also claim that it is a good strategy, to start measuring SES and health as early as possible in the life course, to attribute as much as possible of their interrelation to either causation or selection, instead of using indirect selection as a black-box or residual causal model that absorbs all interactions before observations started.

Some authors describe indirect selection as being more important than health selection (Smith et al. 1994; Valkonen 1996). Other authors describe it as being unimportant (Goldman 2001a; House et al. 1994; Marmot et al. 1995). Generally, in empirical and theoretical research, this causal model is only rarely discussed and tested, maybe because it is even more difficult to verify than the difference between social causation and health selection. However, the unknown importance of indirect selection has been discussed in relation to the social justice aspect of health inequalities, where it seems relevant whether or not health inequalities are due to common background factors (Fritzell 2014; Mackenbach 2012).

The relative importance of the three causal models that exist for health inequalities can be interpreted in terms of the relative importance of biology

and society. For the two opposite models, causation and selection that we tested in our empirical analysis, one can argue that selection would imply more dominance of biology on the social structure and the hierarchy in society. However, we found that in some life stages there is more causation which implies that social processes determine biological outcomes. We can only provide weak empirical results on the relative importance of indirect selection, but one could argue that common background factors for SES and health would also point to a powerful biological influence. Such conclusion would be straightforward if the common background factors were genetically fixed characteristics. However, in times of emerging knowledge on epigenetics and the interplay between environment and the genome, such conclusions are much less obvious. Besides this, common background factors can also be socially determined but unrelated to SES, for example, certain aspect of lifestyle. Our study could only present some empirical findings and some theoretical interpretations, but many questions remain open for further interdisciplinary research.

The strength of our study and its design is a combination of factors that is new in the literature; first we start early in the life course by measuring the very beginning of the development of health and SES, and then go up to high ages. Second, we include many important indicators that are important for a valid measurement of SES. Third, we combine these indicators into measurement models for latent variables, which reduces measurement error. The substantial influence of measurement error on results and conclusions in a cross-lagged panel design has been shown in previous research (Kröger et al. 2016). Fourth, we use structural equation models that can simultaneously model two causal directions (causation and selection) also taking into account indirect selection to some extent.

On the other hand, there are remaining limitations related to our approach. The fact that our data cover a long time span comes at the costs of using retrospective data that in principle might be affected by recall bias (Smith and Thomas 2003). However, several studies have shown that the retrospective measurement of health and SES, including the SHARE data, is relatively valid (Garrouste and Paccagnella 2011; Haas 2007; Mazzonna and Havari 2011). The remaining disadvantages of this data need to be balanced with the fact that it allows the study of longer periods than in previous research based on prospective data (Adams et al. 2003; Stowasser et al. 2011). For the study of the relative importance of causation, selection and indirect selection it is especially important to commence measurement in childhood as the starting point of these causal mechanisms (Heckman 1981).

Besides the question on the type and timing of data production there is the more fundamental problem of how to measure complex partially unobservable concepts such as SES and health. On the side of SES, there is more or less agreement that education, income/wealth and occupation are relevant indicators, but the problem that these indicators may not be equally relevant for all population groups and age groups has not been solved. Decisions on how to measure SES are mostly guided by a mixture of theoretical and pragmatic considerations. On the side of health, we have to deal with a similar problem; that there is no universal definition and clear guidance on how to measure health. More subjective measures such as self-rated health have both advantages and disadvantages compared with more objective indicators such as biomarkers.

Our multiple indicators for SES and health also show differences in terms of their missing values. In general, survey data has high percentages of missing values and this is especially the case for sensitive questions on income. The highest percentage of missing values in our analysis (50.7 per cent) is for the variable average wages that is one of the indicators for adult SES (see Table 26.1). The other indicator is occupation that has 18.1 per cent missing values. Other financial variables (household income and household wealth in old age) have much lower missing values, namely 12.1 and 13.3 per cent, respectively. We performed a sensitivity analysis for average wages by excluding the 50.7 per cent of the sample that did not answer this question and found that this does not substantially change the results and would not lead to any different conclusion with regard to our main research question. We made a second sensitivity analysis excluding 1102 women and 37 men who reported, for at least 75 per cent of the years between age 30 and age 50, to have mainly worked in the household. Also this did not change the results. This is probably because, first, we always use several indicators to define a latent variable and, second, we use the Full-Information-Maximum-Likelihood method that is the preferable estimation method to deal with values missing at random.

The conclusion from our study is that none of the two causal hypotheses can be ruled out and both seem to be part of the creation of health inequalities in the life course. Causation is more important in the second part of the life course. The debate about these two causal directions should not be limited by prior assumptions or political beliefs but open to empirical evidence that can inform our general understanding of interactions between biology and society: our example of health inequalities illustrate how closely they are linked in the human life course, which makes it impossible to define any aspect that would be only biological or only social. More practically, our results can inform about specific strategies how health inequalities can be reduced.

If both causal mechanisms contribute to health inequalities, also both can be used to reduce them (Van Doorslaer and Koolman 2004). Both mechanisms reveal a dysfunction of the social security system but require different political strategies (Adda et al. 2003): selection could be addressed by better health policy and causation by a reduction of social differences in education, health behaviour and material resources.

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Part V

Medicine and Society

27

Universal Biology, Local Society? Notes from Anthropology

Patrick Bieler and Jörg Niewöhner

Introduction

"City living and urban upbringing affect neural social stress processing in humans" write German psychiatrist Andreas Meyer-Lindenberg and members of his group in a letter to *Nature* (Lederbogen et al. 2011, 498). On the basis of functional magnetic resonance imaging (fMRI) of cognitive performance under social stress, they argue that birth and early life in cities has a negative effect on stress response dissociable in adulthood, namely a slower operation of the amygdala control circuit in the brain. In a further study, the same group shows significant correlations between exposure to social stress and mental health problems such as schizophrenia (Lederbogen et al. 2013).

It is studies like these that over the last 15 years or so have begun to problematize the relationship of biology and society in a new way. In a somewhat ironic turn of events, the search for the essential building blocks of human life in the genome and in the brain returns the result that the molecular and cellular levels of analysis are full of references to 'the social'. It turns out that it is not simply matter "all the way down" (Haraway 2008, 32). Inadvertently, the life sciences are not only showing that the material basis of human biology is already heavily impregnated with remnants of culture understood as practice. They are indeed throwing up findings that question the entire conceptual model of a material base with layers of social practice and cultural values

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draped over it. In their confusion and indignation, the life sciences recognize that their object of research exceeds what their methods can see. They turn to the social sciences for help so that these may deliver empirical data about 'the social' to a realist understanding of the human body (Nature 2012). In 2016, this is not working all that well.

At the same time, however, in a further, even more ironic turn, the social sciences have returned from their journey to radical social constructivism and are fully engulfed in discussions about new materialisms (Law and Mol 1995; Barad 1999; Dolphijn and van der Tuin 2012; Latour 1993). A good time to respond to the cries for help from the life sciences one would think. Yet, this is not the case. Social sciences remain wary of biology. For too long, biology, and the human and life sciences more generally, have been (considered) the enemy of critical thought (Tsing 2000). Most domains of cultural and social anthropology—after all part of a discipline with a strong commitment to the scholarly understanding of humankind in all its aspects ("What is Anthropology?" 2016)—still do not show an interest in engaging in discussions about the nature of the human body and indeed the nature of human nature itself (Kuper and Marks 2011). The material dimensions of the human body have been black boxed in most ethnographic research and anthropological (and other social scientific) theory due to commitments to critical and social constructivist thinking considered incommensurable with collaborative leanings toward the life sciences (Blackman 2008; Palsson 2016b). Now, the social sciences are beginning to admit materiality into social inquiry and they are considering the role of the human body in social theory. Yet they remain curiously skeptical of the fact that a person is also an organism (Ingold 2000).

This chapter addresses this skepticism. We are aware of the complicated history that exists between biology and society (Meloni 2016; Meloni et al. 2016) and that is portrayed so lucidly in many of its facets in this volume. And we are aware of the important social science critique of biology (Foucault 1970). Yet as sociocultural and medical anthropologists, we are not entirely happy with the ways the important and multi-faceted relationship between the human material body and social practices is currently being explored. Pragmatic reductionism in the life sciences (Beck and Niewöhner 2006) does not engage generatively with constructivism in the social sciences and vice versa. This is problematic in at least two ways. Firstly, the life sciences are colonizing concepts such as 'social environment' that have long histories in the social sciences (Landecker and Panofsky 2013). Secondly, if social (science) theory acknowledges the fact that the (re)production of material-semiotic practices involves material agency in some shape or form, it seems

imprudent not to engage with biology as a discipline that knows an awful lot about the human material body and material dynamics (Rose 2013; Thrift 2008).

We suggest in this chapter that ethnography is a good way of forging a generative relationship between biology, the human material body, social practices (society) and the social sciences. Ethnography is a relational and humble practice that is happy to stay away from the grand questions of ontology and phenomenology and instead explores the partial connections between matter and meaning in an epistemo-, onto- and phenomenographic mode (Holbraad 2012; Lynch 2013; Niewöhner et al. 2016). Following the ideas of an anthropology of the contemporary (Rabinow 2008; Rabinow et al. 2008), we want to understand the human sciences as forms of practice, which play a central role in shaping the concrete sets of practices within which 'being human' is being done. We think that ethnography carries the potential to produce reflexivity within the epistemic, social and material architectures of late modernities (Law 1994; Boyer 2014). And we suggest that a human body-in-action might work as a boundary object between biological and social scientific research practices (Star and Griesemer 1989).

To argue our case, we revisit four scenes of ethnographic engagement with the intersections of biology and social science from medical anthropology and science and technology studies. We do so by focusing in an exemplary manner on a number of prominent figures in the respective research fields knowing full-well that the fields themselves are much broader and much more heterogeneous than we are able to portray them here. We draw out the role of the material body in each analysis before returning to the case of the relation of mental illness and urban life to outline what we think an ethnographic engagement between biology and society today might sensibly consider of analytical importance. We do not develop a holistic argument or resolve questions of nature-culture, human variation or particularity versus universality. Neither do we mean to suggest a chronology or evolution of inquiry toward some kind of a more comprehensive analysis. On the contrary, ours is an attempt to keep the coevalness of analytical approaches and their tensions and their messiness alive in discussions in the natural and social sciences through constant ethnographic engagement that is self-reflexively aware of its strengths and limits. Rather than defining how to 'think' the body as material-discursive assemblage (Blackman 2007), we propose a set of research practices that we want to engage ethnographically through modest withnessing (Sørensen 2009) in the life-scientific vectors of truth claims that are cutting people's/human lives in significant ways (Rabinow et al. 2008).

The Ethnography of the Body as Narrated

In the 1970s and 1980s, a conceptualization of medical encounters as meaningful, mostly language-based communicative processes has become a dominant strand of medical anthropology. Drawing on cross-cultural empirical studies, anthropologists criticized the biological reductionism and universalism of Western medical practice as well as empiricist versions of ethnomedicine. The social dimension of suffering is emphasized and a heuristic distinction between disease, illness and sickness is introduced for analytical purposes (Eisenberg 1977). Whereas disease is understood as only referring to abnormalities of physical functions of the body and thus belonging to the domain of Western biomedical models, illness and sickness are conceptualized as being culturally shaped. According to Kleinman, "[i]llness is the lived experience of monitoring bodily processes" (Kleinman 1988, 3–4) leading people to seek treatment.

Most authors who follow this narrative are interested in how illness is associated with heterogeneous meanings in a particular cultural setting relating to "the metaphors associated with a disease, the ethnomedical theories, the basic values and conceptual forms, and the care patterns that shape the experience of the illness and the social relations to the sufferer in a given society" (Good and Good 1981, 176). The main concern is to understand how the meanings of symptoms function as expressions of cultural beliefs and social interactions (Good 1977), how causes and courses of illness episodes are (differently) explained by healers, patients and their social environment (Kleinman 1980; Blumhagen 1980) and how narrating establishes a certain kind of coherence to a person's suffering (Kleinman 1988). Illness is thus personal and interpersonal at the same time. It directs the focus of analysis to individual biographies, family dynamics, the community of the sick person and the interaction of patient and medical professional/healer.

The overall aim is to establish models that can be used by clinicians in order to self-critically reflect their own assumptions, better interpret their patients' symptoms and complaints and deal with the social aspects of their patients' suffering (Kleinman 1981; Good 1995; Mattingly 1991). To the medical anthropologists, the illness narratives are both an analytical tool for understanding patients and an important part of the healing process, which can lead to a different perception of the disease opening up possible decisions, actions and futures (Mattingly 2009).

Medical anthropology thus argues for the importance of social and cultural analysis and contextualization of any medically relevant phenomenon instead

of reducing it to a physical process. Opposing a universalizing biomedical model, the particularity of the (individual) illness experience is emphasized. Importantly, this particularization emanates from social structure, symbolic webs of meaning and cultural practices. These are rendered as the anthropologists' field of inquiry and expertise (Kleinman and Kleinman 1991). Medical outcomes are of interest to the practitioner Kleinman and other scholars who argue for engaging with medical practitioners (Mattingly 1991). However, as the focus of these analyses is clearly to argue against biomedical reductionism with a primary focus on disease (Hahn and Kleinman 1983), the material body is almost framed as the universal carrier of cultural variation. It is an asymmetrical perspective that locates agency in culture, rendering the material body somewhat inert.

A changing medical anthropology in the 1980s driven by historical epistemological concerns about the contingent nature of knowledge practices, by matters of political economy and by feminist critique, begins to consider this perspective too individualistic and too narrowly focused on the patient-healer interactions (Young 1982). The strong reliance upon language is also criticized for assuming rational actors (Young 1981). According to Young, the medical system itself is always already socially and historically contingent and should, in effect, be understood as a set of situated practices. Thus, Young argues in favor of a critical reflection of the processes of knowledge production (in terms of power hierarchies) and its social effects (differential access to the medical system as a result of social inequality). Furthermore, he suggests the study of different forms of medical knowledge in their everyday use in order to account for the complex and sometimes contradictory statements of patients. What emerges from these early contributions is a new critical medical anthropology that necessitates an anthropology of biomedicine. Illness narratives are shaped as much by social structures as they interact with processes of knowledge production. The latter processes are no longer seen as a homogeneous body of medical knowledge but rather empirically and analytically pried open to reveal a heterogeneous set of knowledge and healing practices deeply enmeshed with matters of political economy and systems of (biopolitical) governance. The relationship between biology (as a discipline) and society is increasingly investigated in a constructivist and post-structuralist vocabulary that the interaction between science and technology studies, feminist critique and cultural anthropology has brought forth. In this vein, ethnographic research is increasingly conducted within the engines of discovery, production and circulation of medical knowledge primarily in Euro-America (Hacking 2007; Lock and Nguyen 2010). The material body becomes an object of power/knowledge practices and a site of biopolitics.

The Ethnography of the Body as Known

Early critical medical anthropology studied illness and healing in faraway settings. Yet, at the same time as Euro-American anthropologists travelled abroad, their own, 'modern healing systems' radically changed after World War II—and so did the relationship of biology and society. The health care sector grew dramatically through huge state investments in private and public institutions. Medical knowledge production and clinical interventions became more widespread and more powerful. The term 'medicalization' was established in order to describe medicine's expansive tendencies replacing and complementing legal jurisdiction and becoming increasingly implicated in peoples' everyday lives (Zola 1972). Illness and disease were theorized as products of social interaction posing the question how people come to accept and participate in medical institutions. Not only did the reach of medicine expand, medicine itself changed quite dramatically. From the mid-1980s onward, technologically enabled scientific innovations have rapidly changed the organization and practices of biomedicine. Clarke and others have termed this the 'era of biomedicalization' characterized by the inseparability of technological innovations and scientific knowledge production (Clarke et al. 2003). Medicine, health and illness are increasingly economized.

Biomedicine's pervasiveness in modern societies, its embeddedness within political economy and its role in shaping modes of governance rapidly attracted critical attention from the social sciences and philosophy. Most prominently, of course, Michel Foucault demonstrated in his lectures on biopolitics how medicine has introduced a new episteme into modern society that significantly shapes the conditions of possibility for subjectivity and sociality (Foucault and Faubion 2000). Two major strands of empirical investigation operate within this framework of biopolitics that need to be considered in more detail: On the one hand, a focus on the discourse of genetic risk emphasizes its highly individualizing effects and the ethical implications of constant responsibility for one's own individual health (Novas and Rose 2000; Rose 2001); on the other hand, authors emphasize the changes of individual and collective identification processes along new and widely distributed knowledge(s) of the biological body (Rabinow 1992) and its potentialities for political claims (Petryna 2003). These strands provide an epochal analysis of how technologically assisted biological knowledge production (especially in the field of genetics) shapes and changes 'modern' understandings of human bodies and being human (Franklin 2003).

For sociologist Nikolas Rose, the current discourse surrounding genetics leads to the identification of individual risk susceptibilities on the molecular level with the intention to prevent disease and illness. Although Rose argues that this is not a new mechanism of social control and power relations, he identifies a new mode of practicing biopolitics which he calls ethopolitics: "By ethopolitics I mean to characterize ways in which the ethos of human existence—the sentiments, moral nature or guiding beliefs of persons, groups or institutions—have come to provide the 'medium' within which the self-government of the autonomous individual can be connected up with the imperatives of good government. […] If discipline individualizes and normalizes, and biopower collectivizes and socializes, ethopolitics concerns itself with the self-techniques by which human beings should judge themselves and act upon themselves to make themselves better than they are" (Rose 2001, 18). This new form of politics does not operate on the level of coercion but rather through individual choice and the idea of optimizing individual lives, which, in turn, creates an individual responsibility for one's own biological and psychological resilience. Biomedical practices are thus construed to provide knowledge individuals can turn on themselves. However, as Rose also suggests, biopolitical knowledge production relies on technological innovation and thus depends on a biological industry. Biopolitics as ethopolitics is at the same time also bioeconomics and the 'politics of life itself' is at least partially controlled by those actors with financial resources. The human material body known through biology is in many ways at the heart of this critical social inquiry. It is conceptualized as a site of politics involving state and economic agents, medical practitioners and scientists as well as individuals performing technologies of the self. Yet, analytical attention lies on and stays with the biopolitical and economic processes that provide the basis for knowing and regulating the body. How this affects the human material body is of lesser concern.

In introducing the term 'biosociality' in the early 1990s, anthropologist Paul Rabinow has lead the way for a related argument which shifts the focus onto a different facet of biopolitics. Rabinow is interested in the emergence of new collective identities referring to the genetic knowledge(s) made available to the public at large. In a sharp critique of socio-biology (Wilson 1975), that is, the biological explanation of (social) behavior and social structure, Rabinow analyzes how the new biomedical practices (especially of genetics) change the configuration of social domains along biological lines, which are themselves the effects of the entanglements of knowledge production and collective action: "If sociobiology is culture constructed on the basis of a metaphor of nature, then in biosociality nature will be modeled on culture understood as

practice" (Rabinow 1992, 241). This process is highly dependent on technological innovation and scientific classification systems as the material basis for the identification of self and other according to genetic properties. The affected actors are not passive objects of this trend. Rather, knowing their biological body provides a means for collective action. Thus, the possibility is opened for claiming rights and (self-) representation according to the known materiality of the body. This is captured in the terminology of biological citizenship (Petryna 2003; Novas and Rose 2000). The emergence of biosocial identities can call for political action among different actors—affected and non-affected alike—and have an effect on the practices of medical knowledge production and classification (Epstein 2007). Hence, biomedical knowledge(s) and biosocial identification are co-constitutive.

The (bio)medical critique and the specific versions of biopolitics enable an analysis of how biological knowledge shapes society through discourse. In line with Foucault, the material body is first and foremost accessible through knowledge, which is itself bound to its epoch. The known body is a major resource for processes of identification shaping individual and collective experience, self-knowledge and identity, offering subject positions and legitimating rights claims. However, the transformations of the material body as effects of these practices and discourses over time and through different milieus receive little empirical attention.

The Ethnography of the Body as Lived

In the 1980s, it is feminist critique gaining ground within public discourse and beginning to enter the academy and particularly anthropology that starts to explicate the central role of the material body for social inquiry. Particularly, female anthropologists are developing an explicit concern for the human body. This comprises three levels of analysis: the individual body "understood in the phenomenological sense of the lived experience of the body-self"; the social body "referring to the representational uses of the body as natural symbol with which to think about nature, society and culture"; and the body politic, "referring to the regulation, surveillance, and control of bodies (individual and collective) in reproduction and sexuality, in work and leisure, in sickness and other forms of deviance and human difference" (Scheper-Hughes and Lock 1987, 7–8). This agenda is gaining immense traction. While particularly the body politic dimension is investigated discursively at macro-levels as a matter of life itself and biopolitics (see above), ethnographic and anthropological work also focuses on life as such (Fassin 2009). This shifts the level of

analysis to the domain of everyday life. The focus of attention is on the role of the body and biomedical institutions in rendering life meaningful and bearable. Much like Kleinman, illness experience is understood to be closely connected with the healing system. While Kleinman conducted his work within non-Western groups and societies, feminism and science and technology studies carry this agenda into the heart of Euro-American biomedicine. Yet where the ethnography of biopolitical processes focused primarily on sites of knowledge production and medical treatment, this new critical medical anthropology analyses "living and working with the new medical technologies" (Lock et al. 2000). It investigates the emergent biomedical practices and their inherent logics in themselves, their embeddedness within other social and ethical formations as well as their consequences for people whose everyday lives are being cut by these new vectors of truth claims (Martin 1994; Rapp 2004; Konrad 2005). These studies contribute detailed ethnographic material on healing systems and how they shape individual and collective illness and bodily experience. They also lead to a whole series of studies of the diversity of the material body as subject of biomedical (self)knowledge (Lock and Farquhar 2007). While they harbor a deep-seated skepticism toward biology and biomedicine, their ethnographic exposure to clinical and research routines lets them experience the heterogeneity of biomedicine in practice. Consequently, these anthropological approaches problematize the biological and medical(ized) body as lived and situated within diverse and often vernacular social, ethical and epistemic practices.

The Ethnography of the Body as Practiced

Empirical research of biomedicine in practice is also carried out by Dutch empirical philosopher Annemarie Mol (Mol 2002; Mol and Law 2004). While a child of constructivism, her interest does not so much lie with understanding how social configurations and individual experiences of the body are shaped and changed by biomedical practices and interventions. Stemming mostly from fieldwork on atherosclerosis in a medical clinic, Mol instead develops an ontological argument arguing for a multiplicity of the human body in practice: The human body exists only as and when enacted within practice (Mol 2002). Thus, what the body (disease) is depends entirely on how the body (disease) is done at any given moment. According to Mol, this does not mean that the body is fragmentary: Quite the contrary, her concept of the body multiple emphasizes that although the human body (disease) is done always partially in different practices, it is at the same time coordinated

into a whole through the combination of clinical practice and medical knowledge. In Mol's analysis, the materiality of the human body and its ability for resistance are captured as she conceptualizes the observed practices as material-semiotic. Materiality is present in co-producing the body.

Mol's framework is the attempt to ethnographically account for a body which is real and (socially) constructed at the same time. The radical emphasis on practice as the unit of analysis is an ontological argument against the idea of a universal body that is only epistemologically particularized—this is still implicit in the approaches of the experienced and known body. The (materiality of the) body is only accessible through epistemic practices. The perspective questions the separation of inside/outside, nature/culture and subject/object and constructs the body as an amalgam of heterogeneous co-producing human and non-human elements which is never finished but in a constant process of becoming. Finally, to distinguish clearly between the biological and social dimensions of human life is no longer possible from this perspective. This does not mean, however, that they are one and the same either. Yet, the radical practice-oriented approach of enactment is clearly focused on the processes of doing the body in medical practice. This is not a particular blind spot as the argument is not in itself anthropological. Nevertheless, ethnographically examining the material consequences of doing the body should be of interest to anthropologists, we argue, as questions of human development and evolution are in the focus of the discipline's matters of concern (Marks 2013).

Explicitly dealing with this interest is Margaret Lock's concept of local biologies, which develops a cultural anthropological concern for the material body. Although it can probably not solve the questions raised, it is especially interesting because it is not a theoretical concept that tries to explain the relation of nature and culture in producing a particular biological body and its social experience. Rather, it is an ethnographic notion that insists on the historical, social and political situatedness of the co-constitution of material and social phenomena. The notion of local biologies suggests a materialism of lived bodies (Lock and Farquhar 2007) that does not start from nature/culture and subject/object dichotomies but rather starts from the situated practices of doing, experiencing and knowing the body.

"[L]ocal biologies refers to the way in which the embodied experience of physical sensations, including those of well-being, health, illness, and so on, is in part informed by the material body, itself contingent on evolutionary, environmental, and individual variables. Embodiment is also constituted by the way in which self and others represent the body, drawing on local categories of knowledge and experience. If embodiment is to be made social, then history, politics, language, and local knowledge, including scientific knowledge

to the extent that it is available, must inevitably be implicated. This means in practice that, inevitably, knowledge about biology is informed by the social, and the social is in turn informed by the reality of the material. In other words, the biological and the social are coproduced and dialectically reproduced, and the primary site where this engagement takes place is the subjectively experienced, socialized body. The material body cannot stand, as has so often been the case, as an entity that is black-boxed and assumed to be universal, with so much sociocultural flotsam layered over it. The material and the social are *both* contingent—both local" (Lock 2001, 483–484).

This concept has been informed by Lock's long-term ethnographic work in Japan (Lock 1993). Lock noticed striking differences in the way 'menopause' was experienced by Japanese women compared to women in North America; most obvious, perhaps, was the absence of 'hot flashes' as a symptom of hormonal changes so regularly reported by North American women. Rather than reducing this differential experience to either culture (different discourse) or nature (different bodies), Lock weaves a thick ethnographic narrative that draws together differences in media reporting and public discourse, women's accounts of experiences, body images and genealogies of 'aging' narratives within a broader analysis of cultural and political context, including the role of the medical system. The result is not so much an explanation of cross-cultural differences in individual bodily experience but rather an ethnographic account of the diversity and contingency of female aging.

Importantly in this context, this is not a cultural anthropological account of the political, discursive and symbolic variance above a real and universal biological body. Neither is the account ignorant of a biological body. While Lock is a socio-cultural anthropologist with a degree of biological training, she has connected biological anthropological expertise to her ethnographic perspective. The role of soy as a major component of the Japanese diet has been shown to influence the levels of flavonoids in the body, which act as phytoestrogens on the body's hormone system (Melby 2005, 2015; Melby et al. 2005). So the differences in symptoms reported appear to have a biological aspect. In fact, the body becomes materially situated through particular food cultures.

Yet the concept of local biologies is neither interested in grounding social experience in a pre-social material body (Raman and Tutton 2010) nor is it interested in taking these biological accounts of the material body as reified truths. Rather, local biologies insist on investigating the experienced body as a site of epistemic, social, historical and political contestation. In this sense, it is firmly rooted within the social constructivism of the 1970s. Yet the experienced body is also a site of material contestation and knowledge about and

experience of the material body is always situated (Haraway 1988). Thus, Lock's interest in the materialism of lived bodies starts from the situated practices of doing, experiencing and knowing the body.

An Example: Mental Illness in the City

In the previous sections, we have traced the strengths of ethnographic research at the interface of the human material body, biology, medicine and society. Admittedly, we have been selective and have only sketched the different approaches as heuristic markers to make our case for an ethnographic engagement of anthropology (social sciences) and biology. We apologize to specialists in any of these areas for eviscerating what is so dear to them and we refer non-specialist readers to the multitude of volumes on the various facets of the subject (see especially Lock and Farquhar 2007). Our case, however, remains firm: The material body plays an important role in societal matters of difference and deviance. We need to stick with the lived body through practices; we need to take into account its experiences, its 'use' by a person, need to understand how it is known and how this knowledge is situated historically and socially. We need to understand how it is done in practices and how being done in certain ways affects the way subjects, socialities and societies construe themselves.

What we aim to achieve is to open up and engage in a debate on how to ethnographically grasp and account for human (group) life without privileging cultural or biological explanations.

To conclude, we return to the beginning of this chapter and discuss the case of mental illness in cities to outline the different dimensions through which the material body can enter an ethnographic analysis of the case. Taking this specific relation to the center of analysis has two reasons: Firstly, today more than half of the world's population lives in cities. This trend will rapidly increase within the next decades and the design of 'healthy urban environments' is becoming a central paradigm for urban governance. Even more importantly, specific sub-disciplines of biological and epidemiological research have rediscovered the field of mental health in cities and thereby undergone an epistemological shift, while at the same time this topic has a long tradition in ecological thinking within the social sciences (Fitzgerald et al. 2016b). Ethnographic research (especially in medical anthropology and the broad field of science studies) has not explicitly dealt with these questions so far but, as we have argued, provides different useful tools for a better empirical understanding of the materiality of the body in a globalized, yet locally practiced

world. We will therefore end with four propositions for ethnographic research on mental health in cities.

1) Staying with the Body Through Ecologies of Practice

We argue that the conceptualization of the body as being done in practices focusing on medical encounters in clinics can and should be translated into the urban environment. The body-in-action is thus constantly done through its embeddedness in the urban. Taking Mol's account into the city emphasizes the socio-material dimensions of mental health as concrete, observable doings. Even more, Mol's ontological conception of the body is crucial because it is completely different from most psychiatric/neuroscientific conceptualizations. Mental illness is thus not a separated essence from its surrounding environment but constantly enacted within shifting urban cosmopolitics (Blok and Farías 2016). Participant observation in the everyday movements of people diagnosed as mentally ill in the city could point to the centrality of lived space, paying attention to the various trajectories that built environments afford for people with psychiatric symptoms as well as the dynamics of social interactions in the city, while at the same time understanding the urban environment not as stable but as (different) 'niches' which are co-constituted through the bodily practices (Bister et al. 2016). This perspective enables us to think "how bodies are permeated through atmospheres and environments" (Fitzgerald et al. 2016a, 233). The body-in-action implies that it must be ethnographically accounted for in its complex entanglements with the assembled environment instead of trying to measure clearly defined, decontextualized variables (Söderström 2017). It is thus a useful tool for altering the status of the biological body (see section "Altering the Ontological Status of the Biological Body through Co-laboration").

As we are especially interested in thinking with the material consequences of the processes of how the body is done, situated biological knowledge about the different influences of specific environments on brain functions and their long-term effects on human development and phenotypes should be acknowledged (Timmermans and Haas 2008) and tried to be included into anthropological conceptions of human-environment relations (Ingold and Palsson 2013). There is not a straightforward strategy on how to include these findings into ethnographic research. Correlations of social and material environments with neurobiological or (epi)genetic patterns or markers do not readily relate to thick narratives or add up to some kind of comprehensive account (Downey 2016). Yet putting these accounts next to each other and reading

them relationally might produce something interesting and offers the chance to enrich the life sciences as well as the social sciences.

2) Locating the Body Differently Through a Biographical Perspective

As we commit ourselves to thinking about the material consequences of living in cities, we are further suggesting a long-term biographical perspective on the experienced body as an object of ethnographic research. The phenomenological approaches in medical anthropology as well as the concept of local biologies emphasize that experience is not given a priori, grounded in the biological materiality of the human body, but is (historically) embedded in cultural, social, political and epistemic practices. As we have shown, phenomenological approaches tend to focus on a narrative analysis of illness experiences and explanatory models. We find it crucial in this respect that Margaret Lock's research demonstrates in how far different accounts of symptoms should not be explained simply with reference to cultural differences in the representation of sickness—they might well be grounded in biological differences that result from living the body differently. Thus, we find this perspective to be complementary with the inquiry of the body in ecologies of practice. However, the political and moral dimensions of the materiality of the body are more explicitly dealt with—what we believe Didier Fassin is interested in conceptualizing "life as the course of events which occurs from birth to death, which can be shortened by political or structural violence, which can be prolonged by health and social policies, which gives place to cultural interpretations and moral decisions […]" (Fassin 2009, 48). Here, the focus of analysis is strongly directed toward the embeddedness of individuals in local ecologies of care over time (Das and Das 2006). Illness narratives and explanatory models can be a starting point for an analysis of the interactions of (a) social positioning, (b) attitudes and behaviors toward mental illness in relation to biomedical knowledge production, (c) cultural, social, and economic resources, (d) treatment decisions and options, (e) technologies of the self and social moral orders, including specific distributions of shame and blame, (f) social dynamics (e.g. inclusion/exclusion, stigma in families or neighborhoods, etc.) and (g) biosocial identity formation. The role of medical institutions, drug regimen and treatment carriers can be illuminated by trying to reconstruct the 'medical career' of the sick person through documents and interviews with past practitioners and ancestors (Biehl 2005). This can be an extremely laborious task demanding huge personal and ethical commitment from the

researchers. However, this perspective is anthropologically useful because it is dynamic and emphasizes change within the life course. It opens up the possibility to research lifestyle-environment-body trajectories (Bowker and Star 1999) or human-milieu-environment relations (Palsson 2016a).

3) Accounting for the Body in Urban Epistemic Spaces

The human body cannot be separated from knowledge and skill. The way people conduct and experience themselves, the way medical institutions operate, the way epistemic cultures of biology contribute to enacting the body multiple—all this is shaped significantly by knowledge practices. That medical knowledge heavily shapes social relations as well as collective and individual identity formation has been made evident through biopolitical critique. Yet the production of difference and deviance within societies has long ceased to be a matter of specialized medical knowledge being received by a lay public. The vectors of truth claims have long started to operate within much more diverse ecologies of expertise involving public and private actors as well as infrastructures, technology and natural and moral orders (Beck 2015). Knowledge is not just in the mind and affecting technologies of the self. The current neuroscientific domination in psychiatric research begins to approach urban planning (Fitzgerald et al. 2016a). The built environment comes under scrutiny from the medical and the molecular gaze (Landecker and Panofsky 2013). Interventions into the material world might foreshadow a new kind of pastoral power that does not address itself to human subjects any longer, but that constructs healthy environments that govern subjects at a new kind of distance (Kontopodis et al. 2011; Niewöhner 2011). Ethnographic research needs to understand and become involved in these ecologies of expertise in order to understand how matters of fact become matters of concern (Latour 2004) in material, social and cultural contexts. How does this shape and transform urban environments and care infrastructures? What are the effects for people's dwellings (see section 'Introduction')? And how do people's experiences of illness, their treatment decisions and options as well as their social relations develop and change over time (see section 'The Ethnography of the Body as Narrated')? The notion of ecologies of expertise moreover offers a chance for new forms of collaboration between social science, medicine and people with experiences of psychiatry. This might lead to at least local and experimental transformations of psychiatric classifications (see section 'Altering the Ontological Status of the Biological Body through Co-laboration').

4) Altering the Ontological Status of the Biological Body Through Co-laboration

We have outlined the dimensions of the body-in-action that are accessible to ethnography as conventionally understood in anthropology. Yet there might be more to do for ethnographic inquiry. Barry and colleagues point to different modes of interdisciplinary collaboration (Barry and Born 2013). In their agonistic-antagonistic mode of interdisciplinary engagement, one of the partners is trying to change the ontological status of their collaborator's object of research. This resonates with much recent writing about ethnographic projects that co-laborate with (Niewöhner 2016), experimentally entangle (Fitzgerald and Callard 2014; Callard and Fitzgerald 2015), para-sitically attach (Marcus 2010; Deeb and Marcus 2011) or embed (Rabinow and Bennett 2012) the ethnographer in life scientific contexts. We have outlined the body-in-action above and we suggest here that co-laboration between biology and anthropology (social sciences)—understood as temporary joint epistemic work—might lead to research designs that capture this body-in-action as it is lived and used out there, shaped by everyday patterns of practice.

Ever since early enlightenment when moderns started to understand the natural world in terms of laws rather than customs (Daston 2002), biology has been left on the edge of the natural sciences not entirely sure about the epistemological status of the natural order. We have witnessed many universalizing projects, particularly within the recent large-scale genome initiatives. Yet we have also witnessed counter-movements that have focused on emergent embodied phenotypes (Krieger 2013) and emphasized dynamic, ecological and systemic perspectives (Oyama et al. 2001). The fact that certain sub-disciplines within biology and epidemiology have started to open up the discussion on the deep entanglement of body and environment is a valuable entry point for working together on situating biology and differentiation. For social scientists and anthropologists, this momentum is a chance to account for the material dimensions of social differentiation—understood not in terms of measurable variables but as a complex biosocial phenomenon. It is an opportunity to go beyond the idea of situating universal bodies in local contexts and it invites anthropology to empirically translate the theoretical insights of the new materialisms in order to understand in how far and through what practices the materiality of the body is (made) local.

Anthropology should thus neither function in the mode of distant (social constructivist) critique nor as a data delivery machine, which is only appending the 'social context' for natural science research. There is the potential to shift the research methodology and become an epistemic partner that

can generate critique from engagement with its co-laboration partners. We see the potential to develop anthropological theory through approaching the materiality of human (group) life in an experimental mode. The body-in-action, we suggest, might work as a boundary object for biology and anthropology. It might produce a somewhat more integrated perspective, yet, more likely, it will produce bodies in both disciplines that are good to think with and that help to avoid unnecessarily early slips into reductionisms of one kind or another.

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28

Big Data and Biomedicine

Nadine Levin

Introduction

In the late summer of 2012, following the London Olympics, a series of stories came out in British newspapers announcing the inauguration of a "Phenomenal Olympic Legacy" (UK Department of Health 2012). The newspapers announced the opening of the National Phenome Centre, a facility which would take the laboratory equipment used to test for illegal performance-enhancing drugs—over 20 mass spectrometry and nuclear magnetic resonance machines, each costing hundreds of thousand pounds—and transform it into a state-of-the-art biomedical research facility. This multimillion-pound investment was described as a way to further reap economic benefits from the London 2012 Olympics, and as a way to reinvest the cost of the games in the health of the British public. It was proclaimed by its new director, Professor Jeremy Nicholson of Imperial College London, to be "a first for the UK, there's no question about it" (Walter 2012). While the Olympic games had seen the testing of more than 6000 samples, the National Phenome Centre was set to analyze 25,000 samples in its first year (Saini 2012).

The Centre had been founded with the aim of investigating peoples' "metabolic phenotypes." This term refers to the collection of unique, measurable signals that summarize a person's metabolism, and that result from interac-

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tions between genes and environment. Analyzing biological samples like urine, blood, feces, and tissues from hundreds of thousands of people, researchers at the Centre would investigate disease processes, and look for risk factors associated with chronic illnesses like cardiovascular disease and cancer—with the ultimate goal of developing new diagnostic techniques and therapies. "You can gather a huge amount of information about the biochemistry of people just from a drop of blood," said one researcher, "You can tell how old somebody is, their BMI, their gender, diet, what drugs they're taking and the nature of their disease" (TechRepublic 2015).

The Phenome Centre is emblematic of the rhetorics and technological changes that have swept through biomedicine in the early twenty-first century. It is one of a number of investments, at a seemingly unprecedented scope and scale, in molecular approaches to the health of individuals and populations. In July 2013, the UK launched the 100,000 Genomes Project, an ambitious undertaking to sequence 100,000 whole genomes by 2017, with the hope of uncovering rare genetic diseases. Just 18 months later, President Obama announced the "Precision Medicine Initiative" during his State of the Union address, a project which would create a personal healthcare information database, consisting of patient histories and genomic analyses of more than a million individuals, with the aim of creating more "personalized" drugs and medical care.

The desire to peer into the human body at the molecular level—through the Human Genome Project of the 1990s, or the more recent Human Microbiome Project of the 2000s—is not new. The Phenome Centre is unique not because it provides a window onto the metabolism of individuals and populations, but because it uses data to provide an all-encompassing view of peoples' lives. In the study of metabolic phenotypes, what you eat, how well you slept, what drugs you take can all have measurable effects on the body. In this system, where the combined effects of genes and environment can be measured and quantified, metabolism is not just downstream of genes, but also influences and shapes them (see Landecker 2011; Landecker 2015). Metabolism transcends the reductionism of genetics (Keller 2002; Kay 2000), moving beyond the flat textual world of the gene, and toward a world of dynamic complexity (Ackerman et al. 2015; Arribas-Ayllon et al. 2010; Levin 2014a; Wynne 2005). But in order to capture metabolism in all of its complexity, as the sum total of the interactions between genes and environment over time and space, the Phenome Centre must draw on massive amounts of data about bodies and environments. The Phenome Centre therefore represents a science concerned not only with bigger data, but also with data that is

more pervasive, with data that knows no limits to what it can and should measure.

Ventures like the Phenome Centre have been spurred by the explosion of 'omics' and 'omes' (Hotz 13 August 2012): fields like genomics, proteomics, and metabolomics, which are dedicated to the study of the various molecules that make up living organisms. With technological innovations in gene sequencing, microarrays, protein crystallography, and mass spectrometry, these fields have enabled researchers to transcend outward symptoms and peer into the body at the molecular scale (Braun 2007), revealing the genetic mutations associated with cancers, and the metabolic interactions underlying obesity.

But, lurking underneath the surface of press releases and white papers, projects like the National Phenome Centre are also stories of big data; of attempts to define health, disease, people, and populations through increasingly large, complex, and unruly collections of information. The National Phenome Centre is symptomatic of a broader trend in biomedicine, in which governments, researchers, and companies are using data to diagnose and intervene into human health. The complex nature of post-genomic biology captures the imagination of scientists, who then attempt to measure, quantify, and intervene into bodies with data—presenting new opportunities, but also new questions, for the future of biomedicine (Boyd and Crawford 2012).

What if, instead of looking at developments like the National Phenome Centre through the frame of post-genomic science, we approach them through the lens of big data? How can we use the tools of anthropology and Science and Technology Studies (STS) to examine the ways in which data, big and small, is changing medical research and care? How can we also use these tools to critically explore the broader politics and practices of data, i.e. the societal patterns and trends that go hand in hand with data-centric approaches to health and disease?

This chapter provides an overview of the changes that big data is rendering in biomedicine, providing a brief analysis of the term "big data" and its relationship to biomedical paradigms. This chapter explores how data is changing the knowledge practices of biomedicine. Ultimately, this chapter sets out an agenda for an "anthropology of data," as a means to question the norms, politics, and values that get wrapped up in data. To conclude, the chapter introduces the notion of "data binds" as a way to grapple with the ambivalent, uneven effects of data on society and biomedicine.

Big Data and Biomedicine

"Big data," often defined in relation to the "3Vs" of volume, variety, and velocity (Laney 2001; Kitchin and McArdle 2016), first came into being in the published literature in 2003 (Lohr 2013; Diebold 2012). It refers to massive datasets that must be analyzed computationally to reveal patterns and correlations. Big data is therefore about much more than just data: it is about the statistical methods, machine learning, algorithms, complex visualizations, databases, and informational infrastructures which enable data to be cleaned, organized, and made meaningful.

Since the rise of big data in the early 2000s, its role and visibility has grown, leading to fundamental transformations in society, the economy, and everyday life. Companies like Google, Facebook, Twitter, Apple, and Uber—whose very existence is predicated on data—have altered the ways people spend time and money, and relate to one another (Turkle 2012). Beyond the realm of Silicon Valley and Wall Street (MacKenzie and Spears 2014), data is also becoming increasingly central to healthcare (Bender 2015; Topol 2012). In the developed world, a healthcare ecosystem has emerged where individuals turn to phones and other "smart" sensors to track their health (Lupton 2015), and where hospitals and research institutes turn to electronic health records and other sources of digital data to evaluate the cost and effectiveness of health interventions (see Kayyali et al. 2013). Data is a commodity with untapped economic and social value, a resource for improving the health of individuals and population, as demonstrated in a McKinsey report proclaiming that by 2025 health data could account for a worldwide economic impact of over one trillion US dollars (Manyika et al. 2015).

In twenty-first century biomedicine, the development and success of big data is discussed in relation to paradigms such as "translational research," the movement and application of laboratory research to clinical issues (Davies 2012; Levin 2014b; Rajan and Leonelli 2013), and "personalized medicine," the shifting of disease categories away from clinical symptoms to account for individual biologies (Tutton 2012). Within such paradigms, researchers attempt to improve upon the perceived subjectivity of clinical practices like symptom reporting (Whitmarsh 2011), the visual assessment of cells and tissues (Löwy 2009), and the reliance on surgeons' human skills and capacities (Prentice 2012). Such practices are seen as being prone to human error and bias, which—perhaps unsurprisingly—can be ameliorated with the use of more objective data-intensive approaches.

For example, one of the flagship translational technologies being developed by the researchers associated with the National Phenome Centre is the "iKnife." This device uses the surgical technology of electrocautery, which cuts through tissue with a hot element, and a mass spectrometry machine, a biochemistry technology that is used in fields like proteomics and metabolomics (Balog et al. 2013; Imperial NIHR Biomedical Research Centre 2011). The iKnife uses mass spectrometry to detect and analyze the "chemical smoke" generated during electrocautery, giving surgeons a new tool for understanding the molecular composition of the tissues through which they are cutting—about tumor margins or cancerous metastases. Similarly, a widely publicized 2013 study showed how big data techniques, which had originally been developed in astronomy, were able to surpass the "manua[l] scoring of results" of cancer diagnosis via the "age old practice" of histopathology (University of Cambridge 2013; Ali et al. 2013). Consequently, these data-intensive translational approaches are paving the way for the real-time analysis of tissues, reducing the time and cost of surgical interventions, and paving the way for new understandings disease.

Data as we now know it, in its digital and large-scale form (see Leonelli 2016), became central to biomedicine through the Human Genome Project (HGP), which began in the 1990s, and culminated in the full sequencing of the human genome in 2003. The HGP, as the story goes, revolutionized the scale and scope of scientific research. Scientists began to focus on the molecular inner-workings of organisms and cells and over the course of the project, learned to carry out the discovery and analysis of genetic information in increasing volumes and smaller amounts of time. Today, for example, it is possible to sequence a person's genome—which consists of 100 gigabytes of data—for $1000, nearly 1/10,000 of the original $3 billion cost (Hayden 2014). Following the HGP, large-scale governmental initiatives like the 100,000 Genomes Project and President Obama's Precision Medicine Initiative have emerged as ways of harnessing the health-promoting potential of data, while commercial products like FitBit and the Apple iWatch have been developed to mine the economic value of data about lifestyle and health.

Despite this genome-inspired origin myth, data has always been central to biomedicine, albeit in different forms and ways. Bruno Strasser (2012) has argued, for example, that it was only because the experimental sciences became more important than the natural sciences in the late nineteenth century that data-driven research was perceived as a new aspect of twenty-first-century biology. Natural history, according to Strasser, had been data-driven for many centuries before the emergence of post-genomic research, through the cataloging and collecting of organisms and their attributes. Similarly, Joel Hagen

(1998) has argued that the use of computers and statistics in biology, which enabled the generation and analysis of large volumes of data, dates back to work on protein crystallography in the 1950s and 1960s. Appeals to the "data deluge" and "information overload" are not as new as we might be led to believe, as "every age was an age of information, each in its own way" (Darnton 2000). New forms of data—whether they were the punch cards of the 1930s (Edwards 2010) or the population statistics of the 1800s (Foucault 2003; Foucault 1977; Hacking 1990)—have always overwhelmed society with their volume and complexity.

And yet, there seems to be something different about twenty-first century "big" data. I argue that big data is unique not because of the technological changes it brings about, but because of the social conditions in which it operates. In the era of big data, numbers are being afforded greater power and value relative to other forms of knowledge, such as the arts and humanities disciplines' more qualitative and aesthetic modes of inquiry. Big data represents, as Boyd and Crawford (2012) argue, a "computational turn in thought and research," where value is placed on data regardless of the condition of its production, or of the work required to make it meaningful. Big data has heralded a shift in the processes of knowledge production, and consequently in the kinds of knowledge that are produced. If things are not countable, then they do not have a role in the information economy, and effectively cease to matter. At stake in this new data-driven mode of knowledge production is what happens to phenomena which are difficult to count, which are not easy to measure, or which cannot easily be combined with existing datasets. Unfortunately, such phenomena—qualitative and contextual information—are often those which make big data meaningful, or which allow us to ask new questions rather than confirming existing hypotheses.

To see how these kinds of questions and challenges affect society, take the parable of Google Flu Trends (GFT), a project launched in 2008 by the search giant Google to use search query data to track influenza epidemics. By tapping into the fact that people often search for flu-related information when they are sick, Google showed in a 2009 *Nature* paper that for US influenza outbreaks, GFT could match epidemiological surveillance data from the Center for Disease Control (CDC) almost exactly—and with a faster lead time (Ginsberg et al. 2009; Lohr 2014).

GFT was hailed as a major innovation in the realm of healthcare, as a way to improve the dated, costly methods of epidemiology with big data (Krenchel and Madsbjerg 2014; Madrigal 2014). Given its success, it was rolled out in 29 countries, and applied to dengue fever as well as influenza. In subsequent years, however, research revealed that GFT greatly overestimated flu levels in

the 2012–2013 season, when it nearly doubled the CDC's estimate. Previously, the algorithm had underestimated the 2009 swine flu epidemic, forcing Google engineers to tweak the algorithm to account for changes in the nature of search behaviors and influenza biology (Butler 2013; Cook et al. 2011).

It is tempting to claim that such failures are caused by methodological shortcomings, by the fact that the technology needs more time to develop and mature. However, such failures also highlight, on a much broader and theoretical scale, the challenges of using big data to shape healthcare. Firstly, what exactly *are* the various kinds of data being implicated in big data efforts, and what aspects of our social world are they able to capture? Many big data innovations like GFT are based on "found data," on data from cell phones and online activities—data which has not been purpose designed or collected for particular ends (Harford 2014). Although this is often hailed as a strength—enabling companies to generate new insights by transcending existing categories and theoretical paradigms—such found data is often poorly understood and subject to unknown sampling errors. Researchers have shown, for example, that drawing on large volumes of data in social media networks like Twitter does not eliminate bias, but can even exacerbate it (Ruths and Pfeffer 2014). This reveals the untested assumption that big data is using the right categories and measuring the right things—or even that the most important aspects of life can be quantified in the first place.

Secondly, what does it mean to structure health interventions around correlation, and to claim that with data, understanding causation—understanding mechanisms and asking "Why does this happen?"—is not as important as finding patterns (Naughton 2014)? Researchers have increasingly become aware of the challenges of false positives in big data sets that are "short and fat" (MacKenzie 2015), which contain many more variables than data points. As the books *The Signal and the Noise* and *Spurious Correlations* aptly point out (Silver 2012; Vigen 2015), if you test enough correlations in big data sets, you will eventually find one that is true, even if that correlation means nothing—such as the correlation between autism diagnosis and organic food consumption (Doctorow 2013). This leads to "cherry-picking [at] an industrial level" (Taleb 2013), whereby value and power are attributed to data, rather than to the techniques and theories that have created them.

And lastly, what new forms of surveillance and control emerge when data is used not only to describe the past and present, but also to predict the future? Does this promote, as Trevor Barnes (2015, 300) argues, an inherently conservative world that is "stuck with what is rather than what should be"? As algorithms like GFT, which is one of many efforts to use data to predict disease (Butler 2013), are increasingly incorporated into public health and

hospital frameworks, the under- or over-prediction of disease has very real consequences for the allocation of resources and care (Crawford 2013). The allocation of money to particular causes means the lack of funds for other causes—and consequently, to particular groups of people. Big data, ultimately, is political. It entails new forms of biopolitics (Lakoff 2015), new forms of surveillance and control, and fuels new economies of exchange, as incomplete "found" data sets fuel the desire to collect newer, bigger sources of information (Caduff 2014). Thus, the parable of GFT raises important ontological and political questions about the kinds of knowledge practices big data promotes, and about the uneven effects it has on society.

Toward an Anthropology of Big Data

Investigations of "data" and its associated objects have become increasingly central to the field of STS over the last several decades. Scholars have examined the production of scientific images (Daston and Galison 2007; Halpern 2015) and how they come to have historically and culturally specific meanings (Dumit 2003; Beaulieu 2001, 2002). They have also explored the history of bioinformatics and biological computing (Stevens 2013; November 2012), the making of databases (Leonelli 2012), and the rendering of models and visualizations (Myers 2015; Keller 2000; Morgan and Morrison 1999). Less work, however, has explored how the large-scale, complex, and distributed practices of big data are made and come to affect society. What is meant by passing references to "statistics" and "data"? What technologies, techniques, and practices do these signifiers of the dense world of contemporary computation and quantification entail?

In order to open up the black box of big data, I argue that we should expand our scholarly focus beyond the apps and graphs commonly encountered in websites and publication, to encompass the various technologies, people, institutions, and approaches to scientific work through which researchers give meaning and value to data. Digging deeper into the polished end-results of big data, to examine the social processes that are involved in the production, manipulation, and use of data, foregrounds the forms of expertise and politics that enable data to become meaningful in particular ways and contexts. It also shows how data are not just transient digital objects, but are also tied to material infrastructures and networks (Carruth 2014; Starosielski 2015).

Focusing on data as a set of practices, which invoke particular ways of engaging with and understanding the world, draws on a long history of social

scientific work on the social and cultural dimensions of science. It takes inspiration from the work of anthropologists in the 1980s, which critiqued the dominance of Western biomedicine and its claim that scientific knowledge had universal meaning, use, and effects on the body (Lock 1995; Rapp 1999; Good 1994; Kleinman 1982). Similarly, it takes inspiration from the sociologists and historians who turned their attention to scientific laboratories, seeking to unravel the common portrayal of science as an objective form of knowledge, and emphasizing that science, like other forms of knowledge, constitutes particular ways of describing and knowing the world (Latour and Woolgar 1986; Daston and Galison 2007).

All of these scholars were responding to what they saw as the urgent societal issues of the twentieth century: positivism and the rise of "big science" following the second world war, the devaluation of non-Western modes of knowledge with the spread of globalization, and rising health disparities and discrimination against women and minority groups. Motivated by similar concerns about big data—how it can exacerbate existing inequalities (Crawford 2013), how it can lead to discrimination (Dougherty 2015; Miller 2015), and how it can constrain the variability and spontaneity of human experience (see Note to Self 2015)—I want to advocate for an "anthropology of big data" that can unfold the politics that get built into seemingly objective bundles of data. Such an approach moves beyond the commonly discussed issues of data transparency and privacy, and instead questions the norms and values that get built into—and can seemingly disappear within—the digital infrastructures of twenty-first century society.

Although they may seem objective, data are never unbiased, neutral, or raw (Gitelman 2013; Räsänen and Nyce 2013). Giving meaning to data entails choices and judgments—from their collection, to their cleaning, to their interpretation—about how to carry out experiments or do research "correctly." As researchers use strings of code and algorithms to analyze data, seemingly simple decisions are transformed into evaluative statements about how and to what ends knowledge should be produced. As Amade M'Charek (2005, 179) has written, "politics get built into standardized technologies and laboratory routines." The world of big data is pervaded by a "digital objectivity" (Beaulieu 2001, 2004) that places value on statistical calculations and quantitative forms of knowledge, and reflects particular "epistemic virtues" about how knowledge should be produced (Daston and Galison 2007).

