

Mary McEniry

Early Life Conditions and Rapid Demographic Changes in the Developing World

Consequences for Older Adult Health

 Springer

Early Life Conditions and Rapid Demographic Changes in the Developing World

Mary McEniry

Early Life Conditions and Rapid Demographic Changes in the Developing World

Consequences for Older Adult Health

 Springer

Mary McEniry
Inter-university Consortium for Political
and Social Research
Institute for Social Research
University of Michigan
Ann Arbor, MI, USA

ISBN 978-94-007-6978-6 ISBN 978-94-007-6979-3 (eBook)
DOI 10.1007/978-94-007-6979-3
Springer Dordrecht Heidelberg New York London

Library of Congress Control Number: 2013949683

© Springer Science+Business Media Dordrecht 2014

This work is subject to copyright. All rights are reserved by the Publisher, whether the whole or part of the material is concerned, specifically the rights of translation, reprinting, reuse of illustrations, recitation, broadcasting, reproduction on microfilms or in any other physical way, and transmission or information storage and retrieval, electronic adaptation, computer software, or by similar or dissimilar methodology now known or hereafter developed. Exempted from this legal reservation are brief excerpts in connection with reviews or scholarly analysis or material supplied specifically for the purpose of being entered and executed on a computer system, for exclusive use by the purchaser of the work. Duplication of this publication or parts thereof is permitted only under the provisions of the Copyright Law of the Publisher's location, in its current version, and permission for use must always be obtained from Springer. Permissions for use may be obtained through RightsLink at the Copyright Clearance Center. Violations are liable to prosecution under the respective Copyright Law.

The use of general descriptive names, registered names, trademarks, service marks, etc. in this publication does not imply, even in the absence of a specific statement, that such names are exempt from the relevant protective laws and regulations and therefore free for general use.

While the advice and information in this book are believed to be true and accurate at the date of publication, neither the authors nor the editors nor the publisher can accept any legal responsibility for any errors or omissions that may be made. The publisher makes no warranty, express or implied, with respect to the material contained herein.

Printed on acid-free paper

Springer is part of Springer Science+Business Media (www.springer.com)

*To my parents:
Matthew J and Marcella L McEniry
The foundation of my early life*

Acknowledgements

I have been fortunate in many ways. The award that I received from the National Institute of Aging for a five-year grant to study early life conditions and older adult health in the developing world (grant number K25 AG027239) made my research possible. The award arrived at a very critical period in my academic career and thus I will always be deeply grateful.

I have also benefited from the rich scholarly environment found at the Center for Demography & Ecology (grant number R24 HD47873) and the Center for Demography of Aging (grant number P30 AG017266) at University of Wisconsin-Madison and the Institute for Social Research, ICPSR, Population Studies Center (grant number R24 HD041028) at the University of Michigan. The Social Science Computing Cooperative at the University of Wisconsin-Madison provided excellent computing resources. The weekly seminars and other presentations at the University of Wisconsin and University of Michigan, and at national and international conferences organized by the Population Association of America (PAA), Gerontological Society of America (GSA), Social Science History Association (SSHA), American Public Health Association (APHA), and International Union for the Scientific Study of Population (IUSSP) all contributed to my intellectual and academic experience. Through these venues I had the opportunity to meet and interact with many of the senior scholars in the fields of demography, sociology, historical demography, epidemiology, and public health, and this interaction enriched my work.

Throughout the development and writing of this book many people touched my life and work including senior scholars, colleagues and friends such as Alberto Palloni, Bob Hauser, John Mullahy, Michel Guillot, George Alter, Barbara Anderson, Bob Shoeni, Francisco Scarano, Bernard Harris, Maria Muniguerria, Nurul Alam, Gilbert Brenes, John García, Ana Luisa Dávila, Susan DeVos, Jim Walker, anonymous reviewers, Patty Mullins, Carmen Alonso, George and Eileen Reilly, Mary Vardigan, Christine Schindler and Elyce Rotella. Bernadette Deelen-Mans and Evelien Bakker, Sunil Padman and the production unit at Springer were absolutely wonderful in all of their communication and feedback.

There have been a couple of people who have helped tremendously in the support of my research. Sarah Moen was flexible in what she could do and contributed in many ways. She spent many hours diligently editing, making suggestions, and checking and rechecking the many details and facts that go into creating a book. This included analyses to check for inconsistencies and also producing tables and graphs. It also included reviewing and checking the literature contained in the book. Jacob McDermott was very adept at the technical aspects of the project including programming, superb graphical design and statistical analyses. His thoroughness, curiosity and willingness to learn were much appreciated. Both Sarah and Jacob were instrumental in carefully checking the construction of the cross national data that form the basis of the book and in the writing and development of the technical report that describes the cross national data.

Others were also very helpful in terms of editing (Phyllis Stillman); data entry (Aimee Joutras); computer support (Nancy McDermott, Andy Arnold); library support (John Carlson, Lee Ridley); user support (Carol Roan, Joe Savard, Jack Kneeshaw, Elizabeth Breeze, Nirmala Naidoo, Paul Kowal, Alberto García); administrative support (Jeff Petersen, Janet Clear, Carol Tetzlaff, Carol Gilmore); and data archiving (Lu Chou, Karen Sullivan, Tannaz Sabet, Tommaso Pavone, Joseph Lindblad, Joseph Chatham, John Yim, Joshua Goode).

There were also those who, while not directly involved in my work, nevertheless shaped my academic experience (Jim Raymo, Betty Thomson, Giovanna Merli, Guido Pinto, Gay Seidman, Beatriz Novak, Flavia Andrade, Malena Monteverde).

My desire is that my work and what I have written will contribute in a positive way to the advancement of knowledge.

Contents

1 Aging Populations and the Determinants of Older Adult Health	1
1.1 Aging and Its Importance in Low and Middle Income Countries	2
1.2 Early Life Nutrition and Infectious Diseases and Older Adult Health	4
1.2.1 Implications of Poor Early Life Nutrition and Infectious Disease for Older Adult Health	6
1.3 Health as an Accumulation of Effects Across the Life Course	9
1.3.1 Debate About Relative Importance of Early Life Conditions	10
1.4 Long Term Consequences of Mortality Decline in the Early Twentieth Century	11
1.4.1 Setting: Public Health, Standards of Living, and Nutrition	12
1.4.2 Mortality Decline of the Early to Mid Twentieth Century	13
1.4.3 Continued Stagnant Economic Conditions and Poor Nutrition and Risk of Infection	22
1.4.4 Growth Rates and Implications of the Demographic Past	24
1.4.5 Unique Cohorts of the 1930s–1960s and Tip of the Iceberg Countries	24
1.5 The Intriguing Case of Puerto Rico	26
1.5.1 Standard of Living	27
1.5.2 Diet and Nutrition	28
1.5.3 Providing for One’s Family	31
1.5.4 Infectious Diseases in Mothers, Infants, and Children in Puerto Rico	34
1.5.5 Changes in the Late 1920s Through the Early 1940s: Reduction in Exposure to Disease but Stagnant Economic Conditions	38
1.6 Unintended Consequences and Future Policy Implications	45
1.7 Conclusions	46

2	A Contrarian Conjecture, Road Map, Ideal Data, Approach	47
2.1	A Contrarian Point of View	47
2.2	Road Map and Cross-National Comparisons	52
2.3	Ideal Data, Surveys of Older Adults, and Historical Data	53
2.3.1	Population Studies: Early Life	55
2.3.2	Population Studies: Older Adult Health and Life Course Events	59
2.4	Approach	60
2.4.1	Mortality Regimes and Tip of the Iceberg Countries	63
2.4.2	Validity of Early Life Measures and Older Adult Health	64
2.4.3	Morbidity: Population-Level and Individual-Level Analyses	65
2.4.4	Morbidity and Mortality: Season of Birth	65
2.4.5	Mortality	66
2.4.6	Health Inequality	67
2.5	Conclusion	68
3	Data Sources, Measures, Validity, and a Description of the Older Adult Population	69
3.1	Data Sources and Their Limitations	69
3.2	Measures	73
3.2.1	Mortality Regimes and Tip of the Iceberg Countries	73
3.2.2	Selected Measures of Early Life Conditions	78
3.2.3	Adult Lifestyle and SES	79
3.2.4	Adult Health Outcomes	80
3.3	Validity	83
3.3.1	Birthplace and Parental SES	83
3.3.2	Puerto Rico	85
3.3.3	Underestimation of Chronic Conditions	87
3.4	The Older Adult Population Across Mortality Regimes	92
3.4.1	Basic Characteristics	92
3.4.2	Early Life Conditions	95
3.4.3	Adult Lifestyle, Standards of Living and Educational Attainment	98
3.4.4	Prevalence of Heart Disease and Diabetes	101
3.5	Conclusions	104

4	Cross-National Morbidity Patterns	105
4.1	Pooled Models for the Cohort of the Late 1920s and Early 1940s	106
4.2	Population-Level and Individual-Level Analyses	110
4.2.1	Discussion	115
4.3	Long Term Consequences for Older Adult Health: The Case of Puerto Rico	121
4.3.1	Discussion	126
4.4	Conclusions	129
5	Cross-National Mortality Patterns and Health Disparities	131
5.1	Mortality	131
5.1.1	Waller-Type Surfaces and Observed Mortality Rates	132
5.1.2	Case of Puerto Rico	138
5.2	Health Disparities	145
5.2.1	Discussion	150
5.3	Conclusions	152
6	Tide, Trickle, or Flow	153
6.1	Factors Across the Life Course and the Wellbeing of Older Adults	154
6.1.1	Morbidity: Adult Heart Disease and Diabetes	154
6.1.2	Mortality and Health Disparities	155
6.2	Relative Importance of Early Life Conditions	157
6.2.1	Critical Period Versus Accumulation of Effects	157
6.2.2	Cohort vs Period Effects	158
6.2.3	Mediation of Effects of Early Life	159
6.3	Unintended Consequences and Policy Implications	160
6.4	Research Agenda	163
6.5	Strengths and Limitations	164
6.6	The Future: Tide, Trickle or Flow	165
	Appendix A: Sources for Life Expectancy and Infant Mortality	169
	Appendix B: Graphs for Life Expectancy and IMR Across Time and Countries	173
	Appendix C: Description of Cross-National Data on Aging Populations	177
	Appendix D: Descriptive Statistics on Older Adult Heart Disease and Diabetes	181
	References	189
	Index	213

List of Figures

Fig. 1.1	Growth rate of the population aged 60 years and older	3
Fig. 1.2	Early life pathways to adult heart disease	7
Fig. 1.3	Early life pathways to adult diabetes	8
Fig. 1.4	GDP per capita (1990 international dollars) throughout the twentieth century in selected mortality regimes	14
Fig. 1.5	Life expectancy in selected countries and mortality regimes	16
Fig. 1.6	Infant mortality in selected countries and mortality regimes	19
Fig. 1.7	Life expectancy and GDP per capita (1990 per 100 international dollars) from 1900 to 2000	20
Fig. 1.8	Percent of water samples containing <i>E. coli</i> in wealthier municipalities in Puerto Rico, 1929–1944	39
Fig. 1.9	Infant mortality in Puerto Rico (1900–2000)	41
Fig. 1.10	Infant mortality rates in Puerto Rico by selected region and municipality, 1929 and 1943	42
Fig. 1.11	Mortality rate for infants, stillbirths, mothers, and infants less than 1 year with diarrhea	43
Fig. 1.12	Seasonality of death rates due to malaria and dysentery in Puerto Rico, 1936–1944	43
Fig. 1.13	Adult male height in US mainland males and Puerto Rican males	44
Fig. 3.1	Association between growth rate of the population aged 60 years and older and change in IMR, 1930–1960	76
Fig. 3.2	Categorization of countries into mortality regimes	77
Fig. 3.3	Percent born poor by rural and urban area	98
Fig. 4.1	Caloric intake in early life and older adult heart disease	110
Fig. 4.2	Caloric intake in early life and older adult diabetes	113
Fig. 4.3	Seasonal variation in plantation employment and hypothesized exposure during late gestation	122
Fig. 4.4	Predicted prevalence of heart disease by level of exposure during late gestation	123
Fig. 4.5	Predicted prevalence of diabetes by exposure period	124

Fig. 4.6	Kaplan Meier hazard estimates by level of exposure during late gestation	125
Fig. 4.7	Predicted hazard by level of exposure during late gestation	126
Fig. 5.1	Expected relative risk of mortality for males born during the late 1920s–early 1940s	132
Fig. 5.2	Expected relative risk of mortality for females born during the late 1920s–early 1940s	133
Fig. 5.3	Predicted probability of death by exposure, 75 years and older ...	142
Fig. 5.4	Prevalence of heart disease by educational level	147
Fig. 5.5	Likelihood of adult heart disease for those with no education ...	148
Fig. 5.6	Prevalence of diabetes by educational level	149
Fig. 5.7	Likelihood of adult diabetes for those with no education	150
Fig. B.1	Life expectancy vs. ln GDP per capita (1990 international dollars) from 1900 to 2000	174
Fig. B.2	IMR vs. ln GDP per capita (1990 international dollars) from 1900 to 2000	175

List of Tables

Table 1.1	Components of daily caloric intake per capita in the 1930s	15
Table 1.2	Estimated percent of mortality decline in developing countries 1900–1970 due to a particular disease along with strategies for prevention and treatment	18
Table 1.3	Daily caloric supply per capita in selected countries across time	23
Table 1.4	Population, access to medical care, and quality of water by wealth of municipality in the 1930s in Puerto Rico	29
Table 1.5	Yearly per capita consumption of foods in Puerto Rico and selected areas of the United States (in pounds)	30
Table 1.6	Major causes of infant mortality in Puerto Rico in 1934	35
Table 2.1	GDP per capita, standard of living, and health care in selected countries	48
Table 2.2	Demographic regimes and expected health patterns for the unique cohorts of the 1930s–1960s	53
Table 3.1	Cross-national data on aging populations	70
Table 3.2	Description of cross-national sample	71
Table 3.3	Patterns of mortality decline from 1930–1960	74
Table 3.4	Association between father’s parental education and being born in a rural area prior to 1945 (pre-antibiotic cohorts)	84
Table 3.5	Bivariate associations between “no schooling” for respondent’s mother and other childhood and adult variables	86
Table 3.6	Prevalence of heart disease and diabetes according to seasonal exposure and place lived during childhood	87
Table 3.7	Associations between self-reported childhood health and childhood illnesses, and self-reported childhood SES and father education and occupation	88
Table 3.8	Prevalence of health conditions and adult factors known to be associated with heart disease and diabetes	89
Table 3.9	Comparison between self-reports, symptom, and biomarker data	91

Table 3.10	Basic demographic characteristics of the cross-national sample (pre-antibiotic cohorts, born prior to 1945)	93
Table 3.11	Selected variables on early life conditions (pre-antibiotic cohorts, born prior to 1945)	96
Table 3.12	Adult behavior or risk factors (pre-antibiotic cohorts, born prior to 1945)	99
Table 3.13	Degree to which respondent's educational attainment is higher than father's educational attainment	102
Table 3.14	Age-standardized prevalence of adult heart disease and diabetes (pre-antibiotic cohorts, born prior to 1945)	103
Table 4.1	Likelihood of reporting heart disease for those born in the late 1920s–early 1940s	107
Table 4.2	Likelihood of reporting diabetes for those born in the late 1920s–early 1940s	108
Table 4.3	Likelihood of being obese for those born in the late 1920s–early 1940s	109
Table 4.4	Caloric intake and the likelihood of reporting heart disease for those born in the late 1920s–early 1940s	112
Table 4.5	Caloric intake and the likelihood of reporting diabetes for those born in the late 1920s–early 1940s	114
Table 4.6	Caloric intake and the likelihood of being obese for those born in the late 1920s–early 1940s	116
Table 4.7	Effects of being born during the lean season on adult health in Puerto Rico (reference group born during harvest)	122
Table 5.1	Expected relative risk and observed relative risk (males born in the late 1920s–early 1940s)	135
Table 5.2	Excess of relative risk of mortality (Waaler) for males born in the late 1920s–early 1940s	136
Table 5.3	Age-standardized mortality for those reporting heart disease and diabetes for those born in the late 1920s–early 1940s	136
Table 5.4	Comparison of childhood illnesses and SES by cohorts	139
Table 5.5	Likelihood of dying for PREHCO respondents who lived in rural areas as children	141
Table A.1	Sources for life expectancy and infant mortality	169
Table B.1	Growth rate for 60 years and older	176
Table C.1	Description of cross-national data on aging populations as of August 2011	178
Table D.1	Prevalence of heart disease by age and gender	181
Table D.2	Prevalence of diabetes by age and gender	182

Table D.3	Proportion who are obese for those with and without heart disease	183
Table D.4	Proportion who are obese for those with and without diabetes	184
Table D.5	Proportion reporting difficulty with at least one functional task for those with and without heart disease	186
Table D.6	Proportion reporting difficulty with at least one functional task for those with and without diabetes	187

There was a dramatic improvement in life expectancy during the twentieth century, especially for those born between the 1930s and 1960s, primarily due to reductions in infant and child mortality. Widespread public health interventions including advancements in medical technology helped alleviate poor early life conditions – serious infectious diseases, poor nutrition, and harsh living environments – by reducing exposure to disease and treating it more effectively.¹ In some developing countries, this happened largely in the absence of improved standards of living.² The dramatic increase in life expectancy was one of the demographic transitions of the early twentieth century³ and partially explains the growth of aging populations throughout the world, in particular in low- and middle-income countries in the early twenty-first century.⁴ This growth is projected to increase the burden of disease due to chronic conditions such as adult heart disease and diabetes—conditions which in some instances originate in early life⁵—as populations age.⁶

This book examines the degree to which these historical circumstances produced cohorts largely characterized by increased survival of poor early life conditions which are, as a result, more susceptible at older ages to adult heart disease, diabetes, and higher mortality. The unfolding story encompasses a wide spectrum of low-, middle- and high-income countries that had different patterns of mortality decline

¹ Preston (1976).

² Riley (2008).

³ Demographic, epidemiological, and nutritional transitions are related concepts. Development and urbanization leads to a shift from rural, low life expectancy and high fertility to urban, higher life expectancy and lower fertility. Infectious diseases in early life become less dominant and chronic diseases at older ages more dominant. Diets change to those with higher concentrations of saturated fats and sugar. A more sedentary lifestyle may then lead to health problems such as obesity.

⁴ Kinsella and He (2009) and Kinsella and Velkoff (2001).

⁵ Barker and Osmond (1986) and Elo and Preston (1992).

⁶ Murray and López (1996), Lim et al. (2012), and Murray et al. (2012).

leading into the 1930s–1960s.⁷ These different patterns not only tell a story of the relevance of poor early life conditions to the health of older adults, but also how a series of historical circumstances may have led to unintended consequences (beyond the positive intended consequences of better health through improved sanitation, better health education, and improved medical technology) and may shape the health of older adults in some settings well into the future.

1.1 Aging and Its Importance in Low and Middle Income Countries

Aging in the developing world is of growing concern among policy makers and researchers and continues to receive national and international attention for a number of reasons.⁸ First, the growth of aging in the developing world is different. While it took nearly 70 years for US elderly to increase from 7 to 14 % of the total population, it is estimated that it will take only 20–30 years to see similar increases in some developing countries; since the 1970s, the average annual percentage growth rate of the elderly population has been higher in developing than in developed countries.⁹ The growth rate of the population 60 years and older for the US and the UK was 0.9 and 0.2 %, respectively, between 2000 and 2005 and is projected to grow slowly.¹⁰ In contrast, the average growth rate for this population in many developing countries was 2–3 % (even higher in some countries) between 2000 and 2005. It is projected that the rate will increase to 3–4 % in countries with large populations, such as China, India, and Mexico, by 2030.¹¹ Inspection of growth rates and projected growth rates from 1950 through 2050 show this to be the case (Fig. 1.1, also see [Appendix B, Table B.1](#)).

Second, in the developed world, the growth of the aging population occurred in an environment where standards of living were typically high. In the developing world, however, even the most optimistic forecasts indicate the picture of aging is a dismal one. Most developing countries have not had the time or means to generate sustained high standards of living for any of their citizens. For example, no appreciable fraction of time during which the countries of the Latin American and Caribbean (LAC) region are aging rapidly will be characterized by a GNP per capita exceeding \$10,000.¹² Barring unprecedented economic development, countries in the LAC region will be faced with rapid aging paired with a precarious standard of living.

Aging in the developing world will also occur within weaker familial and institutional settings. While rapid aging is occurring, traditional family and kin relationships

⁷ The categorization of countries into low-, middle-, and high-income used throughout the book is based on the World Bank (2011).

⁸ National Research Council (2001).

⁹ Kinsella and Velkoff (2001).

¹⁰ United Nations (2002).

¹¹ UN (2002).

¹² Palloni et al. (2004).

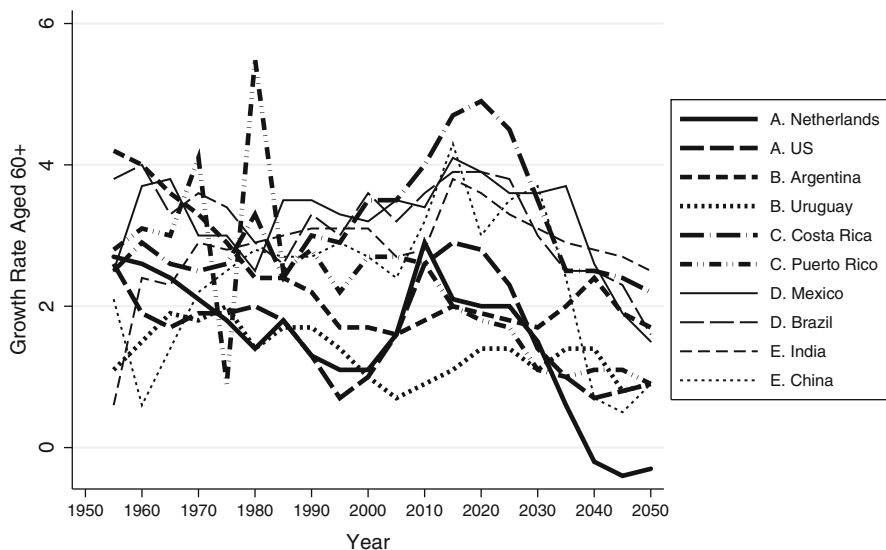


Fig. 1.1 Growth rate of the population aged 60 years and older. *Note:* Long term forecasts past 2020 may not be as accurate as shorter term forecasts (*Source:* Author's calculations based on data obtained from United Nations Department of Economic and Social Affairs (2010))

are gradually being dismantled in some regions such as the LAC.¹³ In addition, there are worrisome signs that formal institutions, such as social security systems, are being reformed in ways that are likely to have a negative impact on the ability of the elderly to secure a safe standard of living.¹⁴ Health care systems in the developing world are not adequately prepared to address the care of a rapidly growing elderly population with an increasing incidence of chronic diseases such as diabetes and heart disease; treatment costs in the LAC region will be staggering.¹⁵

Third, by all indications there will be an increasing number of older adults in the developing countries with chronic conditions such as heart disease and diabetes. These chronic conditions are projected to be important future health concerns in several developing countries; in many instances, they coexist with infectious diseases (e.g., malaria, intestinal infections, and respiratory tuberculosis).¹⁶ Heart disease is now one of the leading causes of death in the developing world.¹⁷ Heavily populated countries such as Brazil and China are projected to see a significant

¹³ De Vos and Palloni (2001) and Palloni (2002).

¹⁴ Mesa-Lago (1994), Barrientos (1997), and Klinsberg (2000).

¹⁵ Barceló, Aedo, Rajpathak, and Robles (2003).

¹⁶ Amos, McCarty, and Zimmet (1997), Beaglehole and Yach (2003), King, Aubert, and Herman (1998), Murray and López (1996), Nikolic, Stanciole, and Zaydman (2011), and World Health Organization (WHO) (2000a, 2000b).

¹⁷ Kinsella and He (2009).

increase in cardiovascular disease.¹⁸ In India, another heavily populated country, by the year 2020, cardiovascular disease will be the largest cause of disability and death; India also has one of the largest populations with diabetes in the world, along with high rates of hypertension and cardiovascular disease.¹⁹ Africa is prone to higher rates of diabetes.²⁰ Researchers anticipate that by the year 2025, China, Indonesia, Mexico, and Brazil will be among the top ten countries in the world in terms of numbers of adults with diabetes.²¹

Obesity is an important risk factor for diabetes and heart disease and is rapidly increasing in developing countries.²² In addition, there have been dietary changes which reflect higher consumption of saturated fats and refined carbohydrates which can result in higher risk of disease.²³ The economic costs of chronic conditions including heart disease and diabetes in the developing world are estimated to be huge and growing. Conservative estimates of the cumulative gross domestic product (GDP) loss (in US dollars) between 2006 and 2015 due to these chronic conditions range from 14 billion in China to 17 billion in India and over 1 billion in Bangladesh²⁴; alternative methods estimate these costs to be even higher.²⁵

A final important reason for the focus on population aging in the developing world is that while much is known about the growth of the aging population, standards of living, and familial and institutional settings, less is known about the determinants of elderly health status. The aging process is shaped across the entire life course,²⁶ and yet there is insufficient evidence for how various factors over the life course influence health and disease across a variety of diverse countries in the developing world.²⁷ Such evidence is of interest and importance to policy makers, researchers, and others engaged in population aging and the implementation of effective programs and policies for older adults.

1.2 Early Life Nutrition and Infectious Diseases and Older Adult Health

There is continued interest in the idea that poor environmental circumstances in early life, such as poor nutrition, infectious diseases and adverse socioeconomic conditions, can have lasting effects into old age (60+ years).²⁸ Several mechanisms

¹⁸ Leeder, Raymond, Greenberg, Liu, and Esson (2004).

¹⁹ Reddy (2009).

²⁰ Prentice (2009).

²¹ King, Aubert, and Herman (1998).

²² Monteiro, Conde, Lu, and Popkin (2004) and WHO (2000a).

²³ Popkin (1998).

²⁴ Abegunde, Mathers, Adam, Ortegón, and Strong (2007).

²⁵ Barceló et al. (2003), Reddy (2009), and WHO (2005, 2008).

²⁶ Kuh and Ben-Shlomo (2004).

²⁷ National Research Council (2001).

²⁸ See for example WHO (2006).

explain how poor early life conditions may affect older adult health: scarring (*in utero*/infancy, infectious diseases of childhood), acquired immunity, selection of the biologically robust, and indirect mechanisms operating through adult socioeconomic conditions and lifestyle.²⁹ Inflammation originating *in utero* may have long term negative consequences for health.³⁰ Poor childhood SES and health can have substantial impacts on adult health.³¹ Strain in childhood but also in youth and early adulthood (e.g., disease, poverty, low education or stressful working environment) increases the risk of early occurrence of chronic disease (e.g., cardiovascular disease), which in turn increases the risk of premature disability and mortality. Some childhood illnesses such as rheumatic fever, respiratory diseases, and severe bouts of diarrhea are clearly tied to older adult health.³² Important background conditions that influence early growth and development could also have independent effects on later health and mortality.³³

Abundant evidence from those born in the late nineteenth century or early twentieth century in the developed world suggests that strong associations exist between early life and older adult health. The 1918 flu epidemic, World War II famines, and early life conditions in England, Sweden, the Netherlands, Ireland, Australia, and the US have all had a long-term impact on adult health beginning before or at conception, continuing *in utero* and early infancy (critical timing for nutritional insults *in utero* and early infancy and for inflammatory processes *in utero*), and extending through childhood (socioeconomic conditions and exposure to infectious diseases during childhood).³⁴ Evidence now accumulating from older adults born during the early twentieth century in low- and middle-income

²⁹ Preston, Hill, and Drevenstedt (1998).

³⁰ Crimmins and Finch (2006).

³¹ Davey Smith and Lynch (2004), Elo and Preston (1992), Gunnell, Davey Smith, Holly, and Frankel (1998), Hertzman (1994), Lundberg (1991), and Wadsworth and Kuh (1997).

³² Elo and Preston (1992).

³³ Huxley, Neil, and Collins (2002) and Joseph and Kramer (1996).

³⁴ There is a large literature from the fields of demography and biology indicating early life conditions are important to older adult health. See for example Almond and Currie (2010), Alter (2004), Barker (1998, 2002), Barker, Thornburg, Osmond, Kajantie, and Eriksson (2010), Bengtsson and Lindstrom (2000, 2003), Bengtsson and Mineau (2009), Blackwell, Hayward, and Crimmins (2001), Case and Paxson (2010); Costa (2002), Crimmins and Finch (2006), Davey Smith et al. (2000), Davey Smith, Hart, Blane, and Hole (1998), Davey Smith and Lynch (2004), Doblhammer (2004), Elo and Preston (1992), Eriksson, Forsen, Tuomilehto, Osmond, and Barker (2001), Ferrie, Rolf, and Troesken (2009), Finch and Crimmins (2004), Floud, Wachter, and Gregory (1990), Forsdahl (1978), Gagnon and Mazan (2006), Gluckman and Hanson (2006); Johnson and Schoeni (2007, 2011), Newnham and Ross (2009), Haas (2008), Hayward and Gorman (2004), Hertzman (1994), Gunnell et al. (1998), Kuh, Power, Blane, and Bartley (2004), Lundberg (1991), Leon, Davey Smith, Shipley, and Strachan (1995), Leon and Davey Smith (2000), Otero-Rodríguez et al. (2011), Palloni (2006), Peck and Lundberg (1995), Regidor, Guitierrez-Fisac, Calle, Navarro, and Domínguez (2002), Svensson, Broström, and Oris (2004), van den Berg, Lindeboom, and Portrait (2006), Wadsworth, Hardy, Paul, Marshall, and Cole (2002), Wadsworth and Kuh (1997), and Wickrama, Conger, and Abraham (2005).

countries also suggests the importance of early life conditions on older adult health.³⁵ These studies have produced evidence for associations with older adult health: poor childhood socioeconomic status (SES) is associated with adult mortality, functionality, and cognition; poor childhood health as reflected in serious childhood illnesses is correlated with adult functionality and chronic disease; specific linkages between childhood illnesses such as rheumatic fever in childhood and adult heart disease and malaria and adult mortality due to stroke; and *in utero*/early infancy exposures to poor nutrition and infectious diseases (independent of other early life and adult conditions) are associated with adult heart disease and diabetes.

1.2.1 Implications of Poor Early Life Nutrition and Infectious Disease for Older Adult Health

Diseases such as adult heart disease and diabetes can be strongly influenced by nutrition.³⁶ The synergistic relationship between nutrition and infection may also play an important role.³⁷ Poorly nourished individuals are at higher risk of infection and this may cause further exacerbate already existing conditions or create further complications.

The idea proposed by Barker and colleagues that the critical timing of nutritional insults *in utero* or early infancy can contribute to the risk of chronic conditions at older ages (the Barker hypothesis) continues to be of interest and debate.³⁸ Barker

³⁵ Since the Elo and Preston (1992) article and the Barker (1998) book, the list of studies conducted in the developing world has grown. See for example Beltrán-Sánchez, Crimmins, Teruel, and Thomas (2011), Brenes (2008), Brenes-Camacho and Palloni (2011), Campbell and Lee (2009), Crimmins et al. (2005), Davis, Campbell, and Lee (2009), Godoy, Goodman, Levins, Caram, and Seyfried (2007), Huang and Elo (2009), Huang, Soldo, and Elo (2011), Kohler and Soldo (2005), Martorell, Stein, and Schroeder (2001), McEniry (2011b), McEniry and Palloni (2010), McEniry, Palloni, Dávila, and García (2008), Monteiro, Moura, Conde, and Popkin (2004), Monteverde, Noronha, and Palloni (2009), Moore et al. (1999), Palloni, McEniry, Dávila and García Gurucharri (2005); Palloni, McEniry, Wong, and Peláez (2006), Sichieri, Siqueira and Moura (2000), Schooling et al. (2011), Schroeder, Martorell, and Flores (1999), Victora et al. (2008), Xu et al. (2009), Zeng, Gu, and Land (2007), Wen and Gu (2011), and Zhang, Gu, and Hayward (2010). In addition, there are now numerous studies conducted on children in the developing world. See for example Van Ewijk, Painter, and Roseboom (2013), Yajnik and Dshmuikh (2008) and the recent review article (McEniry, 2012).

³⁶ Popkin, Horton, and Kim (2001) and Barker (1998).

³⁷ Crimmins and Finch (2006).

³⁸ Barker (1998) and Kuh and Ben-Shlomo (2004). There are also several critiques of the Barker hypothesis in regards to the importance of other life course factors that may explain health (Huxley, 2006; Huxley, Neil, & Collins, 2002; Joseph & Kramer, 1996). Small sample sizes have been a problem in some studies, and some have shown weak or no associations between under-nutrition *in utero* and later adult health (Kannisto, Christensen, & Vaupel, 1997; Stanner et al., 1997).

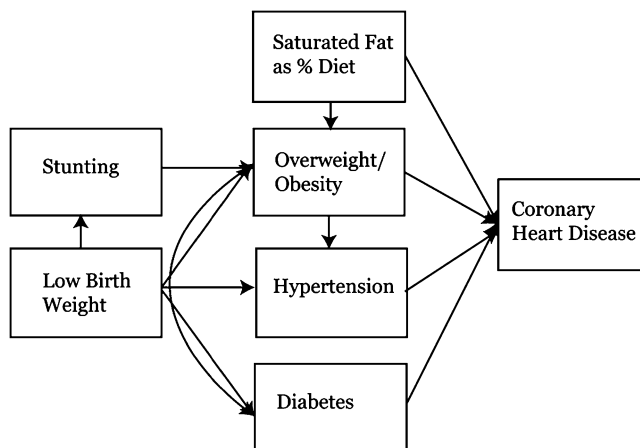


Fig. 1.2 Early life pathways to adult heart disease (*Source:* Adapted from Popkin et al., 2001)

et al.'s argument stresses the importance of disproportionate growth at birth.³⁹ During perinatal or early gestation, the magnitude of fetal demand for nutrients and the trajectory of fetal growth are established. Fetal demand for nutrients is thought to be small until late in pregnancy, a period of rapid fetal growth and organ development particularly sensitive to variations in the supply of nutrients. A favorable nutritional environment in early gestation sets the fetus on a path of rapid growth with higher subsequent nutrient demands which makes the fetus more vulnerable to under-nutrition in late gestation which can permanently affect organ development, generate disproportionate fetal growth, and result in small size at birth. Being born small followed by either slow infant growth or exposure to more abundant nutrition after birth and rapid weight gain can increase the susceptibility to later adult disease. Poor living conditions are not an important confounding factor but may add to the effects of early life conditions.⁴⁰

Figures 1.2 and 1.3 present a framework and reference point by which to interpret results that will be used throughout the remaining chapters. While there are many determinants of adult heart disease and diabetes,⁴¹ the figures suggest that the pathways from early life to adult heart disease and diabetes are slightly different as a consequence of poor nutrition and/or infection *in utero*.⁴² Impaired intrauterine growth as measured by low birth weight has a direct effect on an increased risk of stunting (a measure of malnutrition), diabetes, hypertension, and obesity, some of

³⁹ Barker (2005), Barker, Forsén, Uutela, Osmond, and Eriksson (2001), Barker, Eriksson, Forsen, and Osmond (2002), Barker (1995), Eriksson et al. (2001), Gardiner (2007), Godfrey and Barker (2000); Osmond and Barker (2000), Osmond, Barker, Winter, Fall, and Simmonds (1993).

⁴⁰ Barker et al. (2001) and Barker (1995).

⁴¹ See for example Marmot and Elliott (2005).

⁴² Popkin et al. (2001).

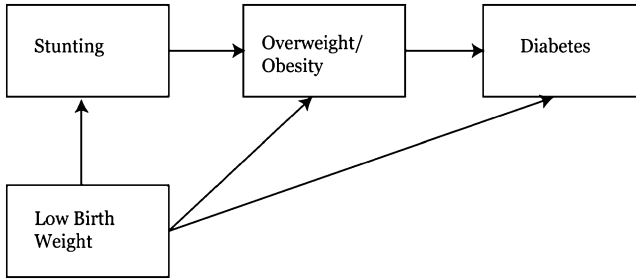


Fig. 1.3 Early life pathways to adult diabetes (Source: Adapted from Popkin et al., 2001)

which lead to adult coronary disease and or diabetes. However, the consequences of poor early life nutrition and infection *in utero* are shown to have a more direct impact on adult diabetes than heart disease. Saturated fat as a percent of one's diet plays a direct role is depicted as a separate important factor that increases the risk of coronary heart disease.

Hypotheses regarding under nutrition and/or infection *in utero* are particularly relevant because most of the population in low- and middle-income countries during the early twentieth century were exposed to less than optimal nutritional environments. These effects may be exacerbated by an adult diet high in saturated fat which is increasingly common.⁴³ There is also growing evidence that early exposure to poor nutrition and/or infection can alter genetic expression without changing the DNA.⁴⁴ A recent examination of data from the Dutch Famine (1944–1945) suggests that poor environmental conditions early in life led to epigenetic changes that persisted throughout life, leading to adult disease.⁴⁵ Adverse conditions *in utero* may also lead to trans-generational epigenetic effects on adult health, just as a grandparent's childhood or adolescent risk behavior can predict negative health behavior in their grandchildren.⁴⁶ Exploring the epigenetic precursors of disease holds promise in developing future therapeutic methods to prevent and/or address disease and produce novel solutions to disease caused by under-nutrition and infection in early life.⁴⁷ Understanding the epigenetic mechanisms that alter genetic expressions may help explain the interaction between genes and environment, particularly at younger ages when sensitivity to the environment may be larger.⁴⁸

⁴³ Bateson et al. (2004).

⁴⁴ Dziadek (2006), Fall and Sachdev (2006), Forrester (2006), Waterland (2006), Whitelaw and Garrick (2006), and Wu, Bazer, Cudd, Meininger, and Spencer (2004).

⁴⁵ Heijmans et al. (2008).

⁴⁶ Pembrey et al. (2006).

⁴⁷ National Institute on Aging (2009).

⁴⁸ Heijmans, Tobi, Lumey, and Slagboom (2009).

1.3 Health as an Accumulation of Effects Across the Life Course

Not all variation in older adult health originates from poor environmental conditions *in utero*/infancy or childhood; rather, older adult health can be an accumulation of adverse events or poor SES across the life course.⁴⁹ In regards to adult heart disease and diabetes, there are many possible intervening variables that may explain their prevalence—prime among them being adult lifestyle.⁵⁰ The risk of adult heart disease and diabetes can be reduced by about 30–40 % through an appropriate diet and lifestyle (e.g., proper exercise, and eliminating smoking).⁵¹

There is little doubt that the transition over the last 30 years or so to diets with higher saturated fat, sugar, and sodium content and to a more sedentary lifestyle throughout the world is an important contributor to changing health patterns at all ages.⁵² The transition has already impacted the developed world. Traditional diets are changing around the world due to increased importation of processed foods. These changes are having adverse effects on health including increased risk of heart disease and diabetes.⁵³

Genetic predispositions and gene-environment interactions may also determine adult heart disease and diabetes, although genetic predispositions may be less important when examining health at a population level.⁵⁴ Economic opportunity and growth, quality of the health care system, and health policy can have important impacts on individual health.⁵⁵ With improved economic opportunity, individuals are more likely to have the resources needed for better nutrition and sanitary conditions and access to medical care. A quality health care system will properly diagnose and treat heart disease and diabetes. Health policy can provide the foundation for programs to increase the opportunity to gain access to quality medical care.

⁴⁹ Aboderin et al. (2002), Kuh and Ben-Shlomo (2004), Otero-Rodríguez et al. (2011), Rockwood and Mitnitski (2007), and Power, Kuh, and Morton (2013), O’Rand and Hamil-Luker (2005).

⁵⁰ Aboderin et al. (2002), Coleman, Ruth, and O’Hanlon (2004), Harper, Lynch, and Davey Smith (2011), Kuh and Ben-Shlomo (2004), Kuh (1999), and Pickles, Maughan, and Wadsworth (2007).

⁵¹ Popkin et al. (2001).

⁵² See for example FAO (2004), Popkin (2006), Schmidhuber and Shetty (2005), Lim et al. (2012), Woolf and Aron (2013), and Basu, Yoffe, Hills, Lustig, and Wagner (2013).

⁵³ De Schutter (2012).

⁵⁴ Case and Paxson (2010).

⁵⁵ Almond and Currie (2010), van den Berg, Doblhammer, and Christensen (2009), Harper, Lynch, and Davey Smith (2011), Bengtsson and Mineau (2009), Davey Smith and Lynch (2004), Elo and Preston (1992), Forsdahl (1978), Hertzman (1994), Lundberg (1991), Wadsworth et al. (2002), and Wadsworth and Kuh (1997).

1.3.1 Debate About Relative Importance of Early Life Conditions

The question of what determines health as an older or elderly adult especially in the developing world is obviously a complex one:

- Is one's health directly a result of *in utero* conditions such as poor nutrition and infectious diseases during critical periods, or is it the result of an accumulation of adverse conditions and events throughout the lifespan?
- Are early life conditions a major contributor to older adult health in the developing world or will recent changes to a diet higher in saturated fat, sugar, and sodium along with an increasingly sedentary lifestyle play a more important role in older adult health?
- Have some individuals been so scarred by their early life circumstances that their health during old age is affected or can other conditions either early or later in life help modify or mediate the effects of poor early life conditions?

The ongoing debate regarding the relative importance of early life continues to be sparked by the difficulty of disentangling the exact mechanisms involved and identifying the relative importance and magnitude of poor early life (*in utero*, early infancy and childhood) circumstances.⁵⁶ Proponents of the importance of a critical period in early life argue that, under certain conditions, the underlying causes of adult chronic conditions such as heart disease or diabetes originate in early life and that circumstances throughout one's life compound or exacerbate but do not confound these effects.⁵⁷ Proponents of health as an accumulation of adverse events throughout the life course argue it is precisely the accumulation which determines older adult health.⁵⁸

The evidence for the relative importance of poor early life nutritional and infectious disease conditions is mixed. In some instances, they have been shown to have a small effect on adult chronic conditions.⁵⁹ In other cases, they have been shown to exhibit equally strong or stronger effects on older adult health than adult lifestyle.⁶⁰ Similarly, the evidence is mixed regarding the degree to which poor early life conditions are mediated or modified throughout the life course. For example, social factors can modify the effects of poor early life conditions, as can interventions before the age of five to supplement nutrition and income.⁶¹ Educational and economic opportunity during young adulthood can also serve as a mediator.⁶² In contrast, famine conditions experienced during

⁵⁶ See for example Otero-Rodríguez et al. (2011).

⁵⁷ Barker (1995, 2001, 2005) and Barker et al. (2001, 2002).

⁵⁸ Kuh and Ben-Shlomo (2004).

⁵⁹ Popkin et al. (2001).

⁶⁰ See for example Huang and Elo (2009), Huang, Soldo, and Elo (2011), and Zeng, Gu, and Land (2007).

⁶¹ Almond and Currie (2010) and Wilkinson and Marmot (2003).

⁶² Davey Smith et al. (1998), Elo, Martikainen, and Myrskylä (2010), and Hayward and Gorman (2004).

early life in the Netherlands during World War II appear to have heavily influenced older adult health even though many of the survivors lived in a highly developed country with good educational and economic opportunity.⁶³ It is yet unclear the degree to which exemplar health care systems⁶⁴ or major health and educational reform⁶⁵ in the developing world can mediate the effects of poor early life conditions at older ages or how a weaker infrastructure, particularly welfare and health institutions, will impact older adult health into the future.⁶⁶ A better standard of living, better health care or a better adult lifestyle (i.e. nutritious diet, exercise, and not smoking) may prolong and improve the quality of life but it is not yet clear the degree to which they completely negate the effects of poor early life conditions.

1.4 Long Term Consequences of Mortality Decline in the Early Twentieth Century

Studies to date regarding poor early life conditions have been mostly country-specific and have, with a few exceptions, focused on individual-level determinants of older adult health.⁶⁷ One set of hypotheses relevant to early life conditions which has yet to be thoroughly tested is based on examining the long term implications for older adult health of the demographic transitions of the early to mid twentieth century which led to the rapid and dramatic increase in life expectancy during the period of the 1930s–1960s. Phases in the demographic transitions and different mortality patterns of the early twentieth century have been described at length reflecting differences in the pace and reason for mortality decline.⁶⁸ However, although these historical circumstances may have long term unintended consequences on older adult health,⁶⁹ only recently has there been comprehensive information on an individual level by across a diverse set of countries by which to examine them.

A few words of introduction are necessary regarding life expectancy in the developed and developing world before fully explaining the reasons for possible long-term unintended consequences for older adult health.

⁶³ Painter et al. (2006), Ravelli et al. (1998), and Roseboom et al. (2000).

⁶⁴ Garnier, Grynspan, Hidalgo, Monge, and Trejos (1997), Rosero-Bixby (1991), and WHO (2000b).

⁶⁵ Bishop, Corbin, and Duncan (1997).

⁶⁶ See for example, Palloni et al. (2007).

⁶⁷ One exception is van den Berg et al. (2009).

⁶⁸ Omran (1971) and Preston (1976).

⁶⁹ Palloni (1981) and Palloni et al. (2007) .

1.4.1 Setting: Public Health, Standards of Living, and Nutrition

The steady improvements in life expectancy in the developed world in the early to mid twentieth century reflect a well-defined pattern where mortality declined at graded or constant rates, although at different stages and due to different causes.⁷⁰ In the early part of the twentieth century, the decline was mostly in deaths of infants and children as a result of public health and sanitation measures which led to reduction in the spread of infectious diseases and better nutritional status.⁷¹ Higher levels of economic development (e.g. better education, housing, and living standards; increased knowledge about exposure and resistance to illnesses) supported broader efforts at sanitation (clean water and waste disposal). This, along with other public health measures and improved nutrition, led to a gradual reduction in infectious diseases and improvements in life expectancy.⁷²

Improvement in poor early life conditions reduced mortality risk and led to better health at older ages. Reduction of infection and inflammation at early ages for those born in the late 1800s led to lower mortality rates at older ages in northern European countries.⁷³ In England, lower early life mortality was associated with lower cause-specific mortality at older ages for those born during the 1920s.⁷⁴ Decreases in infant mortality due to public health programs during the 1940s in Ireland reduced the chances of disability as an older adult, especially in lower socioeconomic groups.⁷⁵

With the introduction of antibiotics and other medical innovations in the mid-1940s, mortality decline became more evenly distributed among different age groups. After the 1960s, high-tech medical interventions, better medications, and a focus on encouraging a healthier adult lifestyle helped further reduce early mortality. Mortality due to chronic conditions such as heart disease in the US, which had increased during the early twentieth century, began to decrease in the 1960s,⁷⁶ likely due to better treatment and improved lifestyles.⁷⁷

The developed world, for the most part, had the resources and economic capacity to invest in public health, sanitation measures, and the research leading to medical breakthroughs such as antibiotics and other therapies. Developed countries were in a more advantageous position due to economic growth, including skilled and educated workforces with rising incomes, allowing their citizens to obtain better nutrition for their families. The fact that many of these countries'

⁷⁰ Cutler and Meara (2001) and Cutler, Deaton, and Lleras-Muney (2006).

⁷¹ Costa (2005).

⁷² Arriaga and Davis (1969).

⁷³ Crimmins and Finch (2006) and Finch and Crimmins (2004).

⁷⁴ Barker and Osmond (1986).

⁷⁵ Delaney, McGovern, and Smith (2009).

⁷⁶ Center for Disease Control and Prevention (2001).

⁷⁷ Ford et al. (2007).

citizens spoke similar languages may have helped disseminate public health information resulting in lower disease rates.⁷⁸ Reduction in infant and child mortality and subsequent improvements in life expectancy paralleled an increase in GDP per capita. Those born in developed countries during the early part of the twentieth century had better access to health care systems which may have helped people born under adverse conditions over the life course. Some adults born in the 1930s in these higher-income countries may have been exposed to poor early life conditions (e.g. the Netherlands during the famine of World War II) but, overall and on average, many improvements in sanitation and public health in the early twentieth century resulted in a higher proportion of the population being in better health at birth by the 1930s.

Conditions were considerably different in the developing world where in the early to mid twentieth century most countries had considerably lower levels of GDP per capita than the developed world (Fig. 1.4) with subsequent consequences for nutrition and diet. Many developing countries (with some exceptions, such as Argentina and Uruguay) experienced serious nutritional deficits (Table 1.1). India and Taiwan were at levels close to or lower than 2,000 cal daily per capita in contrast to 3,000 cal daily per capita in the US, the Netherlands, and England. The diet in these three countries along with Argentina included more animal and dairy products and was better balanced than in the lower caloric countries, where people ate a higher proportion of cereals such as rice. With the exception of Argentina, Uruguay, Chile, South Africa, Brazil, and Russia, daily protein consumption per capita across all developing countries was lower than the recommended international standards of the day (70 g per day).

Public health interventions in the developing world during the early to mid twentieth century were largely patterned after those in the developed world,⁷⁹ but were implemented in settings of lower economic growth and lower levels of GDP per capita illustrated by Fig. 1.4. Improvement in health conditions was focused mainly on the reduction of infectious diseases by improving water and sanitation conditions, malaria or hookworm campaigns, preventive health care, vaccinations and, after 1945, medical innovations such as antibiotics and other therapies.⁸⁰

1.4.2 Mortality Decline of the Early to Mid Twentieth Century

Differences in how public health programs were implemented and the level of resources available produced different patterns of life expectancy and mortality

⁷⁸ Ramasubban (2008) suggests that large developing countries such as India with many different languages and traditions may have encountered more difficulty in implementing successful public health interventions than large developed countries such as the US.

⁷⁹ See for example the description of the Rockefeller Foundation's effort to combat disease throughout the world in the early twentieth century (Farley, 2004).

⁸⁰ Farley (2004).

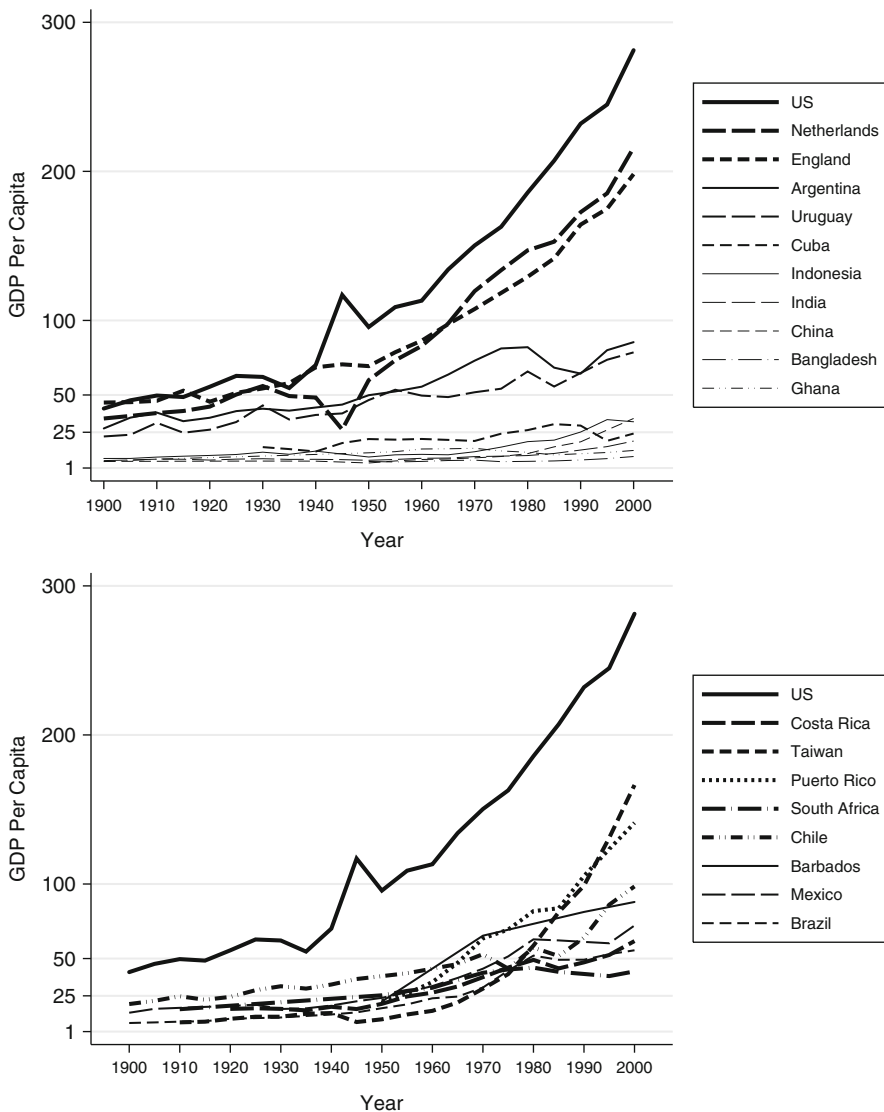


Fig. 1.4 GDP per capita (1990 international dollars) throughout the twentieth century in selected mortality regimes (Source: Maddison, 2006).

decline in the developing world.⁸¹ Thus, while life expectancy converged across many countries towards the end of the 1950s (Fig. 1.5), there were clearly different patterns across countries reflecting differences in the timing, pace, and reason for mortality decline.

⁸¹ Preston (1976).

Table 1.1 Components of daily caloric intake per capita in the 1930s^a

Country ^b	Total calories	Cereals	Roots/tubers	Sugar	Fats ^c	Pulses	F&V	Meat	Milk	Grams of protein	% Protein from animal source
US	3,249	27	4	16	15	3	6	16	11	88	57
England	3,005	30	4	15	17	2	3	19	9	80	54
Netherlands	2,958	33	8	13	19	4	3	11	10	78	47
Argentina	3,275	38	3	11	7	3	3	23	9	111	57
Uruguay	2,902	33	4	11	5	3	4	29	10	102	62
Cuba	2,918	30	20	15	10	7	1	10	7	68	43
Russia	2,827	65	11	4	5	3	1	5	5	88	19
Brazil	2,552	34	17	10	5	8	6	17	4	73	36
Chile	2,481	45	7	11	6	3	5	10	7	70	34
South Africa	2,300	70	2	10	1	3	2	7	6	75	31
Puerto Rico	2,219	36	14	17	9	9	4	9	3	55	31
Taiwan	2,153	51	20	4	6	5	3	11	0	52	27
Costa Rica	2,014	35	1	22	8	7	10	12	1	49	43
Mexico	1,909	53	2	10	6	6	5	10	8	59	34
China	2,201	71	5	1	6	12	2	4	0	68	7
French West Africa ^d	2,311	50	23	1	7	7	2	9	9	68	24
India	2,021	65	2	8	4	10	2	2	0	56	16
Java & Madura ^e	2,040	57	9	2	3	9	3	1	0	43	9

Source: FAO (1946, Appendix 3: Table 2); author's calculation of percentages based on FAO (1946, Appendix 3: Table 2)

Notes:

^aThe number of total calories appearing in this table is an estimation for daily caloric intake per capita prior to WWII

^bInternational standards of the 1960s recommended consumption of 70 g of protein per day (Lanney, 1963). The high income countries (England, Netherlands, US, and Argentina) met this standard in 1939. The Centers for Disease Control and Prevention (2011) now recommend 10–35 % of daily calories should be protein based on age. For example: 13–34 g per day is recommended for children; 46–52 g for adolescents; and 46–56 g for adults

^cThe grouping of countries is roughly according to their level of caloric intake and whether they are a developed or developing country

^dFats includes any visible fats and oils of both animal and vegetable origin

^eInformation for French West Africa was used to provide a reasonable estimate for Ghana

^fInformation for Java and Madura was used to provide a reasonable estimate for Indonesia

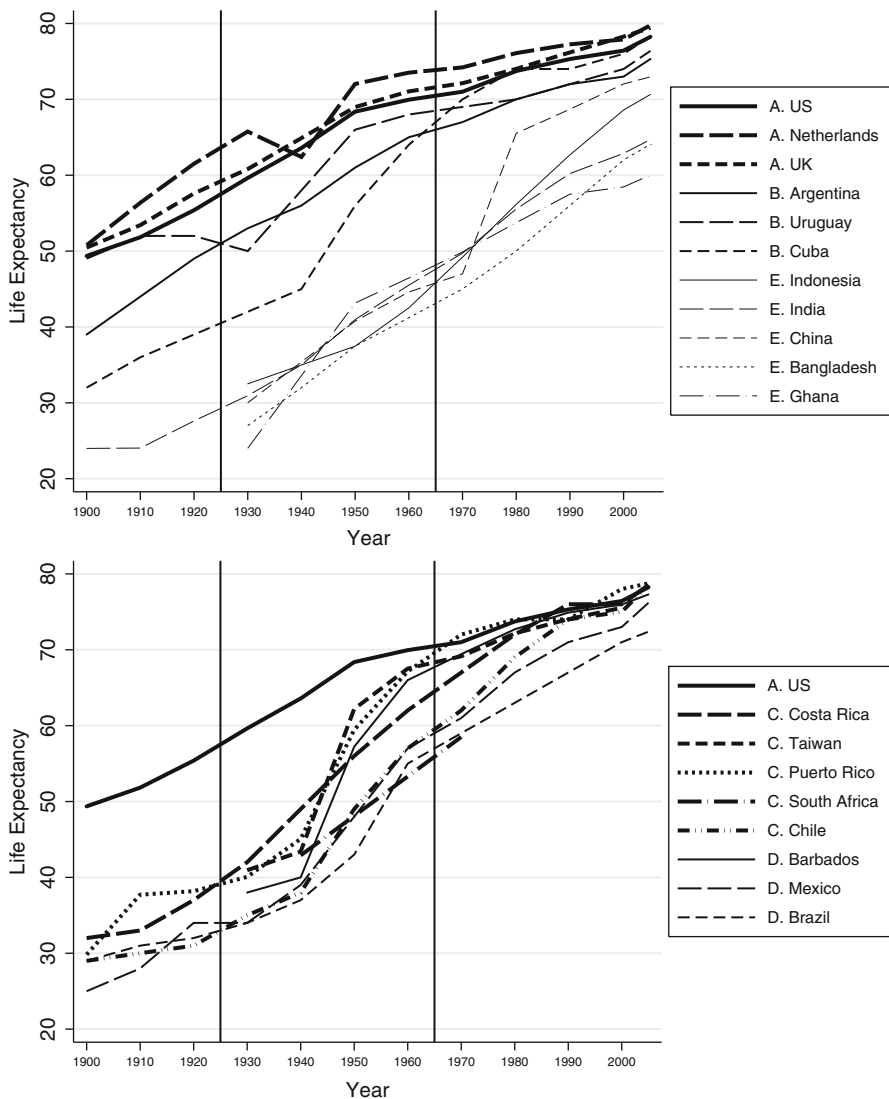


Fig. 1.5 Life expectancy in selected countries and mortality regimes. *Notes:* Shown are patterns of life expectancy (A–E) that reflect different patterns of mortality decline due to the pace, timing and reason for mortality decline during the early to mid twentieth century. Earlier regimes (A–B) experienced an earlier and more gradual mortality decline at higher standards of living; mid and late regimes experienced more rapid decline during the late 1920s through 1940s (C–D) and the very late regimes experienced mortality decline during the late 1940s and 1950s. Vertical lines show the approximate period of interest (*Source:* See [Appendix A](#))

In terms of timing and pace, at one extreme life expectancy increased early before the twentieth century, in high income countries such as England, the Netherlands, and the US (Pattern A), followed by Argentina, Uruguay, and Cuba (Pattern B). Mortality in the Netherlands, and the US had begun to decline in the late 1800s, and in England and Wales this transition was earlier.⁸² By the 1900s the decline appeared as a gradual, steady pattern with increasing levels of life expectancy. Citizens in these countries reached a life expectancy close to 50 years at the beginning of the twentieth century and, throughout the early to mid twentieth century, mortality decline was steady and graded; by 1930, life expectancy had increased to over 60 years. Argentina and Uruguay also experienced a graded improvement in life expectancy at the beginning of the twentieth century.

At the other extreme are countries represented by patterns C–E. While most of these countries experienced large changes in mortality and improvement in life expectancy, the timing of the changes differed. Countries that are now middle-income but at the time were mostly small, poor countries (Pattern C) began to experience more rapidly increasing life expectancy during the late 1920s through the early 1940s, but they began at lower levels of life expectancy. Mexico and Brazil (Pattern D) were very poor large countries which experienced large changes in mortality by the end of the 1940s when public health interventions including medical technology became more widespread in rural areas; in some large urban areas, however, mortality had begun earlier. The most extreme consists of low income countries such as Bangladesh, China, Ghana, India, and Indonesia, which experienced significant mortality decline much later (during the 1950s), and at much lower levels of standard of living (Pattern E).

In terms of the reasons for mortality decline, the large shift in life expectancy during the period of the 1930s through the 1960s has been mostly explained by factors other than the income; these other factors are comprised of public health interventions including medical innovations.⁸³ Even at lower income levels in nations with less growth in national income, some countries experienced a decline in mortality rates. According to some accounts, an estimated 50–70 % of the mortality decline that took place after 1945 in the developing world was associated with medical interventions such as antibiotics and other therapies.⁸⁴ Antibiotics helped reduce mortality due to respiratory diseases such as influenza, pneumonia, and bronchitis but also helped reduce mortality due to measles, diphtheria, whooping cough, and typhoid fever (Table 1.2).

A large fraction of gains in life expectancy occurred early, between birth and age 10.⁸⁵ Thus, accompanying the large increases in life expectancy shown in Fig. 1.5, there were large decreases in infant mortality rates (IMR) and child mortality during

⁸² Riley (2005a) identifies the beginning of the health transition in the US and the Netherlands towards the later part of the nineteenth century. This transition was earlier for England and Wales.

⁸³ Preston (1976).

⁸⁴ Palloni and Wyrick (1981) and Preston (1976).

⁸⁵ Palloni and Wyrick (1981) and Preston (1976).

Table 1.2 Estimated percent of mortality decline in developing countries 1900–1970 due to a particular disease along with strategies for prevention and treatment

	%	Prevention	Treatment	Year treatment developed
Airborne				
Influenza, pneumonia, bronchitis	30		Antibiotics	1945
Respiratory TB	10	Immunization, isolation	Chemotherapy	1947/1948
Smallpox	2	Immunization	Chemotherapy	
Measles	1	Immunization	Antibiotics	1960s
Diphtheria, whooping cough	2	Immunization	Antibiotics	1921
Water, food, and feces borne				
Diarrhea, enteritis, gastroenteritis	7	Better water supply and sewage disposal, personal sanitation	Rehydration	1960s–1970s
Typhoid	1	Better water supply and sewage disposal, personal sanitation, partially effective immunizations	Rehydration, Antibiotics	1949
Cholera	1	Better water supply and sewage disposal, personal sanitation, partially effective immunizations, quarantine	Rehydration	1960s–1970s
Insect borne				
Malaria	13–33	Insecticides, drainage, larvicides	Quinine	1600s
Typhus	1	Insecticides, partially effective vaccines	Antibiotics	1949
Plague	1	Insecticides, rat control, quarantine		

Sources: Adapted from Preston (1980, Table 5.3), Paul (1964), Morley (1973), Hinman (1966). The year treatment was developed was found from various sources including: Achan et al. (2011), Al-Rimawi and Kharaof (2011), Antman (2001), Crofton (1959), Cutler and Meara (2001), Cutts (1993), Guarrant, Carneiro-Filho, and Dillingham (2003), Jayachandran, Lleras-Muney, and Smith (2009), Military Vaccine Agency (2005), Nickel (2005), and Shlaes, Projan, and Edwards (2004).

the 1930s–1960s. Countries in patterns A and B experienced a more gradual, steady pattern of decreasing levels of IMR (Fig. 1.6). Countries in patterns C–E experienced more significant changes, although the timing of the decline was different. The gray shaded area in the figure distinguishes the two different parts of the period:

- Those born in the 1930s–1940s when exposure to disease was being reduced more rapidly in some very poor developing countries.
- Those born in the 1940s–1960s when, in addition to reduced exposure to disease, better treatment of disease was available due to antibiotics and other therapies.

An indication that other factors besides income were responsible for much of the mortality decline of the 1930s–1960s is shown in Fig. 1.7 (see Appendix B for more examples and corresponding figures for IMR). The US (along with England and the

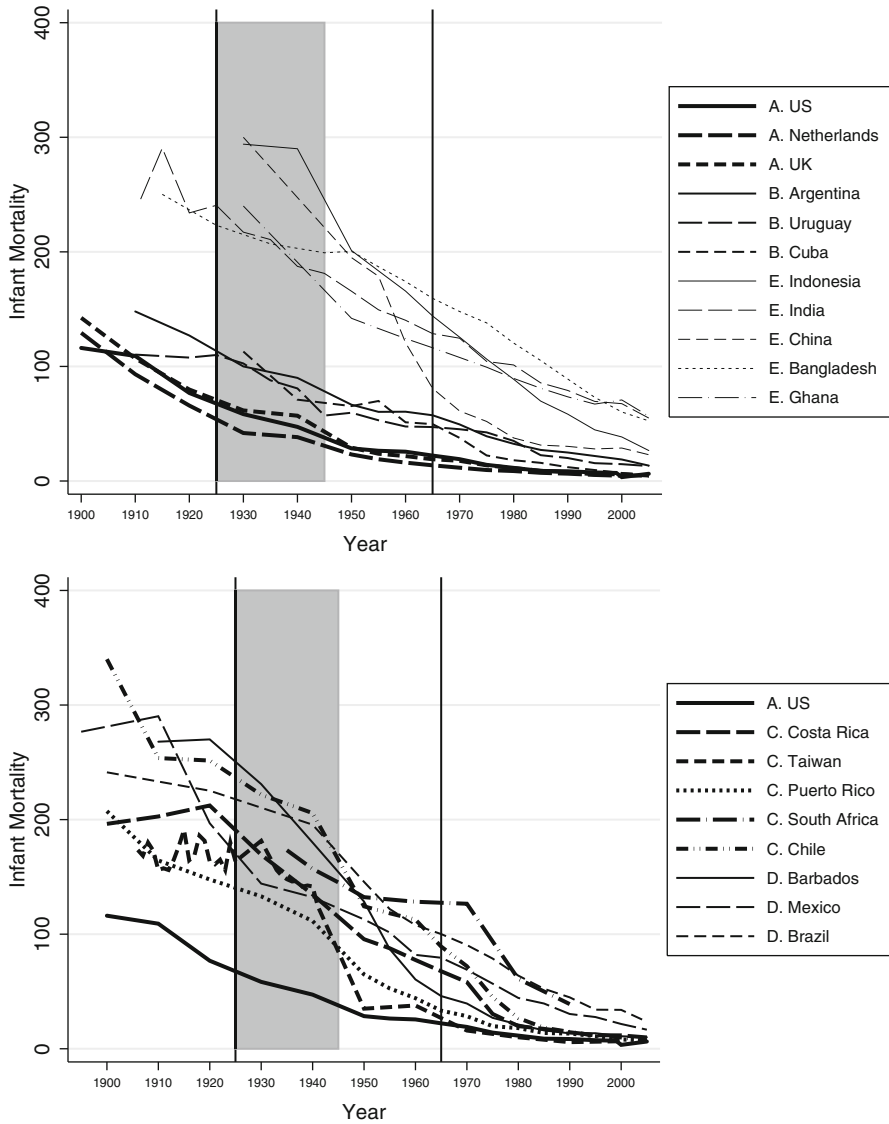


Fig. 1.6 Infant mortality in selected countries and mortality regimes. *Notes:* Countries listed from highest to lowest life expectancy in 1930 by mortality. The shaded gray area from the late 1920s to the early 1940s is the time period of interest. Countries are grouped according to mortality regimes: A Very early, B Early, C Mid, D Late, E Very late. Vertical lines show the approximate period of interest (Source: See Appendix A)

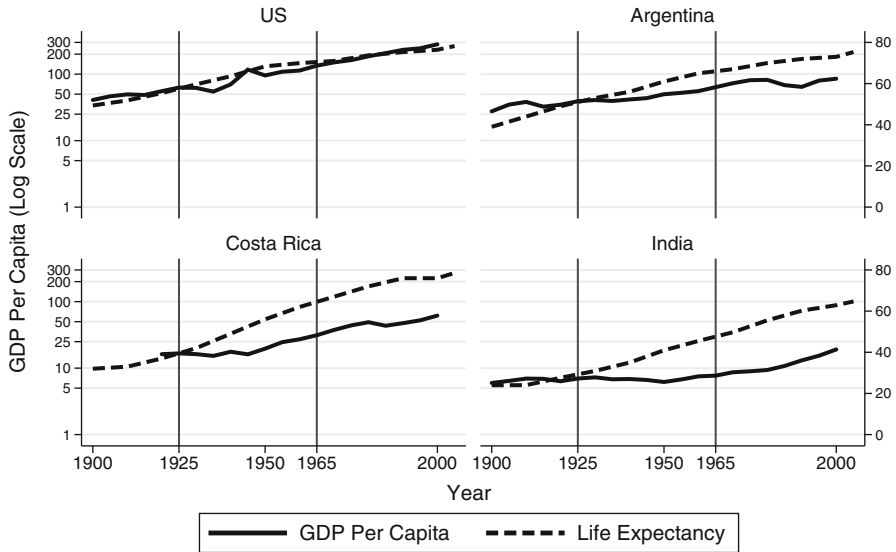


Fig. 1.7 Life expectancy and GDP per capita (1990 per 100 international dollars) from 1900 to 2000. *Note:* Vertical lines show the approximate period of interest (Source: Maddison, 2006; Appendix A)

Netherlands) experienced a graded increase in life expectancy over time. Argentina and Uruguay, which had a better public health infrastructure than most other developing countries,⁸⁶ experienced a more gradual mortality decline at the beginning of the twentieth century, but only at higher levels of GDP per capita. At the beginning of the twentieth century, there was a concerted effort to use sanitary and public health measures to improve infant and child mortality,⁸⁷ and by 1930 a larger proportion of the population of developing countries was in better health at birth as reflected by life expectancy, which was much higher in Argentina and Uruguay, 56–58 years, than in poorer developing countries. Cuba had implemented these major sanitary reforms at the beginning of the twentieth century, which significantly lowered infant mortality;⁸⁸ however, Cuba's pattern was slightly different, although closer to Argentina and Uruguay than other low-income countries.⁸⁹ More infants and children survived during the 1930s–1960s as IMR declined from about 100 to 62 per 1,000⁹⁰; increased

⁸⁶ Diaz-Briquets (1981).

⁸⁷ Birn (2005), Diaz-Briquets (1981), and Mazzeo (1993).

⁸⁸ Diaz-Briquets (1981).

⁸⁹ Cuba is an interesting case because in the early twentieth century its IMR and caloric intakes were similar to that of Argentina and Uruguay but with lower life expectancy (Diaz-Briquets, 1981; Lannoy, 1963).

⁹⁰ McEniry (2009c).

survivorship was more dramatic in countries which started with higher levels of infant and child mortality and lower levels of GDP per capita. Across countries experiencing later mortality decline, there was a more rapid increase in life expectancy after the 1940s when antibiotics and other therapies brought about dramatic improvements in infant and child mortality.

In some poor, smaller countries it is clear that factors other than GDP per capita were strongly associated with improvements in life expectancy and IMR even before the advent of antibiotics and other therapies after the 1940s (Fig. 1.7). In countries such as Costa Rica, Puerto Rico, Chile, and Taiwan, sustained mortality decline appeared as public health efforts began to intensify in the late 1920s and early 1930s, in spite of continued difficult economic times and low levels of income.⁹¹ In Costa Rica, GDP per capita was fairly steady during the 1920s–1940s, but life expectancy increased and IMR declined. Efforts during the late 1920s–early 1930s were preventive in nature and included educational campaigns, the development of public health units, malaria, and hookworm campaigns in tropical countries, vaccinations for small pox, building latrines, and improving laws to regulate public health. Puerto Rico, for example, started to implement public health units at the municipal level during the 1920s; by the end of the 1930s, most of the island was covered. IMR went from about 133 to 43 per 1,000 in Puerto Rico and from 232 to 132 per 1,000 in Costa Rica as more widespread public health interventions were implemented in the 1920s and 1930s. In Taiwan, mortality declined earlier because of foreign interventions.⁹² In South Africa, public health efforts also intensified in the early 1920s, although there were important differences in access for whites and blacks.⁹³

In larger countries such as Brazil and Mexico (Appendix B), which experienced improvements in life expectancy in the early twentieth century,⁹⁴ more concerted public health efforts on the part of the government began by the 1920s; the efforts improved urban areas but did not reach much of the rural population until the late 1930s–1940s.⁹⁵ Barbados, a small island, was able to implement major sanitation and public health measures beginning in the 1920s; strong public education and health reforms produced dramatic mortality decline beginning in the 1930s.⁹⁶

Countries which are now mostly low-income, such as India, Indonesia, and Bangladesh lowered IMR to levels below 200 per 1,000 much later (the 1940s or 1950s) as a result of large scale public health interventions and changes in medical technology. There were slight improvements in life expectancy beginning in the 1920s, but even though mortality began to decline in countries such as China, India,

⁹¹ Garnier et al. (1997), Jiménez de la Jara and Bossert (1995), Palmer (2003), Rigau Pérez (2000), and Rosero-Bixby (1991).

⁹² Barclay (1954) wrote extensively on Taiwan in the early twentieth century.

⁹³ Beinart and Dubow (1995).

⁹⁴ Riley (2005b) contains a comprehensive description of timing of mortality transitions.

⁹⁵ Rodríguez de Romo and Rodríguez de Pérez (1998).

⁹⁶ Bishop et al. (1997) and West India Royal Commission (1945).

Bangladesh, Ghana, and Indonesia in the 1930s,⁹⁷ economic development was slower and the type of public health interventions that could benefit a large proportion of the population didn't develop until the end of the 1940s and into the 1950s. Indonesia's GDP per capita declined along with IMR during the 1950s. In China, GDP per capita was stagnant until the 1940s when it declined; it increased in the 1950s while IMR continued to decline.

Later cohorts (late 1940s–1960s) experienced a greater increase in survivors of poor early conditions than did earlier cohorts because of medical interventions such as antibiotics and other therapies. However, even in the 1930s and early 1940s, more infants and children began to survive in urban areas. China had established a public health infrastructure affecting some urban areas,⁹⁸ and there were some targeted public health efforts during the Dutch rule in Indonesia.⁹⁹ This also happened in India, where there were some limited public health measures in the cities in the early twentieth century during British rule¹⁰⁰ and where conditions in the state of Bengal, India (a portion of which later became Bangladesh) were reported to have been better than in other parts of India.¹⁰¹ Finally, in Ghana, modern public health efforts began to slowly improve in the 1920s, mostly in urban areas, but did not have a wide impact in rural areas until in the 1940s and 1950s after colonial rule ended.¹⁰² In these countries, improvement occurred mostly after the introduction of antibiotics and other therapies (after 1945), for the most part with stagnant economic growth.

1.4.3 Continued Stagnant Economic Conditions and Poor Nutrition and Risk of Infection

In the developed world, the focus on reducing disease during the early twentieth century may have been effective because of the higher standard of living and concomitant improved nutrition and nutritional status of the general population. However, with stagnant economies and lower standards of living in the 1930s–1940s, most of the population in the developing world was still exposed to chronic malnutrition in early childhood, especially in rural areas. Even with economic growth in some countries the distribution of income may not have led to parallel increases in standard of living across all population groups.¹⁰³

⁹⁷ Riley (2005b).

⁹⁸ Banister (1987), Campbell (1997), MacPherson (2008), and Xizhe and Zhigang (2000).

⁹⁹ Hull (2008), Nitisastro (1970), Pardoko (1984), and Widjojo (1970).

¹⁰⁰ Dyson (1997), Guha (2001), and Ramasubban (2008).

¹⁰¹ Guha (2001) and Khan (1984).

¹⁰² Patterson (1979, 1981).

¹⁰³ See for example López-Alonso (2007).

Table 1.3 Daily caloric supply per capita in selected countries across time

Country ^a	Pre-WWII	Post-WWII				
	1934–1939	1948–1950	1951–1953	1954–1956	1957–1959	1964–1969
US	3,280	3,180	3,150	3,150	3,110	3,300
England	3,110	3,130	3,110	3,260	3,280	3,180
Netherlands	2,840	2,930	2,900	2,940	2,890	3,200
Argentina	2,780	3,240	2,980	3,070	3,090	3,160
Uruguay		2,900	2,950	2,960		2,740
Cuba	2,610	2,730				2,500
South Africa	2,340	2,640	2,690	2,610	2,640	2,730
Chile	2,250	2,370	2,430	2,540	2,570	2,560
Brazil	2,190	2,180	2,340	2,600	2,680	2,820
Mexico				2,380	2,440	2,620
Taiwan	1,870	1,980	2,140	2,210	2,330	
India	1,950	1,630	1,700	1,840	1,900	1,990

Sources: Data for the years 1934–1939 and Cuba come from United Nations Statistical Office & the Department of Economic & Social Affairs (1960). All other data prior to the 1960s come from United Nations Statistical Office & the Department of Economic & Social Affairs (1962). Data for the 1960s comes from United Nations Statistical Office & the Department of Economic & Social Affairs (1973)

Notes: The numbers are different from Table 1.1 because they were computed at slightly different points in time. The years for which data are reported vary slightly by country. For instance, Argentina reports data from 1935 to 1939 while Cuba reports data from 1934 to 1938. The caloric supply data for these countries are then reported in the column “1934–39.” This is also true for other columns in the table

^aThe grouping of countries is roughly according to their level of caloric intake pre-WWII

The public health interventions including advances in medical technology during the 1930s–1960s reduced exposure to disease and improved health, but did not directly address deficiencies in nutrition. The synergistic relationship between infectious diseases and nutrition was not completely realized until after World War II,¹⁰⁴ and interventions to supplement household income and nutritional needs in early life came much later in the twentieth century.¹⁰⁵ This may partially explain why nutritional status in the developing world may not have improved as it did in the developed world for those born during this period.¹⁰⁶

Daily caloric supply per capita across some developing countries did not dramatically change during the 1950s or even into the 1960s (Table 1.3). With the exception of Argentina, Uruguay, and possibly Cuba, daily caloric supply per capita was very low in countries which are now low and middle income countries. As suggested by Table 1.3 and Fig. 1.6, nutrition was worse at higher IMR levels in

¹⁰⁴ Fogel (2004).

¹⁰⁵ Almond and Currie (2010).

¹⁰⁶ Godoy et al. (2007), López-Alonso and Porrás Condey (2003), and Meisel and Vega (2007).

developing countries. With low levels of caloric intake also came the risk of infection among families who could not afford proper nourishment.

1.4.4 Growth Rates and Implications of the Demographic Past

Growth rates of a population are partially a function of the demographic past in terms of fertility and mortality changes.¹⁰⁷ The rapid growth of the aging population in the developing world is thus directly tied to the rapid infant and child mortality decline in the 1930s through the 1960s from public health interventions including medical technology.¹⁰⁸ In some parts of the Latin American and the Caribbean (LAC) region, a substantial proportion of the growth rate of the older adult population during this period may have been due to cumulative mortality changes before the age of 60, most of which were concentrated at younger ages (infancy and young childhood).¹⁰⁹ However, the magnitude of the pattern varies across countries according to the timing, pace of, and reason for the mortality decline—higher cumulative mortality changes among those countries where significant and rapid mortality decline occurred during the 1930s–1960s.

The growth rates shown in Fig. 1.1 between the 1990s and the 2020s reflect mortality and fertility changes for those born during the 1930s through the 1960s and the dramatic changes in infant and child mortality in some developing countries during the 1930s–1960s (Fig. 1.6). If indeed a large proportion of this growth rate can be attributed to the cumulative mortality changes which occurred as a result of reduction in infant and child mortality in the 1930s–1960s from public health including medical innovations, then the implications are twofold:

- A large percentage of those born during the 1930s–1960s in some developing countries who survived until the first decade of the twenty-first century also survived poor early life conditions;
- Differences in magnitude of the cumulative mortality changes across countries during the 1930s–1960s are a reasonable way to ascertain the degree to which cohorts are heavily characterized by survival of poor early life conditions.

1.4.5 Unique Cohorts of the 1930s–1960s and Tip of the Iceberg Countries

An argument can be made that the historical circumstances of the 1930s–1960s produced unique cohorts characterized by increasing survival of infants and children mainly due to public health interventions including medical technology

¹⁰⁷ Horiuchi and Preston (1988).

¹⁰⁸ Preston (1976) and Palloni et al. (2007).

¹⁰⁹ Palloni et al. (2007).

often in the context of stagnant economic conditions, so they were still exposed to poor nutrition and infectious diseases.¹¹⁰ The particular nature of the subsequent dramatic mortality decline (i.e., pace, timing, and cause) produced individuals in some low- and middle-income countries who were at risk of having been affected by harsh early childhood experiences but, simultaneously, large probabilities of survival due to their exposure to more widespread deployment of medical technologies and public health measures during the period after 1930. This cohort was less affected by early mortality-driven selection than the group of cohorts that preceded them; however, they may be less robust because they are increasingly characterized by their survival of poor early life conditions. An increasing number of surviving children would have increased demands on poor families who already struggled to provide adequate nutrition.