Take, for example, IBM's recent efforts to use the supercomputer Watson—which became famous in 2011 for beating a human being at the game show Jeopardy—to help doctors cope with large volumes of data being generated in healthcare. One of Watson's main commercial applications is health analytics:

the synthesis of data from scientific papers and patient charts, in the search for patterns and insights (IBM 2015). In collaboration with well-known institutions like the Mayo Clinic and the Memorial Sloan Kettering Cancer Center, Watson aims to "dramatically improve the ability of doctors, researchers and insurers to surface new insights from all the personal health data being created daily" (IBM 2015).

But in asserting that technology can move beyond the capacity of human beings—who can only absorb a certain amount of information, and who are prone to bias based on the information and contexts immediately available to them—Watson is *political*. It places value on the computational capacities of computers over the more interpretive abilities of physicians, and in doing so, changes the way people are diagnosed and treated. In this sense, big data does not merely reflect back an objective reality of disease, but rather intervenes into healthcare to create particular a version of reality, one populated by apps and cost-benefit analyses. Big data naturalizes particular ways of interacting with the world, producing bodies and populations whose health is judged by how well their measurements conform to the statistical norm, rather than by how they qualitatively feel about their experiences. In doing so, big data privileges the average health outcomes that can be measured in large datasets, while it also silences unique or "abnormal" experiences or needs.

Ultimately, at stake in this process are the various forms of expertise and power that are involved in making and making sense of data. These are the politics of data: the ways and extent to which data affects society is determined by the institutions and people who are in positions of power to determine how and to what end knowledge gets produced. The effects of data, like globalization (Appadurai 1996), are uneven and local, favoring the well-being of certain populations over others. Consider, for example, the story of Streetbump, a crowd-sourced app developed by the City of Boston to detect potholes in roads. In a city notorious for the poor quality of its roads, the app uses sensor data from smartphones to detect road condition data, with the ultimate goal of fixing previously undetected road hazards. But as Kate Crawford (2013) has written, Steetbump also had the unfortunate side effect of polarizing pothole data toward more affluent neighborhoods, where people were more likely to own expensive smartphones with sensing capabilities. Cell phone ownership—or the use of digital technology, more broadly—is not even among all demographic groups (Zickuhr and Smith 2012). As a result, digital innovations do not represent or reach everyone equally, polarizing interventions and services toward certain groups of people.

The (Big) Data Bind

Data, it would seem, is becoming an inescapable aspect of modern society and healthcare. It is not only growing in volume, but also social acceptance, as people increasingly rely on computers and cell phones for everyday tasks—even speech and music (Hern 2016). As the world is increasingly oriented around data, society is faced with increasingly complex questions about whether data is good or bad, or about whether digital innovations enhance or damage our well-being. There is the increasing sense, among the scientists and healthcare practitioners I have worked with, that collecting more and more data does not simply lead to more knowledge of biology, or to better health outcomes. Biomedicine is always trying to develop new algorithms, new pieces of software, new databases, new computational platforms, as a means to study disease. But the leap from data to knowledge is neither straightforward nor easy.

Consequently, how can we engage with data in a critical way, one which recognizes that, like all technologies, data has positive and negative effects on society? What kinds of expertise are necessary to make sense of the ever-growing "deluge" of data? Why do researchers persist in developing new tools, when they struggle to make sense of the data they already have? Does collecting more data transcend old ways of thinking about the body, or simply reinforce existing paradigms? Is society investing in the right resources to address the health of individuals and populations across the globe? Data's practices, questions, and effects on society are far from settled.

To examine the complex, ambivalent effects of data on the world, I argue that we should attend to "data binds," to the choices and dilemmas researchers face as they analyze and use data. In data binds, researchers try to make particular aspects of the complex, messy, and unpredictable world—be it the human body, the earth's climate, global inequality—stable and coherent through data. They measure inputs and outputs, turn social problems into numbers, and organizing findings into matrices and databases. Big data gives the illusion of control, but ultimately, many aspects of the world elude quantification. This is the data bind: in order to study complex phenomena, researchers must order and simplify them. But in the process, complex phenomena remain slippery and elusive.

Modern research, through its roots in the scientific method, has always encouraged researchers to question the limits of and assumptions within their data. Big data, however, intensifies the quandaries of research, by binding knowledge workers into a political and economic system of data, where

the production and use of data is increasingly connected to commercial needs and economic forces. Researchers are encouraged to produce certain kinds of outputs—quantitative findings, publications in ranked journals, outputs that are commercially relevant—limiting their abilities to study complex phenomena with alternative methods or inquiries. As they work with big data, researchers are continually presented by data binds of varying kinds and intensities, as the objects they attempt to study elude quantification.

My use of the term "data bind" draws on the notion of the "double bind," which was first developed by social scientist Gregory Bateson in the 1950s, and which has been more recently introduced into the social studies of science literature by anthropologist Kim Fortun. In *Advocacy After Bhopal* (2001), Fortun explores how "enunciatory communities" are called into action in the aftermath of the Bhopal gas disaster. She argues that "double binds," "situations that create dual obligations that are related, are of equal value, and yet are incongruent with one another" (Fortun 2001, 13), give rise to social groupings that are never stable, and are always negotiating compromises around competing needs. Although the term is only discussed in the introduction to her book, for Fortun, double binds are a way to look at the complexities of social forces, showing how they often cannot be resolved, and how they invoke particular decisions and values (see also Fortun and Fortun 2005).

Here it is useful to return to Gregory Bateson's original formulation of the double bind, which he developed through his linguistic work on schizophrenia (Bateson et al. 1956a, b; 1963). Bateson coined the term double bind as a way to think about the complexity of communication during the treatment of mental illness. In his formulation, a double bind might occur when a therapist says to a patient: "I want you to disobey me." In such cases, to obey is to disobey, and to disobey is to obey, such that no matter what a person does, "he can't win" (Bateson et al. 1956b). For Bateson, double binds are not only social situations, but also forms of social control, as they are used strategically to exert control without open coercion. In the complex social milieu of schizophrenia, double binds emerge as an important framework for asking how paradoxes are imposed, who imposes them, and how people navigate them.

Drawing on Fortun and Bateson's work, the notion of the data bind emphasizes the multivalent, complex binds researchers confront as they use data to ask and answer questions about the world. Referring to contemporary paradoxes as "data binds" emphasizes how conflicting obligations are multiple and

evoke many values simultaneously. In the world of metabolic research and the Phenome Centre mentioned at the beginning of this chapter, data binds happen when researchers develop computational methods to embrace the complexity of metabolism over space, time, and health. They render metabolism visible through thousands of molecular measurements, which quantify diet, microbial communities, and the effects of environmental toxins. But because researchers are constrained by numerous sociotechnical problems, they can never make metabolism fully stable or legible. It is impossible to measure everything, so researchers have to make choices: about which aspects of metabolism to render visible, and which to render invisible. They need, in other words, highly stabilized and formalized computational methods like software and statistics, in order to deal with the multicausal and dynamic nature of metabolism.

But this is the tension: in order to study metabolism, researchers have to freeze it. In order to embrace the complexity of disease, they have to simplify it. Faced with large datasets that are impossible to interpret with the naked eye, or by using intuition, researchers turn to statistical techniques to explore the complexity of their data. But, in doing so, they also use these same statistical techniques to reduce and contain complexity. In the search for metabolic phenotypes, researchers try to quantify the statistical boundaries between states of health and disease, but in doing so, they struggle to understand the complex processes underlying cancer relapse, or the individual and environmental variability of drug response.

Big data, in this way, is a self-fulfilling prophecy. It drives the collection of more and more data (see Caduff 2014), co-producing knowledge practices and infrastructures. But data binds are not just a tool for examining the strategic ways in which complexity is frozen and rendered quantifiable. Ultimately, they are also a tool for examining the social forces and values that moments of tension render legible. How, in other words, does quantification entail choices—some overt, some not—about what aspects of life to make known through quantification? How do these choices come about in the first place, and how do they affect human experiences?

Ultimately, data binds are a means to interrogate the norms, values, and politics of big data, the forces that shape how and to what ends knowledge gets produced in the twenty-first century. Examining the moments in which researchers finds themselves in data binds—and the ways in which they articulate and navigate them—provides insight into the contexts in which data binds arise, and also the values and norms of data-intensive science. Examining the strategic ways in which researchers freeze and reify complexity, simplifying it, shows how certain aspects of the world are being made legible. Thinking

about how data binds are imposed, and what people and institutions impose on them, reveals how data binds are political, and how they reflect mechanisms of social change and control. It shows how the quantification of various phenomena entails choices—some overt, some not—about which aspects to make visible, and which to silence.

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29

Personalised and Precision Medicine: What Kind of Society Does It Take?

Barbara Prainsack

Introduction: From Personalised to Precision Medicine

A social scientist studying personalised medicine will meet people along her way who remind her that medicine has always been personalised. Medicine has always revolved around individual patients, she will be told, whose illnesses were seen as expressions of their unique life situations, environments, and physical and mental characteristics. Moreover, throughout most of human history, those diagnosing and treating patients have considered these patients' personal characteristics and needs. The creation of institutions that treated standardised symptoms rather than individual patients—a process that the sociologist Nicholas Jewson famously described as 'the disappearance of the sick-man' (Jewson 1976)—is an invention of modern times. With the proliferation of technologies for molecular diagnosis and digital algorithms, the social scientist's well-meaning colleagues will argue, medicine is now less personalised than it has ever been.

In a way they are right. If we understand personalised medicine as a way of practising medicine that foregrounds the specific characteristics and needs of individual patients, instead of framing them as 'cases' to be treated according to supposedly objectively measurable symptoms that indicate treatments, then medicine has indeed become less personal (see also Vogt et al. 2014).

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Following this rationale, it could be argued that medicine was at its least personal in the late 1990s and early 2000s, when—in the aftermath of the Human Genome Project—people started to be clustered together in different groups on the basis of genetic markers that they shared in common. In many areas of medicine, this method of stratification is still practised. It is driven by the belief that genetic markers reflect characteristics that determine diagnostic categories or treatment success. Those who have celebrated this practice as the advent of personalised medicine have become an easy target of critics pointing out that group stratification is not, in fact, making medicine very personal (Hedgecoe 2004; Tutton 2014).

What these critics ignore, however, is that many of those who refer to the matching of drug treatments to genetic markers of patients as 'personalised medicine' see this as the first step of a much longer path. For them, personalised medicine is a process that has only just started. In the late 1990s and early 2000s, scientists hoped that genetic markers would be a key to understanding individual differences in health and disease more broadly. The Human Genome Project had not delivered the cures and treatments that its most optimistic supporters had expected; but it had yielded important insights into the *relevance* of genetic differences in the aetiology of diseases and the responses to different treatments. Findings from the Human Genome Project did away with the idea that one size fits all. In its aftermath scientists and policy makers argued that individual difference should inform not only treatment but also diagnosis, monitoring, and prevention. What was left to do was to figure out how to measure, interpret, and translate individual differences into decisions in the clinic.

In the late 1990s, looking at genetic markers that corresponded with a type of response, or non-response, to a drug seemed the logical way forward. Adverse drug reactions became one of the biggest killers in some countries (e.g. Lazarou et al. 1998; Cressey 2008; see also Onakpoya et al. 2016). If a person's genetic markers suggested that her version of an enzyme crucial for metabolising a certain drug would not be effective, then it would be beneficial to spare her the side effects. Not treating everybody in the same way but instead testing whether a specific patient had a characteristic on the basis of which she should be included or excluded from a particular diagnosis or intervention, in this light, was indeed a step towards greater personalisation.

In the early 2000s, with the ongoing advance of technologies that made the generation of molecular and other information cheaper and faster, the notion of personalised medicine started to broaden. Henrik Vogt et al. (2016) diagnose a shift towards a more systemic and *holistic* understanding of health and disease. At the same time, they argue:

> [r]ather than representing a medical holism associated with basic humanistic ideas, we find a *technoscientific holism* resulting from altered technological and theoretical circumstances in biology. We argue that this holism, which is aimed at disease prevention and health optimization, points towards an expanded form of medicalization, which we call '*holistic medicalization*': Each person's whole life process is defined in biomedical, technoscientific terms as quantifiable and controllable and underlain [by] a regime of medical control that is holistic in that it is *all-encompassing*. It is directed at all levels of functioning, from the molecular to the social, continual throughout life and aimed at managing the whole continuum from cure of disease to optimization of health. (Vogt et al. 2016, 307; see also Clarke et al. 2010)

What Vogt and colleagues describe is the result of a number of epistemic, technological and practical shifts that the idea of personalisation in medicine has been involved in since the late 1990s—so much so that a new term, 'precision medicine', is now increasingly used to describe the stratification of people on the basis of individual characteristics (NAS 2011; Robinson 2012; Weber et al. 2014). These shifts are (1) the transition from variation to difference; (2) from thick data to big data; (3) from episodic to continuous medicine; (4) from reactive to predictive medicine, and (5) the collapse of the distinction between research and treatment. I will discuss each of them in turn and spell out what notions of people and society they entail. I will conclude by arguing that what all these shifts have in common is that they change how patients are understood, seen, and treated: Their physical bodies, lives, and practices are datafied and digitised, creating new divisions. Moreover, personalised and 'precision' medicine also change what is expected from patients: Working towards the gold standard of precision medicine requires the cooperation of patients who contribute information, time, and self-monitoring efforts. In order to benefit from precision medicine, they need to become 'activated patients' (Britnell 2015) who contribute not only effort and time but also data and information to their own healthcare.

From Human Variation to Radical Difference[1]

A decade ago, Steven Epstein described the battle for adequate representation of different groups in medical research in the United States in his seminal book *Inclusion: The Politics of Difference in Medical Research* (2007). But which were the groups that should be represented adequately, according to what characteristics should these groups be defined? The answer to this question lay in the use of traditional demographic and epidemiological characteristics such

as race, age, and gender. Because of how deeply these labels were engrained in the social and political fabric of our societies, they seemed to be a natural choice to classify people (see also Prainsack 2007, 2015a). They have been used for population census and in epidemiology. They have been very useful for governments also because they are relatively stable: Labels such as race or gender typically stick to people throughout their lives.

In the second half of the twentieth century, genetics was imbued with the hope of reinventing traditional demographic categories such as race and gender/sex. In Gisli Palsson's words, '"[o]bvious" phenotypic traits such as skin colour were now seen as surface differences providing trivial if not misleading information about the deeper realities of the human body' (Pálsson 2007, 258; see also Skinner 2006; Waldby 2000). The hope was thus that genetics would get to the bottom of these categories, to capture their essence; DNA assumed the role of a 'truth machine' (Lynch et al. 2010) producing evidence on the true nature of human life.

Personalised medicine makes a different promise. It undertakes to overcome superficial and stable categories in the stratification of people. During the later phases of the Human Genome Project, individual differences at the level of genetic markers were seen as the key to stratification, and racial and ethnic labels were seen as acceptable proxies for groups within which certain markers were particularly prevalent (Hedgecoe 2004). Throughout the 2000s, this reductionist and mechanistic approach gradually gave way to what Silke Schicktanz, Gabriele Werner-Felmayer and I called 'normative complexity' (Prainsack et al. 2014, 8), namely the programmatic rejection of linear relationships. The understandings of the role of genetic factors in disease aetiology became more nuanced; scientists obtained a better understanding of the many factors that regulate and modulate the workings of a person's genetic 'hardware'. If the human body has become an orchestra, then among all the factors that account for health and disease, the DNA sequence is now seen as, at most, the first violin; it is no longer regarded as the conductor (see also Noble 2008). Helped by advances in the fields of other '-omics' (large-scale and data-rich research into the function and relationships between different types of molecules in the cell of an organism) as well as new opportunities to integrate different types of information, personalised medicine has thus started to be used much more broadly to refer to the adjustment of prevention, diagnosis, and treatment to specific characteristics of patients (ESF 2012; PerMed Consortium 2015). Many authors now include also the consideration of non-molecular differences between people in their definition of personalisation (e.g. NAS 2011; Desmond-Hellmann 2012; Topol 2012; Özdemir et al. 2013). Some even go as far as including credit card purchases

and social media postings in the types of data that they would like to see integrated at the level of individuals (e.g. Weber et al. 2014).

What we see here is not only a shift from presumably stable genetic and genomic information to the inclusion of wider ranges of malleable or dynamic data for the purpose of personalising medicine but also a shift in the meaning of difference (Meloni and Testa 2014; Prainsack 2015a; Mansfield and Guthman 2014; Meloni 2016). Personalised medicine assumes that people are different at so many levels that their diseases cannot be subsumed under shared labels. This means, as argued in one of the most prominent iterations of 'precision' medicine by the US National Academies of Science (NAS), that traditional symptom-based disease taxonomies need to be replaced by data-rich characterisations of individuals at various stages of health and disease (NAS 2011). Within this vision, there are no more shared diseases, only individually unique manifestations of different configurations of characteristics and symptoms.[2]

One of the implications of this stance, which I have called 'radical difference' (Prainsack 2015a), is the need to develop new methods and methodologies to produce evidence. If it is no longer acceptable to lump people together into large groups, then the randomised controlled trial has ceased to be a suitable gold standard for the production of evidence to guide medical practice. Calls for new ways to complement—and in some contexts, replace—traditional group-based clinical studies include the computerised simulation of biological processes (*in silico* modelling) or in-depth $N = 1$ studies (ESF 2012). What the proponents of personalised medicine demand, here, is not that societal groups should be represented more accurately (read: proportionally) in clinical trials but that the idea of putting people together in groups is given up altogether. One articulation of this vision is that every person's health data would be integrated into something resembling a 'Google maps' feature. It would combine static (e.g. gene sequence) and dynamic (e.g. gene expression, lifestyle, etc.) information (NAS 2011; Topol 2015) to render people's health information open to navigation.

But if every person is her own standard, how do we know when she is ill? How do we know when a person's functioning ceases to be 'normal' and how should we even define normal in a context of radical difference? One part of the answer lies in the increasing push towards continuous data collection from patients, also when they do not have a problem. This would establish a baseline of their own individual physiology (Ausiello 2013). Another answer to this question is that individual health information, even if it is not directly compared to any aggregate population or group-based standard, always only makes sense in relation to other people. First of all, despite the rhetoric around radical

difference in personalised medicine, routine clinical practice still operates with population averages. Physicians still refer to 'normal ranges' of blood glucose levels in determining whether or not we need to be concerned, and many performance tests still compare us to groups that are defined according to old epidemiological categories. But even in cases where there is no fixed standard that determines what 'normal' is in terms of a group average, seeing the results of others helps us to put our own results in perspective. We need to see the status of others to know what our own status means. Self-trackers use web-based applications to compare their performance to the aggregate data of 'similar' others (e.g. other women in my age range or at similar fitness level). Patients using the CureTogether (curetogether.com) platform look at what treatments work for other patients who are similar to them—which means that they are of the same gender, of similar age, and have similar comorbidities and symptoms.

In other words, within personalised medicine, groups are still relevant, but they may be less clearly visible, and the criteria according to which groups are defined may be entirely implicit. Especially in cases where the group that a person's data are compared to consists of users of a specific service that is not accessible to many people, it is likely that we end up comparing ourselves to others who are similar to us. Here, the 'filter bubble' effect—the phenomenon that information from our activities is used to 'personalise' what we see, so that two people searching for the same term online, for example, get different results (Pariser 2012; Prainsack 2015a)—enters medicine. The effect of this is that existing inequalities and lines of segregation are reinforced, but in much more silent ways than previously. If the only difference that we see between ourselves and others lies in the severity and combination of our migraine symptoms, or in our genetic predisposition to respond well to blood thinners, then it may remain unnoticed that 80 per cent of us hold a university degree. Here, social inequalities are reiterated by moving people on the other end of the social spectrum out of sight—they become, quite literally, 'missing bodies' (Casper and Moore 2009). Such a personalised medicine operating with radical difference thus presumes and fosters a society comprising of individuals who make their dependencies on, and relations to, others invisible. These 'missing bodies' are still part of society, but they stop to be part of the collective biology of a society.

From Thick to Big Data

As pointed out at the beginning of this chapter, practitioners of medicine have always taken into consideration the specific personal characteristics of their patients; and patients themselves have also brought these into medical deci-

sion-making. These characteristics have taken the form of unstructured, 'meaning-full' information on people's bodies, lives, and feelings that did not lend themselves easily to de-contextualisation and standardisation—we could call it 'thick data' (see also Vogt et al. 2014). But contemporary iterations of personalised medicine require different types of data. The 'big data' paradigm, which is one of the epistemic drivers of personalised and 'precision' medicine, describes both a paradigm and an approach to making predictions. Internet governance experts Viktor Mayer-Schönberger and Kenneth Cukier describe the key tenet of the big data paradigm as a shift away from the 'age-old search for causality':

> As humans we have been conditioned to look for causes …. In a big-data world, by contrast, we won't have to be fixated on causality; instead we can discover patterns and correlations in the data that offer us novel and invaluable insights. The correlations may not tell us precisely why something is happening, but they alert us *that* it is happening. (Mayer-Schönberger and Cukier 2013, 14)

According to these authors, when working with small datasets, it is important for every single data point to be correct. In the context of big data, by contrast, the larger scale makes up for the inaccuracies in single data points. If, for example, a person measured their temperature once a day to obtain a reliable fever curve over the course of a week, it would be important for this measurement to be accurate. If they measured their temperature every three seconds, then it would be practically irrelevant if some of these measurements were wrong. While this example pertains to data taken from a single person, the principle, so Mayer-Schönberger and Cukier (2013, 14) argue, is also applicable to population-level data: 'If millions of electronic medical records reveal that cancer sufferers who take a certain combination of aspirin and orange juice see their disease go into remission, then the exact cause for the improvement in health may be less important than the fact that they lived'. Here, the call to act upon association is not a compromise—because we have not found out what the causal factors are—but it is a matter of principle. People give up the claim to even attempt to understand the dynamics underlying a relationship between two factors; knowing that the association is strong enough is sufficient. In an ironic turn that could be seen as a version of intelligent design, humans surrender their quest to understand the underlying causes assuming that only a higher entity—may it be God, or an algorithm—sees the 'real' causes.

The idea that big data will create better evidence than ever before and lead to unprecedented advances forms an important part of the convictions of

many proponents of personalised and 'precision' medicine. For some visionaries, the temptation of using big data methods and epistemologies is so strong that they see the comprehensive surveillance of people—not only patients—as the new gold standard of medicine (see below, section 'From episodic to continuous medicine'). For them, big data is only truly big if N = everything (Harford 2014; see also Green and Vogt 2016).

What many big data enthusiasts in the context of personalised medicine seem to be untroubled by is that data-mining typically provides probabilistic predictions. To stick with the example of aspirin and orange juice used by Cukier and Mayer-Schönberger earlier: if an analysis of data from 200,000 cancer patients who consumed aspirin and drank orange juice on a daily basis showed that 80 per cent of them went into remission, a doctor would not know whether her specific patient would personally benefit from aspirin and orange juice. This patient could be like one of the 20 per cent of people in the study who did not benefit from this combination. If we do not understand what causes a particular effect, we cannot tell whether something will work for a particular patient or not, regardless of the size of the dataset.

Somewhat ironically, the idea that data-rich medicine will contribute to better health outcomes is accompanied by the fear that we will not be able to manage all the data that we have generated and collected. Metaphors such as 'lake', 'ocean', 'tsunami', or 'drowning' make frequent appearances in discussions on data-driven medicine, and big data more broadly (Lareau 2012; Khoury et al. 2013, 513; Roski et al. 2014, 1117). Such watery metaphors, as Deborah Lupton pointed out, refer to 'uncontrollable entit[ies] possessing great physical power […] as well as their unpredictability and the difficulty of control and containment' (Lupton 2013). Oceanic metaphors in particular also convey the smallness of humans in the face of an awesome force.

Such metaphors suggest that no matter how hard we try, we will never be able to control the flow of data completely. As history has shown, not even the most sophisticated methods and technologies can effectively prevent floods, tsunamis, or droughts. Comparisons of big data to water and other natural forces or resources thus convey to us that data have a life of their own and that their power is bigger than ours. Humans can alleviate the dangers and mitigate the harms of oceans and tsunamis, but we can never fully master them. Moreover, the portrayal of data as natural resources suggests that humans rely on them for their survival (see also Dean 2016).

The metaphors we use are not merely a matter of semantics. As George Lakoff and Mark Johnson (1998) famously argued, the metaphors we use structure our experience of the world. For the field of medicine, such portrayals of the intrinsic and natural 'force' of data have several implications. First, because the very function of medicine is to alleviate or even cure people's suf-

fering, metaphors that highlight the inexorable intrinsic power of data can, in turn, articulate a moral obligation for individuals to harness this power in order to reap clinical benefits. In fact, an increasing number of scholars and activists refer to Article 15 of the *International Covenant on Economic, Social and Cultural Rights* (UN 1966), which defines a human right to 'enjoy the benefits of scientific progress and its applications'. The existence of such a right begs a number of questions: if citizens have a right to enjoy the benefits of scientific research, does this mean that there is an obligation for research to 'open up' and use all available datasets in medical research institutions? Do we also need to 'open' clinical data? Do people have a moral, if not a legal, obligation to allow others to use their personal health information if used for research with likely public benefit? And what does 'opening datasets' mean in this context, does it mean that we need to make these data available to bona fide researchers or to everybody? How do we address the problem that equal access to datasets for everybody makes corporate actors that are already very powerful even stronger (Taylor 2014)?

While we are still deliberating these questions, comparisons of big data with natural resources, oceans, and water continue to suggest that once large datasets are available, we need to mine and analyse them in order to draw conclusions for the treatment of individual patients. We may be unlikely, at this point, to wonder about what meaning these datasets have in a wider context and whether we should have created them in the first place.

From Episodic to Continuous Medicine

Current visions of personalised and precision medicine rest upon the idea of data-intense characterisations of individuals at different stages of health and disease in the course of their lifetime (NAS 2011; ESF 2012). However, as visionaries of precision medicine argue, we are still at a point where most data about people are generated when they have a problem. But what happens in-between doctor's appointments? What happens when people do not experience problems? These visionaries believe that we should move beyond what they call reactive and symptomatic medicine and move towards a situation where patients are monitored continuously and as comprehensively as possible (Ausiello 2013; Agus 2016). The result would be health data maps for individual patients. These data maps would allow probabilistic predictions about people's treatment courses and treatment response not only on the basis of other people's data but also their own. People's data during stages of disease could be compared to their data during phases of good health.[3] In this vision, people are literally becoming their own control group. Such a 'benevolent' surveillance medicine seeks to translate the bodies and lives of

individual patients, not only of populations, into datasets that can be mined, analysed, and used for prediction—by healthcare providers, insurers, patients, scoring bureaus, technology developers, and many other actors. As we have seen with Cukier and Mayer-Schönberger's example of taking aspirin and orange juice to battle cancer, the fact that such prediction is merely probabilistic tends to get lost somewhere on the way to implementation. In other fields where predictive analytics is used, such as credit or consumer scoring, it does not matter that 'predictions' are wrong in some cases as long as they are accurate in most. Leaving the considerable political and social dimensions of such a 'benevolent' digital surveillance medicine aside (I discuss these elsewhere; Prainsack 2015b; Prainsack forthcoming), even its scientific assumptions alone problematic. As David Spiegelhalter reminds us, collecting lots of data does not automatically compensate for poor quality. In Spiegelhalter's words, '[t]here are a lot of small data problems that occur in big data. … They don't disappear because you've got lots of the stuff. They get worse' (cf. Harford 2014, 29). Also communication studies scholar Gina Neff (2013) has argued for years that 'big data won't cure us': in order to overcome the trap of collecting data merely for the sake of collecting data, we also need to pay more attention to the 'social interoperability' of different types of data. This means that we need to try to make different meanings and utilities of different data for different actors in the medical domain more explicit. And we need to obtain a better understanding of the 'differences in how people generate, use and even talk about data' (Neff 2013, 119).

Last but not least, the ideal of 'continuous medicine' with its focus on digital data collection ignores that human contact alone can go a long way in making people feel better. Some of the most effective solutions for people's health problems are thus the simplest, low-tech ones: fight social isolation. In this sense, personalisation contributes to a society where the value of human contact, social interaction, and in fact solidarity is neglected in favour of the currency of big data and continuous (self-)surveillance. Ironically, at a time when human biology is conceptualised as a complex system of relations, the relationality of a patient's being in the world is denied in favour of representing her body and behaviour in digital data.

From Reactive to Predictive Medicine

Closely related to the previous shift in which medicine moves from episodic intervention to continuous data capture is the shift from reactive to predictive medicine. Whereas the former concerns the creation of longitudinal datasets that are supposed to be as comprehensive and continuous as possible (i.e. to

fill in the alleged 'gaps' in data about people in between the time points when they need medical attention), the latter speaks to how we move on that timeline. The very notion of predictive medicine implies that medicine has so far, literally, been stuck in the past: we attended to problems after they have appeared. What may sound like a truism to many of us, many visionaries of personalised and 'precision' medicine want to change. Predictive medicine seeks to move us forward by enabling us to use data from our past and our present to act upon and to anticipate the future: both to know what will happen in the future and to change it. This is supposed to be done by 'looking' (as much as computer software looks at anything) at historic data on various aspects of our life—the ways in which our genes are expressed, our blood pressure, our sleeping patterns—to discern patterns. As soon as our current data start to behave out of the ordinary (i.e. contrary to the pattern), the software analysing our data doubles would ring an alarm bell. This is what precision medicine visionaries mean when they say that our smart phones will know that we will get a heart attack before we do (Hein 2015).

The Marriage of Research and Treatment

The distinction between research and treatment originates in the beginning of laboratory medicine in the late nineteenth century. At that time, the idea that medical practice should be guided by systematically collected and evaluated evidence—preferably from controlled experiments—became an important characteristic of Western medicine. This does not mean, however, that the goal of medical practice based on experimental scientific evidence has been reached: today, less than one-fifth (19 per cent) of published guidelines are based on randomised controlled trials, and such evidence 'may not generalize well in many clinical situations' (Longhurst et al. 2014, 1230; see also Zwolsman et al. 2013). Nevertheless, it has remained the gold standard of medicine. Personalised and precision medicine are changing this: as noted above, if clinicians and researchers take differences between people seriously, then they can no longer lump them together into large groups and assume that these differences do not matter. *In silico* modelling was already mentioned as one of the alternatives that is being proposed as an alternative to the randomised controlled trial; another one is to equip clinical decision-makers—human and non-human ones—with data from electronic health records, observational data, and 'deep phenotyping' data in lieu of evidence from randomised controlled trials (see also Robinson 2012; Jensen et al. 2012; Longhurst et al. 2014).

Greater use of observational instead of experimental study designs could be particularly helpful in gathering evidence on people who are typically underrepresented in randomised controlled trials. The introduction of a 'Green Button', namely a tool allowing clinicians to access aggregate clinical data from other patients similar to the one they are treating, could also help to make decision-making more evidence based in cases where there are few or no pertinent published studies (Longhurst et al. 2014, 1231). Such a Green Button approach would also provide an opportunity to include patient-centric parameters as part of health records into clinical decision-making as well. While there are several technical and other challenges that need to be overcome for observational studies on the basis of existing clinical data to be used for clinical decision-making, visionaries of personalised and precision medicine believe that they can be addressed (Longhurst et al. 2014, 1232).

Whereas medical research and practice have always been overlapping, this development is remarkable as it abolishes even the *ideal* of separating the two. The influential report by the US National Academies of Science *Toward Precision Medicine* programmatically called for the creation of a new disease taxonomy that serves the needs of medical practice and research at the same time (NAS 2011). While it may be tempting to dismisses this as a utopian plan that opinion leaders and policy makers seek to impose top down, such a dismissal would be rash: the boundary between clinical and research uses of health data is blurrier than ever before. Data collected for clinical care are already analysed to inform the care of other patients than those from whom they were collected, and this collapses the research and care distinction entirely (e.g. Bazelier et al. 2012). While there will always be differences between datasets that were collected primarily for research purposes and those that were collected for other purposes (in terms of how they are characterised and where they unfold their biggest utility) against the backdrop of personalised and precision medicine, any dataset is meant to be usable for both ends (see also Abernethy et al. 2014).

Conclusion: What Kind of Society Does Personalised Medicine Require?

In the early 2000s, Andrew Webster (2002) and Sarah Nettleton (2004) saw a new medical cosmology (Jewson 1976) of medicine emerging: medicine started to be seen as information. Drawing upon the work of Jos de Mul (1999), Nettleton in particular saw medicine as trying to become more syn-

thesisable, programmable, and manipulable. As the rhetoric in policy initiatives on personalised and precision medicine in consecutive years has shown, medicine indeed seeks to increasingly rely on evidence that carries these characteristics. Unstructured, narrative data and data that are not available in digital format, including patients' narratives, continue to play a role, but they do not slot easily into the data architectures that are currently built to enable data mining and automation (see also Hartzband and Groopman 2016). Not only has seemingly objective evidence replaced narrative knowledge and unstructured information as the legitimate representation of human experience (Nettleton 2004, 671), but a particular definition of what counts as evidence has crowded out the unstructured sharing of experience and narrative. A decade after Nettleton's analysis, and after the American biologist Leroy Hood had introduced the concept of 'P4 medicine', namely a vision of medicine that is predictive, preventive, personalised, and participatory (Weston and Hood 2004; Hood 2008), computer scientist Bruce Schatz predicted that medicine in the next decade and beyond will be characterised not by 4P but by 3M: monitor, measure, and manage (Schatz 2015, 222).

In this chapter I argued that current visions and iterations of personalised and precision medicine are underpinned by five important shifts: (1) the transition from variation to difference; (2) from thick data to big data; (3) from episodic to continuous medicine; (4) from reactive to predictive medicine, and (5) the collapse of the distinction between research and treatment. All of these shifts have one thing in common: they change how patients are understood, seen, and treated. Patients' physical bodies are increasingly translated into datasets that can be manipulated and navigated; and also their lives and social practices are seen as something that could or should be 'datafied' (Mayer-Schönberger and Cukier 2013). Today's personalised and 'precision' medicine is thus not reductionist in the same way as the genetic medicine at the end of the last century was seen to be. They are, as Vogt et al. (2016) helpfully observed, underpinned by a new kind of holism. This new holism 'sees' some aspects of our bodies and lives better and in a more nuanced way than before, but it leaves out those aspects of our bodies and lives that do not lend themselves to easily to datafication and digitisation. Traditional differences between psyche and soma, between a person's body and her environment, and between physiology and pathology lose relevance compared to the increasingly impactful difference between what can be datafied and what cannot (and related distinctions that indicate the ease with which data are computable: structured vs. unstructured data, digital vs. non-digital data, etc.). Similarly, 'big data' methodologies and epistemologies claim power and effec-

tiveness in any discipline. The crucial difference here is no longer the one between social and life sciences but between 'data science' and everything else.

This development towards a unified scientific method to study human beings is problematic for several reasons. Within personalised and precision medicine specifically, it strengthens existing or even creates new ones: divisions between those who can and cannot be seen digitally; those who are passive data transmitters and those who actively contribute information, analyse, and interpret; and divisions between those whose healthcare benefits from the new algorithmic analyses and decision aides and those who merely contribute the data for the benefit of others.

Personalised and precision medicine also change what is expected from patients and healthcare professionals. Regarding expectations from patients, surprisingly few of the policy papers and initiatives that call for greater personalisation in medicine and healthcare ask the question of where the data will come from. Instead we frequently encounter the assumption that 'empowered' patients will happily surrender themselves to comprehensive monitoring in the name of continuous medicine and will collect data on their smart phones, mood diaries, and wearable sensors to help personalise their healthcare. In current iterations of personalised and precision medicine, attention to difference is not a political choice. It is a scientific, personal, and also societal obligation. It is our moral duty—as medical professionals, as patients, or as healthy 'patients-in-waiting' (Timmermans and Buchbinder 2010) to know what is knowable about our own unique characteristics (see also Rose 1999, 2007; Cetina 2005; Clarke et al. 2010). And the data-rich characterisations of individuals at potentially any stage of health and disease make self-optimisation measurable in novel ways. By changing our diet or exercise levels, for example, our proteome profile may improve or deteriorate. By focusing not only on genomic data, which are stable and thus beyond our control, but by including characteristics that are seen as changeable by our behaviour, personalised medicine produces visible evidence of successful or failed self-optimisation.

Also healthcare professionals are affected by personalised medicine underpinned by data science epistemologies. Computational algorithms are becoming more important in medical practice, and it will be more difficult and riskier for healthcare professionals to ignore algorithmic decision 'aides'. Investments in machine learning will surpass investments in human learning. Despite the strong rhetoric of patient participation and shared decision-making, health professionals and patients alike will be objects and 'operators' within a system that is ruled by other entities not accountable to human beings.

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Notes

1. Part of this section draws upon Prainsack (2015a).
2. Mansfield and Guthman (2014) discern a very similar development in their analysis of epigenetic research: The idea of biological plasticity, which underpins epigenetics, is anti-determinist. Because environmental stimuli influence how a person's DNA is expressed, there cannot be one 'normal'. Difference is thus inscribed into the very concept of epigenetics. But, as Mansfield and Guthman show, this difference is not morally and politically neutral: Some 'variations' are treated as signs of damage and thus treated as a 'disruption' of desirable processes while others are not. That some population groups are much more likely to be exposed to 'damaging' stimuli than others is an issue that epigenetic epidemiology pays increasing attention to (Hanson et al. 2011).
3. Very tellingly, *Google* launched a digital health data platform called 'baseline' in 2014 (Levy 2014).

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30

Emergent Postgenomic Bodies and Their (Non)Scalable Environments

Megan Warin and Aryn Martin

While the term 'the environment' is a relatively recent invention,[1] many of the ideas the concept embodies have extensive histories in philosophy, sociology and the life sciences. As Pearce (2010) notes, 'before the mid-nineteenth century, the idea of a singular, abstract entity—the organism—interacting with another singular, abstract entity—the environment—was virtually unknown' (2010, 241). Organisms were thought to be influenced by external conditions such as air, sunlight and temperature, and these were referred to generically as the force of 'circumstances'. It was the British philosopher Herbert Spencer who introduced the singular term 'environment' in 1855, which not only replaced the plurality of external conditions or circumstances into the one concept but created 'organism-environment interaction' as a conceptual possibility (Pearce 2010, 242).

This concept of a singular environment was part of wider historical debates concerning nature and nurture. While we do not delve into this complex history of nature and nurture here (for a detailed history, see Tabery 2014), it is important to highlight that interaction between genetic and environmental influences was key to explanations put forward by British biologists and statisticians Fisher and Hogben, and developmental geneticists like Waddington.

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Genes and the environment were viewed as distinct, separable causes, interacting in a classic 'gene-environment interactionism' (Meloni 2016, 203–205).

Following the sharp critiques of the Human Genome Project for its reductionist, gene-centric focus and lack of explanatory power to account for non-genetic variance in bodies, health and disease, new understandings of the environment are arising in postgenomics. Rather than genetic mutations being unresponsive to the environment in the short term, postgenomic thinking explores how the environment comes into the body and modulates the genome in relatively short time frames (Landecker and Panofsky 2013, cited in Meloni 2016, 205). The genome is thus thought to be 'far more fluid and responsive to the environment than previously supposed' (Jablonka and Lamb 1995, 26).

Within this change, interactionist discourses based on separated sources of causality are no longer plausible (Meloni 2016, 206). Environments traverse and intertwine across diverse geographical locations and historical times and are characterized as microbiomal habitats, intrauterine, maternal and molecular landscapes. These porous and variegated environments complicate and even undermine classic gene-environment models, compelling a new view of the relationship between genes and environments.

Interpretations of the environment in postgenomics are multiple and conflicting. On the one hand, coming to terms with the limits of late twentieth-century gene-centrism heralds an openness: to extra-genomic and complex causality and to non-molecular disciplines (Pickersgill et al. 2013; Lamoreaux 2016). On the other hand, we see a further reductionism in some fields, where explanations are sought, and thought to be forthcoming, from more and more minute material locations inside the body (Mansfield 2012; Kenney and Muller 2016). Probably both are happening concurrently: the environment is becoming a more salient actor than gene-thinking allowed at the same time that it is becoming known in greater detail, molecularized (Niewohner 2011), miniaturized (Lock 2013, 2015) and digitalized (Meloni and Testa 2014).

In this chapter we focus on 'the environment' and how its 'incessant openness' (Rapp 2011, 669) challenges social scientists to critique their own ideological scaffolds, as well as those that are implicit in scientific and popular discourses. Social scientists are renewing calls to re-conceptualize the environment and asking questions such as: what does 'the environment' mean in the postgenomic age? (Weatherford et al. 2016); what kind of environment is epigenetics constructing? (Pickersgill et al. 2013); and what are the dangers, blind spots and empirical inaccuracies of privileging specific spatio-temporal locations, such as intrauterine environments? (Warin et al. 2015; Richardson 2015). Our goal is not to determine what 'the' environment is (as though that

is an answerable question) but to highlight the ways in which differing versions of the environment are enacted in different situations.

In troubling the concept of the environment, we—a medical/social anthropologist and a sociologist of science, both influenced by feminist philosophers—present two case studies from our fields of interest: developmental origins of health and disease (DOHaD)/nutritional epigenetics and maternal/fetal microchimerism. The first case study arises from author one's work with clinical literature, popular media (Zivkovic et al. 2010; Warin et al. 2012) and ongoing research relationships (2009 to present) with life course and epigenetic scientists at the University of Adelaide, South Australia. Material for the second case study comes from author two's decade-long research engagement with microchimerism research and researchers in the USA and Europe.

In our case studies, we demonstrate how the environment 'is a catchall for a very wide range of factors' (Mansfield and Guthman 2015, 8; c.f. Pickersgill et al. 2013), traveling across persons, time and space, through and into bodies and cells at molecular and metabolic levels. While both cases purport to bring the (social) environment into the (biological) body, we don't rely on spatial categories of the social and biological (or subject and object) in order to bring them back together. Rather than think of the categories inside/outside and gene/environment as inherently spatial (which implicitly entails boundaries and separation), and scalar (which implicitly entails singularity, universality and transferability), we explore the potential of examining *the emergent and unfinalizable relations* between phenomena as a way to tell a different story about the environment in postgenomics.

We draw upon the feminist philosopher and physicist Karen Barad's work (2001, 2007, 2014) to illuminate how we come to know things as sets of fixed differences. Women's reproductive bodies, for example, can too easily be inserted into dominant representations of the environment, conceptualized as containers (with edges, interiors and exteriors) in which other materials/persons (cells, fetuses) circulate and are held. In Barad's theory of intra-action (which we describe later in the chapter), the domains of interior and exterior lose their prior designations and boundaries are shifted, re-worked and re-articulated. We claim that intra-action may thus be a valuable frame for registering different versions of the environment as they emerge and are enacted in different postgenomic situations.

Our second major claim is that these different versions of the environment are not simply different scales of the same thing. The environment is not a stable entity that can be scaled. We propose that these insides and outsides of intertwining postgenomic environments are better understood as nonscalable. Here we take up Anna Tsing's (2012) anthropological critique of scale

which interrogates 'ideologies of scale' (2000, 347) and attends to the ways in which difference is erased and flattened in scale-making projects (2012). 'Scalability', she argues, 'banishes meaningful diversity, that is, diversity that might change things' (2015, 38). Implicit in our critique is a concern about overdrawn dividing lines and simplistic leaps to ground policy or morality on the presumption that environments are, in fact, scalable. What we present is thus not prescriptive but carefully attends to the lure in attempting to redefine or recapture a concept ('the environment') that has been split open, pointing to the complexity of unmaking and remaking boundaries and borders.

Different Versions of the Environment

What constitutes an environment is conceptualized and operationalized differently in different disciplines, research agendas and worldviews. Environments are made up of things with radically different properties, encompassing items as ontologically disparate as neurotransmitters, temperature and love. For example, the environment of a cell includes other cells as well as functionally significant proteins, nanovesicles, hormones, ions, cytokines and more. The spaces between cells, and indeed the other cells they come into contact with, matter to the survival and trajectory of a cell. As one medical geneticist told author two: 'any cell is going to die unless its environment is sending it little goodies in the way of cytokines to say "keep going".'[2] While cells were once thought of as autonomous building blocks, the totality of which makes an organism, a more ecological view is emerging, especially in the philosophy of biology (Tauber 2008).

In pregnancy, multiple environments are at play. At the tiny core, embryonic or fetal genes and cells have environments as described above, and the fetus has an amniotic and placental environment as well as a maternal environment.[3] And the pregnant woman's ambient environment becomes relevant to the developing being insider her. Entities that pass from the environment outside the woman to the 'maternal' environment are highly scrutinized and regulated (more so for those items she selects, like alcohol (Armstrong 2008), than those that are selected for her, like industrial toxins (Murphy 2006)).

A completely different version of the environment is traditionally found in the social sciences, and generally refers to the domains external to the skin of the body, to social and physical environments and exposures such as social networks, poverty, trauma/stressful life situations, famine, feasts, toxins and perceived individual behaviors such as nurturing, diet, sex and exercise (Shostack and Moinester 2015, 194). Shostak and Moinester suggest that this exogenous

environment is amenable to ecological analysis, in which neighborhood-level research focuses on social structures and social processes that shape population health (2015, 202).

The layperson's 'environment' is another thing still: imperiled nature, a quasi-personified entity central to contemporary morality and politics. As a descriptor, the word 'environment' points to not-X, but that which surrounds it. Moreover, when biologists or sociologists talk about X's environment, they usually mean the X (gene, cell, fetus, body) to be more concrete, more effable than its surrounding. When specific elements in an environment take on knowable material attributes, they sometimes cease to be understood as environment at all. In sum, the environment is multiple and nebulous, enrolled contextually to perform particular rhetorical and analytical work.

Despite this heterogeneity, we are in many ways hamstrung in any effort to follow the environments' protean movements by our own inherited legacies of different disciplinary boundaries (Gieryn 1999).[4] In spite of academic cautions to the contrary,[5] we continue to operate much of the time as though research and pedagogy can be carved up—disciplined—by simple spatial lines. These epistemological boundaries operate on frames of Cartesian dualism, in which the relative contributions of nature and nurture have historically been teased out and held apart. While life scientists and social scientists all agree that 'environmental contexts matter' (Olden et al. 2011, cited in Shostack and Moinester 2015, 195), questions of how these multiple environments might be defined or studied pose 'a central challenge in postgenomic knowledge production' (ibid, 202). As a precursor to our elaboration of Barad and Tsing's theories, we now turn to our two case studies and the ways in which they disrupt (yet are simultaneously drawn back into) the simple formulations of inside-outside relations and gene-environment interaction in women's reproductive lives.

Case 1: Environments in Nutritional Epigenetics and DOHaD

Fetal, maternal, neonatal and infant environments are key to major research paradigms that advance the theory that nutritional conditions during intrauterine development can have long-lasting and adverse health effects (such as type 2 diabetes and obesity) in adult life (Gluckman et al. 2005). Early ecological research in the 1980s by Barker and colleagues suggested that the developing fetus was especially vulnerable during critical, prenatal formative

periods and their development could be altered by poor maternal nutrition, with permanent consequences for the baby's physiology and metabolism (Barker and Osmond 1986). Barker's work was based on observation and epidemiological studies, and his hypothesis drew on associations derived from historical records. What was needed to further understand these 'maternal effects' on adult life was a mechanism. The rise of molecular genetics in the 1990s and a number of well-known studies (in particular, the Agouti mice experiment) provided a convergence point in that the molecular mechanisms (methylation) were identified as being nutritionally mediated through the diets of pregnant mice (Waterland and Jirtle 2003).

The hypothesis originally posited by Barker and colleagues has led to extensive research on the effects of fetal undernutrition, low birth weight and development of chronic metabolic disease. Through time, the 'Barker hypothesis' was subsumed into the thrifty phenotype hypothesis, fetal origins of adult disease and early origins and more recently has moved to a more general and contemporary umbrella theory of developmental origins of health and diseases (DOHaD) and nutritional epigenetics. Richardson notes that not all 'developmental origin' or 'fetal origins' research presumes a strictly epigenetic mechanism' (2015, 218) but what unites these different theoretical approaches is a common concern in how 'things outside of the body are transformed into the biology of the body …' (Landecker 2011, 178) and transmitted across generations. The process is thought to be an adaptive response, whereby environmental cues produce a fetal phenotype fitted to the predicted future environment.

The environment in DOHaD literature is rather loosely defined, with some referencing the fetal environment, others referring to the 'intrauterine environment' and many pointing to the relationship of the fetus with the 'actual' (meaning external) environment (Gluckman et al. 2005). The environment here is temporal, spanning the fetal period, the neonatal period and infancy and the 'inherited effects of the interplay of genes and environment' (Jablonka 2004) across generations. Central to all of these environmental performances is the woman's body, referred to as the maternal environment, maternal/fetal environment or sometimes even as 'fertile epigenetic soil' (Heerwagen et al. 2010). This environment, as Richardson suggests, is embodied by 'the fuzzy, receding figure of the maternal' (2015, 227), and the maternal environment is in turn 'conceptualized as an adaptive environment for the fetus in which crucial early developmental cues are transmitted' (2015, 217).

In DOHaD theory it is suggested that during certain 'windows' of development (in utero, in early childhood and in adolescence) the body 'goes through periods of plasticity and openness to the environment' (Landecker 2011,

174). Building on earlier work on poor maternal diets and underweight babies, the idea that prenatal *overnutrition* might affect life-long risk of obesity came to the fore amidst rising global concerns of childhood obesity. From 2000 onward, maternal obesity and its effects on the fetus and early years of development became a growing concern. It is thought that:

> fetal exposure to excess blood lipids, particularly saturated fatty acids, can activate proinflammatory pathways, which could impact substrate metabolism and mitochondrial function, as well as stem cell fate, all of which affect organ development and the response to the postnatal environment. (Heerwagen et al. 2010, 711)

Food that a woman eats can affect the very systems that metabolize food in the growing fetus (Landecker 2011, 176) and becomes mutually constitutive of the nature and functioning of organs and systems through biological 'being-in-each-other' (Martin 2010a, b). Here we have followed our biomedical informants into a research domain that molecularizes environments. But when we step back and invite other disciplinary practitioners to the table, what someone eats is also a function of innumerable environments, such as cultural mores, gender, class and poverty, war and climate change. Reality multiplies (Mol 2002).

The multiple realities of the environment are frequently scaled as one entity and presented in popular discourse as simply an external exposure of food, 'taken in' to the body and inherited across generations. A previous study that examined representations of fetal overnutrition and the reproduction of obesity in Australian print media (Warin et al. 2012) found that women's bodies (spanning both the construction of gender and sex) were positioned as central to the intergenerational transmission of obesity, with women portrayed as responsible for passing obesity on to their children (and grandchildren) via biology and ill-informed 'lifestyle choices'. Women's behaviors and intrauterine environments were constructed as 'smoking guns'—a popular media metaphor for understanding a causal relationship between an exposure and a disease process (Warin et al. 2012). Women who overate and gained excess weight during pregnancy were thus blamed for the 'obesity epidemic':

> Gulp … You are what your Grandma ate … Research by the Victor Chang Institute shows that what mothers and grandmothers ate during pregnancy affects the health of a particular generation through the genes that are passed on. (The Sydney Morning Herald 2006, 34)

This characterization of the environment in DOHaD reverts to a classic example of gene-environment interactions in which food eaten by the mother brings about alterations to DNA expression to the developing fetus and potentially to future gametes of the fetus. Rather than identify what the implications of this 'iterative and open-ended model of relations' (Guthman and Mansfield 2012, 487) are between differing modalities of environments—food systems, eating, genes, cells, bodies and socio-economic status—the environment is situated as a simple matter of individual food choice made by a pregnant woman. This conceptualization not only constructs food as an environmental exposure, but more broadly, it constructs mothers as environments. Mothers are understood as environments of exposure (Landecker 2011), reproducing long-standing discourses that blame mothers for disorders in their children (Warin et al. 2012; Warin 2014; Richardson et al. 2014). As Pickersgill et al. (2013) argue 'in effect, women are framed as the first environment for children, potentially activating and augmenting a range of moral discourses and subjecting [women] to (increased) scrutiny' (Pickersgill et al. 2013, 437). Moreover, women are (erroneously) rendered developmentally dormant, merely containers to the dynamic and vulnerable 'life' inside them.[6]

Case 2: Microchimerism

Like epigenetics, maternal-fetal microchimerism is an instance of reproductive biology that defies, or rather exceeds, the truisms of classical genetics. Chimerism is defined as the existence in an organism of two or more genetically distinct cell populations. Microchimerism occurs when a 'non-self' cell population is very small. While these 'non-self' cells can arise from iatrogenic sources such as transplantation or transfusion, here we're concerned with the naturally occurring kind. It is now well established that cells move, or 'traffic' in both directions between a pregnant woman and the fetus during pregnancy, and routinely persist for many years, even decades, after mother and fetus begin living apart. Since the first report of these so-called fetal cells[7] in a woman decades post-partum (Bianchi et al. 1996), researchers have been finding cellular needles in bodily haystacks and devising ingenious experiments to answer the question: what are they doing there?[8]

Environments are not as explicitly referenced in this domain of postgenomic research as in epigenetics, but this second case brings into relief a number of claims we are making about the inseparability of insides and outsides in postgenomic 'naturecultures' (Haraway 1997). Microchimerism is only a phe-

nomenon of interest in the first place because genetically labeled cells were located *outside* their proper body and *inside* another's. In other words, it was the expectation that these cells can survive inside one body and only that body—and their uncanny defiance of this expectation—that made them scientifically interesting. The discourse of microchimerism is rife with spatial metaphors. One of the most common ways of describing microchimerism is that mothers *contain* cells from their fetus or vice versa. *Trafficking across borders* (both between mother/fetus and inside bodies to diverse organs including the brain) is the dominant metaphorical repertoire in the field, where these cells have been explicitly characterized as 'immigrants' (Martin 2010b; Davies 2012). A number of epidemiological studies aimed at quantifying microchimeric cells in women before and after breast cancer suggest that their presence mitigates the disease (Kamper-Jorgensen et al. 2014; Gadi 2010). In a telling turn of phrase, these cells are labeled as 'exposures' in these studies, though they come from the woman's blood samples and have never existed outside that woman's body.

In traditional sociological and evolutionary terms, a mother is someone who is *outside* a baby (child, teenager, etc.), albeit someone whose behavior (both pre- and postnatally) is extremely relevant to that offspring. Parenting, and especially mothering, looms large in explanations of children's developmental trajectories (especially when they are negative, it seems) (Reimer and Sahagian 2015). Prior to microchimerism and epigenetics, mothers were understood to be exerting influence on their children through their doings; it's the very definition of nurture. How does microchimerism affect this picture? In the words of Harvard evolutionary biologist David Haig, 'microchimerism internalizes the family within maternal bodies' (Haig 2014, 15). This phenomenon is universally read as though material bits of the mother are *inside* the body of the child. Of course mothers and fathers were already thought of as biological contributors to the DNA of their children, but children were still imagined to have a distinct personal genome, theirs alone. However, a common trope in microchimerism discourse is that something we thought of as mere metaphor—the 'mother-child bond'—is *in fact* biological (Martin 2010a). In media coverage of microchimerism, both scientists and journalists frequently use the words 'literally' or 'real' to emphasize the material there-ness of these cells (e.g. Wilson Sayres 2013; Brusie 2015). For example, a recent National Geographic blog post read: 'A mother's children will remain part of her long after they leave her body and enter the world. This isn't just a saying or a metaphor; it's biological reality' (Yong 2015).

Scientists and doctors reaching to make meaning of the unexpected phenomenon tend to combine a sentimentality thick with maternal essentialism

(Martin 2010a) with reflections on the possible evolutionary reason for this cellular exchange and persistence. In an interview with author two, a Canadian medical geneticist and early commentator on the broad biological significance of this phenomenon said: 'the role of our parents, we've always thought of it as environmental and yeah, we have their genes. But now we're talking about it being internal as well. Mom being internal. Dad not. Which is really interesting' (2004). Much is made of the realness of this mother-child bond in ways that threaten to authenticate a conservative backlash against egalitarian parenting and nurturing dads (Martin 2010a). In language informed by epigenetics, this same clinician proposed that maternal cells 'teach' developing babies what the world they will enter is like. In other words, she suggests that perhaps these cells evolved to convey intergenerational knowledge.

Queries about what microchimeric cells are 'doing' entreat evolutionary speculation. Because recognition and elimination of 'foreign' cells seem to be bodies' default immunological position, the question of why a small number of cells would be 'allowed' to live in another usually prompts scientists to propose that they confer some sort of selective advantage. Two recent articles illustrate this move. Harvard evolutionary biologist David Haig (2014), speaking about cells from pregnancies that persist in *mothers* post-partum (rather than children, as above), proposes that fetal cells in women promote a longer inter-birth interval, an advantage to the first sibling. He writes: 'From an evolutionary perspective, *engrafted cells are extensions of the genetic individual of whom the cells are disjunct fragments* and are predicted to evolve effects that increase that individual's inclusive fitness' (Haig 2014, emphasis added). While Haig is fuzzy about the potential mechanisms for this—it is a speculative rather than experimental paper—he proposes that 'offspring cells in mother's bodies should favor their own child at the expense of its sibs' and that these cells may do so by 'interfering with the implantation of subsequent embryos'. Haig's hypothesis is presented as sibling rivalry writ small; a subtitle in the paper, for example, is 'Battling Brothers'.

Haig's work is emblematic of a much larger trend in microchimerism discourse: scientists and popularizers alike render cells as *one and the same thing* as the people from whom these cells originated (Martin 2007). This is true not just of chimerism, but it is a feature of cellular discourse more broadly, harkening to a long history of conflating the body with the body politic, and cells with individuals (Reynolds 2007). The unexpected liveliness of cells exceeding their 'own' bodies is met, more often than not, with a re-inscription of a reductionist equivalence between genes, cells and organisms. The potential for microchimerism to unsettle this picture by introducing difference at a molecular level (and creating something new, neither 'mother' nor 'fetus') is

neutralized by its representation: cells have a priori (genetic) identities and interests, regardless of where they are found. While we are keen attendants to how the emergent field of microchimerism will settle into scientific knowledge, at the same time, we are skeptical that simple scaling between cells and people, and easy drawing of insides, outsides, causes and effects, will do justice to the liveliness and complexity of microchimerism, which itself acts in concert with heterogeneous sorts of temporal and material forces in the unfolding of biological development. Below we draw on the work of Barad and Tsing to further articulate this critique and reach toward alternative renderings of postgenomic life.

The Environment as Intra-action

In the first half of the chapter, we have demonstrated that simple scaling (e.g. between a woman's food 'choices' and methylated nucleic acids in DOHaD, or between a full-fledged son and a cell with a Y chromosome embedded in a woman's liver in microchimerism) is a prominent feature of the discourses we examine. We have further made the claim that this scaling is likely flawed. Next, we draw on the work of Barad to provide a theoretical scaffolding to support this claim. The key to this step in the argument is that in order to scale, one needs fixed and definable entities. We employ Barad's theory of agential realism to reconceptualize the environment in postgenomics, unsettling the well-worn oppositions that are explicit in our case studies. Fox-Keller suggests that it is a grave mistake to think of 'the development of traits as a product of causal elements interacting with one another' (2010, 6) as it 'presupposes an a priori space between elements'. This, she states, is 'precisely what the character of developmental processes precludes' (ibid). Epigenetic processes (although poorly understood):

> depend on the complex orchestration of multiple courses of action that involve interactions between many *different* kinds of elements—including not only preexisting elements (e.g. molecules) but also new elements (e.g. methylation) that are formed out of such interactions, temporal sequence of events, dynamic interactions. (Fox-Keller 2010, 6, emphasis added)

This complexity signals that it is impossible to hold things apart (for long) as phenomena are constituted in their relations of inseparability.

The concept of difference is key to Barad's desire to unsettle dichotomies and move away from what she refers to as some of the most 'sedimented and

stabilized/stabilizing binaries' (2014, 168), such as inside/outside and self/other. Rather than thinking of bodies and environments interacting as separate entities that come together, Barad suggests that these ostensible dualisms are 'intra-actions', 'cuts that make separations—not absolute separations, but contingent separations—within phenomena' (ibid, 175). Cuts, for Barad, are agential ad hoc arrangements of material and semiotic matter from the entanglements that constitute phenomena (where phenomenon is a Bohrian term to 'designate particular instances of wholeness' (2007, 119)). Dichotomies are not pre-existing, they are made in particular moments, where words and practices arrange the world, fleetingly, into seemingly separate things.

Agential cuts require, in the cases of microchimerism and epigenetics, the constant enrolment of scientific practices or apparatuses. Borrowing from Barad again, apparatuses (e.g. experimentation, measurement or staining) do not reveal pre-existing properties of the world, but participate in a performative intra-action to elicit those properties (and not others). An elaborate multi-step ritual enacted by a skilled human in concert with instruments and organic materials makes 'fetal cells' and DNA methylation visible and countable. The observer (scientist), agencies of observation (e.g. a microscope or a real-time polymerase chain reaction machine) and the things observed (fluorescently labeled Y chromatin or light signals) are held apart by a deliberate, contingent cut enacted through the experimental performance. For microchimerism, the output is the statement 'fetal cells were found in this sample' often accompanied by a photograph of a hard-won 'fetal cell', a brightly shining blob against a backdrop of differently colored 'maternal' cells.

Barad does not revert into a romantic holism, though, that denies particular onto-epistemological cuts, partial and non-innocent though they may be. In the play of indeterminate entanglements that make up the world and specific sites in it (like cells and bodies), entities like outsides and insides are not flattened to become one and the same, nor are differences erased (as Ingold (2013) might suggest). She writes 'the point is that the specificity of entanglements is everything' (Barad 2007, 74). We hold on to this point of difference here, as we do not want to suggest that boundaries don't matter or that the particular intra-actions performed in epigenetic and microchimeric laboratories are spurious or illusory. In wrestling with the same conundrum of how to conceptualize the environment, Ingold argues against what he calls a simple addition of one thing to another—in his example of conventional ecology, it is the organism + environment. Like genes + environment, this presupposes independence of living things, rather than their 'indivisible totality' (Ingold 2000, 19) or 'mutually conditioning relations' (2013, 11). From this, Ingold

proposes that the '*domain of the social and the biological are one and the same*' (2013, 9, italics in original).

But they are not *just* one and the same. Barad agrees up to a point in that she doesn't segregate differences into separate entities; she 'does not take separateness to be an inherent feature of how the world is' (2007, 136). But, she continues, 'neither do [I] denigrate separateness as mere illusion' (ibid); 'differences are within; differences are formed through intra-activity, in the making of "this" and "that" within the phenomenon that is constituted in their inseparability (entanglement)' (2014, 175). In conceptualizing the environment in postgenomic fields, we want to preserve '*both* the fundamental inseparability of the biological and the sociocultural, *and* the possibility of a subsequent cut' (Fitzgerald and Callard 2015, 20).

The Lure (and Limits) of Scaling

In this final section, we work with Anna Tsing's articulation of 'scalability' and, in particular, her recent call for a theory of 'nonscalability' (2012). We propose that Barad's notion of intra-action and Tsing's theory of nonscalability are complementary (not equivalent) while putting them in service to our case studies and vice versa. We draw attention to scalar gestures in both of our field sites and approach these moves with an intuition that things are not as neat and tidy as they are made out to be. Tsing has given us a vocabulary in which to articulate this doubt.

To lay some groundwork, by 'scale', Tsing is referring to a design property, long used in mapping ('scale = 1:200'), that turns on the seamless movement between large and small, with a presumption of equivalence in every property other than size.

> As in digital media, with its power to make the great tiny and the tiny great in an effortless zoom, scale has become a verb that requires precision; to scale well is to develop the quality called scalability, that is, the ability to expand—and expand, and expand—without rethinking basic elements. (2012, 523)

For Tsing, scalability—as a design premise in digital pixelated worlds and as an imperial fantasy—has at its core the value of expansion without transformation. Tsing introduces the historical accomplishment of scalability as a prelude to her call for a *theory of nonscalability*, which aims to denaturalize scalability as an inherent property of the world. Scaling is a move that's often attempted, but doesn't always work. This (counter)theory resonates with us,

as it seems to characterize the emergent epigenetic body. According to Tsing, scalability fails in its goal of self-same expansion when *relationships introduce unforeseen difference*. The failure of 'precision nesting' in these cases, though, might be the very thing that enables nonscalable projects to thrive. While engineered growth—expansionism—is built on a biological metaphor, Tsing challenges the assumption that biology or nature are inherently and wholly scalable. It is scholars, she worries, that risk being left behind the lively world, 'holding on to the aesthetic pleasures of scalable precision even when it projects only our fantasies' (2012, 506).

Nonscalability can help us make sense of our case studies. Specifically it enables us to tease apart popular rhetoric that operates as though bodies and environments can be scaled from empirical findings that suggest emergent and indeterminate properties instead. As an exposure, food is and does much more than what Landecker (2011) calls the 'old metabolism': a simple process of digestion where nutrients from food enter a body and are used for energy and growth. In epigenetic thinking, food is now also 'a conditioning environment that shapes the activity of the genome and the physiology of the body' (ibid). Yet this new conceptualization of food and its complex connections to molecular mechanisms, and correlations between nutritional states and adult diseases, reverts back to the 'old metabolism with a twist', as it is frequently referred to in public discourse as 'you are what you eat' or 'you are what your mother/grandmother ate'. How food transforms the body (e.g. by influencing the production of proteins that regulate how cells divide during development or the induction of different phenotypes) is not considered. As an environmental exposure, food is scaled from an outside environment to inside the cells of bodies via molecularization, through and across different bodies and times.

With regard to scaling and microchimerism, consider this title of an article about microchimerism in the *Journal of Pediatrics*: 'So you think your mother is always looking over your shoulder?—she may be IN your shoulder!' (Hall 2003). Here, in a half-joking register, cells are scaled to persons and back again. Haig's hypothesis about micro-level sibling rivalry is another of the many examples we could cite where this move is unproblematically made, as though it is common sense. The move is predicated on an old notion of a stable genetic code in every cell throughout one's life, and the 'selfish gene' inspired plausibility of reducing a person to her genetic code (Dawkins 1989). Applying Tsing, we propose that this expansion and contraction doesn't work because the human body is far more materially complex and heterogeneous (genetically and otherwise) than genetic reductionism allows. Microchimerism and epigenetics themselves are our evidence.

Interestingly, given the cases we are bringing together here, one reason the scalar relationship imagined in microchimerism does not hold could be change over time in the genome of the 'maternal origin' cells living in mother and those in the body that the fetus became in its extrauterine environment (in other words, the son[9] in the world). The genetic code is not a closed and inviolable nugget passed on from cell to cell over years of growth but undergoes constant change in response to its multiple environments and to errors in replication. In a rare nod to this temporal lability, a long-time microchimerism researcher told author two that she would be willing to 'bet a six pack' that if she were to mix together a woman's cells from her own blood sample with 'her' cells isolated from her 15-year-old son's blood, they would not recognize each other as the 'self'. In other words, they would react as immunologically incompatible. 'So I'm kind of an environmentalist there … in some aspect … they would see something different in each other due to the environment'. Unlike this scientist, most commentators on the 'real' meaning of microchimerism uphold an atemporal gene-centric version of events, which fails to account for subtleties of biosocial experience over time recorded in the cells' materiality.

The complexity of the environment has thus far been constructed as a scalable entity in our case studies, an insight corroborated by studies that have attended to media discourses and the narratives of what scientists say they do. In epigenetics, scale making is easy to comprehend, as the oft-used nested Russian dolls present a continuum of food and bodies in which the 'small is encompassed neatly by the large' (Tsing 2012, 507) and the relations between cells and persons are thought to hold still. Moreover, scaling is valued in academia by multiple sources—including grant-funding institutions and scientific reviewers who like to see the environment as 'a doable problem' (Weatherford et al. 2016, 59). Tsing suggests that 'scale-making projects compete for the scholar or world-builder's attention; the trick is to trace or make relationships between projects' (2012, 509). This scalar trick disguises complexity, ambivalence and differences.

Conclusion

In this chapter we have put forward some cautions, questions and (we hope) generative suggestions concerning 'the environment' in postgenomic research. There are many different ways of imagining and perceiving the environment in this wide-ranging field, and while we do not claim to present all possibilities, through our two case studies, we have elucidated underpinning tropes

that support environment rhetoric making. Environments entail different contexts, and we have suggested that they are most often presented as discrete spatial objects. Despite the opportunities to rethink the environment in relational terms, a spatial lens has meant that environments remain tied to boundaries, and they are positioned as inside/outside, as self/non-self and as fetal/maternal. Understanding nutritional epigenetics, for example, as gene x environment (GEI) exemplifies this separation of environments.

This spatial view of environments conforms to long-held assumptions about the construction of bodies and the lines that are drawn between inner and outer spaces, between nature and nurture. Barad's concept of intra-action has been valuable in reconsidering the environment not as a separate thing to begin with, but as phenomena that entail *prior* 'entanglements … not unities' (2014, 176). In her rearrangement, boundaries are not dichotomous; nor do they infer absolute separations, clear dividing lines or geometries of exclusion that position the self on one side and the other—the non-self—on the other side (ibid, 169). Like fetal cells in multiple bodies, pregnant bodies and the intergenerational transmission of epigenetic processes, 'there is no absolute outside; the outside is always already inside' (ibid). Moreover, her attention to the ways in which knowledge is shaped in the sciences (in which we would include social sciences) further draws our conversation toward how the social and the biological are co-constituted (and not entirely separate).

It is not surprising that our case studies demonstrate a tendency to zoom in on molecular/cellular environments and out on bodies. Rose (2006) has been the most prominent scholar pointing to the contemporary molecularization of the life, and epigenetics and microchimerism are easily enfolded into this schema. Tsing's theory of (non)scalability has helped us to critique this molecular scaling. Barad and Tsing can be brought together here with the assertion that to scale properly—a precise mathematical operation—boundaries around the things-to-be-scaled need to be neatly circumscribed, not muddled together with other things. The postgenomic body can be characterized as the intermingling of scalable and nonscalable elements (Tsing 2012) and like Tsing, we worry about the ways that scaling, seemingly demanded by much modern science, disguises our ability to notice heterogeneity in the world.

Two pragmatic antidotes to some of the problems we raise are specificity and interdisciplinarity (Fitzgerald and Callard 2015). By specificity we mean careful attention to rhetorical shorthands (like 'the environment') where empirical precision, or further work (at the lab bench but also linguistic), can allow better characterizations. This often means more cautious or qualified ones. Interdisciplinary collaboration seems a necessary way forward as scien-

tists and social scientists with different epistemological loyalties perform local and partial inquiries that might come together productively.

In a pregnant woman, microchimeric cells from the woman's mother and previous pregnancies introduce cellular and molecular *relationships* to biological life in ways not thought of before. A woman's diet does the same, in that epigenetic mechanisms rearrange relations between biochemical processes and circulations of food production and consumption. Yet there is sometimes (in media especially) a rush to pin these relationships down. Simple discursive moves (like 'your mother's always with you' or 'you are what your grandmother ate') are not likely to capture the nonscalable complexity of living things entangled in myriad relationships (molecular and otherwise) through time. We conclude with Tsing, who herself nods to developmental biology as a 'spark' to ignite a shift to thinking with nonscalability:

> Because relationships are encounters across difference, they have a quality of indeterminacy. Relationships are transformative, and one is not sure of the outcome … Scalability is never complete. If the world is still diverse and dynamic, it is because scalability never fulfills its own promises. (2012, 510)

Notes

1. We use 'invention' here in the manner of historical ontology, to signal 'the coming into being of the very possibility of some objects' (Hacking 2002, 2).
2. This interview, of a Canadian pediatrician, was conducted in 2004 by author two in the context of a project about the social and philosophical significance of microchimerism.
3. This phrase thrives in postgenomic life in spite of early and frequent feminist objections to this effect: "As fetuses in their 'maternal environments' become ubiquitous, women seem to vanish" (Morgan and Michaels 1999; also Petchesky 1987; Duden 1993).
4. Note that these dominant understandings of the environment do not acknowledge or accommodate indigenous cosmologies of landscapes, social relations and personhood that often incorporate humans, animals and objects (c.f. Zavala et al. 2015).
5. Latour (1993); Callard and Fitzgerald (2015); Griffiths and Stotz (2013)
6. While these representations of the environment are taken for granted in popular accounts of epigenetics, scientists working in the field have a more complex understanding of the environment (c.f. Weatherford et al. 2016).
7. The quotation marks here are meant to denaturalize this nomenclature which is based on genetic identity alone. As we will elaborate later in the chapter, the

decades passed embodied by another are relevant to the materiality of these cells, and it is genetically reductive to see them as simply fetal, as though unchanged by their time in the mother's body.

8. Much evidence from clinical, epidemiological and molecular research suggests that the answer is 'probably something'. See Boddy et al. (2015) for a recent review from a biomedical viewpoint and Martin (2010b) for a detailed history of the field.
9. By far the most common method of elucidating microchimerism is finding molecular sequences from gene-rich areas on the Y chromosome. Because women are presumed to be XX in all their cells, Y chromosomal genes are an efficient proxy. Hence, sex-mismatched pairings are sought in clinical trials, while female fetal cells are presumed to behave in the same ways as their brothers.

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31

The Vitality of Disease

Ayo Wahlberg

Introduction

Despite long-standing scepticism towards the biological within the social sciences (Meloni et al. 2016), in recent decades, social studies of medicine have attained a definite *bio*-bent as scholars have set out to map and analyse processes of biomedicalisation, molecularisation, geneticisation and pharmaceuticalisation (Lippman 1993; Clarke et al. 2009; Rose 2007; Williams et al. 2009). Such processes have urged social scientists into the laboratories, clinics and patient associations within and through which biological knowledge, biomedical practice, biosocial groupings and biological citizens are being formed or co-produced. Anthropologists, sociologists and historians alike have taken up the task of studying biomedical practices of research, therapy and counselling both within biomedical settings and outside as biomedical knowledge, products and practices leave its laboratories, factories and clinics to circulate in communities and households (Novas 2006; Callon and Rabeharisoa 2008; Lock 2013).[1] We have learned how biosocial communities form around specific genetic mutations, how biological citizens negotiate access to entitlements and demand health rights by tactically using biological knowledge, how individuals increasingly relate to and act upon themselves in terms of their genetics or neurochemistry and how biotech CEOs mobilise capital by selling future 'visions of life' in the bio-economy (Rajan 2006; Petryna 2002; Rabeharisoa & Callon 2002). In

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this strand of research, "the vital politics of our century… is concerned with our growing capacities to control, manage, engineer, reshape and modulate the very vital capacities of human beings… at the molecular level" (Rose 2007, 3–4). As a result, we have a seen a proliferation of social scientific bio-concepts—from bio-sociality to bio-value, bio-regulation, bio-capital, bio-economy and biological citizenship—just as journals like *BioSocieties* and *New Genetics and Society* have appeared as venues for social scientific scholars to engage with the ways in which a 'new' biology impacts upon society and vice versa.

Yet this turn to the social study of biology and biological knowledge production practices in medicine has not been embraced by all. Medical anthropologists (and other qualitative health researchers) who study the lived experience of their informants as they fall ill and struggle to get better, devising coping strategies and mobilising therapy support groups along the way, have pointed not to the growing circulation of biomedicine and biomedical concepts, but rather to the chronic failures of biomedicine in terms of its availability, accessibility, quality, efficacy and relevance (see Biehl and Petryna 2013; Manderson and Smith-Morris 2010). From these scholars, we have learned how biomedicine is but one among many medical strategies pursued by the ill and also how stark inequalities continue to shape medical landscapes throughout the world. And so, if the aforementioned bio-turn has seen social scientists take up biology and biological knowledge production as the objects of their study, 'classic' medical anthropologists have retained subject-centred approaches to understanding therapeutic quests, illness understandings and relationalities.

In more recent years, a third form of social scientific engagement with biology and society in medical fields has emerged (Pickersgill et al. 2013; Meloni and Testa 2014; Lock 2015). As Meloni et al. (2016, 10) have asked, "if some of the convenient notions that polarised the separation between the social and the biological as two distinctive fields are becoming increasingly untenable in the light of the new biology, what shall we make of our reassuring disciplinary division?" Indeed, the goal of the present *Handbook of Biology and Society* is to move beyond the acrimonious debates that have characterised the biology/society border in biology and the social sciences alike. This is a biosocial (as opposed to a bio-) turn which is characterised not so much by social scientists taking biology as an object of study, but rather by attempts to (re)incorporate notions of the biological body into social investigations and vice versa.

These developments notwithstanding, there is one line of analytical and empirical pursuit which remains strikingly idle, namely that which relates to the formation of knowledge of *living* (understood as a social activity and personal experience) and related practices of *living*. How biology and society

intersect in the twenty-first century is not only a matter of how developments in genetics, neuroscience, reproductive technologies and regenerative medicine are profoundly transforming the ways in which we organise our societies and relate to ourselves. Nor is it only a matter of how social lives and individual biographies profoundly shape our situated biologies. It is also a matter of methodologies. The medical life sciences are those which take organs, tissues, cells and DNA as their object of study predominantly with the help of microscopes, magnetic resonance imaging, polymerase chain reaction techniques, pipettes, cell cultures and the like. Social scientists on the other hand study people, their forms of organisation, their practices, behaviours and self-understandings, more predominantly with the help of databases, collected documents, surveys, interviews, participant observation and more. Biology and society intersect in numerous spheres (see Villadsen and Wahlberg 2015), albeit health and medicine is perhaps one of the most salient and it is within this sphere that I will show how knowledge of (biological) life and knowledge of living—generated in and through different methodologies—have come to co-circulate and inform practice in ways that increasingly enfeeble the kinds of epistemological hierarchisations that are embedded in the notions of 'biological reductionism', 'biomedical hegemony' or Cochrane-style hierarchy of evidence models (which place qualitative research on their lowest rungs).

Canguilhem once wrote: "Human life can have a biological meaning, a social meaning, and an existential meaning. In an assessment of the modifications that disease inflicts on the human being, all these meanings can equally be retained. A man does not live only like a tree or a rabbit" (2008, 121–122). Point being: not only is life sustained, it is also lived. If Canguilhem's archaeological readings of biologists and medical scientists have generated path-breaking insights into the formation of 'knowledge of life' in its biological meaning, what then of its social and existential meanings? I would suggest that the time is ripe for empirical analyses of the ways in which knowledge of living—that is to say knowledge of living as a social activity and a personal experience—has come to be formed, as well as how it has transmogrified or spilled over into practices of living (and vice versa) within medical arenas. For, alongside molecularisation and biomedicalisation, we have seen an upsurge in the production of knowledge about how it is to live with disease—morbid living—a subset of what I have more broadly called knowledge of living.

In distinguishing between knowledge of life and knowledge of living, I am not alluding to the distinction between 'bare life' (or *zoe*) and 'qualified life' (or *bios*), which has been extensively fleshed out by Agamben (1998), Fassin (2009), Biehl and Petrtna (2013) and many others who rightly point out that there is more to the politics of life than the *bio* prefix indexes. Neither am I

pointing to the styles of ethical activity that make up contemporary 'regimes of living' (Collier and Lakoff 2007). Instead I am making a methodological distinction between how knowledge of life and knowledge of living are generated as well as between the DNA, cells, tissues and organs that biomedical therapies target and the identities, quality of life, daily living or well-being that strategies aimed at improving the lives of those living with disease target. My suggestion is that we need to study the productive effects of our own knowledge production practices as social scientists—that is, we need to be as attentive to the effects of our interview techniques and observation as we have been to the effects of sonography or DNA amplification.

I am not the only one to have noted this blind spot within contemporary social studies of medicine. Rabeharisoa and colleagues recently put together an important special issue on what they call 'evidence-based activism' in which they argue that "patients' organisations… collect experiences and build experiential knowledge, and that is how they give shape to concerned groups and delineate their preoccupations" (2014, 115). Likewise, Angela Martin and colleagues held *Experience as Evidence? A Symposium on the Sciences of Subjectivity in Healthcare, Policy and Practice* at the University of Oxford in October 2014 to explore "What does it take to turn experience into evidence? What new methods and expertise are emerging in this field?" More broadly, Charles Camic and colleagues have called for an expansion of STS attention to the social sciences by suggesting further studies of "the mundane actions and processes by which the makers of social knowledge carry out their work[,] … the daily routines of knowledge production, evaluation, and use" (2011, 8). While I agree that we must look to the ways that qualitative evidence is currently being produced in patient associations or government agencies for use in policy and practice, I suggest that we must in a sense be bolder than this. Qualitative health research, including that of medical anthropologists and sociologists, has been around for at least a century now, and it has certainly not remained within the ivory tower. Rather (much like biomedical knowledge), it has left the Academy to circulate within hospital consultations, patient schools, households and more, as I will show. It is for these reasons that we need to attend to the effects of knowledge of living with the same commitment that the effects of knowledge of life have been cartographically and analytically pursued in recent decades.

In this chapter, I sketch out a possible outline for such an analytics of what we might conceptualise as the vitality of disease. I begin by arguing that morbid living has by now settled alongside pathological life as a crucial site of therapeutic intervention in many different medical settings. I then move on to argue that biological diseases today are as much understood as 'kinds of

living' (Wahlberg 2009) as they are failing biologies in these same settings. Finally, I suggest that in recent decades, morbid living has come to be disciplined, by what I mean processes through which knowledge of living with a disease transmogrifies into practices of morbid living and vice versa (cf. Foucault 1995). I point to three instances of the disciplining of morbid living; firstly the emergence of patient schools aimed at teaching patients to learn how to live with their disease; secondly the standardisation of subjects through rating scales used in clinical trials to measure treatment effect on the 'quality of life' of patients; and finally the dissemination of practical advice through systematised, disease-specific 'Living with' guides aimed at patients and carers which are often prepared by patient or disease-advocacy associations. What these instances have in common is a focus on patient living as something that can be improved in terms of 'quality of life', 'well-being' or 'healthy life' as therapeutic objects. Indeed, quality of life has become a container concept.

Morbid Living and the Birth of 'Quality of Life'

According to epidemiologists, more people than ever before are living with (especially chronic) diseases. Against a backdrop of aging populations, 'lifestyle disease' epidemics and advances in medicine, epidemiological calculations have suggested that by 2004, "18.6 million were severely disabled and another 79.7 million had moderate long-term disability" because of the diseases they suffered from (WHO 2008, 34; Manderson and Smith-Morris 2010). In countries like the United Kingdom and Denmark, healthcare officials report that soaring numbers of people with long-term medical conditions such as diabetes and dementia have pushed the proportion of treating and caring costs for the chronically ill to between 70% and 80% of national healthcare budgets (Campbell 2014; MandagMorgen 2011). At the same time, as many scholars have pointed out, low-income countries are currently struggling with a 'double burden' of both communicable and non-communicable disease, indeed so much so that acute-chronic and communicable-non-communicable dichotomies are becoming blurred (Rosenberg and Golden 1992; Whyte 2012; Manderson and Smith-Morris 2010). And so, just as healthcare workers in the global North and South continue their efforts to reduce morbidity and mortality rates through preventive medicine, lifestyle interventions or curative treatment, they must also *improve the lives of those living with disease*, or as put in a national healthcare strategy from Denmark, they "must focus much more than hitherto on *both* length *and* quality of life" (MoH 2002, 6). As we will see, within contemporary biomedicine and health-

care, it is apparent that life is *treated* as much more than a cellular and molecular activity. Notwithstanding the important findings that have resulted from studying the effects of biomolecularisation, as I have already noted, there has been less attention within medical science and technology studies to the equally conspicuous and contemporaneous (with molecular biology) emergence of 'quality of life'.

This is not to say that quality of life is understudied in any way, quite the contrary. It is around the middle of the twentieth century that we can locate the birth of 'quality of life', not so much as a term but as something that can and ought to be measured, monitored, audited and improved in the medical field (see Armstrong and Caldwell 2004; Brooks 2013; Dokumaci 2014; Wahlberg 2007; Wahlberg and Rose 2015). Three books in particular stand out as emblematic for this birth: John Galbraith's *The Affluent Society* (1958), Rachel Carson's *Silent Spring* (1962) and Ivan Illich's *Limits to Medicine* (1976). Each of these books highlighted the ways in which longer, more affluent lives (in the West) were at the same time afflicted by the smog, toxins and iatrogenic side effects that industrial societies had brought in their wake (see Wahlberg 2007, 2012). Galbraith, Carson and Illich were of course not alone; rather they participated in what, by the 1960s, had become a chorus of critiques of modernity's growing inventory of *-isations* (industrialisation, technologisation, bureaucratisation, rationalisation, globalisation, medicalisation, etc.). In the field of medicine, the developments of renal dialysis and heart transplantation have in particular been highlighted as signposts in the consolidation of concern about 'health-related quality of life'. While these two forms of therapy clearly saved lives, as Armstrong and Caldwell have shown, early commentators argued "only at considerable sacrifice to the quality of life" (2004, 364).

In tandem with these critiques, an incipient field of 'quality of life' (QoL) research coalesced within the fields of medicine, public health and health economics. This type of research was devoted to the definition and measurement of quality of life in the form of indices and instruments (whether generic or disease-specific) used: to measure "a patient's ability to carry on his normal activity and work" (Karnofsky and Burchenal 1949, 197); to measure the state of health of a particular individual, group or population (Fanshel and Bush 1970); to assess the health outcomes of a particular health intervention (Carlens et al. 1971); or to evaluate a particular health policy (Patrick and Erickson 1993). Among the most important of these instruments has been the EQ-5D measure of health outcome (Brooks 2013), the QALY (Quality Adjusted Life Years) which is used to assess the extent of the benefits gained from a medical intervention in terms of survival and quality of life and the

DALY (Disability Adjusted Life Years) which is used to estimate the burden of disease in a given population with particular emphasis on non-fatal outcomes, which is to say 'loss of healthy life' as distinct from the loss of life (see Wahlberg and Rose 2015). Much of the debates and critiques that have ensued in the wake of these and other instruments of measurement have concerned the assumptions about health and valuing of life which underpin them (see Gold et al. 2002; Round 2012; Kleinman and Kleinman 1996).

More recently, Anne Marie Mol and colleagues' (2002, 2008, 2015) important work on how different enactments or versions of atherosclerosis entail different ontologies as well as on how logics of care and choice clash in the treatment of diabetes patients has highlighted the tensions that emerge when "it is mostly [patients'] so-called 'quality of life' that improves" (2008, 70). Still, for Mol, 'quality of life' indicators and measures are not analysed for what they do in terms of making up a particular version of atherosclerosis or diabetes; rather she suggests that when "quality becomes a quantity" controversies are stifled (2002, 174).

Such critiques notwithstanding, it is clear that with the improvement of 'quality of life' firmly on the medical agenda, there is much more to the politics of vitality in the twenty-first century than molecular biology. For, to acknowledge such a thing as 'loss of healthy life' is to show epistemological partiality towards what I have called morbid *living*; when healthy life is lost, it is morbid living that takes its place which in turn calls for knowledge about how it is to live with disease. This partiality is achieved through a methodological distinction between somatic, biological life (i.e. 'life itself' as many have put it; see Franklin (2000)) and personal, social life (i.e. living as a personal and social activity or experience) as separate, albeit intimately interlinked, objects of knowledge. While "knowledge of life" (Canguilhem 2008) stemming from the life sciences certainly constitutes a key component of the ways in which vitality is understood and intervened upon today in healthcare, we must also examine how *knowledge of living* generated especially, but not only, through qualitative social science is also contributing to the emergence of novel forms of measurable life—for example, 'healthy life', 'quality of life' or 'well-being'.

Indeed, alongside biomolecularisation, we have seen an upsurge in knowledge of how it is to live with disease, a subset of knowledge of living. Medical anthropology and sociology have, of course, been at the forefront. Beginning in the first half of the twentieth century, anthropologists began generating insights into the ways in which individuals and communities experience, cope with and tackle disease and illness (Rivers 1924; Evans-Pritchard 1937). Indeed, the distinction between disease and illness proposed by medical

anthropologists was exactly intended to shed light on an individual's experience of living with a disease: "Disease… in the… biological terms of the biomedical model is… an alteration in biological structure or functioning" whereas "illness refers to how the sick person and the members of the family or wider social network perceive, live with, and respond to symptoms and disability" (Kleinman 1988, 3, 5–6; See Mol 2002). Consequently, as Susan Whyte has argued, "research on chronic conditions… has been central for the development of methods, concepts, and theories in medical anthropology" (2012, 64). It was long-lasting rather than acute illness that gave rise to therapeutic quests and the unfolding of personal narratives as patients looked for meaning, support and ways to cope with their conditions (Janzen 1978; Kleinman 1980; Mattingly 1998; Steffen et al. 2005; Manderson and Smith-Morris 2010). Hence, what I am also calling for are archaeological readings of the key concepts of medical anthropology—not reflex, regulation or pathology but rather coping, suffering, navigation, symbolic efficacy or coherence (see Wahlberg 2008).

Moreover, if morbid living begins the moment healthy life is lost, then we are led to ask just what it is—what kinds of vitality—that is considered lost and, conversely, what there is to be (re)gained via healthcare interventions. Canguilhem famously argued that "a pathological state is never a state without norms—such a thing is impossible. Wherever there is life there are norms" (Canguilhem 1994, 351). Following Geroulanos and Meyers, we can see how Canguilhem relocates this lesson into the realm of 'knowledge of living' when he shows how "disease makes it impossible to live without constant reference to norms and to deficiency vis-a-vis these norms" (2012, 3). We might say then that there is never a state of ill health without norms, meaning that every form of (disease-specific) morbid living has its norms. As long as one is alive, not only is life *sustained* biologically, it is also *lived* socially and personally however 'diseased' this living might be. It is in this sense that morbid living is quintessentially biosocial. Every disease has its specificities and characteristics in terms of the kinds of biological failing that are at stake (e.g. uncontrolled cell division, faulty immune response, ineffective production of insulin) and it is these failing biologies that inevitably lead to certain kinds of living characterised by therapy, regular medication, visits to the hospital and/or reliance on medical devices which in turn can impede upon or shape daily living. Diseases are debilitating to varying degrees and in differing ways, hence, qualitative health research has shown how becoming sick will eventually impact on daily life through a range of restrictions, limitations, constraints, discomforts and/or apprehensions. These restrictions and limitations can, at the same time, be addressed more or less effectively with the help of medical therapies

and/or social support, allowing some to live chronically with a given disease for years if not decades.

For all Canguilhem has taught us about how knowledge of life and knowledge of living presuppose each other, we have yet to see the kind of archaeological readings of the knowledge of living that has been generated by medical anthropologists and other qualitative health researchers over the last century or so. If Claude Bernard, René Leriche and Marie François Xavier Bichat were central empirical sources for studies of the consolidation of a knowledge of life in the eighteenth and nineteenth centuries (Canguilhem 1994), then William H.R Rivers, Edward E. Evans-Pritchard, Erwin Ackerknecht and many other qualitative social scientists become essential empirical sources for similar studies of the consolidation of a knowledge of living with disease in the twentieth century (see Wahlberg 2008). It is by studying the norms of morbid living archaeologically and genealogically that we gain further insight into questions of: what is a life of 'good quality', what is a 'healthy life' and what is 'well-being'?

And so, however urgent a priority saving the lives of those who die from (especially preventable) disease remains (and let us not for a second discount this urgency, not least in these times of Ebola and scandalously grave health inequality), what I am arguing is that therapeutic objectives today increasingly involve improving the daily living, quality of life or well-being of individuals through specific practices which can be shared, taught, trained and propagated. For, not only have we seen an upsurge in the production of knowledge about how it is to live with disease (not least by medical anthropologists and sociologists), we have also seen a swarming of practices aimed at improving the lives of those living with disease. Efforts to improve the lives (as something further to saving lives, which, as I have said, remains crucially urgent) of those living with disease are to be found globally. Let us now turn our attention to three specific arenas wherein practices aimed at improving the lives of those living with disease are currently playing out.

Learning to Live with Disease

In a hospital leaflet distributed to discharged heart patients at Hvidovre Hospital in the west of Copenhagen, an offer is made:

> When you come home from the hospital after treatment for a heart disease, it can be difficult to imagine how daily life will be. Recovery takes time and you need to know how to prevent your heart disease from getting worse. In this

> brochure you can read about our offer of courses and training for heart patients after discharge. (Hvidovre Hospital 2010)

Such courses have become a mainstay of many hospitals and healthcare centres (Taylor and Bury 2007; Lindsay and Vrijhoef 2009). The idea is to cultivate so-called expert patients who, in learning how best to take care of themselves and their chronic conditions, will "improve [their] quality of life and health status" (MandagMorgen 2011, 35) while also hopefully reducing strains on healthcare budgets. In a 1998 Working Group report, the World Health Organization argued that such:

> therapeutic patient education should enable patients to acquire and maintain abilities that allow them to optimally manage their lives with their disease. It is therefore a continuous process, integrated in health care. It is patient-centred; it includes organized awareness, information, self-care learning and psychosocial support regarding the disease, prescribed treatment, care, hospital and other health care settings, organizational information, and behaviour related to health and illness. It is designed to help patients and their families understand the disease and the treatment, cooperate with health care providers, live healthily, and maintain or improve their quality of life. (WHO 1998, 8)

This 'outsourcing' of care by helping patients to help themselves is a crucial site in which to observe the different ways in which practices of morbid living are currently playing out. In a welfare state like Denmark, municipalities are in charge of offering patient courses to their citizens, as in the case of a municipality in Southern Zealand which offers courses for seven different patient groups: cancer, lower back pain, chronic obstructive lung disease, type 2 diabetes, obesity, stress and heart disease. If we take a closer look at the patient course for type 2 diabetes, stated vital objectives are to help patients:

- Achieve better regulated blood sugar levels
- Feel increased physical and psychological well-being
- With their motivation to be physically active
- Gain more energy
- Get a good social network which can be used after the course

We can say, then, that such patient courses are focused on improving the vitality of type 2 diabetes patients, both by coaching patients to regulate their bodies appropriately while also working to improve their well-being and social life. What the emergence of therapeutic patient education in the form of

systematised courses highlights is a sentiment that getting a diagnosis in a hospital is not 'merely' a biomedical matter—adhere to doctor's orders, comply with treatment regimens and schedules—but it is equally a social and personal affair. With chronic disease, patients can expect to live with the condition for the rest of their lives, and their primary challenge is therefore learning how to live within the more or less narrowing constraints that a disease is seen to bring with it in the most optimal of ways. As Mattingly and colleagues have shown, such courses stand somewhat in contrast to the daily difficulties faced by many, especially those who are socio-economically marginalised (Mattingly et al. 2011). They contrast therapeutic patient education which is very often compliance and adherence oriented (sometimes called treatment literacy), with the kinds of 'chronic homework' that patients struggle with in their daily lives. Nonetheless, what I wish to point out is that patient courses have become an institutionalised component of healthcare as something further to the ambulatory visits and check-ups at the General Practitioner that have long been a part of life with a chronic condition. In so-called 'resource poor' parts of the world, such courses can take the form of community treatment literacy training or workshops to help, for example, individuals with HIV to 'live positively' (Whyte 2014). We need to continue studying patient courses and training, not only with a view to assessing their strengths or shortcomings but also to examine their productive effects in the constitution of new forms of living with particular diseases.

Standardised Subjects

Another arena within which we can observe the consolidation of the improvement of daily living as a therapeutic objective is that of clinical trials. Therapeutic claims for a drug or therapy are increasingly linked to the improvement of a patient's 'quality of life'. For example, six out of the top ten blockbuster drugs from 2006 purported to improve patients' quality of life as their main claim: for example, "Advair significantly improved and maintained health-related quality of life"; "Subjects treated with venlafaxine [Effexor] noted an…improved quality of life as compared to those receiving placebo"; "After treatment with Nexium 91% of patients with reflux esophagitis in the study shown here were free of heartburn, resulting in a considerable improvement in many aspects of their daily lives"; and "Olanzapine [Zyprexa] demonstrated a superiority over risperidone in… improving patient quality of life and interpersonal relationships" (see Herper and Kang 2006). Such therapeutic claims are not possible without rating scales and indices. Since treatments

are not only used to save/lengthen life, researchers must be able to *measure* improvements in the way a patient experiences or lives with a disease, which in turn requires scales to rate what is sometimes called the 'health-related quality of life' of a patient. As noted earlier, we can trace the emergence of rating scales to the work of Steinbrocker and colleagues (1949) who proposed classificatory schemes for measuring the functional capacity in rheumatoid arthritis as well as Karnofsky and Burchenal (1949) who developed a performance scoring system for measuring the ability of cancer patients to carry out activities of daily living (see Wahlberg and Rose 2015; Dokumaci 2014). Since then, the construction of such scales has become a veritable industry in itself as a cascade of generic as well as disease-specific rating scales have emerged.

What they have in common is that they attempt to quantify the subjective experience of what it is like for someone to live with a disease, that is, what is morbid living like? Put in another way, they attempt to quantify the 'loss of healthy life'. If disease is considered to be debilitating in varying degrees and becoming sick will eventually impact on your daily life through a range of restrictions, limitations, constraints, discomforts and apprehensions, by scoring and ranking these, rating scales provide a numerical basis for assessing disease impact along axes of severity. While not exhaustive, it is possible to identify four important domains of daily living that are assessed through rating scales:

1. Functional ability—many rating scales give priority to 'performance' or the ability to carry out daily activities such as self-care (washing, toilet visits), mobility, cleaning, cooking, shopping and so on. Continuums go from 'bedridden' or 'entirely dependent' to 'fully mobile' or 'independent'. In Karnofsky and Burchenal's words, performance scales "measure the usefulness of the patient or the burden that he represents to his family or society" (1949, 195–197) and have been criticised for this utilitarian view of disease impact.
2. Discomfort—not only is sickness seen to limit or constrain, it is also seen to generate discomfort, a point that many rating scales attempt to capture by asking patients to what extent they feel pain or discomfort. Continuums go from extreme pain/suffering to no pain or discomfort.
3. Unease—some rating scales attempt to capture the many apprehensions that sickness can generate in a patient. Continuums range from 'very anxious or depressed' to 'not anxious or depressed'.
4. Relationships—a less common domain is that of relationships which suggest that a 'healthy' person is one who is involved in a number of positive

relationships with family, friends and colleagues. Continuums can be organised along degrees of isolation (i.e. number of relationships) as well as on how individuals self-assess their relationships (e.g. as good or bad).

Social studies of randomised controlled trials have shown how such trials rely on the construction of standardised inclusion criteria, treatment regimens as well as clinical outcomes (Lakoff 2007; Petryna 2007; Wahlberg 2008). Indeed, it is not uncommon for trial conductors to include a battery of clinical outcome measures in the form of rating scales as a way to maximise the possibility that some kind of 'signal' is detected, that is, statistically significant changes in the rating scale scores of trial subjects over time. Rating scales make it possible to quantify the experience of living with disease. Yet each disease has its specificities in terms of how it is seen to impact on a patient and/or carer. As such disease-specific scales such as the St. George's Respiratory Questionnaire (SGRQ) or the Quality of Life in Reflux and Dyspepsia (QOLRAD) have been developed for use in clinical trials for asthma and reflux, respectively. Predictably, such efforts to quantify the qualitative have been subject to countless critiques. My task here is not to assess their suitability or validity; rather I am highlighting the fact that knowledge about morbid living (about how it is to live with a disease) is now routinely generated in clinical trials. The rating scales themselves have been developed by clinicians, yet a key question emerges around how disease impact has come to be conceptualised around the four domains of functional ability, discomfort, unease and social isolation. Here we see one of the immediate methodological differences when compared to those social studies of biomedicine which have studied the co-production of biomedical knowledge and practice: while clinicians and laboratory scientists are in specific medical settings on a daily routine basis (and therefore can more readily be observed and studied in these settings), social scientists are usually not. Moreover, while biological disease categories and substances are surrounded by visible materialities in the form of laboratory equipment, biopsies and medical devices, knowledge of quality of life is most often paper-based in the form of QoL questionnaires, study results or clinical trial protocols. The question I would pose then is when clinicians, epidemiologists, health economists and others have developed rating scales for measuring a patient's quality of life when living with a disease, has this development been entirely independent of medical anthropologists' and other qualitative health researchers' long-standing engagements with similar questions? Future studies could productively track down the genealogies of QoL rating scales while also attending clinical meetings in which clinical trials are designed and treatment outcomes determined. The ironies of quantifying the

qualitative as patients and carers are modelled into standardised subjects are obvious. Yet, we should nevertheless investigate how it is that clinicians, epidemiologists and health economists have come to invest so much time and resources into quantifying the qualitative in ways that render 'quality of life' and 'well-being'—the patient perspective—auditable.

Kinds of Living

> Life with Alzheimer's is a shared concern for both the one who has the disease and her or his loved ones. Alzheimer's infiltrates deep into family life. Roles transform and ways of being together change character. Hence, when a diagnosis has been given, the challenge is to find out how you will live a life with Alzheimer's together… Whether you have Alzheimer's or are a loved one, you will need good advice as well as help and guidance to the different phases that you will go through as the disease progresses. (From carer guide "Lev med demens [Living with dementia]", Alzheimer Association 2012)

How can one live a life of good quality—or at least the best life possible—with Alzheimer's, diabetes, chronic obstructive pulmonary disease or ischemic heart disease? Such questions lie at the heart of a new style of advice or guidance that is emerging out of especially patient associations. So-called 'Living with' guides are now a standard offer from most patient associations[2] as they seek to provide recently diagnosed patients and their carers with insights into the 'new' life that awaits them. Take Alzheimer's disease, a disease that is viewed by many public health experts as a major healthcare challenge in coming years as populations age. 'Living with dementia' guides are often introduced with the following kinds of statements: "When receiving a dementia diagnosis, you are inevitably thrown off course"; "Receiving a diagnosis of Alzheimer's is never easy—it's life changing" (Alzheimer's Association 2012, 3; Alz.org 2014). Such guides from patient associations contribute to the stabilisation of Alzheimer's as a 'kind of living' (Wahlberg 2009; see also Moreira et al. 2014). While each patient is unique as are his or her circumstances, there are nevertheless a range of experiences that are seen as common to the kind of living that Alzheimer's brings with it. So what kind of living is Alzheimer's? Let us take a closer look at how Alzheimer's living is sketched out in such guides as well as how advice and tips are provided: "There are some practical things that you can do to help you live as well as possible" (Alzheimer's Society 2012, 42).

Common to 'Living with' guides is their emphasis on daily living and on providing tips for easing this in the face of the challenges that Alzheimer's

living can bring with it. As a form of morbid living, Alzheimer's has its specificities related to memory loss, mood changes, loss of balance, medicines, doctor's appointments, the making of wills and eventually loss of ability to carry out tasks in everyday life. As a degenerative disease with no known cure, a life with Alzheimer's is characterised by a series of deteriorations which can hopefully be slowed down and tackled through medication, tips of keeping daily routines, staying active, labelling things, using a calendar and the like.

Such guides are also characterised by their emphasis of bringing patient experiences to the fore, through quotations such as "You must carry on doing the things you enjoy, and not side line yourself from your friends and family or clubs and groups. We've always led busy lives, going out and about—and we still do" (Alzheimer's Society 2012, 42). Indeed, as Rabeharisoa and colleagues have argued, "patients' organisations… collect experiences and build experiential knowledge, and that is how they give shape to concerned groups and delineate their preoccupations" (2014, 115). This, they suggest, is different from the forms of biosociality that bring people together because of, for example, a shared genetic mutation (see Epstein 1998; Rabinow 1996; Rose and Novas 2005; Gibbon and Novas 2008). However, while Rabeharisoa and colleagues point to the 'evidence-based activism' that experiential knowledge then feeds into, I would argue that we might speak of socialities which are coalescing around shared kinds of morbid living or *lebens*-socialities. The point being that through the generation of knowledge about what it is like to live with disease, patient associations are preparing practical advice on how *best* to live with that disease. It is therefore exactly at the intersections of knowledge and practice that we can observe what I have called a disciplining of morbid living. Morbid living is disciplined in the dual sense of coming to be the object of specialised bodies of expertise while at the same time being subject to normalising interventions aimed at promoting 'good' ways of living with a particular disease through 'Living with' guides, training courses or state-led information campaigns. Much like hospitals, one Alzheimer Association organises weekend courses as well as patient schools, as they suggest growing numbers of people:

> receive a dementia diagnosis while they are still leading active lives. For many, the diagnosis may lead them to stop exercising or to withdraw from their social lives. Further to decreasing a person's quality of life, isolation and inactivity can worsen the disease. That's why Alzheimer Association organises patient schools which are tailored for people with dementia. The courses are especially targeted at younger persons with early onset Alzheimer's. (Alzheimer Association 2012)

The methodological challenge here for future studies relates to examining how living with guides have been developed as well as how patient schools are designed and held. For example, in Denmark patient associations have become an important employer for anthropologists and other social scientists, not least because of their interest in patient perspectives and the ways in which patients and carers live with disease on a daily basis. Who are the experts hired to put together 'Living with' guides, and what forms of knowledge and expertise are invoked to qualify daily living advice? Once again methodological challenges arise, ones requiring detective-like skills of tracking down how morbid living manuals and patient schools have been developed.

Conclusion—Living

Why research the ways in which morbid living has come to be known and practised when in many ways it is its opposite in 'healthy living' that has recently grabbed headlines and shaped numerous national health agendas in the form of preventative medicine (e.g. cholesterol and blood pressure medicines), lifestyle interventions (targeting exercise, alcohol and smoking) or nutrition campaigns (e.g. five fruits a day) (see Greene 2007; Dumit 2012)? Firstly, as pointed out in this chapter, notwithstanding continued efforts to reduce morbidity and mortality, when it comes to healthcare, it is long-term disease treatment and management that is taking a growing share of annual healthcare expenditure. We can say that the management and improvement of morbid living is one of the most pressing tasks for healthcare workers today. Indeed a national healthcare administrator in the United Kingdom has warned that "this is the biggest problem facing the health system and the care system and the costs are growing year on year… unless we change the way we address the problems, [it] will overwhelm the system" (McShane cited in Campbell 2014).

Secondly, by studying how morbid living is known and practised, we can also generate further insight into how healthy living and quality of life (as healthcare objectives) are understood and how they are valued. At stake are efforts to enable the best possible lives within the constraints that particular diseases are seen to bring with them not by privileging either biomedicine or social science, rather by acknowledging that both are requisite. In the biosocial turn that this book is in part a response to, methodological debates often concern how social and biological research techniques can be modified to take the other into account, for example, in the form of a bio-ethnography "which integrates biological and ethnographic data about the larger histories and life

circumstances that shape health" (Roberts 2015). What I am proposing is to make ethnographic and other forms of qualitative research the object of study in much the same way that social scientists have made biological knowledge production the object of research in recent decades. If medical practice today is informed in important ways by qualitative insights into how it is to live with disease, then social studies of medicine will do well to train their analytical gaze in equal measure towards the production of knowledge that takes place within the social sciences.

How then should one design research that will allow one to research the disciplining of morbid living? To begin with, we must ensure that we are empirically equipped to study the ways in which knowledge of morbid living is generated on the one hand and, on the other, how practices of morbid living are shaped and 'routinised' which requires homing in on the knowledge/practice nexus. Chosen empirical sites must therefore, on the one hand, provide an opportunity to study the ways in which 'quality of life' or 'healthy life' is known, measured and invoked as a metric of morbid living along good/bad or better/worse continuums. They must also allow one to examine how patients and carers seek out, share and receive tips, advice or training about how best to live with a particular disease—how to get on when afflicted by a particular condition. Such an approach marks a break from hitherto social studies of biomedicine which have focused on molecularisation and the forms of biosociality and biological citizenship that have emerged in its wake.

I should reiterate once again that it is not my claim that 'quality of life', 'well-being' or 'healthy living' has been understudied in any way. As I have underlined, medical anthropologists and other qualitative social scientists have been researching such themes for over a century. Indeed, in a sense I am suggesting that perhaps qualitative health research deserves far more credit than it has received. Medical anthropology, for example, is not 'merely' a subjective discipline operating in the shadows of and critiquing a hegemonic biomedicine. It is a field of expertise that has contributed in concrete ways to the transformation of medical practice, in GP consultations, hospitals, patient associations as well as homes. If this is the case, what we need are methodological tools to trace the ways in which knowledge of morbid living has come to co-circulate (alongside biomedical knowledge of disease) and transmogrify into practices of morbid living.

Of course, one of the open questions that such an approach raises is the extent to which we can find links between the qualitative insights that have been generated over the past century or so about how people live with, cope with and tackle disease and illness and the ongoing efforts to know morbid living through, for example, clinical trials or patient associations. This will

require empirical studies of the co-production of medical practices which aim to improve the quality of life of patients and carers.

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Notes

1. Small sections of this article have appeared as an entry on the Somatosphere blog (http://somatosphere.net/2014/11/knowledge-of-living.html). I thank the editor for permission to reproduce these sections.
2. Tip: Type 'Living with…' followed by almost any disease into a search engine and see what happens.