Any of the early life mechanisms described earlier in the chapter could help predict health at older ages: A higher proportion of individuals who survived poor early life conditions might translate into a higher proportion of individuals who are in poor health at older ages. The focus in the coming chapters is on older adult heart disease and diabetes—projected to be important health concerns in the developing world. The nutrition argument relating to adult heart disease and diabetes by Barker and colleagues (the Barker hypothesis) continues to be of interest although, as noted earlier, it is not the only potential explanation for *in utero* exposures associated with adult health. The synergy between nutrition and infection are important.¹¹¹ Given the importance of major dietary changes and a transition to a more sedentary lifestyle in the last 30 years or so,¹¹² there is also particular interest in the claim accompanying this hypothesis that living conditions and other life course events do not confound the effects of early life exposure to poor nutrition but compound them.¹¹³

Those born in the unique cohorts after 1945 when most of the mortality changes occurred have not yet reached the age of 60. However, older adults in the unique cohorts born prior to 1945 have already reached the age of 60. This includes adults born in poor countries previously described by Patterns C–D (Fig. 1.5) which in the early to mid twentieth century experienced significant mortality decline prior to antibiotics and other therapies which came after the late 1940s.

Older adults born in Pattern C and to some extent Pattern D countries prior to 1945 represent the first of a burgeoning wave of adults who have increasingly survived poor early life conditions due to reduction in disease but who nevertheless continued to be exposed to poor economic conditions. These mostly now upper-middle-income countries in which these older adults were born during the late 1920s through early 1940s could be considered tip of the iceberg countries because

¹¹⁰ Palloni et al. (2007).

¹¹¹ Crimmins and Finch (2006).

¹¹² Popkin et al. (2001).

¹¹³ Barker et al. (2001) and Barker (1995).

of this growing elderly population. Previous work set the foundation for developing the concept of tip of the iceberg countries.¹¹⁴ The tip of the iceberg countries included:

- Smaller and poor developing countries that were able to implement widespread reforms in public health prior to 1945 amidst stagnant economic conditions (e.g. Puerto Rico, Costa Rica, Chile);
- Urban areas in some larger poor developing countries which began to experience significant mortality decline prior to 1945 (e.g. Mexico, Brazil).

Older adults born during this period from very late regimes (Pattern E) were born in severe mortality regimes with high infant and child mortality whereas older adults born in the Patterns A–B countries experienced a more graded mortality decline at higher income levels.

A comparison of older adult health across different mortality regimes of the early to mid twentieth century with a particular focus on older adults from the tip of the iceberg countries may possibly illuminate the merit of the conjecture and help ascertain the degree to which those born after 1945 will be affected or not. This assumes that it is possible to obtain adequate measurement of the key components of health and early life conditions and control for confounding factors such as differences in economic growth and development, increasing urbanization, migration, and exposure to different events throughout the life course.

1.5 The Intriguing Case of Puerto Rico

Puerto Rico helps further illustrate the historical circumstances of mortality decline in the early to mid twentieth century. It is a compelling case study because there are readily available detailed historical accounts regarding health and economic conditions on the island in the early twentieth century which complement survey data on older adults.¹¹⁵ Puerto Ricans themselves present a unique mixture of US, Latin American, and Caribbean cultures. While differences may exist in disease etiology, a large proportion of older adults born in tropical Puerto Rico during the early twentieth century experienced similar early life circumstances and demographic forces to those in other developing countries.¹¹⁶

¹¹⁴ Palloni et al. (2006).

¹¹⁵ Mostly from annual reports from the Health Commissioner of Puerto Rico, published articles in Puerto Rican public health journals, and published books during the early twentieth century, but also from Ayala and Bernabe (2007) and Steward et al. (1956) who provide a broader context of the events that occurred during the 1930s–1940s in Puerto Rico.

¹¹⁶ Palloni et al. (2005).

1.5.1 Standard of Living

In the early to mid twentieth century, Puerto Rico, like other developing countries, experienced very low levels of economic development (Fig. 1.4). In the 1930s, GDP per capita in Puerto Rico was about 50 % lower than GDP per capita in the US, England, and the Netherlands. In the first part of the period between the 1930s–1960s, which produced dramatic improvements in life expectancy, economic growth was mostly stagnant in Puerto Rico but as the country began to industrialize after 1945, GDP per capita improved significantly. However, inequalities in the distribution of income may not have led to equal increases in standard of living across all population groups.

Like many countries in the developing world, Puerto Rico's population was predominantly rural and dependent on agriculture prior to the late 1940s. Agricultural employment was thoroughly dominated by the sugar cane industry. In 1929, over half (52 %) of the total acres of sugar cane, tobacco, and coffee for the international market (the main cash crops in Puerto Rico) were devoted to sugarcane, compared with 36 % for coffee and 12 % for tobacco; sugar cane made up almost 99 % of the total tons of production of these three crops.¹¹⁷ Sugar cane was predominant in the coastal regions of Puerto Rico while tobacco and coffee were found inland. More than half of the population in 1930 (56 %) lived in sugar producing areas.

Slightly over 70 % of the population lived in rural areas in 1930 according to the US census.¹¹⁸ The mostly rural population was chronically malnourished and exposed to serious infectious diseases such as malaria and hookworm.¹¹⁹ In 1930, about 66 % of male wage earners worked in agriculture and, of those, 81 % were farm laborers.¹²⁰ The illiteracy rate was high (45 %)¹²¹ although there were differences among municipalities (*municipios*) with literacy rates ranging between 20 and 57 %.¹²² Illiteracy did not significantly improve until after the mid-1940s in Puerto Rico and, for the most part, educational opportunities in rural areas were limited. Education was often restricted to 3 years, after which many children left school to return to work in the fields.¹²³

¹¹⁷ US Census Bureau (1932).

¹¹⁸ The US census (US Census Bureau, 1932) defined “rural” as communities with fewer than 2,500 inhabitants. Using the US census definition, 78 % of Puerto Rico's population was identified as rural in 1920 and about 72 % in 1930. Only the municipalities of San Juan, Bayamón, and Ponce had predominantly urban populations in 1920. By 1930, Cataño and Mayagüez had become predominantly urban as well although Bayamón became more rural than urban.

¹¹⁹ Allen and Hunt (1914), Ashford and Gutiérrez Igaravidez (1911), Clark (1930), Mountin, Pennell, and Flook (1937), and Rigau Pérez (2000).

¹²⁰ US Census Bureau (1932).

¹²¹ Clark (1930).

¹²² Today, Puerto Rico is divided into 78 *municipios* governed by an elected mayor and legislative assembly. The current location of *municipios*, for the most part, conforms to how Puerto Rico was divided in the late 1920s-early 1940s.

¹²³ Rosario (1930, page 565).

Puerto Rico had one of the highest population densities in the world during the early twentieth century.¹²⁴ Reputable surveys of the day point to overcrowding in both urban and rural areas.¹²⁵ Tuberculosis was a major health concern during the early twentieth century, especially in urban areas; public health officials described overcrowding as one of the major reasons.¹²⁶

Socioeconomic surveys of sugar cane, tobacco, and coffee plantations and US census data provide an indication of the general characteristics of rural housing conditions for field workers and their families.¹²⁷ Housing in rural areas for “peasants” was of poor quality. The average reported size of a house ranged between 250 and 500 square feet, with an average of five to six persons sharing the space; an estimated two thirds of houses in some areas did not have latrines.¹²⁸ In some cases, employers provided a house free of charge, and many families did not own their own homes. Data from the US census show that home ownership in some municipalities was lower than 50 %.¹²⁹

There were large discrepancies between municipalities in Puerto Rico in the early 1930s in terms of wealth, literacy, access to health care, per capita spending on public health and the quality of the water treatment system (Table 1.4).¹³⁰ The most affluent municipalities (6 % of all municipalities and 24 % of the total population) had lower rates of illiteracy, better access to medical care, and better water treatment. Sixty four percent of the municipalities were classified as least affluent according to their population, assessed value, and government income. Almost 45 % of the total population and 48 % of children under the age of 5 years lived in the least affluent municipalities, which were mostly rural and had high illiteracy rates; only 15 % of the hospitals and 19 % of the physicians in Puerto Rico were located in these municipalities. Average per capita spending on public health was low in the less wealthy areas. Water treatment facilities were almost non-existent in the least affluent municipalities.

1.5.2 Diet and Nutrition

The Puerto Rican diet was deficient compared to that of mainland US during the early twentieth century (Table 1.5). Puerto Ricans consumed less meat, eggs, milk, cheese, fresh fish, green and leafy vegetables, and wheat, and consumed more rice, beans, and starchy vegetables (e.g. sweet potatoes). If the overall Puerto Rican

¹²⁴ Bureau of Vital Statistics (1926).

¹²⁵ Clark (1930), Morales Otero and Pérez (1939), Morales Otero et al. (1937, 1939).

¹²⁶ Clark (1930).

¹²⁷ Morales Otero and Pérez (1939), Morales Otero et al. (1937, 1939).

¹²⁸ Rosario (1930, pages 549 and 551).

¹²⁹ US Census Bureau (1932).

¹³⁰ Clark (1930, pages 123–124), Fernós Isern (1932), Mountin et al. (1937) and US Census Bureau (1932).

Table 1.4 Population, access to medical care, and quality of water by wealth of municipality in the 1930s in Puerto Rico

	Most affluent (class 1 ^a)	Middle (class 2 ^a)	Least affluent (class 3 ^a)	Total number
Population				
Municipalities (%)	6	30	64	77
Population (%)	24	33	44	1,543,506
Population <5 years of age (%)	20	32	48	226,468
Rural population (%)	11	34	55	1,116,677
Education				
Illiterate (%)	20	33	47	450,729 ^b
Medical care				
Hospital beds (%)	50	35	15	3,533
Physicians (%)	51	30	19	450
Mean expenditure per capita spent on charity (public welfare)	\$1.10	\$0.70	\$0.29	
Water treatment system^c				
Acceptable (%)	23	36	41	22
None (%)	0	27	73	55
Public Health Units^d				
Operating in 1929 (%)	17	50	33	6

Sources: Municipal class used in Puerto Rico in 1930 to classify municipalities was described by Clark (1930, pages 123–124). Population data are from the United States Census Bureau (1932). Access to medical care data are available in Mountin et al. (1937). Water data are available in the Report to the Commissioner of Health of Porto Rico for fiscal year 1930–1931 (Fernós Isern, 1932).

Notes:

^aMunicipalities were grouped into three administrative classes in the 1930s according to their population size, assessed value, and government income. The five municipalities that comprised class 1 included: Arecibo, Caguas, Mayagüez, Ponce, and San Juan. There were 23 municipalities in class 2 and the remaining 49 municipalities were in class 3

^bThere were a total of 1,093,423 individuals 10 years and older in the 1930 census (the number of illiterate persons includes only those 10 years and over). Of these, 450,729 (41 %) were identified as illiterate

^cThe best water treatment system available in Puerto Rico in the early twentieth century was sedimentation, coagulation, filtration, and chlorination. Other acceptable options included liquid chlorine, rapid gravel, and chlorination, and the emergency use of chlorine. Some municipalities had no water treatment system at all

^dThose with public health units in 1929 included Caguas (class 1), Cayey (class 2), Río Piedras (class 2), Yabucoa (class 2), Adjuntas (class 3), and Cataño (class 3)

population was nutritionally deficient, it was worse for those living in rural areas.¹³¹ Cook, Axtmayer, and Dalmau¹³² analyzed the chemical composition and nutritive values of three typical diets in Puerto Rico and found that a rural diet in particular

¹³¹ Rosario (1930, page 563).

¹³² Cook, Axtmayer, and Dalmau (1943).

Table 1.5 Yearly per capita consumption of foods in Puerto Rico and selected areas of the United States (in pounds)

Class of food	Puerto Rico	North Atlantic	South East	
		(843 white families)	Central (282 white families)	Southern cities (222 black families)
Total, all foods	982.6	1,289.0	1,145.7	886.1
Meats	33.4	113.6	70.5	85.6
Eggs	4.4	32.9	33.2	15.7
Milk (fresh)	67.7	261.4	151.8	24.8
Milk (processed)	14.3	31.2	98.0	58.2
Cheese	1.6	7.7	4.6	2.3
Butter	0.4	21.0	8.4	6.6
Fats	20.0	22.8	54.3	63.4
Fish	21.7	20.9	12.2	40.2
Codfish	16.0	–	–	–
Fish, fresh	2.5	12.9	4.7	23.4
Green and leafy vegetables	58.4	155.4	170.1	136.3
Rice	144.6	3.9	4.4	15.1
Wheat	51.0	177.9	149.5	123.4
Beans	29.2	4.3	8.8	8.2
Starchy vegetables	283.7	156.8	100.5	91.4
Fruits and nuts	134.8	150.2	134.5	63.6

Source: Suárez (1943, Table 1)

was deficient in various essential nutrients, one of which was Vitamin A.¹³³ Vitamin A deficiency can increase the risk of maternal mortality, poor outcomes at birth or during infancy, reduce the growth rate, and can lessen the body's ability to fight infection leading to higher fatality rates for infectious diseases and increased mortality rates.¹³⁴ Case studies also point to riboflavin, niacin, and Vitamin C deficiencies in preschool-aged children.¹³⁵

Several other studies were conducted during the early twentieth century which give an indication of the nutritional deficiency of the population, especially children. Although these studies were small in scope, they echo the widely-reported

¹³³ Cook et al. (1943) examined three typical diets in Puerto Rico: continental (Puerto Ricans that followed US dietary habits); caterer (urban areas); and country (rural areas). A typical continental diet included: juice, cream, sugar, coffee (breakfast); egg and toast, sugar, milk, banana, iced tea, lime juice (lunch); avocado, plantain, fried cabbage, potato cake, fried pork chops, cream, butter, sugar, pineapple ice cream, cake, coffee (dinner). A typical urban diet included chicken meat, potatoes and gravy, macaroni, polished rice, red kidney beans (lunch) and fried bread meat, stewed eggplant, polished rice, and rice soup (dinner). A typical rural diet included polished rice, red kidney beans, pork, onions, chick peas, salted cod fish, olive oil, annatto seeds, lard, salt, sugar, garlic, black pepper, yautías (a root), sweet potato, tomatoes, and pigeon peas.

¹³⁴ WHO (2012).

¹³⁵ Robinson and Suárez (1947).

observations by public health officials of the day for much of the population, especially those in rural areas.¹³⁶ Measurement of over 7,000 school children in the 1920s found that, on average, Puerto Rican children were shorter and lighter than children of similar age in mainland US¹³⁷ In Puerto Rican communities with higher living standards, children were taller and heavier whereas in communities where a high percentage of the population was infected by hookworm, children were shorter and lighter. Measurements of over 2,700 children in the late 1920s found that a high percentage of boys and girls between the ages of 6 and 16 were underweight.¹³⁸ In another study of 157 children and adults from a rural community close to a main highway that had a grocery store, the average intake of dietary essentials (e.g., calories, protein, and calcium) was low for everyone, but lower for children than adults, and a large percentage of preschool children showed signs of malnutrition.¹³⁹

1.5.3 Providing for One's Family

The strong cyclical nature of employment due to the dominance of the sugar cane industry most probably affected a family's ability to obtain proper nourishment. Employment on the island was highest during the first 6 months of the year, the harvest season for sugar cane; this was followed by a lean season lasting most of the second half of the year. A large proportion of Puerto Ricans were unemployed or underemployed during extended periods of the year, and, as previously described, many of them lived in rural areas where conditions were precarious.¹⁴⁰ As many as 34 % of males of wage earning age were unemployed or underemployed at some time during the year¹⁴¹; because Puerto Rican society was primarily rural, much of this unemployment or underemployment occurred in rural areas.

Although the temperature may not have greatly varied between seasons, the period of highest rainfall coincided with the lean season for the sugar cane industry.¹⁴² The hurricane season also peaked between August and October, bringing with it a long

¹³⁶ See annual reports to the health commissioner of Puerto Rico (Fernós Isern, 1932, 1933, 1946, n.d.; Garrido Morales, 1935, 1936, 1937, 1939, 1940, 1941; Ortiz, 1927, 1929, 1931), Clark (1930), and Gayer, Homan, and James (1938).

¹³⁷ Bary (1923).

¹³⁸ Payne, Berríos, and Martínez Rivera (1929).

¹³⁹ Robinson and Suárez (1947).

¹⁴⁰ Rosario (1930).

¹⁴¹ Clark (1930, page 50).

¹⁴² Food and Agriculture Organization of the United Nations (1985) Agroclimatological Data for Latin American and the Caribbean. Average annual precipitation for Puerto Rico: 1471 mm (57.91 in). Maximum precipitation: 162 mm. Minimum precipitation: 62 mm. Average annual temperature: 25.6 °C (78.1 °F). Maximum temperature: 30 °C. Minimum temperature: 21.2 °C.

period of hot and humid weather, ideal conditions for infectious diseases such as dysentery, diarrhea, malaria, and dengue fever.¹⁴³ These climactic conditions coincided with the lean season for work, when people had less money to purchase good food, so poor nutrition was combined with increased exposure to infectious diseases.

Thus, providing an adequate nutritional environment for one's family was difficult in Puerto Rico, especially for farm laborers who comprised a large proportion of the population. Because many rural families, especially those in sugar cane producing areas, did not have a garden plot nor did they own much livestock, wages were the most important means of purchasing food and providing a proper diet for one's family. Wages were low: The typical rural agricultural worker earned less than US\$1.00 a day in 1930 or approximately US\$182–\$207 per year,¹⁴⁴ and most did not work full time throughout the year. Most of the daily wages went to food with little left over, as surveys of socioeconomic conditions on sugar, tobacco, and coffee plantations show.¹⁴⁵ When the worker did not work the full day, he could purchase only part of that day's food. Puerto Rico imported staple food items such as rice from the US, which made it expensive. With these wages, there is little to suggest families were able to save and plan for hard times. Environmental conditions were substandard¹⁴⁶ which compounded the problem of poor nutrition. There were no uniform programs to augment nutrition or income for Puerto Rican families during the early twentieth century—programs which may have mediated the effects of early life conditions.¹⁴⁷ Wages were generally higher in the sugar cane regions, but there few alternative sources of employment to supplement income during the lean season. Puerto Rican farmers (*colonos*) who produced sugar cane in the northern part of the island may have helped their workers

¹⁴³ Rigau Pérez (2000).

¹⁴⁴ Clark (1930).

¹⁴⁵ Morales Otero and Pérez (1939) and Morales Otero et al. (1937, 1939). A US dollar per day in 1930 was not enough to sustain a living for a family. Rosario (1930, page 563–564) provides several examples of the purchasing power of earning less than \$1 per day: (1) A coffee farm with a full time worker making 60 cents per day with his wife and eight children. The 60 cents were sufficient to buy food for lunch (3 lb of corn meal, half of a pound of cod fish, salt) and supper (two and a half pounds of rice, beans, salt pork, sugar) with eight cents left over which had to be saved for Sunday purchases of food when the man was not working. If he worked only a half of a day he could only buy lunch and no supper for the family. (2) In contrast, because wages in the sugar region were higher, a family of four in the sugar region with 75 cents per day could purchase for a single meal 4 lb of sweet potatoes, olive oil, sugar, codfish and coffee leaving an average of 44 cents for other meals and for Sunday; a family of five in the sugar region could potentially have a reserve of 46 cents left for other meals and for Sunday after purchasing a pound of rice, lard, cigarettes, two pounds of sweet potatoes, and coffee; and a family of eight with similar wages spent 35 and a half cents on food for a single meal with the remaining for purchasing food for other meals and for Sunday.

¹⁴⁶ Clark (1930), Morales Otero et al. (1937, 1939); Morales Otero and Pérez (1939), and Pérez (1941).

¹⁴⁷ Almond and Currie (2010).

supplement their nutritional needs during the lean period. This probably did not occur in the larger sugar cane plantations in the southern part of the island, but there may have been opportunities for some to work in the mills during the lean period. In some cases, employers provided rent-free housing for workers,¹⁴⁸ and in some areas workers received coffee with milk and lunch, which would have helped working fathers but not mothers at home. For the most part, however, favorable circumstances were available only among those employed in the large sugar plantations along the southern coast where mostly men worked, where mothers and children did not actively participate in the harvest, and where there were fewer opportunities to own land or have a garden and livestock.¹⁴⁹

There were more opportunities for coffee and tobacco workers to supplement their income, including participating in the sugar cane harvest.¹⁵⁰ Mothers and children worked in the coffee and tobacco harvest, and they may also have had other opportunities to earn extra money. It was also common for families in the coffee region to be given land to grow food for household consumption such as fruit (bananas or oranges) and have a few animals (chickens, cows, and/or goats) that could supplement household nutrition. Some families received an allotment of bananas per day when available in the coffee region. However, wages were lower in the coffee and tobacco regions compared with the sugar region. A few areas may have been helped by non-profit organizations, but these efforts were not widespread.

Family networks may have improved to some extent the nutritional status of rural families because extended families shared limited resources such as food. However, it is difficult to make an objective assessment of the impact of family networks given there are few historical accounts.¹⁵¹ Rural families tended to be poor, scattered and isolated (especially in the mountainous regions), with weaker formal social organizations. Many couples were not married, leading some to claim that these “irregular ties” reflected weaker family structures and individual attachments.¹⁵² While nuclear families were more common in rural areas,¹⁵³ there is no indication these families were any less cohesive than urban families. The numerous descriptions of socioeconomic conditions in sugar, coffee, and tobacco plantations in the late 1920s and early 1930s suggest difficult socioeconomic conditions for most workers which certainly impacted their ability to provide a suitable environment in which to raise a family.¹⁵⁴

¹⁴⁸ Although only a small percentage of housing was provided rent free (Rosario, 1930, page 554).

¹⁴⁹ Clark (1930).

¹⁵⁰ Clark (1930).

¹⁵¹ Scarano and White (2007).

¹⁵² Clark (1930).

¹⁵³ Scarano and White (2007).

¹⁵⁴ Morales Otero and Pérez (1939) and Morales Otero et al. (1937, 1939).

1.5.4 Infectious Diseases in Mothers, Infants, and Children in Puerto Rico

The serious diseases of the early twentieth century which produced high mortality and morbidity rates in Puerto Rico and many other tropical countries were malaria, hookworm, and tuberculosis.¹⁵⁵ However, public health reports of the early to mid twentieth century in Puerto Rico focus particularly on infants and mothers. About 26–27 % of all deaths were children under the age of one during the late 1920s.¹⁵⁶ Overall, infant mortality was above 100 per thousand during this period and the under-5 mortality rate was also high.¹⁵⁷ All of these early life indicators were much worse than those on the United States mainland and in other tropical countries such as England and Wales.¹⁵⁸ High temperatures year round in tropical Puerto Rico nurtured infection and resulted in generally higher IMR than those of northern climates.

There were four major reported causes of IMR during the late 1920s–1940s, some of which are also associated with poor nutrition.¹⁵⁹ Table 1.6 describes the causes of IMR in the year 1934: diseases of the digestive system (about 35 % of deaths), of which diarrhea and gastro-enteritis were predominant; diseases of early infancy (about 24 %), of which congenital disability predominated; diseases of the respiratory system (about 18 %), of which acute bronchitis was most common; other infectious diseases (about 16 % of cases).

Factors impacting IMR differed with the age of the infant. Congenital disability was the most common cause of death during the first month of life; diarrhea, acute bronchitis, and other infectious diseases were predominant causes of death during post neonatal life (1st–11 months). Poor economic and environmental conditions which affected the mother, the unborn child and the infant, together with a largely illiterate and uneducated population with little knowledge of preventive health measures probably compounded the problem.

There was a strongly held belief among public health officials of the day that even though there were difficulties in the reliability of cause of death data, feeding

¹⁵⁵ Farley (2004).

¹⁵⁶ Fernós Isern (1932) and Ortiz (1931).

¹⁵⁷ See annual reports to the health commissioner of Puerto Rico (Fernós Isern, 1932, 1933, 1946, n.d.; Garrido Morales, 1935, 1936, 1937, 1939, 1940, 1941; Ortiz, 1927, 1929, 1931).

¹⁵⁸ In 1924–1925, IMR, neonatal and post neonatal mortality rates in Puerto Rico were 148.6, 43.3, and 105.4 per 1,000 live births, respectively. Neonatal deaths accounted for about 29 % of total IMR (Fernós Isern, 1928). In 1924 for the state of New York excluding New York City, comparable numbers were: 69, 40.74, and 28.29, with about 59 % of the total IMR being neonatal mortality (Fernós Isern, 1928). In 1921–1925 in England and Wales, comparable numbers were: 76, 33.4, and 42.6 with about 44 % neonatal mortality (Barker & Osmond, 1986). In 1928–1938 in England and Wales, comparable numbers were: 61.7, 30.9, and 30.8 with 50 % being neonatal mortality (Woolf, 1947).

¹⁵⁹ Fernós Isern (1925, 1928).

Table 1.6 Major causes of infant mortality in Puerto Rico in 1934

Category	All ages (%) ^c	Under 1 month (%) ^c	1st–11th month (%) ^c
Digestive system ^a	35	12	48
Early infancy ^b	24	59	5
Respiratory system ^c	18	9	22
Infectious diseases ^d	16	13	17
Other	7	7	8
Total %	100	100	100
Total deaths	7,442	2,615	4,827

Source: Report of the Commissioner of Health of Puerto Rico to the Governor of Puerto Rico for the fiscal year ending June 30, 1935 (Garrido Morales, 1935, page 81)

Notes:

^aDiseases of the digestive system include: diarrhea, gastro-enteritis, and dysentery

^bDiseases of early infancy include: congenital disability, other diseases of early infancy, and congenital malformations

^cDiseases of the respiratory system include: acute bronchitis, pneumonia, and whooping cough

^dInfectious diseases include: malaria, tetanus, tuberculosis, measles, syphilis, meningitis, and diphtheria

^ePercentages for other years in the 1930s are similar. A very high percentage of congenital disability, syphilis, infantile tetanus, other diseases of early infancy, and infantile convulsions fell within 1 month of life. All other mentioned diseases fell predominantly in 1–11 months of life

habits were an important cause of diarrhea.¹⁶⁰ It was noted by officials that breastfeeding, which is natural and important in the first 6 months of life, was often impeded due to mothers (especially poor mothers) having to return to work 10–12 h per day leading to irregular breastfeeding. Because cow's milk was scarce in Puerto Rico, mixed or artificial feeding (canned milk and mixtures of herb teas, sometimes in dirty bottles) during the first month occurred.¹⁶¹ Unclean breasts may also have been a contributing factor. The cause of diarrhea and dysentery under the age of two was perceived to be feeding habits, not understanding the importance of boiling water and milk, and not being careful with cooking and storage of food. Mortality from diarrhea under the age of two was not associated with climate change or rainfall variations with the seasons of the year, but it was for children over the age of 2,¹⁶² thus leading to a stronger argument that feeding habits during the first year of life may have been an important cause of death whereas environmental factors became important at older ages.

¹⁶⁰ Fernós Isern (1928).

¹⁶¹ Fernós Isern and Rodríguez Pastor (1930). Wegman, Fernández Merchante, and Kramer (1942) also found that in the rural *municipio* of Ciales, a very high percentage of mothers who used the public health unit breastfed for up to 6 months (page 237). They noted two unusual facts about IMR in Puerto Rico: (1) there was a rapid decline in neonatal mortality rates (first month of life) but not a corresponding rapid decline in post neonatal mortality rates (1st month through the 11th month of life); (2) there was higher IMR in western and coastal regions and lower IMR in central and eastern regions (page 244).

¹⁶² Fernós Isern and Rodríguez Pastor (1930) and Phelps (1928).

Infections such as malaria and hookworm in the mother were an important explanatory factor for the second leading cause of infant death in Puerto Rico during the 1920s–1940s, congenital weakness. Malaria was prevalent in the very hot coastal regions where sugar cane was planted.¹⁶³ An association between congenital mortality and malaria in the mother was noted by many Puerto Rican public health officials of the time.¹⁶⁴ The highest IMR during the late 1920s and early 1940s tended to be in the coastal southern and western regions of Puerto Rico where malaria was common. Prevalence of hookworm was also high in rural areas and it appears many lived with a chronic infection throughout the year.¹⁶⁵ A high percentage of adults did not wear shoes in rural areas which contributed to hookworm infection. In a typical sugar plantation, about 60–69 % of those above 15 years old did not wear shoes.¹⁶⁶ While hookworm tends to be associated more in men and with certain occupations (coffee in the case of Puerto Rico and mining in the case of England and Wales), adult women in Puerto Rico may also have been more severely affected than men because of soil conditions close to housing.¹⁶⁷ Hookworm is associated with intrauterine growth retardation, premature delivery and low birth weight among newborns born to infected mothers and can lead to anemia especially if there is poor nutrition (protein and iron).¹⁶⁸

The third leading cause of infant death was respiratory infection. Public health officials in Puerto Rico at the time generally thought that the major cause of respiratory infections was direct contact with older persons with bronchitis and influenza who may not have understood the importance of limiting their contact

¹⁶³ Examples of *municipios* with high malaria rates were Barceloneta, Fajardo, and Ponce (Earle, 1925). The annual reports of the health commissioner frequently mentioned *municipios* with lower and higher malaria rates. See for example Fernós Isern (1932, 1933, 1946, n.d.), Garrido Morales (1935, 1936, 1937, 1940, 1941), and Ortiz (1927, 1929, 1931).

¹⁶⁴ See Fernós Isern and Rodríguez Pastor (1930), although they note that mortality from congenital disability was decreasing by the late 1920s.

¹⁶⁵ Hill (1926) describes hookworm infection in early twentieth century Puerto Rico as a chronic condition in the rural areas that was not seasonal. A high prevalence of hookworm infection does not reflect severity; severity is measured by worm burden (number of worms). Historical records describe a high severity in certain regions and *municipios* of Puerto Rico especially in the coffee regions. In the mountainous interior of the island, infestation averages were estimated at 500 parasites per person whereas in the coastal areas estimates were closer to 200 per person. The severity of the infection in other regions is not clear. Nevertheless, the high infestation averages confirm that hookworm infection was a serious health issue in Puerto Rico during the late 1920s–1930s. In terms of the effect of hookworm infection on maternal health, Gilles and Ball (1991) point out that hookworm infection in mothers may cause nutritional problems for the developing child, although hookworm infection alone may not be as serious as other nutritional factors which affect disease (Gilles & Ball, 1991).

¹⁶⁶ Morales Otero et al. (1937, page 460).

¹⁶⁷ The highest rates were in coffee cultivation areas, with an average infestation rate of 90 % and an extremely high prevalence of hookworm disease—a disease prevalent in Puerto Rico since 1530 (Daengsvang, 1932; Hill, 1926; Howard, 1928). It is difficult to identify the severity of hookworm disease since the worm burden is important in terms of morbidity risks for the mother. A pregnant woman could be infected but not have a high worm burden, but even low worm burdens can lead to anemia (Hill, 1926).

¹⁶⁸ WHO (2011a).

with other people when they were sick¹⁶⁹; undernourished children exposed to adults with the disease were more susceptible to infection.

The fourth leading cause of infant death was from epidemic, endemic, and other infectious diseases affecting newborn infants, such as malaria and tetanus. However, other diseases such as tuberculosis and measles could have escaped detection as causes of death or have been hidden when either gastrointestinal or respiratory were listed as the primary cause of death.

Maternal mortality and stillbirth rates during the early twentieth century suggest that maternal health status was substandard, especially in rural areas. In 1927, maternal mortality and stillbirth rates in Puerto Rico were 32.6 maternal deaths per 10,000 total births and 86 stillbirths per 1,000 total births.¹⁷⁰ Although there was variation among municipalities, the number of stillbirths was still high in the 1930s¹⁷¹ as were neonatal (during the first month of life) deaths.¹⁷²

Women mostly delivered their babies at home with midwives, not at hospitals with doctors. Although there are little historical data on low birth weight—a measure of intrauterine growth—indirect measures such as the number of physicians per capita and expenditures on public health in early twentieth century Puerto Rico along with poor nutrition and high infection suggest the risk of a low birth weight baby must have been high, especially among more vulnerable populations such as the families of rural laborers. In 1930, there was one doctor per 6,829 people¹⁷³ and most of the doctors were in urban areas. Expenditures on public health and charity were low.¹⁷⁴

The chronically undernourished state of the rural population during the late 1920s-early 1940s probably had serious effects on the ability to fight infectious

¹⁶⁹ Fernós Isern and Rodríguez Pastor (1930).

¹⁷⁰ Ortiz (1929, page 134).

¹⁷¹ As a way of comparison, Corsica in France in the 1950s had an endogenous mortality of 26.4 per 1,000 and a stillbirth mortality of 19.9, for a total perinatal mortality rate of 46.3 per 1,000, 43 % of which was stillbirths (Pressat, 1961).

¹⁷² Early research on the distribution of deaths during the first year of life (Bourgeois-Pichat, 1952; Galley & Wood, 1999) shows that neonatal mortality rates (deaths during the first month of life) are strongly associated with non-environmental or endogenous causes. Endogenous infant mortality is defined as “cases in which the child bears within itself, from birth, the cause resulting in its death, whether that cause was inherited from its parents at conception or acquired from its mother during gestation or delivery” (Bourgeois-Pichat, 1952). Barker and Osmond’s (1986) classic geographical analysis in England and Wales in the 1960s showed strong positive associations between neonatal mortality and adult mortality due to coronary heart disease. Nearly 80 % of overall neonatal mortality was due to congenital causes. Endogenous mortality thus became an important clue as to the source of early life conditions which led to adult mortality. Barker later concluded that, “coronary heart disease is associated with past infant mortality because it originates in conditions *in utero*, rather than poor conditions in childhood, though these contribute” (Barker, 2002, page 309).

¹⁷³ According to Ortiz (1927, page 99): “Three hundred and twenty-one physicians are practicing in Porto Rico, according to our records. The estimated population of the Island is 1,417,646. The average number of persons for each physician is about 4,500. Considering that of these 321 physicians 113 are practicing in San Juan and about 25 in Ponce, the second largest city in the Island, there are only 183 left for the rest of the Island or an average of 6,829 people for each physician. This situation is particularly serious in small towns.”

¹⁷⁴ Mountin et al. (1937).

diseases such as diarrhea and dysentery. Perinatal exposure to maternal infections, possibly aggravated by poor maternal nutrition, may have placed the unborn child at risk. Perinatal exposure to tuberculosis and malaria is associated with low birth weight and fetal growth retardation.¹⁷⁵

1.5.5 Changes in the Late 1920s Through the Early 1940s: Reduction in Exposure to Disease but Stagnant Economic Conditions

During the 1930s–1940s, several forces combined to move Puerto Rico toward improvements in public health and reduction in disease.¹⁷⁶ As part of the New Deal, the US began to invest more heavily in Puerto Rico starting in 1933. Events in Europe during the 1930s led to an increased US military presence in Puerto Rico; as a result, there were additional efforts made to eradicate malaria in order to reduce troops' exposure while stationed there. However, the Puerto Rican government also played an important role, implementing interventions to improve conditions throughout the island. Taken as a whole, these forces form the background in which the Puerto Rican population experienced rapid improvement in public health during the 1930s–1940s during stagnant economic times.

It was in the 1930s when public health efforts intensified and spread to more of the island. The period of the late 1920s–early 1940s saw increased sanitary, hygienic, and other preventive efforts. There was an increased number of public health units equipped with a medical team dispatched throughout the *municipios* beginning in 1926 with Río Piedras and Yabucoa. There were rural sanitation efforts through public health education using rural teachers and the building of latrines,¹⁷⁷ laws improving the quality of midwives,¹⁷⁸ improvements in the quality of milk, improvements in water supply and sewerage systems,¹⁷⁹ and help from non-profit international organizations such as the Rockefeller Foundation along with US government assistance as part of the New Deal in the late 1930s.¹⁸⁰ The interventions were not aimed at directly improving nutrition, with the exception of a few milk stations in larger urban areas.¹⁸¹ The Department of Health built thousands of latrines in the rural areas in the 1930s,¹⁸² inspected rural school children for

¹⁷⁵ See for example Jana et al. (1994) and Guyatt and Snow (2004).

¹⁷⁶ Ayala and Bernabe (2007), Perloff (1952), and Steward et al. (1956).

¹⁷⁷ Fernós Isem (1932, 1933, 1946, n.d.), Garrido Morales (1935, 1936, 1937, 1939, 1940, 1941), and Ortiz (1927, 1929, 1931).

¹⁷⁸ Belaval (1945).

¹⁷⁹ Ortiz (1929).

¹⁸⁰ Farley (2004).

¹⁸¹ Fernós Isem (1932, 1933, n.d.), Garrido Morales (1935, 1936, 1937, 1939, 1940, 1940, 1941), and Ortiz (1931).

¹⁸² See for example the annual reports of the health commissioner of Puerto Rico in the late 1920s and 1930s (Fernós Isem, 1932, 1946, n.d.; Garrido Morales, 1936, 1937, 1939, 1940); Ortiz, (1927).

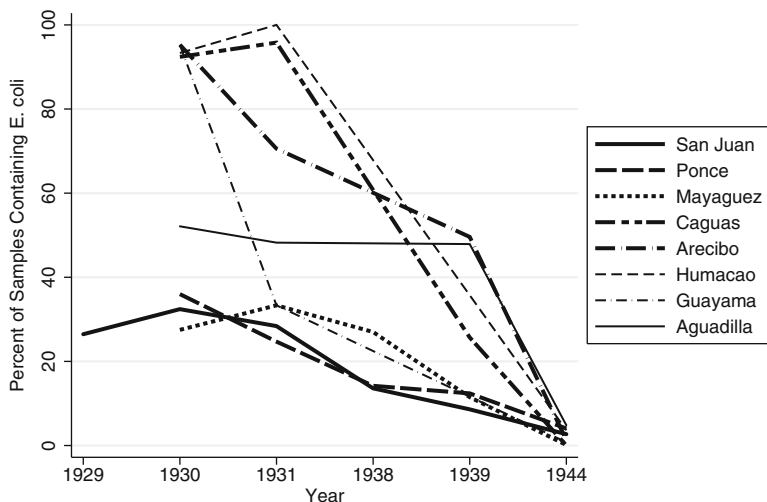


Fig. 1.8 Percent of water samples containing *E. coli* in wealthier municipalities in Puerto Rico, 1929–1944. *Notes:* *Bolded lines* of any pattern are class 1 (wealthiest) municipalities; *unbolded lines* are class 2 (middle class) municipalities. In each of the municipalities, acceptable water systems were developed in 1930, except in Humacao in which a system was developed in 1938 (*Sources:* Reports of the Commissioner of Health of Porto Rico 1929–1945 (Fernós Isern, 1932, 1933, 1946; Garrido Morales, 1940, 1941; Ortiz, 1931))

infections,¹⁸³ and educated people in the countryside regarding wearing shoes and using latrines to prevent hookworm infection. Targeted malaria campaigns drained swamps in coastal areas to reduce stagnant water where mosquitoes could breed.

Water treatment improved by the end of the 1930s, but mostly in the larger urban or wealthier areas.¹⁸⁴ In 1930, the five most affluent municipalities all had acceptable water treatment systems including the use of chlorine. By 1940, these wealthier municipalities all used sedimentation, coagulation, filtration, and chlorination. Water quality reflected by the presence of *E. coli* in water improved dramatically in wealthier *municipios* (Fig. 1.8).¹⁸⁵ In contrast, in 1930 and 1940 about 82 % of the least affluent municipalities had no water treatment system at all.

¹⁸³ Fernós Isern (1932, 1933, 1946, n.d.), Garrido Morales (1935, 1936, 1937, 1939, 1940, 1941), and Ortiz (1927, 1929, 1931).

¹⁸⁴ Fernós Isern (1932) and Garrido Morales (1941).

¹⁸⁵ Historical sources refer to the high percent of *B. coli* (later renamed *E. coli*; see Todar's Online Textbook of Bacteriology, 2008) in water supplies during the early 1930s (Fernós Isern, 1932, 1933, 1946; Garrido Morales, 1940, 1941; Ortiz, 1931). See also United States Environmental Protection Agency (2010).

Figure 1.9 shows the decline of infant mortality in Puerto Rico. Prior to the advent of antibiotics and other therapies after 1945, there were at least two periods of decline: one at the beginning of the twentieth century, probably as a result of US-instituted rigorous public health measures,¹⁸⁶ and another between the 1920s and the early 1940s attributed largely to more widespread public health interventions throughout the island and improvements in medical technology.¹⁸⁷

Changes in IMR during the 1930s occurred unevenly (Fig. 1.10) with large variations among municipalities across time, possibly reflecting the resources available to reduce disease and the type of public health intervention implemented. The wide variation may also partially reflect the accuracy of vital statistics prior to the 1930s.¹⁸⁸ Higher IMR in the late 1920s tended to be found in coastal areas; temperatures were consistently high through the year, and there was a high prevalence of malaria. Higher IMR also existed in urban centers because of overcrowding. Lower IMRs in the late 1920s were found in the highlands where tobacco was grown, but the lowest IMRs were found in the center of the island between the coffee and tobacco regions but close to the sugar cane region. Some

¹⁸⁶ Rigau Pérez (2000).

¹⁸⁷ Fernós Isern (1932, 1933, 1946, n.d.), Garrido Morales (1935, 1936, 1937, 1939, 1940, 1941), Ortiz (1927, 1929, 1931), and Rigau Pérez (2000).

¹⁸⁸ The collection of vital statistics in Puerto Rico in the early twentieth century was the responsibility of *municipio* clerks under direct supervision from the mayor of each municipality. While law required that daily statistics be kept which contained information determined by the Director of Health (Pérez, 1926), the Department of Health did not have direct oversight into the local registers and how data were collected until the early 1930s. In fact, it is dubious whether municipal authorities inspected the civil registers in the early 1920s. However, Puerto Rican laws established during this period clearly outlined fines for not reporting deaths and required that a physician sign the death certificate. Thus the Department of Health viewed the numbers of deaths collected by each *municipio* as fairly accurate and believed that almost all deaths were reported. According to the Department of Health, over 90 % of births were reported (Bureau of Vital Statistics, 1926). The 90 % estimate agrees with official estimates of underreporting in later annual reports to the governor. The problem of underreporting births appears to be related to missing birth reports for infants who died before they were a year old. In the Fiscal Year 1930 report, an examination of December 1929 deaths of children under 1 year with matching birth reports found that about 45 % of associated births were not reported (Fernós Isern, 1932, page 8): "According to law death reports are 100 % complete as no burial permits are issued without a certificate of death from the physician, but on the other hand, birth reports, taking the month of December, 1929, as a basis are only about 55 % complete. . . The infant mortality rate. . . would be 125 per 1,000 live births after proper checking 118 per 1,000 live births." Thus, in this example, there was about a 6 percentage point difference between the higher calculated and lower corrected infant mortality rate for December 1929. It is not clear from the annual reports to the governor the degree to which underreporting of births may have occurred when the infant did not die before 1 year of age. An important weakness of the system was also the classification of births and deaths by race, domicile, age, and occupation and the reporting of the cause of death and morbidity because there were no uniform standards across *municipios*. In the 1920s, the Department of Health actively classified deaths according to international standards of disease classification, but many Puerto Rican physicians did not conform or did not report communicable (transmissible) diseases as required by law.

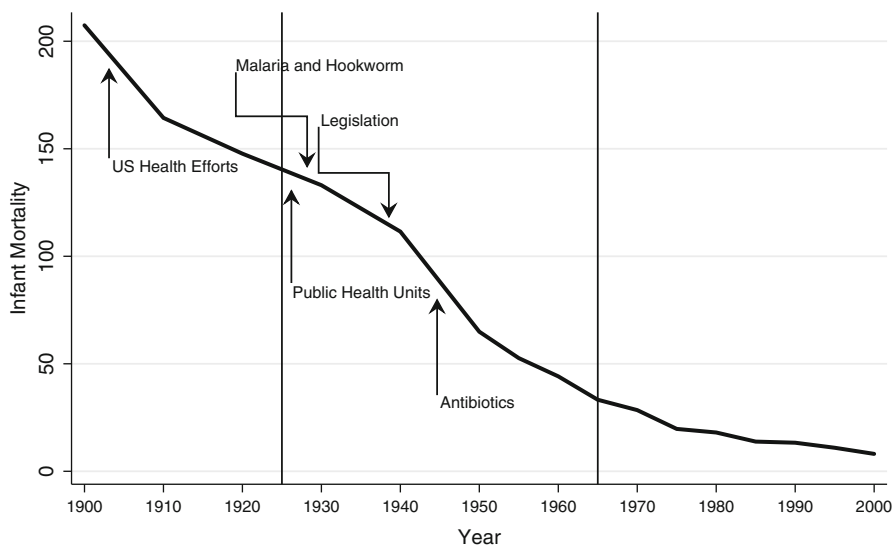


Fig. 1.9 Infant mortality in Puerto Rico (1900–2000). *Notes:* Appearing on the graph are a few key events that likely impacted the decline of IMR in Puerto Rico in the early twentieth century. The vertical lines at 1925 and 1965 delineate the period of the 1930s–1960s (*Sources:* Original sources are the annual reports of the Commissioner of Health in Puerto Rico in the early twentieth century (Fernós Isern, 1932, 1933., 1946, n.d.; Garrido Morales, 1935, 1936, 1937, 1939, 1940, 1941; Ortiz, 1927, 1929, 1931))

public health officials attributed lower IMR in the central region to the milder climate in the mountain ranges and although population density was associated with higher IMR in Puerto Rico¹⁸⁹ population density may not fully explain the lower IMR in the central region.

IMR was high during the late 1920s in many *municipios* and then improved during the 1930s as more public health units¹⁹⁰ were constructed and preventive public health interventions took place. The *municipios* which experienced the most dramatic changes were those along the coastal regions, especially areas with some major cities. The average percentage change in IMR between 1927 and 1943 ranged from 28 % to as high as 70 %.¹⁹¹ Although there was improvement during the 1930s in IMR, IMR was still high and many of these same *municipios* with high IMR still had among the highest IMR in the early 1940s.

¹⁸⁹Fernós Isern and Rodríguez Pastor (1930)

¹⁹⁰The first public health unit was created in 1926 in Río Piedras and was partially funded by the Rockefeller Foundation (Ortiz, 1929). By 1938, each municipality had its own public health unit. Annual reports throughout the 1930s refer to the gradual coverage of the entire island with public health units staffed by a medical team of a doctor and nurses.

¹⁹¹Author's calculations from the late 1920s to 1943 using *municipio*-specific IMR obtained from the annual reports of the health commissioner of Puerto Rico.

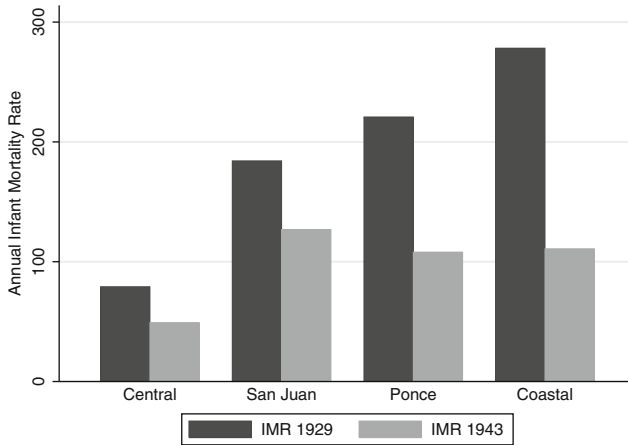


Fig. 1.10 Infant mortality rates in Puerto Rico by selected region and municipality, 1929 and 1943. *Notes:* *Municipios* showing IMR greater than or equal to 200 per 1,000 births in 1929 came mostly from the southern or western coastal regions which where sugar cane was grown. They included: Aguada, Añasco, Arroyo, Camuy, Guánica, Guayama, Hormigueros, Juana Díaz, Lajas, Las Marías, Maricao, Mayagüez, Moca, Patillas, Ponce, Rincón, Sabana Grande, Salinas, Santa Isabel, and Yabucoa. All *municipios* in 1929 which had an IMR less than 100 per 1,000 births came from the central regions of Puerto Rico where mostly tobacco was grown. These included: Aguas Buenas, Aibonito, Barranquitas, Ciales, Cidra, Coamo, Jayuya, Naranjito, Orocovis, Vega Alta, and Villalba (*Sources:* Original sources are the annual Reports of the Commissioner of Health in Puerto Rico in the early twentieth century (Fernós Isern, 1932, 1933, 1946, n.d.; Garrido Morales, 1935, 1936, 1937, 1939, 1940, 1941; Ortiz, 1927, 1929, 1931). For the year 1929, data were obtained using the Porto Rico Review of Public Health and Tropical Medicine which reported monthly births and deaths for each *municipio*; yearly infant mortality rates by *municipio* were then calculated using these numbers. For 1943, infant mortality rates were obtained using the Puerto Rican Commissioner of Health annual report to the governor of Puerto Rico which contained already calculated yearly infant mortality rates for each *municipio*)

By the late 1930s, maternal mortality rates were also declining (Fig. 1.11) and, as environmental conditions began to improve, there was weaker seasonality of diseases such as dysentery and malaria (Fig. 1.12). Yet, in spite of environmental improvements and reductions in IMR, the leading cause of death in Puerto Rico at the end of the 1930s continued to be dysentery and diarrhea in children under the age of 2¹⁹²; mortality rates for several infant and childhood diseases also remained high.¹⁹³

By reducing disease, nutritional status may have improved to some extent in that less energy was spent fighting disease and more could be spent nurturing the body. However, an examination of improvement in height across the early twentieth

¹⁹² Garrido Morales (1941) and Vázquez Calzada (1988).

¹⁹³ Fernós Isern (1933), Garrido Morales (1935, 1936, 1937, 1939, 1941).

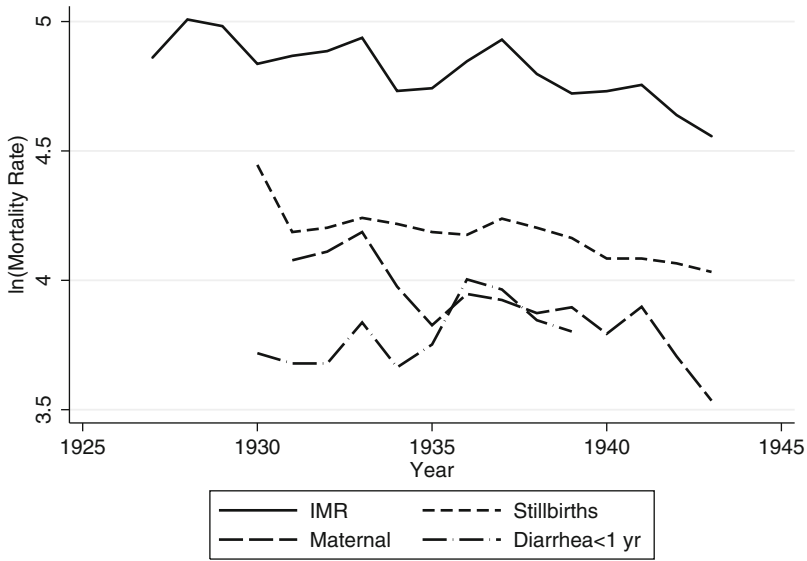


Fig. 1.11 Mortality rate for infants, stillbirths, mothers, and infants less than 1 year with diarrhea (Sources: McEniry, 2009a. Original sources are the annual Reports of the Commissioner of Health in Puerto Rico in the early twentieth century (Fernós Isern, 1932, 1933, 1946, n.d.; Garrido Morales, 1935, 1936, 1937, 1939, 1940, 1941; Ortiz, 1927, 1929, 1931))

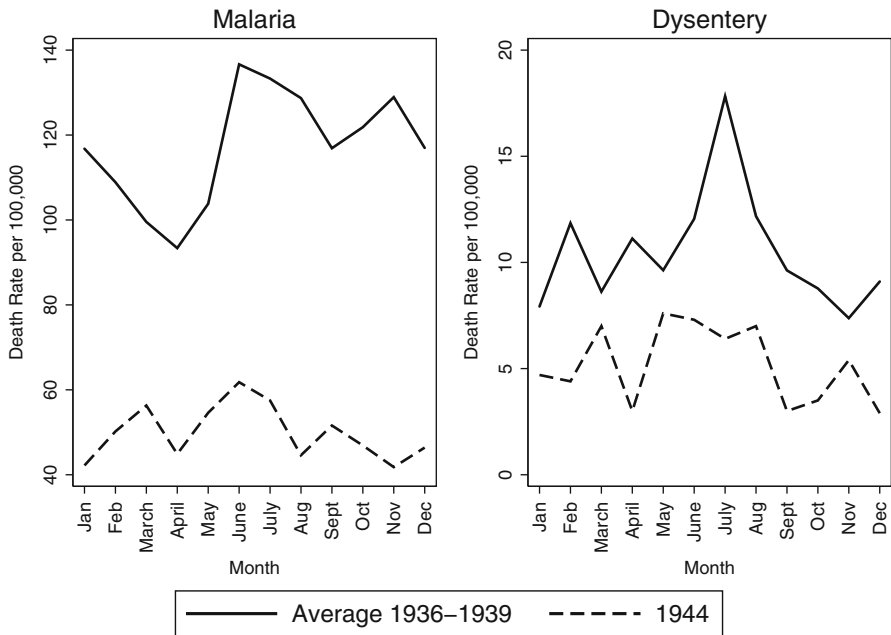


Fig. 1.12 Seasonality of death rates due to malaria and dysentery in Puerto Rico, 1936–1944. Notes: Death rates are per 100,000 for all ages, although dysentery primarily affected infants and children (Sources: Reports to the Governor of Puerto Rico for Fiscal Years 1937–1938, 1939–1940, and 1944–1945 (Fernós Isern, 1946; Garrido Morales, 1939, 1941))

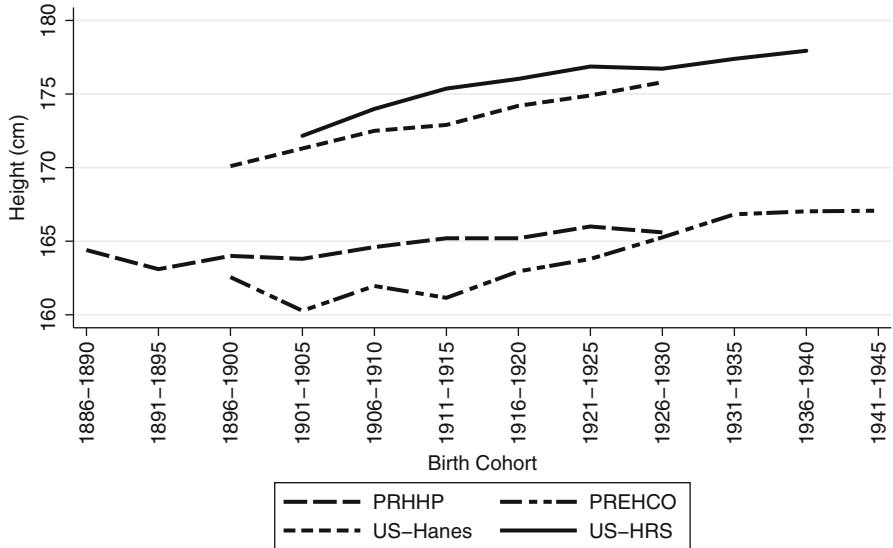


Fig. 1.13 Adult male height in US mainland males and Puerto Rican males. *Notes:* Health and Retirement Study, Public Use Dataset (2000–2006) survivors now are taller than the HANES (1971–1975) survivors suggesting that taller individuals survived, but Puerto Rican survivors from PREHCO (2002–2003) are shorter than the PRHHP (1965) data suggesting perhaps that taller individuals migrated to the US (*Sources:* National Heart, Lung and Blood Institute, 2008; Puerto Rican Healthy Heart Program (PRHHP), 1965–1968; US HANES, 1971–1975 as reported in Godoy et al., 2007; Puerto Rican Elderly Health Conditions (PREHCO), 2007; Health and Retirement Study, Public Use Dataset (2000–2006)

century among Puerto Rican males suggests that there was not tremendous improvement in nutritional status.¹⁹⁴ Using available data on height, Fig. 1.13 shows that from 1900 to 1940, US mainland non-Hispanic white males gained almost 7 cm (about 2.76 in.) in height whereas, during the same time period, males in Puerto Rico gained only about 3 cm (slightly over one inch).¹⁹⁵ The slower increase in height partially reflects the minimal improvements in standard of living in Puerto Rico in the early to mid twentieth century, similar to other Latin American countries.¹⁹⁶ Yet, improvements in public health and sanitation and reduction in exposure to disease did result in mortality decline during this period.¹⁹⁷ While there are no comparable

¹⁹⁴ Fogel (2004).

¹⁹⁵ Godoy et al. (2007), Health and Retirement Study, Public Use Dataset (2000–2006), and PREHCO (2007).

¹⁹⁶ See for example López-Alonso and Porras Condey (2003) and Meisel and Vega (2007).

¹⁹⁷ See the series of reports by the health commissioner of Puerto Rico throughout the 1930s. For example Fernós Isern (1932, 1933, n.d.), Garrido Morales (1935, 1936, 1937, 1939), and Ortiz (1931). Also see Morales Otero and Pérez (1939), Morales Otero et al. (1939), and Morales Otero et al. (1937).

published data for female height during the period, conditions for females were not any better than for males and could have been worse since some males who worked received at least one good meal per day as part of their employment.¹⁹⁸

As a tip of the iceberg country, Puerto Rico is a relevant case study and precursor by which to examine the long term consequences of poor early life conditions on older adult health for those born during the 1930s–1960s in the developing world. The circumstances in Puerto Rico, a small and poor island during the early to mid twentieth century, illustrate the precarious nature of living conditions and stagnant economic conditions. In spite of these environmental conditions, there was movement in mortality due to investments in public health efforts producing an increasing number of infants and children who survived.

1.6 Unintended Consequences and Future Policy Implications

The historical circumstances of the 1930s–1960s present a relevant example by which to examine policy and interventions that promote health over the long run. It is one of the few opportunities that exist to examine the long term consequences of policy on older adult health across a diverse range of countries. The example is relevant for many parts of the developing world. It is particularly relevant for those born in the 1930s–1960s but may also be relevant for those born afterwards where conditions mirror what occurred in the 1930s–1960s.

Public policy and well-intentioned public health interventions including medical innovation saved lives at young ages and dramatically increased survival during the 1930s–1960s. However, if early life conditions are indeed important to older adult health, the rapid survival of infants and children during the 1930s–1960s in low- and middle-income countries due to these policies and interventions in the midst of continued stagnant economic conditions may have long-term unintended consequences for their health at older ages.

The question of what types of policies to pursue in the improvement of life expectancy at early ages in the developing world is a complex one which hinges to some degree on the questions presented earlier regarding the relative importance of early life conditions for older adult health. An additional question looms based on the historical circumstances of the 1930s–1960s which is relevant today:

- What are the long term consequences on adult health of rapid improvement in survivorship of infants and children based primarily on public health interventions including medical innovation but not based on parallel improvements in standard of living?

¹⁹⁸ Clark (1930).

1.7 Conclusions

The growth of the aging population in many developing countries today is directly linked with the particular characteristics of the mortality decline in the early- to mid-twentieth century. The timing, pace, and reason for mortality decline produced rapid demographic transitions during the 1930s–1960s that resulted in unique cohorts that included an increasing numbers of infants and children who survived poor early life conditions due to public health including medical technology. However, many survived in the developing world amidst stagnant economic growth resulting in continued exposure to poor nutrition and infectious diseases. If the hypothesized importance of early life conditions (nutrition in particular) in the development of certain adult health chronic conditions such as heart disease and diabetes has merit, then the historical circumstances of the early- to mid-twentieth century may have produced unintended long term consequences. The unique cohorts of the 1930s–1960s may be more susceptible to the effects of poor early life conditions at older ages. In contrast to the health of older adults in the developed world, it is conjectured that early life conditions may be an important determinant of the projected growth in adult heart disease and diabetes, as important as the major dietary changes (higher consumption of saturated fat and sodium) that have taken place along with an increasingly sedentary lifestyle. An examination of the health of older adults across mortality regimes of the early twentieth century with a particular focus on older adults born in the tip of the iceberg countries such as Puerto Rico may shed light on the merit of this conjecture.

There is a tide to come comprised of increasing numbers of older adults in the developing world with chronic conditions such as heart disease and diabetes. It remains to be seen if the health of those in the tide to come can largely be explained by the rapid survivorship of poor early life conditions at younger ages due to well intentioned public policy, interventions including medical innovations but largely in the context of little improvement in standard of living. The upcoming chapters provide some evidence that suggests that the conjecture may not be so far off the mark.

The previous chapter laid out the ground work for a conjecture regarding life expectancy and older adult health for the unique cohorts of the 1930s–1960s in some low- and middle-income countries. In the next section, more detail is provided for a contrarian conjecture and related hypotheses, a roadmap and the “ideal” data along with an introduction to the overall approach used to examine the hypotheses.

2.1 A Contrarian Point of View

There are opposing viewpoints regarding the limits of life expectancy even in the developed world, some optimistic¹ and others pessimistic.² For the most part, life expectancy in the developed world presents an optimistic viewpoint; however, recent evidence in the US and some parts of Europe suggests that trends in life expectancy may be reversing or leveling off.³ There is also the issue of what will happen to quality of life as humans live longer.⁴

However, in general, people across the world are living longer because of improvements in early life conditions; chronic conditions such as heart disease and diabetes among older adults are projected to thereby increase.⁵ On the surface, it would seem that as developing countries undergo a transition to an increasingly older adult population, older adults in those countries will experience life expectancy and health patterns similar to those in developed countries.⁶ Table 2.1 shows

¹ Oeppen and Vaupel (2002) and Floud, Fogel, Harris, and Chul Hong (2011).

² Gwatkin (1980) and Manton and Singer (1994).

³ Crimmins, Preston, and Cohen (2010) and Woolf and Aron (2013).

⁴ World Health Organization (2011b) definition of Healthy Life Expectancy (HALE): “Average number of years that a person can expect to live in ‘full health’ by taking into account years lived in less than full health due to disease and/or injury.”

⁵ Murray et al. (2012) and Lim et al. (2012).

⁶ See for example, Barreto et al. (2012).

Table 2.1 GDP per capita, standard of living, and health care in selected countries

Country	GDP per capita (1990 international dollars)			Income group 2000	Health care rating 2000	Life expectancy at age 60 (M, F) 2000	Human development ranking 2000	Gini index 1995–2005	Caloric intake 2000
	1930	1940	1960						
Barbados	1,815	1,698	4,034	High	46	18.6, 22.6	31	39	3,025
England	5,441	6,856	8,645	High	18	18.8, 22.7	13	36	3,370
Netherlands	5,603	4,831	8,287	High	17	18.9, 23.7	8	31	3,215
Puerto Rico	815	896	3,421	High	37	19.6, 23.1	–	56	–
Taiwan	1,150	1,134	1,353	High	–	20.0, 22.9	–	33	–
US	6,231	7,010	11,328	High	37	19.6, 23.1	6	41	3,732
Argentina	4,080	4,161	5,559	Upper middle	75	17.8, 22.8	34	53	3,272
Brazil	1,048	1,250	2,335	Upper middle	125	16.2, 19.6	73	59	2,885
Chile	2,859	3,236	4,270	Upper middle	33	18.3, 22.7	38	55	2,806
Costa Rica	1,626	1,763	2,715	Upper middle	36	19.3, 22.8	43	47	2,804
Cuba	1,505	1,208	2,052	Upper middle	39	19.3, 21.5	55	41	3,051
Mexico	1,618	1,852	3,155	Upper middle	61	19.7, 21.7	54	53	3,172
South Africa	2,247	2,496	3,041	Upper middle	175	14.5, 17.1	107	58	2,886
Uruguay	4,301	3,661	4,960	Upper middle	65	17.1, 22.2	40	45	2,831
Bangladesh	659	637	545	Low	88	14.7, 15.7	145	31	2,125
China	568	1,000	662	Lower middle	144	16.6, 20.4	96	42	2,908
Ghana	878	686	1,378	Low	135	14.5, 16.5	129	41	2,596
India	726	686	753	Lower middle	112	14.6, 17.7	124	37	2,314
Indonesia	1,141	1,165	1,012	Lower middle	92	15.5, 17.5	110	38	2,498

Sources: Maddison (2006, 1990 international dollars), WHO (2000b, 2002), World Bank (2011), United Nations Development Program (2002), World Bank (2012), and FAO Statistics Division (2010); see <http://sowf.moi.gov.tw/stat/english/elist.html> for Taiwan life expectancy

Notes: Countries are alphabetized within income group. Bangladesh was part of India until 1947. Puerto Rico and Barbados GDP per capita numbers are estimated through linear extrapolation. The values displayed in the table are the closest available Gini indices to the year 2000. When multiple values were available, the one closest to the survey year was chosen. The values come from the World Bank (2012) with the exception of Barbados (John & Firth, 2005), Cuba (Ramis & Kosack, 2004), Puerto Rico (Toro, 2008), and Taiwan (CIA, 2012). Lower health care ratings indicate better health care systems

the countries studied in terms of historical information on GDP per capita along with recent information regarding income, health care, life expectancy at age 60, and per capita caloric intake. GDP per capita has improved for the countries that were poorer in the 1930s; although income inequalities still exist, many are now upper-middle-income countries. A few (Costa Rica, Cuba, and Chile) have a similar rating for their health care system as the US, and a few (Costa Rica, Cuba, and Mexico) have life expectancy at age 60 close to that of the US. With the exception of the poorer countries, most countries have a relatively high caloric intake. Barbados and Costa Rica are noted success stories in improvement of social indicators.⁷

Palloni and colleagues present a contrarian point of view.⁸ They argue that future prospects for life expectancy for those born during the 1930s–1960s may not materialize in the developing world precisely because of the circumstances described in Chap. 1—rapid mortality decline primarily due to public health interventions including medical technology amidst stagnant economic conditions which left many still exposed to poor environmental conditions in early life. The mortality decline of the 1930s–1960s was not random relative to conditions affecting health status; it was predominantly due to improvements in infant and childhood environmental conditions. Those affected by poor early conditions who benefited most from public health interventions most likely were the less privileged. The health status of the poorer segment of the population was worse than average to begin with, and thus the rapid and dramatic mortality changes of the 1930s–1960s, especially after 1945, potentially contributed to producing a frailty composition worse than that of more evenly spread survival gains.⁹ Even though nutritional status may have improved due to reduction in disease and better treatment, stagnant economic conditions hampered large improvement in nutritional status—a supposition illustrated in recent studies.¹⁰

The contrarian conjecture of Palloni and colleagues assumes strong cohort effects marked by poor early life conditions for the unique cohorts born in the 1930s–1960s in the developing world. It assumes to some degree that the main hypothesized mechanisms driving adult morbidity and mortality differences for a unique cohort of individuals in the 1930s–1960s are based on their exposure to early life nutrition and infectious diseases. In this scenario, adult lifestyle (diet, smoking and drinking habits, and exercise) may also be important but cannot fully explain their health. If early life hypotheses such as Barker’s have merit,

⁷ Mehrotra and Jolly (1997).

⁸ Palloni (2011), Palloni, Noronha, and McEniry (2009), Palloni, Pinto-Aguirre, and Peláez (2002), and Palloni and Pinto-Aguirre (2004).

⁹ Palloni, McEniry, Wong, and Peláez (2006), Alter and Riley (1989), and Vaupel, Manton, and Stallard (1979).

¹⁰ For example, see López-Alonso (2007), Godoy, Goodman, Levins, Caram, and Seyfried (2007), and Harris (2000).

then one implication is that living conditions may compound the effects of poor early life conditions but not confound them.¹¹

However, the opposite may be true: It could be that the changes across entire populations to a diet with higher saturated fat, sodium and sugar content and shifts toward a sedentary lifestyle¹² more adequately explain older adult health even in the developing world. Diet continues to be identified as an important factor influencing health at all ages around the world,¹³ and a diet high in saturated fat and sodium can contribute to heart disease.¹⁴ This type of unhealthy diet already poses risks for adult health in the developed world. Unhealthy processed foods are increasingly being imported into the developing. These foods have, in many cases, replaced traditional diets and increased health risks.¹⁵

In terms of the contrarian conjecture, differences across countries in the growth of the elderly population attributable to early mortality decline provide clues as to the degree to which cohorts may have been affected by the historical circumstances of the 1930s–1960s. If the Palloni et al. contrarian conjecture has merit, and adequate measurement of health and its determinants can be assumed, there should be observable differences in the health of older adults across mortality regimes according to the degree to which the growth of the elderly population attributable to early mortality decline is due to these historical circumstances.

Unequal societies tend to also have larger SES inequalities in health.¹⁶ There were already very large disparities between upper income and other income groups in many of the societies in low- and middle-income countries during the early to mid twentieth century.¹⁷ An increasing number of individuals scarred by poor childhood health may have led to an increasing amount of individuals with difficulty in attaining a better socioeconomic position in adulthood, thus producing sharper SES inequalities.¹⁸

Because larger changes in life expectancy and infant and child mortality were more heavily concentrated in low- and middle-income countries during the 1930s–1960s,¹⁹ these countries are more likely to have been affected by these historical circumstances. Given the rapid changes of the 1930s–1960s and the subsequent growth of the aging population, it maybe possible to observe differential effects of early life conditions on older adult health through comparisons across and within different mortality regimes of the period, controlling for important confounding factors throughout the life course.

¹¹ Barker, Forsén, Uutela, Osmond, and Eriksson (2001) and Barker (1995).

¹² Popkin, Horton, and Kim (2001) and Basu, Yoffe, Hills, Lustig, and Wagner (2013).

¹³ Murray et al. (2012) and Lim et al. (2012).

¹⁴ Marmot and Elliott (2005).

¹⁵ De Schutter (2012).

¹⁶ Wilkinson (1996).

¹⁷ López-Alonso (2007).

¹⁸ For a longer discussion about health selection—see for example, Lundberg (1991) and Palloni et al. (2009).

¹⁹ Preston (1976).

It may also be possible to discern the degree to which unique cohorts more heavily characterized by early life conditions are also in poorer health at older ages, particularly chronic conditions such as heart disease and diabetes.

The Palloni conjecture encompasses not only the potential importance of early life nutrition for older adult health, but also early experience with illnesses, other deprivations experienced during childhood, and the synergy between nutrition and infection.²⁰ The conjecture is particularly relevant for those who survived after the advent of antibiotics and other therapies in the mid- to late-1940s when most of the mortality decline occurred.²¹ However, many adults born after 1945 and before the late 1960s have yet to reach the age of 60 and thus it is not possible to fully examine the conjecture for this group. Nevertheless, older adults born during the first half of the period, between the 1930s and 1960s, have reached older adult ages (60+). Some of the tip of the iceberg countries were able to reduce exposure to disease during the late 1920s through the early 1940s due to more investment in public health interventions and increased sanitation, eradication of malaria and hookworm, education campaigns, and preventive measures, despite stagnant economic conditions; this also occurred in the urban areas of some larger poor countries. The unique cohorts born in the tip of the iceberg countries experienced less exposure to disease but continued exposure to poor nutrition. They were less affected by mortality-driven selection than those who preceded them, and they may provide insight into the conjecture.

The following hypotheses restate and expand upon the Palloni et al.²² hypotheses regarding the health of older adults across the mortality regimes of the early twentieth century, paying particular attention to older adults born in the tip of the iceberg countries during the late 1920s through early 1940s:

1. The profile of older adult health in the tip of the iceberg countries will be found to be worse than what is observed in developed countries, even after removing the potential effects of current disparities in standard of living.
2. Current health conditions, but particularly the prevalence of certain chronic conditions such as heart disease and diabetes in the tip of the iceberg countries, will be closely associated with individual history, including nutritional status, early experience with illnesses, and deprivations experienced during early childhood.
3. Levels of mortality of older adults in the tip of the iceberg countries should exceed what one would expect; it will be higher than that experienced by populations with equivalent socioeconomic conditions but where the growth of the elderly population attributable to early mortality decline was due to improvements in standards of living.
4. Significant socioeconomic disparities in health status and disability of older adults in the tip of the iceberg countries will be found. Keeping everything constant, social and economic disparities will be more salient in areas where the contribution of past mortality decline associated with deployment of novel medical technology is higher.

²⁰ Scrimshaw (1968) and Scrimshaw (1997).

²¹ Preston (1976).

²² Palloni et al. (2006).

2.2 Road Map and Cross-National Comparisons

Table 2.2 provides a general road map for examining the hypotheses by comparing the health of older adults across mortality regimes. At one extreme are countries of mortality regime pattern A, which experienced an early and more graded mortality decline in the early twentieth century at higher levels of GDP per capita (mostly high-income countries). At the other extreme are countries of mortality regime pattern E (low-income countries) which experienced almost the opposite: mortality declined much later (during the 1950s) at lower levels of GDP per capita and was primarily due to widespread public health interventions including medical technology (antibiotics and other therapies). In between the extremes but not shown explicitly in the table, are other regimes (Patterns B–D). The expected results of differences in the pace, timing, and reason for mortality decline are shown at the bottom of the table. In the case of graded mortality decline, the overall health for cohorts at older ages is better, and poor early life conditions (nutrition, infectious diseases, poor SES) are hypothesized to have less effect on a cohort; the opposite is expected for the very late E regimes, with the B–D regimes somewhere in between.

Several cross-national comparisons are relevant across and within mortality regimes assuming it is possible to control for possible confounding factors such as adult lifestyle and life course events such as migration and access to quality health care. The first is to compare older adult health across mortality regimes within similar income groups to help control for disparities in standard of living. The health of older adults in the tip of the iceberg countries, which are now mostly middle-income countries, can be compared with the health of older adults in the other middle-income countries by mortality regime. A second but more difficult comparison is to examine the health of older adults in the tip of the iceberg countries with high-income countries at a similar point in their demographic transition. The timing of mortality decline due to heart disease during the 1960s in high-income countries with a similar but later moment in time for tip of the iceberg countries could help identify similar moments in the demographic transition.²³ The historical prevalence of (or mortality due to) chronic conditions such as heart disease and diabetes in the developed and developing world could provide a benchmark by which to examine a higher than expected prevalence at similar moments in the demographic transition after controlling for possible confounding factors. A third comparison involves examining health and its determinants across time by age for the unique cohorts and those preceding them. The health of the older adults of the unique cohorts should be related to their survivorship of poor early life conditions.

²³ See for example Harper, Lynch, and Davey Smith (2011). Palloni, McEniry, Dávila, and García Gurucharri (2005) graphically show crude mortality rate over time for Puerto Rico. It appears that consistent mortality decline occurred after 1980 about 20 years later than in the US.

Table 2.2 Demographic regimes and expected health patterns for the unique cohorts of the 1930s–1960s

Regime type A		Regime type E
Nature of mortality decline during the early twentieth century		
Very early (1800s)	Timing	Very late (1945–1960)
More graded	Pace	Rapid
Higher standards of living	Primary reason	Public health, medical technology affecting infants and children
Expected consequences in the twenty-first century		
Smaller	Pool of survivors Older adult health	Larger
As expected	Fragility	More than expected
As expected	SES differentials	Sharper
Weaker	Effects of poor nutrition and infectious diseases in early life	Stronger
As expected	Expected mortality risk	Higher than expected

Source: Author constructed table

Note: “Pool of survivors” includes the survivors of poor nutrition and serious infectious diseases experienced in early life

2.3 Ideal Data, Surveys of Older Adults, and Historical Data

The ideal type of information needed to examine early life hypotheses is cohort data collected throughout the life course with extensive detail on early life and other critical periods, and with accurate information regarding older adult health. This would permit a better understanding of health not only for a particular cohort but also for different cohorts at similar ages across time.

Good indicators of the timing and nature of *in utero* and infancy exposures and data such as mother’s health during pregnancy, maternal height/weight, birth weight/length, and childhood growth patterns at critical ages would be needed, along with good measures of SES throughout the life course precise enough to distinguish groups at risk. Detailed information on the nature, timing, and severity of all infections, illnesses, and diseases during infancy and childhood for individuals would be needed. An older brother who contracted hookworm and survived but now suffers health consequences in old age should be distinguished from a younger sister who did not contract hookworm because latrines were built and she wore shoes. Similarly, the youngest brother should be distinguished from his siblings after he contracted respiratory diseases after 1945, when he could be treated with antibiotics or other therapies and survive. Each of these examples (exposure to disease, reduced risk of disease, and survival due to medical innovations) reflects a potentially different set of circumstances which could better explain later adult health. Postnatal and childhood family environment or young adult living arrangements can also have

important effects and may moderate the effects of poor early life conditions.²⁴ Detailed information on adult occupation and occupational hazards, migration, timing, and severity of illnesses, critical events, and adult lifestyle would be needed. Valid and robust measures reflecting the complexity and severity of older adult health in terms of chronic conditions, comorbidities, functionality, and self-reported health which can be tracked over time would strengthen the examination of early life hypotheses in relation to older adult health.

Of course, “ideal” data do not exist. There are longitudinal cohort and panel data from the developed world by which to test hypotheses regarding early life and older adult health. While economic historians have produced several studies regarding early life conditions and older adult health in the developed world based on the collection of data from extensive historical census or registration systems,²⁵ these data are not often readily available in the developing world (with a few exceptions).²⁶ There are some cohorts in the developing world from which information on early life conditions is available, but these cohorts have not yet reached older adult ages.²⁷ Therefore, until recently it was not possible to examine hypotheses regarding the determinants of older adult health in low- and middle-income countries.

In the absence of cohort data on older adults in the developing world, country-specific data from representative surveys of older adults are available. The large sample sizes, a longitudinal approach, linkages to administrative data, quality data (probability sampling, good interviewer training, good questionnaire design, high response rates), and the possibility of replication make existing survey data attractive. With the increase in population-based studies on older adults over the last 10 years, cross-national comparative studies are now possible which can improve understanding of overall patterns in health around the world²⁸ and reveal patterns not observed in country-specific data. Interest in the fields of demography and population research have resulted in several studies on early life conditions and older adult health from the developed and, more recently, the developing world. Demographers have become increasingly interested not only in the social and behavioral but also in the biological mechanisms of early life health.²⁹

²⁴ Lin, Schaeffer, Seltzer, and Tuschen (2004) and Almond and Currie (2010).

²⁵ Bengtsson and Lindstrom (2000) and Gagnon and Mazan (2006).

²⁶ Campbell and Lee (2009).

²⁷ There are five well-known birth cohorts from studies in low- and middle-income countries that have not yet reached the age of 60: (1) the Institute of Nutrition of Central America and Panama Nutrition Trial Cohort (INTC-Guatemala), (2) the Cebu Longitudinal Health and Nutrition Survey cohort (CLHNS-Philippines), (3) the 1982 Pelotas (Brazil) Birth Cohort Study, (4) the Birth-to-Twenty (Bt20; Soweto-Johannesburg, South Africa), and (5) the New Delhi Birth Cohort (India). See for example Stein et al. (2010) or MRC Lifecourse Epidemiology Unit (2011). See Adair et al., (2013) and Batty, Horta, Davey Smith, Barros, & Victory (2009) for an example of research with these cohorts.

²⁸ National Research Council (2001).

²⁹ See for example Crimmins, Kim, and Vasunilashorn (2010).

Survey data from population studies cover different aspects of the life course and are limited in their ability to fully test early life hypotheses, especially those pertaining to *in utero* or early infancy risk factors. At a population level, genetic background may be less relevant,³⁰ although those types of data would be of interest. Most surveys do not have measures reflecting genetic background other than respondent height³¹; however, some contain information on whether a parent or sibling had diabetes, an important risk factor that can reveal either a genetic component or shared lifestyles.³² Nevertheless, strong associations between early life environmental conditions and older adult health have surfaced among older populations in low-, middle-, and high-income countries.³³ These associations have been found mostly by using retrospective and anthropometric measures of early life along with self-reported adult health outcomes which, in some instances, have been validated by biomarkers.

2.3.1 Population Studies: Early Life

Retrospective questions asked in surveys regarding SES have been shown to have strong validity,³⁴ but in some cases may not provide a sufficiently precise definition of SES to thoroughly examine its effects on older adult health. Asking respondents to rate their early SES³⁵ does not permit an understanding of the occupational status of their parents, nor do general questions about parental occupational status that result in broad categories such as “work in agriculture” (e.g., laborer, land owner, type of crop); neither can distinguish social stratification in the predominantly rural and agricultural societies of the early twentieth century. Likewise, even if parental educational attainment is available it may not be possible to distinguish among non-educated fathers which ones were economically well-off and which ones were not.

Rural or urban birth place provide a general indication of early life nutritional and infectious disease environment. It reflects family SES and parental educational attainment. In the 1930s, most populations in the developing world lived in rural

³⁰ See Case and Paxson (2010).

³¹ Eveleth and Tanner (1990).

³² American Diabetes Association (n.d.) There is a genetic component to diabetes but there is the possibility that shared lifestyles also contribute to diabetes.