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32

Bioethnography: A How-To Guide for the Twenty-First Century

Elizabeth F.S. Roberts and Camilo Sanz

This chapter describes our efforts to develop what we call "bioethnography," a research platform that combines data derived from biological and ethnographic methods to arrive at a better understanding of the larger histories and life circumstances that shape health, disease and inequality. Bioethnography is intended as a contribution to the growing insistence across the social sciences on the relationality of phenomena instead of the autonomy of objects (Barad 2007; Mol 2002; Strathern 2004). The bioethnographic research platform discussed here is made possible through our collaboration with environmental health scientists involved in a longitudinal, pregnancy-birth-cohort, chemical-exposure study ongoing for nearly 25 years in Mexico City, and collaboration between ourselves (Roberts and Sanz)—two medical anthropologists at different career stages. Platforms are raised—level surfaces on which to stand.[1] We are working to develop a platform, so to speak, combining two different methodological bundles—ethnographic observation and biochemical sampling—in a synthetic, symmetrical analysis that understands environment-body interactions as always relational, contingent and constructed phenomena.[2]

We are modeling our bioethnographic platform on forms of knowledge production distinguished, not by their objects of study, but by their methods

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for knowing the world (Mol 2002, 153). With this methodological focus, we conduct analyses that suspend in advance prevalent domaining practices that designate some phenomena (like blood-lead levels) as biological/natural, and others (like family meals using lead-glazed ceramics) as social/cultural. Our bioethnographic platform allows us to regard a phenomenon such as lead exposure and its effects as knowable through methods of both blood-lead measurement and observation of household mealtimes. Within bioethnography, then, we work against separating phenomena such as blood-lead levels and household dynamics, BMI and class hierarchy, police violence and circadian rhythms, as well as biostatistical data and coded ethnographic data. Instead, we are focused on how these phenomena emerge in coordination with each other.

Coordination, which we take from Annemarie Mol (2002), is an apt description for our attempts to bring together biostatistical and ethnographic data. In the *Body Multiple, Ontology in Medical Practice*, Mol argues that diversity of objects that go by a single name, like in her case atherosclerosis, or in our case lead exposure, involves various modes of coordination. Mol demonstrates how atherosclerosis is "enacted" through different practices such as clinical findings, blood pressure measurement, social inquiries, statistical data and angiographic images—and brought together in a patient's file. Ideally, our bioethnographic platform will coordinate the knowledge making practice of lead exposure, sleep and neighborhoods and so on, so that practices and relations involved in enacting these objects are accounted for.

Coordinating knowledge making practices while accounting for their enactment is a slow process. Inspired by the philosopher of science, Isabelle Stengers's call for a "cosmopolitics" that "slows down" "the construction of "the common world," we understand bioethnography as a method, to slow down knowledge making practices about bodily conditions. In slowing down the reasoning that makes knowing, cosmopolitics brings together disparate practitioners and practices, constituted through "multiple divergent worlds" that contribute to the making of knowledge about an issue at stake (Stengers 2005, 995). The focus on *an* issue makes the process specific, not general, as it is focused on the concrete situations in which the practitioners operate, which of course are always political. We understand bioethnography then as an exploratory cosmopolitical attempt, in which environmental health scientists' and anthropologists' obligations and hesitancies, their politics, slow down and then open out into different practices that might eventually resonate (or not) with one another (Stengers 2010). This chapter focuses on the logistics and (dis)agreements through which distinct scientific worlds (Knorr-

Cetina 1999) might be brought together into a collaborative and symmetrical work we call bioethnography.

The fact that bringing these worlds together is such a slow process makes the title of this chapter jokingly grandiose. In reality, we have much more work to do in order to engage in meaningful bioethnographic analysis. A more accurate but less tantalizing title would be "A Detailed Description of the Endless Logistical Minutiae Involved in Building a Bioethnographic Research Platform." Although Roberts has been working on the project since 2012 and Sanz since 2016, it seems as if this logistical work has only just begun. In the future we plan to write a more comprehensive guide addressing both life scientists and social scientists. In this chapter we reflect on a few key issues that have arisen so far using rather mundane examples from three specific bioethnographic investigations within the larger project (neighborhood dynamics, sleeping and eating), to provide some preliminary thoughts for other social scientists contemplating similar projects.[3] The central and simple point running throughout our discussion is that within bioethnography, logistics and methods are always theoretical and theory is always logistical and methodological. In other words, the logistical minutiae and methodological challenges we encounter along the way—such as the difficulties of meshing blood-lead levels with field notes about lead-glazed dishes in working-class households—are themselves relational phenomena that must somehow be coordinated in order to understand the relational, contingent and constructed phenomena that shape health and disease.

ELEMENT and Mexican Exposures

In l993 a team of US-based environmental health researchers partnered with research scientists and public health officials in Mexico to form ELEMENT (Early Life Exposure in Mexico to ENvironmental Toxicants). The project—primarily funded through successive NIH awards, and with administrative and some financial support from the state-funded Instituto Nacional de Salud Pública (INSP) in Mexico—aimed to study the effects of chemical exposures, particularly lead, on fetal and childhood growth and neurological development. Mexico City provided a relatively easy location to recruit cohorts that likely had some exposure to lead. Since then ELEMENT project staff have collected and conducted molecular epidemiological analysis of blood, urine, hair, toenails, breast milk and teeth samples drawn from nearly 2000 participants, mostly working-class mother-and-child pairs recruited through *Seguro Social* clinics in Mexico City. As ELEMENT continued, its scope expanded to

collect data on additional toxins (e.g., bisphenol A or BPA, mercury and fluoride) and new health concerns (e.g., premature sexual maturation and obesity), using new methods (e.g., epigenetic[4] and telomere[5] data analysis) (Afeiche et al. 2011; Ettinger et al. 2009; Tellez-Rojo et al. 2013).

In 2012 Roberts began conversations with project PIs about the possibility of conducting ethnographic observations of study participants in Mexico and the study itself, because ELEMENT's long history, vast bio-repository and current research questions appeared to offer the opportunity to develop a relational understanding of the larger histories and life circumstances that shape health, disease and inequality. ELEMENT Project PIs, Karen Petersen and Howard Hu, were intellectually welcoming of Roberts's exploratory mode and soon after she began observing ELEMENT staff meetings and spending time in ELEMENT laboratories at the University of Michigan. In March 2013 she began to conduct observations of interactions between project staff and project participants in Mexico City. In 2014 Roberts obtained NSF and Wenner-Gren funding for a 3-year project entitled "Mexican Exposures: A Bioethnography of Six Urban Families." Mexican Exposures involved 14 months of ethnographic research with ELEMENT study participants and then 2 years of follow-up work to develop a bioethnographic research platform with which to combine her ethnographic findings with biostatistical ELEMENT data. The title of the project, "Mexican Exposures," reflected Roberts's aim to expand the concept of exposure through ethnographic work, not only with ELEMENT study participants but the project's scientists as well (Roberts 2015a; Roberts 2017).

The "cosmopolitics" of Mexican Exposures so far has been shaped by the fact that Roberts came to ELEMENT nearly 25 years after its inception, meaning that her efforts have necessarily involved enrolling ELEMENT researchers in an agenda that is not their own. As we describe throughout this chapter, the "enrollment" process (Latour 1987) of environmental health scientists into a symmetrical bioethnographic research platform has been slow and full of expected asymmetries that involve divergent resource ecologies, academic positioning (i.e., hard and soft sciences) and the time the ELEMENT research team had already spent with the project. Perhaps ideally, life scientists and ethnographers would form bioethnographic projects together, finding common ground from "the ground up", so to speak, and some similarly conceived projects already have (Eisenberg and Trostle 2013). Nevertheless, the temporal and resource asymmetry that comes with collaborating with ELEMENT scientists has provided 25 years of data gathered and analyzed by intellectually innovative scientists, collected from the bodies of people in Mexico City, where Roberts had long-term interests and commitments. Thus,

developing bioethnography with an already existent project had has several advantages.

In 2014–15 Roberts worked with six ELEMENT participant families living in two different working-class neighborhoods in Mexico City. Her participatory observation focused on household and neighborhood environments and histories relevant to the production of the bodily states of these families and their neighbors. While the scope of Roberts's observations was broad—what she came to call *todologia* (described below)—much of her participatory fieldwork observations centered on the relationship of economic conditions, urban infrastructures, neighborhood environments, religious practice, kinship relations and eating to participants' well-being.

At the same time, Roberts examined the process and effects of scientific knowledge production of ELEMENT itself in accord with a fundamental science-technology studies (STS) insight—that phenomena do not merely mark the epistemological inseparability of "observer" and "observed," technology and experiment; rather, phenomena are produced through the looping ontological inseparability of "agentially intra-acting components" (Barad 2003, 815). Intra-action means *action from within* (*intra*) and refers to the mutual constitution of entangled elements, by contrast to the *inter* of interaction, which means "among" objects that maintain a degree of independence. Put another way, instead of presupposing interactions between separate entities derived from nature/culture or micro-/macrodualisms where some set of factors *determines* others, Mexican Exposures focuses on intra-dependence "between people, situations and practices (regardless of their presumed scales)" (Jensen 2007, 845). The question of what public health researchers discover about exposed bodies cannot be separated from the way researchers ask questions. Taking intra-dependence seriously meant that Roberts documented how participants' lives were shaped by their involvement in ELEMENT research (and her own), and how ELEMENT data is shaped in turn by participants' life conditions, which have been shaped by their involvement with ELEMENT.

The ultimate aim of Mexican Exposures is to develop a bioethnographic research platform that will allow us, together with ELEMENT investigators, to ask new kinds of research questions that could not be asked with ethnographic or biological data alone. From the outset it has seemed that our anthropological approach to the interplay of specific life conditions and bodily states in particular neighborhoods in Mexico City would generate a novel kind of data for ELEMENT, which, like most epidemiological studies, had previously paid relatively little attention to the specificity of participants' bodies and life worlds as located in larger histories, neighborhoods and geo-

political processes. Additionally, as in most environmental epidemiological, health-exposure studies, ELEMENT researchers have tended to investigate the mechanisms of ill health through the linear examination of the effects of single-chemical variables within individual bodies understood as universally the same (Lock 2013). We hope that our bioethnographic platform will transform standard linear data analysis models like these, which examine one variable (e.g., exposure event, telomere length or methylation pattern) at a time, by conducting multivariate analysis (data analysis that assumes that phenomena are caused by more than one variable), allowing for an understanding of how phenomena, including geopolitical processes and participant's life conditions, are created through intra-active looping, examples of which we describe below (Barad 2007; Hacking 1999; Miller and Page 2007).

In 2015 Roberts returned to the University of Michigan and soon after recruited Camilo Sanz to work as a postdoctoral fellow for the project. Together Roberts and Sanz's—our—first tasks were to set up systems to organize and manage the Mexican Exposures data collected in Mexico City and identify key directions for developing a bioethnographic research platform. This time- and personnel-intensive process involves seeking new funding sources, and constant and ongoing consultation with ELEMENT researchers about how the Mexican Exposures ethnographic data might be productively deployed to ask more nuanced research questions. Additionally, Roberts has continued fieldwork in Mexico City and incorporated Sanz into this ethnographic research.

Currently we are developing a variety of directions with an aim of combining Mexican Exposures' ethnographic data with ELEMENT's biostatistical data. We describe three of these projects (neighborhood dynamics, sleeping and eating), briefly here to give a sense of the potential for bioethnographic research.

Neighborhood Environments and Biomolecular Markers

During fieldwork in Mexico City, Roberts found that while the working-class neighborhoods of ELEMENT participants might share similar socioeconomic status (SES), differences between neighborhood environments seemed to matter in shaping bodily being. Roberts made the, perhaps, counterintuitive observation, concerning the two neighborhoods where she lived, that some forms of toxicity might be protective. In the first neighborhood, toxic boundaries such as a sewage-filled dam, cement factories and a freeway interchange served to keep out police, who are arguably the most acute threat

to the well-being of working-class people in Mexico City today. In the other neighborhood, where boundaries are unmarked and easy to cross, residents felt deeply insecure because of the constant circulation of the police.

Working with a subset of ELEMENT researchers possessing expertise in epidemiology, environmental exposure, telomeres, and epigenetic data, we are developing a method to carry out multivariate analysis that deploys ethnographic insights like these, about neighborhood differences. This will be the first time ELEMENT data will be used to examine how specific neighborhoods shape chemical exposures, allowing us to ask questions of ELEMENT's stored biological samples in novel ways. We will examine how specific conditions like social cohesion, housing stability, the presence or absence of police violence and the built environment affect the uptake of chemical exposures and produce biomarkers that index underlying physiological processes, such as stress, diabetes and sleep deprivation. In order to accomplish this task, we will conduct a neighborhood assessment scale that characterizes all of the neighborhoods where ELEMENT participants live. Currently, converting neighborhoods into numbers seems necessary for the coordination of ethnographic with biostatistical data, but we also feel hesitant about the kinds of simplifications this process will involve. We also wonder how making these correlations will complicate our sense of neighborhood environments, possibly compelling us to reassess the anthropological truism that numbers only simplify and decontextualize.

A Bioethnography of Sleep in Mexico City

In response to clinical and behavioral research that demonstrates that sleep is fundamental to our well-being,[6] ELEMENT investigators have begun to focus on sleep patterns in Mexico City. They are now in the midst of gathering and analyzing accelerometry[7] data and sleep questionnaires, seeking to examine possible links between sleep, micronutrients and smoking. In the specific case of smoking, ELEMENT postdoctoral fellow Erica Jansen has found a correlation between girls reporting having tried smoking and shorter sleep duration. If this correlation holds, physiological mechanisms might explain it, but there are also a myriad of socio-economic and neighborhood environmental conditions that might *intra-act* to shape sleep that would also serve to reshape normative scientific concepts such as "sleep quality," "sleep deprivation" and "normal sleep time."

We are now working to coordinate ELEMENT participants' sleep data to our ongoing ethnographic observations and our anthropologically informed

sense that sleep itself is a contingent relational process, varying in place, time and among differently situated sleepers (Ekirch 2006; Koslofsky 2011; Williams 2005; Williams 2011; Wolf-Meyer 2012). Roberts collected some ethnographic data on household sleep patterns during 2014–15. Her observations have already informed ELEMENT sleep questionnaires that were initially developed with measures based on US-based instruments, which tend to assume that household members have their own bedrooms and beds. With bedrooms and beds shared between siblings, parents and children, sleep in working-class households in Mexico City is often a less solitary experience than in the USA. Additionally, the predominance of informal-sector work that takes place at all hours of the day means that many working-class families have sleep patterns that would be considered nonoptimal or exceptional by normative US-based standards, but crucial for these families' well-being.

In our future ethnographic work with the six families, and with their neighbors, we plan to focus more specifically on how sleep is organized within households, when it happens, with what patterns of sleep and wakefulness. Several of the households' sleeping spaces are roomy enough that we can spend time there while inhabitants are sleeping, to observe sleep dynamics. Our bioethnography of sleep will incorporate prenatal lead exposure, phthalate levels, sleep diaries, people's abiding fears about receiving extortion calls, emotional stress resulting from job instability, use of electronic devices (smartphones, tablets, etc.), and interactions with each other during the night and day. Our goal with this research is to better understand the *intra-actions* that produce the embodied, daily rhythms of working-class life worlds in Mexico City.

Eating in Mexico City: A Bioethnography

Although the central focus of the ELEMENT study has been toxicant exposure, understanding participants' diets has been core to ELEMENT's investigation since its inception. Not only diet can be a direct source of toxicants, it can also affect the body's uptake of toxicants (e.g., calcium affects the uptake of lead). Furthermore, diet is connected to anthropometric outcomes such as obesity, which is especially relevant in the context of Mexico's designation as the world's fattest industrial nation by the WHO in 2013. Currently ELEMENT gathers diet-related data through food frequency questionnaires and anthropometric measures like BMI and body fat indices.

Roberts spent much of her fieldwork with ELEMENT participants engaged in food-related activities such as shopping, meal preparation, and eating. She

noted that in a precarious world, sharing cheap sugary and fatty foods (increasingly available through globalization) is central to forming and maintaining the social density *necessary* for survival (Roberts 2015b). Meanwhile, the Mexican public health apparatus exhorts working-class people to make "better food decisions" by halting their consumption of these types of so-called *unnecessary* foods. Roberts's qualitative data, in its documentation of everyday life in working-class Mexico City, provides insight into how the transformation in food landscapes transforms eating.

Recently ELEMENT postdoc Erica Jansen, began working with Hannah Marcovitch, an anthropology undergraduate in our Mexican Exposures data analysis lab, to bring together Mexican Exposures ethnographic data and ELEMENT epidemiological data in order to more fully understand eating among ELEMENT participants in Mexico City. The two of them are developing a way to manage, code and categorize the data that Roberts gathered and thus to provide a broader understanding of eating, food preparation and sociality. Although this effort is still in the preliminary stages, Jansen and Marcovitch are exploring different ways to coordinate these data sets into a diet analysis that examines both statistical trends and eating environments. This joint analysis will also help to identify future ethnographic focus areas for continued research with the six families involved in the Mexican Exposures project. In particular, Jansen and Marcovitch plan to trace corn in all its Mexican and globalized iterations (e.g., tortilla *masa*, corn syrup and corn snack foods, all produced at different scales (Lind and Barham 2004)) as a specific benchmark for understanding eating in contemporary working-class Mexico City households.

Building a Bioethnographic Research Platform

Building a bioethnographic research platform that will allow us to carry out projects like those described above is slow partly because the platform's development involves the epistemic, temporal and logistical coordination of disparate and differently positioned intellectual research environments. Our bioethnographic endeavor requires that we follow how lead is relationally enacted in different sites and across them, for instance, as it travels from participants' accounts about their eating habits and family meals, to blood samples and biostatistical databases and graphs. In each site, this toxicant is enacted differently and represented through various technologies; as beans are cooked and served on lead-glazed ceramics, which makes them sweeter, then eaten during family meals in Mexico City; later lead is located inside millili-

ters of frozen blood inside a test tube, which are shipped to the University of Michigan for biochemical analysis; then as free-floating microscopic particles in blood's plasma that are counted by lab technicians through specialized equipment and measuring devices; then, biostatisticians "do" lead by populating their databases with these numbers, creating graphs and tables for further epidemiological analysis. At different points in this chain, families maybe be notified if their children have high lead levels, enacting lead within households in whole new ways.

The coordination of these enactments resonates with one of our key commitments as bioethnographers: foregrounding the specificity of life in working-class households in Mexico City in relation to ELEMENT data. We do this, not to prevent the production of generalist knowledge about health, but instead to slowly allow for the comparative production of knowledge about bodily states as relational and contingent phenomena across time and space. As with lead exposure, we seek to coordinate sleep, through accelerometer data, participants' sleep diaries and ethnographic field notes—and work to understand how and why these phenomena are difficult or impossible to coordinate. We hope to investigate how sleep, as a relational phenomenon, is enacted across biostatistics and anthropology, thus satisfying, to some degree, the epistemological requirements of each world while also transforming them.

In addition to coordination, we also deploy a fractal approach that presupposes complexity regardless of the scale on which one focuses (Callon 1989; Jensen 2007; Latour 1999; Strathern 1991). Fractals are geometrical images that can be infinitely broken into smaller parts, each of which will retain similarity with the original. With a fractal approach to ELEMENT and Mexican Exposures data, we refrain from relying on a specific prioritized scale with which to evaluate how toxic substances like lead shape bodies. Instead, we work with both biostatical data about bodily phenomena and anthropological data about the same phenomena without assigning in advance a scale to either. Through fractals that assume complexity at every site, we can zoom into the microscopic particles of chemicals found in participants' blood or urine, for instance, and zoom out into people's practices. Lead, for example, may emerge not only as a conglomerate of particles embedded in bone tissue, reflecting chronic exposure, and blood, reflecting acute exposure, but also as a player in quotidian household activities—from the dinner table where household members eat beans served in lead-glazed ceramics to the couch where breastfeeding mothers may transmit lead to babies through their milk.

It is through the coordination of isomorphic fractals that our bioethnographic work emerges. ELEMENT biological sample data and data about people's daily lives can take many forms. Instead of analyzing each kind of

data as disparate and as either more or less complex than the other, we focus on the complex relationships that produce each kind of data as we work to combine them. Below we outline four phenomena that require us to coordinate complex worlds as we work to assemble a research platform that will, eventually, allow us to conduct bioethnographic analysis. Our discussion of this coordination does not fall in any order of importance, because each phenomenon is embedded within complex fractal relationships that loop back on each other intra-actively. As we described above, intra-action insists that these phenomena do not precede their relations. Rather, they emerge through them (Barad 2003, 2007).

Variable Research Ecologies, Temporalities and Concepts

Mexican Exposures and ELEMENT are located in two specific, disparate research environments that can be difficult to coordinate, spatially and temporally. Most of the ELEMENT researchers in the United States are located in the University of Michigan School of Public Health, where they work incessantly to bring in large grants for team-based research that generate vast amounts of mostly numerical data. These grants fund a wide array of master's and PhD students who provide labor for these team efforts. Faculty researchers in public health have no undergraduate teaching responsibilities (although that is about to change with a new undergraduate major in public health) and relatively few formal teaching requirements, although they do spend much of their time training students who work on their own larger projects.

Roberts and Sanz are located in a relatively well-resourced anthropology department in the College of Literature, Science, and the Arts at University of Michigan. The effort to develop a bioethnographic research platform that combines Mexican Exposure and ELEMENT data is much larger than most projects in cultural anthropology, which tend to focus on individual production and analysis of qualitative data. While Roberts's professorship is divided between teaching, administration and research, Sanz's postdoctoral work is primarily dedicated to developing bioethnography. Roberts's teaching focuses on undergraduate liberal arts education and the teaching and training of PhD students who must carry out research independent from that of their advisers. These teaching responsibilities do not provide an ideal environment for bioethnographic research: the coordination and quantity of the Mexican Exposures data alone is beyond one person, and student labor is not readily available. One solution to this labor problem has been for us to establish a qualitative coding laboratory, using Mexican Exposure data, where we can

train undergraduates, who receive course credit for their efforts. We will discuss the workings of this laboratory below, but a key point is that the project could not exist without this student labor, and the lab could not exist without Sanz's work paid for by grant funding, because the logistics of lab coordination are so extensive.

In moving our bioethnographic endeavor forward, we face other temporal pressures unusual for cultural anthropologists. To carry out a project of this magnitude, we need to spend much of our time seeking funding for nonstandard anthropological research. We also spend a large percentage of our time at the School of Public Health attending ELEMENT meetings, as a means to understand ethnographically and participate in ELEMENT's research endeavors. These meetings demonstrate how the temporal patterns embedded in each discipline (anthropology and public health) can be difficult to coordinate. Even though ELEMENT is a long-term study, it has been funded in short-term, 3–5-year chunks that require proof of publishing productivity to receive more funding (Jackson et al. 2011). Thus, publishable results come from the analysis of biostatistical data at a faster pace than we are accustomed to in anthropology. Since ethnographic data gathering and analysis proceed at a slower pace, it might appear to ELEMENT researchers that we have few results to show after 3 years of effort. The work of coordination, once again, is useful for understanding these different temporalities. There is an enormous amount of rhythmic action going on at any time in collaborative work; knowing what, where and when the action is can be an enormous challenge to us, both as researchers attempting a novel kind of collaboration and as anthropologists trying to theorize collaboration across different methodologies.

Our observations and experiences at these meetings suggest that the world of collaborative science is in fact full of such mismatches and that many of our efforts (small and large, local and systemic) to coordinate, manage or to simply live with these mismatches are constitutive of bioethnographic analysis. This means that assembling a bioethnographic research platform requires what feels like a series of compromises, and frequent acceptance of the subjection "of our work and interests" to the interests of our environmental health colleagues "in which we seek to entangle them" (Callard and Fitzgerald 2015, 105). At the same time that it feels like we must relinquish some of our own intellectual commitments (at least temporarily) to go forward, we can also see willingness on the part of ELEMENT researchers to create a more expansive and critical approach to ELEMENT data. Thus, for now: we are willing to use BMI (body mass index) as a measure to compare body size across neighborhoods despite the biopolitical moralism embedded within it (Wright and Harwood 2009); we are willing to translate our findings to the service of mak-

ing better questionnaires to speak to ELEMENT scientists, even though translation diverges from the intra-active looping that guides our efforts; and we are willing to try to turn thick descriptive Mexican Exposures ethnographic data into numbers in order to coordinate it with ELEMENT data—although it can feel like numbers deplete context. At the same time, ELEMENT researchers have been willing to reassess their use of standard individualizing public health concepts like "food decisions" and "sleep quality" as they explore with us questions of how specific neighborhood environments might affect chemical exposures or how geopolitical processes like NAFTA might contribute to diet and bodily conditions like diabetes.

A Problem of Scope and Scale: The Ethnography of *Todologia*

With over 25 years of data and counting, ELEMENT is an enormous project with researchers, staff, biological samples and statistical data distributed over three nations, at least four universities and multiple departments.[8] A method, maybe *the* method, for managing this data is turning each data point into numerical values, which has the tendency to decontextualize lived worlds and assumes that data points speak about processes internal to individual bodies. By contrast, Mexican Exposure data was collected with a commitment to the bioethnographic goal of intra-active looping, so ethnographic data collection was not limited to individuals or to a few aspects of life in two neighborhoods. Instead we have come to characterize the method of Mexican Exposures data collection as *todologia.* Built on the Spanish root word *todo* (all), *todologia* is the study of everything.[9] The term sometimes means knowing a little about a lot and at other times means a study that connects everything.

The problem is that, for now, Mexican Exposure data, gathered through *todologia*, must be organized, managed and coded in ways that will facilitate coordination with public health data. In over 4 years of fieldwork, we have produced hundreds of thousands of pages of field notes and transcripts plus over 30,000 photos that must be managed in order to be coordinated. We have organized a laboratory to harness undergraduate labor for this task, which in turn involves obtaining space where students can engage in the collective coding of the data and in training and management.

One consequence of this team approach to data management is that Roberts's field notes are read and analyzed by the 8–10 students who work in the lab at any given time. Since Roberts had never engaged in a team-based ethnographic project before, her notes about daily life in Mexico City included

accounts of her own bodily phenomena. In the spirit of *todologia*, she paid attention to neighborhood water infrastructure, plumbing, bodily conditions and gendered street dynamics and concurrently recorded her own GI illnesses, access to water for showers and bathroom conditions. At this point, making data available to students for coding seems more important than protecting Roberts's privacy. Ethnographers contemplating a project of this scale might anticipate this issue and in fact plan for team-based ethnography as well as team-based analysis as they write their field notes. An upside of the process overall has been that, almost right away, students began to note important patterns in the field notes that neither of us perceived.

Another challenge has been deciding on the most appropriate coding software. Given the volume of our data and the collective and real-time nature of the work of managing and coding it, there is no ideal program for this task. We experimented with online programs that allowed access to multiple users but could not find one able to handle the quantity of our data.[10] To deal with these issues, we turned to ATLAS.ti, an offline software package that can handle thousands of field notes and transcriptions, although it cannot handle large quantities of photos. For that, we need another program.[11] Working with offline software has pushed us toward more connectivity and intensive sociality[12]—that is, toward developing and maintaining an ongoing web consisting of a lab space with set lab hours filled with dry-erase boards, snacks, music, coffee, endless conversation, note taking, scratch paper and pens. All of these elements are intimately intertwined with the interactive practice of coding, which means intensive collective conversation as we decide on new codes and merge or delete old ones.

With respect to developing codes, *todologia* poses another challenge. Most qualitative projects focus on only a few key issues, for example, labor movements or gender and adolescence or cancer diagnosis practices and racial formation, house construction methods, herding economies and so on. While these foci radiate out into multiple arenas, a kind of filtering usually happens that helps researchers identify the relevant subset of issues that should be coded. Within the aims of bioethnography—which do not exclude any observable phenomena or maintain distinctions between the social and the biological—we have already generated over 2000 codes (and counting) and yet have coded less than 7% of the Mexican Exposures ethnographic data. These numbers are already much larger than what ATLAS.ti is used to handling. Thus, ATLAS.ti staff and developers frequently chastise us for creating too many codes. In line with an interest to expand the capacity of their product for large-scale projects, the ATLAS.ti team is working with us to develop new methods to handle this issue. As we continue to organize our bioethno-

graphic research platform, we worry, however, that our ability to manage the ethnographic portion of this data might eventually be short-circuited by the amount and breadth of what the data contains.

The main axis of our coding endeavor is the so-called Master Project. This is an ATLAS.ti file that contains all of the field notes and transcriptions amassed during fieldwork. Students in the lab are assigned satellite projects (usually by date), which contain field notes and transcripts they need to code at the sentence and paragraph level.[13] As students code, new phenomena emerge, which are assigned codes that are immediately entered into a Google.doc shared by all the team members. The Google.doc helps us create a temporary base layer of connectivity between lab members in real time. During our weekly lab meetings, we discuss all newly created codes, accepting some, renaming others and deciding against the inclusion of still others into the Master Project.[14] After this meeting Sanz merges each of the satellite projects into the Master Project and cleans the resulting master code list, a process that usually takes an entire day. When this process is complete, Sanz sends the updated master code list back to students, along with either brand new or updated satellite projects.

Synchronizing and updating the Master Project requires that *all* members of the team send their projects to the administrator at the same time. If just one satellite project is left out, the resulting list of codes will be incomplete, out of date, and unable to reflect all the most recent changes discussed during the lab meeting. This points to perhaps one of the greatest challenges of managing ATLAS.ti: the need to work in sync, coordinating different coding rhythms. Hence coding in ATLAS.ti is about constant synchronization of the collectively harnessed labor of a group of dedicated and engaged students.

Data Request #1

Currently we are working to develop the ability to link Mexican Exposure ethnographic data with ELEMENT chemical-exposure data concerning neurological development, sexual maturation and body mass. We have multiple visions for what kinds of next-order correlations we could pursue as we engage in the projects described above, but we have found that obtaining the data to carry out these correlations requires a careful coordination between our research questions and the research ecology of public health, starting with the actual act of requesting the data itself.

Originally our plan was to obtain all of the ELEMENT biostatistical data about the six Mexican Exposures families. Given our small *n,* any data about

these participants seem like it would be helpful for understanding their overall health and lives. We wanted to start making linkages between biological samples—like blood-lead levels and BPA levels—and the lives of these people whose biological substances these numbers are supposed to represent. We knew we would need to learn how to read the biological data like environmental health scientists learning, for instance, what BPA or phthalates do to a urine gravity number or how to interpret a negative blood-lead level (which seemed at first to make no sense). With the help of ELEMENT scientists, we also planned to educate ourselves on how to interpret the data of this small participant subset in relation to the larger cohort.

While our request for all the variables, as in *todologia*, about the small group of participants included in Mexican Exposures seemed simple and straightforward to us, this was not the case for ELEMENT researchers. Data managers would frown warily when confronted with our repeated requests.[15] They would immediately respond with a question that seemed more like a statement: "You want *all* the data, *all* the variables? Do you really think you would be able to work with so much data?" Requesting all the available data on our small *n* did not make sense to them—despite our explanations that, because our work is not hypothesis driven, we anthropologists cannot know in advance which specific variables are relevant and despite our assertions that the mass of biostatistical data about a small *n* would provide hunches to allow more refined research questions and to generate hypotheses about the whole cohort.

Our approach simply did not work, because database managers need to follow statistical protocols when providing information to researchers. When fulfilling a data request, for example, ELEMENT database managers need time to find the right variables within their vast data repositories and to filter, merge and arrange them in specific ways. Based on the request, the database managers decide how to organize the data distribution—whether to set it up in vertical or horizontal arrangements in spreadsheets and which variables should remain fixed in time. There is not just one arrangement for each variable within a database; there are many. The same numbers will be arranged differently to tell different stories about lead, BPA or phthalates. What conveys meaning is not the numbers per se but the relations among them and with the variables.

Thus, requesting *all* variables for a small number of participants would mean providing a massive quantity of numbers untethered to any specific question or hypothesis—which runs counter to how biostatistical databases are designed. In this world, untethered numbers lack specific meaning or scientific value. ELEMENT researchers formulate their requests in such a way that the biological data, the questions asked, and the anticipated answers

expected in return (their hypotheses) come into coordination. Data requests will not yield "meaningful" information if researchers fail to coordinate these elements or bypass data-sharing protocols and rhythms.[16]

The correct way to make a data request resonates with the work that Sanz did with oncologists at hospitals in Colombia (Sanz 2017). When Sanz asked physicians about the protocols that helped them to diagnose a cancer, their explanation boiled down to the importance of knowing how to ask the right questions. For instance, if doctors request a bone scanography of patients' bodies without first specifying what they want to obtain from the resulting image, technicians would not know how to modulate the intensity of the "camera" or where to take the picture. Likewise, according to the oncologists Sanz interviewed, it is necessary for doctors to coordinate their diagnostic suspicions and specific imaging requests with the way that technicians and radiologists conduct these exams. Similarly, specific requests for ELEMENT data questions set the conditions for different ways of arranging data and produce different answers (Bowker and Star 2000; Dumit 2004). We have had to learn that when we seek to access ELEMENT data, we need to know in advance *why* we are asking for certain variables and to specify what we expect to get from them. In time, we are fairly sure that through repeated requests for specific variables, we will have received all the data that we wanted initially but had to ask for piece by piece.

Data Request #2: Neighborhoods

We modified our data-request approach by asking for a smaller number of variables (lead levels and BMI) for all the participants in the two neighborhoods where Roberts conducted her ethnographic research. With a larger *n* we hope we might be able to provisionally examine whether ethnographic insights about difference between these two neighborhoods might appear in the biostatistical data and perhaps in relation to the cohort overall.

But then again, generating a list of participants who live in these two neighborhoods has proven to be an exceedingly complicated task, taking over 5 months to accomplish. Part of the problem has been that ELEMENT data had not before been parsed by neighborhood, and deciding the status of a neighborhood and participants' residence required intense coordination between various team members. Another issue was that our data request involved both mothers and children instead of just one or the other, across all three cohorts in the larger ELEMENT project. The majority of typical data requests are limited to one cohort and to either mothers or children, which

makes fulfilling the request simpler, since the available data is more consistently collected within a cohort.

Not surprisingly, our first data request for a list of participants by neighborhood did not go well. The data we received turned out to be inconsistent: the total number of participants per cohort did not add up to the total number of participants from both neighborhoods. Inexplicably there were hundreds of duplicates. We suspect this problem was partly due to the mismatch between our ethnographic approach and the design of the database. But it could also be the result of an inaccuracy in the merging of the data set. ELEMENT investigators have assured us, however, that initial data requests, even from experienced requesters, often don't result in usable data the first time, especially when they involve data across cohorts.

And then there is the fact that our request to arrange participants by neighborhood presented a completely a new kind of query, by introducing a new variable, neighborhood, in which to organize other variables. There were no pre-established protocols on how to merge this combination of variables. Database managers kept going back and forth between the raw database and our request. They worked to correlate space (i.e., location, neighborhood) with time (i.e., cohort recruitment dates and participant ages). Lack of coordination did not mean that data managers had failed to understand our requests. Rather, it meant that the required correlation of addresses and participants simply had not existed within the database or had not been attempted before. While the addresses, the participant's biographical information, and the toxicant levels *did* exist as variables in the database, nobody in ELEMENT had formulated the type of question that would correlate them as a precondition to generate new information. We understand our novel queries as a new apparatus that shapes the reality it measures (Barad 2007). The way we formulate our questions and submit the neighborhood data request might not only create a new merging of numbers but also possibly the conditions for new public health policies in Mexico City and around the world, geared toward neighborhoods.

One challenge has been identifying the time points when each participant was recruited to each cohort, so as to decide on the addresses that would determine their place on the list—accounting for the fact that their residential address might have changed between phases of the study. In a longitudinal study like ELEMENT (actually a study of three separate birth cohorts and, later, further follow-up studies with subsets of participants from each of those three cohorts) some participants may have been enrolled in more than one study and changed their address between studies. While limiting participants to one address reduces the complexity of their life experience, this simplification is crucial to the making of a working list that will allow us to conduct neighbor-

hood comparisons. As we tried to parse the data to produce a working list, moreover, we observed that changes in address could be a sign of housing instability—which in turn might shape bodily conditions. This is the kind of insight we plan to investigate further in our bioethnography of neighborhoods.

To fulfill our neighborhood data request, one ELEMENT database manager, Maritsa Solano, provided a manual to explain the data set we requested. Manuals allow people to agree on what is being "seen" in the data sets. Yet "agreeing" takes time and requires a learning process. When accessing biostatistical data, for instance, we must learn to "see" biological measurements through the variables and definitions included in the manual, learn how to read them (vertically, horizontally, chronologically, and otherwise), and learn to "recognize" confounders and identify values that are considered out of the normal range for specific toxicants—which for untrained observers like us would go unnoticed. "Vision" in this text, following Orit Halpern, "operates as a holding term for multiple functions: as a physical sense, a set of practices and discourses, and a metaphor that translates between different mediums and different communication systems" (Halpern 2015, 21).

In trying to master this process, we recall Bruno Latour's essay on learning to recognize different smells in the perfume industry (Latour 2004). Latour describes how trainees become a "nose," that is, someone able to discriminate more and more subtle differences and able to tell them apart from one another even when they are masked by or mixed with others. Before the teaching session, Latour writes, "odors rained on the pupils without making them act, without making them speak, without rendering them attentive, without arousing them in precise ways: any group of odors would have produced the same general undifferentiated effect or affect on the pupil" (Latour 2004, 207). Each of these trainees viscerally learned to be affected by seemingly unidentifiable differences through the mediation of the kit. In our encounters with ELEMENT biostatisticians and database managers, we are being trained to become not "noses" but "eyes." We are learning how to *see* data in hopes of coordinating ethnographic knowledge with public health knowledge, thus making new relationships possible and generating more nuanced questions and spaces for speculation and (dis)agreement.

Conclusion

As we write these lines, we have received word that our request for a list of participants in the two neighborhoods has been fulfilled. Preliminary statistical analysis by ELEMENT epidemiologist Martha Tellez-Rojo indicates that

there might be meaningful differences in children's blood-lead levels in the two neighborhoods, as was predicted by our ethnographic observations on how toxicity might serve as protection from the police. This very preliminary and tentative finding—from one rather small data set—of a difference in children's blood-lead levels between two neighborhoods is an initial modest outcome from our half-built bioethnographic platform.

By now it should come as no surprise that myriad complexities are embedded in that one preliminary comparison. These complexities involve, to name just a few things: disparate temporalities embedded in how different bodily systems engage in chemical uptake, specific relations that produce lead exposure in neighborhoods and households and the relevance of neighborhood and household in the first place. The initial finding described above compares blood-lead measures in children instead of bone-lead measures, which were also available. The two measures relate to different temporalities. Bone lead is a measure of long-term exposure and is difficult to obtain. ELEMENT researchers collected this measure from mothers at only one time point. Blood lead indicates acute exposure, and ELEMENT has blood-lead measures for both mothers and children from multiple time points. Conversations, with Dr. Tellez-Rojo, clarified that our research question about neighborhood difference would drive how to select which samples to compare—whether long term or acute exposure. We settled on the latter.

Besides temporality, our efforts to compare bodily lead levels between neighborhoods—indeed, to decide whether it makes sense to think in terms of a "neighborhood lead burden" at all—is structured by the complex relations that produce lead exposure in Mexico City. Lead exposure in Mexico tends not to be associated with the built environment in the same way it is in United States, where lead comes from house paint and plumbing pipes.[17] Since lead was banned from gasoline in 1997, some of the main known sources of lead exposure in Mexico City are leaded pottery (*trastes de barro*) and certain snack foods (e.g., candies with chili) (Tamayo y Ortiz et al. 2016). The use of leaded pottery and the consumption of these foods link to socioeconomic status, and neighborhoods are robustly correlated to SES, all of which lends support to our pursuit of data linking neighborhoods to bodily conditions.

We will face complexity, too, in our ongoing work to link biological samples to lived worlds within neighborhoods. In the future we will need to decide whether we should select and compare all the measures taken around the same calendar date, when air quality might have been the same across neighborhoods. Alternately, if we decide to focus on candy as a source of exposure, we will need to know if candies from different confectioners con-

tain similar amounts of lead. Or we can select blood-lead levels by the age of the child, since age might determine the child's vulnerability to lead. This approach allows us to know only a small thing about what's inside children's bodies at one time without reference to externalities like neighborhood or study conditions at different times. We are still in the process of making these decisions. While we continue to investigate whether it makes sense to think in terms of comparative neighborhood lead burden, we know that we need to think carefully about what we mean when we invoke neighborhoods.

Through these processes we hope to learn more, both about lead exposure and neighborhoods and about how ELEMENT biostatisticians assemble data and make knowledge. At the same time, participating in these processes provides us with an opportunity to observe how we have become interpolated in the complex work of biostatical analysis whereby a particular data request might produce knowledge. In our attempt to make a bioethnographic account, we are also generating new biostatistical knowledge. This is somewhat uncomfortable; as we are unaccustomed to being responsible for making numbers, because no matter how fervently we might insist that numbers are produced through contingent relations, we know that they tend to be treated as autonomous things in themselves (Nelson 2015). Thus, even as we offer a preliminary guide to the deliberate coordination involved in bioethnography, our ultimate goal is to keep numbers tethered to larger accounts that insist on the intra-active complexity of bodily, neighborhood and geopolitical phenomena at work within them.

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Notes

1. Our vision of a bioethnographic research platform involving observation and biochemical sampling--in a synthetic, symetrical analysis, is different and similar to Keating and Cambrosio's "biomedical platforms," a concept describing the merging of biology and medicine post-WWII, that recast the normal and pathological in productive and powerful ways (Keating and Cambrosio 2003). While bioethnography works to coordinate methods associated with the domains of nature and culture, perhaps understood as more different than biology and medicine were before their platforming, we hope that bioethnography is both powerful and productive in providing more nuanced means to understand relationships between health, disease and inequality that biomedical platforms have so often obscured.
2. A bioethnographic research platform might sound similar to what American biological anthropologists have formulated in recent decades as a "biocultural synthesis," which explores the role of culture in shaping human biology and behavior. But there are important differences. Biocultural anthropologists have contributed to the understanding of a wide range of phenomena including HIV transmission, high-altitude adaptation and lactase persistence (Beall 2006; Brabec et al. 2007; Hadley et al. 2010; Lindstrom et al. 2011; Wiley 2008). By maintaining culture as distinct from biology, however, the approach yields a synthesis that remains asymmetrical. It leaves unexamined the historical and economic conditions that continually shape biological processes and scientific study itself. In effect, this reification of culture as separate from biology is similar to social constructionism, which posits an object world separate from the ideational social world.
3. We imagine other qualitative social scientists might be interested in developing other kinds of "bioqualitative" methods to look at phenomena besides health, disease and inequality. Here we describe a model for a bioethnography of health, disease and inequality because (1) ethnography of health and disease is what we do and (2) because ethnography is arguably the most different qualitative method from what our public health collaborators already use. Their methodological tool kit already contains surveys, questionnaires and

structured observations—all of which are easily quantified. Ethnography could be made more numerical, in an ethological tradition, but up until this point, at least, we have taken a different approach.

4. Epigenetics refers to molecular mechanisms which effect gene expression located in the chromatin that envelops DNA.
5. A telomere is a region at the end of the chromosome, which displays varying levels or weathering thought to be signs of bodily insults. This weathering is measurable.
6. Researchers now claim that over 30% of the medical problems that doctors are faced with are rooted directly or indirectly to sleep alterations (Buysse 2013; Camey et al. 2012; Hoevenaar-Blom et al. 2011). From obesity to diabetes and from heart disease to cancer, sleep shapes most aspects of our health.
7. The accelerometer is a wearable technology—similar to a Fitbit— that is attached to participants' wrists for extended periods of time and allows researchers to monitor their movements' frequency, intensity, patterns and periods of rest, as well as exposure to light or darkness (through a built-in photo detector). Accelerometers also record the steps taken, distance traveled, calories burned and, of course, sleep patterns.
8. There are members of the ELEMENT research team at the University of Michigan, the University of Toronto, INSP in Cuernavaca Mexico, the Instituto Nacional de Perinatología and Hospital ABC in Mexico City, University of Indiana and Harvard University.
9. This term was provided to us by one member of an ELEMENT participant family. When she heard that we were interested in more than blood samples and food questionnaires, and that we wanted to know as much as possible about participants' lives, economic pursuits and family dynamics— but were still also interested in what the blood samples had to say, she commented, "So, you're doing "*todologia*."
10. Additionally, online programs can pose problems if more than one person is working at once. For instance, if one user is coding a data segment and 5 minutes later another user modifies that data by erasing the first user's codes.
11. For now we are coding photos through an online program called Smug Mug, but its capacities are quite limited.
12. Because it is offline, ATLAS.ti software depends on students' individual computers—their own hardware and storage capacity. Thus, when building our research team, we make sure students have enough free disk space on their computers. Because we started using the ATLAS.ti for Mac in 2015, all the technical and logistical platforms of our coding endeavor have been developed and adapted for this particular operating system. Due to incompatibilities between Mac and PC, attempting to use ATLAS.ti for PC has been not only logistically challenging but technically impossible. ATLAS.ti software had only allowed for a one-way transfer of data—from PC to Mac, but not

the other way around. For this reason, we recruited students who owned Mac computers. While drafting this chapter, however, we received good news from ATLAS.ti. After several months' wait and multiple postponements, a new version of the software was finally released, and it included the much-needed Mac-PC two-way data transfer capability.

13. This coding process is facilitated by the fact that it's relatively easy to find students who can code notes and transcripts in Spanish. Our efforts to harness student labor would be more difficult if our field language was less commonly taught in US high schools and universities.
14. Weekly lab meetings consist of 2 to 3 hours of conversations like this; during the previous week a student, Hailey Briscoe created a new "baby" code to apply to the action of a neighborhood resident covering a smelly sewer drain with a plastic bucket. Hailey located the new baby code "changing environment" under a pre-established "mother code" *BUILT ENVIONMENT*. As a group we talked about whether a clearer and more useful "baby" code, that would apply to a variety of situations in other notes, might be *BUILT ENVRIONMENT manipulating*, or *BUILT ENVRIONMENT transforming*, or more capaciously, *BUILT ENVIRONMENT manipulating/transforming*. We are well aware of the gendered and normatively kinned coding framework language we have developed in conjunction with the students (MOTHER and baby codes). It reflects both the make-up of the ELEMENT study in terms of mother/child pairs and the make-up of our lab, which is mostly female.
15. We also had to learn to identify the most appropriate database managers to whom we should pose our questions. Some are located in the USA and some in Mexico, and each has different responsibilities and database jurisdictions.
16. We also learned that data-request protocols are safeguards that seek to protect intellectual property, maintain standards of research ethics, and guarantee accuracy of information. They normalize the use of data and help data managers (and PIs) decide which variables will be shared with whom, based on researchers' questions and hypotheses.
17. Due to the use of different building materials in Mexico (cement instead of wood), paint does not contain lead. There is no research that we know of determining whether water pipes in Mexico contain lead.

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Part VI

Contested Sites/Future Perspectives

33

The Postgenomic Politics of Race

Catherine Bliss

New genetic sciences focused on the sequencing and interpretation of genomes have developed a reputation for engaging the public about racial difference in humans (Henig 2004; *Nature Genetics Supplement* 2004). Since the early 1990s, scientists have published their research alongside ethical statements on matters of race, discussed social equality in media interviews, and fostered meetings and debates about specific issues pertaining to various racial groups (Cheng and Canfield 2005; Goldstein and Willard 2005; Rosenberg et al. 2005; Rotimi and Jorde 2010). But genomic engagement with matters of race intensified with the 2000 publication of the draft map of the human genome and into the "postgenomic" era, the period following the Human Genome Project. The start of the new millennium was a time when the US federal government, the leading sponsor of the Human Genome Project and all subsequent global genome projects, was implementing inclusionary race policies throughout public health. In the late 1990s, Congress had extended the Office of Management and Budget's Directive No. 15 (OMB 1997) to all publicly funded research so that minority participation could be monitored and encouraged. Other governments around the world involved in the project were also implementing new racial justice policies. At the Human Genome Project's 2000 White House celebration, President Clinton celebrated the field's new insights into the biology of race, saying that genomics showed that

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"in genetic terms, all human beings, regardless of race, are more than 99.9 percent the same" (Office of the Press Secretary 2000). His remarks pitted genomics as a leader in egalitarian science right when public health agencies began opening up funding to research into racial health disparities from a genomic perspective (HHS 2000).

Statements about race began lighting the pages of the field's main media immediately after (*Nature Genetics* December 2001; *The Pharmacogenomics Journal* March 2001; *Scientific American* June 2003). Though experts had differing opinions on the significance of race itself, all believed that genomics, with its knowledge of the true meaning of humanity's DNA, would explain the true nature of racial difference (Schwartz 2001; Wood 2001). Amid affirmative action debates, genomics became the new science of race that would bring about a definitive taxonomy based in biological fact (Duke 1998; Winant 2004). Genome project leaders did their part by taking their views to Capitol Hill, United Nations forums, and other global political forums (Angier 2000; Chandler 2001; Stolberg 2001). At this time, they also founded the biotechnology and pharmacogenomics companies that would come to use drugs and technologies to battle racial health disparities (Bliss 2015). Just as genomics was forming its mainstay journals, associations, and funding mechanisms apart from its parent fields in genetics, and as it was embarking on forming the "postgenomic" terrain of gene-environment science, it was called to political action to define the fundamental order of mankind.

Scholars of various backgrounds responded by voicing fears about a new form of scientific racism (see Abu El-Haj 2007). They worried that new bioscientific maps of population difference would reinscribe older typological notions of race that had been proven inaccurate (Gannett 2001; Reardon 2005). Others warned of a new eugenics (Duster 2003; Nelkin and Lindee 2004; Nelkin and Tancredi 1994). Still others highlighted the political course that fields navigate as they respond to present day changes in conceptions of equality and citizenship (Duster 2006; Epstein 2007; Fujimura and Ramagopalan 2011; Jasanoff 2011; Rose 2007). Genomics, the science positioned as the contemporary science leading the fraught reconstruction of human taxonomy, was being tasked with the responsibility of creating new avenues for solving the problem of racial health disparities (Bliss 2011; Marks 2002). Thus, analysts of the ensuing postgenomic era have continued to question the significance and consequences of genomic efforts to drive a wedge between lay notions and scientific definitions of difference by making statements and policies against racism and patriarchy (Braun 2002; Fullwiley 2008; Montoya 2011; Nelson 2015), as well as its systematic implications for biology and human difference (Chow-White 2008; Chow-White and Duster 2011; Roberts 2008, 2011).

In this chapter, I examine some of the key structures of racial politics in this fast-evolving postgenomic climate. I argue that a collective sense of responsibility to promote antiracist values and to establish postgenomic sciences as different from prior genetic sciences has driven scientists to speak on race beyond the limits of their scientific milieu and to do so in ways that have taken up common political strategies of mass activism. Yet, as I will show, tactics of consciousness-raising, integration, strategic essentialism, and self-representation have been appropriated and advanced in ways that boost the field's image and influence in society without addressing the root causes of racial inequality. Scientists working in the postgenomic moment have delimited their social justice endeavors to the sphere of biomedicine, and as a result, they have recast the politics of race as the biology of race, something essential to humanity's DNA.

Data and Methods

The findings presented here are based on semi-structured, hierarchical interviews with 36 world-leading genome scientists and content analysis of all articles on genomics and race published in 1986–2010 (see Bliss 2012 for sampling and analysis protocols). This research was part of a larger ethnography of genomics consisting of participant observation of genomics and in-depth interviews with other scientists, experts, and policymakers. The scientists studied were identified as the core senior and lead scientists of the world's global human genome projects and large-scale genomic cohort studies and developers of the field's mainstay technologies (Collins and Evans 2007).[1] I conducted 1–2-hour digitally recorded interviews at scientists' North American headquarters, in which we discussed their leadership of global studies, deployment of population classifications, perspectives on pharmacogenomic therapies and biotechnologies, and experiences with consumer genomics, gene-environment research, behavior genomics, whole genome sequencing, and other subfields. I then shadowed many of these scientists, studied their labs and the genotyping facilities they employ, and observed them at meetings and conferences. Recorded data was transcribed verbatim and analyzed using a grounded theory approach (Charmaz 2005).

Racial Consciousness-Raising

At many of the world's leading research centers, elite genome scientists expressed to me their commitment to racial consciousness-raising in the general public. Craig Venter, the leader of the private effort to map the human

genome and the first scientist to create synthetic genomes, discussed the progressive importance of consciousness-raising:

> The ideal is that we all try to use our power, our positions if we have any, to try and influence the world around us. For me it is very hard to be in truly a modern society looking at the past fore-history of our species and not to be upset, be ashamed of it, extremely bothered by it.

Francis Collins, the leader of the public Human Genome Project and current Director of the National Institutes of Health, agreed. He said that scientists working in the wake of the Genome Project had a particular responsibility to educate the public about "the genetics and race interface." Both leaders positioned their field vis-à-vis a racist genetic past, a past that they believe will have to be overcome by increasing public awareness about the scientific truth of race, a truth best understood by studying DNA. While these scientists believe that DNA is but one contributor to the differences we see, they see it as a critical one that geneticists must "elaborate for the public."

Elite scientists also expressed the belief that the emerging genomic sciences owe it to the public to consciousness-raise, because they are the new face of public health. Stephen O'Brien, of the National Cancer Institute, argued:

> [I]t's the public who supports our work. It's the public who benefits from it. It's the public whom evaluates it and needs to understand it…You need to understand who's paying, who's writing the checks—the taxpayers, the community and the beneficiaries of the research that we do.

O'Brien said that the field possessed a special expertise that "the clinician or the pathologist" and "the epidemiologist" did not for solving scientific aporias of diversity and disease.

Some scientists who I spoke with went so far as to say that scientists who have not actively publicized this expertise are not true genome scientists. Rick Kittles, founder of the genetic genealogy company African Ancestry and researcher at the University of Arizona, spoke more broadly about being a scientist in the postgenomic era:

> I think that if you want to be a successful scientist and you want to contribute, you have to be very conscious of what you're saying and how you say it. And you should be able to articulate what you are doing to the lay community in a fashion that they understand and appreciate, because if you can't do that then you should probably stay on the sideline.

On television and in interviews with major news sources, like Kittles most scientific elites have criticized lay concepts of race (e.g., CNN 2001; CBS 2003). Indeed, my content analysis of genome scientists in the press demonstrated that, by 2003, they were making monthly headlines on the topic (see also Bliss 2012). Genomics had also become a leader in the trans-institute efforts at the National Institutes of Health to ramp up gene-environment health disparities research, as well as the Health and Human Services initiative to bring more racial minorities into research positions, issuing intramural statements and extramural grand challenges that foregrounded their scientific acumen and activism about race (NHGRI 2001, 2003, 2004).

The problem with this form of consciousness-raising is that unlike mass movements that have deployed speeches and public debate campaigns to get out their message, genome scientists have realized their consciousness-raising in terms of methods and engagement activities specific to genomic science's disciplinary identity and interests. Genomic consciousness-raising pivots on publicity about genomic taxonomy alternatives to common folk definitions of race. Scientists build their social justice cache on their human variation studies and their unique ability to map human variation. Many have, for example, iteratively tested the validity of folk categories of race with biostatistical technologies like admixture mapping and principal components analysis (e.g., Bamshad 2003; Goldstein and Hirschhorn 2004; Manica, Prugnolle, and Balloux 2005). Others have used their exalted position within the biosciences to engage in major media reporting on their findings in order to consciousness-raise. Esteban Burchard, of the University of California San Francisco, a researcher who routinely visits under-resourced black and Latino community centers and has appeared on shows like *The Tavis Smiley Show* (2009) and *Science Friday* (2010), for one, explained that the field's elites were in agreement that it was their responsibility "politically, and as a global community, that we have safeguards in place that don't allow for the manipulation of [genomic racial] information." He and others shared numerous cases of lab chiefs consulting each other on critical science reporting that could publicize a more ethical message about race (e.g., Burchard 2014). They criticized "clinically oriented" scientists, biomedical practitioners, and even non-genomic bioscientists, as being less aware of the true basis for human variation and its interaction with social-environmental factors. Genomic biotechnologies are thus being offered to subvert problematic commonsense notions of human difference in ways that protect and enhance the jurisdiction of genome science. Through this public engagement, scientific elites have positioned the field as the new science of race, and other experts and laypeople as those unable to correctly analyze human difference.

Such strategies are enacted to engender social equality, but they elide common consciousness-raising strengths. For one, postgenomic consciousness-raising is conducted first by technical means exclusive to genomics, and later by a public engagement equally based in the field's unique expertise. While such consciousness-raising may be enacted for the general public, this form of activism draws on data that only genomic experts can interpret. Secondly, as scientists police public appropriations of their work, they engage in a form of boundary work that reifies their authority over other fields. Thus, this form of postgenomic politics escapes an inclusive and socially oriented mass activism.

Indeed, genome scientists have not engaged in public disputes that would threaten the field's status. Scientific elites have avoided policing internal divisions, and few have challenged findings that characterize race in deterministic ways (Richardson 2011). In 2005, when Bruce Lahn and colleagues published a study that claimed that the evolution of big human brains originated in non-African populations (Evans et al. 2005), very few papers took issue with it (Timpson et al. 2007; Woods et al. 2006; Yu et al. 2006). Instead, ethical debates ensued behind closed doors (cf. Kinchy 2006; Wright 1994). Genome scientists merely continued offering up race-evaluative studies that portrayed the field as the beacon of antiracism and ensured good press for the field.

The Fight for Integration

Genome scientists, like other health advocates, are concerned about health equity; therefore, the integration of underserved populations, particularly blacks and Latinos, has become a basic social justice strategy. But scientific elites have chiefly envisioned integration in terms of access to genomic research, genetic tests, and cutting-edge therapies. Scientists like Life Technologies researcher Francisco De La Vega and Stanford University researcher Carlos Bustamante demonstrate how leaders who criticize biomedical segregation actually think of integration in terms of DNA sampling. The two began studying the genomic admixture in people of Latin, indigenous American, and African descent in 2008 so they could chart "the contribution of native American genetic variants to the disease burden in the Americas of today" (cf. European Society of Human Genetics 2010). When asked why they launched the study, Bustamante, a Venezuelan national who has received a MacArthur Genius Award, said:

> One of the reasons that researchers say they study white populations is that they're easier to study, they're more homogeneous, blah-blah-blah…But, it's

> really that they haven't really done enough to engage minority populations. (Cooper 2011)

Bustamante's comments show the frustration some minority scientists feel at the lack of integration and the paucity of effort on the part of their colleagues.

Charles Rotimi, founder of the National Institutes of Health Center for Global Genomics, also shared this sentiment. He railed against recent failures in large-scale sequencing projects, saying:

> There was an example of a new study going on, in one of the universities like Hopkins or something like that, where they did not really have good access to the African-American community. And over about a year or two years periodically they only called in one or two families!

Although these scientific elites demonstrated their sensitivity to the need for rectifying prior injustices, their comments suggested that study recruitment was the only target of change needed. Indeed, Bustamante and Vega successfully petitioned the 1000 Genomes Project, the world's largest-scale sequencing project, to include at minimum 500 genomes from African Americans from the Southwest and Southeast United States, Afro-Caribbeans from Barbados, Mexicans from Los Angeles, Peruvians from Lima, Colombians from Medellín, and Puerto Ricans from Puerto Rico. Rotimi was able to establish his own National Institutes of Health center dedicated to studies of racial health disparities and genomics in racial minorities, with a focus on people of African descent. From his post as President of the African Society on Human Genetics and leader of the first international African genome project, he marshaled researchers to create the Human Heredity and Health in Africa Project (H3Africa 2012). This large-scale international project is entirely focused on the African diaspora, being the first project to dedicate itself to a specific racial group, a group that is not geography-based but rather identity-based.

Such efforts signal a top-down institutionalization of integration in ways that bond the field to antiracist struggle. With inclusionary DNA sampling, scientists create a racially integrated network of DNA databanks that all researchers of the postgenomic era can use. They also align the field's politics with governmental policies that attempt to redistribute resources along racial lines (NIH 1993; FDA 1998). But these efforts, like consciousness-raising, wage a politics that boosts the field's reputation while popularizing genomics as a social justice science, as opposed to creating broad-based integration for the underserved.

Scientific elites also fight for integration by establishing databanks devoted to the study of particular minority groups. Esteban Burchard, Neil Risch, Hua Tang, and others at Stanford and the University of California San Francisco have banked the DNA of African Americans and Puerto Ricans, while University of Pennsylvania's Sarah Tishkoff and Yale's Ken Kidd have banked the DNA of sub-Saharan African ethnolinguistic groups. These scientists have come to be known as representatives and go-betweens for the populations they recruit and biomedicine. All the while they advance their own career interests and build the field's image. Burchard, for one, is renowned for his advocacy for Bay Area Latinos, while Tishkoff is renowned for shedding light on the variation within sub-Saharan Africa. So while scientists have adopted more pointed equal-access platforms developed by like-minded non-expert mass movements like the medical civil rights movement, they have used such platforms in uniquely genomic ways. In place of rallying community members to establish alternative healthcare systems or knowledge, genome scientists use research integration to level the playing field.

Integration through DNA sampling is markedly different than integration into basic medical institutions. The latter form of integration has the power to alleviate disparities in access and treatment, leading to greater equality between groups. Integration into genomic research does not ensure these social and physiological benefits. In fact, inclusion by race only leads to a systematization of race in research and a geneticization of folk groups in the media, taking us further from the sociopolitical bases for inequality.

A Strategic Form of Essentialism

Attempting to expand into the developing world while facing accusations of fostering a "genomic divide," genome scientists search for ways to promote egalitarian research (see Genographic 2012; Hayes 2011). In doing so, many put a postcolonial spin on their activism, further attempting to desegregate biomedicine using strategic essentialism—the positing of an essential groupness in order to garner resources for said group. With antiracist and anticolonial advocates, genome scientists argue that the most sensitive way for researchers to bring minorities into the fold is to create a sense of racial fraternity. Many adopt particular characterizations of race to embody in their own interactions or they support the strategic modeling of racial groupness on the part of minority scientists.

One avenue for strategic essentialism is in the field's minority researcher recruitment programs (HHS 2000; NIH 2002) and its special support for

institutes and individuals that capitalize on their ties to specific minority communities (NHGRI 2004). Kittles is an example in point. He has used his identification as an African American to target groups like Chicago's South Side African American community and the Kpelle and Bubi of West Africa, meanwhile garnering National Human Genome Research Institute funds to do research at Historically Black Colleges and Universities. He spoke at length with me about his political outlook on his research and then said:

> [Some researchers] don't know how to operate. They don't know how to talk to people. They don't know how to build relationships. They don't know about trust. They don't know about respect. When I go into West Africa, people see themselves in what I do and I make sure of that. I'm very sensitive, even in the studies that we do in the African-American populations. So I think that there is a level of fraternity and actual egos that create barriers to sample collection.

Kittles wants to embody a humanistic racial sensitivity in his research and create a new paradigm for enlisting minorities, but through a racially embodied sensibility—what Nelson (2008) has called "authentic expertise." Researchers like Kittles work hard so that subjects will "see themselves" in what they do and are. Kittles's remarks express the prevailing view that recruitment is a way to promote a more just and welcoming biomedicine.

Rotimi, who followed his words on integration, above, with talk of correcting past legacies of scientific racism and colonial injustice by researcher-research subject racial parity, insisted on the need for "relatable" scientists in the field:

> And somebody says, "We cannot or we do not want to participate in the study,"—but forgetting that the reason maybe you are not being successful is that you do not understand this community. You don't have somebody that they can relate to, to participate in a study like these. And of course you may also have a history of collecting this kind of information and never going back to them with your results. And so why should they come again?

Rotimi said that the outcome of a study was determined by the researcher's identity. He stressed the need to have minority scientists involved in inclusionary research (see also his statements in Goldstein and Weiss 2003). He and others discussed how the sordid history of research abuses, in which scientists measured the skulls of indigenous Americans, fatally tested or withheld drugs on Puerto Ricans and US blacks, and sampled DNA from central African populations without concern for their well-being, was the result of a failure to include minority researchers.

Indeed, Rotimi himself was advanced as a frontline minority scientist in the International HapMap Project. As David Altshuler, HapMap's Sequencing and Analysis Director at the Whitehead Institute/MIT Center for Genome Research and founding member of the Broad Institute at Harvard and MIT, explained:

> There was a discussion about who should we sample—we could've done it all in America, and that was felt to not be a good idea. That for political, more than scientific reasons, that if you were going to have a sample from Japan it would be good if it was collected in Japan as opposed to first generation Japanese Americans. It's sort of a self-determination thing, you know, that Charles [Rotimi]—somebody who is African and worked in Africa for many years—do it as opposed to someone else.

Altshuler's remarks, which move from "political" and on to "self-determination," signal the commonplace assumption that minority-led research, and research that contributes to sovereignty, is superior research. The HapMap Project's planners believed that African samples would be better collected by someone with native ties to the subject population communities, because that person would have firsthand knowledge of their political structures and social needs. They also wanted to foment lasting political ties with subject populations. Deploying researchers like Rotimi or University of California's Pui Yan Kwok, a Chinese scientist appointed to the Steering Committee who served as ambassador in Asia, enabled the project to simultaneously build human and social capital in postcolonial contexts effecting a "by the people, for the people" image of the science (cf. NIH 2010).

Strategic essentialism is thus utilized for political reasons, but in ways that advance the field's jurisdiction. Scientists advance notions of "Latino" or "black" to expand their reach across the globe in ways that stratify collections by race. While scientists see this as socio-politically sensitive and responsible science, they adopt strategic essentialism in ways that only change research. Genomics comes out appearing as a humanistic science par excellence without tackling issues outside the domain of science where inequality is born.

Self-Determination by Self-Representation

The belief that identity-based groups must represent themselves has come to permeate our postgenomic world, and nowhere is it more evident than in the new DNA science. The field first learned of the need to heed groups' own

conceptions when its second international project, the Human Genome Diversity Project, failed to take seriously the self-representations of indigenous groups and was prematurely brought to a close (Knight 1997; UNESCO 1995). Its founders came to agree that groups should be enrolled on their own terms, but the damage to its reputation was irreversible (cf. NHGRI 2002, 2004). The National Institutes of Health has since fostered self-determination by self-representation with its Ethical, Legal, and Social Implications and Community Genetics programs (GenoCommunity 2010; NHGRI 2012a,b).

Scientific elites have published widely on self-representation and its meaning for equality (e.g., Risch 2002; Rosenberg et al. 2002; Tang et al. 2005). These published views were reflected in my interviews with these and other scientists who all agreed that it was their duty to permit subjects to identify using their own affiliations. Scientists remarked that group identity needed to be affirmed in biomedical research even if this meant explicitly bringing sociopolitical categories into the research domain that may not have been present. Content analysis also showed the ways that scientists later transposed subject self-identifications to study reports in science reporting and public engagement, and the ways that their classifications got looped into journal publishing policies (Litt 2001; Phimister 2003; Winker 2008).

Scientific elites posited self-representation in overt political terms as a way of promoting diversification and prohibiting Eurocentrism. They argued that marking boxes or free-writing racial affiliations on a research form was a political act. As Pennsylvania State University's Mark Shriver illustrated:

We don't want to insult the people who are being sampled, the population, by using completely academic or terms that they would not use or terms that they find offensive!

Shriver and others criticized past research abuses, especially the field's own history of scientific racism, the academic study of indigenous Americans, and pre-inclusionary genome project foibles. They voiced awareness of researcher and research subject power differentials but were even more concerned that racial hierarchies would be reproduced if groups weren't allowed to represent themselves.

Elad Ziv of the University of California, San Francisco, expressed the common justification that self-determined labels help researchers get at social aspects of health that feed into health disparities:

> Self-report tells you a lot about social aspects or cultural aspects of that person or sometimes it correlates with socio-economic things.

The Broad Institute's Mark Daly similarly remarked:

> We can substitute racial labels in a purely genetic study with actual hard data now, but we can't do that when we're looking completely across the board and in terms of how people are treated and what access they have had to medical care and what access they have had to early life advantages, nutrition and so forth.

Daly and Ziv's remarks provide a window into the field's widespread interest in self-representation toward building a health disparity genomics. Ziv is an epidemiologist studying cancers in minority populations, while Daly is a computational biologist with no direct ties to classification debates. Yet both argued the importance of self-representation.

Encouraging self-identifications brings the potential for the perpetuation of folk racial classifications like "black" or "white"—categories that scientists have time and again criticized and which do not match the statistical groupings of genomic mapping software. In fact, it is an extra step to translate a sample's biostatistical identification to a social label. But genome scientists feel that they can be trusted to appropriately use subject self-identifications to get at a more holistic view of a person's everyday life experience. Collins, for example, remarked:

> I think realistically you would want to know for every participant, okay, what do they self identify with as far as race or ethnic group? And I think self-identification is the right answer, not what you think somebody is by looking at him across the table. And you also want to know: where did their grandparents come from? You want to know: What was their socioeconomic status and what has been their level of educational opportunity? What environment did they live in, not just the zip code, but in their local environment? What are they exposed to? You want to know about their diet. You want some measure of what degree of social stress they are experiencing.

These comments most clearly show that even folk categories are of interest. Scientific elites believe that they are capturing the widest array of health information while establishing a bond of trust between the researcher and research subject, science community, and lay community.

Scientists thus appropriate the structure of representation of social movements of race, but they again limit its application to scientific domains like recruitment and reporting. They say they are redressing racial inequality, yet they do not address stereotypes or negative representations in the political mainstream. Instead, they reduce self-representation to the marking of boxes

and the reporting of research data, two aspects of genomic practice that do not fundamentally change the terms of racial inequality.

Conclusion

This chapter has shown that while scientific elites in the postgenomic moment have used mass activism tactics to promote broad socially just outcomes in the general public, ever so consciously mobilizing around the politics of race, they have done so in ways that have primarily enriched their reputation on scientific and social fronts. In introducing more biologically essentialist notions of race, these struggles have been a detriment to the very movements with which they have aligned.

This depoliticization of race comes at a time when sciences are being asked to solve deeply entrenched social problems. Scientific elites are increasingly being recruited into positions of policymaking and science advising (Jasanoff 2005; Moore 2008; Moore et al 2011). The relationship between genes and the environment, chemicals and biosystems, and cellular development and senescence are all top agenda items for governments, who look to scientists to solve public health and environmental quandaries. Civil rights, environmental, and health activism is increasingly inextricable (Brown, Morello-Frosch, and Zavestoski 2012). And yet popular activism still does not have the backing of governmental or medical establishments, much less the global publicity that fields like genomics are afforded (Clarke et al. 2010; Nelson 2011).

Replacing political activism with science activism also has consequences for stratification, because it fosters a monopolistic principle of expertise. Postgenomic activism lays the foundation for a world where social equality can only be arbitrated by a highly select group of experts (Benjamin 2013). Such activism encourages the molecular gaze while widening the lay-expert gap and is contrary to prior community-based health activisms in which scientists enacted radical politics outlined and governed by specific social movements. In the 1970s, when American physicists used their science to counter anti-ballistic missiles and the Vietnam War with the New Left antiwar movement (Moore 2008) and physicians and genetic counselors mounted a health civil rights movement with the Black Panther Party (Nelson 2011), for example, scientists honed their efforts in support of mass activism, tailoring their work and their message to politicize issues.

When research is taken as the be-all end-all of social action and responsibility, structural harms are obscured. Postgenomic tactics have encouraged governments and the public to accept race-based pharmaceuticals as a solution

for health disparities (Kahn 2012). Indeed, the US Food and Drug Administration and the Patent Office have set new legal precedents to promote race-based drugs (Kahn 2011), despite scientists' attempts to administer race-based drugs to patients regardless of their race (Bartlett 2009; Singer 2009). This makes for a system in which drugs and diagnostics are the front-line solutions to problems that have social roots, and it absolves governments from making structural change.

In sum, science activism must be evaluated against the possibilities of more direct forms of mass activism. It is all too easy for scientists working in the current postgenomic climate to create self-serving social justice tactics that foster inequality. Knowing how science activism stops short and exacerbates injustice can help illuminate better alternatives, those that actually subvert racial hierarchies and ameliorate inequality in meaningful ways.

Notes

1. Because they are public intellectuals who are easily identifiable, I obtained their permission to report their statements with their names and titles.

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Of Rats and Women: Narratives of Motherhood in Environmental Epigenetics

Martha Kenney and Ruth Müller

Introduction

"Darwin and Freud walk into a bar. Two alcoholic mice—a mother and her son—sit on two bar stools, lapping gin from two thimbles. The mother mouse looks up and says, "Hey, geniuses, tell me how my son got into this sorry state." "Bad inheritance," says Darwin. "Bad mothering," says Freud.

For over a hundred years, those two views—nature or nurture, biology or psychology—offered opposing explanations for how behaviors develop and persist, not only within a single individual but across generations. And then, in 1992, two young scientists following in Freud's and Darwin's footsteps actually did walk into a bar. And by the time they walked out, a few beers later, they had begun to forge a revolutionary new synthesis of how life experiences could directly affect your genes—and not only your own life experiences, but those of your mother's, grandmother's and beyond."

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Mice, mothers, nature, nurture, groundbreaking new research and scientific mavericks—these are some of the stock characters that populate current media representations of epigenetics, the research field heralded above as a "revolutionary new synthesis" of formerly separate research areas that explore the developmental effects of nature and nurture. Epigenetics, as many readers may already know, is a rapidly expanding branch of molecular biology that studies changes in gene expression that are not caused by underlying DNA mutations, that is, a change in the genetic code itself. Rather, researchers explore how chemical modifications *on* the DNA, such as methylations, effect changes in gene expression by regulating which genes can be accessed and transcribed and to what degree. These modifications have been found to initiate, silence, increase or decrease gene expression. While epigenetic regulation is being investigated as fundamental to many different processes within an organism, for example, cell differentiation in basic development, the branch of epigenetics most prominent in public discourse is environmental epigenetics. This subfield investigates how stimuli from the environment can change gene expression via epigenetic mechanisms. This notion of environmental stimuli includes chemical signals, such as toxins or food, but also social experiences and lifestyle. In the media and public discourse, the subfield of environmental epigenetics has by and large come to stand in for epigenetics in general.

Experiments on the influence of maternal biology, behavior and experiences on the epigenetic make-up of her progeny have attracted particular attention. Like the article quoted above, virtually all media reports on epigenetics start their stories about its groundbreaking potentials for better understanding gene-environment interaction by recounting a series of epigenetic experiments that studied how a mother's biology and behavior affect the physical and psychological well-being of her offspring. Due, in part, to this considerable public interest, research on how the experiences and behaviors of mothers, grandmothers and great-grand-mothers "sculpt" and "program" (McGowan and Szyf 2010, 66) the psyche and physique of their progeny over generations is receiving significant scientific attention and is currently expanding. Hence, while epigenetics itself is a much more heterogeneous field, Richardson (2015) rightly argues that both in the popular media and within the primary literature of environmental epigenetics, "[t]heories, data, and experimental paradigms arising from studies of maternal effects have at this

time become canonical to the science of epigenetics writ large" (218). The effects of a mother's biology, behavior, lineage and life circumstances on her offspring's physiological and psychological composition have come to, almost synecdochically, stand in for the provocative proposition that the field of epigenetics will be able to resolve the nature/nurture dualism and show how our nurture becomes our nature, too.

Various authors have drawn attention to how a focus on mothers in epigenetic research and in the media reflects and perpetuates problematic scientific and societal histories of rendering mothers crucially responsible for the health and character of their children. In a commentary in *Nature*, a group of social and biological researchers recently warned against tendencies to "blame the mother" (Richardson et al. 2014) that have been surfacing in and around epigenetics. Others have shown how these tendencies are particularly pronounced in studies that address women with low income (Singh 2012) and women of color (Mansfield 2012). In this article, we contribute to this emerging critical debate about the paradigmatic role of research on mothers and maternal effects in epigenetic research and its representation in society. We provide a fine-grained analysis of figures of motherhood that arise in a selection of research papers and news articles in key scientific journals, as well as in teaching materials. Focusing on key experiments that are cited again and again as paradigmatic to the emerging field of environmental epigenetics, we will investigate narratives about mother-offspring relationships as they are both produced by and inscribed into experimental designs, data interpretation and epigenetic narratives that appear in scientific journals and the popular media. In this analysis, we will pay particular attention to how these new stories are old stories, too, reflecting long-held societal and scientific "pre-ideas" (Fleck 1979 (1935))[1] about good and bad mothers and mothering practices.

We are writing this paper with three different audiences in mind: our colleagues in Science and Technology Studies (STS) and neighboring fields, who are working on epigenetics and related developments in the life sciences; life science scholars seeking critical engagement with current developments in their field; and interested students across the disciplines, who are being introduced to critical analysis of scientific knowledge production in undergraduate and graduate university classes. Concerning this third group, we believe that there is currently a lack of articles that both engage with new developments in the sciences and take interdisciplinary teaching into consideration in their style of writing and their analytic approach. This often forces STS scholars to resort to older texts when searching for easily accessible case studies to teach in their classes.[2] Training students for interdisciplinary conversation and col-

laboration requires that we both sensitize them to historical continuities in scientific practices and help them critically engage with current developments. Emerging fields like epigenetics, which by definition cross the boundaries of disciplinary territories, make it ever more important to invest in interdisciplinary dialogue and training at the student level. We hope to contribute an article that can support these kinds of engagements across the university.

The Biology of Sex, Gender and Motherhood

In the field of feminist STS, we find a rich tradition of investigating how cultural preconceptions about sex and gender enter scientific research processes and data interpretation in the biological sciences. Scholars such as Ruth Hubbard (1979), Evelyn Fox Keller (1983), Donna Haraway (1989), Londa Schiebinger (1993) and Anne Fausto-Sterling (2000) have written influential, field-shaping accounts of how preconceived ideas about the nature of the sexes and their social roles have shaped biological and biomedical knowledge production about the sex/gender complex in fields as diverse as anatomy, endocrinology, genetics and primatology.

One important focus of analysis within this literature is how throughout the history of modern biology the sexes have been constituted as binary and dichotomous, having distinct physiological, psychological and behavior traits based on the perceived biological/social roles. For women, sexual difference has most predominately been figured through reproduction and motherhood. Ruth Hubbard (1979) and Joan Roughgarden (2004, 2009), for example, show how notions of sex difference across species in the work of Charles Darwin are inspired by Victorian stereotypes, which featured active, aggressive males and passive, coy females. In Darwin's theory of sexual selection, the males of any given species compete for access to females, while females are concerned with choosing a good mate and raising the young. Competition between males becomes the driving force of evolution, leading to development of physical and mental qualities that increase mating frequency and offspring number; females merely inherit these traits through their fathers.[3] Female biology, on the other hand, is understood to primarily evolve to best serve the role of mother and caregiver, producing a body that is physiologically, psychologically and behaviorally adapted to and for maximizing the survival of her offspring. These assumptions about the active male and passive female remain present in the twentieth century figures of Man the Hunter and Man the Toolmaker, where men are credited with evolutionary innovation and women, figured as cave-bound mothers, are hidden from view (see also Haraway 1989).

Feminist science studies scholars have shown that these Darwinian notions of sex difference organized around reproductive function are not limited to evolutionary biology; in many different fields of biological research, female bodies have often been understood and constructed first and foremost as maternal bodies. Oudshoorn (1994) shows how in the conception of the so-called male and female sex-hormones (e.g. estrogen, progesterone and testosterone), similarity and variability between the sexes have been erased in favor of a clear-cut dichotomous concept of hormones that emphasizes and standardizes difference and ties female hormones ever more strongly to reproductive functions. Richardson (2013) recently demonstrated that similar processes are at work in the constitution of what is today known as the female X and the male Y chromosome, while Fine (2010) investigated and dismantled the continuous construction of the brain as sexed in terms of men and women showing innate structural and cognitive differences.

This Darwinian concept of women's bodies as primarily shaped by and adapted to motherhood influences how research questions are asked and investigated and how results are written up and circulated (Roughgarden 2004, 2009). When women are understood as evolutionarily adapted to being optimal gestational environments and caregivers, women's bodies become de facto sites for research and intervention in relation to the health of her offspring. For example, recent childhood obesity research focuses predominately on mothers, from the health of the pregnant women, to breast feeding, to how she feeds, cares for, and parents her child, often to the exclusion of larger social, economic and structural factors (Maher et al. 2010; Boero 2009). The biological understanding of a woman as the natural and most important environment for her offspring can lead researchers to focus their research questions on mothers without questioning the pre-ideas about sex, gender and sexuality that have informed the underlying framework (Richardson et al. 2014).

"Metaphors into Hardware": The Politics of Experimental Design

These pre-ideas about sex, gender and sexuality are not only visible in the gendered language used to describe women's bodies or the morally charged language around motherhood but also in the way that scientific experiments are designed and interpreted. Donna Haraway (1989) uses a historical example to illustrate how psychological research conducted on model organisms has often been designed to answer unresolved social questions and thus incor-

porates available social categories into the experiments themselves. In her chapter, "Metaphor into Hardware: Harry Harlow and the Technology of Love," Haraway analyzes the experiments of influential American psychologist Harry Harlow (1905–1981), showing how the experimental design and laboratory apparatuses embodied post-WWII anxieties about changing American families. Using rhesus monkeys as model organisms, he studied attachment (often coded as "love") between infants and their mothers, fathers and peers. In Harlow's famous experiment in which a baby rhesus monkey is given a choice between a "wire mother" (which dispenses milk but provides no tactile comfort) and a "cloth mother" (which is soft but does not provide milk), the experimental design reflected concerns about the adequacy of biparental care as women entered the workforce. In later experiments, Harlow introduced the baby monkeys to dolls of differing temperatures known as the "ice cold mother" and the "hot mama," literalizing the common characterizations of good mothers as "warm" and bad women as "frigid" (240). Haraway argues that this range of surrogate mothers demonstrates how Harlow and his fellow researchers were not only studying attachment; they were turning "metaphor into hardware" (236).

In order to make these studies on rhesus monkeys relevant to the human problems that concerned Harlow, the experiments needed to be designed to suggest an analogy between the lives of laboratory monkeys and the social world of humans. For example, Harlow devised a cage dubbed "the nuclear family apparatus" that was designed to measure how the post-WWII US nuclear family affects childhood development: "Harlow speculated that the enriched environment, the nuclear family, produced confident primate children, ready to excel, in metaphoric contrast to lower class human children whose family deprivations might result in impaired personalities and so low achievement" (242). Given that rhesus monkey social structures in the wild and in the lab do not resemble a 1950s American nuclear family, the "nuclear family apparatus" actively created a resemblance that allowed the results to travel between Harlow's laboratory and the social context he sought to intervene in. With these and other experiments, Harlow's laboratory produced powerful scientific stories about mothers, fathers and infants that informed how people thought about good and bad parenting practices in the post-WWII United States. Haraway concludes: "In the measure of love is the literalization of sexual politics" (243).

Recent accounts of Harlow's experiments have emphasized how the rhesus monkey did not always conform to the expectations of the researchers, which lead to new research questions, for example, about the importance of peer relationships (Vicedo 2013). What remained a constant, however, was that

the rhesus monkeys and their social life were always seen as stand-ins for humans. Haraway's analysis draws our attention to how the language and laboratory apparatuses used in Harlow's lab reflected contemporary social questions and were designed to produce parallels between humans and model organisms. This is instructive for our own analysis of figures of motherhood in the literature on the epigenetic effects of maternal care, which are based primarily on experiments conducted in rodents. Following Haraway, we will show how social questions are built into laboratory apparatuses and trace the translations made between humans and model organisms. As we look at the gendered politics of epigenetic research and storytelling, we will highlight these often-silent analogies and examine their epistemological and political consequences.

STS Engagements with Epigenetics

In recent years, scholars in STS and neighboring fields have begun to engage with environmental epigenetics (e.g. Niewöhner 2011; Landecker 2011; Singh 2012; Mansfield 2012; Landecker and Panofsky 2013; Meloni 2015). A number of authors (e.g. Pickersgill et al. 2013; Meloni and Testa 2014) suggest that environmental epigenetics is a topic that deserves more attention from STS scholars, not only because it is an increasingly prominent research field but also because it works with social categories that have traditionally been understood to lie within the purview of the social sciences and the humanities. Rather than signaling the beginning of a "turf war," a growing number of scholars have argued that environmental epigenetics can offer new occasions for "constructive and creative collaboration between social sciences and developmentalists" (Singh 2012, 319). They suggest that social scientists and bioscientists can combine their expertise and work together to create novel accounts of biosocial complexity (Niewöhner 2011; Singh 2012; Rose 2013; Meloni 2013).

This is an important project that we are also committed to. Yet, we are unconvinced by recent suggestions that for this endeavor to succeed, social scientists will need to leave behind a critical stance toward the biosciences (Fitzgerald et al. 2014; e.g. Rose 2013). Hence, we would like to align ourselves with scholars such as Singh (2012) and Niewöhner (2015), who suggest that critical and collaborative approaches are not mutually exclusive. Singh in particular argues that both approaches are useful and necessary for productively engaging the field of environmental epigenetics, as it is crucial to both *reflect on* and *contribute to* the shaping of social categories as they enter bio-

logical research. We would add that these practices are mutually dependent and can invigorate and enliven one another. For example, a feminist STS analysis that is critical of reductionism in the biosciences can create openings to elaborate rich explanatory models that take adequate account of biological and social complexity of sex and gender. Critique in this sense becomes not an act of judgmental distance but of intimate engagement (Puig de la Bellacasa 2011).

This understanding of the practice of critique draws attention to an important aspect of the history of feminist STS. Many feminist STS scholars, including those discussed above, have a background in the natural sciences and a deep love of that material-semiotic hybrid we think of as "the natural world."[4] For us, the purpose of critique is to draw attention to the political and epistemic limitations of current scientific practice in order to offer "a more adequate, richer, better account of a world" (Haraway 1988, 584). In other words, critique is not about dismissal or remaining above or outside but about inhabiting and participating meaningfully in technoscientific worlds. As Donna Haraway writes, "I will critically analyze, or 'deconstruct,' only that which I love and only that in which I am deeply implicated" (1997, 151). Within this tradition, we argue that in the case of epigenetic research on maternal effects, it will first be essential to develop a detailed understanding of the historically and culturally situated pre-ideas of sex, gender and motherhood and their implicit moral discourses that are currently active in epigenetic research *in order to* contribute to the development of better categories in collaboration with epigenetic researchers.

Current epigenetic notions of sex, gender and motherhood also need to be understood within broader epistemic frameworks characteristic of epigenetics. Niewöhner (2011) argues that environmental epigenetics can be understood as a form of "molecularization of biography and milieu"—a concept Meloni and Testa echo when they talk about the "digitization" of the environment (2014). Both notions point to the ways in which epigenetics experiments seek to make the environment both measurable and directly accountable for molecular effects within the body. In order to quantify and measure the environment, researchers look for proxies to stand in for the complexity of the social and material environment: single toxins or dietary components, or quantifiable activities. In the case of the influential experiment on "Epigenetic programming by maternal behavior" (Weaver et al. 2004), which we will explore in detail in the next section, the licking and grooming of pups by their mother comes to stand in for the value-laden category of "maternal care."

We see two major problems with operationalizing the environment in this way. The first one is epistemic: while valuable in some cases, in others it is

questionable in how far such reductionism can offer analyses that are capable of withstanding the actual complexity of the environment and its multiple, ongoing relations and interactions—a concern that appears to be shared by some epigenetic researchers as the field expands and gains more scientific and popular attention (Darling et al. 2016). The second is socio-political. In a case study of the controversies surrounding methylmercury exposures that result from eating fish, Becky Mansfield (2012) shows how linking health risk to single molecule instead of more complex social and environmental phenomena can easily shift responsibility onto already vulnerable individuals. In the US context, Native Americans, immigrant populations from Southeast Asia and the Caribbean and urban subsistence fishers are more likely to be exposed to high levels of methylmercury that can affect fetal neurodevelopment. However, rather than address environmental concerns related to industrial pollution, the US Environmental Protection Agency (EPA) has issued fish consumption warnings targeting women from these groups, who are of "reproductive age." Here we can see how the molecularization of an environmental problem shifts the burden from the collective to the individual enforcing social norms around diet. Mansfield argues that policies like these "change the problem from contamination itself to the abnormal diets of these women" (2012, 352). The environmental justice framework, which links uneven toxic exposures to socio-economic and racial inequalities (e.g. Bullard 2011), is de-emphasized in favor of an individualized approach. Political scientist Maria Hedlund (2012) argues that it will be vital to frame arising notions of "epigenetic responsibility" in terms of political rather than individual responsibility and attribute it to those actors able to make systemic rather than individual changes, such as states or corporations.

Mothers are often at the center of these debates about emerging forms of epigenetic responsibility. Sarah Richardson has argued that in an epigenetic discourse that is mostly concerned with how maternal physiology, exposures and behaviors can harm the fetus and the infant, mothers become primarily "vectors of developmental or epidemiological risk" (2015, 227) within an emergent "explanatory landscape of postgenomic science" (211). Our article sets out to analyze the specificities of this maternal figuration as it is being produced by and inscribed into experimental designs, data interpretation and scientific narratives in epigenetics, thereby refiguring, refuting and reinforcing pre-held ideas about the role of mothers in the psychological and physical health of their offspring. In this process, we show how key assumptions and knowledge claims about the epigenetic effects of maternal care and their wider social significance are created and upheld through dense speculative cross-traffic between epigenetic studies in rodents and psychological and epidemio-

logical studies in humans. We trace how in these multiple steps of translation and circulation between different species and different disciplines, simplified and remarkably stereotypical notions of maternal agency and responsibility often travel between contexts without much scrutiny and are, in the process, reinforced and solidified rather than critically questioned and opened up for novel interpretation. As epigenetic reasoning is increasingly invoked in public health debates, we identify a need for critical interventions from fields such as feminist STS that challenge these all too smooth translations and instigate interdisciplinary conversations about how to elaborate thicker, less familiar, more complex and situated forms of exploration and experimentation in epigenetic research.

Mother Nurture: Maternal Care and Epigenetic Programming

If there is one experiment that has become paradigmatic for the science of epigenetic maternal effects, it is the experiment on "epigenetic programming by maternal behavior" (Weaver et al. 2004) conducted by researchers from the labs of neuroscientist Michael Meaney and geneticist Moshe Szyf at McGill University in Montreal, Canada. This experiment explores the epigenetic effects of how much a rat mother licks and grooms her pups—what the researchers call "maternal care." The researchers at McGill argue that the degree to which rat mothers lick and groom their pups when they are young shapes the pups' epigenetic profile in genetic regions related to brain development. These epigenetic modifications change gene expression and, consequently, the number of glucocorticoid receptors in their brain. Frequent licking and grooming leads to a high expression of the glucocorticoid receptor gene and hence to a high number of receptors, and less frequent licking and grooming to a lower expression and low numbers of receptors. The McGill group argues that a lower number of receptors alters stress responses and induces more anxious and aggressive behaviors in the offspring. This change is believed to remain stable throughout the offspring's lives.

Below is a transcript of a video of Moshe Szyf explaining this influential experiment to high school teachers from all over the United States during a summer school at the Genetic Science Learning Center of the University of Utah in 2008. The video can be found online at the interactive public learning platform "Learn.Genetics" hosted by the University of Utah's Genetic Science Learning Center,[5] a website that received almost 20 million visits in

2013. Information is mainly geared at students and teachers, providing them with learning materials to understand basic principles of, as well as latest development in, genetic science. Szyf's re-telling of the experiment is a characteristic instantiation of how narratives about rats, humans, species-typical behaviors and broader social concepts such as care and love intermingle in the epigenetic study of maternal effects:

> So, the way a mother rat takes care of its pups is by licking and grooming, nipple switching and arched-back nursing. So there are rats that do a lot of licking and grooming, and there are rats that do very little, but most rats are in between. So that resembles human behaviour as well. Right, you have mothers that are highly mothering and mothers that could not care less. And most mothers are somewhere in between. So if you look at these rats, so all you do is you observe them and you put them in separate cages, so you put the high lickers in one cage—not the mothers, but the offspring—and the low lickers in another cage. And then you let them grow and they are adults now. The mothers are long buried. And you look in the brain and you see that those who have high licking mothers express a lot of glucocorticoide receptor gene and those who are low lickers express low. That reflects the number of receptors and that results in a different stress response. But this is not the only difference. We found later on there are hundreds of genes that are differently expressed. So if you get a [genetic] mutation, you know a polymorphism once in a million… here, just the motherly love changes hundreds of genes in one shot. And it changes them in a very stable way so that you can look at the old rat and you can say whether it was licked or not. But you can also say by behavior. So if you walk to the cages, to the room, the rats that were poorly licked are highly anxious, hard to handle, aggressive, and the rats that were very well handled as little pups, they are much more relaxed, much easier to handle. So, you know, like every technician in the lab knows looking at the adult rat, how it was licked when it was a little pup. And the question of course is—mechanism. How does this work?
>
> (Moshe Szyf at "Beyond the Central Dogma Summer Institute," Genetic Science Learning Center, University of Utah, 2008. http://learn.genetics.utah.edu/content/epigenetics/rats/)

There is one sentence here that offers a particularly striking condensation of this multi-step translation: "Just the motherly love changes hundreds of genes in one shot." In this one sentence, a well-defined set of quantifiable rat behaviors—"licking and grooming, nipple switching and arched-back nursing"—is quickly translated into "motherly love," a human term that is arguably not quantifiable.[6] Szyf also tells us that there are rat mothers who lick and groom their pups intensely and others who don't—just like in humans, he continues,

where some mothers are highly "mothering" and others "could not care less." The amount of licking and grooming—or love as Szyf suggests—changes "hundreds of genes in one shot," shaping the pups' mental and physical well-being for life. Everyone knows which rats have been licked more or less as pups, as those who have been licked less are aggressive, anxious and hard to handle.

Attending to the narrative dimension of Szyf's account, we want to explore figurations of motherhood that are emergent in this scientific story. A first striking figuration is what we call the *expanding mother*. By focusing on licking and grooming as the key decisive factor for epigenetic development regarding stress, while at the same time black-boxing the rest of the environment (e.g. the cage, the food, the other rat pups), the mother increasingly comes to stand in for the whole environment of the infant rat. Her actions determine what kind of rat her pup will become as they shape its epigenetic profile in very stable ways. She is not only the first environment it has ever known, but she remains with it throughout its life, shaping its ways of relating to every environment it will ever encounter. In this version the mother is inherently powerful but also dispersed and depersonalized. She comes to stand in for "nurture" in ways that are similar to how nature is traditionally personified as female: just as Mother Nature engulfs her children, follows her own laws and is sometimes cruel and sometimes merciful, Mother Nurture programs her children's epigenome and determines much of their fate.

In Weaver et al. 2004, the McGill Group focused their attention on "natural variations" of maternal care. They observed which rat mother licked and groomed their pups more or less, separated the pups according to how much they were licked and then examined their behavioral response to stress caused by being placed in small (8.5 x 21.5 cm) plexiglass boxes for 20 minutes at a time. The rat brains were also dissected and analyzed for differences. However, the McGill Group assumed that variations in maternal behavior do not just occur at random but ultimately have environmental causes. The working hypothesis was that the difference in maternal behavior represents different environments, some that are more threatening and some that are less threatening. Weaver et al. call this "a forecast of the environmental conditions" (2004, 852) through maternal behavior.

In later papers this hypothesis was experimentally explored by exposing pregnant rats to stressful environments during the last third of their gestation period and observing how much they licked and groomed their pups (Champagne and Meaney 2006). Rat mothers were put in the plexiglass restrainers for 30 minutes 3 times a day at random intervals for the last 7 days of pregnancy. Rat mothers who had undergone this procedure were subsequently observed to lick

and groom their pups less, even if they had shown different behaviors with previous pups. This, the researchers argued, confirmed that maternal behaviors act as cues that help the offspring predict environmental conditions.

Here mothers are figured quite differently than above, and inherently less powerful: rather than agents themselves, they become pliable mediators of the environment that they themselves are experiencing. Behaviorally vulnerable to environmental experiences, this figure of the *mediating mother* is programmed by her environment in the same way as she programs her offspring. Her behavior is not her own; it is a mediation of the environment.

Harm as Adaptive: Epigenetic Programming and Class Mobility

It is the combination of both these figurations—the figure of the expanding mother, who stands in for the entire environment, and the figure of the mother passively mediating this environment—that allows for a specific line of interpretation. The dominant scientific narrative about why less maternal care leads to heightened stress response suggests that the infant rats are not in fact harmed by the uncaring maternal behavior that causes them to become more anxious and aggressive. Instead, these behavioral traits become refigured as adaptations to the environments that they are most likely to live in. Michael Meaney writes: "These patterns of transmission likely reflect very adaptive patterns of development. Children inherit not only genes from their parents but also an environment." He goes on to quote Francis Galton, co-founder of the eugenics movement: "Englishmen inherit England, as Galton remarked." (Meaney 2001, 1181). This passage is remarkable on a number of levels: First, we find it surprising that Meaney would quote Galton without acknowledging that Galton was one of the founders of the eugenics movement; since the racist and colonialist implications of eugenic science are now widely known (e.g. Roberts 1997; Stern 2015), it is equally surprising that this quote passed through peer-review unchallenged. Second, it demonstrates once again how quickly researchers often move between the rat and human context. Finally, it is exemplary of how in this line of research arguments *initially* about harm to the infant are often conceptually reframed as adaptive to specific environments, particularly, as we shall see, in the context of narratives that focus on individuals of low socio-economic status.

Examining these three features helps us understand some of the key practices through which the publications and teaching materials about the epigenetic effects of maternal care perpetuate troublesome narratives about

class and ultimately racial belonging.[7] Consider this passage from the Learn. Genetics homepage that follows the description of the experiment on the epigenetic effects of maternal licking and grooming:

> Anxious Behavior Can Be an Advantage
>
> In our society, we think of anxious behavior as being a disadvantage. But that's because, for the most part, we live in a nutrient-rich, low-danger environment. In the rat equivalent to our world, the relaxed rat lives a comfortable life. It is likely to reach a high social standing, and it doesn't have to worry about where its next meal is coming from. An anxious rat, on the other hand, doesn't do so well. It is more likely to have a low social standing and suffer from diabetes and heart disease.
>
> In another environment, however, the tables turn. The anxious, guarded behavior of the low-nurtured rat is an advantage in an environment where food is scarce and danger is high. The low nurtured rat is more likely to keep a low profile and respond quickly to stress. In the same environment, a relaxed rat might be a little too relaxed. It may be more likely to let down its guard and be eaten by a predator.
>
> (http://learn.genetics.utah.edu/content/epigenetics/rats/)

As in much of the scientific literature, here the epigenetic modifications induced by less maternal licking and grooming are framed as adaptive traits that prepare the rats for their harsh future environments. If the mother is stressed because of her environment, then it is probable that the offspring will also live in a stressful world. Anxious and aggressive behavior might offer better chances of survival in such an environment than being calm and peaceful. With this argument the text interweaves rat lives and human lives almost seamlessly: while reporting on rat experiments, it speaks of "high and low social standing," of "keeping a low profile" and of proneness to "diabetes and heart disease"—all anthropomorphic categories. The text argues that "anxious rats don't do well in relaxed worlds," while a well-licked and hence more relaxed rat might "let its guard down" too easily in the life world its less licked cousin is adapted to.

Again, we want to stress that these class inferences are not only present in more popular scientific representations of the epigenetics of maternal effects, they echo the language used in the primary peer-reviewed literature. Particularly in the introduction and conclusion sections of articles in this field, researchers support the relevance of their model organism experiments by offering comparison to problems of human society, for example, class-based differences in stressful experiences like violence and crime. Here we often find less-than-rigorous citation practices that align the experimental

findings in rats with previous studies in primatology and human psychology, often leaving aside questions of translatability between species and between the lab and human society.

Below is an example from the above mentioned 2001 paper by Michael Meaney. His article, reporting mainly epigenetic findings in rodents, includes this paragraph early in the concluding section:

> The key issue here is that of the potential adaptive advantage of the increased level of stress reactivity apparent in the offspring of low LG-ABN[8] mothers. […] In the current context, the research of Farrington et al. (1988) and Haapasalo and Tremblay (1994), for example, on young males growing-up in low socio-economic status (SES) and high crime environments provides an excellent illustration of the potential advantages of increased stress reactivity. In this environment, the males who were most successful in avoiding the pitfalls associated with such a "criminogenic" environment were those who were shy and somewhat timid. Under such conditions, a parental rearing style that favored the development of a greater level of stress reactivity to threat would be adaptive. (Meaney 2001, 1182)

Here the relevance and meaning of the rat findings for human society is constructed through comparison to psychological studies in humans. The specific studies of Farrington et al. (1988) and Haapasalo and Tremblay (1994) are frequently cited by researchers of the McGill Group (Meaney 2001; Champagne et al. 2003; Meaney 2010; Champagne and Meaney 2006; Szyf et al. 2005; Cameron et al. 2005; Zhang et al. 2006; Zhang et al. 2004; Meaney 2004; Caldji et al. 2000; Meaney et al. 2007; Nguyen et al. 2015; Meaney et al. 2015). However, in the literature on the psychology and sociology of crime, these studies are not undisputed. For example, critics have argued that Farrington's influential study of crime causation is overly centered on the individual and therefore does not sufficiently address larger structural issues, which tends to "pathologize the individual while ignoring both the propensity for change over the life course, as well as how social factors operating at both the macrostructural level and at the micro level (i.e. intimate relationships) contribute to criminal and antisocial behaviour." (Buffone 2012, 908). Yet, the way in which Farrington's work is referenced by the McGill group seems to suggest that there is a uniform consensus between psychology and epigenetics about these issues, a consensus that is neither true on the side of psychology nor on the side of epigenetics (c.f. Pickersgill 2016).

Nevertheless, by means of this comparison in this and similar articles, human "parental rearing style," just as rat parental rearing style, is figured as epigenetically programmed and human parents are figured as obediently

mediating their environmental conditions to their child, through abuse and neglect, just as the high and low licking and grooming rat mothers. What we see emerging are narratives about how the social and material environment becomes hard-wired into human bodies and minds. In line with the epigenetic and psychological proposition that the first years of life—often figured as a key window of developmental plasticity—are particularly formative for who we will become, epigenetics opens a door for thinking of individuals—rodent as well as human—as well adapted to one environment, and at the same time poorly adapted for another. As the rodent and human studies are implicitly made analogous in the article, the stressful cage environments of the rats become the "criminogenic" neighborhoods of humans of low socio-economic status. The failure of class mobility or the demographics of prison populations no longer become linked to discriminating and excluding structures in society, but can now be explained by biological difference. Even if many epigeneticists explicitly state that they hope their work will further social and environmental justice aims, the ways in which rodent studies, primate studies and human psychological studies are casually but meaningfully linked might work instead to stabilize and reinforce social inequalities and segregation than to subvert them.

Doting Mothers, Prudish Daughters: Sexual Stereotypes in Environmental Epigenetics

As with any type of politics, class politics are always also gendered. The McGill group has also investigated how maternal licking affects the sexual behavior of female offspring. In Cameron et al. 2008, the authors suggest that maternal licking might affect the expression of an estrogen receptor gene in female pups. They reported that in their experiment the more female rats were licked as pups, the less likely they were to give the back-arching response that is required for successful sexual reproduction (lordosis) when mounted by males. On the level of the individual, they argue that maternal care is a key factor in the "sexual receptivity" of female offspring. On the level of the population, they conclude that this single epigenetic mechanism allows rat populations to shift reproductive strategies to adapt to environmental conditions, giving birth to more offspring in hazardous conditions.

This research on the maternal programming[9] of sexual behavior in female rats was the topic of a 2010 news feature in *Nature* entitled "Neuroscience: In their Nurture." In this article, science writer Lizzie Buchen presents these findings in human terms, making use of familiar gender stereotypes:

> Doting mothers have prudish daughters, whereas the daughters of inattentive rats cavort around like mini Mae Wests. At the heart of these differences lies the sex hormone oestrogen, which drives female sexual behaviour. Champagne says that neglected rats might respond to it more strongly than those raised by attentive mums. (Buchen 2010, 146)

Here, through anthropomorphic analogy, the rat dams and rat pups are figured as "prudish daughters," "doting mothers" and "mini Mae Wests." Despite the fact that humans do not lick their young, nor do they require back arching for sexual reproduction, the terms "sexual receptivity" and "maternal care" flow more easily into the human context. By using cultural short-hands like "mini Mae Wests," Buchen translates the rat findings into human findings, thus naturalizing these narrow roles for women as loving or frigid mothers or promiscuous or prudish sexual prospects (an echo of Harlow's "hot mamas" and "ice cold mothers").

Again, these kinds of translations are not only found in science journalism for public consumption but also in peer-reviewed journal articles. In the original article published in the *Journal of Neuroendocrinology*, Cameron et al. spend most of the article discussing the rat experiments with special reference to rat reproductive behavior and the rat endocrine system, but conclude with this sentence about humans:

> These findings provide a stunning parallel to the human literature (68) in which the onsets of both reproductive function and sexual activity are influenced by parental care in early life. (Cameron et al. 2008, 798)

Importantly, the citation that is used to support the "stunning parallel" between rats and humans (reference number 68) is incorrect. It actually links back to Weaver et al. 2004, "Epigenetic Programming through Maternal Behavior," which does not discuss the onset of puberty in humans.[10] Nevertheless, this citational error passed peer-review, lending scientific authority to common stereotypes about absent mothers and the sexual development of their daughters, which offers another example that illustrates the political and epistemic effects of poor citation practices. Here, simplified and unsupported analogies offered between humans and rats serves to reduce the complexities of parenting and sexuality to one bio-behavioral mechanism, thereby limiting the biological and social agency of both species. Because these findings contribute to moral and biological discourses about parenting and sexuality, it is important to consider what makes these cross-species comparisons appear common sense (to readers and peer reviewers alike) and to think about how we might collectively ruffle the smoothness of these translations.

Disciplinary Translations: Epigenetics and DOHaD

So far we have discussed how preconceived notions of sex, gender, sexuality and motherhood intersect with narratives about race and class in epigenetic research on maternal effects itself. However, the importance of epigenetics for scientific discourse goes beyond the epigenetics lab. Increasingly, epigenetic research also provides a frame of reference for studies in epidemiology and biological anthropology, particularly those in the subfield focused on the developmental origins of health and disease (DOHaD). This research field explores how the socio-economic environments individuals experience during gestation and early life significantly shape their susceptibility to a range of diseases in later life. Researchers in this field increasingly postulate epigenetic mechanisms as plausible causal explanations for their epidemiological and bio-anthropological observations. Often, however, this does not imply that there is currently concrete epigenetic research regarding their specific topics of study. Rather the general "explanatory order" (Richardson 2015, 211) of epigenetic reasoning is invoked. We see the claim, for example, that epigenetic processes constitute a "key mechanism that underpins the developmental origins of chronic noncommunicable disease" (Gluckman et al. 2009, 401) being called upon to support new approaches to disease prevention that target the "prenatal and postnatal periods" (402).

The invocation of epigenetics has become so common that it is included in the mission statement of the field as expressed on the front page of the web presence of the International Society for DOHaD:

> A poor start to life is associated with an increased risk of a number of disorders, especially non-communicable diseases, throughout the lifecourse. […] The DOHaD concept describes how during early life (at conception, and/or during fetal life, infancy and early childhood), the environment induces changes in development that have long term impact on later health and disease risk. Environmental exposures including parental lifestyle and diet, smoking, obesity and exposure to endocrine disruptor chemicals/ toxin, have been shown to modulate disease risk. […] It is thought that some of these developmental alterations come about through changes in the activity of genes through epigenetic processes. […] Timely interventions may reduce such risk in individuals and also limit its transmission to the next generation. (http://www.mrc-leu.soton.ac.uk/dohad/index.asp)[11]

It is crucial to note that to date a majority of researchers in the DOHaD field do not conduct epigenetic research themselves. However, relating their

research to epigenetic studies increasingly serves to lend molecular credibility to their work. Scoping papers (e.g. Loi et al. 2013) propose epigenetics as an important partner discipline for DOHaD researchers, while other studies zoom in on particular phenomena, such as heart disease (Kuzawa and Sweet 2009) or obesity (Wells 2010), and connect them to epigenetic findings in rodents. In this context, the rodent research on maternal effects often serves to illustrate how these processes work *in general*—even if researchers have not identified the specific mechanisms for this *particular* phenomenon. Loi et al., for example, cite the research on the epigenetic effects of maternal care in rats to support the position that "stress responsivity, cognitive ability and response to reward are highly sensible to early-life events, especially maternal care" (145). Although they state that these results "were *so far* obtained [only] in animal models" (145, emphasis ours), they nonetheless argue that they "might open up new policy avenues to tackle disadvantage" (145). While we support the researchers' agenda to address local and global health disparities caused by social injustice and elucidate the role of epigenetics in this context, we are wary of how simplified and decontextualized notions of maternal care and responsibility travel between the experimental settings in epigenetics labs and disciplines with a more immediate impact on public health policy (Pickersgill 2014). We suggest that particularly as epigenetic studies become influential for fields that interact more closely with policy, such as DOHaD, it is imperative to scrutinize the social assumptions that shaped the epigenetic studies in the first place.