³³ Beltrán-Sánchez, Crimmins, Teruel, and Thomas (2011), Brenes (2008), Brenes-Camacho and Palloni (2011), Campbell and Lee (2009); Crimmins et al. (2005), Huang and Elo (2009), Huang, Soldo, and Elo (2011), Kohler and Soldo (2005), McEniry, Palloni, Dávila, and García (2008), McEniry and Palloni (2010), McEniry (2011b), Monteverde, Noronha, and Palloni (2009), Moore et al. (1999), Palloni et al. (2005, 2006), Schooling et al. (2011), Zeng, Gu, and Land (2007), Zhang, Gu, and Hayward (2010); Victora et al. (2008); see review article McEniry (2012).

³⁴ Hauser, Tsai, and Sewell (1983).

³⁵ See for example PREHCO (2007).

areas where conditions were often precarious,³⁶ and thus those born in rural areas may have been at higher risk being exposed to these conditions.

The measurement of early childhood health in population surveys of older adults relies on retrospective questions asking respondents to rate their childhood health; these measurements have been associated with *in utero* growth³⁷ and childhood illnesses³⁸ which suggests they have some validity. Some surveys obtain information regarding variables known to be associated with childhood health such as household density, number of children under the age of 5 in the household, household composition, age of mother and birth order, all of which may be associated with increased chances of exposure to disease during childhood and long-term poor health.³⁹

Population surveys of older adults have limited measures of *in-utero* and early infancy exposure. Although researchers have criticized it as being contaminated by the influence of other life course factors,⁴⁰ birth weight is an indicator of *in utero* growth,⁴¹ but it is rarely available in population surveys of older adults. In settings such as Puerto Rico, it is almost impossible to obtain historical data from the early twentieth century on birth weight, especially for those born in rural areas where midwives delivered babies at home without recording their birth weight. Adult height partially reflects genetics but also reflects early life conditions. It is a marker of nutritional status throughout childhood and adolescence, is associated with height at early ages in some settings⁴² and is found in almost all surveys of older adults. As depicted in Figs. 1.2 and 1.3, poor *inutero* growth can lead to stunting. However, adult height may not be a sensitive measure of *in utero*/early infancy exposure to poor nutrition and infection⁴³ although it may be related

³⁶ Most historical accounts of countries such as China (Banister, 1987; Campbell, 1997; MacPherson, 2008; Zeng, Gu, & Land, 2007); India (Dyson, 1997; Guha, 2001; Ramasubban, 2008), Ghana (Patterson, 1979, 1981), Puerto Rico (Fernós Isern & Rodríguez Pastor, 1930), Chile (Garnier, Grynspan, Hidalgo, Monge, & Trejos, 1997; WHO, 2000b), Indonesia (Hull, 2008; Nitisastro, 1970), Taiwan (Barclay, 1954), South Africa (Beinart & Dubow, 1995), Barbados (Bishop, Corbin, & Duncan, 1997; West India Royal Commission, 1945), Brazil, Mexico, and Puerto Rico (Clark, 1930; Huang, Soldo, & Elo, 2011; Rodríguez de Romo & Rodríguez de Pérez, 1998; Scazufca et al., 2008) bear out the idea that in most of the selected countries, conditions during the early twentieth century in rural areas were worse in terms of nutrition, educational attainment and economic opportunities. Exceptions are Argentina and Uruguay. There were undoubtedly problems in urban areas in terms of crowding which produced disease but access to better nutrition was probably better in urban areas during the twentieth century in these settings. In terms of the demographic transition it is assumed to occur first in more urban areas and then spread to the rural areas as efforts to improve conditions in rural areas increased.

³⁷ Haas (2007).

³⁸ McEniry et al. (2008).

³⁹ Wilkinson and Marmot (2003) and Campbell and Lee (2009).

⁴⁰ Huxley, Neil, and Collins (2002), Joseph and Kramer (1996), and Rasmussen (2001).

⁴¹ Barker (1998).

⁴² Schmidt, Jorgensen, and Michaelsen (1995) and Stein et al. (2010).

⁴³ See for example Palloni et al. (2005) or Brenes (2008).

to adult health.⁴⁴ Leg length and knee height are perhaps more sensitive markers of nutritional status during early childhood,⁴⁵ but leg length is rarely measured and knee height is not captured in most surveys of older adults.

A few studies have attempted to disentangle the effects of *in utero* and early infancy conditions on adult health by using local IMR as an indicator of disease load during the first year of life, and local food prices as an indicator of access to food and nutrition during pregnancy⁴⁶; however these data are hard to obtain for the early to mid twentieth century especially in the developing world.

Season of birth is a potentially viable broad indicator of early life exposures and is easily obtained in most population studies of older adults. It has been shown to be a good indicator for early life conditions *in utero* and early infancy that may precipitate poor adult health in the developed world.⁴⁷ It is for the most part largely independent of social class of origin.⁴⁸

Availability of food has been closely related to the harvest season and to rainfall.⁴⁹ In decades past, food supply (i.e., quantity, variety, and freshness) varied sharply by season. Marked seasonality in the supply of food can lead to seasonal patterns in maternal nutrition which can then affect the unborn child *in utero*. Being born during or right after a harvest, when nutritional supplies are more plentiful, is associated with longer life.⁵⁰ Late gestation (third trimester of pregnancy) is particularly important in terms of later adult heart disease⁵¹ and diabetes.⁵²

In addition to nutritional status, season of birth may also reflect increased risk of infectious and parasitic diseases affecting both the mother and the fetus, which may work in tandem with nutritional deficiencies. Climate conditions may be favorable to the reproduction of vector borne infectious diseases thus augmenting exposure to infectious and parasitic diseases. To the extent that maternal nutritional status is harmed by infectious or parasitic diseases, normal intrauterine growth will be impaired. By the same token, as increased exposure translates into increased incidence of infectious and parasitic diseases, deterioration is expected in the

⁴⁴ Maurer (2010).

⁴⁵ Davey Smith et al. (2001), Gunnell, Davey Smith, Holly, and Frankel (1998), Leitch (1951), and Wadsworth, Hardy, Paul, Marshall, and Cole (2002).

⁴⁶ Bengtsson and Lindstrom (2000), Doblhammer (2004), Svensson, Broström, and Oris (2004), and van den Berg, Lindeboom, and Portrait (2006).

⁴⁷ Doblhammer (2004), Costa (2005), Gavrilov and Gavrilova (2005), Moore et al. (1999), Muñoz-Tuduri and García-Moro (2008), McEniry et al. (2008), Mazumder, Almond, Park, Crimmins, and Finch (2009), and Prentice and Cole (1994).

⁴⁸ Doblhammer (2004).

⁴⁹ For historical patterns of rainfall throughout the world see Food and Agriculture Organization of the United Nations (1984, 1985, 1987).

⁵⁰ Bengtsson and Lindstrom (2003), Costa (2005), Doblhammer (2004), Gavrilov and Gavrilova (2005), Prentice and Cole (1994), Gavrilov and Gavrilova (2011), and Gavrilov and Gavrilova (2012).

⁵¹ Barker (1998) and Gardiner (2007).

⁵² Ravelli et al. (1998).

quantity and quality of nutrition received by babies from breastfeeding, the main source of nutrition in early life.⁵³ In fact, being born at the beginning of seasons with more moderate temperatures is beneficial, at least with regard to risk of infectious diseases in early infancy.⁵⁴

As health and nutritional conditions improved throughout the twentieth century, the influence of seasonal differences weakened in the developed world⁵⁵ and became less relevant as an indicator of early life exposures. However, nutrition and infectious diseases still follow strong seasonal patterns in developing countries, especially around the rainy and dry seasons in tropical regions,⁵⁶ and these patterns interact with early growth and development and later health.⁵⁷ In the developing world, there are several studies with children or younger adults but only a few studies which examine older adult health based on season of birth.⁵⁸ Some differences found have been attributed to the importance of the timing of nutritional insults during late gestation⁵⁹; for example, being born in the lean season is associated with low birth weight.⁶⁰ However, season of birth is, of course, not a perfect indicator because so many other factors may be involved; for example, differences among populations in immune functions and exposure to infections or environmental risks. Season of birth has also not always produced anticipated associations with adult health and thus it remains a measure useful only in some restricted circumstances.⁶¹ Early SES,⁶² adult SES,⁶³ macro-environmental, economic, and/or weather conditions⁶⁴ could confound the effects of season of birth. Other indicators of *in utero* exposure to poor nutrition⁶⁵ in addition to more specific data on the early life environments in which people were exposed are thus needed to more fully understand the effects of early life on older adult health.

Historical data can help complement measures of early life found in surveys of older adults even if the data are not available on an individual level. One example is

⁵³ John, Menken, and Chowdhury (1987).

⁵⁴ Doblhammer (2004) and Kevan (1979).

⁵⁵ Costa (2005), Gavrilov and Gavrilova (2005), and Kevan (1979).

⁵⁶ Cole (1993), Hauspie and Pagezy (1989), Marin et al. (1996), Moore et al. (1999), Tomkins (1993), Trowbridge and Newton (1979).

⁵⁷ Moore et al. (1999).

⁵⁸ Studies done in Brazil, Gambia, India, and Bangladesh have focused either on children or younger adults (González, Goncalves, & Victora, 2009; Hawkesworth et al., 2009; Richards, Fulford, & Prentice, 2009; Veena et al., 2009). For older adults, see McEniry et al. (2008).

⁵⁹ Doblhammer (2004).

⁶⁰ Ceesay et al. (1997).

⁶¹ See for example Gamble (1980), Guerrant, Lima, and Davidson (2000), Moore et al. (1999), Moore et al. (2004), Richards et al. (2009), and Simondon et al. (2004).

⁶² Buckles and Hungerman (2008).

⁶³ Huxley, Neil, and Collins (2002) and Joseph and Kramer (1996).

⁶⁴ van den Berg, Doblhammer, and Christensen (2009).

⁶⁵ See for example Doblhammer (2003).

country-specific daily per capita caloric intake which provides a broad indicator of the nutritional environment of the overall population,⁶⁶ including maternal, infant, and childhood nutrition.⁶⁷ Nutritional status is reflected in the ability to fight infectious diseases, and very low caloric intake reflects a higher consumption of staple foods due to the inability to purchase more nourishing food and the real possibility of hunger and malnutrition.⁶⁸ Lack of adequate food affects maternal health and the mother's ability to properly nourish the unborn and infants resulting in higher infant deaths.⁶⁹ It can also leave children vulnerable to infectious diseases and poor childhood health which may have longer term consequences during adulthood.⁷⁰

2.3.2 Population Studies: Older Adult Health and Life Course Events

While most surveys of older adults have collected longitudinal data or are planning to do so, in some instances even when mortality data are available, there is often no detailed information regarding cause or timing of death in surveys carried out in the developing world. On the other hand these surveys do contain information about morbidity of chronic conditions.

Surveys of older adult health have, until recently, relied almost entirely on self-reported adult health questions to assess health status. Self-reported health questions can be problematic for a number of reasons. They try to measure disease prevalence but may conceal differences in the incidence, duration or prognosis of disease making it difficult to interpret results from analyses. Survey questions regarding heart disease may be broad and inconsistent across countries in terms of specific types of heart disease. Treatment and detection of chronic conditions have changed over the years and care must be taken when comparing historical levels of prevalence.

Self-reported questions also assume that people have good understanding of their own health either because they visit doctors on a regular basis or have access to good quality health care. Some studies show that, under certain conditions, under estimation provides slightly more conservative estimates of self-reported health but not dramatically so.⁷¹ However, morbidity data from self-reported health obtained

⁶⁶ See for example FAO (2004).

⁶⁷ Almond and Mazumder (2011).

⁶⁸ Jensen and Miller (2011) and Hunger Notes (2011).

⁶⁹ Lannoy (1963).

⁷⁰ Elo and Preston (1992).

⁷¹ See for example Baker, Stabile and Deri (2004), Banks, Marmot, Oldfield, and Smith (2006a, 2006b), Beckett, Weinstein, Goldman, and Yu-Hsuan (2000), Brenes (2008), Goldman, Lin, Weinstein, and Lin (2003), and Riosmena and Wong (2008). However, other studies suggest that in the case of Latin America and the Caribbean self-reports may grossly underestimate the true prevalence of chronic conditions (Andrade, 2008).

from surveys of older adults may be problematic in some settings. Information obtained from surveys asking if a medical doctor has ever diagnosed the respondent with a particular health condition may reflect respondents who infrequently go to a doctor or live in an area with restricted quality health care and therefore do not know if they have a particular condition rather than reflecting low prevalence of the disease. Respondents may believe they have heart disease when they actually have other conditions such as heart burn, chest pain due to pneumonia, or anxiety. Surveys may deduce illness from symptom questions, but some conditions such as heart disease and diabetes may be asymptomatic for many years. Morbidity data may also reflect the ability of a health care system to maintain life or the ability of a population to pay for better care to maintain life. The more recent inclusion of biomarkers in surveys of older adults in low and middle income countries is helpful in that, with biomarkers, it will be possible to more accurately understand the health status of adults. This will permit a more confident analysis of morbidity across time. Armed with this information, it will then be possible to better interpret the results from studies examining the effects of early life conditions on older adult health.

2.4 Approach

Cross-national patterns at the population level can be powerful in pointing to possible causal relationships.⁷² Under certain conditions, population studies can provide valid insights into associations between individual behaviors and outcomes.⁷³ Yet, the data and the array of measures for examining early life hypotheses and older adult health in population studies of older adults are clearly not optimal and limit the ability to make all of the comparisons in the road map. It is not possible to compare the health of different cohorts at similar ages across time. Securing a valid measure of early life nutritional and infectious disease environment for cross-national comparisons, the potential for underestimation of adult chronic conditions, the absence of mortality data in some instances, the diversity of countries compared, the largely untested nature of cross-national comparisons of older adults in low- and middle-income countries all present challenges in making valid cross-national comparisons. Cross national comparisons of the health of older adults in the developing world are only now beginning to appear and are mostly descriptive in nature.⁷⁴

Given these circumstances, the approach used in this study was conservative in terms of analysis but also ambitious in terms of compiling as much historical and more recent data as possible by which to make cross-national comparisons regarding the health of older adults across mortality regimes. Historical data were collected and pieced together on life expectancy, infant mortality, literacy, and

⁷² Gruber and Wise (1998).

⁷³ Greenland and Robins (1994).

⁷⁴ He, Muenchrath, and Kowal (2012).

GDP per capita and analyzed to describe trends of the early- to mid-twentieth century and to identify mortality regimes. Data from 14 major surveys of older adults from a diverse group of high-, middle- and low-income countries were compiled and then harmonized⁷⁵ to increase comparability across countries. Historical data were collected on caloric intake prior to WWII were collected to complement measures of early life found in surveys of older adults and the prevalence of adult heart disease and diabetes in the developed world after WWII on into the 1970s. In Puerto Rico, historical contextual information on socioeconomic conditions and infectious diseases from the early to mid twentieth century were also compiled to gain a better understanding of the early life socioeconomic and health conditions of older adults from one of the tip of the iceberg countries.

Given the nature of the data, several cross sectional analyses were conducted that were designed to explore basic associations between early life and older adult heart disease and diabetes.⁷⁶ A study of associations is a first step in understanding the complex and recently compiled data in an area of research that is relatively new. Longitudinal analyses which could help account for study heterogeneity were not incorporated into the study because they will have more meaning when biomarkers become available in more surveys of older adults in the developing world so that there is more confidence about analyzing the prevalence of heart disease and diabetes across time.

Issues of validity and missing values of certain measures of early life and health outcomes were examined as were underestimations of adult heart disease and diabetes. Multivariate models were used to compare differences in the magnitude and direction of associations in covariates when using adult health outcomes based on self-reports and symptom or biomarker data. The models used data from a subset of countries which had symptom data on adult heart disease and biomarker data on diabetes. Pooled country data were used in the analysis of symptom data, and models included country dummies so as to compare across countries. The models also controlled for variables important to adult health such as age, gender, education, current state of health, obesity, and adult lifestyle.

Those born in the late 1920s through the 1940s were selected to examine the hypotheses because this group had already reached the age of 60 by the year 2000 and were born in the first part of the 1930s–1960s. A series of multivariate models were then estimated based on country pooled data in order to examine basic associations between the risk of adult heart disease, diabetes, or obesity across countries. These models included most of the surveys in the cross-national data and controlled for basic variables available across all countries such as age, gender, education, and smoking.

⁷⁵ Differences in response scales and wording may affect how respondents answer questions in surveys. Harmonization uses methods to attempt to correct for these differences and to develop questions that are more directly comparable across survey instruments.

⁷⁶ See McEniry working papers (2009–2010).

Another series of multivariate models were estimated for those born in the later 1930s through the 1940s to examine associations between early life and older adult health using caloric intake and rural birthplace as indicators of early life. A series of related models were built to predict the likelihood of adult heart disease and diabetes in order to determine the degree to which the effects of early life conditions were attenuated by other important factors across the life course. Models initially controlled for age and gender, then variables from early life (e.g. caloric intake, rural/urban birthplace) were added followed by adult SES, adult lifestyle, and adult health, and whether or not the respondent had visited a doctor within the last year. Appropriate interactions between caloric intake in early life and rural birthplace and between caloric intake in adult life and adult education were included in the models.

Given the possibility of underestimation of morbidity (i.e., chronic conditions), models were estimated using not only heart disease and diabetes but also outcomes that could be more objectively measured such as obesity and functionality which are known to be associated with heart disease and diabetes. The use of health services was used as a way to examine associations in light of possible under estimation of chronic conditions. Data were imputed in some cases. Comparisons were made across mortality regimes of similar income groups, and some comparisons were made regarding historical levels of prevalence of adult heart disease and diabetes.

A similar approach of model building was used in the case of Puerto Rico for adult heart disease, diabetes, and mortality. In the analysis of SES health disparities, multivariate models were used to assess the likelihood of heart disease and diabetes for respondents with no education. These models included most countries and controlled for age and gender only.

In the case of Puerto Rico, analyses for cross-national comparisons of health inequalities used imputation methods based on procedures proposed by Rubin⁷⁷ which require that missingness is ignorable—that is, that the pattern of missingness for each variable does not depend on the value of the missing item after conditioning on other observed variables available in the data and that any parameters that may govern missingness are not related to the parameters in the models of interest. As part of the multiple imputation procedures,⁷⁸ all relevant cases were used, including item non-response, as well as target individuals who were interviewed with proxies and for whom there was no information on early conditions. To the extent that different types of missingness are predicted with the available data in (e.g. item missingness and proxy responses), the estimations for multiplying imputing missingness take into account many of the observed factors that could explain missingness. This made it possible to maximize the opportunities for satisfying the assumption of ignorable missingness.

⁷⁷ Rubin (1987).

⁷⁸ Raghunathan, Reiter, and Rubin (2003), Rubin (1987), and Van Buren, Boshuizen, and Knook (1999).

2.4.1 Mortality Regimes and Tip of the Iceberg Countries

Mortality regimes and tip of the iceberg countries were defined based on the timing, speed, and reason for mortality decline during the early- to mid-twentieth century.⁷⁹ An initial classification was obtained by examining changes in life expectancy and IMR over the period and grouping countries accordingly. Previous analyses identified some tip of the iceberg countries in the LAC region such as Puerto Rico, Costa Rica and Chile.⁸⁰ From analyses of changes in life expectancy it was thus possible to identify other countries with similar patterns of life expectancy and IMR.

Countries were then classified using two other measures: the percent of change in life expectancy during the 1930s–1960s due to factors exogenous to national income, and cumulative changes in mortality throughout the period. To obtain an estimate of the percent of change due to factors exogenous to national income, a shift analysis was conducted similar to that used by Preston to examine the relationship between income and life expectancy between 1930 and 1960.⁸¹ This analysis was redone using data from several sources⁸² along with updating data used by Preston. Life expectancy and GDP per capita data were collected from as early in the twentieth century as possible for as many countries as possible. Countries were first identified using the United Nations life expectancy numbers for the year 1960 then data on previous years were obtained from other sources. Complete data on life expectancy and GDP per capita were obtained for 12 countries for 1900, 29 countries for 1930, 36 countries for 1940, and 53 countries for 1960. When literacy rates were added, sample sizes changed slightly. The basic model examined life expectancy as a function of the natural logarithm of GDP per capita; models that included literacy rates were also estimated.

Changes in infant mortality were used as a rough proxy to obtain an estimate of cumulative mortality changes during the 1930s–1960s. As noted earlier, the growth rate of adults 60 years and older is, in part, a function of past historical circumstances. The rate of increase of the population aged 60+ during the period 1990–2025 depends on three determinants: (1) changes in fertility during 1930–1965; (2) changes in mortality before age 60 during 1930–1965; and (3) changes in mortality after age 60 during 1990–2025.⁸³ An examination of the second determinant provides an indication of the degree to which a cohort was shaped by infant and child mortality decline. Arguably, in the case of the developing countries examined here, this contributed more to the growth rate for the cohorts of the 1930s–1960s because fertility changes were much smaller during this period. Furthermore, most of the changes in mortality were concentrated at younger ages

⁷⁹ Omran (1971), Palloni (1981), and Palloni, McEniry, Wong, and Peláez (2007) proposed general classification schemes for mortality regimes.

⁸⁰ Palloni et al. (2007).

⁸¹ Preston (1976), pages 72–73.

⁸² UNESCO (1953, 1965, 1977), Preston (1976, 1980), and Maddison (2006); Appendix A.

⁸³ Palloni et al. (2005).

due to improvements in IMR and child mortality.⁸⁴ With this in mind, a rough estimate of the magnitude of the second determinant was obtained by using changes in infant mortality between 1930 and 1960, the period of the most dramatic changes in life expectancy. An alternate measure for distinguishing the magnitude of mortality changes at younger ages is annual absolute gains/reductions; annual percentage decline/increase in IMR or the magnitude of change in IMR during 1930–1960s produced a similar association (results not shown).

2.4.2 Validity of Early Life Measures and Older Adult Health

The validity of using measures such as rural birthplace as an indicator of low parental SES was assessed by examining associations between father's education and rural/urban birthplace. The validity of season of birth using quarter of birth as an indicator of nutritional status and infectious disease exposure was examined by testing the statistical significance of the association between prevalence of heart disease and diabetes according to birth quarter and place of residence during childhood. In the case of diabetes, the prevalence of the disease for those respondents with and without family members with diabetes was also compared. The convergent validity of self-reported childhood health was examined by testing the statistical association between self-reported child health and a series of questions about childhood illnesses and periods of health problems. The broad measure of childhood SES in the Puerto Rican Elderly: Health Conditions (PREHCO)⁸⁵ survey data have face validity. The convergent validity of childhood SES was examined through the association between this variable and father's education level and occupational status. These associations were tested using both the complete scale (child health, five items; child SES, three items) and the dichotomous variables created for childhood health and SES.

There were limited biomarker data based on blood samples to examine the degree by which underestimation is problematic. Thus, underestimation of adult chronic conditions such as heart disease and diabetes was assessed using Study on Global Aging and Adult Health (SAGE) data⁸⁶ because they include symptom questions on angina based on a well-tested instrument.⁸⁷ The prevalence of adult heart disease using self-reports alone was compared against the prevalence of heart disease using symptom data. Health utilization over the last year was used to control for possible underestimation of chronic conditions. Biomarker data for diabetes were available in Costa Rica and were used to examine differences in the associations between predictors of adult diabetes using self-reported diabetes and measured diabetes in multivariate models which controlled for age, gender, education, adult health, adult lifestyle, and countries.

⁸⁴ Preston (1976).

⁸⁵ McEniry, Moen, and McDermott (2013).

⁸⁶ McEniry et al. (2013).

⁸⁷ Rose (1962) and Rose et al. (1977).

2.4.3 Morbidity: Population-Level and Individual-Level Analyses

Country-specific estimates of daily per capita caloric intake prior to World War II from the Food and Agriculture Organization of the United Nations (FAO) were compared against the overall prevalence of heart disease and diabetes for each country. Age-standardized prevalence of adult heart disease and diabetes were calculated for those adults born during the late 1920s and early 1940s using the UN standard population.⁸⁸ These comparisons produced a population level estimate of associations between early life nutrition and older adult health. Historical data on the prevalence of heart disease and diabetes in the US, England, and the Netherlands were obtained from various sources⁸⁹ and compared with current levels of prevalence across countries.

Analysis using population data to reach conclusions about individuals is appropriate under certain conditions, but can be problematic due to the reliance on population averages along with other difficulties present in individual-level analyses such as selection bias, insufficient information on potential confounding covariates, and errors in measurement.⁹⁰ Therefore, multivariate models were also estimated using individual-level data. Basic pooled multivariate models controlling for age, gender, and smoking were estimated with country dummy variables. Models were also estimated, creating dummy variables for countries according to caloric intake per capita. A series of models were then estimated, first controlling for gender and age followed by education, caloric intake as an adult, use of medical services, adult functionality, and poor self-reported health. Interactions between early life caloric intake and parental SES were investigated as were interactions between late life caloric intake and respondent SES.

2.4.4 Morbidity and Mortality: Season of Birth

In the first of a series of analyses,⁹¹ logistic regression models for heart disease and diabetes were estimated for older Puerto Rican adults to better assess the total and net effects of exposure to poor nutrition and infectious diseases (using season of birth), childhood health, childhood SES, rheumatic fever (in the case of heart disease), and low knee height. In particular, there was interest in examining the degree to which the effects of childhood conditions such health and knee height are attenuated by level of exposure. The baseline models estimated the effects of the more detailed definition of exposure to poor nutrition and infectious diseases (level of exposure) on health including controls for only age and gender followed by models with childhood health and knee height controlling for age and gender. The models ultimately included

⁸⁸ Ahmad et al. (2001).

⁸⁹ See for example Ford and Giles (2003), Gordon (1964), GarcíaPalmieri et al. (1970), Wilkerson and Krall (1947), Hadden and Harris (1987), and Haris et al. (1998).

⁹⁰ Greenland and Robins (1994).

⁹¹ McEniry et al. (2008).

education, season of birth, poor childhood health, poor childhood SES, knee height, and adult obesity (included as a control in the models for diabetes and heart disease). Interactions were tested between childhood conditions (health, SES, important infectious diseases such as malaria and dengue), adult obesity, and, for diabetes models, family member with diabetes and suitable interaction terms in our final models. Results were examined by estimating models with the broader definition of exposure to poor nutrition and infectious diseases, exposure period (July–December). Finally, the best model was compared with a similar one estimated using respondents who had not lived in the countryside as a child. Imputed and non-imputed results were compared to ensure there were no important discrepancies in model estimation. To assess the actual magnitude of effects of season of birth, predicted probabilities of experiencing heart disease or diabetes for an individual with average attributes were calculated and then poor childhood conditions and adult obesity were added. The typical respondent was 66 years old, had 7 years of education, was not obese, reported that he or she was not poor and did not have poor health as a child, and had not experienced rheumatic fever (in the case of heart disease). Using comparable control variables, these analyses were followed by examining age at diagnosis of heart disease,⁹² season of birth according to higher or lower IMR,⁹³ season of birth and mortality,⁹⁴ and season of birth and migration to the United States mainland.⁹⁵

2.4.5 Mortality

For countries with mortality data, standardized mortality rates were computed across health conditions (heart disease, diabetes, and obesity) and by gender. The overall age-specific mortality rates estimated from survey mortality data were compared against 2000 life tables⁹⁶ and reported research.⁹⁷ However, not all countries had mortality data so modified Waaler-type surfaces were estimated to show expected mortality risk. A Waaler-type surface is a useful tool in depicting expected mortality risk based on height and weight.⁹⁸ These surfaces were used to first examine expected relative risk and then to calculate excess of relative risk of mortality in countries where mortality data were available.

Mortality data from individuals in Norway and Costa Rica were used to construct Waaler-type surfaces and used in cross-national comparisons with the assumption that Norwegian and Costa Rican mortality risk are suitable standards or benchmarks.

⁹² McEniry and Palloni (2010).

⁹³ McEniry (2012).

⁹⁴ McEniry (2009b, 2011c).

⁹⁵ McEniry (2012).

⁹⁶ WHO (2002).

⁹⁷ Zimmer, Kaneda, and Spess (2007) and Dow (2011).

⁹⁸ Fogel (2004), Palloni et al. (2007), Waaler (1984), and Floud et al. (2011).

The Waaler-type surface based on Waaler (1984) data was particularly robust in serving as a benchmark because the estimates of relative risk were based on a large sample of individuals. Costa Rica is a small, upper-middle-income country, but could also be a suitable standard for making cross-national comparisons because of its relatively high life expectancy.

The expected relative risk of mortality was estimated by taking the ratio of the mortality risk for a particular gender and age group given a particular weight-height and the overall mortality risk for that group. Waaler relative mortality risk across broad groups of height and weight for males and females was already available,⁹⁹ but relative mortality risk had to be first estimated using the Costa Rican data. From this information a quadratic regression model was used to estimate mortality risk as a function of height and weight and to construct Waaler-type surfaces; the resulting surfaces were then used to make cross-national comparisons. Mortality risk was estimated by country using the average height and weight of respondents with different types of health conditions (e.g., obese and non-obese adults; adults with and without heart disease or diabetes).

Waaler-type surfaces were also used to estimate excess of relative risk of mortality, deviation between the observed and expected risk, for those countries with actual mortality data. First, observed relative risk of mortality was estimated using actual mortality data for each of the countries where these data were available. The observed relative risk by gender for those born during the late 1920s and early 1940s was estimated as the crudemortality for a particular health outcome divided by overall crude mortality for that gender; the corresponding observed average height and weight was also computed. Excess of relative risk of mortality was then calculated by using the Waaler-type surface to predict weight given a particular height and observed relative risk of mortality. The calculation (observed weight—predicted weight/observed weight) produced an estimate for excess of relative risk of mortality¹⁰⁰; positive numbers indicated the observed relative risk was higher than the expected relative risk and larger numbers indicated a larger deviation from the average weight for a particular subgroup.

2.4.6 Health Inequality

Several analyses regarding SES disparities in adult heart disease and diabetes were conducted using the cross-national data on education and per capita household income level with both non-imputed and imputed data.¹⁰¹ Health disparities according to education are depicted because information regarding education attainment can be more reliable than personal income. The strategy was to first describe the differences in educational attainment between countries and then show

⁹⁹ Waaler (1984).

¹⁰⁰ Palloni and McEniry analyses carried out during 2007.

¹⁰¹ McEniry (2010a).

the disparities in the prevalence of adult heart disease and diabetes by country according to adult educational attainment across countries. Multivariate models controlling for age and gender were used to further examine associations between having no education in comparison with having at least a secondary or higher level of education and the prevalence of heart disease and diabetes.

2.5 Conclusion

A contrarian and rather pessimistic point of view was postulated regarding the future health of the unique cohorts of the 1930s–1960s in low- and middle-income countries because of their early life circumstances. Specific guiding hypotheses with a particular focus on the tip of the iceberg countries were presented along with key assumptions and a road map for analyses. Existing data are by no means ideal for fully examining the conjecture. However, large scale survey data on older adults are comprehensive in many ways, based on standards that improve the quality of the data often with high response rates. Complementing survey data with historical data and a suitable in-depth case study such as Puerto Rico increases the potential value of the data in examining the hypotheses. All of these efforts increase the chances that the results will be of value in providing broad insights into hypotheses regarding early life conditions and older adult health. A study of associations has its limitations because it cannot make inferences about causality; however, given the nature of the data, a cross-national study of associations examining patterns of health is appropriate as the first of many steps and make it possible to observe general cross-national patterns that either support or do not support the merit of the conjecture.

Data Sources, Measures, Validity, and a Description of the Older Adult Population

3

The degree to which analyses of the determinants of health using surveys of older adults are relevant hinges partially on the quality of the data – the degree to which the data accurately reflect the older adult population across countries and the degree to which the data are valid. Although there are limitations, the data are consistent with what is already known about the older adult population and about associations between adult chronic conditions and related health conditions; thus inferences can be made.

3.1 Data Sources and Their Limitations

The data come from two major sources: (1) historical data on life expectancy, infant mortality rates (IMR), literacy rates, GDP per capita, and caloric intake, and (2) comprehensive national representative surveys of older adults or household surveys. The historical data on life expectancy and IMR come from a variety of sources which are listed in Appendix A. Survey data come from a recently compiled cross-national dataset of 20 low-, middle-, and high-income countries comprising over 147,000 older adults (Table 3.1, Appendix C).¹ About 75 % of the selected adults were born prior to 1945 and the discovery of antibiotics and other therapies.

This dataset includes many of the major studies conducted on older adults in the last 10 years. The studies in the dataset and the countries represented have an aging population that is either a large proportion of the population or is rapidly growing. Most studies are representative of the older adult population either nationally, in major urban centers, or in major provinces. Some studies are representative samples of households from which older adults were selected. Most studies obtained high response rates in the first and subsequent waves (Table 3.2, Appendix C). In some cases, the complete set of survey questions were asked only of a subset of respondents

¹ See Appendix C (RELATE, 2013) for more details.

Table 3.1 Cross-national data on aging populations

Birth cohort	Observations	%
Pre-antibiotics		
1915	12,507	9
1916–1920	7,719	5
1921–1925	10,531	7
1926–1930	14,092	10
1931–1935	17,903	12
1936–1940	28,536	20
1941–1945	14,222	10
Post-antibiotics		
1946–1950	16,117	11
1951–1955	13,272	9
1956–1960	9,070	6
Total	143,969	100

Source: RELATE (2013)

Notes: Cases with missing values for age or birth year are not shown. Also not shown are the 100 cases born after 1960. The full sample of the RELATE data includes 147,278 individuals. See Appendix C for more details

(e.g., in Indonesia with the so-called main respondents and in Bangladesh with older adults some of whom received maternal child interventions in the 1970s). The selected surveys were conducted between 2000 and 2005, with the exception of Bangladesh (1996) and SAGE (2007–2009). Many of the surveys are or will be longitudinal in nature, and some have or will have linkages to administrative data. Multiple imputation methods were used in some instances to address the issue of missing values² which, in some settings, were more pronounced than in others.

There are limitations with historical data on life expectancy and IMR used to define mortality regimes. Historical data on life expectancy and IMR – especially during the early to mid twentieth century in the developing world – are not precise and may be based on differing methods of estimation. The magnitude of IMR decline can thus vary depending on the source used. It is difficult to obtain a time series of IMR and life expectancy from the early twentieth century, but estimates were obtained from trusted sources which often described the quality of the estimates (see Appendix A). When there were multiple data sources for a particular year or period, averages were taken. GDP per capita was based on the work of Maddison (2006), and missing years of GDP per capita in the early twentieth century for some countries were at times estimated using linear regression. The classification of countries into mortality regimes must be considered broad and a rough approximation.

The surveys of older adults are comprehensive in that they cover many aspects of adult life. As noted in the previous chapter, there are a number of limitations with population surveys of older adults especially in terms of using retrospective

² Raghunathan, Reiter, and Rubin (2003), Raghunathan, Solenberger, and Van Hoewyk (2007), Royston (2004, 2006), Rubin (1987), Van Buren, Boshuizen, and Knook (1999).

Table 3.2 Description of cross-national sample

Survey (survey year)	Number of respondents	Response rates (%)	
		Survey year	Follow-up survey ^c
National survey of older adults			
China CLHLS (2002)	16,064	96	96
Costa Rica CRELES (2003)	2,827	95	93
England ELSA (2004–2005)	8,780	82	75
Mexico MHAS (2001)	13,463	90	93
Multiple-SAGE (2007–2008) ^a	37,725	85 and above ^b	
Netherlands SHARE (2004–2005)	2,979	88	89
Puerto Rico PREHCO (2002–2003)	4,291	94	94
Taiwan SEBAS (2000)	1,023	68	
US HRS (2000)	12,527	85	89
Urban survey of older adults			
Multiple-SABE (2000) ^a	10,597	62–95	
National household & community			
China CHNS (2000)	6,452	83	88
Indonesia IFLS (2000)	13,260	95	91
Rural household & community			
Bangladesh MHSS (1996)	6,973	95	
1957 high school graduates			
US WLS (2004–2005)	10,317	92	
Total	147,278		

Sources: Number of respondents from RELATE (2013). This reflects all cases, even those with missing information on age or birth year. Shown are individual response rates for the survey years selected. See McEniry, Moen, and McDermott (2013) for detailed description of survey years selected and references for response rates

Notes:

^a“Multiple” means that there were multiple countries involved in the survey. SAGE countries include China, Ghana, India, Indonesia, Mexico, Russia, and South Africa. SABE countries include Argentina, Barbados, Brazil, Chile, Cuba, Mexico, and Uruguay

^bFor the most part, all SAGE surveys had a high response rate with the exception of Mexico

^cThere were some surveys for which there were as of the time of publication either no publicly available follow up survey data (MHSS, SEBAS, WLS) or no available follow-up survey data (SAGE, most of SABE). A second round of the Bangladesh Matlab Health and Socioeconomic Survey (MHSS) was being conducted in 2012. Data were to be available in May 2013 and could not be included in the cross-national data set. SEBAS data are restricted; the most recent wave of WLS has not yet been released (2010–2012); SAGE will be collecting a second round of data; most of SABE cities have not collected follow-up data

questions to ascertain early life conditions and self-report of health status. There are a few more specific observations to be made about the selected surveys.

Comparisons across countries are not perfect for reasons including:

Age: Most surveys were conducted in the early to mid 2000s, but respondents in the SAGE countries were interviewed when they were older (2007–2008) while respondents in Bangladesh were interviewed when they were younger (1996).

Generalizability: SABE respondents come from major cities and thus are not representative of the country-wide population of older adults. Many of the older adults in SABE were born in urban areas during the early to mid twentieth century. The Bangladesh survey was conducted only in a particular rural area of Bangladesh. The US-WLS respondents are a cohort of high school graduates from the late 1950s and are mostly Caucasian; respondents for the most part are slightly healthier than the general US population such as can be found in the US-HRS study which includes African Americans and Latinos. The Chinese studies (CHLHS, CHNS, and China-SAGE) cover different provinces and thus care must be taken in comparing older adult health across different samples. The Mexican studies (MHAS, Mexico-SAGE, and Mexico-SABE,) also cover different areas—the first two are nationally representative surveys while the third is a survey in Mexico City. Response rates for the Mexico-SABE survey were also lower than other SAGE countries. Chinese, Indian, and Indonesian surveys include respondents from a diverse group of provinces and states so averages at a country level may not adequately capture differences among them. Taiwan has a lower percentage of females due to the selective migration of males after World War II when the Nationalist army came to Taiwan from the Mainland.³ There are a high percentage of single older adults in Barbados.

Country exceptions: Both Taiwan and Barbados were very poor countries in the early to mid twentieth century but in contrast with the other selected countries are now high income countries. Barbados but also Costa Rica is considered to be an example of high achievers.⁴ Migration is important in countries such as Mexico, Puerto Rico and Barbados. The legacy of apartheid is an important component when examining the health of older adults in South Africa. Historical events of the early to mid twentieth century in the Russian Federation make it a complex case to examine.⁵

Early Life Conditions: Some surveys have more information than others regarding early life conditions. The US-HRS and the LAC surveys have questions regarding childhood health and ask questions regarding childhood SES which do not appear in other surveys. The Taiwan-SEBAS study has only adult height as a marker of early life conditions making it difficult to include Taiwan in all analyses.

Nutrition and Diet: Individual-level data on caloric intake are impossible to obtain across all countries for those born prior to World War II from these surveys. As noted in the previous chapter country-specific data provide rough measures of early life nutritional environment. Few of the surveys have detailed enough information on adult diet to be able to fully examine the effects of a more enriched nutritional environment and a sedentary lifestyle later in older adulthood either on a country-specific level and or on a cross-national perspective. CHNS offers a wide spectrum of information on nutrition at older ages but most other surveys of older adults do not.

³ Weinstein and Goldman (2003), page 18.

⁴ Mehrotra and Jolly (1997).

⁵ Anderson and Silver (1986) and Prokhorov (2005–2009).

Other: Information such as migration from rural to urban (difficult to obtain for all countries using these survey data). There is also inconsistent information about adult occupation which limits the ability to disentangle the effects of early life conditions from older adult conditions. Cultural idiosyncrasies in dietary habits and customs and critical events throughout the life course important to health are often not adequately captured in these surveys.

3.2 Measures

A brief description of the measures used in analyses appears below; a more detailed description appears elsewhere.⁶

3.2.1 Mortality Regimes and Tip of the Iceberg Countries

Table 3.3 describes the initial classification of countries into mortality regimes based on descriptions from the literature⁷ and an examination of sources of historical life expectancy and infant mortality across the twentieth century (see Table 2.1 for reference). Average yearly gain in life expectancy was used to classify countries into broad mortality regimes according to the pace and timing of mortality decline during the 1930s–1960s:

1. Very early – High income countries which experienced a more graded mortality beginning in the late 1800s;
2. Early: Developing countries which also experienced a more graded mortality decline prior to the 1920s (mostly upper middle income countries now);
3. Mid-paced: Mostly smaller developing countries which experienced a quicker pace of mortality decline around the late 1920s through the 1940s – tip of the iceberg (mostly upper middle income countries now);
4. Late: Mostly larger developing countries that experienced more widespread mortality decline in the 1940s; urban areas could be tip of the iceberg (mostly upper middle income countries now);
5. Very late: Very poor countries that experienced shifts in mortality during the 1950s–1960s (mostly low income countries and lower middle income countries now).

The table shows the two distinguishable parts to the period of the 1930s–1960s—prior to the advent of antibiotics and other therapies (1930s–mid 1940s), and after (post 1945) when there was a dramatic mortality decline. The average yearly gain in life expectancy was larger in the 1940s–1960s than in the 1930s–1940s, and important variations within mortality regimes exist. Reductions in IMR show a similar pattern, although there are some exceptions. Linear regressions by year for IMR show similar differences in the magnitude of decline across time (results not shown).

⁶ McEniry, Moen, and McDermott (2013).

⁷ Palloni, McEniry, Wong, and Peláez (2007).

Table 3.3 Patterns of mortality decline from 1930–1960

Regime	Life Expectancy						IMR							
	Year			Pace			Year			Pace				
	1930	1940	1960	30–40	40–60	30–60	Level	1930	1940	1960	30–40	40–60	30–60	Level
Very early decline, graded (late 1800s)														
Netherlands	66	62	73	-0.40	0.55	0.23	Vhigh	51	39	18	1.20	1.05	1.10	Vlow
England	61	62	71	0.08	0.47	0.34	Vhigh	60	57	22	0.30	1.75	1.27	Vlow
US	61	64	70	0.27	0.30	0.29	Vhigh	61	49	25	1.20	1.19	1.19	Vlow
SA-whites	66	68	70	0.20	0.12	0.15	Vhigh	67	50	30	1.70	1.00	1.23	Vlow
Early decline, graded (prior to 1920s)														
Argentina	53	56	65	0.30	0.45	0.40	High	100	90	62	1.00	1.40	1.27	Low
Uruguay	50	58	68	0.80	0.50	0.60	High	100	86	47	1.40	1.95	1.77	Low
Cuba	42	45	64	0.30	0.95	0.73	Mid	113	104	59	0.90	2.25	1.80	Low
Early decline, mid-paced (1920s–1940s)														
Puerto Rico	40	45	69	0.50	1.20	0.97	Mid	133	114	43	1.90	3.55	3.00	Mid
Costa Rica	42	49	62	0.70	0.65	0.67	Mid	155	132	71	2.30	3.05	2.80	Mid
Taiwan	41	43	68	0.25	1.21	0.89	Mid	181	158	45	2.30	5.65	4.53	Mid
SA-blacks	38	40	59	0.20	0.93	0.68	Mid	197	180	129	1.70	2.55	2.27	High
Chile	35	38	57	0.30	0.95	0.73	Mid	234	217	125	1.70	4.60	3.63	High
Early/late decline, rapid (1930s–1940s)														
Mexico	34	39	57	0.50	0.90	0.77	Low	144	132	81	1.20	2.55	2.10	Mid
Brazil	34	37	55	0.30	0.90	0.70	Low	262	239	117	2.30	6.10	4.83	High
Barbados	38	40	66	0.20	1.30	0.93	Low	231	180	60	5.10	6.00	5.70	High

Later decline, rapid (1950s)

India	31	35	46	0.41	0.53	0.49	Vlow	226	188	140	3.80	2.40	2.87	Vhigh
Bangladesh	31	35	41	0.41	0.31	0.34	Vlow	226	220	174	0.60	2.30	1.73	Vhigh
China	30	32	45	0.20	0.63	0.49	Vlow	300	290	121	1.00	8.45	5.97	Vhigh
Ghana	30	31	46	0.10	0.75	0.53	Mid	240	230	124	1.00	5.30	3.87	Vhigh
Indonesia	33	40	43	0.74	0.13	0.33	Vlow	294	290	166	0.40	6.20	4.27	Vhigh

Sources: See Appendix A for sources of life expectancy and infant mortality. There are different points of view regarding life expectancy in some countries and when it began to consistently improve (see Riley, 2005a). Analyses using 1970 as the ending point of the period shows most major changes occurred during the 1940s–1950s

Notes: SA = South Africa. Pace is the average annual increase (decrease) in life expectancy (IMR) over the identified period. The Russian Federation is a more complex case and although it does not appear in the table it has been roughly classified as a late and rapid decline regime based on historical accounts (Anderson & Silver, 1986; Prokhorov 2005–2009)

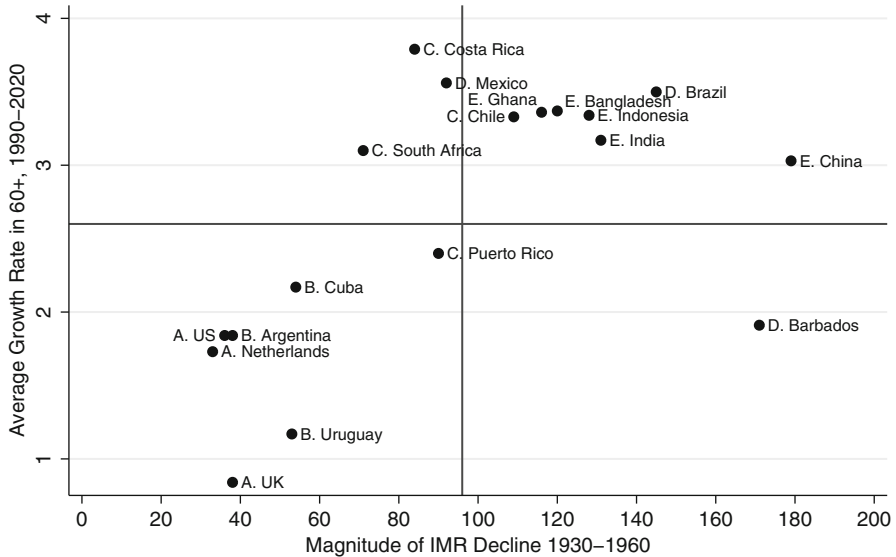


Fig. 3.1 Association between growth rate of the population aged 60 years and older and change in IMR, 1930–1960. *Note:* Horizontal and vertical lines are plotted at average values. Shown is the period 1930–1960. Although the graph does not include the 1960s, the association between growth rate and magnitude of IMR decline does not dramatically change when using a slightly expanded time period, 1930–1970. The magnitude for India, Bangladesh, and Mexico shown above are slightly different from what can be computed from Table 3.3 because of different sources used. However, even with different sources the nature of the association with growth rate does not change (*Source:* Author's calculations)

The earlier regimes of the developed world and countries like Argentina and Uruguay experienced smaller gains in life expectancy than did the mid-paced regimes. The mid paced regimes (tip of the iceberg countries) are the small group of poor countries which experienced a more rapid pace of improvement in the late 1920s through the mid 1940s. Brazil and Mexico, classified as late regimes experienced significant and rapid mortality decline throughout much of the country during the late 1940s, but experienced improvement in urban areas earlier.⁸ Urban areas within these late regimes could also be considered tip of the iceberg settings. The very late regimes, with the exception of Bangladesh, showed larger reductions in IMR towards the later part of the 1930s–1960s.

Changes in infant mortality during the 1930s–1960s were used as a rough proxy to indicate cumulative changes in mortality for those born during this period and as a way to further identify mortality regimes. The strong association between changes in infant mortality between 1930 through 1960 with the growth rate of those 60 years and older (Fig. 3.1) suggests the appropriateness of the proxy. Countries with a higher growth rate of those 60 years and older are also countries with a larger magnitude of change in IMR in the period 1930s–1960s.

⁸ López-Alonso (2007).

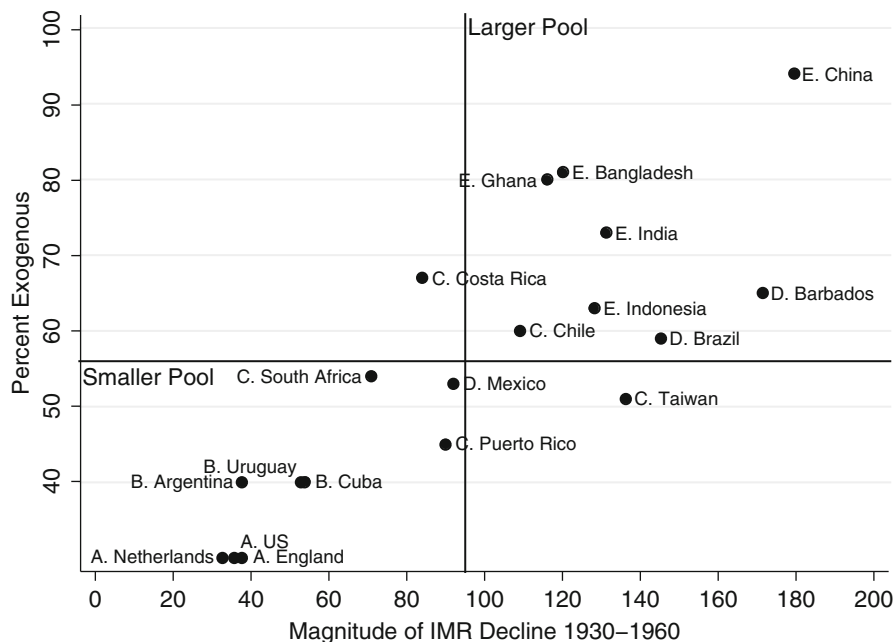


Fig. 3.2 Categorization of countries into mortality regimes. *Note:* Shown are countries according to the nature of mortality decline during the 1930s–1960s: timing and pace (magnitude of IMR) and reason (decline due to factors exogenous to improvements in standard of living). *Horizontal* and *vertical* lines are plotted at average values. Moving from *left* to *right*, cohorts are increasingly characterized by their survival of poor early life conditions. Shown are mortality regimes: *A* Very early, *B* Early, *C* Mid, *D* Late, *E* Very late. Shown is the period 1930–1960. The pattern of countries is similar when using a slightly expanded time period, 1930–1970, which was used to included data from the 1960s. Magnitude of IMR decline can vary depending on source used. The magnitude for India, Bangladesh, and Mexico shown above are slightly different from what can be computed from Table 3.3 because of different sources used. However, the resulting categorization of countries in mortality regimes remains the same (*Source:* Author’s calculation)

A re-examination of Preston’s shift analysis was used to identify the percent change in life expectancy due to factors exogenous to income in the period of the 1930s–1960s.⁹ This quantity along with changes in infant mortality is depicted in Fig. 3.2. To some degree, the findings provide support for the classification of mortality regimes presented in Table 3.3. On the x-axis, the magnitude of the decline in IMR across countries is smaller (less than 60) for the very early regimes than it is for the mid-paced to later regimes (greater than 60). The y-axis shows, as expected, that a high proportion of the reason for mortality decline during the period of the 1930s–1960s can be attributed to public health interventions including medical advances; this proportion ranges from about 30 % in the US, Netherlands, and England and Wales to nearly 100 % in China. The early, graded mortality decline regimes cluster in the left-hand portion of the graph; the early, mid-paced regimes appear

⁹ Preston (1976); see also McEniry (2009c).

toward the center of the graph; and the later, rapid decline countries appear on the right-hand side of the graph. When countries are grouped according to the average yearly decrease in IMR between 1930 and 1960, the pattern remains the same except for Bangladesh which becomes more of an outlier (not shown). The classification is a reasonable one although it is rough at best because the data on early life expectancy and IMR from the early twentieth century for some developing countries are imprecise and often incomplete making it difficult to obtain a continuous time series.

Older adults in tip of the iceberg countries were born in the late 1920s through early 1940s. Representative surveys of older adults from Costa Rica, Puerto Rico, Taiwan and South Africa were used for the tip of the iceberg countries in the mid-paced mortality regimes. In the case of the so-called urban tip of the iceberg sample within countries late mortality regimes of Barbados, Brazil and Mexico, the SABE survey was used; SABE respondents reflect urban dwellers: older adults born in urban areas during the early to mid twentieth century and older adults who migrated at some point in time from rural areas to urban areas.

3.2.2 Selected Measures of Early Life Conditions

Rural birthplace, as reflected by questions asked of respondents regarding their birthplace and residence during childhood, was used as a proxy for low parental socioeconomic status (SES).¹⁰ Historical country-level caloric intake was used as a crude measure reflecting early life nutritional environment¹¹; countries were grouped as:

- Low: less than 2,100 cal reflecting severe nutritional deficiency,
- Mid: 2,100 but less than 2,800,
- High: 2,800 or higher

The low-caloric countries of the 1930s were very late mortality regimes (early twentieth century); these countries are now lower- middle-income or low-income countries. The mid-caloric countries were mid-paced and late regimes, and now mostly upper middle income countries. The high-caloric countries were early mortality regimes; these countries are now upper-middle-income or high-income countries.

Caloric intake was also used to reflect current nutritional environment for older adults; a dichotomous variable was created to classify countries with a good level of intake (2,700 daily caloric intake per capita).

For analyses with only Puerto Rico, season of birth was used as the primary marker of exposure to early life nutrition and infectious disease along with low knee height, self-reported child health and self-reported child SES as additional measures.¹² Different levels of exposure were defined according to the degree of

¹⁰ A more suitable measure would have been questions regarding the educational attainment of the respondent's father or mother. An insufficient number of countries in the sample collected data on parental education. Where available in the cross-national data, rural birthplace was strongly associated with lower educational attainment. Thus, birthplace was used as a proxy for low parental SES. The limitations of using rural/urban birthplace are that it is very broad and there may be important rural and urban differences in epidemiology environment which confound the meaning of rural birthplace.

¹¹ Food and Agriculture Organization of United States (1946).

¹² McEniry, Palloni, Dávila, and García (2008).

overlap between the third trimester of gestation (calculated from month of birth) and the months of the slack season (July through December) in Puerto Rico during the early to mid twentieth century. An indicator of exposure to poor nutrition and infectious diseases, level of exposure, was defined as follows: Full exposure (fourth quarter of birth) means that the third trimester fell completely within the slack period, partial exposure means that the third trimester of gestation fell partially within the window defined by the slack months either early (third quarter) or late (first quarter), and no exposure during the third trimester was reserved for those whose third trimester of gestation fell completely outside the window of slack months. Binary variables were created to represent levels of exposure, with the reference group being the no exposure group. A broader definition of exposure to poor nutrition and infectious diseases was also created in model estimation called exposure period, which identified whether the respondent had been born during the lean season after the sugar cane harvest (July–December).

3.2.3 Adult Lifestyle and SES

A harmonized measure for smoking and drinking behavior was created across countries.¹³ Questions regarding exercise varied considerably across countries

¹³ Smoking was defined as only cigarette smoking for England, US-HRS, US-WLS, Puerto Rico-PREHCO (100 cigarettes or more), Taiwan, Mexico-MHAS, and China-CHNS. In the remaining countries, smoking was defined in the following manner:

Netherlands: Respondent smoked cigarettes, cigars, cigarillos, or a pipe daily for at least one year.
 SABE cities: General smoking question. Follow-up question asks about number of cigarettes, cigars, or pipes smoked per day.

Costa Rica: Respondent smoked more than 100 cigarettes or cigars during his/her lifetime.

SAGE countries: Respondent smoked tobacco products such as cigarettes, cigars, pipes, chewing tobacco, snuff, or smokeless tobacco.

Bangladesh: Respondent smoke hookah, biri, cigarettes, or cigars.

China-CLHLS: General smoking question.

Indonesia: Respondent smoked cigarettes or cigars.

Exercises refers to the percent of respondents who indicated that they participate in some type of exercise. In US-HRS, SABE cities, Costa Rica, Puerto Rico, and Mexico-MHAS, this is defined as vigorous physical activity (including sports, heavy work, or other physical work) three or more times a week. In the Netherlands, England, and US-WLS, this is defined as vigorous physical activity, without a specified timeframe. In the remaining countries, exercise was defined in the following manner:

SAGE countries: Respondent participates in vigorous-intensity activity that causes large increases in breathing or heart rate (e.g., heavy lifting, digging, chopping wood) for at least 10 min continuously.

Taiwan: Respondent participates in exercise (not defined further).

Bangladesh: Survey does not ask about exercise behaviors.

China-CLHLS: The type or degree of exercise is not specified in survey.

China-CHNS: Respondent participates in heavy or very heavy physical activities during the work day in a week (e.g., farmer, athlete, dancer, steel worker, lumber worker, mason, etc.).

Indonesia: Survey does not ask about exercise behaviors.

making comparability difficult, but a variable was created which indicated whether a respondent participated in some type of rigorous exercise.¹⁴ Visits to a medical professional within the last 12 months were captured by means of a dichotomous variable indicating at least one visit.¹⁵ Years of education completed were obtained from most surveys by asking the respondent, but in some instances, the calculation of years of education was based on categorical variables for education level. Per capita income was calculated by dividing household income by household size (or in the case of SABE, the number of dependents). Taking 2000 to be the year of interest, each country's annual per capita income was first converted to local currency using the GDP deflator from the World Bank World Development Indicators and then converted to purchasing power parity (PPP) international dollars for the year 2000 using the PPP conversion table published by the World Health Organization.¹⁶ A wealth indicator was created based on household characteristics and the number of assets the respondent owned if information was obtained on these items; this indicator varies slightly from country to country as country-specific surveys asked some what different questions regarding household assets.

3.2.4 Adult Health Outcomes

All surveys had a question asking respondents to rate their current state of health, although surveys used a different scale to define self-reported health as excellent to poor health. A harmonized variable for poor self-reported health was created for comparisons across countries.¹⁷

Although wording of questions differed slightly, most surveys asked whether or not respondents had ever been diagnosed by a doctor or medical professional with a list of specific diseases. The self-reported heart disease question in almost all countries referred specifically to the most common heart diseases, coronary heart disease (CHD) or coronary artery disease (angina, heart attack or myocardial infarction).

¹⁴ Drinks refers to the percent of respondents who indicated that they drink alcoholic beverages. The timeframes for drinking habits varied, anywhere from drinking in the last 3 months (SABE cities, Puerto Rico), the last 6 months (Netherlands, Taiwan), the last year (England, China-CHNS), to anytime in their lifetime (US-HRS, US-WLS, Costa Rica, Mexico-MHAS). In the remaining countries, drinks was defined in the following manner:

SAGE countries: Respondent drank an alcoholic beverage in their lifetime (past drinker) or in the last 30 days (present drinker).

Bangladesh: Survey does not ask about drinking behaviors.

China-CLHLS: Respondent drinks alcohol at present or drank alcohol in the past.

Indonesia: Survey does not ask about drinking behaviors.

¹⁵ Visits to a doctor refers to the percent of respondents who indicated that they visited a medical doctor at least once in the previous year. The Indonesian survey included any visits in a 5-year time period.

¹⁶ McEniry (2008) and RELATE (2013).

¹⁷ See McEniry, Moen, and McDermott (2013).

However, in some surveys the question was worded broadly to include other heart problems or worded in a very general way to identify heart disease. For example, the self-reported question in the SAGE survey asked only about angina while the CRELES, SEBAS, SAGE, and most recently the CHNS surveys had biomarkers or symptom questions which helped address the problem of underestimation of self-reported heart disease and diabetes. Questions on symptoms based on the Rose questionnaire¹⁸ were used to improve the estimation of prevalence in the SAGE surveys. Biomarker data based on blood samples were used to complement self-reported diabetes in Costa Rica. Age-standardized prevalence rates were calculated in some cases.

A composite index for overall adult health was constructed based partially on the approach used in the creation of the internationally recognized generic indicator of health status, the Short-Form 12 (SF-12), a series of 12 questions which measures physical and mental health dimensions based on 3- or 5-item scales. Its validity and reliability have been well tested across many different countries.¹⁹ The SF-12 questions were not asked in all the surveys; however, it was possible to create an indicator of “good health” that was objective in nature rather than relying, for example, solely on questions of self-reported health. The indicator of health status included three dimensions: (1) self-reported health; (2) body mass index (BMI); and (3) frailty (functionality). To combine the three dimensions of health into a composite index of good health, a simple rule was followed in the SF-12: for each respondent, the non-missing number of responses were summed for all three dimensions of health and then all responses were rescaled on a 0–100 scale, normalizing the scale to the minimum and maximum sum possible within the non-missing items. To have a valid score for health, respondents had to have at least two out of the three responses. Scores closer to 100 reflect less difficulty with functionality, more normal BMI and better self-reported health. The composite measure has shown good construct validity.²⁰

Categories for adult weight were defined using international standards.²¹ The body mass index was calculated using height and weight. Adult obesity was defined as having a body mass index equal to or greater than 30.

Functionality was based on ADLs developed by Katz²² and disability scales developed by Nagi.²³ There is no consensus on the definition of frailty,²⁴ but physical limitation questions were used. ADLs reflect impairments associated with underlying conditions that induce physiological limitations and deterioration; they provide a useful benchmark to calibrate demand for care, assistance, and support.

¹⁸ Rose (1962) and Rose et al. (1977).

¹⁹ McDowell (2006).

²⁰ McEniry (2008).

²¹ Centers for Disease Control and Prevention (CDC) (2012).

²² Katz and Akpom (1976).

²³ Nagi (1976).

²⁴ Bergman et al. (2007) and Rockwood and Mitnitski (2007).

ADLs are good probes of physical functioning, particularly lower body functionality,²⁵ and reflect impairment created by chronic conditions as well as cognitive and affective functioning.²⁶ ADLs were harmonized according to Pluijm et al.²⁷ by selecting items in common across most countries (dressing, bathing, toileting, and transferring). In the case of countries which did not have a particular item, items were constructed using physical performance measures following guidelines developed by Pluijm et al. Country-specific Cronbach alpha and the Kuder-Richardson coefficient of reliability²⁸ were used to test the internal consistency and the reliability of the items. A dichotomous variable was defined for reporting at least one ADL (1 = at least one difficulty, 0 = none) and then the construct validity was assessed by examining the country-specific relationship between disability and age group. Results from harmonizing the ADLs show very good country-specific reliability and validity of the harmonized ADL measure. In all countries, internal consistency was very high (kr20 ranged from 0.66 to 0.93), age was significantly associated with reporting at least one ADL, and there were significant associations between poor self-reported health and disability. In most countries there were significant associations between disability and adult heart disease, diabetes, and obesity.²⁹

The Waaler-type mortality surfaces for gender from Waaler (1984) data and CRELES data were based on the following models (numbers are rounded):

Waaler 1984 mortality surfaces:

Waaler male

$$\ln(RR) = 6.30 + (1.96 * 10^{-2} * W) + (-6.54 * 10^{-2} * H) + (4.69 * 10^{-4} * W^2) + (2.71 * 10^{-4} * H^2) + (-5.52 * 10^{-4} * H * W)$$

Waaler female

$$\ln(RR) = 2.44 * 10^1 + (-1.67 * 10^{-2} * W) + (-2.75 * 10^{-1} * H) + (5.11 * 10^{-4} * W^2) + (8.72 * 10^{-4} * H^2) + (-3.72 * 10^{-4} * H * W)$$

CRELES mortality surfaces:

CRELES male

$$\ln(RR) = 1.23 * 10^2 + (4.95 * 10^{-1} * W) + (-1.71 * H) + (3.02 * 10^{-3} * W^2) + (6.38 * 10^{-3} * H^2) + (-5.65 * 10^{-3} * H * W)$$

CRELES female

$$\ln(RR) = 1.20 * 10^2 + (-7.52 * 10^{-2} * W) + (-1.56 * H) + (1.43 * 10^{-3} * W^2) + (5.24 * 10^{-3} * H^2) + (-5.96 * 10^{-4} * H * W)$$

²⁵ Smith, Branch, and Scherr (1990).

²⁶ Stump, Clark, Johnson, and Wolinsky (1977), Wray, Herzog, and Park (1996), and Wray and Lynch (1998).

²⁷ Pluijm et al. (2005).

²⁸ Kuder and Richardson (1937).

²⁹ See McEniry (2011a) for more detail.

Where mortality data were available, excess of mortality risk and standardized death rates were calculated based on health outcomes such as heart disease, diabetes, and obesity. Excess of relative risk of mortality (deviation between the observed and expected risk) was calculated using Waaler-type surfaces in the following way. First, observed relative risk of mortality was estimated using actual mortality data for each of the countries where these data were available. The observed relative risk by gender for those born during the late 1920s and early 1940s was estimated as the crude mortality for a particular health outcome divided by overall crude mortality for that gender; the corresponding observed average height and weight was also computed. Excess of relative risk of mortality was then calculated by using the Waaler-type surface to predict weight given a particular height and observed relative risk of mortality. The calculation $(\text{observed weight} - \text{predicted weight})/(\text{observed weight})$ produced an estimate for excess of relative risk of mortality³⁰; positive numbers indicated the observed relative risk was higher than the expected relative risk and larger numbers indicated a larger deviation from the average weight for a particular subgroup.

3.3 Validity

3.3.1 Birthplace and Parental SES

There is some indication that rural/urban birth place as a proxy for parental SES has validity. In this study, a very strong association between rural birthplace and low parental educational attainment was found across a diverse group of countries (Table 3.4, Panel A). Excluding Bangladesh where the survey was administered only in rural areas, 51–88 % of older adults were born or lived in rural areas as a child and have fathers who had no formal education. This association is not surprising given the conditions in many rural areas during the early twentieth century in low- and middle-income countries where educational opportunity was limited and not encouraged as compared with urban areas.³¹ Similar to other studies where height was shown to reflect better SES conditions,³² taller individuals tended to have parents with more education (Panel B) and have more education themselves (results not shown). Individuals whose fathers had more education also tended to have more education as adults (Panel C).

Table 3.5 shows the most salient bivariate associations between no schooling for respondent's mother and other childhood and adult conditions. Of particular note are the consistent strong associations between mother's lack of education and low height, knee height, poor childhood health, the respondent being born and living in

³⁰ Palloni and McEniry analyses carried out during 2007.

³¹ Take the example of Puerto Rico (Clark, 1930).

³² See for example Case and Paxson (2010), Davis-Kean (2005), Kuh and Wadsworth (1989).

Table 3.4 Association between father's parental education and being born in a rural area prior to 1945 (pre-antibiotic cohorts)

Regime/country	Father's education				p-value
	No schooling	Primary	Secondary	Higher	
Panel A: Rural born (%)					
<i>Very early</i>					
US-WLS		31	13	5	0.000
<i>Mid</i>					
Puerto Rico	71	59	29		0.000
South Africa	51	29	22		0.000
<i>Late</i>					
Mexico-MHAS	60	34	11		0.000
Mexico-SAGE	40	27	10		0.000
Russian Federation	60	47	26		0.000
<i>Very late</i>					
Bangladesh	99	99	95		0.000
China-SAGE	64	42	22		0.000
Ghana	65	45	30		0.000
India	85	67	46		0.000
Indonesia	88	79	55		0.000
Panel B: Height (avg)					
<i>Very early</i>					
US-HRS	166	169	170	171	0.000
US-WLS		170	171	172	0.001
<i>Mid</i>					
Puerto Rico	157	160	160		0.000
South Africa	156	157	161		0.000
<i>Late</i>					
Mexico-MHAS	159	161	163		0.000
Mexico-SAGE	154	156	157		0.012
Russian Federation	162	162	163		0.226
<i>Very late</i>					
Bangladesh	153	155	154		0.002
China-SAGE	157	159	161		0.000
Ghana	161	162	160		0.065
India	156	157	156		0.229
Indonesia	152	153	155		0.000
Panel C: Years education (avg)					
<i>Very early</i>					
US-HRS	8	12	13	15	0.000
US-WLS		13	14	15	0.000
<i>Mid</i>					
Puerto Rico	6	10	12		0.000
South Africa	3	7	10		0.000
<i>Late</i>					
Mexico-MHAS	2	5	10		0.000
Mexico-SAGE	3	6	11		0.000
Russian Federation	7	9	12		0.000

(continued)

Table 3.4 (continued)

Regime/country	Father's education				p-value
	No schooling	Primary	Secondary	Higher	
<i>Very late</i>					
Bangladesh	1	3	4		0.000
China-SAGE	4	7	10		0.000
Ghana	2	6	8		0.000
India	2	5	9		0.000
Indonesia	2	6	10		0.000

Source: RELATE (2013), unweighted

Notes: Shown in the table are averages (% born in rural area, height, years of adult education) for each level of father's education. For developing countries, secondary and above level of education was combined into the "secondary" category that appears in the table. Example: In Puerto Rico, 71 % of older adults with fathers with no education were born in rural areas compared with 29 % of older adults with fathers with at least a secondary level of education. Also shown are p-values from either chi-square (Panel A) or analysis of variance (Panels B–C). Mother's education and adult height produced similar results but are not shown in the table. Comparisons using US surveys also included educational levels higher than high school which are not shown in the table. For US-WLS, 5% of older adults with fathers with higher education were born in rural areas (Panel A). Older adults from US-WLS and US-HRS with fathers with higher education had an average height of 171 and 172 cm respectively (Panel B). Older adults from US-WLS and US-HRS who had fathers with higher education had an average 15 years of formal education (Panel C)

a rural area as a child, and the respondent's education. Significant associations between the mother's lack of education and respondent being underweight appeared in Mexico, Indonesia, and Bangladesh and the mother's lack of education and being overweight or obese in Indonesia. Mother's education was associated with the respondent's age of death in Puerto Rico.

3.3.2 Puerto Rico

Several analyses to examine the validity of season of birth and childhood health were carried out using the Puerto Rican Elderly: Health Conditions (PREHCO) study (n = 4291, waves 2002–2003 and 2006–2007), historical data on nutrition, infectious diseases, infant mortality and other early life conditions in Puerto Rico,³³ and looking at previous research regarding Puerto Rico.³⁴ The validity of birth

³³ See for example Fernós Isern (1932, n.d.), Garrido Morales (1935, 1941), Ortiz (1927, 1931), and US Census Bureau (1932).

³⁴ Palloni, McEniry, Dávila, and García Gurucharri (2005).

Table 3.5 Bivariate associations between “no schooling” for respondent’s mother and other childhood and adult variables

Variables	Mid-paced		Late	Very late	
	Puerto Rico (n = 2,693)	Costa Rica (n = 1,317)	Mexico (n = 5,440)	Indonesia (n = 3,495)	Bangladesh (n = 1,879)
<i>Childhood</i>					
Low height	11.3***	11.1***	80.2***	17.5***	11.4***
Low knee height	20.7***	1.9	0.3	–	–
Poor child health	12.7***	15.0***	0.3	–	–
No school-father	595.3***	–	2,100***	1,400***	364.1***
Rural	117.8***	2.1	170.1***	54.8***	40.2***
<i>Adult</i>					
Education	284.1 (2)***	97.3 (2)***	995.9 (2)***	50.3 (3)***	143.7 (3)***
Heart	5.0*	2.4	0.1	–	0.1
Diabetes	1.5	0.8	0.1	–	3.9*
Underweight	3.5	0.4	7.7**	62.9***	4.0*
Overweight	3.5	2.0	2.5	34.8*** ^a	2.0
Obese	1.5	1.5	2.4	26.2*** ^a	0.1
Dead	10.9***	1.4	0.1	13.7*** ^a	0.4

Source: McEniry (2010b, Table 2)

Notes:

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. Shown are chi-square associations with degrees of freedom in parentheses. Unless specifically noted in parentheses degrees of freedom are equal to one

“–” means that the indicated variable was not asked in the country-specific survey

^aMore education is associated with overweight, obesity, and death in Indonesia

quarter (season) and place of residence during childhood as indicators of maternal nutritional status and the extent to which the unborn child is exposed to higher risk of poor nutrition *in utero* was examined by testing the statistical significance of the association between birth quarter and place of residence and heart disease and diabetes (Table 3.6). There were statistically significant differences between prevalence of heart disease by level of exposure to poor nutrition and infectious diseases for those who lived in the countryside as a child, $\chi^2(3) = 11.44$, $p = 0.010$ but not for those who did not. In the case of diabetes, significant associations appeared only in those respondents who did not have diabetes and had no family member with the disease ($\chi^2(3) = 9.48$, $p = 0.024$).

The convergent validity of self-reported childhood health was examined by testing its statistical association with a series of childhood illnesses and periods of health problems as a child. The convergent validity of childhood SES was tested by examining the association between a general question regarding childhood SES and the father’s education level and occupational status (Table 3.7), which showed that self-reported childhood health (5-item scale) was statistically associated with the more prevalent serious diseases in Puerto Rico from the 1920s through the early

Table 3.6 Prevalence of heart disease and diabetes according to seasonal exposure and place lived during childhood

Condition	Level of exposure (birth quarter)				χ^2 (df)	p-value ^a
	None (2nd)	Partial early (3rd)	Full (4th)	Partial late (1st)		
<i>Heart disease</i>						
Rural	14	19	24	20	11.44 (3)	0.010
Urban	14	16	15	13	1.72 (3)	0.632
<i>Diabetes—no family member with diabetes</i>						
Rural	17	26	21	13	9.48 (3)	0.024
Urban	12	13	14	15	0.30 (3)	0.960
<i>Diabetes—family member with diabetes</i>						
Rural	41	40	42	41	0.35 (3)	0.951
Urban	43	40	35	38	2.03 (3)	0.565

Source: Puerto Rican Elderly: Health Conditions project (PREHCO 2007), imputed, unweighted; all 60- to 74-year-olds born in Puerto Rico. Published with permission from *Journal of Gerontology* (McEniry, Palloni, Dávila, & García, 2008)

Notes: These results are unweighted. Sample sizes: lived as a child in the countryside (n = 1,457), did not live as a child in the countryside (n = 1,147), lived in the country side as a child and had a family member (parents or siblings) with/without diabetes (n = 869, n = 588), did not live in the countryside as a child and had a family member (parents or siblings) with/without diabetes (n = 626, n = 521)

^aAll p-values were calculated from the chi-square analysis

1940s such as malaria ($\chi^2(4) = 25.87, p \leq 0.001$), typhoid fever ($\chi^2(4) = 9.52, p \leq 0.05$), and dengue fever ($\chi^2(4) = 23.73, p \leq 0.001$). This was also true when self-reported childhood health was dichotomized.

3.3.3 Underestimation of Chronic Conditions

It is clearly evident that obtaining the best possible measurement of older adult health from surveys is critical to better understanding the precursors of health. The underestimation of conditions such as heart disease and diabetes produces conservative results especially in some low- and middle-income countries. Yet, to some degree while they may lack precision, self-reported questions regarding adult heart disease and diabetes have general validity.

Strong bivariate associations appeared between heart disease and diabetes and conditions or behaviors which have been shown in previous studies to be associated with heart disease and diabetes.³⁵ These included hypertension, stroke, obesity, functionality, and exercising (Table 3.8). For those reporting heart disease, about 65 % also reported hypertension (versus 35 % for those without heart disease), 25 %

³⁵ Kuh and Ben-Shlomo (2004).

Table 3.7 Associations between self-reported childhood health and childhood illnesses, and self-reported childhood SES and father education and occupation

Variable	Scale ^a chi-square	Scale (0/1) chi-square
<i>Self-reported childhood health^b</i>		
Periods of deprivations due to health ^c	173.46 (8)***	114.55 (2)***
Highly prevalent illnesses in Puerto Rico (1920s–1930s)		
Malaria	25.87 (4)***	11.48 (1)***
Tuberculosis ^d	4.78 (4)	4.63 (1)*
Typhoid	9.52 (4)*	4.42 (1)*
Other serious illnesses		
Asthma	40.04 (4)***	39.84 (1)***
Bronchitis	24.49 (4)***	17.77 (1)***
Dengue fever	23.73 (4)***	17.92 (1)***
Hepatitis	3.99 (4)	1.33 (1)
Pneumonia	25.33 (4)***	22.15 (1)***
Polio	1.04 (4)	0.23 (1)
Rheumatic fever	30.44 (4)***	21.78 (1)***
Smallpox	22.94 (4)***	18.37 (1)***
<i>Self-reported childhood SES</i>		
Father's education ^e	36.58 (4)***	30.84 (2)***
Father's occupation ^f	17.61 (6)***	14.73 (3)***

Source: Puerto Rican Elderly: Health Conditions (PREHCO, 2007), imputed, unweighted; all 60- to 74-year-olds born in Puerto Rico and who lived in the countryside as children. Published with permission from *Journal of Gerontology* (McEniry, Palloni, Dávila, & García, 2008)

Notes: Numbers in table are from chi-square analyses with degrees of freedom indicated in parentheses. SES socioeconomic status. * $p \leq .05$, *** $p \leq .001$

^a Scale is 1–5 for self-reported childhood health and 1–3 for self-reported childhood SES

^b Respondents were asked if they had ever experienced a list of childhood illnesses (yes/no). Only the more serious self-reported illnesses are reported here. However, there were no associations or marginal associations between self-reported childhood health and the more common childhood illnesses such as mumps, measles, and chickenpox. The source for identification of diseases with very high prevalence rates during the 1920s and 1930s was Clark (1930)

^c Frequent, some, none

^d The prevalence of tuberculosis was very high in urban areas of Puerto Rico; it was more prevalent among Puerto Rican adults than children (Clark, 1930, p. 62)

^e None, eighth grade, more than eighth grade

^f Respondents were asked the occupation of their father using an open-ended question, and responses were coded into occupational categories (i.e., agricultural, urban service, professional, other) according to standards used by the International Labor Organization (2010)

were obese (versus 20 % for those without heart disease), 28 % had diabetes (versus 11 % for those without heart disease), 28 % had difficulties with functionality (versus 17 %), and 44 % had ever smoked (compared to 41 % for those without heart disease). For those reporting diabetes, about 70 % also reported hypertension compared with 36 % for those without diabetes. A much larger percent with diabetes were obese (31 %), and had difficulties with functionality (24 %) than those without diabetes (18 and 18 %, respectively). For both heart disease and

Table 3.8 Prevalence of health conditions and adult factors known to be associated with heart disease and diabetes

	Heart disease		Diabetes	
	Yes	No	Yes	No
Hypertension (%) ^a	65	35	70	36
Stroke (%)	19	4	19	5
Obesity (%)	25	20	31	18
Diabetes (%)	28	11		
Functionality (%)	28	17	24	18
Mortality (%)	27	17	24	18
Ever smoked (%)	44	41	39	42
Exercises (%)	28	31	28	31

Source: RELATE (2013), for those born prior to 1945, data not weighted

Notes: All bivariate associations between health conditions and adult factors with either heart disease or diabetes were significant at $p \leq 0.000$. Although there were some exceptions (especially in the case of ever smoked), individual country analyses produced similar results

^a Example: for those reporting heart disease, 65 % also reported hypertension. Of those who did not report heart disease only 35 % reported hypertension

diabetes, the percent who exercised was slightly higher for those not reporting the condition (31 % versus 28 %). In Costa Rica, China, Indonesia, Bangladesh, and the US, mortality data were available from panel studies which showed that mortality was significantly higher for those who reported heart disease and diabetes (27 and 24 %) compared with those not reporting these conditions (24 and 18 %, respectively). For the most part, country-specific analyses within these countries produced similar results with some exceptions, suggesting the importance of further exploration.

An examination of prevalence across age and gender provides another piece of evidence that self-reported questions for heart disease and diabetes have validity even if there is underestimation. Table D.1 in Appendix D shows the prevalence of self-reported heart disease across countries by age and gender. For the most part, the findings confirm expectations: The prevalence of heart disease increases with age; high-income countries have the highest prevalence of heart disease, not surprising since better prevention and treatment of targeted risk factors for heart disease have increased the life span of those with heart disease; the earlier mortality regimes of Argentina and Uruguay are similar to high-income countries; surprisingly, low- and middle-income countries also appear to be similar, with the exception of Chile, Brazil, and Puerto Rico. Females in low income countries show a high prevalence of heart disease which may be due to SAGE respondents being slightly older than other adults from the same cohort. A declining prevalence of heart disease as individual's age is shown in some instances which may reflect problems in underestimation or selection.

The pattern for diabetes is different. Table D.2 in Appendix D reveals a strikingly higher prevalence of diabetes in the mid-mortality regimes. This is particularly true for females, and it is true even for countries whose economic growth and

health care systems improved after the 1940s (e.g., Taiwan and Costa Rica). A closer examination shows that in the very early mortality regimes the prevalence of diabetes generally increases with age—a finding expected in these settings given better access to treatment and better options for prevention and control of diabetes than in the developing world. In contrast, in the mid-mortality and some later regimes (e.g., Costa Rica, Puerto Rico, Taiwan, Mexico City residents, Barbados City residents, and South Africa) diabetes is seen at an earlier age which may indicate that individuals with diabetes in these countries do not survive to old age because of lack of proper medical treatment and/or lifestyle factors.