Epigenetics is currently not only invoked as an important mechanism for studies of DOHaD by researchers themselves but also by science journalists even when these links have *not* been made by scientists themselves. For example, a recent article in *Nature News* (Reardon 2015) discusses a US-based study that links childhood poverty to decreased brain surface area and reduced cognitive abilities. Among all the possible causes, the article focuses on parental behavior as the most likely cause and renders parental neglect and abuse as the main medium transducing poverty to the brain. Besides the problematic fact that in this narrative being poor becomes almost equivalent to being a bad parent, when discussing the possible mechanisms behind the observed phenomenon, the discussion immediately turns to epigenetics. Jamie Hanson, psychologist at Duke University, is quoted as suggesting that "epigenetics—modifications to DNA caused by environmental factors such as stress—could also be playing an important role, and can be passed down through generations." Epigenetics clearly has found its way into the scientific and popular scientific explanatory landscape of research that focuses on the biological impacts of social position.[12]

In return, epigenetic researchers tend to use studies that engage with questions of DOHaD to lend a human framework to their rodent experiments, as the example below illustrates:

> Environmental adversity, such as poverty, is associated with patterns of parental care that involve abuse, neglect, and harsh, inconsistent discipline (Conger et al. 1994; McLoyd 1990, 1998). Such forms of parenting are, in turn, associated with impaired cognitive development, as well as an increased risk for mood disorders, substance abuse, and posttraumatic stress disorders in the offspring (Ammerman et al. 1986; Brown and Anderson 1993; Feehan et al. 1991; Holmes and Robins 1988; Trickett and McBride-Chang 1995). (Champagne and Meaney 2006, 1227)

Here, these findings, which broadly support the hypothesis that poverty causes poor parenting, which in turn, causes developmental and mental health disorders, are called upon to validate Champagne and Meaney's claim that the rodent experiments are relevant to human health. Importing these claims into an article about the epigenetic effects of maternal care in rats, the original studies are "black-boxed," and any limitations, uncertainty or complexity (e.g. how they account for kind, loving and supportive parents living in poverty) from the original studies are eliminated.

This circular referencing between epigenetics and fields such as DOHaD lends authority to epigenetic explanations across disparate fields, while simultaneously strengthening the social categories and assumptions used in the epigenetic literature. As epigenetics becomes an increasing common mechanism for explaining and exploring correlations between social position and health, research questions and experimental designs that are not readily compatible with this type of molecular reasoning might become harder to pursue, to fund and to argue for (Darling et al. 2016). It therefore becomes important to question how epistemic practices like circular referencing gloss over the complex histories and heterogeneities of different fields and might thereby reinforce overly-simplified understandings of class, race gender, sexuality and motherhood.

Environmental Epigenetics and the Social Sciences: Critique, Conversation and Collaboration

In this article, we aimed to show how epigenetic research on maternal effects brings together questions of gender, class, race and motherhood both in its experimental designs and in the narratives it produces. Although this research

appears to challenge genetic determinism and refigures traditional categories like "nature" and "nurture," other categories remain unquestioned such as the binary, heteronormative and functionalistic notions of sex and gender. We suggest that feminist STS can make distinct analytic contributions to research on maternal effects in epigenetics. In order to begin to develop a shared vocabulary for this potential conversation, we will summarize the main challenges we see with regard to epigenetic research on maternal effects as it stands so far.

First, we see a tendency to translate between experimental results of model organism studies and human health without adequate empirical evidence. These translations are facilitated by vague categories such as "sexual receptivity," "social standing," "maternal care" and "parental rearing style" that are applied to both rats and humans, obfuscating important differences between species (e.g. humans often practice bi-parental care, whereas rats do not). Commonsense stereotypes (e.g. the idea that single mothers raise promiscuous daughters) are often called upon to support these translations and consequentially circulate uncritically in both the peer-reviewed literature and media reports on environmental epigenetics.

Second, we find that these entanglements of human and rodent research give rise to specific figurations of motherhood—the expanding mother and the mediating mother—that focus the responsibility/blame for the health of the offspring on the mother, but paradoxically grant her no personal agency for "breaking the cycle" of environmental deprivation and contributing to positive change.[13] In these stories, the epigenetic profile of children who have experienced poverty and other forms of social and material deprivation are figured as "adaptive," making poor children seem epigenetically conditioned for disadvantaged, stressful living conditions. As poverty has marked their bodies by increasing anxious and aggressive stress responses, they become rendered biologically unfit for a "better life."[14] These narratives emerging in the epigenetic research on maternal effects increasingly interact with epidemiological studies from fields such as DOHaD. Here they serve to lend a general explanatory framework to phenomena for which the precise causal mechanisms are unknown as of yet. As studies in these fields aim to inform public opinion and public policy on the implications of early environments on human health, pointing out sex, gender and class bias in the epigenetic studies seems especially important.

As feminist STS scholars who foreground questions of social and environmental justice in our own work, we are sympathetic to researchers in environmental epigenetics and DOHaD who are interested in understanding "the material consequences of social dynamics and inequality" (Niewöhner 2015, 228) (e.g. McGowan and Szyf 2010; Kuzawa and Sweet 2009; Wells 2010; Loi

et al. 2013). Identifying the somatic effects of poverty, racism, and colonialism across generations is an important task; health inequalities are central to the "increasingly racialized [and] gendered disparities in wealth and the distribution of life chances" (Spade 2011, 57) that feminists and other scholars in the critical humanities and social sciences have sought to address. However, the ways in which the molecularization of the environment in environmental epigenetics privileges narratives and practices of individualization (Darling et al. 2016; Mansfield 2012; Niewöhner 2015, 221) means that the research on maternal effects tends to pathologize women's bodies and figure "bad mothers" as the locus of public health intervention[15] (Richardson et al. 2014; Richardson 2015). Historically, attributing biological difference and deviance to the bodies of marginalized groups has not worked in favor of the groups (e.g. by improving their living conditions) but rather contributed to further marginalization (Rose 2009; Roberts 1997; Meloni 2015). If the new biosocial accounts coming from environmental epigenetics do not take these histories into consideration, they risk working against the social justice goals that motivate the research.

To proactively address these challenges, collaborative work with critical humanities and social science scholars might be both necessary and rewarding. As we stated earlier, there have been many calls for social scientists to "work closely with actual researchers" (Rose 2013, 21), to risk new "experimental entanglements" (Fitzgerald and Callard 2015), or to create hybrid fields like the "biohumanities" (Stotz and Griffiths 2008 in Meloni 2013, 743). However, there have been few practical suggestions about *how* these collaborations could be pursued, organized and institutionally supported in the specific field of environmental epigenetics. As STS scholars have been increasingly integrated in research fields that are understood to have "social implications" (e.g. nanotechnology, synthetic biology, neuroscience), it has proven difficult to create collaborative relationships in research settings that are already structured by specific disciplinary cultures and institutional norms that regulate what counts as valuable work (e.g. in terms of attracting funding and producing high-impact publications). For example, STS scholars have noted that they are often interpreted as "a representative of the public" rather than a scientific colleague who could contribute relevant expertise to the research process itself (Fitzgerald and Callard 2015, 16; Viseu 2015; Balmer et al. 2015). Calls for collaboration are therefore important, but not enough. Rather, creating the kinds of complex biosocial accounts scholars envision will require dedicated institutional spaces and practices that, among other things, upend traditional hierarchies between the natural and social sciences (see e.g. Reardon et al. 2015) and provide the time and resources for a mutual in-depth engagement. This is a particularly challenging task given the current fast-paced and highly competitive working contexts of life scientists and other

researchers, in which swift, high-impact publication is key to achieving institutional recognition (Müller, 2014). For the long winding practices of in-depth collaboration to take hold and prosper, specific structures of incentive and reward need to be established in order not to disadvantage the life science scholars truly interested in collaboration with the social sciences.

Taking into account these institutional and epistemic challenges facing biosocial collaboration in and around environmental epigenetics, we have two concrete suggestions for how to begin to create spaces for such shared conversations. First, we suggest including STS scholars with knowledge of epigenetics as additional peer reviewers for articles that draw on social categories in their experiments and findings. As our analysis shows, STS scholars have the skills to point out both political and epistemological problems with epigenetics articles that were not caught by peer reviewers in the biosciences. Having STS scholars as peer reviewers for similar articles would also increase the likelihood that social scientists will become attractive partners for planning and conducting such research. Secondly, when environmental epigenetics is being taught at universities, social scientists could be invited to the classroom as discussion partners and co-teachers to anchor the idea of possible collaboration early in life science education. As we laid out in the introduction to this article, we believe as STS scholars that it is valuable both to develop and write for classes that train students for interdisciplinary conversation and collaboration. By addressing the relationship between the social and biological from the very beginning, we argue that teaching critical scientific literacy in interdisciplinary classrooms has the potential to lay the groundwork for collaborative research.

Moving beyond existing disciplinary frameworks is a challenging endeavor. However, environmental epigenetics is just one field that shows how research questions are already butting up against the limited confines of the disciplines whether we are ready or not. We hope that environmental epigenetics can become a successful case for mutual efforts to catch up with the liveliness of our questions and develop interdisciplinary research practices that can begin to modestly address the complex biosocial relations that affect human health and well-being.

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Notes

1. Ludwik Fleck, a Polish molecular biologist and early theorist of science, developed the notion of "pre-ideas" (1979/1935) to account for how scientific processes often depart from pre-set beliefs about the matter they investigate. These include taken for granted assumptions that build on folk knowledge, findings in other scientific fields and earlier, possibly outdated work in one's own field. In his own work, Fleck uses the example of syphilis to show how the pre-modern folk idea of bad blood crucially shaped how syphilis was investigated. This idea of "bad blood" was both inaccurate and generative of important research. Fleck understands pre-ideas as inherently necessary for knowledge production as they allow for hypotheses to emerge, yet he also points out that they are prone to reflect societal bias and often are hard to overcome, even if empirical evidence to the contrary accumulates. He argues that a sustained reflection of pre-ideas and their historical and social situatedness is hence crucial. He writes: "Whether we like it or not, we can never sever our links with the past, complete with all its errors. It survives in accepted concepts, in the presentation of problems, in the syllabus of formal education, in everyday life, as well as in language and institutions. Concepts are not spontaneously created but are determined by their "ancestors." That which has occurred in the past is a greater cause of insecurity—rather, it only becomes a cause of insecurity—when our ties with it remain unconscious and unknown" (1979/1935, 20).
2. As many other STS scholars, both authors regularly teach classes for students in natural sciences, social sciences and humanities that explores the social, ethical and political aspects of life science research. For example, Kenney teaches a class called "Genetics, Biotechnology, and the Politics of Difference" that regularly enrolls undergraduate and graduate students in Women and Gender Studies, Biology, and American Indian Studies. Such classes require introductory texts that teach "critical scientific literacy" to students with a wide range of backgrounds and expertise.
3. Despite the changing politics of gender in the twentieth century, Neo-Darwinian accounts of animal behaviors and morphology still rely on these Darwinian gender roles, especially in the sexual selection literature (see Roughgarden 2004, 2009). For example, evolutionary biologists hypothesize that large human brains are "male ornaments" (like the peacock's feathers) used to compete with other men to attract mates, whereas women developed large brains so that they could appreciate the large brains of men and choose the best specimen (e.g. Rice 2007).
4. Feminist science studies scholars with graduate degrees in the natural sciences include: Karen Barad (physics), Anne Fausto-Sterling (developmental genetics), Donna Haraway (biology), Ruth Hubbard (biology), Evelyn Fox Keller

(physics), Lilly Irani (computer science), Natasha Myers (environmental science), Deboleena Roy (neuroscience), Astrid Schrader (physics), Banu Subramaniam (zoology genetics) and Ruth Müller (molecular biology).

5. http://learn.genetics.utah.edu/content/epigenetics/rats/
6. Beyond the question of whether *love* is quantifiable is the basic question of whether the human category "motherly love" can apply to the licking and grooming behavior of rats, since human mothers tend not to lick their young. Although it's easy to imagine human analogues (cuddling, breastfeeding, etc.), we argue that these parallels should be investigated rather than assumed.
7. Narratives about pathological motherhood are not just about class but are racialized as well. In the US context, the Moynihan Report (1965) declared that unwed black mothers damaged their families by "demoraliz[ing] black men" and "transmitt[ing] a pathological lifestyle to their children, perpetuating poverty and anti-social behavior from one generation to the next" (Roberts 1997, 16; see also Collins 1999, 69–96). Although the epigenetics literature refigures this transmission as biosocial rather than just social, it requires special care to avoid these racialize archetypes in telling scientific stories about pathological inheritance.
8. LG-ABN = Licking, Grooming, Arch-Back-Nursing.
9. The relationship between the environment and the genome is often expressed using the metaphor of "epigenetic programming" (Landecker 2011), which, like many dominant biological discourses of the twentieth century, renders life as information (Fox Keller 1995). As in the genomic era, these metaphors can lead to modes of deterministic thinking. Rather than genetic determinism, the metaphor of "epigenetic programming" leads to a new form of "environmental determinism" (Müller, forthcoming) where the psychological and physiological make-up of an organism is defined by its environment.
10. Our best guess is that they had intended to cite Belsky et al. 2007 "Family rearing antecedents to pubertal timing," although this does not appear anywhere in their works cited. However, without the correct reference it is difficult to assess the epistemological strength of the parallels between the human study and the rat experiments.
11. During final revisions for this article, the DOHaD Society homepage has undergone a relaunch. The archived link can be found here: http://web.archive.org/web/20150802235357/; http://www.mrc-leu.soton.ac.uk/dohad/index.asp.
12. In the *Nature News* article (Reardon 2015), the study about poverty and brain size that appears in *Nature Neuroscience* is joined by "unpublished research" from another lab in order to strengthen the explanatory claims of the researchers. Thus, a peer-reviewed study, unpublished data and a speculation about epigenetic causes are all called upon to support a specific biosocial explanation. This kind of reporting contributes to cycles of hype where poorly substantiated claims are repeated without critical scientific or social scientific analysis.

13. This happens at a historical moment in which actual class mobility is found to be on the decline in many Western countries for the first time in generations (Altzinger et al. 2013, Bukodi et al. 2015). This phenomenon is particularly pronounced for people of color, ethnic minorities and second- and third-generation immigrant groups.
14. This figuration is familiar from such "fish out of water" narratives as Mark Twain's *The Prince and the Pauper* (1881) or Will Smith's *The Fresh Prince of Bel Air* (1990–1996). These epigenetic narratives take what is usually coded as cultural difference, and render it as biological.
15. Women of color feminists have been especially important critics of public health interventions that focus on mothers and "pathological" family formations. Angela Davis, for example, writes: "While the difficulties besetting the family should by no means be dismissed, any strategies intended to alleviate the prevailing problems among poor Black people that methodologically target the family for change and leave the socioeconomic conditions perpetuating Black unemployment and poverty intact are doomed to failure at the outset" (1990, 81).

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35

Ancestors and Identities: DNA, Genealogy, and Stories

Jessica Bardill

The concept of "Native American DNA" is in and of itself constructed through pursuits including the genetic ancestry testing, or mapping of genomic patterns, of individuals and of ancestral remains, reciprocally contributing to the identities of those tested (TallBear 2013; Bardill 2014). However, literary imagination and indigenous sciences offer alternative ways to construct identities, specifically how a given people define themselves. These alternatives often reframe the genetic and genomic knowledge—questioning even if that data does constitute knowledge—and center attention to relationships as opposed to individual inquiry. While our ancestors influence our present and potential identities, their classifications based on genetic testing do not have to limit the possibilities of being, belonging, and becoming for us both as individuals and communities. This chapter will explore both actual genetic testing and handling of remains as well as creative work that has been generated out of this nexus of genetics, identity, and society. The specific examples include: the rhetoric around the Genographic Project in its interactions with the Uros and their claims to indigeneity, including the legibility of those claims with and without genetic testing to the nation-state; the discovery and subsequent disputes about the disposition of the ancient individual known as Kennewick Man, as well as the testing and then re-interment of the ancient individual called Anzick-1 or the Anzick child; and finally the capacity building for both scientific inquiry and for tribal nations of training indigenous scientists.

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Genetics and Identity

Genetic ancestry testing involves a variety of inferences usually between present-day populations standing in for ancestral populations (Royal et al. 2010). Those present-day populations are also constructed by genetic scientists with a specific and limited definition of indigenous or as narrowly representative of a nation or "genetically similar population" instead of as a people (TallBear 2013; Barker 2002). In those constructions, in relation to American Indian peoples, Kim TallBear brings us to the important point that tribes are sociopolitical groupings (TallBear 2003), and therefore even if someone has demonstrable American Indian heritage (through genealogy or genetic testing), that information does not mean that someone necessarily belongs to the sociopolitical group. As socially determined, belonging requires more than personal assertion but also communal assertion and acceptance of particular identities (Andersen 2014).

The definition of indigenous peoples from genetic scientists as "Such groups that have children only among themselves and retain a unique culture are termed 'indigenous'" (Lewis 2010, 4) does not conform to the many ways that indigenous peoples choose to define themselves. However, it should also be made clear that one cannot use genetic ancestry testing to successfully seek tribal enrollment or belonging, to claim being and belonging. Even as many tribes have opened up the determination of parentage to genetic tests and affidavits of declared parentage, that acceptance of information generated by a scientific test sacrifices knowledge already had within the community about notions of family and parentage (Bardill 2010; TallBear 2013). Information here requires inference and interpretation whereas knowledge refers to the resultant culturally situated and interpreted information. If the community replaces communally held knowledge with a method of information derivation, including genetic testing, and then conflates that information with knowledge, the community relinquishes its sovereignty over data and the stories generated therefrom.

"Story" is consciously extended here to describe particular products of genetic and genomic sciences, including socially constructed ideas of relation. While not the only way to characterize the relationship between scientific data and social affiliation, and not meant to put the two in opposition, bringing attention to the formation of narratives by scientists allows for attention to the cultural work done by those narratives alongside literary production. From biospecimens, DNA is extracted, amplified, and sequenced, and that sequence information is then compared against large databases of other

sequences to determine similarity and distinction, which scientists then package into articles or other presentations—their stories—about genetics. Information can be packaged in many ways to convey data about relationships, and attention to the rhetoric of those packages and the assumptions of the method of packaging allows us to see a range of stories emerging in the genomic era. While humanity has constructed figurative understandings of relation and inheritance about blood and what would become known as genes for centuries, the advances in genetic and genomic science in the last two generations have led to a variety of stories, some of which parallel their predecessors in blood rhetoric or racial logics, and some of which emerge as truly novel (Dennison 2012). For example, if a genetic test infers an extremely close relationship between two individuals, such as parentage, when the socially constructed family relationship had existed between them for years, that test and its related story do not add much to the information already had only serving to reinforce that existing relationship. At a larger level of the community, genetic testing has been used to show relation between ancient individuals and living individuals (Rasmussen et al. 2015) even though the community had declared and argued for their relation for over a decade. In this case, and in others explored in this chapter, community knowledge of self and surroundings may be ignored in favor of published data, thereby both artificially creating (through ignoring other claims and evidence) and limiting space (through lack of consultation and collaboration) for the scientific hypothesis to exist. By failing to take into account indigenous knowledge, for example, genetic science can appear to have discovered something already known by indigenous people, when collaboration could have produced a greater insight for all beneficiaries.

Importantly, the above definition from genetic scientists of "indigenous" comes forth from larger systemic projects of nation building as well as power differentiation, for example, in the settler colonial nations of Japan and the US during the formation of their branches of anthropology. Ann-Elise Lewallen explains, "In Japan, the colonizing moment coincided with the introduction of modernist discourse, and anthropology in Japan originated from a search for ancient Japanese origins through Ainu and prehistoric ancestors' bodies" (2013). While the foundation of American anthropology with Franz Boas is rooted in the idea of culture, these attempts to use present-day indigenous individuals and their ancient relatives occurred and still occur today, often to diminish the existence of settler colonialism and to draw connections between an ancestral population and the whole of the national, or provincial (Leroux 2015), population. These examples demonstrate that the construction of various anthropological projects reflects efforts to generate

evidence of the settler colonial population as emerging from the indigenous even as they are no longer the same people groups, an erasure and replacement seen often in efforts of "playing Indian" (Deloria 1994). This power to define indigeneity permeates across a range of projects and places.

Uros—Indigeneity and Resource

In exploring how "being indigenous" has come into conversation and tension with genetic testing, we first turn to the example of the Uros people of Bolivia. The Uros live on floating islands they build of reeds on Lake Titicaca and they have consistently maintained their differentiation as a people genealogically from the Quechua and Aymara of the lands around the lake, an assertion to which the other indigenous peoples agree. However, before 2008, state actors were unwilling to accept their sovereignty and with it the rights to the reed beds, which they had been using to build their polity. At least they were unwilling to accept this truth until the National Genographic Project, then led by Spencer Wells, performed genetic testing on all three of these peoples (Kent 2013).

The full quality of the "informed" nature of "consenting" these group members to testing is not known, as the Genographic Project has previously provided only cursory information before sampling individuals, and this lack emerges as a concern generally with how researchers account for translation, local social practices, and community consultation around consent (Wald 2006; De Vries et al. 2011). Further, the results of this research have then been used to distinguish political groupings, on the basis of their genetic distinction from one another. Based on the results, the Uros were able to successfully petition for proper recognition of their indigeneity in Bolivia, including access to the reed beds they had historically used. This political recognition based on genetic testing, while fairly rare, remains questionable in regards to the power thus given over to scientific processes as opposed to community histories and statements (Benjamin 2009; Palsson and Rabinow 1999). Particularly for indigenous peoples, oral histories and storytelling provide valuable and valid evidence of being and belonging to a place (TallBear 2003). This battle over resources and home space is not just a disagreement, but a demonstration of how identity politics are tied in to economics and systemic management of peoples. After this recognition, Bolivia capitalized on the unique (genetic) identity of the Uros, incorporating them strongly into tourism advertising; however, given that tourism to the reed islands is a chief economic enterprise of the Uros, this national promotion (as opposed to denial of identity and access rights) also supports the people (Kent 2013).

This rare case demonstrates the power of the scientific story—the power to gain recognition where it was otherwise denied, a denial not because of lack of truth but for lack of evidence legible to the state in the story told by the communities in and around the lake, who also lack power within the national society. This scientific story is based on access to and interpretation of biospecimens from indigenous peoples, which genetic scientists argue are scarce (Chatters 2001; Kelly 2008), particularly in the USA where tribal nations have by and large refused participation in research that constructs their bodies as resource, or continues older narratives of "vanishing Native Americans", and this refusal from the communities and individuals reflects tribal sovereignty. The demand and desire for indigenous samples was so great, and the scientific practices of engaging with community so limited in the 1990s and 2000s, that many tribes in the USA and Canada particularly opted against giving any kind of sample to research scientists for genetic testing, or other research (Reardon 2004). The Navajo Nation, the largest of the 567 federally recognized tribal nations in the USA, issued a moratorium in 2002 that was to have lasted for 5 years, allowing time for review of the issue and capacity building about genetic science within the nation; however, the moratorium still stands today (NNC 2002). This moratorium cannot be separated from research abuses including those seen in the *Havasupai Tribe vs. Arizona Board of Regents* (2009), when a researcher from ASU used samples given by the Havasupai in secondary studies of schizophrenia and human migration, to which the tribal members did not consent and which went against their own beliefs and produced further stigma toward the community (Mello and Wolf 2010; Garrison 2012). This refusal further limits representation of indigenous peoples from the USA in genetic ancestry testing databases, which are then supplemented with indigenous samples from throughout the Americas, as well as publicly available data on ancient individuals.

Karitiana and Poetry

Poetry, like many forms of literature, gives voice to both emotions and identities often rendered invisible through scientific narratives that seek an unbiased presentation; however, both stories from science and poetry from indigenous authors demonstrate our investment in defining our identities, with a wide set of evidence from DNA bases to cultural figures. Heid Erdrich (Turtle Mountain Chippewa) has meditated and expounded upon these particular issues of being, belonging, and becoming as a poet, exploring both scientific and lay understandings of identity, relationship, ownership, and sovereignty.

In her poem, "Vial", she reacts to the situation reported in the *New York Times* of the Karitiana people of Brazil (Rohter 2007), specifically their experiences with exploitation and power relationships between researchers and participants as well as between medical doctors and patients. The poem's epigraph references this 2007 article, including how the people found that their "blood and DNA samples are being sold online" (Rohter 2007). Erdrich then explores this event in six stanzas.

The first two stanzas emphasize that the samples were "stolen", an interaction reported in the *New York Times* article wherein two encounters were had between the Karitiana and outsiders seeking blood: first, a set of researchers in the 1970s came to take blood samples and more recently, in 1996, a second group of researchers came and made promises of medicine that were never fulfilled. The characterization of stolen then refers to the broken contract and lack of benefit, as well as the undisclosed uses of their blood—the internet sales. Erdrich places into sharp relief that the samples have been commoditized not by the people themselves, but by another actor, likely the researchers who obtained those samples. The third stanza hones in on how the people, "offered / blood and got / nothing", echoing the title of the *NYT* piece and furthering Erdrich's use of negating language in the poem, visible earlier in phrases like "non-bought / non-paid for" but here used to put forward the unmodified absence, the failure to compensate, to engage in reciprocal exchange, including "promised medicine" (Erdrich 2012, 134). The fourth and fifth stanzas return to the imagery of the first stanza, the blood in the vial, but turn on the costs to the Karitiana who believe the body has to travel whole into the next life, "each drop accounted for…" (Erdrich 2012, 134), and this final ellipsis leaves open the tensions of needing medicine and needing to be whole, along with the costs to try to get the former without fully sacrificing the latter. The final stanza of "Vial" takes up the issue of greed, that has been seen for centuries in extracting from and appropriating indigenous bodies, but leaves open how the Karitiana or any indigenous peoples should or could react in the face of these extractive technologies.

This poem demonstrates injustice experienced by the Karitiana not only as symptom of capitalism but also in relation to a specific Anishinaabe cultural figure, relevant to Erdrich's identity and culture. Jessica Kolopenuk identifies the wiindigo that emerges in these actual and representational genetic projects, where the desire for indigenous bodies and blood, the literal and figurative consumptions of the body—come from actors that are no longer quite human—but instead driven mad with their desire and insatiability (Kolopenuk 2014). This consumption conditions the possibilities of being and belonging, through the invocation of a power differential in the interactions. Further this

consumption constructs its desire for indigenous body as resource through asserting an outside definition of identity, where the wiindigo desire, instead of reason, drives the collection practices.

The Uros and Karitiana examples allow us to consider how genetic research projects in South America have taken indigenous knowledge and bodily resource. Further their bodily integrity may have also been disrupted, a concept that Puneet Sahota explored in a study with a USA tribal community regarding genetic research (Sahota 2014), but that has potential for understanding the disintegration that occurs particularly in the place of the Karitiana and which may also be a compromise made by the Uros for their recognition. However, these manipulative practices occurred both throughout the Americas and globally, contributing to the objectification and dehumanization of indigenous groups. For example, Ann-Elise Lewallen discusses how these practices impacted the Ainu, who do not have the benefit of wide-scale media coverage like the *New York Times*. In a ruse the Karitiana would find familiar, Lewallen reveals that "To obtain blood samples from Ainu persons, researchers masqueraded as medical professionals or deceived Ainu into cooperating with their research by suggesting that these tissue samples would be used to solve epidemics of smallpox that had decimated the Ainu population" (Lewallen 2013). Again, we see that "promised medicine" leads to the fragmentation of the body and its integrity for another indigenous people (Sahota 2014). Further, Lewallen quotes Ainu activists who articulate their view of the situation, for example, Cikap Mieko, who "want[s] scholars to stop labelling Ainu as "valuable data" and treating us as "research materials", because we are a living people" (Lewallen 2013). This powerful resistance to objectification and the gaze of the researcher shows another of the possibilities of the partnerships that can emerge and strengthen communities and science when research relationships begin with respect.

Both the example from "Vial" and this Ainu example demonstrate how power is commodified by those in the dominant position (researcher, physician), instead of being negotiated as an ongoing interaction; however, both examples show how the community and the researchers could partner to journey toward health instead of having promises made and broken. The Uros example clearly shows the interrelation between the people and the reed beds, a part of their environment that they rely on to construct their homeland on the lake. Given their need for the reeds, the Uros also care for and tend to the reed beds, in a reciprocal relationship. However, this reciprocity in relationships has not been seen often regarding the claiming of ancestral remains by scientists over the centuries, particularly given how they are highly valued, as well as how they are used by researchers.

In genetic ancestry testing, one aspect as to why modern human populations are used to stand in for ancestral populations has to do with a purported "scarcity" of ancestral remains; however, that notion obscures the fact that for ancient individuals in the Americas there have been between 1200 and 1500 ancestral remains found (Bolnick 2015). However, that figure of 1500 refers to remains that are hundreds and thousands of years old, while there are literally thousands if not hundreds of thousands of AmerIndian human remains housed in museums and collections throughout the world, more still when we count in Indigenous peoples not of the Americas. These remains, like the blood and knowledge above, for many peoples should be returned home to allow for rest of the individual. For example, in 10 years, starting 15 years after the passage of the Native American Graves Protection and Repatriation Act (NAGPRA 1990), one small collective of five tribes has had over 12,000 ancestors returned. That collective and their claim of belonging for an ancient individual stand at the center of our next case.

Belonging and Ancient Remains

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In 1996, while attempting to sneak into a hydroplane race on the Columbia River (and drinking Budweiser), two teenagers noticed a skull peaking out of the river bank and called in a local anthropologist. Once aware, at least five local tribal nations (Confederated Tribes of the Colville, Confederated Tribes of the Umatilla Reservation, Yakama Nation, Wanapum Band of Priest Rapids, and Nez Perce) in Washington State made claim to the remains under the 1990 legislation known as NAGPRA. It is important context that the finding, claiming, valuing, testing, and destroying of Native American remains was quite common during the rise of racial science including phrenology (or the measuring of skulls), building on the taking and destruction of Native grave sites as those lands were claimed as property over the course of Manifest Destiny. After decades of disputes, NAGPRA emerged to remedy past practices and the circulation of remains through labs and museums without community consultation—however, through biospecimen collection practices the legacy of this scientific work continues, including samples from remains that may one day be repatriated (Trope and Echo-Hawk 1992). James Chatters, an anthropologist widely quoted in news outlets about the discovery of these particular ancestral remains, known popularly as Kennewick Man, provided

rich rhetoric that Heid Erdrich would later use in her poetry. Turning now to excerpts of his words and her own, we can view this ongoing tension between scientists and tribal communities.

In the first of her Kennewick Man poems, "Kennewick Man Tells All", Erdrich begins with an epigraph quoting Chatters from the *New Yorker* in 1997—"We didn't go digging for this man. He fell out—he was actually a volunteer. It would be wrong to stick him back in the ground without waiting to hear the story he has to tell" (Preston 1997, 73). For Chatters, that story would be told through genetic and other molecular testing that requires the destruction of the remains, but for the claimant tribes, geography told the story. For Erdrich, she takes Chatters' poetic license and runs with it, demonstrating how little of a story his methods can reveal through a short public address and going beyond the first poem to animate a more lively Man, who engages with the more than scientific testing in the present-day world. The language of "volunteering" and inferred consent should also be noted here as in using these particular word choices, Chatters invokes ethical research standards and their authority, even as he fails to engage in community consultation for consent from the living or to consider the inapplicability of the current ethical framework to the deceased. He then claims to speak for the remains from a position that he has rhetorically constructed as ethically justified, despite evidence to the contrary, including community objections (Kaestle and Horsburgh 2002). More precisely, Chatters attempts to be the scientific oracle through which Kennewick Man speaks by interpreting his genetics and potential biological relations (TallBear 2015). Erdrich also takes up that possibility of speaking for the Ancient One, but to a somewhat humorous effect.

In "Kennewick Man Tells All", Erdrich prefaces Kennewick Man's statement with a press conference style introduction, calling the audience to attention and order, and including an invocation that "he will answer questions as time permits" (Erdrich 2012, 128). If the remains can volunteer and tell a story, she contends it will be with words and moving about in our current world, even as she calls attention to the idea of time and permission, invoking how Chatters and other scientists do not have permission, as well as their concern about the need to find information quickly even though the Ancient One has been there for millennia. Kennewick Man's brief statement follows and reflects all that the initial analysis, which has recently been elaborated in a nearly 700-page text (Owsley and Jantz 2014), showed about the remains: his age, what remains of him, and his loneliness (Erdrich 2012, 128). These two lines of the poem leave much to be desired of Kennewick Man's story, so Erdrich continues to narrate the story of this man in additional poems, includ-

ing having him swimming and cyberdating. Karen Poremski argues that Erdrich's Kennwick Man poems "do the work—unfinished by [NAGPRA]—of turning objects into beings; they thereby reverse the work of colonialist rhetoric in science and academia that has turned humans—and, specifically, Native people—into objects to be collected, sold, studied, and narrated" (Poremski 2015, 2). Like the Ainu activist's call for her and her people to be considered more than "research materials", Erdrich's poetry makes room for the humanity of the ancient and living individuals instead of their continued objectification. This relationship differs from one of objectification, consumption, or observation and requires an assessment of responsibilities instead of an assertion of "rights" to scientific study.

In "Kennewick Man Attempts Cyber-date", Erdrich explores the modern way of forging relationships through online dating. The poem centers attention on Kennewick Man's desire for identity and social interactions; yet the poem also leads the reader to question how particular identifiers (including age and race) are informed by technology and society (Erdrich 2012, 129). To begin, she returns to Chatters and his story about Kennewick Man and the modern man whom he may resemble: "And then one evening I turned on the TV, and there he was, Patrick Stewart—Captain Picard of *Star Trek*, and I said, 'My God, there he is! Kennewick Man!'" (Preston 1997, 77). This epigraph pulls our attention to the effort to visualize Kennewick Man's features, and how that vision has taken on markers normalized in America of a middle-aged European (or white) male leader, associated with the future even as he is tied to the past. Erdrich's further examination brings that scientific imagination into question through answers he cannot give to the dating survey.

This connection by Chatters between an ancient individual and a European is not without its precedents, as the Solutrean hypothesis imagines that the Clovis people were descended from the Solutrean people of France, settling the Americas perhaps before American Indians; however, this hypothesis has gained little evidentiary support making it appear more fantastical than factual. While this hypothesis fails in the face of oral traditions, scientific investigations, and other facts, it persists. I argue it does so because to prove it correct, to show Kennewick Man as European, as Patrick Stewart, would allow settler peoples and their descendants to make claims to indigeneity in America. They would no longer be historical invaders but instead making a homecoming, though a violent one at that. In this interpretation of historical events and bloodlines, EuroAmericans would have original claim to these indigenous lands, through a biologically evidenced manner of "playing Indian", a familiar narrative being propagated within the supposedly objective sciences (TallBear 2013; Whitt 1999; Haraway 1991).

Oyt.pa.ma.na.tit.tite—or the Ancient One, how the Five Claimant Tribes refer to Kennewick Man—was only recently, nearly 20 years after his unearthing and subsequent exhumation, shown to be "Native American" in Rasmussen et al.'s (2015) article, "Ancestry and affiliations of Kennewick Man". The fight over these remains centered around the different stories and studies distinct groups wanted to tell or carry out, as well as the questions they felt should be asked at the time: the scientific community claimed the remains were a boon for knowledge about human history, going on to claim they might be European or Ainu (but not Native American), and as a result they wanted to keep the remains for testing (to extract information or data that is often conflated with knowledge), whereas the Claimant tribes want to rebury their ancestor, with the only question emerging being to wonder why Oyt.pa.ma.na.tit.tite chose to show himself at that particular time, in that way (Minthorn 2015). This question from Minthorn takes into account much larger structural and interactive ideas of time, place, circumstance, and relation. These aspects are important to keep in mind as we consider the battle of entitlement over the remains, the ethical disputes and moral stands taken on multiple sides, and the spinning of a story that allows scientists to be "right" in their construction of the relationship of the remains to both ancestral and present-day communities, instead of seeking multiple truths about the relations of the remains (TallBear 2015).

The Anzick Child(ren)

The next story and ancient individual emerged in Montana in 1968, though this individual was not fully studied by scientists, nor initially known about by the local tribes, until 2000, with further consultation occurring in the 2010s. In 1968, a contractor working on the Anzick family farm encountered the remains of an approximately 2-year-old child, covered in red ocher, as well as many other ancient artifacts. The family who owned this private property invited an anthropologist to come in and look at the remains; he dated the tools found at the site to the Clovis period and then took most of the site contents after excavation into his laboratory. In 1999, well after NAGPRA went into effect, and after Sarah Anzick, who had been about 2 years old when the remains were discovered, had trained in genetic and genomic analyses, the remains and other objects found on the Anzick farm were returned to the Anzicks. During a 2014 symposium at the National Museum of the American Indian sponsored by the National Congress of American Indians, Sarah Anzick notes that the family reached out to local tribes in 2000 about

the disposition and potential study of the remains. Anzick claimed in her presentation that these remains "were a gift", ostensibly to science, and "had a story to tell" (Doyle and Anzick 2014)—remarkably similar language to that used by Chatters around consent and the idea of "gifting" or donating a body to science. By referring to the remains as "a gift", Anzick demonstrates that given the remains had a clear value to scientific study of human ancestry and were unearthed on her family's land, she did not find that consent needed to be sought, particularly because their uncovering was good fortune not an ethical dilemma. This invocation also serves to negate her responsibility for consultation with or compensation to the tribes.

Subsequently, on her "personal time and independent of [her] position at NIH (National Institutes of Health)", Anzick traveled to Eske Willerslev's lab in Denmark to extract and sequence the nuclear genome of the remains (Doyle and Anzick 2014). While this statement likely indicates that the ancient individual was disambiguated into samples and then taken across international lines, this explanation permits Anzick to deny culpability to any invocation of NAGPRA were the remains to enter a federal lab at the NIH, or to have been handled by her during her time as a federal employee. Instead, through this method she was able to fulfill what she "felt [was] an obligation to humanity" to provide a better understanding of an exceptional site, given that it is the only one like it currently known (Doyle and Anzick 2014).

Once the lab sequenced the genome, they found that the ancient individual was a male child, and when placed in the context of modern populations—about which, again, quite limited data and samples exist particularly for North America—they showed the highest relatedness to American populations from North and South of the USA, as there was no DNA available for US comparison due to historical disrespect and subsequent sovereign refusal (Reardon 2004; Rasmussen et al. 2014). Interpretations based on the genetic analyses of the remains place the young boy as ancestrally genetically related to all American Indians and position his remains as the oldest set found to date in the Americas, with an age of 12,600 years old (Rasmussen et al. 2014). After their analysis was complete, Anzick and Willerslev contacted the Montana tribal nations with the information. Anzick noted that none of the tribal representatives objected to the information presented about the analysis, despite an earlier lack of consensus about how the remains could or should be studied, though they did clearly request the child to be laid to rest.

Shane Doyle, a member of the Crow tribe of Montana, presenting at the symposium with Sarah Anzick, noted that he and others were intrigued by the scientists' findings but that the boy deserved reburial—specifically, he responded to the results with "well that's nice, but I think we should put him

back now" (Doyle and Anzick 2014). In 2014, they did just that, with a joint ceremony of tribes and researchers—46 years after he had been exhumed from the spot where his family had laid him to rest. Anzick and her family agreed that it was their "moral obligation" to return him, particularly because "he has given us a generous gift" (Doyle and Anzick 2014). We are left to consider whether, when asked after the analysis had been performed, did the tribal nations and their representatives truly have room to object or did their silence represent a kind of assent to what had happened and could not be undone, so that they could then move forward with the reburial. The *New Scientist* also quotes Doyle regarding his acceptance of genetic testing personally and for tribal peoples: "I don't believe there is anything wrong with genetic testing and I don't think most Native Americans would either", and further Doyle contends "What all of us agree on is that you should have respect for people who have been put in the ground. Eske's message is that this work is not intended to be disrespectful" (Brahic 2014). However, intent does not correlate to impact, and the lack of awareness around the consequences one's actions may actually have as opposed to those you intend them to have invokes a self-blindness that maintains the false promise of scientific objectivity (TallBear 2013; Haraway 1991). This case shares many similarities with the case of Oyt.pa.ma.na.tit.tite, and also demonstrates how scientists close to a case can ignore the need for consultation with tribal communities until they obtain what they came for, at which point there is no going back.

Similar pressure for the community to become involved or the research would happen without them is also seen in the earlier case. The conclusion that Oyt.pa.ma.na.tit.tite is related most closely to the Colville in Rasmussen et al.'s (2015) paper was only made possible because current members of that tribal nation were willing to provide samples for the study; however, the Colville's decision to participate forced the other four claimant tribes to have to decide about participation. Subsequently, instead of abstention, all four of those other tribes decided against participation in the genetic testing. Important to note as well is the fact that both genetic analyses occurred in Willerslev's lab in Denmark, and other projects there include sequencing the genome a lock of Australian Aboriginal hair from a museum (Rasmussen et al. 2011; Callaway 2011), and sequencing the genome of Sitting Bull, which collectively demonstrate both the lab's keen interest in the indigenous and the potential to continue to pressure indigenous peoples into cooperation instead of full and proper consultation, particularly as they and their collaborators lead the vanguard of population genetics.

Genetic testing and population genetics assert the ability to provide definitive evidence for claims of racial, personal, and group identity formations over

time, whereas many anthropologists and other communities draw attention to the fact that each and every one of these identities is constructed, shifting/fluid, and dependent on context. Yet the evidence from genetic testing has become sacralized (Schwartz-Marín and Restrepo 2013), perceived as the best and most unassailable truth, which trumps family history, traditions, and stories. This view of the truth offered by genetic testing is reflected in a recent Ancestry.com commercial in which a man knew his family to be German all his life, he loved sauerkraut and other German cultural and culinary traditions, but when his test came back and he found out he was Scottish, he "traded in his lederhosen for a kilt" (Merker 2015). When identity is so easily influenced by genetic inference, trading one story for another becomes normalized for a set of peoples, creating a slippage of ethnicity.

Though this chapter has highlighted tense relationships between indigenous peoples and genetic researchers and genetic data, it is important to remember that by and large indigenous peoples are not anti-science, and so many actually want the promised benefits of genetic testing (e.g. addressing health disparities, establishing an additional line of evidence to support indigenous knowledge, and helping learn from the past and each other), but on shared terms. Our next example allows us to explore how those terms are being renegotiated among indigenous peoples becoming scientists throughout the world.

Becoming Indigenous Scientists

Contrary to the notion that story and science stand in opposition to one another, they are mutually constitutive and require our awareness. A story worth amplifying is that of indigenous scientists, who are bringing together a range of worldviews with scientific inquiry. One such leader of indigenous led genetic research, Hopi geneticist Frank Dukepoo asserted that DNA "is part of the essence of a person" and Native community members, and communities, have protected that essence against scientific abuses (Dukepoo 1998). These and other cultural concerns, as well as problematic historical relationships, emphasize that scientists and community members can gain from training together and contribute in new and necessary ways to biomedical studies.

Instead of ethics violations as seen in cases of the Karitiana and the Havasupai samples being used for non-consented studies, or even with the ancestral remains of Kennewick Man and Anzick-1, another kind of partnership is possible. By designing research studies with the community from the

beginning via consultation and community-based participatory research (CBPR), tangible benefits can be created for scientists and the community members in the forms of access, training, and in understanding the results of data analysis from the research. Overall the science can be both accurate and culturally informed. Reciprocally, communities can ask for research and create projects themselves, with the most beneficial relationships creating researchers within the communities who can carry out or collaborate on the work.

In the summer of 2011, the Summer Internship for Native Americans in Genomics (SING) workshop was developed at the Institute for Genomic Biology at University of Illinois, Urbana-Champaign. Now known as the Summer Internship for INdigenous peoples in Genomics, this workshop allows students to learn more about DNA extraction and amplification, the analysis of DNA data with bioinformatics, and the ethical, legal, and social implications of genomics research, particularly with indigenous people. It has the additional benefits of creating networks of young indigenous scientists, and training all involved about the diversity of indigenous cultures and approaches to genomic science. This workshop in turn has started to "bridge the gap" of the divide between non-indigenous scientists and indigenous communities, providing immediate benefits to both groups. It has also resulted in researchers better equipped to deal with cultural practices and concerns in the laboratory, including when those practices and cultures are their own, such as how to respect samples and avoid destruction of them. There have been three additional SING workshops in the USA, training 50 indigenous peoples from 44 different nations (SING 2011), and a program has started Aotearoa, led by a group of Maori scientists and social scientists. This workshop continues to grow and expand, responding to both desire and need, as well as self-determination from the communities about who they want to become. Overall, the training of non-indigenous scientists alongside indigenous scientists, at all levels, enables better science through deeper cultural knowledge, that in turn informs better research questions, methodology, interpretation, and analysis.

In order to extend benefits of genomics to indigenous communities, indigenous peoples cannot only be considered the subjects of research or as biological resources, but scientists should work to collaborate with indigenous peoples, gaining from the insights of other worldviews and knowledges. Indigenous people's roles in science must be expanded to being researchers, investors in research, and as comprising indigenous nations with economies that—like cities, states, and nations—can benefit from the research enterprise and the technologies and information that result from research work.

Indigenous sovereignty enables benefit and cost sharing, but also helps balance the relations between researchers and tribes. In order to begin this balance, scientists can focus on creating immediate benefits (e.g. jobs and training on projects for community members, healthcare, compensation for participation, and retained ownership over their samples) rather than distant potential benefits (e.g. better health for all and advancement of scientific understanding). These beneficial relationships and a changed story—the active storytelling of a narrative alternative to those that pit science and indigenous peoples against one another—enable preservations and evolutions of identities, continuations of sovereignty, and enriched scientific practices.

Identities are complex and multilayered, shifting aspects of our lives. They have relation to our ancestors but also our lived experiences, our languages, our physical bodies, and the systems into which we are born and move. For American Indians and global indigenous peoples, those systems have largely been created to take away land and resources, without consent and without attention to the ways that peoples have shared in reciprocal relationships with particular environments for millennia. While American Indian tribal nations are sociopolitical units who have the right to determine belonging, which may or may not be based on biology, the construction of indigenous identity is yet another way to manage and limit those definitions from the outside and to seek another resource, within the bodies and ancestral remains, within the genes of the people. Through literature and concrete partnerships, better ways can be imagined and constructed, and both new and traditional identities can emerge including new possibilities for indigenous scientists.

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Species of Biocapital, 2008, and Speciating Biocapital, 2017

Stefan Helmreich and Nicole Labruto

Introduction

The store of science studies work theorizing the conjuncture of economic action and biotechnology is well stocked. Scholars in anthropology, sociology, history, and literary theory have generated a variety of concepts: biovalue, genetic capital, the biotech mode of (re)production, the organic phase of capitalism, genomic capital, life as surplus, the bioeconomy, and, perhaps most prominently, *biocapital*, which is becoming the prevailing coin in academic exchanges about contemporary unions of biological science with profit-oriented enterprise. A taxonomy of *species of biocapital* is in order.

The word *species* refers not just to durable, though mutable, life forms but also to 'a particular kind or sort of coin or money' (OED), so that a classification of kinds of biocapital may take the form of an intellectual phylogeny or of an accounting or both. Following Pierre Bourdieu (1991 [1982]), who first defined four 'species of capital' (economic, cultural, social, and symbolic) and showed how they might be convertible into one another, such a classification

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could also manifest as a table of exchanges between different coinages. I consider all these possibilities here.

What is biocapital? Scholarship in the social study of biology has suggested that in the age of biotechnology, when the substances and promises of biological materials, particularly stem cells and genomes, are increasingly inserted into projects of product-making and -seeking, we witness the rise of a novel kind of capital: biocapital. The term, paging back to Marx, fixes attention on the dynamics of labor and commoditization that characterize the making and marketing of such entities as industrial and pharmaceutical bioproducts. It gives a fresh name to a phenomenon that Edward Yoxen, writing at the dawn of the biotech revolution in 'Life as a productive force: capitalizing upon research in molecular biology', described as 'not simply a way of using living things that can be traced back to the Neolithic origins of fermentation and agriculture' but 'a technology controlled by capital, … a specific mode of the appropriation of living nature—literally capitalizing life' (1981, p. 112). Biocapital also extends Foucault's *biopolitics*, that practice of governance that brought 'life and its mechanisms into the realm of explicit calculations' (Foucault 1978, p. 143). Theorists of biocapital posit that such calculations no longer organize only state, national, or colonial governance but also increasingly format economic enterprises that take as their object the creation, from biotic material and information to value, markets, wealth, and profit. The biological entities that inhabit this landscape are also no longer only individuals and populations—the twin poles of Foucault's biopower—but also cells, molecules, genomes, and genes.

Stem cells have been potent objects on this landscape because of their, well,…potency—or better, their potential potency, their capacity, under finely tuned circumstances, to grow into diverse sorts of cells, cells that might be employed as resources for regenerative medicine. One might argue that stem cells are animated by a double fetishism—infused with vitality because of the erasure of the labor and regulation that allows them to appear 'in themselves' in such places as laboratories and simultaneously imbued with life because of their origin in living things. Whether such fetishism dovetails with commodity fetishism is a complicated question—certainly stem cells' relation to market, gift, and national economics and imaginaries is multiple—but one of the more general claims of the present chapter will be that biological potency as such, in biocommerce, is often (mis)taken to be a primordial ontology upon which biocapitalism merely elaborates.

This chapter began as a review of two books. Kaushik Sunder Rajan's 2006 *Biocapital: The Constitution of Postgenomic Life* (Duke) and Nikolas Rose's 2007 *The Politics of Life Itself: Biomedicine, Power, and Subjectivity in the*

Twenty-First Century (Princeton) each propose a diagnosis of scientific, ethical, and cultural transformations in the way we think of life—biological and social—in the era of capitalized molecular biology, biotechnology, and stem cell and genomic medicine.

Sunder Rajan's *Biocapital* argues that life science commodities—for example, therapeutic molecules, genome sequences, and pharmaceuticals that promise future health—require analysis of capitalist practices as well as of the correlated citizen, corporate, and scientific subjectivities materializing alongside such activities. University and corporate biosciences have become porous to one another, with the circulation of biomaterials between labs governed by novel regimes of buying and selling—regimes set in place by regulatory transformations permitting the holding of intellectual property in biological matter and knowledge. Contouring this landscape, too, are infusions into genomics of money from venture capital. Speculative finance mirrors the speculations of biotechnology. The subjectivities in the making—for scientists, doctors, and patient advocacy groups—also tune to future-looking financescapes. The biocapitalist ethos takes nationally particular forms, too. US rhetoric organizes around sentiments of *salvation*, seeing the promise of genetic medicine in millennial terms, powered by languages of hope and hype; Weber's *The Protestant Ethic and the Spirit of Capitalism* (2001 [1905]) is the key intertext here. In India, a narrative that highlights the importance to the nation of biotechnology prevails; bioproducts promise to make India a 'global player'. Biocapital also depends on older, colonial structures of subordination as well as on new logics, requiring examination of 'where value resides as biology becomes an information science' (p. 41). Sunder Rajan thus tracks permutations in the 'explicit calculations' about 'life and its mechanisms' that Foucault saw as key to biopower.

In *The Politics of Life Itself*, Rose explores how novel forms of personhood, citizenship, race, brain/mind, and crime are under construction as people position themselves in relation to technologies of genetic mapping, genetic diagnosis, genetic counseling, genetic therapy, and genetic profiling. Contemporary biopolitics operates at the level of the *molecular* and from that seat organizes landscapes of risk and ethical subjectification. Family, personhood, race, and crime are refigured as the stuff of biology is made malleable. Rose concludes *The Politics of Life Itself* with a meditation on 'The Spirit of Biocapitalism', detecting an 'elective affinity' between the new molecular bioeconomics of 'life itself' and the modes through which, for example, doctors and patients work on human corporeal being, a corporeal being increasingly fungible and multiple.

Sunder Rajan and Rose have not been alone in their analyses. Below, to make sense of the genesis of discussions of biocapital is a timeline of publications, starting with Marx:

1867

Karl Marx in *Capital* defines *use value* and *exchange value* as, respectively, the value of things in use and the value that things acquire when set against one another as commodities. For Marx (who inherited these terms from Aristotle, Luther, and Smith), use value could be natural or conventional, though Marx sometimes described 'nature'— materialized in such substances as cultivated soil or the human body—as containing 'means of production already produced' (quoted in Franklin 2007, p. 106). This framing posits generativity (or reproductivity) as an elemental property of the natural.[1]

1884

Frederick Engels in *The Origin of the Family, Private Property, and the State* theorizes a distinction between means of production and means of reproduction, suggesting that women's subjection in marriage is aided by their domination as a class of unpaid workers responsible for the material reproduction of persons in households.

1905

Max Weber in *The Protestant Ethic and the Spirit of Capitalism* suggests that in post-Reformation Europe, Calvinist ethics of hard work and rationality underwrote the assignation of moral meaning to capital accumulation, which could be read by believers as a secular sign of salvation for which they were already predestined.

1976

Michel Foucault in *The History of Sexuality, Vol. 1*, theorizes *biopower* as that which made it possible for nation-states to bring 'life and its mechanisms into the realm of explicit calculations', that is, to summon forth the bodies of individuals and populations as elements to be governed and managed in the service of such social imperatives as nation-building and colonial expansion.

1981

Feminist scholars Olivia Harris and Kate Young, commenting on Engels in 'Engendered structures: some problems in the analysis of reproduction', argue against naturalizing—that is, locating in the ground of the biological—a distinction between reproduction and production.

1981

Marxist scholar Edward Yoxen publishes 'Life as a productive force: capitalizing upon research in molecular biology', in which he argues that a shift in the 'appropriation of living nature' takes place when capital begins to operate on biotic stuff at the molecular level.

1987

Literary critic Hortense Spillers (1987) in 'Mama's baby, papa's maybe' examines how the reproductive capacity of slaves under chattel slavery in the antebellum American South was conscripted by slaveholders into producing more slaves as property and as potential capital.

1988

Rural sociologist Jack Kloppenburg (1988), in *First the Seed: The Political Economy of Plant Biotechnology, 1492–2000*, offers a history of the capitalization of plant matter.

1992

Anthropologist Paul Rabinow in 'Artificiality and enlightenment' coins the term *biosociality*, arguing that genetics, immunology, and environmentalism are 'leading vehicles for the infiltration of technoscience, capitalism, and culture into what the moderns called "nature"'(1992, p. 245).

1992

Anthropologist Marilyn Strathern in *After Nature* (1992a) and *Reproducing the Future* (1992b) describes biological substance modified and capitalized as 'nature, enterprised-up'.

1993

Ecologist Walter V. Reid (1993) publishes 'Bioprospecting: a force for sustainable development' in *Environmental Science and Technology*. The term, a compression of 'biodiversity prospecting', refers to scouting in 'natural' settings (e.g. rainforests) for biological material (e.g. from plants) or information (e.g. traditional or indigenous knowledge) that may provide leads for natural products that can be industrialized or commercialized.

1995

Historian Harriet Ritvo (1995) in 'Possessing Mother Nature' offers a history of the remaking of livestock breeding in eighteenth-century Britain, when curated pedigrees emerged as tools to establish markets in what she terms *genetic capital* (see also Derry 2003 on the profit motive in breeding cattle, dogs, and horses beginning in 1800).

1997

In *Modest_Witness@Second_Millennium*, the historian of biology Donna Haraway (1997) discusses a shift 'from kind to brand' in the taxonomy of living things in the days of biotechnology. OncoMouse™ is an exemplar of the new branded biology.

1997

Physicist and critic of development Vandana Shiva (1997) publishes *Biopiracy: The Plunder of Nature and Knowledge*, building on then recent activist analyses of bioprospecting that construe the activity as a neocolonial practice of resource extraction, in which wealthy nations or companies dispossess

poorer nations or people of their territorial, organic, or ethnobotanical inheritances, often at profit.

2000

Historian Hannah Landecker (2000), in 'Immortality, *in vitro*', examines the case of the immortalized cancer cells of Henrietta Lacks, showing how they were serially imagined as valuable as the gift to science of an unknown woman, as the property of science, and, when they were discovered to originate in the body of a black woman, as two things: by some scientists, in line with racist visions of black sexuality, as hyperfecund, and by Lacks's family and advocates as a sign of an historical and continuing dispossession in the United States of black women from their bodies as property. Landecker made early versions of this argument in 'Between beneficence and chattel: the human biological in law and science' (1999).

2000

Medical sociologist Catherine Waldby coins the term *biovalue*, 'generated wherever the generative and transformative productivity of living entities can be instrumentalized along lines which make them useful for human projects' (2000, p. 33).

2001

Anthropologist Chaia Heller (2001) in 'McDonalds, MTV, and Monsanto: resisting biotechnology in the age of informational capital' theorizes 'biotechnology as a mode of production', argues that scholars might name a new moment in capitalism, *the organic phase of capitalism*, in which 'capital targets the reproductive dimensions of cultural and biological life as loci for intensified production and commodification'.

2001

Science studies scholar Mike Fortun (2001) in 'Mediated speculations in the genomics futures markets' suggests that understanding the business of genomics requires attention to its speculative logic, which he examines by demonstrating the role of 'forward-looking statements' in generating investment and profit. This work elaborates his earlier interest in the rhetoric of speed in genomics (1999) and sets the stage for his 2002 argument that genomics operates in the 'future anterior', the *what-will-have-been*—the promise—an argument he will elaborate in *Promising Genomics: Iceland and deCODE Genetics in a World of Speculation* (2008).

2001

Anthropologist Margaret Lock's 'The alienation of body tissue and the biopolitics of immortalized cell lines' (2001) fuses political economic analysis with Foucauldian attention to body politics.

2001

Sociologist Nikolas Rose argues that new markets in health create a circumstance in which 'biopolitics becomes bioeconomics' (2001, p. 15).

2003

Anthropologists Sarah Franklin and Margaret Lock define *biocapital* as a kind of wealth that depends upon a 'form of extraction that involves isolating and mobilizing the primary reproductive agency of specific body parts, particularly cells, in a manner not dissimilar to that by which, as Marx described it, soil plays the "principal" role in agriculture' (2003, p. 8). Franklin and Lock understand this biocapital to be underwritten not only by production but also by reproduction. Their thinking emerges from a May 2000 conference at the School of American Research, which they recall thus: 'Imagining ourselves (re)writing volume 1 of (bio)*Capital*, we attempted to specify as precisely as possible the range of forces at work in the transformation of life and death into means to (re)production and, in turn, into component parts that together compose an emergent global biological economy' (p. 13). Franklin's contribution to *Remaking Life and Death: Toward an Anthropology of the Biosciences*, the volume that emerged from this workshop, was entitled 'Ethical biocapital'.

2003

Sociologist Charis Thompson argues that the *biotech mode of (re)production* operates with 'promissory capital', 'capital raised for speculative ventures on the strength of promised future returns' (quoted in Franklin and Lock 2003, pp. 6–7). In her *Making Parents: The Ontological Choreography of Reproductive Technologies*, Thompson turns her attention to what she calls the 'biomedical mode of reproduction' (2005).

2003

Science studies scholar Kaushik Sunder Rajan in 'Genomic capital: public cultures and market logics of corporate biotechnology' defines 'biocapitalism' as that which asks, 'how "life" gets redefined through the contradictory processes of commodification' (2003, p. 87). His biocapitalism has five features: a rhetoric of speed, corporate/university connections, porosity between commodity and gift economies in labs, excessive production, and biosocialities tuned to market logics.

2003

Anthropologist Cori Hayden in *When Nature Goes Public: The Making and Unmaking of Bioprospecting in Mexico* shows how bioprospectors often seek to create capital through channeling biodiversity through 'slightly choppy' (2003, p. 10) networks that mix economies of purchase, benefit-sharing, dispossession, profit, and promise, many of which turn out to be situated in larger frames of North-South political economic inequality.

2005

Literary theorist Eugene Thacker in *The Global Genome* (2005) fixes on how the fluidity of genetic information as data permits it to be used as a currency in globalization. Thacker draws on the Marx of the *Grundrisse* as well as on Foucault to develop a theory of 'biological exchange' that aligns information management with moments in the movement of capital: encoding/production, recoding/circulation, and decoding/consumption. Thacker thinks through the excess of bio-information using the work of Georges Bataille (1967) in *The Accursed Share*, which argues that the accumulation of surplus is not always fed back into production, but is often spectacularly spent on lavish wastage.

2006

Kaushik Sunder Rajan in *Biocapital: The Constitution of Postgenomic Life* follows Marx in parsing *biocapital* into industrial, commodity capital (such as therapeutic molecules) and speculative, commercial capital (such as stocks), which later are often underwritten by quasi-religious sentiment, in the way Weber argued that the rise of merchant capital was motored by the Protestant ethic. Sunder Rajan uses Bataille to think about how speculation underwrites and permits practices of excess, particularly in the over-the-top expenditures of biotechnology start-ups in the United States.

2006

Anthropologists Adriana Petryna, Andrew Lakoff, and Arthur Kleinman in *Global Pharmaceuticals: Ethics, Markets, Practices* (2006) examine the inequalities that organize world distribution and markets in pharmaceuticals, a global economy in which access and excess are often inversely related. They draw on Bourdieu's notion of capital to locate pharmaceutical economies—of patents, products, and promises—in regimes of economic, cultural, material, and symbolic capital. They do not put the package together as *biocapital*—though Lakoff argues that in pharmacogenomics, 'Biopolitics and the market were to be brought together through the application of genomic knowledge' (2005, p. 171).

2007

Nikolas Rose extends earlier arguments of his that a 'mutation' from *biopolitics* to *bioeconomics* characterizes the dominant social order in at least the United States and Europe, writing that 'vitality has been decomposed into a series of distinct and discrete objects, that can be stabilized, frozen, banked, stored, accumulated, exchanged, traded across time, across space, across organs and species, across diverse contexts and enterprises, in the service of bioeconomic objectives' (2007, 67). Rose notes that biocapital is already a phrase circulating in the world of pharmaceuticals, frequently as a company name or service.

2007

Sarah Franklin in *Dolly Mixtures* looks at the history of 'stock' in livestock to think about the braided logic of breeding and wealth creation, from pre-capitalist to capitalist modes of accumulation, writing that 'capital in the older sense of stock derives out of a combination of genealogy, property and instrumentality' (2007, p. 57).

2007

Science studies scholar Joseph Dumit theorizes *surplus health* as that which pharmaceutical companies conjure in order to 'add medications to our life through lowering the level of risk required to be "at risk"' (quoted in Sunder Rajan 2007, p. 81). Dumit's Biomarks (or, sometimes, BioMarx) experiment operates by substituting 'health' for 'labor' in *Capital* (consult Dumit 2012).

2007

Political theorist Melinda Cooper (2007) in 'Life, autopoiesis, debt: inventing the bioeconomy' argues that capitalist culture operates through 'delirium', in which the drive of capital to overcome its own material limitations not only finds new resources but also constantly redefines the 'nature' of resources (e.g. through turning debt or other crisis moments into value) in order to create surplus. Her later book *Life as Surplus* (2008) elaborates this argument.

This chronology does not take in as many vectors of origin for biocapital as it might. Missing are chronicles of molecular biology (e.g. Kay 1993; Wright 1994; Keller 1995; de Chadarevian 2002), histories of modernist agricultural technique (e.g. Fitzgerald 1990; Boyd 2003), studies of colonial and postcolonial enterprise (e.g. Sidney Mintz's 1985 analysis of the plantation as a fusion of farm and factory in *Sweetness and Power*), anthropologies of organ donation and trafficking (e.g. Hogle 1999; Scheper-Hughes 2001), and social analyses tracking the rise of markets in racialized genomics (e.g. Fullwiley 2007; Montoya 2007; TallBear 2008). Also absent are works theorizing transformations in capitalism and governance more generally (e.g. Harvey 1989; Comaroff and Comaroff 2000; Maurer 2000; Jasanoff 2005).

The timeline, though organized stratigraphically, also does not indicate which writers relied upon which to develop their arguments—which could trace how the concept of biocapital has developed. Co-citation or co-word analysis might make common links clear (see Cambrosio, et al. 1993), though a search for 'biocapital' in Thomson Scientific's Web of Science database in 2008 yielded only five journal articles. Plugging 'biocapital' into Google Scholar in 2008 picked out Franklin and Sunder Rajan as key exponents of

the concept, showing 24 citations to Sarah Franklin's 2003 articulation and 28 to Sunder Rajan's 2006 book and, strikingly, since both publish in anthropological venues, no cross-citations between the two, suggesting that there may be two scholarly conversations in motion here. A simple scientometric approach, of course, would be difficult to cash out as a full tracing of influence. Different scholars cast more and less finely meshed citation nets.

These caveats in mind, just below is a tentative genealogy, full, as all genealogies are of repetitions, omissions, mistakes, surprises. Its nodes are the names of authors of peer-reviewed, published works that contribute to discussions of biocapital. Names are keyed to years and each scholar appears only once (in connection with either their first articulation of a concept important to biocapital or their most significant statement on the matter). The lines represent direct, more-than-in-passing citation. Turning away from the automatic information gathering of citation analysis, I sought to locate authors' developments of concepts central to biocapital by using an antique method: reading (Fig. 35.1).

Gillian Beer has suggested that Darwin's forking figure in the final pages of *Origin* 'could as well be interpreted by the eye as a shrub, branching coral, or

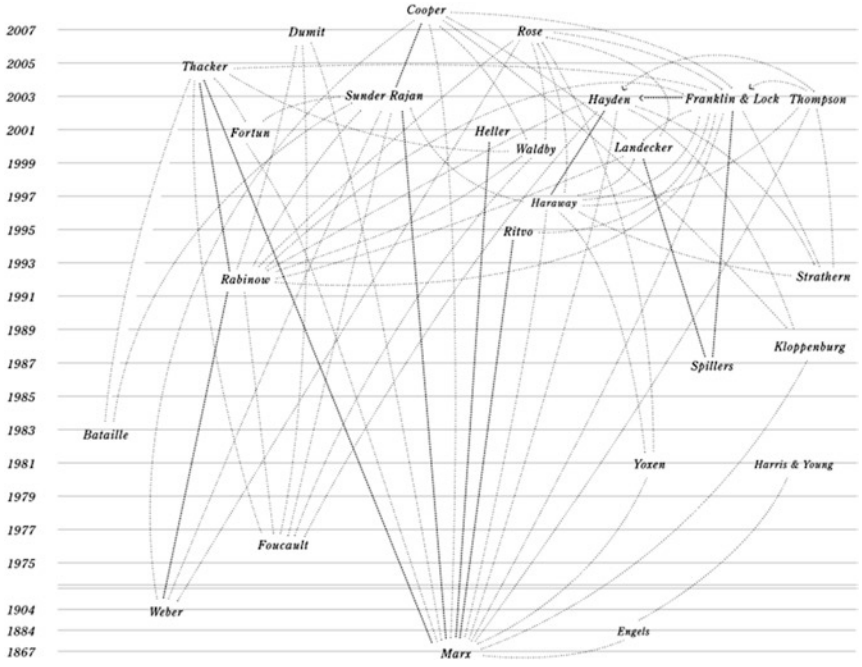

Fig. 35.1 With apologies to Charles Darwin, a diagram of the divergence and convergence of taxa of biocapital. Rendered by Michael Rossi

seaweed' (2000, p. 86). The figure above is even weedier than Darwin's, but even within this thicket, two clusters of writing on biocapital can be discerned.

One cluster—around Sarah Franklin, Margaret Lock, and Charis Thompson, and drawing on Marilyn Strathern, Donna Haraway, and Paul Rabinow—might be called Marxist feminist. Here the binary of *production* versus *reproduction* is key, as are questions to do with sex/gender and race (particularly in work about reproductive technology). The remaking of boundaries between *nature* and *culture* is a central concern—one reason attention to the changing substances and generativities of biology, emblematized by Hannah Landecker's work on the history of tissue culture, is also a signature feature of this scholarship.

A second cluster—around Kaushik Sunder Rajan, Eugene Thacker, and Michael Fortun, and drawing on Haraway and Rabinow—pays attention to questions of meaning, though less to biomatter. Focusing on questions of information management and speculation, this scholarship has a Weberian flavor. Call it Weberian Marxist; relations of production are described alongside accountings of ethical subjectivity.

Strains of each line are present in the other. And Marx's political economy and Foucault's biopolitics operate as crucial conditions of possibility for each. Melinda Cooper's work marks a fusion of the lines.[2]

Another feature of the discussion that leaps out is the acceleration of the discussion in the late twentieth century. For scholars interested in new kinds of financial speculation of genomics, biocapital tracks biotechnological innovation (recombinant DNA, PCR) as well as the history of legal agreements between universities and companies about the commercialization of university property, which begins in 1980 with the passage in the US Congress of the Bayh-Dole Act, which permitted universities and their employees to retain rights in patented inventions developed with federal monies and, if desired, to license or sell those inventions to private business. Academic-industrial biotech hybrids became common in the United States after the Supreme Court in 1980 permitted the patenting of modified organisms in *Diamond v. Chakrabarty*. For theorists of biocapital interested in the intercalation of reproductive technologies (IVF, cloning, pre-implantation genetic diagnosis) with new kinds of relations of commoditization (of women's reproductive labor, most notably), biocapital is entangled with changing relations of reproduction and kinship.

The two schools of thinking on biocapital also have distinct orientations: they represent two sides of what, once upon a time, was called the *substantivist* position in economic anthropology. Against *formalist* economic anthropolo-

gists who believed that a common rational logic animated all exchange, *substantivists* sought to examine logics of exchange with respect to the cultural values that motivated them—values to do, for example, with kinship or prestige (see Isaac 1993).

The cluster of which Franklin, Landecker, Lock, Thompson, and Hayden are a part, I suggest, represents a substantivism interested in the changing substances of biology. Associates of this cluster attend to matters of generativity and reproduction. But they are careful not to take generativity and reproduction as 'natural laws' (as Marx did). In earlier work, Franklin, writing with Helena Ragoné (1998, p. 2), cautioned against 'the relegation of "reproduction" to a domain of "natural" or biological facts … considered prior to, and separate from, sociality'—an argument that echoes an earlier position in Marxist feminist anthropology, in a piece by Olivia Harris and Kate Young (1981), entitled 'Engendered structures: some problems in the analysis of reproduction', in which the authors argue against positing, as did Engels in *The Origin of the Family, Private Property, and the State*, a fundamental difference between reproduction and production.

A scholar like Sunder Rajan, meanwhile, may be read as a substantivist who looks at moral economies, joining Marxist political economy with a Weberian attention to meaning. Though he offers clear analyses of molecular biology lab practices, he is less interested in the substances of the biological, calling attention instead to the constructedness of biological facts upon which speculative exchange value is predicated.[3] In *Biocapital*, he takes care not to impute any particular ontology to biological material—though by not engaging the arguments of writers like Franklin and Lock about the new substances of 'life itself' which (via such materials as stem cells) contain and morph histories of sex/gender, race, colony, and nation, he misses a chance to dig into the politics of generation and reproduction that are in the remaking in biotechnology. Together, however, Sunder Rajan's and Franklin and Lock's attention to the making of facts and the remaking of generativity can complicate such analyses as Eugene Thacker's, which argues that bioengineering relies on a '"molecular species being," a species being in which labor power is cellular, enzymatic, and genetic' (2005, p. 40). That formulation is a molecular rewrite of Engels's famous 1876 reflection on 'The part played by labor in the transition from ape to man', in which Engels naturalized labor, via evolutionary theory, as that process at the heart of anthropoid organisms' self-making (see Engels 1884).

But let me cut across these substantivisms and offer a less nit-picky classification. Taking a cue from evolutionary biology, I'll pick an analogous structure that operates in the bodies of all the work I've discussed: the very concept

of *biocapital* (and its similarities). Comparing how the concept fares in different bodies of work may permit us to set up a series of exchanges among them.

What is biocapital? My sense is this:

In *Capital*, Marx describes the circulation of money as capital—in which 'More money is finally withdrawn from circulation than was thrown into it at the beginning' (1867, p. 251)—using the formula M-C-M', where M stands for money, C for commodity, ' for the surplus value gained in a profitable exchange of a commodity for money, and M' for the total capital produced by that exchange. For the biotech imagination, I suggest an analogous formula to describe the making of biology into capital: B-C-B', where B stands for biomaterial, C for its fashioning into a commodity through laboratory and legal instruments, and B' for the biocapital produced at the end of this process, with' the value added through the instrumentalization of the initial biomaterial.

What does B-C-B' look like for the theorists discussed above? How do different species of biocapital organize the metabolic pathway that makes B into B'? What 'primes' biology?

I have suggested that the sentiment of many biotech boosters has them imagining B' already to be latent in B—to believe that biological process itself already constitutes a form of surplus value and profit production (Helmreich 2007). This logic naturalizes biotech. Biological generativity is configured as accumulated labor power, the products of which can be harnessed to create productive futures. This belief is based, it bears emphasizing, on a metaphor: that organisms are laborers (an equivalence declared even by Marx, who saw the natural consumption of eating entailing production of the body [1857–58, p. 228]). The negative image of biocapital then becomes *necrocapital*, dead matter, like fossil fuel, put to unregenerative, zombie-like work. But we must be careful not to imagine reproduction as a transparently 'natural' process, as though organisms' coming-into-being straightforwardly designates them as what Marx would have called 'means of production already produced', as though their productivity is the essence of their *species being*. To see matters this way is to see organisms as natural factories or assembly lines, when in fact they only become so in certain relations. As Landecker argues, contemporary biology has become expert at stopping, starting, suspending, and accelerating cellular processes, wedging these dynamics into processes that look like a molecular version of industrial agribusiness. But biotech geese cannot lay golden eggs without daily tending.

What does thinking in terms of B-C-B' permit us to do? To begin, this accounting points back to Bourdieu—a figure mostly absent from theories of biocapital—and allows us to name how B' corresponds to economic, cultural,

social, and symbolic species of capital (also, in the bargain, making explicit the Darwinian, Marxist, Weberian, and Foucauldian ancestries in play in different theorists' formulations). It could allow us to draw up a table of exchanges between different B primes, species of biocapital.

But—to draw upon the evolutionary biology idiom once again—such a classification assumes the neatness of the species concept, which is these days in crisis; recent research has seen the creation of transspecific hybrids and contemporary molecular biology has discerned thick lateral gene transfer tangling up taxonomic boundaries almost everywhere. But more, sorting biocapital into species has the effect of holding stable the item against which different species of biocapital exist at all—namely, *capital* itself. What if we asked not what happens to biology when it is capitalized, but asked whether capital must be the sign under which all of today's encounters of the economic with the biological must travel? It is certainly the case, as medical anthropologists such as Margaret Lock (2002), Lawrence Cohen (2005), and Leslie Sharp (2006) have shown, that the circulation of organs is not in every instance overdetermined by capitalism (the fraught language of donation and trafficking is a giveaway). Cathy Waldby's *biovalue*, Sarah Franklin's *breedwealth* (1997; see also Franklin 2006 on *biowealth*), and Donna Haraway's 2008 *encounter value* are germs of theory that undo the capitalocentrism of so much writing on biocapitalism—and also, perhaps, the emphasis in such writing on the commodity form. Emerging social histories of 'bioeconomy'—looking back to early population sciences to think through political economy—might be another place to look for analyses that include but reach beyond capitalism (see Larsen 2005). What if we imagined biovalue and bioeconomy through J. K. Gibson-Graham's *The End of Capitalism (As We Knew It): A Feminist Critique of Political Economy* (1996), which seeks to break away from the delirious reinscription of capital that happens even in its Marxist critique? What if, refusing to make capital into the coin of exchange across these concepts—and, more, refusing to trust that exchange as such can permit the adequation of different values—we found that capital itself, like the species concept, was unstable, was not so easily reproduced, or so generative, or omnipresent, after all?

Speciating Biocapital

In 2008, Stefan Helmreich identified two genealogies of writing on *biocapital* that examined how biological materials were being leveraged into profit-oriented undertakings: a 'Marxist feminist' lineage that described change and

continuity in economies of reproduction, transformations in the boundaries between nature and culture, and manipulations of biotic substance; and a 'Weberian Marxist' cluster that attended to relations of production, ethical subjectivity, and economic sentiment. While some scholars have offered the *bioeconomy* (Birch and Tyfield 2013; Birch 2016; Hauskeller and Beltrame 2016) as a more expansive analytic, biocapital has continued to gain traction. At the same time, in the age of the post-genome, what counts as 'the biological' has multiplied, with epigenetics and microbiomics, to take two examples, complicating the genetic determinism organizing early attempts to aim biology at market ends. As Hannah Landecker writes, the 'economics and politics of life are changing, but so are biologies' (2016, p. 44). This postscript identifies four primary speciations of biocapital since 2008:

1. Into ever-more international and transnational contexts, as scholars have taken the concept into domains outside the West, often into global South, postcolonial, decolonial, and other settings.
2. Into domains concerned with biological processes beyond the scale of the genetic and cellular, reaching toward accounts of how full organisms and their relations—non-human and multispecies—are being newly capitalized.
3. Into discussions of embodiment that extend feminist concerns with sex/gender, now centering not only on such practices as IVF and stem cell science but also on different exploitative uses of human bodily, affective, reproductive, and procreative labor—in sex work, in surrogacy, in domestic labor, and more.
4. Into discussions of environments, particularly in the age of the Anthropocene and the moment of epigenetics and microbiomes.

Biocapital is therefore no longer so centered on molecular processes isolated and elaborated in US or European laboratories (see Vermeulen et al. 2012 on 'economies of life'). New trajectories reflect increased concern with non-Western and postcolonial science, multispecies engagements, post-genomic biology, and value beyond the economic sphere.

Non-Western/Global South Elaborations

Scholars have continued tracking biocapital in non-Western/global South contexts. Sunder Rajan (2012) examines the 'global knowledge formations' that transnational life sciences create. Aihwa Ong in *Fungible Life* (2016) fol-

lows scientists in Singapore's Biopolis as they make genomic information interchangeable across markets. Biopolis scientists rely on British colonial racial categories to create treatment options for what they claim to be particularly Asian infectious diseases and the health outcomes of Singapore's populations (see Waldby 2009 on Biopolis as brokering a 'utopian vision of a regenerative bioeconomy' and Fischer 2013's attention to the everyday ways biosciences are employed in Singapore not only in market frames but also as tokens in cross-national science diplomacy). Moving explicitly away from genetically animated visions of biocapital, Jean-Paul Gaudilliere (2014) considers how traditional Indian Ayurvedic knowledge is mobilized to build pharmaceutical markets, in marked distinction to the molecular paradigm that has characterized the drug industry.

Biocapital has also received attention in non-English-language literatures as translations of the biocapital conversation become available (Turrini 2011). From a study of pharmaceuticals and risk in Brazil (Rodrigues et al. 2015) to a theorization of transspecies reproductive technologies in Italy (Balzano 2015) to an investigation of the making of biocapital using biosafety scenarios in Spain (Marco et al. 2015), *biocapital*/*biocapitale*/*biocapitalismo* have become concepts with futures of their own, naming changing landscapes of national and transnational science, ethics, and risk.

New Scales and Species

Biocapital has been applied to new biotic scales and a widening range of species. In *When Species Meet* (2008), Donna Haraway imagines a 'Marx-equivalent' writing *Capital* today as *Biocapital*, volume 1, transcending Marx's human exceptionalism and accounting for the multispecies encounters that shape labor and commodities. To Marx's *use value* and *exchange value*, Haraway adds *encounter value*, a genre of interspecies value that, pace Gibson-Graham, does not subsume all value to the market. In *The Mushroom at the End of the World* (2015), Anna Tsing attends to 'unpredictable encounters' between humans and nonhumans in the lifeworlds of matsutake mushrooms as they are grown, gathered, and traded in spaces of 'capitalist ruin.'

Cutting-edge biotechnologies, such as the gene-editing technology CRISPR (Clustered Regularly Interspaced Short Palindromic Repeats), promise to reorganize multispecies relations; mythical hybrids, enhanced transspecies, and programmed extinctions become possible. Eben Kirksey (2015) reports on synthetic biologists creating bioengineered mice that may eradicate Lyme-disease-bearing ticks and mosquitoes crafted to blunt the travel of

malaria through mosquito-human populations. Scientists marry these gene-editing projects to aspirations for new 'transspecific' bioeconomies; researchers claim to want to undo the proprietary secretiveness of biotech companies like Monsanto in order to make what they call 'open source' and even anti-capitalist research platforms (and see Roosth 2013). The future of multispecies markets may transcend conventional economic exchanges of biocommodities, relying instead on minimally regulated private donor-funded projects that alter inter-species interactions—from the scale of ecosystems down to organisms' genomes. Use, exchange, and encounter value may be joined by what we could call *transaction value*, where such transactions are not only about economic exchange or affective encounters but rather and also about as-yet unmarked relations of sharing, transfection, contamination, and displacement.

Embodied Biocapital

The feminist genealogy of thinking about biocapital has extended traditions of examining sex and gender as formations that involve biological and social reproduction, arguing that biocapital is generated from these exploitative forms of embodied labor (Happe 2015. See also Taussig, Hoeyer, and Helmreich 2013 for an introduction to an issue of *Current Anthropology* on the new political economy of biomedical 'potentialities'). Authors in this vein also tend to look to the global South as sites for investigating unequal exchanges of affect and capital. Nurul Ilmi Idrus and Anita Hardon (2015) adapt the concept of biocapital to describe technologically enhanced human bodies and the relations into which they enter. In their research on sex workers in Indonesia, they look at how contemporary technological interventions into the bodies of sex workers and waitresses—in the forms of cosmetics, psychoactive drugs, and antibiotics—produce economic power but also dependency. In another domain in which the well-being of bodies is at stake, David et al. (2015) worry about the biocapitalization of human bodies in the context of medical aid to HIV patients. Their study of the new industry-oriented funding philosophy of the Global Fund, a key distributor of subsidized drugs to infected patients in non-Western countries, raises ethical concerns about the evaluation of bodies that are 'captive' to programs that keep them alive but with uncertain future commitments and treatment policies.

Kalinda Vora (2015) focuses on the unidirectional movement from India to the United States of the 'vital energy' produced by human labor in gestational surrogacies, call centers, domestic labor, and more. As affective and

reproductive labor are commoditized and outsourced, human biocapital from India supports life in the United States, producing new socialities alongside economic value (see Murphy 2017 for an historical analysis of rubrics that quantify the cost and value of populations).

Environments

In the era now marked as the Anthropocene, in which human activities have irrevocably degraded the livable world, Anthropocenic concerns increasingly drive the development of new biologically derived commodities, as biocapitalist forms define interventions, solutions, and ethics related to social-ecological problems. Nicole Shukin (2016) looks to a group of Fukushima residents who defied government orders to evacuate as a way to evaluate 'resilience' as an embodied biocapitalist resource, one that is 'consciously cultivated and valorized by corporate and state institutions' that benefit economically from populations' ability to manage life amid disaster conditions. Here are entwined human biocapital, resource extraction, and economic imperatives, oriented toward an activist, anti-capitalist politics in a technologically mediated landscape (see also Acero 2012 on environment, gender, and 'citizen controlled' biotechnology).

Concomitant with new understandings of the role of humans in making environments come post-genomic research agendas that see extracellular factors playing crucial roles in shaping biologies (Richardson and Stevens 2015). Parallel to CRISPR's genetic reductionism are postgenomic biologies such as epigenetics (Landecker and Panofsky 2013; Meloni and Testa 2014), genres of life science that modulate assumptions of genetic fixity to account for how organic and social forces combine to render 'the biological' plastic and porous to 'the environment' and to history (Landecker 2016). Think, for example, of how our very food bears 'the traces of scientific and economic rationalizations of plant and animal bodies' (Stassart 2003: 449), a dynamic that brings earlier bioeconomies into the multicellular, metabolic processes of today's populations (and that in turn has been narrated in ways that suggest that economic theorizations and measures of human biological life chances may be reanimated in light of new biologies; see Almond and Janet 2011; Pentecost 2016 and see Meloni and Testa 2014 on new attempts to capitalize epigenetic understandings of nutrition). 'Environment'—global, bodily—is coming to matter in new ways as scholars investigate research on the factors that influence biological, social, and economic outcomes (Heckman 2007).

If the concept of 'species' is becoming ever more ontologically unstable, so are 'species' of biocapital, edited and spliced into more heterogeneous social, political, and economic relations. As scholars have shown, biocapitalist forms and operations increasingly rely on the intracellular/intraecological exchanges, encounters, transactions, and drives that biotechnologies make possible. Most contemporary biocapital emerges from exploitative, neoliberal models of commodification and circulation—trans-infecting biologies, ideologies, and markets from the inside out. And as post-genomics, environmental remediation, climate change amelioration, global disease eradication, and resource scarcity motivate new biological research platforms, biocapital will see new inventions, edits, contaminations, and wirings, yielding new species of biocapital for scholars to probe critically.

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Notes

1. For Aristotle, *generativity* was such an essential property of nature that he saw the application of its logic to the artifice of exchange as an ethical problem. In *Politics*, he wrote, 'Currency was intended to be a means of exchange, whereas interest represents an increase in the currency itself. Hence its name [*Tokos* ('offspring')] for each animal produces its like, and interest is currency born of currency. And so of all types of business this is the most contrary to nature' (I x 1258a27) (1981). Martin Luther had a similar view: 'I do not understand how a hundred guilders can make twenty profit in a single year, or even one guilder make another. Nothing like this takes place by cultivating the soil, or by raising cattle, where the increase does not depend on human wits, but on God's blessing' (1961 [1520], p. 482).
2. The tree representation overlooks important mechanisms and vehicles for the travel of concepts. It leaves out the lateral transfections and endosymbiotic fusions consequent on classes taken, conference papers heard, drafts circulated, and readers' reports rendered (Rabinow started giving a biosociality talk in 1990; Fortun was speaking on 'Projecting Speed Genomics' as early as 1994; Thompson's notion of the promissory circulated at a 2000 conference; and Sunder Rajan's dissertation, with the same title as his book, was finished in 2002, etc.). It also leaves out the fact that authors' positions change over

time.Any model of the inheritance of properties would also map out a story of the transmission of what Bourdieu called *academic capital* (with credit and credibility not far behind—see Latour and Woolgar's (1986, p. 201) circle diagram of cycles of conversion between types of capital, in which recognition →grant → money → equipment → data → arguments → articles → recognition → and so on…).

3. Compare social theorists of finance as far back as Gabriel Tarde, who in 1902 looked to organic metaphors to think through capital as a relationship between potentialities of invention and accumulation. Tarde developed the metaphors of *germ capital* and *cotyledon capital* to account for the origin and maintenance of capital not exclusively in accumulated labor but in ratios of difference and repetition realized in reproduction and production imagined as contingent collaborations of human, machine, and nature (Lépinay 2007b). Complicating another biological metaphor in social studies of money, the work of Vincent-Antonin Lépinay (2007a) critiques the notion that financial formula packages such as Capital Guarantee Products are 'parasitic' on the industrial goods to which they putatively refer, arguing that such products circulate in the same sphere of valuation as the 'organisms' to which they are calibrated. Such a critique of how 'parasitism' is employed to describe derivative financial instruments could be extended to direct attention to the parasite metaphor's anti-Semitic resonances in the history of finance in the West (particularly in characterizations of lending money at interest) (see Raffles 2007).

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37

Human Tendencies

Ed Cohen

Thinking Species

Until the eighteenth century, human beings did not actually appear as a species among other species. Indeed, as Michel Foucault taught us, until the Classical age humans dwelling in the "the West" did not actually inhabit the same representational space as other living beings (Foucault 1970). Things changed radically during the period in which Linnaeus formally nominated us *Homo sapiens* (Linné 1806).[1] In the tenth edition of his *Systema Naturae* the esteemed Swedish taxonomer established his famous binomial nomenclature and used the specification *sapiens* to qualify the genus *Homo* for the first time (Linnaeus 1758). In so doing, he designated us the supreme example of his highest class: primates. At the same moment that our new moniker was formalized another key modern parsing of humanness also coalesced: "population." Following on late-seventeenth century innovations in "political arithmetic," promulgated by John Graunt and William Petty, among others, "population" crystallized in the mid-eighteenth century as a demographic technology that evaluates—and hence *values*—aggregates of people as a state's most vital assets. Population makes these vital valuations and evaluations possible insofar as it considers people "statistically" (i.e., literally as a matter of

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"statecraft") (Buck 1977, 1982; Stigler 1986; Porter 1986; Hacking 1990). Population thus accounts for a nation's living citizens by representing them abstractly as a simultaneously vulnerable and valuable assemblage that requires governance, or as the contemporary idiom had it: police.[2]

Since as concepts both the human species and human populations seek to evoke how human beings live—and die—together, it makes sense that they quickly fused into two sides of the same coin. Population makes species makes sense as a way of sorting out who we are as living beings; conversely, species represents the statistical assemblages that figure human populations as if they were natural sorts. Hence, if the biologization of the human species implicates humans within the field of all living species, then because of its reciprocal articulation with population as an analyzer of state power, species also divides this political field in a *specific* way—in a way that following Foucault (1978, 2007) we might call biopolitical.

While we largely take for granted the idea that the human species naturally nominates us as living organisms, the concept of species actually renders the "natural" being of "the human" fairly problematic—if only because the "species concept" itself constitutes something of a conundrum. Although we use it unthinkingly, the real referent for species remains disputed, especially between those who hold genetic and ecological models of living beings.[3] While distinguishing between species as taxa and species as category seeks to reconcile some of these differences, species thinking still provokes much gnashing of epistemic teeth. Given its epistemological and ontological uncertainty, we might wonder what we actually *do* when we consider "being human" to mean "belonging to the human species." What sense do we make of ourselves when we make sense in these terms? And what sense do these terms, in turn, make of us?

Species is a peculiarly Western style of construing living beings. Its Latin etymon, *specere*, means to look at, behold, regard, or contemplate, but the concept that the Latin term translates actually descends from how Aristotle revised Plato's notion of *eidos*. Plato had conceived *eidos* as a transcendent, immutable, and perfect Idea from which all materialized being decisively falls away. Rejecting Plato's otherworldly bias as metaphysically unwarranted, Aristotle recasts the notion of *eidos* in two important respects: first, he uses *eidos* to designate the form that informs matter (so *eidos* in contrast to *hyle*); and second, he uses it to constitute the basis for logical classification (so *eidos* in contrast to *genos*). Thus Aristotle introduces a "tension between *eidos* as individual existence and *eidos* as known in definition." (Grene 1978, 128)[4] This defining tension underwrites the extension of *eidos*—and its Latin translation *species*—to the field of living beings: *eidos* and *species* fold together not

only the atemporality of form and the temporality of existence, but also the singularity of the idea and the multiplicity of being(s). Put otherwise, *eidos*-as-species finesses the relentless tensions between continuity and change and between singularity and multiplicity that underlie the dynamism of living being(s). However, in so doing, it conceals these essential paradoxes, which animate all life forms.

As a concept, species specifies the specific way that we as living (human) beings grasp the life worlds in which we live. Once it infuses our thinking about human existence—and our existence as "humans"—species thinking increasingly informs not only how we live, but also living as such (e.g., via the extinction of species). However, if we do not assume that species-being exhausts our (human) nature, we can begin to reflect on what belonging to a species does to or for "the human."[5] At the most literal level, being a species *secularizes* us. Within the ambit of early modern Europe, Christian eschatology, which oriented time toward the apocalypse to come, informed the temporal horizon of existence both theologically and politically. Its doctrine of the resurrection supposed a bifurcation not just of body and soul but also of life and afterlife. A human soul's travails within the earthly veil of tears merely prefigured an eternal temporality during which its "real life" would take place. Among the manifold implications of this pre-modern dogma, two concern us: (1) the most important thing about being human was not having a body (as we largely assume today) but being a soul, and (2) the soul constituted not only the otherworldly ground of personhood, but also the this-worldly basis for political belonging. Throughout the course of the sixteenth and seventeenth centuries, however, these constitutive precepts began to falter before the period's complex religious, political, economic, technological, philosophical, and military upheavals. Needless to say, a detailed account of these transformations exceeds my scope here. However, let me note two relevant sequela: (1) As Foucault (2007, 298) argues, following the Treaty of Westphalia in 1648, the eschatological frame for European politics, which hitherto imagined a universal Christian Empire as its apotheosis, no longer commanded unconditional allegiance; instead Europe fell into secular history as a coincidence of coeval nation-states "having a relation of utilization, colonization and domination to the rest of the world"; and (2) "The body" replaced the soul as the proper metonym for political, legal, and economic subjectivity such that the personification of "the body" underwrote a new secular political philosophy (C. B. Macpherson (1962) famously named this personification "possessive individualism").[6]

The emergence of the human species as the collective incarnation of these individual bodies naturalized our modern secular orientation. It literalized the

concept's etymological significance (the Latin *saeculum* means "the average duration of a man's life," a "*life*-time," or a "generation") in contrast to Christian eschatology's non-secular valorizations of *soul*-time (Robert et al. 1892, 497). Prior to the eighteenth century, if humans were referred to as a species, it was almost exclusively in a logical rather than a biological sense. Moreover, since species functioned primarily as a categorical determination that established identity-within-difference, it applied indiscriminately to all being(s). Thus Linnaeus, who first named our species-being as such, classified minerals as well as plants and animals as species, recognizing no essential distinction between species' animate and inanimate forms.[7] However, once the notion of species took a distinctly organic turn, as it did in the work of Georges-Louis Leclerc, Comte de Buffon—who explicitly disputed Linnaeus' usage and argued that only living beings speciate—species became conceived for the first time as a vitally *secular* matter of generation and generations (Sloan 1976, 1987, 1995; Farber 1972; Lovejoy 1959).

According to Comte de Buffon (1749, 2, 18), a species' temporal dissemination, its "faculty of producing its fellow creature (*semblable*)," constitutes its "real existence." If in order to be a species at all a species must endure through a multitude of successive iterations, then the generation(s) of new individuals constitutes its vital crux. Furthermore, a species only exists as "the constant succession and the uninterrupted renewal of the individuals who constitute it" (Comte de Buffon 1753, 384). From this perspective, sexual reproduction becomes—again for the first time—a means of reproducing "the species," rather than simply of engendering offspring.[8] Thus, Comte de Buffon (1753, 386) posits: "the species then is nothing other than the constant succession of similar individuals who can reproduce themselves together."[9] This reproductive criteria provides a simultaneously inclusive and exclusive notion of species: individuals belong to the same species if and only if they reproduce offspring who can reproduce more offspring—a criteria that essentializes sexual difference, foregrounds the troubling notion of hybridity, and casts "race" as a biological category.[10] Once the human species appears both to exist and to insist through generation and generations (i.e., through fleshly iterations that manifest in and through time) our "secular nature" actually comes to make sense to us *by making sense of us*.

Concomitantly, our species' secular self-characterization as a species carries an unacknowledged corollary: we only speciate as individuals. While the species endures through generations, it passes, or indeed *takes place*, through the temporal and spatial passage of individuals across a lifetime. In the *Premier Discours* that introduced his magnum opus, Comte de Buffon (1749) epistemologically commits himself to the individual as the only "real" natural unit:

"In nature only individuals really exist; genuses, orders, and classes only exist in our imagination." (38) Hence as he writes in a later volume: "All the similar individuals that exist on the surface of the globe are regarded as composing the species of these individuals." (Comte de Buffon 1753, 384) Individualism thus constitutes a logical, bio-logical, and biopolitical condition for conceptualizing the human species as a species. When Buffon incorporates the individual into natural history as the medium in which and through which the species inheres, he expands the domain proper to the possessive or liberal individual (a.k.a. "the personified body") from politics to nature.

Emerging in seventeenth-century England as a political response to monarchal absolutism and its discontents, liberal individualism introduced an ontological basis for rejecting the monarch's God-anointed prerogatives by affirming the individual's originary ownership of *his* own body (and of course individuals were inevitably male). Liberal political philosophy holds that "the body" represents the individual's natural property and therefore, as John Locke famously avowed, it cannot be alienated without due process of law. Hence, individualism functions to undermine the metaphysical claims of political theology by "naturally" grounding itself upon a political ontology of the human body that held "the body" to represent "nature" in and for the person. However, it also introduced a new question—hitherto unknown to political theology—into political life: that is, once you affirm human beings as discontinuous individuals who "naturally" own their bodies as their property, how do you reaffirm and reanimate their collective coexistence? Or, to turn the question another way, how do you make sense of the collectiveness of a political collective if you suppose that its members naturally bear their individuality within themselves as their most essential attribute?

The possible answers generated to this question were numerous: contract theory, human nature, the economy, society, and culture, among others, all emerged during the late seventeenth and eighteenth centuries as responses to the ontological quandary that possessive individualism introduced. Behind, beneath, or perhaps beyond all these various attempts to reconcile continuity with—and within—individuality, the "human species" appears to offer a natural resolution insofar as it incarnates a vital force that sustains both the production and the reproduction of individuals. In order to appreciate how this reconciliation works, consider for a moment what individualism tries to do. As a political strategy, individualism seeks to isolate people from the life world and reorient their "natural" being inwardly, as if "life itself" were spatially and temporally localized within an epidermal envelope. Individualism proposes a cleavage of the universe *around* "the body" (which is after all what the "environ" of environment signifies). The body hence becomes the proper metonym

for the person only insofar as it bears no other essential relation to anything else—family, kin, clan, tribe, rank, estate, nation, territory, place, God, and so on. Yet, as much as this fiction might make sense logically or strategically as a political reaction to monarchal prerogative, it does not necessarily follow biologically or ecologically, let alone ontologically. All living beings must be both bounded and open, localized and distributed, focused and enmeshed (Varela 1991). Moreover, organisms only exist insofar as they coexist—as, for example, our own coexistence with the commensal bacteria that flourish in our guts and skin reveals. The affirmation of the individual as a simultaneously political and biological unit, then, far from constituting a natural fact, instead betrays an *unnatural* attachment to the historical and cultural assumptions on which it leans.

To put this another way: individualism limns the terrain of the biopolitical by specifying "the body" *in lieu of* the person, that is to say by personifying the body as *the place in which* the human (species) exists. This personification attaches "life itself"—or what we might call the *immanence* of a life (Deleuze 2001)—to "specific life"—species life properly localized within individual bodies capable of reproducing new individuals through their (sexual) relations with other specific bodies. Needless to say, this vital formulation hinges on an implicit doubling of "the human body" as at once an individual and a species body (a doubling which recalls and recasts Kantorowicz's (1957) famous theologico-political icon, "the King's two bodies," in secular terms.) The individual's body thus conceals an intrinsic human duplicity insofar as "it" identifies us, both individually and collectively, as human. *The modern duplicity of the human body is a real paradox*, simultaneously a general singularity and a singular generality. If we regard it as such, we glimpse some of the economic, political, and philosophical investments in our own "specialness" as humans that underwrites belonging to the human species.[11]

Calculating Habits

In the *German Ideology,* Karl Marx and Friedrich Engels (1970, 51) declare that humans "relate to" other living beings, whereas non-human organisms just are. In positing this specifically human relating, Marx extends Hegel's precept that consciousness exists through—and *as*—the negation of nature's immediacy. Instead of drowning in our animality, humans distinguish ourselves from the world in order to relate to it, and we thereby become human. The different forms that such relations take inform the conditions within which human beings live together (with human and non-human others) at

different moments in history. Within the compass of modernity, wage labor constitutes the dominant economic form assumed by the human relations informed by possessive individualism. Before its modernization, feudalism presupposed a continuous metaphysical hierarchy among ranks, estates, and degrees of people that also assumed a material continuity between serfs and the land. As these historical and metaphysical continuities were destabilized, wage labor restabilized them as aggregates of self-owning individuals who can contractually self-alienate themselves in exchange both for remuneration and for political and legal rights. Appearing within this juridico-politico-economic horizon, population becomes a means to count *and to account for* the vital assets that these individuals represent to and for the state. However, population is never merely a matter of counting and thereby accounting. In order for population to take on statistical value, that is, in order for it to orchestrate state power, it must concern itself with the manifold individual changes that occur among the population over time (Foucault 2007, 344–345). Birth rates, death rates, unemployment rates, rates of mortality and morbidity are just that: rates of change through time. They represent singularities quantitatively assimilated into generalities though calculating practices. Moreover, because such rates are themselves comparable as calculations, taken together they make it possible to evoke relative rates of change between two or more populations; hence they gesture toward an even more general population that in turn contains them all.

Population, then, like species, encompasses both production and reproduction. Indeed, population, like species, only produces itself by reproducing itself through individuals through time. This conjunction marks what Foucault (1978, 143–144) describes as "the entry of life into history, that is, the entry of phenomena particular to the life of the human species into the order of knowledge and power, into the sphere of political techniques." Conversely, these political techniques incorporate the desire to make the imperatives of human life calculable—and potentially governable—by making them amenable to economic and biological regulation. They simultaneously render population a vital metonym for species, and specify "the economy" as the natural domain within which our species imperatives must be fulfilled. Leaning on his familiarity with the natural histories of Linnaeus and Buffon, Adam Smith provided the seminal articulation of this politico-economic perspective. Indeed, rather than his famous "invisible hand," Adam's Smith's particular stroke of genius might reside in the invisible sleight of hand through which he cast political economy as that which knits population and species together, thereby envisioning economics as biology by other means.

Classical political economy emerges as a biopolitical domain in part via the assumptions that Smith imports from natural history into his economic thought (Schabas 2003). In the first place, Smith's debts to Linnaeus are manifold, which is not surprising since the Swedish taxonomist explicitly combined natural and economic interests.[12] Indeed, Linnaeus regarded his work in natural history as a vital national resource intended to promote agricultural innovation and development (Koerner 1999; Muller-Wille 2003). In his essay "The Oeconomy of Nature," Linnaeus (1749) affirms this useful and reciprocal link between social policy and natural history, prefiguring Smith's notion of a self-regulating political economy. Linnaeus' assessments of the "oeconomy of nature" then explicitly inspire Smith's (2002) fusion of natural history and moral philosophy in *The Theory of Moral Sentiments*. In Smith's estimation, the oeconomy of nature:

> not only endowed mankind with an appetite for the end which she [nature] proposes, but likewise with an appetite for the means by which alone this end can be brought about, for their own sakes, and independent of their tendency to produce it. Thus self-preservation, and the propagation of the species are the great ends which Nature seems to have proposed in the formation of all animals. Mankind are endowed with a desire of those ends, and an aversion to the contrary; with a love of life, and a dread of dissolution; with a desire of the continuance and perpetuity of the species, and with an aversion to the thoughts of its entire extinction. (Smith 2002, 90)

Smith's text binds up Linnaeus' notion of the economy of nature with Buffon's reproductive criteria for species (with which Smith was also familiar[13]) in order to posit a natural basis for his moral theory. In Smith's estimation, the species imperative acts through individuals, "independent of their [own] tendency," and it is precisely this imperative, divorced from individual agencies but which nevertheless acts "for their own sakes," that constitutes the "oeconomy of nature."

Needless to say, this formulation anticipates Smith's ideas about political economy, published in 1776 as *An Inquiry in the Nature and the Causes of the Wealth of Nations.* However, in this text the market, rather than the oeconomy of Nature, produces the ends conducive of the continuance and perpetuation of the species. Smith (1776, 64) embeds this paradox of "economic nature" in his chapter "Of the Wages of Labor," where he synthesizes his ideas about natural history and political economy in order to explain why, "[t]he produce of labour constitutes the natural recompense or wages of labour." In Smith's exposition, this mode of "natural recompense" leans on an anthropological

narrative that moves from an "original state of things, which precedes both the appropriation of land and the accumulation property, [in which] the whole produce of labor belongs to the laborer," to an articulated division of labor predicated on contracts between "masters" and "workmen." Given its juridico-political investments in such contractual relations, labor becomes subject to, and regulated by, market dynamics that determine the "demand for those who live by their wages." In this telling phrase Smith seeks to explain how and why wages fluctuate by collapsing the distinction between living and wage earning; he assumes that for a substantial portion of the population living becomes tantamount to wage earning, and vice versa. This economic indistinction not only affirms the collapse of "natural recompense" into "wages of labour," but also conversely locates the political basis for national wealth in the living dimension of wage labor—that is, in the *population* of wage laborers considered as a subset of the human species (Smith 1776, 70).

Using the rudimentary statistics available to him, Smith asserts a correlation between changes in population and changes in wages. Moreover, he avers that if the former derive from natural fluctuations, so do the latter, even when the population's "natural" changes reflect the impact of wages on subsistence. Thus, the market dynamics of wage labor, according to Smith (1776, 79–80), reflect the natural patterns of reproduction that we call species. Smith traces the movement from species to population to human reproduction to commodity production, on whose basis *the discourse of* political economy claims that it manifests the oeconomy of nature. However, unlike Linnaeus' oeconomy, the nature of Smith's economy no longer inheres in the "all wise disposition of the Creator in relation to natural things, by which they are fitted to produce general ends and reciprocal uses." Rather the general ends and reciprocal uses produced by wage relations determine the natural reproduction of the human species. In other words, following Adam Smith, political economy weaves together population and species as if they naturally belonged to the same social fabric, a social fabric that we call "the market." Furthermore, with this articulation of population and species, modern political economy "appears"—or rather deems itself—the "natural" locus within which the "human species" necessarily lives. It thereby legitimates itself as nature by other means, an egregious overreaching to be sure, which it then unfortunately realizes (e.g., in climate change).

If Smith adumbrates the theoretical nature of political economy, Thomas Robert Malthus exalts it mathematically when he famously elaborates his "Malthusian" precepts in *Essay on the Principle of Population* (1798). Given his indebtedness to Smith's vision of "multiplication in proportion to the means of subsistence," Malthus obviously leans on Smith (who he credits) for his

conceptual background. However, despite this clear borrowing, Malthus also dramatically increases the return on Smith's theoretical investment when he inflates the scope of Smith's biopolitical conjecture by channeling it through Newton's calculus (Waterman 1998; Cremaschi and Dascal 1996). Whereas Smith's text does not rely on any mathematical techniques to portray political economy, Malthus brings Newton's mathematical invention to bear on Smith's social nature in order to calculate *and thereby constitute* a ratio between human reproduction as a species and human reproduction as a population.[14] He then affirms this ratio as political economy's natural rationality and at the same time introduces mathematical modeling as the *sine qua non* for economic thought (Stengers 2005, 999). Although Newton's calculus entails numerous epistemological and metaphysical subtleties, suffice it to say that Malthus relies on Newton's "fluxions and fluents" to motivate his pessimistic comparison between the rates of increase of food supply and of mouths to feed (Waterman 1998, 582). By invoking the strategies and the authority of Newton's calculus to simultaneously *derive and legitimate* the mathematical bio-logic of political economy, Malthus renders his "principle of population" a biological and economic "fact," one that (even today) motivates political decisions on a planetary scale.[15]

The bio-logical motivation for Malthus' (1798, 3) politico-economy derives from his desire to rebut William Godwin's and the Marquis de Condorcet's incredibly optimistic speculations about human progress. Malthus bases his explicitly "melancholy" refutation on the assumption that the phenomena adumbrated by the concepts "population" and "subsistence" can be compared in terms of two mathematical functions that trace the changes in these phenomena over time. Newton's differential calculus not only underwrites Malthus' reflections on the proportion between these two different rates of change—a proportionality Smith merely asserts as self-evident—but also authorizes Malthus' famous claims about the irreconcilable tension between, or the dilemma of, two competing natural tendencies: the tendency of population to increase geometrically and the tendency of food supply to increase arithmetically. Malthus thus invokes Newton's calculus, which had proved itself capable of encompassing the movements of astronomical bodies, precisely in order to affirm the (supposedly) biological or natural conditions of human existence as inevitable political and economic limits.

When Malthus posits his famous dilemma, he does so by applying the mathematical strategies that Newton developed to calculate instantaneous rates of change. However, whereas Newton use the calculus to plot physical movement (e.g., of planets), Malthus extrapolates Newton's technique to the vital transformations that arise within collectivities of living human beings.

This extrapolation enables Malthus, as it did Newton, to finesse the conceptual distinction between an aggregation of points (i.e., population as data set) and a continuous interval (i.e., population as natural species). Just as Newton was able to use his mathematics to establish an ontological continuity between the infinite points that comprise a line by asserting that the line traces the movement of a point such that the differences between the points are differences that make no difference *and hence make all the difference*, so Malthus ontologically constitutes population as the mathematically discernable traces of individual changes through time. Malthus' calculus renders the infinitesimal transformations of human existence (those infinite differences that make no difference and thus make all the difference) into functions of four variables: food, labor, sex, and death. Their temporal entanglement can be mathematically affirmed as a simple "natural law": following its own tendencies, population will outstrip food supply. Furthermore, in Malthus' case, the application of this natural law enabled him to advance his political and economic claims that, given the human species' relentless biological necessities, providing out of door relief to the poor would "naturally" increase their suffering and immiseration. Indeed, despite the fact that his argument lacked almost any empirical substantiation whatsoever, Malthus' Newtonian prognostication so convinced the Prime Minister that he withdrew the bill to amend the Poor Law, providing the first example of a policy decision based on mathematical modeling.