The validity of self-reported heart disease and diabetes can also be observed by examining their prevalence with obesity and functionality, more objective measures of health known to be associated with these conditions. While there are gaps in the data due to small cell sizes in some cases, Tables D.3 and D.4 in Appendix D show the generally positive association between obesity and heart disease and diabetes in many of the selected countries, especially among those reporting diabetes at younger ages. There is an overall downward trend in obesity as individual's age, especially among those reporting heart disease and/or diabetes. The variation in obesity for those who report heart disease and diabetes across countries may indicate underestimation of prevalence, but could reflect differences in the determinants of these conditions in particular settings.

Tables D.5 and D.6 in Appendix D use the harmonized measure of functionality and show, for the most part, there are more functional problems reported by those with heart disease and diabetes. There is a general expected trend of increasing difficulty with functionality as individual's age. A closer examination shows that, in some countries (Costa Rica, Brazil, the US, England, and Mexico), problems with functionality consistently increase with age, whereas in other countries there is a more sporadic pattern. In countries such as Indonesia, difficulties with functionality are fairly constant until late in life.³⁶ There is more variation in difficulty with functionality for those obese individuals reporting heart disease and diabetes which may also reflect problems in underestimation of these conditions.

Underestimation of adult heart disease and diabetes may produce differences in the strength of associations, but not in their direction. The well-validated symptom questions for angina in the SAGE surveys partially address concerns regarding underestimation of adult heart disease, especially in low-income countries. Pooled country analyses clearly show an increase in the prevalence of angina using only a self-reported question and when using the symptoms questions from the Rose questionnaire³⁷: India self-reported angina 6, 23 % with symptoms questions; Ghana 4 % versus 15 %; China 12 % versus 17 %; South Africa 6 % versus 9 %; Mexico 2 % versus 11 %; and Russian Federation 45 % versus 59 %. There are large differences in the odds ratios between models (Table 3.9, Panel A) for diabetes, where the odds of having heart disease are 91 % using self-reported heart disease but

³⁶ Sample sizes less than or equal to 20 in any age group were omitted from the graphs.

³⁷ Rose (1962).

Table 3.9 Comparison between self-reports, symptom, and biomarker data

Panel A: heart disease in SAGE	Self-reports	Self-reports and symptoms	
Age	1.02***	1.01**	
Female	1.36***	1.38***	
Education (years)	1.06***	1.01	
Obesity	1.21*	1.19*	
Functionality	1.32***	1.72***	
Diabetes	1.91***	1.46***	
Poor health	2.56***	2.22***	
Ever smoke	1.02	1.00	
Exercises	0.89	0.99	
China	3.93***	1.22*	
Ghana (reference)	1.00	1.00	
India	1.61***	1.69***	
Mexico	0.44***	0.64***	
Russian Federation	13.97***	6.57***	
South Africa	1.15	0.51***	
Log likelihood	-3886	-5,436	
Total observations	12,235	12,235	
Panel B: diabetes in Costa Rica	Self-reports	Glucose	Hemoglobin
Age	0.97***	0.98***	0.98***
Female	1.25	1.38**	1.25
Education (years)	0.99	1.01	1.00
Obesity	2.16***	2.18***	2.46***
Functionality	1.37**	1.30*	1.44***
Poor health	1.70***	1.37**	1.57***
Ever smoke	0.85	1.01	0.87
Exercises	0.70*	0.75*	0.73*
Log likelihood	-1,005	-1,206	-1,084
Total observations	2,197	2,197	2,197

Source: SAGE surveys, 2007–2008, for those born prior to 1945, unweighted

Note: Panel A shows the odds of reporting heart disease using self-reports and symptom questions and Panel B shows the odds of diabetes using self-reports and biomarker data. Prevalence of angina using self-reports versus self-reports with symptoms (weighted): China (14 %, 15 %), Ghana (5 %, 15 %), India (7 %, 23 %), Mexico (2 %, 10 %), Russian Federation (51 %, 57 %), South Africa (6 %, 10 %)

CRELES used two biomarkers to measure diabetes: glycosylated hemoglobin levels ($HbA_{1C} \geq 7\%$) and fasting serum glucose levels ($SG \geq 200$ mg/dL). See Brenes (2008) for more information

* $p \leq 0.05$, ** $p \leq 0.01$, *** $p \leq 0.001$

46 % when using a measure based on symptoms questions. China and Russia have very strong odds of reporting heart disease when using only the self-reported measure (OR 3.93 and OR 13.97, respectively), but although the odds remain strong, they are reduced in both cases when using the symptom questions (OR 1.22 and OR 6.57, respectively). These results indicate that although broad conclusions are similar

in terms of describing the direction of associations between variables and heart disease, in some instances the self-reports alone appear to sometimes produce an overly optimistic portrayal of the magnitude of associations and sometimes an overly pessimistic one. Country-specific analyses produce similar results, although country differences are apparent (results not shown) which suggests that caution must be taken in interpretations from cross-national comparisons.

A limited number of countries had biomarkers for diabetes by which to examine differences. Diabetes does not appear to be greatly underestimated in settings such as the UK, US, and Taiwan.³⁸ Even in middle-income countries such as Costa Rica, with a quality health care system, differences between self-reports and biomarkers may not be as great as with other poorer countries with less health care coverage.³⁹ Brenes⁴⁰ reported an increase in the prevalence of diabetes when using self-reports (20 %) and biomarkers (26 %). While this is a large increase (over 25 %), it is not clear how it translates into practical significance when reaching broad conclusions about associations using multivariate models. A comparison of logistic models across different measures of diabetes (self-reports, biomarker data using glucose, biomarker data using hemoglobin) in Table 3.9, Panel B suggests that, as with the case of heart disease in Panel A, the direction of the association is similar across models using different measures, but the magnitude may not be.

3.4 The Older Adult Population Across Mortality Regimes

3.4.1 Basic Characteristics

Table 3.10 shows basic characteristics of the sample obtained across all countries. For the most part, the table parallels results from other cross-national comparisons of older adults.⁴¹ The lower percentage of females in the Mexico-SAGE survey may reflect the lower response rates obtained in this survey. The average age is similar across all countries, although it is important to point out that this is the age at the time of the survey and thus not directly comparable across countries since the surveys were done during different time frames.

Not surprisingly, most older adults were married at one time; the percent of single, never married individuals is very low with the exception of Barbados (21 %) and South Africa (10 %). The average years of education shows the expected finding of better educational opportunities in countries with more resources. A comparison with the median household per capita income expressed in PPP international dollars also shows large differences between developed and developing

³⁸ Baker, Stabile, and Deri (2004), Banks et al. (2006a, 2006b), Beckett, Weinstein, Goldman, and Yu-Hsuan (2000), and Goldman, Lin, Weinstein, and Lin (2003).

³⁹ See Brenes (2008) but also Andrade (2008).

⁴⁰ Brenes (2008).

⁴¹ He, Muenchrath, and Kowal (2012).

Table 3.10 Basic demographic characteristics of the cross-national sample (pre-antibiotic cohorts, born prior to 1945)

Regime/country	Female (%)	Age	Still married (%)	Widowed/ Separated/ divorced (%)	Never married (%)	Children	Years education	Median house income(%) ^a	Household head (%)	House size (min, max)	Urban residence (%) ^b
<i>Very early</i>											
Netherlands	56	70 (8)	62	32	6	3	11 (4)	102	68	2 (1, 6)	
England	55	71 (9)	62	33	5	3	14 (3)	67		2 (1, 11)	
US-HRS	59	71 (8)	58	39	3		12 (3)	Reference		2 (1, 18)	
US-WLS	52	65 (1)	78	18	4	3	14 (2)	133		2 (1, 9)	
<i>Early</i>											
Argentina	62	71 (7)	55	39	5	2	7 (4)	12	63	3 (1, 11)	100
Cuba	59	71 (9)	40	57	3	3	7 (4)	4	72	4 (1, 22)	100
Uruguay	64	71 (7)	49	48	4	3	6 (4)	12	72	3 (1, 21)	100
<i>Mid-paced</i>											
Chile	60	70 (8)	57	26	7	4	6 (5)	6	64	4 (1, 11)	100
Costa Rica	52	70 (8)	60	32	8	6	5 (4)	10		3 (1, 21)	63
Puerto Rico	56	71 (8)	51	43	6	4	8 (5)	26	70	2 (1, 10)	
South Africa	61	70 (8)	51	40	10		6 (5)	7	79	4 (1, 16)	63
Taiwan	43	67 (8)	74	23	3		5 (5)			5 (1, 23)	45
<i>Late</i>											
Barbados	61	73 (8)	35	44	21	4	5 (3)	11	85	3 (1, 10)	100
Brazil	59	69 (7)	57	38	5	4	3 (3)	11	62	3 (1, 13)	100
Mexico-MHAS	53	66 (9)	64	32	4	6	4 (4)	6		4 (1, 19)	44
Mexico-SABE	56	70 (8)	56	40	4	6	5 (5)	4	63	4 (1, 18)	100
Mexico-SAGE	49	73 (7)	62	31	7		4 (4)	4	61	4 (1, 15)	63
Russian Fed	61	73 (7)	47	51	2		10 (4)	14	78	2 (1, 13)	64

(continued)

Table 3.10 (continued)

Regime/country	Female (%)	Age	Still married (%)	Widowed/ Separated/ divorced (%)	Never married (%)	Children	Years education	Median house income(%) ^a	Household head (%)	House size (min, max)	Urban residence (%) ^b
<i>Very late</i>											
Bangladesh	52	61 (8)	74	26	0	5	2 (3)	2	53	6 (1, 20)	0
China-CLHLS	53	72 (6)	57	41	1	5	3 (4)	6		3 (1, 32)	55
China-CHNS	53	66 (8)	74	22	5		4 (4)	9	53	4 (1, 11)	37
China-SAGE	53	71 (6)	75	24	1		6 (4)	15	63	3 (1, 11)	55
Ghana	51	72 (8)	52	47	1		4 (5)	4	83	6 (1, 16)	40
India	50	70 (7)	64	60	1		4 (5)	3	54	7 (1, 40)	30
Indonesia	54	65 (9)	65	34	1	5	4 (4)	1	58	6 (1, 23)	41

Source: RELATE (2013), weighted where relevant

Notes: All values are either percentages, where indicated, or averages with either standard deviations or minimum-maximum values in parentheses. Basic demographic information was mostly complete. Education and household income were the two major variables where missing values made up greater than 5 % of the data for those born prior to 1945. Missing values for education ranged from 6 % in Mexico-SAGE and Taiwan to 17 % in South Africa. Missing values for household income ranged from 6 % in China-SAGE to 33 % in the Russian Federation. Household head is the percent of the sample that were heads of households

^a Income is expressed in relation to the median per capita household income of the US (country median per capita income/US median household income per capita PPP 2000 international dollars). For example, the median household per capita income in the Netherlands is 102 % of the median household per capita income in the US using Health and Retirement Study, Public Use Dataset (2000–2006)

Annual per capita income was obtained in the following manner: in the case of household surveys, either the household income was calculated by survey staff (WLS, HRS, ELISA), target and spouse income was calculated for the user (Mexico-MHAS), questions were asked to calculate yearly household income (Bangladesh, China-Popkin) or wages were calculated (Indonesia). For surveys of older adults, there were various measures: one question for target and spouse (Costa Rica), calculated income based on several questions regarding income for target and spouse (Puerto Rico), or calculated income based on several questions regarding individual income for a randomly selected older adult in the household (SABE)

^b Rural/urban residence information was mostly obtained from surveys in the developing world

countries. Gini indices computed for the income of older adults using these survey data (results not shown) showed the lowest income inequality in high-income countries such as England and the Netherlands (Gini index of 35 and 37, respectively), and the highest inequality in a mix of low- and middle-income countries such as Bangladesh, Barbados, India, Mexico, and South Africa (Gini indices in the 1970s).

The percentage of the population currently living in an urban area partially reflects the nature of the survey—for example, SABE respondents came from major cities within Latin America and the Caribbean, and the Bangladesh survey was carried out in a rural area. Current living arrangements reflected in household size show, as expected, the increasing number of people in households as one moves from high- to low-income countries. Current urban residence also reflects the degree of urbanization experienced in some countries. Ethnicity, although not shown in the table, was obtained in some surveys.⁴² Education and income are two variables with a large degree of missing values (see notes in Table 3.10).

3.4.2 Early Life Conditions

Table 3.11 shows selected early life conditions. Not surprisingly, the percent born and raised in rural areas was high in low-income countries, but in some cases reflects the nature of the survey as noted above. From further analysis of the cross-national data, it is apparent that a large proportion of respondents were born in poor SES conditions, many of whom lived in rural areas (Fig. 3.3). Because height and knee height act as markers of early nutritional status and standard of living,⁴³ average height in Table 3.11 is thought to mirror early life economic circumstances. For example, taller individuals tend to live in higher-income countries. Knee height was measured in the Latin American and Caribbean (LAC) regions, although measurement protocols differed slightly between regions and thus the resulting measurements should be interpreted with caution. There were wide differences between countries in terms of season of birth, reflecting likely differences in exposure to early under-nutrition and/or infectious diseases. Season of birth does not appear in the table because cross-national comparisons are less meaningful. Information about parental educational attainment was available in some countries. While parental occupation was obtained in some cases, a comparison across countries is more difficult due to incongruences in the categories used. Respondents from LAC countries were more likely to have poor early SES conditions and poor early childhood health, although in some cases different scales were used; the measures for early childhood health and SES were harmonized as much as possible. A limited number of countries asked whether respondents had

⁴² For examples of ethnicity in different surveys see RELATE (2013).

⁴³ For height and knee height see Eveleth and Tanner (1990), Gunnell, Davey Smith, Holly, and Frankel (1998), and for season of birth see Doblhammer (2004).

Table 3.11 Selected variables on early life conditions (pre-antibiotic cohorts, born prior to 1945)

Regime/country	Born rural (%)	Male height-cm (sd)	Female height-cm (sd)	Male knee height-cm (sd)	Female knee height-cm (sd)	No education- father (%) ^a	No education- mother (%) ^a	Poor SES (%) ^b	Poor self-reported health (%) ^c	Hungry as a child (%) ^d
<i>Very early</i>										
Netherlands		176 (7)	165 (7)							
England		172 (7)	158 (7)							
US-HRS		177 (7)	153 (7)					32	6	
US-WLS	18	179 (7)	164 (7)						4	
<i>Early</i>										
Argentina	38									
Cuba	51	167 (7)	153 (7)	53 (5)	49 (6)			56	3	11
Uruguay	43	169 (7)	155 (7)	51 (5)	47 (5)			74	5	24
<i>Mid-paced</i>										
Chile	50	165 (7)	150 (6)	51 (3)	46 (2)			57	7	20
Costa Rica	72	164 (7)	150 (7)	52 (3)	47 (3)		31	57	9	
Puerto Rico	57	166 (7)	153 (7)	49 (5)	44 (4)	37	43	76	21	
South Africa	42	160 (13)	155 (12)			54	65	54		
Taiwan		163 (6)	152 (6)							
<i>Late</i>										
Barbados	50	170 (8)	158 (7)	54 (5)	51 (5)			82	2	18
Brazil	63	165 (7)	152 (7)	52 (3)	49 (3)			70	6	20
Mexico-MHAS	74	165 (9)	154 (8)	50 (5)	46 (4)	58	66	73	10	
Mexico-SABE	55	162 (7)	148 (7)	51 (3)	47 (3)			78	4	30
Mexico-SAGE	31	163 (8)	148 (7)			58	67			
Russian Fed	48	170 (8)	158 (6)			19	25			

<i>Very late</i>							
Bangladesh	99	159 (6)	146 (8)		74	94	
China-CLHLS	86			49 (5)	45 (5)		66
China-CHNS	63	164 (6)	152 (6)			92	
China-SAGE	54	163 (8)	152 (7)		74	96	
Ghana	61	164 (9)	156 (7)		88	91	
India	72	162 (8)	148 (7)		69	83	
Indonesia	84	158 (6)	147 (6)		69	83	

Source: RELATE (2013), weighted where relevant

Notes: Cells in the table without data are from countries where questions were not asked regarding that variable. Some countries had additional questions regarding childhood illness and circumstances. For example, China-CLHLS measured arm length which is a marker of early life nutrition. Parental occupation was not consistently asked across countries and is not presented in table. The reasons for missing values varied across countries. In general, there was a higher percent of missing values in older respondents and low income countries. In some countries, male respondents and respondents with less education had a higher degree of missing values

^a Father's and mother's education is the percent of respondents whose fathers/mothers had no education

^b Poor SES refers to the mean number of respondents who reported poor or fair SES in childhood

^c Poor self-reported health refers to the mean number of respondents who reported poor or fair health in childhood

^d Hungry as a child refers to the percent of respondents who indicated that they went hungry as children

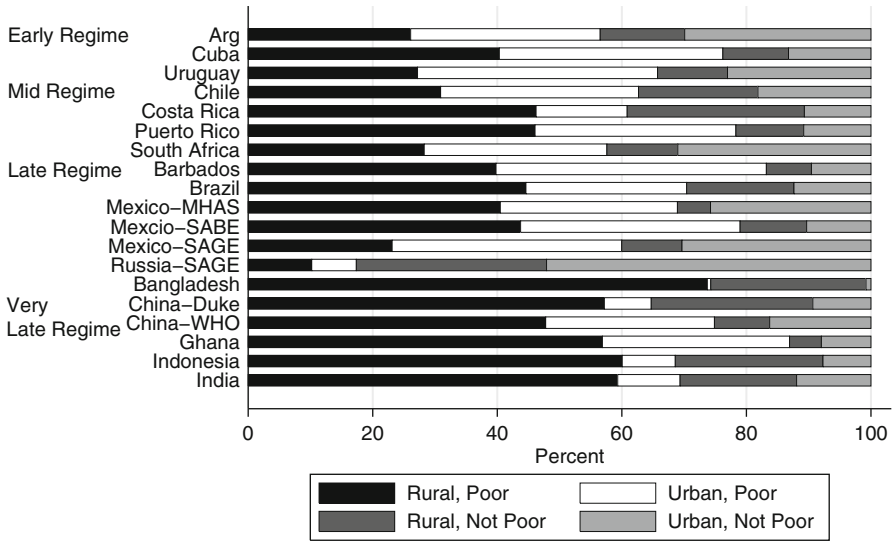


Fig. 3.3 Percent born poor by rural and urban area. *Note:* Calculations of born poor estimated by author using responses from surveys of older adults; calculation of born rural and urban was based on survey responses of older adults (*Source:* RELATE, 2013)

experienced being hungry during childhood. Surveys from Puerto Rico and China asked additional retrospective questions about childhood, and China measured arm length to reflect early life nutrition. These measures are not reported in the table for purposes of space, but do appear in a more detailed description of the cross-national data and recent reviews.⁴⁴ The degree of missing values for variables such as birthplace and height tended to be much more variable across countries than that of demographic variables (see notes in Table 3.11).

3.4.3 Adult Lifestyle, Standards of Living and Educational Attainment

Table 3.12 shows adult behavior and conditions which may impact health.

In most instances, the majority (over 50 %) of the respondents stated that they had never smoked; this may be partially due to the predominance of female respondents. The highest percentage of current smokers tended to be in low-income countries with the exception of Cuba and Taiwan. The wide variance in responses regarding exercise partially reflects the inconsistency in the way the question was asked across countries which ranged from very general questions to questions

⁴⁴ McEniry, Moen, and McDermott (2013), McEniry (2012).

Table 3.12 Adult behavior or risk factors (pre-antibiotic cohorts, born prior to 1945)

Regime/ country	Smoking			Adult weight ^a					Visits to a doctor (%)	
	Never smoked (%)	Used to smoke (%)	Current smoker (%)	Exercises (%)	Drinks (%)	Under-weight (%)	Normal (%)	Over-weight (%)		Obese (%)
<i>Very early</i>										
Netherlands	43	38	19	51	69	1	41	42	16	84
England	36	51	13	14	87	1	27	44	28	
US-HRS	60	28	12	43	46	2	38	39	21	95
US-WLS	42	45	13	14	74	1	25	42	32	92
<i>Early</i>										
Argentina	57	29	14	14	44					84
Cuba	47	22	32	23	23	12	44	30	14	74
Uruguay	57	28	16	15	41	3	30	32	35	76
<i>Mid-paced</i>										
Chile	53	34	13	21	40	1	26	42	31	73
Costa Rica	57	33	10	31	64	3	33	40	24	93
Puerto Rico	66	26	8	39	18	3	29	41	27	87
South Africa	69	10	21	12	23	4	22	27	46	68
Taiwan	76		24	62	24	3	56	34	7	34
<i>Late</i>										
Barbados	73	20	7	43	27	6	39	32	24	90
Brazil	52	32	16	27	32	3	36	39	22	83
Mexico-MHAS	57	27	16	30	92	3	36	40	21	64
Mexico-SABE	57	26	18	32	21	1	27	43	29	79
Mexico-SAGE	58	23	19	11	50	1	32	41	25	40
Russian Fed	74	13	13	27	73	1	29	38	32	72

(continued)

Table 3.12 (continued)

Regime/ country	Smoking		Adult weight ^a					Visits to a doctor (%)		
	Never smoked (%)	Used to smoke (%)	Current smoker (%)	Exercises (%)	Drinks (%)	Under-weight (%)	Normal (%)		Over-weight (%)	Obese (%)
<i>Very late</i>										
Bangladesh	69	10	21			64	33	2	1	66
China-CLHLS	61	13	26	41	33					91
China-CHNS	69	2	29	32	32	9	64	22	4	
China-SAGE	67	9	24	15	31	6	60	28	6	60
Ghana	74	15	11	37	55	20	56	17	8	68
India	43	6	51	24	16	45	44	8	2	89
Indonesia	50	8	42			31	55	12	2	10

Source: RELATE (2013), weighted where relevant

Notes: The reasons for missing values varied across countries. In general, there was a higher percent of missing values in older respondents and low income countries. In some countries, male respondents and respondents with less education had a higher degree of missing values

regarding rigorous and moderate exercise to very detailed questions regarding time spent on different types of exercise per week (see notes in Table 3.12). As noted in Chap. 2, all countries had questions regarding drinking with the exception of Bangladesh and Indonesia. However, there is little comparability across countries for consumed alcohol beverages because of differences in the wording of questions. In terms of obesity, it is clear that the highest prevalence of obesity is among older adults in the LAC region and the highest prevalence of underweight appears in low-income countries. A high percentage of respondents stated they had visited a doctor within the last year, with the exception of those in Taiwan, Mexico-SAGE, and Indonesia. The prevalence of missing values varied across countries (see notes in Table 3.12).

Not surprisingly, there are large differences across countries in the degree to which older adults have the same educational level as their fathers (Table 3.13). In Puerto Rico and the Russian Federation, respondents were likely to have more education than their fathers: 91 % percent of older adults in Puerto Rico and 94 % of respondents in the Russian Federation whose father had no education had at least some education, and 71 % of respondents in Puerto Rico and 78 % in the Russian Federation had achieved higher than a primary level of schooling (six years of education). On the other hand, 70–74 % of respondents in Bangladesh, India, and Ghana whose father had no education also had no education, and between 23–49 % of older adults in these low-income countries whose fathers had primary school education were educated beyond the primary level. Downward changes to a lower educational attainment appear to be most likely in Mexico, Bangladesh, India, China, and Ghana.

3.4.4 Prevalence of Heart Disease and Diabetes

Table 3.14 shows age-adjusted prevalence for adult heart disease and diabetes for those born prior to 1945 as measured by self-reported questions across countries. The highest prevalence of heart disease is in the Russian Federation at slightly over 50 % followed by Chile, with over 30 % of the sample reporting heart disease. The lowest reported prevalence of heart disease is in China-CHNS and Indonesia. For diabetes, Puerto Rico, Costa Rica, Mexico-SABE, and Barbados have the highest prevalence, with the lowest prevalence in China, India, Ghana, and Indonesia. The composite measure of health based on functionality, BMI, and poor self-reported health suggests a U-shaped distribution of health, with the US, England, and the Netherlands along with some lower-income countries (early mortality regimes) being in a better position than the mid- and later mortality regimes. There were more missing values for adult heart disease and diabetes in low-income countries (see notes in Table 3.14).

Table 3.13 Degree to which respondent's educational attainment is higher than father's educational attainment

Regime/country	Father's education			
	No schooling	Primary	Secondary	Higher
<i>Very early</i>				
US-HRS	100 % better	95 % better	48 % better	79 % same
	0 % same	5 % same	50 % same 1 % worse	21 % worse
<i>Mid</i>				
Puerto Rico	91 % better	71 % better	88 % same	
	9 % same	27 % same 2 % worse	12 % worse	
South Africa	48 % better	60 % better	86 % same	
	52 % same	27 % same 13 % worse	14 % worse	
<i>Late</i>				
Mexico-MHAS	56 % better	27 % better	72 % same	
	44 % same	61 % same 13 % worse	28 % worse	
Mexico-SAGE	67 % better	26 % better	65 % same	
	33 % same	62 % same 12 % worse	35 % worse	
Russian Federation	94 % better	78 % better	95 % same	
	6 % same	20 % same 2 % worse	5 % worse	
<i>Very late</i>				
Indonesia	45 % better	30 % better	77 % same	
	55 % same	57 % same 13 % worse	23 % worse	
Bangladesh	26 % better	23 % better	29 % same	
	74 % same	37 % same 40 % worse	71 % worse	
India	30 % better	40 % better	69 % same	
	70 % same	33 % same 27 % worse	31 % worse	
China	55 % better	47 % better	74 % same	
	45 % same	36 % same 16 % worse	26 % worse	
Ghana	27 % better	49 % better	63 % same	
	73 % same	13 % same 38 % worse	37 % worse	

Source: RELATE (2013), unweighted

Notes: Shown in the table is the educational attainment of older adults in the cross-national data comparing father's education with the respondent's education. For each category of father's education, the percent of respondents is shown whose educational attainment was the same, better or worse than their father's education. For developing countries, secondary and above level of education was combined into the "secondary" category that appears in the table. For example, in Puerto Rico 91 % of older adults whose fathers had no schooling achieved a higher level of education than their fathers

Table 3.14 Age-standardized prevalence of adult heart disease and diabetes (pre-antibiotic cohorts, born prior to 1945)

Regime/country	Heart disease	Heart disease (%)	Diabetes (%)	Composite health
<i>Very early</i>				
England	CHD+	27	10	69
Netherlands	CHD+	15	11	75
US-HRS	CHD+	24	15	74
US-WLS	CHD+	8	16	
<i>Early</i>				
Argentina	CHD+	20	12	
Cuba	CHD+	24	15	64
Uruguay	CHD+	23	13	67
<i>Mid</i>				
Chile	CHD+	32	13	57
Costa Rica	HD, MI*	12	21	66
Puerto Rico	CHD+, MI*	17	28	59
South Africa	ANGINA	9	11	67
Taiwan	HD	17	17	80
<i>Late</i>				
Barbados	CHD+	10	22	68
Brazil	CHD+	20	18	64
Mexico-MHAS	MI	3	17	61
Mexico-SABE	CHD+	10	22	57
Mexico-SAGE	ANGINA	11	18	74
Russian Federation	ANGINA	53	11	69
<i>Very late</i>				
Bangladesh	N.A.	N.A.	20	60
China-CHNS	MI	1	4	65
China-CLHLS	HD	13	9	62
China-SAGE	ANGINA	15	8	82
Ghana	ANGINA	14	5	77
India	ANGINA	23	7	70
Indonesia	CHD+	3	2	75

Source: RELATE (2013), weighted where relevant

Key: CHD+ coronary heart disease plus other heart problems, MI myocardial infarction, HD general heart disease question, ANGINA angina or angina pectoris

Notes: Prevalence was weighted in each country using sample weights where relevant and then age-standardized using the WHO standard (see Ahmad et al., 2001). Composite health is a continuous variable comprised of BMI, functionality, and poor self-reported health where 1 = worst and 100 = best. The self-reported heart disease questions in almost all countries refer specifically to the most common forms of heart disease, coronary heart disease (CHD) or coronary artery disease (angina, heart attack, or myocardial infarction), but also includes other heart problems. In some cases, individuals were only asked if they had heart disease (HD). SAGE only asked about angina but symptom questions using the Rose questionnaire (Rose, 1962) were combined with self-reports to obtain the prevalence of heart disease

3.5 Conclusions

Survey data on older adults and historical data from the twentieth century have limitations in terms of their preciseness by which to gauge health and its determinants. Nevertheless, the survey data on early life conditions such as urban/rural residence in early life, season of birth, and childhood health show some validity, and underestimation of older adult heart disease and diabetes may not necessarily lead to erroneous conclusions about the direction of associations. Historical data used in the classification of mortality regimes are imprecise but appear to be reasonable in the resulting grouping of regimes. The use of historical caloric intake while also imprecise is a reasonable alternative to retrospective measures of early life obtained from surveys of older adults. Innovative techniques such as Waaler-type surfaces provide a way to examine mortality risk in cases where actual mortality data are not yet available.

There are exceptions that may influence differences in patterns of health such as the high percentage of men in the Taiwan study or the high percentage of never married older adults in Barbados, and these exceptions require more scrutiny. However, the overall picture obtained of the older adult population across a diverse range of countries using surveys of older adults presents few surprises in most cases:

- An older population, the majority of whom are female, were married at some point in time and have children;
- The pattern in poorer countries was of more children, less educational attainment (or changes in educational attainment), less income, larger household size, higher percentage born in rural areas, shorter height, higher percent underweight, less parental education, and a large percentage saying they experienced poor SES and health and were hungry as children.
- There is an increasing prevalence of chronic conditions and obesity especially in the Latin American and Caribbean countries in contrast to some countries of Asia where being underweight is more of a problem than obesity.

The pieces of evidence implying to some degree the validity of survey and historical data give rise to the expectation that the analyses in the forthcoming chapters will provide at least a modest clue as to the merit of the contrarian conjecture. An examination of cross-national data from so many diverse and important studies on aging in low-, middle- and high-income countries has the value of providing an overall picture, confirming the expected, and uncovering unexpected patterns.

The profile of older adult health in the tip of the iceberg countries will be found to be worse than what is observed in developed countries, even after removing the potential effects of current disparities in standards of living.

(Hypothesis #1)

Current health conditions, but particularly the prevalence of certain chronic conditions such as heart disease and diabetes in the tip of the iceberg countries, will be closely associated with individual history, including nutritional status, early experience with illnesses, and deprivations experienced during early childhood.

(Hypothesis #2)

Adapted from Palloni, McEniry, Wong, and Peláez (2006)

One piece of evidence for the merit of the conjecture regarding the unique cohorts of the 1930s–1960s is the comparison of morbidity patterns across countries and mortality regimes. There are two immediate questions based on the hypotheses: (1) Is the profile of older adults in low- and middle-income countries (and in particular the tip of the iceberg countries) worse than what is observed in developed countries, even after removing the potential effects of current disparities in standards of living?; and (2) Are current chronic health conditions closely associated with past individual history, including nutritional status, early experiences, illnesses, and deprivations experienced during early childhood? In answering these two questions another question arises: (3) What is the appropriate time period to make comparisons across countries which may be at very different points in their health transition?

A comparison across countries at the time of the surveys shows the current prevalence of chronic conditions. To observe if there are differences in historical patterns between the developed and developing world, a comparison of morbidity patterns based on the timing and pace of mortality decline during the twentieth century becomes relevant. The question then becomes: Is the prevalence of chronic

conditions such as heart disease and diabetes in later mortality regimes higher today than what was ever observed in the U.S., the Netherlands, and England at an earlier, similar point in time in their demographic transition? A higher prevalence of chronic conditions at a comparable moment in time might indicate that the conjecture regarding the impacts of a larger pool of survivors of poor early life conditions has merit. It may also indicate that present conditions (e.g., adult lifestyle, SES, better treatment, and prevention efforts) play a role in the higher prevalence of chronic conditions in low- and middle-income countries.

In the first part of this chapter, cross national morbidity patterns are presented. The later part of the chapter then presents the specific case of Puerto Rico. The chapter ends with a summary of findings.

4.1 Pooled Models for the Cohort of the Late 1920s and Early 1940s

Pooled models for adults born in the late 1920s through early 1940s (Table 4.1, Model 1) show that, not surprisingly, compared with the relatively healthier population of the US-WLS, the chance of reporting heart disease is higher in the US-HRS sample, England, Cuba, Uruguay, Chile, the Russian Federation, and India. This is particularly the case for Chile and the Russian Federation, where the likelihood of reporting heart disease is about 2.5 and 6 times that of the US-WLS, respectively. When the developed countries are omitted (US-WLS, US-HRS, England, and the Netherlands) and Uruguay is used as a reference (Model 2), a similar pattern emerges where the likelihood of heart disease is higher in a relatively better off developing country of Uruguay than it is in other developing countries although the strength of the associations is smaller. When the likelihood of heart disease in SAGE countries (where the identification of heart disease is based on a more robust approach using symptom questions) is contrasted with that of a high income country of the US-WLS (Model 3), a similar pattern emerges; in most cases there is a greater likelihood of heart disease in the US than in SAGE countries with the exception of Russia and India. The odds of heart disease are smaller in South Africa (OR 0.47), Mexico (OR 0.49), China (OR 0.86), and Ghana (OR 0.82) than they are in the US-WLS. In contrast, the odds of heart disease in Model 3 are much higher in the Russian Federation and India compared with US-WLS (OR 6.09 and OR 1.37, respectively). When compared against Uruguay (Model 4), the effects are weaker but nevertheless in the same direction for these two countries (OR 4.05 and OR 1.05, respectively). A model using only the self-reports for heart disease in SAGE countries (Model 5) shows differences in magnitude, particularly for Russia and India, when compared with models using symptoms to obtain the prevalence of heart disease (Models 3–4). In most cases, while there are differences in magnitude the direction of the associations are similar across models; the exception is India which in Model 3 shows a positive association with heart disease but in Model 5 the likelihood of reporting heart disease is lower.

Table 4.1 Likelihood of reporting heart disease for those born in the late 1920s–early 1940s

Regime/country	Model 1 ^b	Model 2 ^{a,b}	Model 3	Model 4	Model 5
Very early					
Netherlands	0.76**				
England	1.63***				
US-HRS	1.48***				
US-WLS	1.00 (ref)		1.00 (ref)		
Early					
Argentina	1.22				
Cuba	1.46***	1.02			
Uruguay	1.40***	1.00 (ref)		1.00 (ref)	1.00 (ref)
Mid-paced					
Chile	2.50***	1.63***			
Costa Rica	0.59***	0.43***			
Puerto Rico	1.03	0.71***			
South Africa	0.48***	0.36***	0.47***	0.32***	0.21***
Taiwan	0.87				
Late					
Barbados	0.52***	0.37***			
Brazil	1.14	0.75*			
Mexico-MHAS	0.21***	0.13***			
Mexico-SABE	0.51***	0.34***			
Mexico-SAGE	0.48***	0.37***	0.49***	0.35***	0.08***
Russia-SAGE	5.94***		6.09***	4.05***	2.54***
Very late					
China-CHNS	0.08***				
China-CLHLS	1.12				
China-SAGE	0.84**	0.68***	0.86	0.62***	0.59***
Ghana	0.82*	0.60***	0.82*	0.60***	0.17***
India	1.33***	1.03	1.37***	1.05	0.29***
Indonesia	0.22***	0.15***			
Log likelihood	−24,249	−8,413	−7,713	−5,605	−4,190
Total observations	59,946	21,672	17,144	12,012	12,012

Source: RELATE (2013)

Notes: Odds ratios are shown. All models controlled for age, gender, education, and smoking. The dependent variable for Models 1–4 is self-reported heart disease with the exception of the SAGE countries where Rose symptom questions for angina were used (Rose, 1962). The dependent variable for Model 5 for SAGE countries is self-reported heart disease

* $p \leq 0.05$; ** $p \leq 0.01$; *** $p \leq 0.001$

^aModel 2 only includes countries where information on obesity was available

^bStatistical tests showed that models with country dummies shown in the table significantly improved model fit

Pooled models for diabetes in adults born in the late 1920s through early 1940s (Table 4.2, Model 1) show that the chance of reporting diabetes is higher for older adults in Costa Rica, Puerto Rico, Mexico, Barbados, and Brazil than it is for older adults in the healthier US-WLS population, in some cases twice as high. In contrast,

Table 4.2 Likelihood of reporting diabetes for those born in the late 1920s–early 1940s

Regime/country	Model 1	Model 2
Very early		
Netherlands	0.74**	
England	0.80***	
US-HRS	1.40***	
US-WLS	1.00 (ref)	
Early		
Argentina	1.09	
Cuba	1.21*	1.11
Uruguay	1.10	1.00 (ref)
Mid-paced		
Chile	1.21	1.21
Costa Rica	1.99***	2.05***
Puerto Rico	2.88***	2.61***
South Africa	0.94	1.05
Taiwan	1.10	
Late		
Barbados	1.97***	1.85***
Brazil	1.62***	1.57***
Mexico-MHAS	1.59***	1.53***
Mexico-SABE	2.01***	2.06***
Mexico-SAGE	1.89***	2.03***
Very late		
Bangladesh	1.20*	1.24
China-CHNS	0.27***	
China-CLHLS	1.15*	
China-SAGE	0.66***	0.75*
Ghana	0.31***	0.34***
India	0.61***	0.70**
Indonesia	0.31***	0.30***
Log likelihood	−24,569	−9,961
Total observations	63,257	25,665

Source: RELATE (2013)

Notes: All models controlled for age, gender, education, and smoking. Taiwan data did not identify rural/urban birthplace and was excluded from Model 2. China-CLHLS and Argentina did not measure height and, because obesity could not be calculated for later models with caloric intake, were also excluded from Model 2. Statistical tests showed that models with country dummies significantly improved model fit

* $p \leq 0.05$; ** $p \leq 0.01$; *** $p \leq 0.001$

the odds of reporting adult diabetes are reduced for older adults from countries such as Ghana, India, and Indonesia. Excluding the high income countries (US, England, and the Netherlands) and using Uruguay as a comparison (Model 2), a similar pattern emerges.

Table 4.3 Likelihood of being obese for those born in the late 1920s–early 1940s

Regime/country	Model 1	Model 2
Very early		
England	1.46***	
Netherlands	0.70***	
US-HRS	1.16***	
US-WLS	1.00 (ref)	
Early		
Cuba	0.76**	1.98***
Uruguay	1.94***	1.00 (ref)
Mid-paced		
Chile	2.02***	5.05***
Costa Rica	1.36***	3.37***
Puerto Rico	1.60***	4.08***
South Africa	2.70***	6.70***
Taiwan	0.32***	
Late		
Barbados	1.65***	3.99***
Brazil	1.35***	3.31***
Mexico-MHAS	1.30***	3.32***
Mexico-SABE	2.00***	5.11***
Mexico-SAGE	1.89***	4.98***
Very late		
China-CHNS	0.17***	
China-SAGE	0.25***	0.58***
Ghana	0.37***	0.91
India	0.13***	0.34***
Indonesia	0.12***	0.29***
Log likelihood	−25,340	−9,816
Total observations	55,952	27,105

Source: RELATE (2013)

Notes: Odds ratios are shown. All models controlled for age, gender, education and smoking. Statistical tests showed that models with country dummies significantly improved model fit

* $p \leq 0.05$; ** $p \leq 0.01$; *** $p \leq 0.001$

There is a very strong likelihood of being obese in the early to late demographic regimes in comparison with the very early regime of the US-WLS (Table 4.3); with the exception of Taiwan and Cuba, the odds were between 1.3 and almost 3 times the odds of being obese in the US-WLS (Table 4.3, Model 1). These odds were, for the most part, higher than what was observed for England and the US-HRS. Omitting the developed world and using Uruguay as a point of reference produces odds of anywhere between 2 and almost 7 times that of Uruguay, with the middle and late regimes showing much stronger positive associations (Table 4.3, Model 2).

Being born in the very late regimes greatly reduced the odds of being obese by anywhere between 63 % and almost 90 % when compared with the US-WLS (Model 1) and between about 40 and 70 % when compared with Uruguay.

4.2 Population-Level and Individual-Level Analyses

In terms of caloric intake in early life, a graphical representation selecting only those born during the late 1920s through early 1940s depicts an increasing positive association between country-level caloric intake in early life and the prevalence of adult heart disease for those born during the late 1920s through the early 1940s (Fig. 4.1).