Today we understand that Malthus's tendentious bio-logic underwrites the enterprise of political economy insofar as it constitutes the risks and vulnerabilities inherent in a living population as calculable and hence predictable (though apparently not preventable). However, while Malthus founds the science of economics by representing human nature and human biology as *tending* toward calculable predictability, this economic strategy itself constitutes, as Bruno Latour and Vincent Lépinay (2010, 63) have recently noticed, an affective or psychological response to fear and anxiety and thus motivates what they call an "entirely psychological passage from uncertainty to probability." (In Freudian terms, this psychological passage is called "fantasy.") To make this passage make sense, Malthus' *Essay* conflates population with species as if this conflation represents a natural fact. It then utilizes the mathematical analysis of population to make claims about the natural conditions within which humans necessarily coexist. Population thus seems to evince the dynamics of species-being as a matter of counting and accounting for human coexistence (both with other humans and with other others) insofar as these relations are "naturally" regulated by economic means. Yet it is important to remember that the "oeconomy of nature" does not describe how nature really

is, but rather affirms one particular version of what Latour (2004, 28–29) calls "nature-in-general" as a strategy for universalizing the assumptions to which it is indebted. In this case, the belonging of human beings to the human species offers a way of dividing up the sensible world that forecloses non-calculating habits of thought which do not assume that population and species mean the same things or in the same ways. Nevertheless, the force of this largely unremarked equivalence continues to elude us, in part because these calculating habits now appear to us as the very nature of who we are.

Coda: Special Tendencies

One of the most vital consequences of Malthus' putatively natural calculus arises when it rebounds into bioscience itself via Charles Darwin's theory of evolution. As historians of evolution frequently remark, Darwin explicitly incorporates Malthus's political and economic bio-logic of population into his theory of evolution. Indeed, Darwin (1859) credits Malthus for inspiring his dynamic synthesis of evolutionary theory, for example, when he declares in *On the Origin of Species by Means of Natural Selection*: "It is the doctrine of Malthus applied with manifold force to the whole animal and vegetable kingdoms." (63)[16] The eminent twentieth-century biologist Ernst Mayr neatly framed Darwin's debt to Malthus in terms of what Mayr (1989) calls Darwin's "population thinking"[17] since Darwin's (1859) conceptual breakthrough supposes that species incorporate, as he writes in *Origin of Species*, "the full effects of many slight variations, accumulated during an almost infinite number of generations" (481). When these slight variations that preponderate through almost infinite generations are thought in terms of population, they appear as what Darwin and Alfred Wallace (1858) characterized, in the title of their famous co-authored essay, as "the tendency of species towards variation." Though Darwin himself was fairly nominalistic about the notion of species,[18] understanding that he could not provide a well-bounded definition since by definition species evolve, nevertheless misgivings about the political implications of his intellectual genealogy abound—including those of Marx and Engels in his day, along with many subsequent scholars.[19]

Whatever we think of Darwin's investment in bourgeois political economy (which was quite material since he lived off his inheritance), we can say that evolution's political bio-logic persists insofar as Darwin conflates species with population. He therefore relies on an unremarked mathematical operation borrowed from Newton by way of Malthus that extrapolates evolving tendencies from aggregates of individuals.[20] In order to conceptualize distinct ten-

dencies toward speciation from infinitesimal organismic variations over time, Darwin leans on Malthus' use of Newton's infinitesimal calculus as a natural logic that resolves change into continuity, thereby incorporating the same at once mathematical and ontological sleight of hand. By relying on Newton's mathematical precept to translate infinitesimal changes into determinant life forms, both Darwin and Malthus elide the manifold biological differences that living beings evince so that these differences appear to make no vital difference despite the fact that the elision of these differences makes all the difference. To put this more succinctly, we might say that both Malthus and Darwin treat population as a "black box" where individuals go in and the human species comes out and where change disappears into the conceptual darkness that lies in between.

So why try to shine some light into this black box? Whether we realize it or not, our political, economic, or military policies are now almost exclusively calculated through population paradigms. More than just evincing an underlying political rationality, we could say that these modes of decision-making actualize a political bio-logic that considers species and population as entangled concepts and in so doing incalculably effects multifarious living beings—human and otherwise. Insofar as states, banks, corporations, and NGOs, for example, make decisions concerning the lives of people and other living beings in terms of populations figured as vital aggregates, they unreflectively materialize the political and economic assumptions that population thinking casts as natural, or even as "our nature." Furthermore, the mono-naturalist and universalizing assumptions underwriting this political rationality unreflectively endorse the Western characterization that "being human" entails "belong to the human species." Thus, they purport to describe the most *natural* way of being human, if not "human nature" itself. However, if we reflect on the population-species conjunction as a historical rather than a natural accomplishment, we begin to apprehend that it only coalesced across the eighteenth and nineteenth centuries in Europe as a politico-economic strategy for dividing and sharing the *human* world—Capitalism for short—that then morphed into a putatively natural way of dividing up and sharing the *life* world. Moreover, as we begin to appreciate that the coin of species-population amalgamates mathematics, natural history, political economy, and liberal political philosophy, we recognize how it legitimates secularized humanness as a viable form of life.

We might say then that political economy provides the bio-logic of capitalism and that the human species makes this bio-logic make sense as the dominant calculus through which we partition and participate in the world. If the sensibility that political economy underwrites enables a fantasmatic shift from

uncertainty to probability, as Latour and Lépinay suggest, it does so by rendering vital processes subject to calculations that mathematically exclude incalculable variation as insignificant or without value. Indeed, this is precisely what Malthus' extension of Newton's calculus to population thinking achieved: Malthus claimed that human tendencies are fully calculable and that this calculability justified specific social policies, in this case eliminating out-of-door relief to the poor. Yet tendencies need not be totalized by their calculations, just as species need not be totalized by their populations.

In its etymological sense, tendency comes from the Latin *tendere*, to stretch; hence, by definition tendencies encompass tensions that trouble fixed boundaries and settled formulas. If species constitute tendencies, as post-Darwinian evolutionary theory tells us, they literally name a tension that they must also contain: that is, they must encompass that which stays the same even as it changes, and then incorporate this sameness-in-difference within themselves as that which is proper to them. The tension between sameness and difference means that species must inevitably be properly improper, which is just to say that they must be self-differing or evolving—a self-difference which species' conflation with population obscures or elides. Yet this tension, this tendency, must endure as the incalculable substrate of both our vitality and our humanness, especially if we are to have any hope of continuing to evolve at all. Thus we might need to appreciate why, rather than investing so much in our sense that "being human" means "belonging to the human species," we could consider instead that we are the human tendencies that we could yet become.

Notes

1. For the distinctions between Linnaeus' and Buffon's theories of species, especially with respect to the natural history of humans, see Philip Sloan. 1995. "The Gaze of Natural History." In *Inventing Human Science: Eighteenth-Century Domains*, edited by Christopher Fox, Roy Porter, and Robert Wokler, 112–151. Berkeley: University of California Press.
2. On Foucault and "police," see Ed Cohen 2009. *A Body Worth Defending: Immunity, Biopolitics and the Apotheosis of the Modern Body*. Durham, NC: Duke University Press, 93–98.
3. For an overview of the debates see the entry on "species" in the *Stanford Encyclopedia of Philosophy*: http://plato.stanford.edu/entries/species/ (Accessed April 2, 2011). For an extended treatment of the positions, see David Stamos. 2003 *The Species Problem: Biological Species, Ontology, and the Metaphysics of Biology*. Lanham, MD: Lexington Books. On humans as a species, see John Dupré. 2002. *Humans and Other Animals*. Oxford: Oxford University Press.

4. See also Sloan, Phillip. 1987 "From Logical Universals to Historical Individuals: Buffon's Idea of Biological Species." In *Histoire du Concept d'Éspece dans les Sciences de la Vie*, edited by Scott Atran, 101–139. Paris: Foundation Singer-Polignac.; Balme, D.M. 1962. "Genos and Eidos in Aristotle's Biology." *The Classical Quarterly*. N.S. 12 (1): 81–98; Grene, Marjorie. 1974. "Is Genus to Species as Matter to Form? Aristotle and Taxonomy." *Synthese* 28 (1): 51–69; and John Mouracade, ed. 2007. *Aristotle on Life*. Kelowna, B.B.: Academic Printing and Publishing.
5. For a recent reflection on the contemporary implications of human species-being, see Michael Dillon and Luis Lobo-Guerrero. 2009. "The Biopolitical Imaginary of Species-being." *Theory, Culture, and Society* 26 (1): 1–23.
6. For my extended elaboration how "the body" becomes a proper metonym for the person, Cohen. 2009. *A Body Worth Defending*.
7. The full title of Linnaeus' text was *Systema naturae sive regna tria natura*: that is, *The System of Nature or the Three Kingdoms of Nature*—meaning mineral, plant, and animal.
8. If we consider that this reproductive valence introduces the possibility for conceiving the individual and population as "related" through production and reproduction, then we understand better why Foucault claims that sexuality exists at and as the interface "the anatomo-politics of the human body" and "the biopolitics of populations," the former focused on "the body as machine" and the latter on "the species body, the body imbued with the mechanics of life and serving as the basis of the biological processes." Foucault, Michel. 1978. *The History of Sexuality: An introduction, Volume I*. Translated by Robert Hurley. New York: Pantheon, 139.
9. This criteria also founds Buffon's critique of Linnaeus' more expansive use of species to include inanimate as well as animate being. The quote continues: "it is clear that this denomination must only extend to animals and plant and it is by an abuse of terms or ideas that the taxonomers [*nomenclateurs*] use it to designate different sorts of minerals" (Comte de Buffon 1753, 386).
10. On Buffon and race: Nicholas Hudson. 1996. "From 'Nation' to 'Race': The Origin of Racial Classification in Eighteenth-Century Thought." *Eighteenth-Century Studies* 29 (3): 247–264; Sloan, Phillip R. 1973. "The Idea of Racial Degeneration in Buffon's *Histoire Naturelle*." *Studies in Eighteenth-Century Culture* 3: 293–321; Curran, Andrew. 2009. "Rethinking Race History: The Role of the Albino in French Enlightenment Life Sciences." *History and Theory* 48 (3): 151–179.
11. Special is the adjectival form of species.
12. Linnaeus developed his famous binomial nomenclature precisely to facilitate his students' ability to discern the plants on which cows, pigs, and sheep feed and thereby to improve animal husbandry (Lisbet Koener. 1999. *Linnaeus: Nature and Nation*. Cambridge: Harvard University Press, 101–104).

13. Smith cites Buffon in his very first publication, "Letter to the Authors of the Edinburgh Review." *The Works of Adam Smith*. Vol. 5 London: T. Cadell and W. Davies, 1811. 567–584. On the letter see: Jeffrey Lomonaco. 2002. "Adam Smith's "Letter to the Authors of the Edinburgh Review." *Journal of the History of Ideas* 63 (4): 659–676.
14. Malthus is a "Newtonian" for whom empirical data (supposedly) provides the ground for theoretical formulation. He critiques Godwin and Condorcet as Cartesians who seek to make the data fit the theory. Waltzer, Arthur. 1987. "Logic and Rhetoric in Malthus's Essay on the Principle of Population, 1798." *The Quarterly Journal of Speech* 73(1): 1–17; Winch, Donald. 1996. "Malthus versus Condorcet Revisited." *The European Journal of the History of Economic Thought* 3 (1): 44–60; and I. B. Cohen. "Newton and the Social Sciences." In Philip Mirowski, ed. *Natural Images in Economic Thought*. Cambridge: Cambridge University Press, 1994.
15. David McNally argues that it was precisely Malthus' Newtonianism that enabled his theory of population to be taken up as "natural law." See McNally, David. 1990. *Political Economy and the Rise of Capitalism: A Reinterpretation*. Berkeley: University of California Press.
16. On Darwin's relation to Malthus, see Young, Robert. 1985. *Darwin's Metaphor: Nature's Place in Victorian Culture*. Cambridge: Cambridge University Press; Vorzimmer, Peter. 1969. "Darwin, Malthus and the theory of natural selection," *Journal of the History of Ideas* 30 (4): 527–542; Herbert, Sandra. 1971. "Darwin, Malthus and selection," *Journal of the History of Biology*, 4 (2): 209–217; Bowler, Peter. 1976. "Malthus, Darwin and the Struggle for Survival." *Journal of the History of Ideas* 37(4): 631–650; Ariew, André. 2007. "Under the Influence of Malthus' Law of Population Growth: Darwin Eschews the Statistical Techniques of Adolphe Quetelet." *Studies of History and Philosophy of Biology and Biomedical Science* 38(1): 1–19.

    In 1839 after reading Malthus, Darwin cites the Essay in the following in his Notebook E:
    'And since the world began, the causes of population & depopulation have been probably as constant as any of the laws of nature with which we are acquainted.'—this applies to one species—I would apply it not only to population & depopulation, but extermination & production of new forms.—their number & correlations. (3; http://darwin-online.org.uk/content/frameset?viewtype=text&itemID=CUL-DAR124.-&keywords=e+notebook&pageseq=1. Accessed April 1, 2016)

17. "Darwin's solution for the multiplication of species and his discovery of a theory of common descent were accompanied by a number of other conceptual shifts. The most important one was his abandonment of essentialism in favor of gradualism and population thinking" (Mayr 1989, 176).
18. See Darwin's (1859) demurral in *On the Origin of Species*:

> "From these remarks it will be seen that I look at the term species, as one arbitrarily given for the sake of convenience to a set of individuals closely resembling each other, and that it does not essentially differ from the term variety, which is given to less distinct and more fluctuating forms. The term variety, again, in comparison with mere individual differences is also applied arbitrarily, and for mere convenience sake." (52)
> Needless to say, as with all things Darwin, there is much controversy about Darwin's position on "species." For a detailed summary of the history of the arguments and an attempt at resolving them, see Stamos, David. 2007. *Darwin and the Nature of Species*. Albany: SUNY Press.

19. For a survey of non-Malthusian theories of evolution, see Todes, Daniel. 1989. *Darwin without Malthus: the Struggle for Existence in Russian Evolutionary Thought*. New York: Oxford University Press; Sapp, Jan. 1997. *Evolution by Association: A History of Symbiosis.* New York: Oxford.
20. As early as 1842 in his "First Pencil Sketch of the Species Theory," Darwin writes the following series of notes to himself:

> But considering the enormous geometrical power of increase in every organism and as every country, in ordinary cases, must be stocked to the full extent, reflection will show that this is the case. Malthus on man—in animals no moral restraint […] the pressure is always ready … a thousand wedges are being forced into the economy of nature. This requires much reflection; study Malthus and calculate rates of increase and remember the resistance—only periodical. … In the course of a thousand generations infinitesimally small differences must invariably tell. (Quote in Young 1985. *Darwin's Metaphors. 41.* For facsimile of the original see http://darwin-online.org.uk/content/frameset?viewtype=image&itemID=CUL-DAR6.1-13&pageseq=1, Accessed April 1, 2016)

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Ten Theses on the Subject of Biology and Politics: Conceptual, Methodological, and Biopolitical Considerations

Samantha Frost

Introduction

Recent findings in the life sciences trace how social and material elements of our environments shape human growth and development. In the fields of endocrinology, developmental systems biology, immunology, epigenetics, and neuroscience, among others, researchers are finding that humans are not simply embedded in their lived environments but that they compose and recompose themselves biologically—at molecular and cellular levels—in response to them (Cole 2014; Keller 2010, 2015; Robinson et al. 2005; Slavich and Cole 2013). Interestingly, the dynamics between embodied self and lived environment that scientists have begun to trace have been anticipated over the past decades in efforts by feminist theorists and social and political theorists to explain the phenomenon of subject formation—the processes through which a human organism comes to be recognized as and to see him- or herself as a specific person within a historically and geographically particular community (Foucault 1982). Although the modes of knowledge production mobilized by life scientists and social and political theorists are distinct, the logics and constituting forces of the movements between environment and self they analyze resonate quite profoundly.

The aim of this chapter is to develop a set of concepts and methodological principles that researchers might draw on as they try to elucidate the processes

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by which the engagement of humans with their social worlds, material environments, and historical cultures results in the formation of a self in subjective, corporeal, as well as biological dimensions. The idea here is not finally to reconcile findings in the life sciences with theories promulgated by critics of social and political life along the lines proposed, for instance, by Edward O. Wilson (1999). Rather, the idea is to forge a conceptual language and rules for analysis that will enable scholars to negotiate productively the commonalities, similarities, and tensions in converging accounts of how the social norms, lived environments, and power relations through which people develop a sense of self are bound up with the processes through which biological organisms compose and recompose themselves over time.

In this chapter, then, I will begin with a sketch of a conceptual map of the points of provocative contact or resonance between life science and political theoretical understandings of the social and material processes through which people develop. This map will situate—conceptually and logically—the subsequent unfolding of ten theses. In providing such a map and set of theses, the chapter will not present a survey of the history of these scientific and theoretical developments or of the burgeoning literatures on this topic—a project far too unwieldy for the task at hand (Meloni 2016; Meloni and Testa 2014; Pickersgill et al. 2013). Rather, the chapter will provide a conceptual—and in many ways ontological—foundation and related guidelines for studying humans conceived as biocultural creatures.

The Map

The theoretical insights that anticipate and help us make sense of the scientific findings about biocultural humans are many; here, I draw brief attention, in particular, to two. The first is the insight, articulated well by Judith Butler, that the subject does not pre-exist the linguistic and cultural forms through which it represents and symbolizes itself (1992). When social and political theorists make such a claim, they mean that an individual does not become a person and then, subsequently, use language or enjoy culture. Rather, individuals become persons only through using some form of language and being acculturated and acknowledged in some form of community with symbolic means of self-representation. This first insight tells us, then, that symbolic and representational forms, as manifest through language, cultural practices, institutional imperatives, economic, social, and political activities, and forms of self-understanding, together shape behavior, identity, and desire, making us who we are.

The second insight to which I draw attention is the relatively recent move by a range of theorists to refuse the posture of human exceptionalism (Agamben 2003; Braidotti 2013; Grosz 2011; Wolfe 2010). This refusal takes aim at the notion that humans are possessed of some quality—rational, willful, linguistic, moral, cultural—that exempts them from the forms of conditioning, dependency, and vulnerability that attend living life as a creature. Through analyzing and refusing what Agamben (2003) calls "the anthropological machine" or the desperate (and ultimately futile) effort definitively to distinguish humans from animals and from the material world of their support, theorists call into question moral, political, and economic ideas that derive human prerogative or privilege from humans' putatively exceptional status. The corollary of this refusal of human exceptionalism is an insistence on foregrounding humans' animality, embodiedness, fleshiness, and materiality, their embeddedness in very particular social and material environments, as well as their dependence upon various technologies (Alaimo 2010; Chen 2012; Grosz 2005, 2011; Haraway 2012; Hird 2009; Ingold 2000; Stiegler 1998). This posture of refusal, combined with the insight that subjects are constituted, paves the way for political theorists and cultural analysts to conceptualize subject formation not just in terms of language, culture, and politics but also in terms of embodiedness, creatureliness, and the material and technological elements of the environment that make living possible.

As theorists have elaborated these theoretical insights together, they have crept closer and closer to living flesh, seeing social norms, political imperatives, institutional organization, and symbolic forms of self-understanding infuse the embodied subject to an extent that theorists talk of the embodiment of norms, the materialization of power, or the corporealization of culture. These kinds of analyses show us, for example, that gender and race are not merely socially informed modes of understanding that can be self-consciously transformed through better information, but rather are solicitous and coercive modes of social address and cultural and political practice that shape social expectation, perception, encounter, and relation so thoroughly that "gender" and "race" as socially and politically significant markers appear to originate in the body as natural phenomena (Alcoff 2005; Butler 1992; Lee 2014; Weheliye 2014). Yet, as Birke (2000), Roberts (2007), Frost (2014), and Wilson (2015), among others, have noted, in spite of manifold and theoretically rich efforts to trace how the forces constituting subjects make us inhabit, move in, and experience our bodies in particular ways, many social and political theorists have been wary of exploring the extent to which the biological processes and biological matter of the body might be shaped, formed, or deformed by the vagaries of social and political life.

This wariness about including biological processes in accounts of subject formation has many causes, not least of which is the ongoing effort of social and political theorists to challenge the use of biology as an index of social and political status—and the corollary use of sex, race, and ability as the hinge upon which political dignity and freedom rest. Elizabeth Wilson (2015) argues that as theorists have pushed against a figuration of the biological body as a phenomenon that could determine the contours of social and political life, they have produced as a conceptual accretion of their refusal a figuration of biology as "a passive substrate…that culture animates" (31). Which is to say that, constrained by a sense of political danger, many social and political theorists have cleaved to the terms of social constructivism to an extent that, with the exception of the administration of pharmaceuticals or surgical intervention, they have been reluctant to entertain the possibility that biology conceived as the life processes of an organism might be shaped by or be a factor in social and political phenomena.

It is only quite recently that social and political theorists have begun to explore whether the concepts of "embodiment," "incorporation," and "materialization" that are intended to capture the processes through which bodies are shaped by social and cultural factors might also have specifically biological dimensions (Fausto-Sterling 2004, 2005, 2008, 2012; Grosz 2005, 2011; Lee 2014; Saldanha 2009; Warin et al. 2015; Wilson 2004, 2015). In this emerging genre of scholarship, biology is conceived not as a stable something that subsists doggedly beneath its varied social and political guises but rather as a collection of dynamic processes that are sensitive and responsive to the series of habitats through which humans sustain themselves—and thus as a contributing element in the formation of humans as persons.

Intriguingly, the effort by such theorists to consider biological processes in non-reductive, creatively variable terms is being met by developments in various fields of the life sciences—especially epigenetics as it is pursued through many disciplines (Cacioppo et al. 2013; Cole 2014). In a review of contemporary studies, George Slavich and Stephen Cole (2013) note a trend among scientific researchers in which the "deeply engrained…perception of biological stability and impermeability" is being challenged (331). They recount that according to findings in fields such as neuroscience, immunology, and endocrinology, the molecular and cellular scale composition and work of the body is increasingly seen as a function of "the physical and social environments we inhabit," with "people's subjective perception of their social environment" having more transformative effect than "the 'objective' features" (331). Evelyn Fox Keller (2015) similarly explains that the genome is increasingly recast as an "exquisitely sensitive and reactive system" (10) that responds to "the con-

stantly changing signals it receives from its environment" (25). So, as scientists explore how social and material factors shape the composition and recomposition of biological bodies, they are abandoning the notion that biological bodies are relatively closed off from the world with their inner workings impervious to and protected from the environment (Landecker and Panofsky 2013; Lock 2013; Meloni 2014; Niewohner 2011; Rose 2013). Instead, genes, proteins, cells, and organisms are viewed as open to the environment, as constantly changing in response to the environment (Jablonka and Lamb 2005; Lewontin 2002; Rabinow and Caduff 2006; Robinson et al. 2005; Slavich and Cole 2013). And stupendously, the environment here is construed not simply as chemical or material but also as social, representational, symbolic, and imagined, including such phenomena as family dynamics, workplace inequalities, perceptions of social inequality, anticipated loss of a relationship, and the daily micro and macro politics of living in a racialized polity (McEwen 2012; Muscatell et al. 2016; Robinson et al. 2005; Romens et al. 2015; Rose 2013; Slavich 2016; Slavich and Cole 2013; Thayer and Kuzawa 2011). I cannot emphasize this last point enough, since it is here that findings in the life sciences are beginning conceptually to meet insights in social and political theory: through tracing the particulars of how felt psychosocial experiences evoke various biochemical shifts that affect gene transcription and reverse transcription—and thereby the making of the proteins and such that enable our bodies to function—scientists are beginning to assay the ways that experiences of social interaction, and the anticipation and imagination of social interaction, have a constituting effect upon the biological body.

Before going any further, I want to clarify that the openness of bodies to their environments identified by scientists is not just an expansion of the factors to which bodies *respond*, that is, an increase in the number of factors that provoke transformations in the biological *functioning* of bodies. More, as Guthman and Mansfield (2013) explain, this openness to the environment is one through which bodies are "active in [their] own remaking"—an openness through which environmental factors provoke transformations in the *composition and recomposition* of bodies, in the ways they are *produced and reproduced* on a daily basis as well as across life times and generations (497). I emphasize this point because, in some areas of the social sciences, it is fashionable to try to capture the relation between environment and body through invoking the notion of *interaction*, as if human subjects are a product of a kind of push-and-pull encounter between biological and cultural factors. Under such a rubric, the aim of studying biology along with culture is to sort out which factors are biological, which cultural, and how they come to some productive

balance (e.g. Alford et al. 2005; Kandler et al. 2012; and contra Beckwith and Morris 2008; Charney 2008, 2012; Charney and English 2012).

There are a couple of problems with this interactionist framework to draw out here. The first is that the idea of interaction presumes a coherence and integrity to the living subject that it brings to an encounter with a habitat, as if the subject were not ensconced in a habitat all along (Keller 2010; Latour 2004; Oyama 2000). That is, it presumes a subject effectively unencumbered by a habitat who, with a fixed genome and fully grown—with a settled "constitution" as Fowler and Schrieber (2008) might put it—stumbles into an environment to which it proceeds to respond. The second is that, as noted above, in presuming such an unencumbered extra-environmental subject, its encounter with an environment is conceived as provoking a response that changes how the body *works*, but has no effect on how or with what the body is *made* (Guthman and Mansfield 2013).

The problem with the conceptual framework of interaction is similar to the problem identified in cultural and political theory of positing the existence and features of a subject anterior to its action in a field or context. In working around this problem, theorists point out that social ideas, cultural norms, and disciplinary imperatives are not just *representational* (i.e. painting a picture of a thing that is *there*), but are also *constitutive* (i.e. actively involved in the *formation* of the subject through inciting and delimiting the identifications, attentions, and desires that make the subject intelligible to and able to navigate in its social and political situation).

We have to do a similar correction when critically engaging the interactionist framework. As Lappé and Landecker (2015) explain, "time and context have come (in)to the genome" such that the genome is now conceived as having a varied temporal horizon or a "life span" within which changes in environmental provocations are registered at the level of chromatin (152). These temporally and contextually sensitive dimensions of the genome mean that biological processes do not *pre-exist* the environments in which they proceed and persist but are *constituted through* responsiveness to those environments. Thus, Landecker (2016) says, it is not simply the case that the body responds to the environment but rather that the environment provokes the making and remaking of "the responsive body" (95). If we keep this correction in mind, we can see that, in contradistinction to the interactionist framework, biology is not a substance antecedent to environment and culture whose existential priority is the limiting condition for cultural and environmental effects. Rather, cultural and environmental forces and phenomena together constitute the enabling and provoking conditions for biological bodies to compose,

decompose, and recompose themselves in ways that reflect and anticipate those conditions.

What is remarkable about this moment in social and political theory and the life sciences is that in both genres of research, scholars endeavor to articulate and trace the processes and mechanisms by which social and material environments shape our development and growth as human persons, as acculturated subjects. Findings produced in the contemporary life sciences could give social and political theorists the means to explore how ideas, culture, and the vagaries of material environments shape living matter; how disciplinary norms, cultural imperatives, and sinuous forms of power shape biological processes at the same time as they constitute subjects. And the work that theorists and cultural critics have been doing for the past decades giving spatial and temporal depth and texture to the experiences of power, culture, and identity could serve as a resource for scientists as they endeavor to trace how, as McEwen (2012) puts it, the "social gets under the skin." In other words, this moment represents the possibility for informative, productive intellectual exchange across research domains where heretofore there has not been much.

One of the things that hobbles such admittedly difficult interchange is the lack of a common conceptual vocabulary and shared methodological principles for guiding research across zones of familiarity (Callard and Fitzgerald 2015; Pickersgill et al. 2013). Providing such a conceptual vocabulary and sketching some such principles is the aim of the remainder of this chapter. And again, the point of this project is not to facilitate a final reconciliation of the sciences, social sciences, and humanities but rather to provide something of a path, a way in, for researchers interested in developing more expansive accounts of human development and subject formation.

To facilitate the use of this chapter as a waypoint on an interdisciplinary trip, I will use a "thesis" format rather than presenting a continuous narrative argument. For those looking for a fuller, more textured experience, many of the points articulated here appear in long-winded form in my recent book (Frost 2016). What I do here is refine and distill from that project discrete claims or theses that can serve as discrete points of reference even as, at the same time, each is a moment in the unfolding of the logic of a larger idea. Being schematic in this way enables me to be concise in laying out an idea and then to explain the theoretical and political significance of the claims along with the logic that binds those points into a coherent conceptual map for investigating humans in their world. In fact, it is the logic of the concepts that underlies many of the prescriptive and proscriptive claims that I make along the way.

As a schema, the theses start minutely and somewhat abstractly, and then they build step by step a concept of humans that demands different, and differently detailed, figures of movement and interchange between body and environment. Each thesis is substantively distinct; at the same time, each is logically linked to the ones that precede and follow. I will elaborate the thinking that undergirds each thesis as I go. Note that theses 1 through 7 make substantive (one might want to say ontological) claims, building a theoretical picture of humans as biocultural creatures. Theses 8 through 10 address the possibilities and cautions for research presented within a framework in which humans are conceived as biocultural creatures. The earlier theses have conceptual and methodological implications, the later, methodological and political.

The Ten Theses

Thesis 1 *All living organisms, including humans, are porous*. This thesis captures the openness of living creatures to the material and social environments that culture them and enable them to grow. The porosity, here, is meant in both the metaphorical sense in which human subjects are constituted in and through linguistic and cultural forms, and in the literal sense that living organisms, including human subjects, are constituted and compose themselves with and through their engagement with their habitats (Alaimo 2010; Fausto-Sterling 2012; Guthman and Mansfield 2013, Hoffmeyer 2008; Landecker 2011; Lock 2013; Niewohner 2011). What is rejected in the stipulation of this thesis are the suppositions, mentioned above, that the bodies of living organisms are in some aspect closed to their environments, that living organisms are distinct from their habitats and thereby merely interact with them, and that human subjects precede and thus merely enter into and move in the field of their action.

At the center of this thesis is the commonplace scientific insight that the membranes of each and every cell in a living creature are permeable in such a manner that there is an influx and efflux, a prolific traffic, of biochemicals into and out of them at every moment. This traffic is more than a mere passive diffusion of biochemicals. It is provoked and made possible by the myriad proteins and biomolecules made by organisms in response to stability or change in their social and material environments as well as to their perception or imagination of stability or change in their social and material environments. There is a continuous movement of substances across the porous boundaries of cells, across the porous boundaries of bodies—and it is this

movement and traffic that underpins the processes of living and of subject formation.

Thesis 2 *What distinguishes the inside of a porous human creature from the outside that is its social and material habitat is the specific concentration and concatenation of activities and processes composing and decomposing it.* This thesis answers the vexing possibility that the porosity of living organisms—and all the traffic that is implied by cell membrane permeability—makes it impossible to specify a distinction between the inside and the outside of a cell or a body. That is, it addresses the concern that porosity—whether meant metaphorically or literally—results in a kind of environmental reductionism. It is indeed the case that, because there is a constant traffic of biochemicals across cell membranes, we cannot say that the distinction between the inside and the outside of a cell, between the inside and the outside of a body, is a substantive one, a matter of substance. Evan Thompson (2007) marks this insight when he observes that "because its material composition is constantly renewed," the identity of an organism "cannot be based on the constancy of matter" (150). But, clearly, cell membranes are boundaries, and they serve an important boundary function, which is to say that cell membrane porosity is not equivalent to a complete lack of boundary. The question is: what is the boundary function of porous cell membranes?

Jesper Hoffmeyer (2008) notes that the traffic of biochemicals back and forth across porous cell membranes creates "a basic asymmetry between an inside and an outside, making the membrane a potential interface structure" (34). These asymmetries or cross-membrane differentials in biochemical concentrations and electrochemical charges provoke and make possible the protein-rebuilding, chemical transforming, moving-stuff-around activities inside cells that constitute the manifold processes of living. Or to put the point differently, the cellular activities that constitute living are impossible without the precise changes in biochemical concentration and electrochemical charges made possible by cross-membrane traffic. Because of the dependence of cellular activities upon cross-membrane traffic, we can say that rather than being a substantive distinction, the distinction between the inside and the outside of a cell, between the inside and outside of a body, should be seen as one characterized by the specific forms of activity made possible by the permeable boundary. In construing the distinction between the inside and the outside in terms of activity, we can retain a claim about the ontological singularity of each organism even as we acknowledge that each is likely symbiotic with and embedded among many others and all manner of constituent forces and elements.

Thesis 3 *A living organism is, at any one moment, a temporally particular configuration of processes of composing and decomposing*. This thesis elaborates the idea that the distinction between the inside and the outside of a living organism is a distinction in activity. When biochemicals flow into and out of cells across permeable cell membranes, their changing concentrations provoke a myriad of cellular activities. These cellular activities build proteins and other biomolecules that make possible an increase or decrease in that cross-membrane flow, a change that in turn provokes more cellular activities. Cells are precisely responsive to the cross-membrane traffic of biochemicals, and their responses take the form of creating the conditions for further traffic and response—composing, decomposing, and recomposing molecules that together enhance or diminish further traffic of biochemicals into and out of cells. Because the processes of composing and decomposing provoked by cross-membrane traffic are the condition for further processes of composing and decomposing—that are the condition of further processes of composing and decomposing—we should avoid thinking about living bodies as staid matter, as stolid, static, and given. For similar reasons, we should not think about what crosses cell membranes or what is generated in response as merely a deposit, a sedimentation, or an imprint. The responses to the cross-membrane traffic of biomolecules are as much activities of decomposing and recomposing as they are activities of composing.

This thesis holds before us the notion that what we refer to as the biological body is not pure or fixed but rather is constantly building and rebuilding itself. The constant cross-membrane traffic and related cellular activity mean that the form that living human creatures live and experience as ourselves in any given moment is one instantiation of the processes of composing, decomposing, and recomposing that are continuously under way.

Thesis 4 *All organisms, including humans, are biocultural*. This thesis blends the first three to provide a figuration of humans that captures how bodies compose and recompose themselves in response to provocations of the traffic of biochemicals across cell membranes. The amalgam term "biocultural" is used here to evoke the insight, as articulated by Elizabeth Wilson, that "All worlds are alloyed; no object is purebred" (2015: 29). In other words, it is used to reference the insight that there is no aspect of a living organism that is not cultured, that persists in its activities of composition and decomposition of its own accord rather than through its interrelation, absorption, and transformation with other substances, organisms, and creatures (Fausto-Sterling 2012; Haraway 2012; Jablonka and Lamb 2005; Lewontin 2002; Lock 2013; Niewohner 2011).

Such bio-culturing means that bones, cells, DNA, genes—or whatever putatively "really on the inside" bio-things we can imagine—exist and persist only through the processes of composing and decomposing made possible by the traffic of stuff into and out of cells, across the permeable boundaries of the body. In other words, the "bio-" of human organisms exists and persists only because stuff on the outside, in the environment, traverses to the inside—and back. Such bio-culturing also means that the perception of and identification with norms in historically and culturally specific places, their internalization and experience as a sense of self, and experiences of relating and working in accordance with those norms as they manifest in the social and material organization of life are in the turgid mix of processes through which living human organisms compose and recompose themselves as they live and grow.

We can list four general groups of phenomena that are integral to and are the conditions for the composing and decomposing of living human subjects:

(a) Matter from the outside of the body enters the body, and biochemicals inside exit, and both directional movements support or transform the activities of composing and decomposing. We can think here of chemicals, minerals, microbes, gases, nutrients, and toxins (Guthman and Mansfield 2015; Landecker 2011).
(b) The shifting organization and disposition of matter and energy, such as light, sound, space, heat, vibration, gravity, and radioactivity, enters into and impinges upon the modes, speeds, and manners of activity by which organisms compose, decompose and recompose (Faber Taylor and Kuo 2006; Fitzgerald et al. 2016).
(c) Humans' perception of, experience in, and active engagement with their social and material worlds generate intellectual, emotional, psychological, and behavioral responses that involve, in part, the circulation of hormones, steroids, neurochemicals, and other biochemicals that shape and reshape cellular activities of composing and decomposing (Cole 2009; Fitzgerald et al. 2016; Kuzawa and Sweet 2009; McEwen 2012; Mendenhall et al. n.d.; Muscatell et al. 2016; Romens et al. 2015; Saldanha 2009; Thayer and Kuzawa 2015; Wilson 2011). We can think here of social interactions, labor activity, cultural practices, eating patterns, hygienic habits, exercise regimes, institutional involvements, military service, arts training, transit and travel, access to health care, exposure to violence, and so on.
(d) Human's anticipatory, imaginative, and "inner-worldly" engagements with their social and material worlds generate intellectual, emotional, and

behavioral responses that involve, in part, the circulation of hormones, steroids, neurochemicals, and other biochemicals that shape and reshape cellular activities of composing and decomposing (Davis and Morris 2007; McEwen 2012; Muscatell et al. 2016; Slavich and Cole 2013). We can think here of expectations about upcoming events, insights into inequality, affective suasion, transitory identifications, memory, guilt, reading, aesthetic inspiration, and other things that are a mix of this group and the one prior.

The point of this thesis is that matter, energy, sensuous and social perception, and varieties of memory and imagination all together make possible, provoke, redirect, and delimit a living human subject's processes of composing and decomposing. Because these factors coincide in variously productive, disruptive, and intensifying ways to condition and make possible a living human subject's processes of composing and decomposing, we must think of humans as not just biological and not just cultural but instead as biocultural.

Thesis 5 *The habitats that culture living organisms, including humans, are biocultural.* This thesis is an extension and logical corollary of the prior thesis. The environment in which humans live and come into being as subjects is not merely the background for action, nor only an object of action, but also, ineluctably, a compositional factor in human subjects' composing, decomposing, and recomposing activities. The sense in which I am using the term "habitat," then, draws on the notion of "milieu" developed by Jakob von Uexkull (2010) and Georges Canguilhem (2008) according to whom a milieu is not merely an object-filled space in which a creature moves but a meaningful field constitutive of life and sense of self for the organisms who grow and live within it.

Social and political theorists tend to get half of the point of this thesis when we think in terms of the ways that aesthetics, cultural practices, norms, and institutions shape modes of identity and subject formation. And life scientists tend to get another half when thinking in terms of the ways that nutrients and toxins shape development and growth within lifetimes and across generations. But we have just seen that the factors that condition and make possible the processes of composing and decomposing are mineral, chemical, energetic, spatial, organic, microbial, social, perceptual, normative, aesthetic, and imaginative. Since each and every one of these factors together—synergistically and discordantly—make possible, redirect, and recalibrate a living human subject's processes of composing and decomposing, the habitats that culture humans qua biocultural creatures are best conceived not as just one aspect (social/symbolic/representational) nor as just one kind (material/chemical/

microbial). All of them co-occur as the biocultural habitats that culture humans.

Thesis 6 *The responses of biocultural creatures to bio-culturing are noncontemporaneous with their current habitats.* This thesis points to a time lag between environmental provocations and the activities of responsive composing and recomposing. This time lag trips up any easy turn to behaviorism or environmental reductionism in thinking about how humans as biocultural creatures live in and engage with their worlds. As noted above, a living body responds to the constant inflow and outflow of matter, energy, and percepts by adjusting the forms and the frequencies of its activities of composing and decomposing. When a living body responds to its habitat by adjusting its activities of composing and recomposing, those adjustments "set the template for the future" (Landecker 2016: 95) in the sense that they prepare that living body to respond well to similar future habitats. A body's adjustment of the activities of composing and recomposing is anticipatory (Meloni and Testa 2014), a carrying forward of current responses in preparation for future responses (Frost 2014, 2016; Oyama 2000). These adjustments are variably durable, having short-term, medium-term, and long-term effects upon the activities of composing and decomposing in individual lifetimes as well as in the composing and decomposing activities of subsequent generations (Champagne 2011; Guerrero-Bosgana and Skinner 2009; West-Eberhard 2005). Because responsive adjustments endure in these ways, they affect subsequent responses of a living body to subsequent inflows and outflows of matter, energy, and percepts. These anticipatory adjustments mean that, at any point in time, the array of a living body's possible responses to its immediate habitat is not wholly contemporaneous with that habitat. A body's responses to inflows and outflows of matter, energy, and percepts are the material and energetic trace of that body's or of previous generations of bodies' past responses to past habitats. Because any future habitat inevitably is different from in the sense that it does not coincide exactly with the one that provoked the preparatory responsiveness, a creature's biology is noncontemporaneous with its habitat.

Thesis 7 *In the noncontemporaneity of organisms vis-à-vis their habitats lies the distinction of living organisms, including human subjects, from the habitats that culture them.* This thesis refines the second thesis concerning the distinction between the inside and the outside of the body and reinforces the rejection of behaviorism and environmental reductionism seen in the sixth thesis. By adding the temporality of the processes of living to the account of the ontological singularity of each organism, this thesis heads off the concern that the selective porosity of living organisms and their responsiveness to their habitats entail that living creatures be conceived as *merely* responses to habi-

tats, smears emerging from the background environment. The noncontemporaneity of a living body's responsiveness to its habitat means that a living organism is not reducible to its current habitat. Because responsive adjustments to the activities of composing and decomposing are carried forward through time to shape subsequent responses to subsequent habitats, a living organism's capacity to respond to a contemporary habitat is an effect of its past responses to past habitats. Meloni and Testa (2014) explain this noncontemporaneity in remarking that the body of a biocultural creature "is at once inhabited by the traces of its past and seeded with traces of its future" (15). From within the perspective that construes organisms as biocultural, then, the fleshy "itness" of living bodies—what we experience as the physical form and substance of the embodied self—is a product of that anticipatory carrying forward of past responses. Which is to say that the existence of living bodies as discrete ontological phenomena is an effect of their noncontemporaneity with their habitats.

Thesis 8 *Biocultural habitats, and the biocultural creatures cultured in them, are multiscalar, both temporally and spatially.* This thesis highlights the idea that the conditions for the activities of composing and decomposing within biocultural creatures occur at the fine molecular and cellular scale all the way up through the macro spatial scales of global political economy and global climate change, at temporal scales reaching from fractions of a second through lifetimes, generations, and eons (Oyama et al. 2001; Fujimura 2005). I pose this as one of the theses because the deployment of restrictive temporal and spatial scales in research on humans as biocultural creatures can have a distorting effect on findings. As Shostak and Moinester (2015) observe, the huge spatial and temporal variability in the way that researchers conceptualize the environment generates "'regimes of perceptibility' in which particular aspects of the environment become more or less visible" (195). So, for example, Landecker (2011) argues that "the experimental formalization of food" in epigenetics research can lead to food "stand[ing] in for the environment in the dyad of 'gene-environment interactions'" (168). Similarly, Darling et al. (2016) contend that because "biospecimen collection" is seen as "a marker of robust science," researchers often use the body as a "surrogate" for the environment (55) with the effect that they "reduce social processes to bodies" (57). In both cases, a narrow focus on a specific spatial scale obscures from analytic view the broader social and political milieux in which food is produced and rendered available or in which psychological and biological stressors are generated and experienced.

Below, I outline three interrelated problems already anticipated and observed in some of the related literature when the temporal and spatial mul-

tiscalarity is ignored, when biocultural creatures and biocultural habitats are construed very narrowly and within a limited time frame.

One problem is a kind of environmental determinism in which the spatial and temporal multiscalarity of biocultural habitats is narrowed sharply with the effect that members of particular neighborhood or section of a city might be judged as producing and reproducing, by themselves, malignant social relations, persistent poverty, endemic violence, or toxicological health crises (Duster 2006a; Mansfield 2012; Rose and Abi-Rached 2013). In focusing on how patterns of behavior, illness, or trauma "become ingrained within a specific population"—geographically or demographically defined—researchers ignore myriad spatial and temporal scales of social and material environments, generalizing claims across rich and variegated communities and obscuring the historical and structural forces that constitute the community as such (Meloni and Testa 2014: 17). Some of the scales rendered imperceptible in such monoscalar research approaches can be personal and interpersonal, such as intergenerational family and kinship networks, community organizations, policing practices, educational institutions, population migration, and participation in popular and sub-cultures. Others scales can be structural, impersonal, and political, such as patterns of commercial and residential zoning, labor market transformations, national and governmental ideologies, wars, and so forth. The point here is that at all of these scales together, biocultural habitats create the conditions for diverse, cross-textured forms of subjectivity at the same time that they contribute to regionally and locally persistent patterns of poverty or violence. To ignore the temporal and spatial multiscalarity of biocultural habitats is to risk homogenizing a population and misconstruing social and political phenomena, often in ways that perpetuate ideologically saturated assumptions about class, race, ethnicity, gender, sexuality, and national origin (Duster 2006a; Mansfield 2012).

Another problem with ignoring the temporal and spatial multiscalarity of biocultural creatures and biocultural habitats is the mobilization of a neoliberal conception of the individual according to which individuals are held to be responsible for anticipating and ameliorating the health and developmental effects of toxins or poverty or stress by managing their homes or workspaces or illnesses by themselves (Darling et al. 2016; Mansfield 2012; Meloni and Testa 2014; Rose 2007). To focus on the individual without considering the many scales of the social and material environments that shape their lives is to individualize and moralize problems like pollution, economic transformation, and political violence that are properly conceived as collective problems demanding collective, political solutions (Brown 2005). To proceed by granting the full spatial breadth and temporal depth of biocultural habitats

and the humans cultured in them is to put oneself in a position to discern patterns and trends in subject formation, behavior, culture, environment, and political economy—and possibilities for transformation—that otherwise might be missed or overlooked.

And a third problem that follows from ignoring the temporal and spatial multiscalarity of biocultural creatures and biocultural habitats is the figuration of the maternal body—or the uterus—as the environment in which babies grow (Daniels 1997, 2006; Martin 1991; Richardson 2015; Richardson et al. 2014). This spatial and temporal narrowing ignores both men's and women's exposures to reproductive, developmental, and teratogenic toxins over their lifetimes. It also obscures the intimate, social, political, and economic contexts that shape how women live when they are pregnant or raising children. Such a spatial and temporal narrowing renders women singularly responsible for fetal and child health, often in ways that are clinically incomplete as well as morally, socially, and legally punitive.

In attending to the spatial and temporal multiscalarity of biocultural creatures and biocultural habitats, researchers are better prepared to detect and account for these problematic kinds of scale slippage. Rachel Lee (2014) argues that if we can hold before our analytic attention these combined or transecting scales, we will be able to discern those instances in which research that includes biocultural factors might rely on the "affective, psychic importance" of extant racial, gender, and sexual categories and their "materialization in legal, clinical, commercial and civil social contexts" (54). We will also, she claims, be more readily alert to the creation of "micro-scale risk factors as new markers of difference" (57). Indeed, Lee speculates that as the notion that humans are biocultural creatures diffuses through different research and policy fields, it is quite possible that "race—as a synecdoche for exploited, expendable bodies and bodily parts/habits—must make room for finer articulations of how disabled, diseased, or virally positive, impoverished, imprisoned, or otherwise debilitated classes are constituted as the new 'aliens'" (57).

In pointing to the need to attend to the multiple spatial and temporal scales at which bio-culturing occurs, this thesis nevertheless does not demand that everything and all time frames should be put in the front and center of analysis—a paralyzing and likely impossible task. As a range of scholars have suggested, distinctions and priorities must be made so as to be able to engage in study, but the parsing of those lines and the circumscription of analysis should be carefully considered, explained, and revised if they delimit rather than facilitate understanding (Barad 2007; Connolly 2013; Latour 2004, 2013; Oyama et al. 2001).

Thesis 9 *Research on humans conceived as biocultural creatures must take into consideration, if not into full account, the combined material, spatial, social, and representational dimensions of the processes of bio-culturing.* This thesis is an insistent reminder of what it means to say that humans are biocultural creatures. According to the prior theses, living human subjects are composed and recomposed, formed and reformed, through the material, chemical, nutritive, toxic, gaseous, spatial, and microbial constituents of living processes *as well as* the desires, norms, ideas, principles, identifications, disciplines, and forms of resistance that attend collective social and political life. To study subject formation or the development of persons, then, we need to account for all the biocultural constituents formative of living human subjects and to trace the characteristics of those biocultural formations.

So, for instance, to study the embodiment of norms or the circulation of affects might require going further in one's analysis than elucidating the ways that social norms, ideological expectations, political upsets, and institutional inequalities give a dispositional tenor to the experience of being a subject. It might also require that one explore how affects' varied durability and mutability in social and political subjects reflect and provoke hormonal, steroidal, neurochemical, immune, and other transformations that induce a subject's body to compose and recompose in ways that are experienced as habitual anger, periodic stress, a spate of irritability, or a spell of what José Muñoz (2006) called "feeling brown." Alternatively, as Elizabeth Wilson (2011) has argued, the study of depression might include not only biochemical imbalances, pharmaceutical prescriptions, dietary patterns, and personal trauma but also social and familial bonds, the experience of health, employment opportunities, institutional inequalities, displacement, cultural norms, political and economic shifts, and cultural or collective trauma.

This thesis, then, pushes researchers to consider the fullness and multidimensionality of biocultural creatureliness and biocultural habitats, even in projects that have a primary orientation in cultural critique or scientific investigation.

Thesis 10 *The ontological distinctness and historical singularity of each human conceived as a biocultural creature can only be understood through considering how the social and political differentiation of people into groups creates patterns in the biocultural habitats in which each individual grows and lives.* This thesis is a response to the possibility that the idea that humans are biocultural creatures will be taken as license to champion the irreducible biological singularity of each individual. Within such a scenario, the fact that biological processes track the singular history of each living individual's series of habitats is taken to mean that demographic group categories, like race and ethnicity, can be tabled

as analytically irrelevant (Hibbing 2013). However, as Rajagopalan and Fujimura (2012) point out, while the prospect that each person's epigenomic health profile is "unique" can be "potentially democratizing" in medical fields that historically have apportioned diagnosis and care according to broad political racial categories, research that consequently ignores socially and politically potent demographic categories does not take adequate account of the fact that "differences in disease incidence among different race and ethnic groups" correlate "with socioeconomic disparities that exist between different race and ethnic groups in the U.S." (661, 659). To track how social and political life shapes biological processes is to have to conceive of race, for instance, as not merely a way of looking at or telling stories about particular people (a form of representation) but also as a patterned set of political, cultural, and economic institutions and practices that differentiate groups and structure the places and the means by which people live their lives (Haywood 2013; Weheliye 2014). Because social and material worlds are constituent conditions for the living persistence of biocultural creatures, and because those social and material worlds are organized in accordance with complex political formations like race, class, and gender, the biological processes of composing, decomposing, and recomposing are ineluctably, if variably, constituted through the experience of living as members of historically and geographically particular groups (Duster 2006b; Fujimura et al. 2014; Hancock 2013; Saldanha 2009).

This dimension of the tenth thesis may seem tricky to navigate because thinking about demographic groups in conjunction with biology is fraught with awareness of various political histories of racial biologism and eugenics (Esposito 2008, 2012; Fujimura and Rajagopalan 2011; Mbembe 2003; Meloni 2016; Meloni et al. 2016; Montoya 2007, 2011; Roberts 2011; Schiebinger 1993). However, in consonance with some of the theses outlined here, there are a number of scholars venturing to recalibrate the temporal and spatial frameworks within which race is imagined and analyzed. Such temporal and spatial recalibrations allow us to account for the constituted character of race as well as for the ways that it is experienced phenomenologically and lived bioculturally. For such scholars, race emerges as a lived reality through the social and geopolitical relations and institutions that are organized in accordance with symbolic and representational modes of differentiating groups (Fausto-Sterling 2008; Lee 2014; Kuzawa and Sweet 2009; Saldanha 2009, Thayer and Kuzawa 2011; Wright 2015).

In such analyses, the variegated spatial scale compels us to take into account the specificities of racial formation at the level of identity, norms, and interpersonal relations as well as in the geographies and cultures of institutions, neighborhoods, cities, regions, nations, and digital communities. What is per-

tinent here ranges from the micro-aggressions of everyday life to gross patterns of political, economic, legal, and cultural distinction and separation. The variegated temporal scale compels us to take into account not only the historical specificity of the forms of symbolization and representation that mobilize and reference differentiation but also the means through which those histories are carried partially forward and oftentimes mixed in various political and economic institutions and in a range of cultural and legal practices. Because these approaches foreground the complex spatial and temporal dimensions of racial formation and racialization, they provide a means to understand the embodiment of race as a dynamic process that has enduring although not intransigent effects on the processes that constitute living for humans conceived as biocultural creatures.

What is particularly striking is these analyses that recalibrate the spatial and temporal dimensions of race is the way in which what we know as "race" is doubled in the sense that it is neither just cultural nor just biological but rather both—or biocultural as the concept is developed through this chapter. So, in one facet of analysis, race consists in an astonishingly complex variety of symbolic, social, material, and oftentimes violent forms of differentiation. In another corner of analysis are the effects that these forms of differentiation have on the circulation of stress hormones and steroids, the sensitivity of inflammatory and immunitary processes, the regularity of sleep patterns, the digestion and metabolism of nutrients, and vulnerabilities to injury, sickness, and depression, all of which in turn affect how people live with and among one another, shaping them as social and political subjects (Mendenhall et al. n.d.; Thayer 2014; Thayer and Kuzawa 2015). From within this doubled perspective, race as social and political differentiation and organization produces biocultural habitats that culture biocultural creatures in ways affected by that differentiation and organization. To deploy the terms of Troy Duster's (2006a, b) argument, racialized biocultural subjects are produced as such through living in racially striated biocultural habitats.

One of the dangers in pointing out this kind of doubling is that the social and political marks of representation might be taken as a proxy, for example, for the kinds of biological traces of stress that are induced by features of the social and material environments organized in keeping with those representational forms and norms. I am thinking here of the forms of racialized medicine that generalize health predicaments via racial categories (Duster 2005, 2015; Fujimura 2015; Fullwiley 2008; Inda 2014; Montoya 2007; Roberts 2011; Tallbear 2013). To mitigate this slippage from "the biocultural formation of racialized subjects" to "race as a cause or marker of disease," we need to attend (again) to the multiple scales and varied topologies and temporalities of biocul-

tural habitats. We need to keep in mind that simply being racialized in a racially differentiated biocultural habitat is not determinative for a demographically delimited form of good or ill health. Racial forms of differentiation are crosscut by class, gender, and myriad other micro and macro dimensions of biocultural habitats, not only in the material organization of life in the form of work, diet, shelter, toxins, medical care, and green space but also in the patterns of ideas, icons, friendships, and kinship relations that provide inspiration, consolation, desolation, and hope. Furthermore, the evental, periodic, habitual, or chronic persistence of these biocultural factors, the timespan of life within which they provoke each biocultural subject, their concurrence, synchronicity, or asynchronicity with each other all have an effect on how a biocultural creature develops and persists, in sickness or in health. The complexity of biocultural factors involved in composing, decomposing, and recomposing biocultural creatures means that even when human subjects live together in similar biocultural habitats, they will not necessarily have similar lives or similar selves. Perhaps another way to state the point of this thesis is that we cannot appreciate or apprehend how living subjects' biological processes are responsive to their biocultural habitats without taking into account what those biocultural habitats are.

PAUSE.

With nods and winks to Karl Marx's *Theses on Feuerbach* (1978), I am often asked what the 11th thesis might be. It is not logically necessary. But an 11th thesis would likely petition scholars to formulate their questions and present their findings in ways that enhance rather than detract from the conditions for people's political or collective self-determination.

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