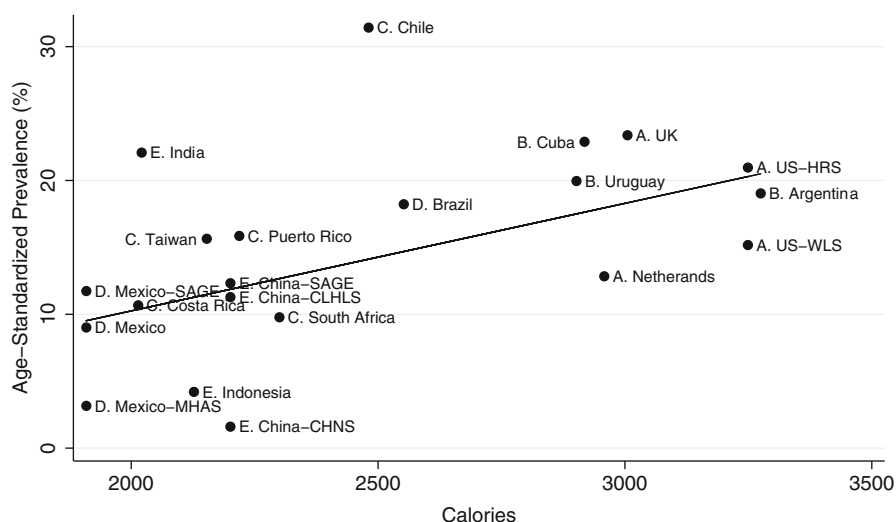


Fig. 4.1 Caloric intake in early life and older adult heart disease. *Notes:* The best-fit equation is heart disease = $[(8.039 * 10 - 3) * \text{calories}] - 5.824 +$ with an R-squared = 0.37. Russia and Chile were omitted from the best-fit regression because they appear as outliers. Most countries used a self-report question to obtain information about coronary heart disease which includes angina pectoris and heart attack. Some countries only used a question about heart attack (Mexico-MHAS, China-CHNS) or a general question about heart disease (Taiwan-SEBAS). SAGE used the Rose questionnaire for angina pectoris. SAGE adults born in the late 1920s–early 1940s are older at the time when they took the survey (2007–2008) than those from other surveys (2000–2005). Data for heart disease in Indonesia were obtained in 2007. The SABE survey represents older adults in major cities of Latin America and the Caribbean. The Russian Federation was excluded from this graph as the prevalence of heart disease was clearly an outlier (0.51). The age-standardized prevalence for Ghana and Barbados was 14 and 9 % respectively. FAO (1946) did not identify caloric supply for these countries although historical evidence describing environmental and nutritional conditions during the 1930s indicate that caloric supply must have been very low (see Caldwell, 1967; Patterson, 1979, 1981 for Ghana; see Bishop, Corbin, & Duncan, 1997 for Barbados). FAO (1946) identifies surrounding countries in French West Africa as having a caloric intake of 2,311. Mortality regimes along with countries are depicted in the graph as: A Very early, B Early, C Mid, D Late, E Very late (*Sources:* Age-standardized prevalence based on RELATE (2013) for those born during the late 1920s through early 1940s using WHO standards (Ahmad et al., 2001). Daily caloric intake per capita based on FAO (1946))

A linear regression shows an R -squared of about 0.37. The prevalence of heart disease for older adults is lower in countries with a reduced daily per capita caloric intake prior to World War II such as Mexico, China, South Africa, Costa Rica, Taiwan, and Puerto Rico. The prevalence of heart disease is greater in the developed countries (US, Netherlands, and England) and the developing countries of Uruguay, Argentina, and Cuba; Chile and the Russian Federation appear to be outliers. The prevalence of heart disease in the Russian Federation was very high (51 %) possibly reflecting special circumstances in the determinants of heart disease.

Table 4.4 shows the likelihood of reporting adult heart disease using individual-level data controlling for potential confounding factors. The odds of reporting heart disease were higher among those countries with higher caloric intake in the 1930s (Table 4.4, Model 1). In general, being born in low- and mid-caloric supply countries (India, China, Ghana, Costa Rica, Puerto Rico, and Chile) greatly reduced the odds of reporting heart disease later. Being born in a rural area (proxy for low parental SES) and being born in a low-caloric country reduced the odds of heart disease by a little over 30 % in some cases (Models 2–5). However, being born in a rural area in a mid-caloric country increased the odds of heart disease by 40 %. The results remained consistent after adding adult education and currently living in a high caloric intake country (Model 2), smoking and obesity (Model 3), other health problems (Model 4), and health systems and visits to a doctor (Model 5).

Females were more likely to report heart disease than males (17–31 % higher); living in a high calorie country reduced the odds of reporting heart disease by 55 %, but the combination of living in a high calorie country and having more education increased the odds of heart disease by about 10 % (Model 2). Being obese increased the odds of heart disease by 30–38 % (Models 3–5), and previously smoking increased the odds of heart disease by 33–46 % (Model 3). Having difficulty with at least one functional task and reporting poor health greatly increased the odds of adult heart disease (Models 4 and 5). Having visited a doctor or medical professional at least once within the last year increased the odds of adult heart disease by over 70 % (Model 5).

In terms of diabetes, at first glance a graphical depiction of pre-World War II caloric intake and the prevalence of adult diabetes among those born during the late 1920s through early 1940s suggest a weak linear association (Fig. 4.2). However, when the very late mortality regimes of China, India, and Indonesia (marked by the large arrow) are excluded, a clear negative association appears between the prevalence of diabetes in older adults and caloric intake in early life. Puerto Rico has the highest prevalence of diabetes (about 28 %), with other countries close behind. Older adults in SABE cities in Brazil, Mexico, and Barbados born during late mortality regimes also have a higher prevalence of diabetes. The very late mortality regimes of China, Indonesia, and India have a prevalence of diabetes lower than 10 % similar to that among older adults in the 1970s in the US.

These graphical associations are confirmed with multivariate analyses. Being born in a mid-caloric supply country during the 1930s increases the odds of reporting diabetes by 68 % compared with being born in a higher caloric country, but no significant interaction effects with rural birthplace (Table 4.5, Model 1). In contrast, being born in a country with a low caloric supply during the 1930s and in a rural area (proxy for low

Table 4.4 Caloric intake and the likelihood of reporting heart disease for those born in the late 1920s–early 1940s

Variables	Model 1	Model 2	Model 3	Model 4	Model 5
Demographic					
Female	1.26***	1.27***	1.31***	1.20***	1.17***
Age	1.05***	1.05***	1.05***	1.05***	1.05***
Years education		0.96***	0.95***	0.96***	0.96***
Childhood					
Rural birthplace ^a	1.02	1.07	1.09	1.05	1.07
Low caloric intake ^b	0.70***	0.61***	0.69***	0.91	1.04
Mid caloric intake ^b	0.40***	0.41***	0.40***	0.40***	0.41***
High caloric intake (ref) ^b	1.00	1.00	1.00	1.00	1.00
Low X Rural birthplace	0.75*	0.69**	0.70**	0.70**	0.70**
Mid X Rural birthplace	1.40**	1.41**	1.40**	1.34*	1.28
Adult lifestyle					
High calorie country ^c		0.45***	0.45***	0.49***	0.59***
High calorie X Education		1.07***	1.08***	1.08***	1.08***
Used to smoke			1.46***	1.35***	1.33***
Smokes now			0.92	0.89*	0.91
Never smoked (ref)			1.00	1.00	1.00
Obese ^d			1.38***	1.32***	1.30***
Adult health					
Difficulty with functionality ^e				1.72***	1.70***
Poor reported health				2.38***	2.25***
Systems					
Visited a doctor within the last year					1.72***
Log likelihood	−8,760	−8,693	−8,643	−8,342	−8,284
BIC ^f	17,599	17,495	17,426	16,843	16,738
Total observations	21,672	21,672	21,672	21,672	21,672

Source: RELATE (2013) excluding high-income countries – US, Netherlands, and England. Taiwan is not included because its survey did not have information on birthplace. Russia is excluded because its historical circumstances make it a more complex case to categorize (Anderson & Silver, 1986; Prokhorov (2005–2009))

Notes: Odds ratios are shown

* $p \leq 0.05$; ** $p \leq 0.01$; *** $p \leq 0.001$

^aBorn and lived in rural area during childhood (1 = yes, 0 = no)

^bCountry-specific daily caloric intake per capita in the 1930s were defined as follows:

Low = less than 2,100 (China, Ghana, India, and Indonesia)

Mid = greater than 2,100 and less than 2,800 (Barbados, Brazil, Chile, Costa Rica, Mexico-MHAS, Mexico-SABE, Puerto Rico, South Africa)

High = greater than 2,800 (Cuba, Uruguay)

^cLiving currently in a high-calorie country was defined as higher than 2,700 daily caloric intake per capita (1 = yes, 0 = no). Bangladesh, India, and Ghana were classified as lower caloric intake countries using data from 2000. Most higher caloric intake countries were in the mid caloric category in the 1930s

^dObese (1 = yes, 0 = no) based on BMI greater than or equal to 30

^eDifficulty with functionality was a harmonized measure using ADLs. Poor self-reported health was also based on a harmonized version of the self-report question asked about one's general health

^fBIC is a criterion for model selection. Models with smaller BIC indicate better fit

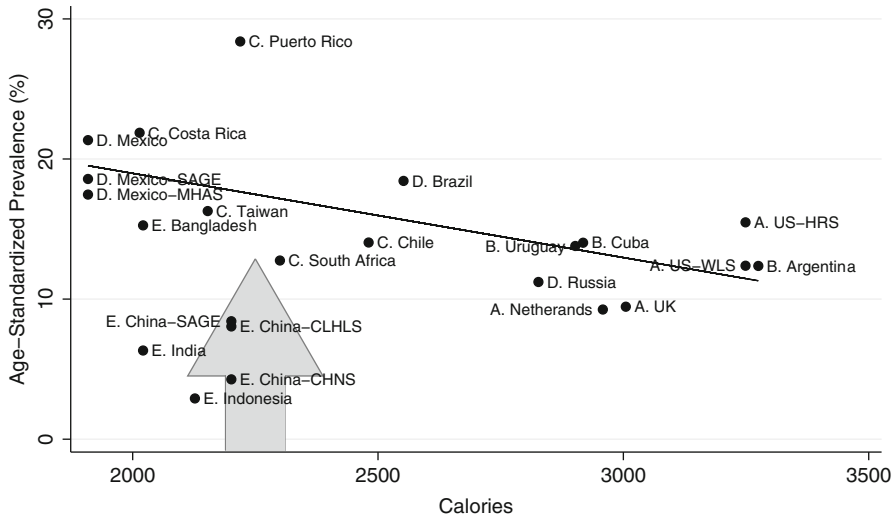


Fig. 4.2 Caloric intake in early life and older adult diabetes. *Notes:* The best-fit equation is $\text{diabetes} = [(6.011 * 10 - 3) * \text{calories}] + 3.100 * 101 +$ with an R-squared = 0.40. China, India and Indonesia were omitted from the best-fit regression line because they appear as outliers. The arrow in the figure highlights low income countries. The prevalence of diabetes was expected to be low in countries which had not yet experienced a demographic change to decreasing mortality in the late 1920s through early 1940s. The arrow suggests that the prevalence may increase when it becomes possible to observe older adults of the unique cohorts born in the 1940s–1960s as their countries experienced significant demographic transitions to lower mortality. SAGE older adults born in the late 1920s–early 1940s are older at the time of the survey than older adults from other surveys. Data for heart disease in Indonesia were obtained in 2007. The SABE survey represents older adults in major cities of Latin America and the Caribbean. The Russian Federation was excluded from this graph as the prevalence of heart disease was clearly an outlier (0.51). The age-standardized prevalence for Ghana and Barbados was 5 and 22 % respectively. FAO (1946) did not identify caloric supply for these countries although historical evidence describing environmental and nutritional conditions during the 1930s indicate that caloric supply must have been very low (see Caldwell, 1967; Patterson, 1979, 1981 for Ghana; see Bishop et al., 1997 for Barbados). FAO (1946) identifies surrounding countries in French West Africa as having a caloric intake of 2,311. Mortality regimes along with countries are depicted in the graph as: A Very early, B Early, C Mid, D Late, E Very late (Sources: Age-standardized prevalence based on RELATE (2013) for those born during the late 1920s and early 1940s using WHO standards (Ahmad et al., 2001). Daily caloric intake per capita based on FAO (1946))

parental SES) reduces the odds of reporting diabetes by about 45 %. The effects for the mid-caloric supply countries remain consistent even after adding potential confounding factors such as currently living in a country with higher caloric supply and education (Model 2), adult smoking and obesity (Model 3), other adult health problems (Model 4), and visits to a medical professional in the last 12 months (Model 5).

As was the case for heart disease, females have a higher chance of reporting diabetes than males, although this effect becomes weaker with the addition of adult health and health system variables. Living in a higher caloric country and having higher education reduces the odds of diabetes slightly (Model 2), being obese

Table 4.5 Caloric intake and the likelihood of reporting diabetes for those born in the late 1920s–early 1940s

Variables	Model 1	Model 2	Model 3	Model 4	Model 5
Demographic					
Female	1.25***	1.31***	1.16***	1.09*	1.05
Age	1.00	1.01	1.00	1.01*	1.00
Years education		1.06***	1.05***	1.06***	1.05***
Childhood					
Rural birthplace ^a	0.96	0.99	1.00	0.95	0.97
Low caloric intake ^b	0.93	0.82	0.83	1.02	1.20
Mid caloric intake ^b	1.68***	1.72***	1.61***	1.66***	1.69***
High (reference) ^b	1.00	1.00	1.00	1.00	1.00
Low X rural birthplace	0.55***	0.56***	0.59***	0.60***	0.60***
Mid X rural birthplace	1.07	1.07	1.06	1.03	0.98
Adult lifestyle					
Good calorie country ^c		0.83*	0.84*	0.85*	1.06
Good calorie X education		0.96***	0.96***	0.96***	0.96***
Used to smoke			0.99	0.92	0.90*
Smokes now			0.67***	0.66***	0.67***
Never smoked (ref)			1.00	1.00	1.00
Obese ^d			1.30***	1.27***	1.25***
Adult health					
Difficulty with functionality ^e				1.11*	1.10
Poor reported health				2.18***	2.02***
Systems					
Visited a doctor within the last year					2.01***
Log likelihood	−10,119	−10,088	−10,040	−9,825	−9,715
BIC ^f	20,320	20,287	20,223	19,812	19,603
Total observations	25,665	25,665	25,665	25,665	25,665

Source: RELATE (2013), excluding high income countries – US, Netherlands, and England. Taiwan is not included because its survey did not have information on birthplace. Russia is excluded because its historical circumstances make it a more complex case to categorize (Anderson & Silver, 1986).

Notes: Odds ratios are shown. The dependent variable is self-reported heart disease with the exception of SAGE countries where the Rose symptom questions for angina were used to complement self-reported heart disease (Rose, 1962).

* $p \leq 0.05$; ** $p \leq 0.01$; *** $p \leq 0.001$

^aBorn and lived in rural area during childhood (1 = yes, 0 = no)

^bCountry-specific daily caloric intake per capita were defined as follows:

Low = less than 2,100 (Bangladesh, China, Ghana, India, Indonesia)

Mid = greater than 2,100 and less than 2,800 (Barbados, Brazil, Chile, Costa Rica, Mexico-MHAS, Mexico-SABE, Puerto Rico, South Africa)

High = greater than 2,800 (Cuba, Uruguay)

^cLiving now in a good calorie country was defined as higher than 2,700 daily caloric intake per capita (1 = yes, 0 = no); Bangladesh, India, and Ghana were classified as lower caloric intake countries using data from 2000. Most higher caloric intake countries were in the mid caloric category in the 1930s

^dObese (1 = yes, 0 = no) based on BMI greater than or equal to 30

^eDifficulty with functionality was a harmonized measure using ADLs. Poor self-reported health was also based on a harmonized version of the self-reported question asked about one's general health

^fBIC is a criterion for model selection. Models with smaller BIC indicate better fit

increases the odds of diabetes by about 25–30 % (Models 3–5), reporting poor health doubles the odds of diabetes (Models 4 and 5), and having visited a doctor or medical care professional at least once within the last year also doubles the odds of diabetes (Model 5).

In terms of obesity, strong positive associations appear between poor early life nutritional environment (country-level daily caloric supply per capita) and adult obesity for older adults born in mid-caloric intake countries (e.g., Puerto Rico, Costa Rica, Chile) (Table 4.6, Model 1). The odds of being obese as an adult for those born in the mid-caloric intake countries are about 1.5 times the odds of being obese in a country where early life caloric intake was much higher (Uruguay and Cuba). In contrast, being born in a very low-caloric country (e.g., China, India, and Ghana) reduces the odds of adult obesity dramatically. Being born in a low- or mid-caloric country and in a rural area (proxy for low parental SES) also reduces the odds of being obese; the effect is much stronger in the low-caloric countries (Model 1). The reduced odds of obesity for the mid-caloric countries suggest that those who are now at increased risk of adult obesity are those born in urban areas whose parents had a better SES. The results are consistent when adding years of education and currently living in a high calorie country (Model 2), adult smoking (Model 3), other health problems such as difficulties with functionality and poor self-reported health (Model 4), and visits to a medical professional within the last year (Model 5).

Other factors are also important in predicting adult obesity. Notably, the odds of being obese for females are over two times that for males. Each additional year of education increases the odds of being obese as an adult by 8 %, and living in a high calorie country increases the odds of being obese by 40–64 % (Models 2–6). Yet, living in a high calorie country and having more years of education decreases the odds of being obese slightly. Having ever smoked reduces the odds of being obese (Models 3–5), but having at least one difficulty with functionality increases the odds of being obese by 57–58 % (Models 4 and 5); having visited a doctor within the last year increases the odds of being obese by about 15 % (Model 5).

4.2.1 Discussion

Data from major surveys representative of older adults and from historical data were used to examine associations between early life nutritional and infectious disease environments and older adult heart disease and diabetes. Multivariate models using individual-level and country-specific data were then estimated controlling for potentially confounding factors. In general, the profile of heart disease for many older adults in low- and middle-income countries was not worse than what is observed in today's developed countries, although it may be slightly worse in some countries such as Chile and Puerto Rico when compared against historical estimates during the 1960s–1970s in the US when the US began experiencing decreasing mortality due to heart disease. There was an overall higher

Table 4.6 Caloric intake and the likelihood of being obese for those born in the late 1920s–early 1940s

Variables	Model 1	Model 2	Model 3	Model 4	Model 5
Demographic					
Female	2.41***	2.46***	2.23***	2.17***	2.15***
Age	0.99**	0.99**	0.99**	0.99***	0.99***
Years education		1.08***	1.08***	1.08***	1.08***
Childhood					
Rural birthplace ^a	1.04	1.05	1.06	1.07	1.08
Low caloric intake ^b	0.26***	0.26***	0.24***	0.25***	0.27***
Mid caloric intake ^b	1.52***	1.53***	1.44***	1.45***	1.46***
High (reference) ^b	1.00	1.00	1.00	1.00	1.00
Low X rural birthplace	0.29***	0.33***	0.33***	0.33***	0.33***
Mid X rural birthplace	0.76*	0.76*	0.74*	0.73**	0.72**
Adult lifestyle					
Good calorie country ^c		1.40**	1.41**	1.54***	1.64***
Good calorie X education		0.93***	0.93***	0.93***	0.93***
Used to smoke			0.94	0.92	0.92
Smokes now			0.58***	0.58***	0.58***
Never smoked (ref)			1.00	1.00	1.00
Adult health					
Difficulty with functionality ^d				1.58***	1.57***
Poor reported health				1.03	1.01
System					
Visited a doctor within the last year					1.16***
Log likelihood	−9,604	−9,590	−9,543	−9,505	−9,499
BIC ^e	19,290	19,292	19,220	19,163	19,161
Total observations	27,105	27,105	27,105	27,105	27,105

Source: RELATE, 2013; excluding high income countries – US, Netherlands, and England. Taiwan is not included because its survey did not have information on birthplace. Russia is excluded because its historical circumstances make it a more complex case to categorize (Anderson & Silver, 1986; Prokhorov, 2005–2009)

Notes: Odds ratios are shown

* $p \leq 0.05$; ** $p \leq 0.01$; *** $p \leq 0.001$

^aBorn and lived in rural area during childhood (1 = yes, 0 = no)

^bCountry-specific daily caloric intake per capita were defined as follows:

Low = less than 2,100 (Bangladesh, China, Ghana, India, and Indonesia)

Mid = greater than 2,100 and less than 2,800 (Barbados, Brazil, Chile, Costa Rica, Mexico-MHAS, Mexico-SABE, Puerto Rico, South Africa)

High = greater than 2,800 (Cuba, Uruguay)

^cLiving now in a good calorie country was defined as higher than 2,700 daily caloric intake per capita (1 = yes, 0 = no); Bangladesh, India, and Ghana were classified as lower caloric intake countries using data from 2000. Most higher caloric intake countries were in the mid caloric category in the 1930s

^dDifficulty with functionality was a harmonized measure using ADLs. Poor self-reported health was also based on a harmonized version of the self-report question asked about one's general health

^eBIC is a criterion for model selection. Models with smaller BIC indicate better fit

likelihood of reporting heart disease in countries with higher caloric intake prior to World War II. However, there was also a strong association between being born in mid caloric intake countries (also the tip of the iceberg countries) and low parental SES and heart disease. The effects of childhood conditions do not appear to be confounded by later-life events such as adult smoking or caloric intake.

The profile of adult obesity and diabetes requires a slightly different interpretation. The pattern for adult diabetes and obesity was worse than that of the developed world in the low caloric countries; this was even more true of the mid caloric countries, where it was higher than in high-income countries and some middle-income countries such as Argentina and Uruguay. Being born in the late 1920s–early 1940s in a tip of the iceberg country was strongly associated with adult diabetes and obesity; in some instances these effects were as strong as the effects of adult conditions and lifestyle such as obesity and smoking. These effects were not attenuated after controlling for caloric intake at the time of the older adult surveys or were they compounded when adding adult lifestyle.

The observed patterns for heart disease and diabetes support the idea that poor nutrition and infectious diseases in early life are important to later adult health. As noted in Chap. 2, lower caloric intake can affect maternal health and the mother's ability to properly nourish the unborn and infants resulting in higher infant deaths.¹ It can also leave children vulnerable to infectious diseases and poor childhood health which may have consequences during adulthood.² Although it is not possible to disentangle the effects of poor nutrition and infectious disease or the timing of effects, the results do suggest the importance of a critical period during early life whether it be *in utero*, infancy or childhood, because the effects of early life remained consistent even after controlling for adult lifestyle and caloric intake. In this regard, the argument that adult lifestyle or changes to a diet with higher levels of saturated fat accompanied by a more sedentary lifestyle could be the only major contributor of adult diabetes and heart disease countries may not completely be accurate, especially in the tip of the iceberg countries.

The results partially confirm existing evidence suggesting that the health of the unique cohorts in the tip of the iceberg countries is worse than that of cohorts from other countries,³ at least in terms of diabetes and obesity. The prevalence of diabetes in older adults has never been as high historically in the US, the Netherlands, and England as that depicted in Fig. 4.2 for developing countries. Even into the 1980s and 1990s, the prevalence of diabetes and heart disease among older adults 50 years and older in the US was lower than what some developing

¹ Lannoy (1963).

² Elo and Preston (1992).

³ McEniry et al. (2008), McEniry and Palloni (2010), and McEniry (2011b).

countries are currently experiencing.⁴ (Recent increases in diabetes in the US point to a younger generation born after 1945 that is at greater risk.⁵)

It may be that the prevalence of heart disease noted in Fig. 4.1 in the developing world is higher than what was experienced in the developed world at similar moments during the demographic transition. One point in the demographic transition worthy of note is that at which countries experienced mortality decline due to heart disease. This generally occurred in the developed world in the 1960s and in some developing countries several years later.⁶ The prevalence of heart disease among older adults in the 1960s–1970s in the US, England, and the Netherlands is lower than the observed prevalence among some low- and middle-income countries shown in Fig. 4.1.⁷ For example, in the early 1960s, the prevalence of heart disease in the US among older adults (aged 55–64) was slightly greater than 10%.⁸ From the early 1970s through the early 1990s, the prevalence rate for angina pectoris for US men and women aged 65–74 ranged between 3 and 8% using a well-validated set of symptom questions⁹; the prevalence for those aged 40–64 ranged between 4 and 7% during this same time period. While not a perfect comparison, it is also noteworthy to point out the prevalence of heart disease comparing Puerto Rican adults at approximate similar ages. The prevalence of heart disease for Puerto Rican males born at the turn of the century when they were 55–64 years of age during the 1960s was

⁴ For example, in the US see Gordon (1964), Wilkerson and Krall (1947), and for England and Wales see Allender, Scarborough, O’Flaherty, and Capewell (2008), García-Palmieri et al. (1970), and Walker and Kerridge (1961). For later periods, in the US see Alexander, Landsman, Teutsch, and Haffner (2003), Cowie et al. (2006), Gregg et al. (2004), Hadden and Harris (1987), Harris et al. (1998), and for the Netherlands see Ubink-Veltmaat et al. (2003). Wilkerson and Krall (1947) estimated the prevalence of diabetes for 55–64 years old in the US was about 4% overall and higher for females (7%) compared with men (2%). The prevalence of diabetes for US men aged 55–64 during the 1960s was 3.3% (Gordon, 1964). In the 1970s, the prevalence of diabetes in the US among older adults began to increase. Hadden and Harris (1987) reported a prevalence rate of 12.8% for adults aged 55–64 and 17.7% for adults aged 65–74 from 1976–1980 data. Harris et al. (1998) described the prevalence of diabetes in the US for adults aged 60–74 to be about 13% overall and about 24% for Mexican-Americans in the year 1988–1994. In another study (Flegal et al., 1991), the prevalence of diabetes in Mexican-Americans and Puerto Ricans aged 45–74 years old in the early 1980s was about 14.3% for both groups. The prevalence of diabetes was higher for women and higher than that of non-Hispanic whites.

⁵ Day (2001).

⁶ For example, in the case of Puerto Rico, mortality decline due to heart disease declined beginning in the 1980s (Palloni, McEniry, Dávila, & García Gurucharri, 2005).

⁷ For the US see Dawber, Moore, and Mann (1957), Ford and Giles (2003), Gordon and Garst (1965), Harper, Lynch, and Davey Smith (2011); and for England and Wales see Allender et al. (2008) and García-Palmieri et al. (1970). Even into the 1980s and 1990s, the prevalence of heart disease among adults 50 years and older was lower than what some developing countries are currently experiencing (for example, in the US see Ford & Giles, 2003).

⁸ The prevalence of all coronary heart disease (myocardial infarction, angina pectoris, coronary insufficiency) among 55–64-year-old men in the US National Health Survey (NHS) in 1960–1962 was 14%.

⁹ Prevalence based on the Rose questionnaire (Ford & Giles, 2003; Rose, 1962).

about 8 %¹⁰ – slightly less than what was observed in the US during the same period. In contrast, the prevalence of heart disease for the cohort of Puerto Rican males born in the late 1920s–1940s when they were 60–74 years old is approximately double this amount (results not shown but based on Fig. 4.1).

The contrasting patterns of heart disease and diabetes in Figs. 4.1 and 4.2 are initially puzzling. However, as described in Chap. 1, heart disease and diabetes are two very different health conditions that may result from poor nutrition and/or infectious disease in early life. Poor *in utero* early life nutritional environments reflected by low birth weight are more directly associated with adult diabetes than with adult heart disease, whereas a diet high in saturated fat is more directly associated with heart disease.¹¹ Clearly, the graphical depiction of simple associations at a population level does not tell the complete story and reflects the limitations of population-level data for reaching conclusions about individual-level associations.¹² The supposition that the pattern in Fig. 4.1 partially reflects a direct association between diets high in saturated fat and cholesterol in early life and adult heart disease was supported by individual-level data. In addition, a strong association with early life nutritional environments and adult heart disease among adults with poor early SES in the tip of the iceberg countries became apparent.

The pattern for heart disease could reflect the importance of composition of diet in early life. A diet with a higher proportion of calories from animal products is a recognized contributor to adult heart disease.¹³ As described in Chap. 1, the diet in higher-caloric countries prior to World War II had a higher proportion of calories from animal products (meats and dairy) and, therefore, a higher protein level than poorer countries; this was especially true in Argentina and Uruguay. In contrast, the diet of countries that are now middle income consisted of more consumption of roots and tubers and, in some cases, sugar, whereas in very poor countries a large proportion of caloric intake came from staples such as rice. On the other hand, the pattern may reflect confounding between early life caloric intake and current higher consumption of saturated fat; diets high in saturated fat may not yet be as widespread in the developing world as they are in the developed world.

The fact that heart disease is more prevalent in developed countries may partially reflect the increasing survivorship of those with heart disease due to medical advances and high quality medical care that can better diagnose, enhance, and prolong life. Many older adults in poorer countries may not live long enough to manifest major problems related to heart disease. The noted lower historical prevalence of heart disease could simply be an indication of better diagnostic

¹⁰ The prevalence of coronary heart disease in Puerto Rico from the Puerto Rico Heart Health Program (PRHHP) for males aged 55–64 in 1965–1968 was 8 % (García-Palmieri et al., 1970, Table 10; National Heart, Lung, and Blood Institute, 2008).

¹¹ Popkin, Horton, and Kim (2001).

¹² Greenland and Robins (1994).

¹³ Marmot and Elliott (2005).

methods for heart disease since the 1960s. Nevertheless, survivorship does not completely explain the contrasting patterns precisely because of the pattern of higher prevalence of diabetes in older adults in developing countries with low caloric intake prior to World War II.

The results may reflect the differences in the ages at which different sets of symptoms become apparent, but it may be too early to observe a higher prevalence of heart disease in some developing countries. The compounding effects of a poor early life nutritional environment with a rapid transition to a diet high in saturated fat may have a more immediate effect on the prevalence of chronic conditions such as diabetes than it does on heart disease, where it may take several generations.¹⁴

There are differences in how each gender is affected by early life conditions. Females may be more influenced by maternal protein metabolism as reflected in shorter height.¹⁵ Under nutrition *in utero* may have a more negative impact on males than females because of differences in growth patterns; males grow faster than females *in utero* and therefore may be more at risk for suffering the consequences of nutritional deprivation at particular times. However, it is likely that the higher risk of heart disease and diabetes for females is partially explained by differences in accessing the health care system and taking care of personal health. Women may tend to visit a doctor more often than men in some settings and thus may be more aware of their health problems.

The strong effects of smoking on adult heart disease and the importance of adult obesity, functionality, overall health, and visits to a medical professional are not surprising. Smoking, obesity, and functionality are strongly associated with chronic conditions¹⁶; overall health and number of medical visits may reflect that those who visit medical professionals simply have a higher chance of knowing they have heart disease or diabetes.

The data are cross-sectional, and it is not possible to fully examine, for example, the meaning of the positive association between caloric intake and heart disease. It could reflect that high energy intake in early life may influence heart disease development and prognosis. It could also reflect just the opposite, that those who survived heart disease have a higher energy intake. Other limitations exist. The broad and crude measure of caloric intake makes it more difficult to adequately control for adult caloric intake especially when most of the selected countries now have a much higher level of caloric intake and are wealthier.

Underestimation may not be problematic. The use of a well-validated instrument for angina by SAGE countries produced a lower likelihood of heart disease than in highly developed countries such as the US, England, and the Netherlands or in developing countries such as Argentina and Uruguay. Models using obesity, a less subjective measure of health, as a dependent variable paralleled results from

¹⁴ Trowell and Burkitt (1985).

¹⁵ Eriksson, Kajantie, Osmond, Thornburg, and Barker (2010) and Ravelli et al. (1998).

¹⁶ Kuh and Ben-Shlomo (2004).

models with diabetes as the dependent variable which indicates that self-reported diabetes may not be far off the mark in reaching general conclusions about cross-national patterns. Even if the prevalence of diabetes was much higher than reported in low- and middle- income countries, the resulting changes illustrated in Fig. 4.2 would strengthen the noted negative observation between low early life caloric intake and adult diabetes. Judging by the multivariate results, this may not have made a difference in several SAGE countries.

4.3 Long Term Consequences for Older Adult Health: The Case of Puerto Rico

Puerto Rico complements the cross-national patterns described in the previous sections. Figure 4.3 shows employment across the year for the sugar, tobacco, and coffee industries in Puerto Rico from the 1920s.¹⁷ These employment numbers reflect the seasonality of the predominant sugar cane industry, in which periods of higher employment occurred during the sugar cane harvest (January–June) followed by periods of below average employment (July–December). Marriages and fertility revolved around the agricultural seasons (especially the sugar cane season)¹⁸; prior to the mid-1940s, marriages were common after the sugarcane harvest (June) and births peaked in the spring.

Superimposed on Fig. 4.3 are descriptions of the timing of exposure to poor nutrition and infectious diseases during late gestation. As explained in Chap. 2, season of birth is a good indicator of early growth and development under certain restricted conditions. If early life hypotheses regarding critical timing of nutritional and infectious diseases have merit, those born during or at the end of the lean season should be at higher risk of older adult heart disease and diabetes.

The results of several analyses suggest that season of birth under certain restricted conditions provides insight into very early life exposures and their impact on older adult heart disease and diabetes in Puerto Rico. **First**, the effects of season of birth on adult Puerto Rican health have been remarkably strong, robust, and in the expected direction, especially for heart disease (Table 4.7). Being born during or at the end of the lean season was associated with older adult heart disease and diabetes for those Puerto Ricans born and raised in the countryside.¹⁹ It was not associated with any other predictor variable (e.g., childhood health, childhood SES, low knee height, low height, education, age or gender), nor did competing hypotheses appear to explain the results (e.g., family planning, breast feeding or timing of mother's work). In the first analysis (Table 4.7, Source #1), being born at the end of the lean season increased the odds of reporting heart disease by almost

¹⁷ Clark (1930).

¹⁸ Vázquez Calzada and Rivera Acevedo (1982).

¹⁹ McEniry et al. (2008).

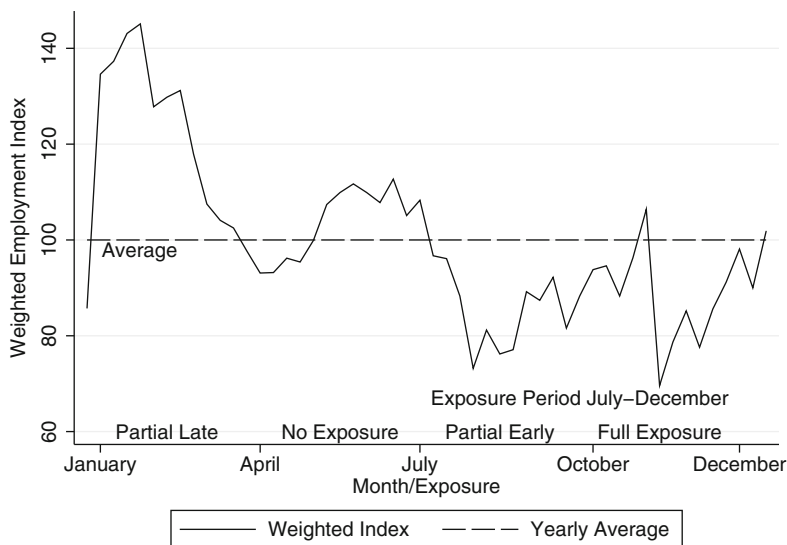


Fig. 4.3 Seasonal variation in plantation employment and hypothesized exposure during late gestation. *Notes:* Data are for sugar, tobacco, and coffee plantations, 1924–1926 (*Sources:* Published with permission from *Journal of Gerontology* (McEniry, Palloni, Dávila, & García, 2008). Original source is Clark, 1930)

Table 4.7 Effects of being born during the lean season on adult health in Puerto Rico (reference group born during harvest)

Health outcome	OR/HR (95 % CI)
1. Heart disease	1.89 (1.30–2.75)
1. Diabetes	1.81 (1.15–2.85)
2. Heart disease	1.65 (1.18–2.33)
3. Heart disease	2.20 (1.38–3.50)
3. Diabetes	2.09 (1.08–4.07)

Sources: (1) McEniry et al. (2008); (2) McEniry and Palloni (2010), and (3) McEniry (2011b)

Note: All studies examined 60- to 74-year-olds born in Puerto Rico who lived in the countryside as children

90 % in comparison with being born at the end of the harvest season; for diabetes, odds were increased about 80 %. When the effects of season of birth on adult heart disease and diabetes were examined according to IMR (Table 4.7, Source #3), the likelihood of reporting heart disease or diabetes for those born during the lean season was a little over two times that of those born during the harvest when IMR was lower.

Second, strong effects of season of birth on adult health were found even after controlling for other childhood conditions (Table 4.7, Source #1), adult conditions and risk behavior (Table 4.7, Sources #2–3). Poor childhood health and rheumatic fever were associated with older adult heart disease and diabetes, but poor

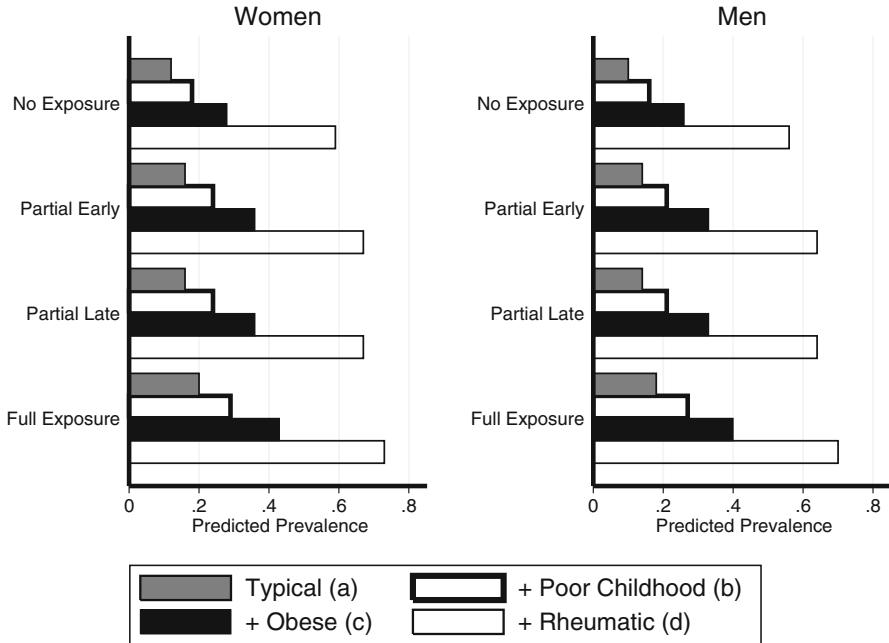


Fig. 4.4 Predicted prevalence of heart disease by level of exposure during late gestation. *Notes:* The predicted probabilities corresponding to the (a) typical respondent (between the ages of 65 and 69 with 7 years of education), (b) plus poor childhood conditions (poor child health, poor child socioeconomic status, low knee height), (c) plus adult obesity, and (d) plus rheumatic fever during childhood follow: Women: no exposure (0.12, 0.18, 0.28, 0.59), partial early exposure (0.16, 0.24, 0.36, 0.67), partial late exposure (0.16, 0.24, 0.36, 0.67), full exposure (0.20, 0.29, 0.43, 0.73). Men: no exposure (0.10, 0.16, 0.26, 0.56), partial early exposure (0.14, 0.21, 0.33, 0.64), partial late exposure (0.14, 0.21, 0.33, 0.64), full exposure (0.18, 0.27, 0.40, 0.70) (Source: PREHCO (2007), imputed; all 60- to 74-year-olds born in Puerto Rico and who lived in the countryside as children ($n = 1,457$). Published with permission from *Journal of Gerontology* (McEniry et al., 2008))

childhood SES was not.²⁰ The risk of heart disease was 65 % higher among those born during the lean season in comparison with those born during the harvest, controlling for other childhood and adult conditions (Table 4.7, Source #2).

Third, the magnitude of effects for seasonal exposure reflects an arguably substantial impact on adult heart disease and diabetes. For example, for the typical respondent, a change from the best to the worst exposure seasonal category increased the probability of self-reported heart disease (diabetes) by about 80 % (73 %) for men and 66 % (60 %) for women (Table 4.7, Source #1, Fig. 4.4 for heart disease and Fig. 4.5 for diabetes). The predicted probability of heart disease increased when poor childhood conditions (e.g., health, knee height, and SES) and adult obesity were added. The increase became even more pronounced when rheumatic heart fever was introduced as a risk factor. In the case of diabetes, the

²⁰ McEniry et al. (2008) and Palloni et al. (2005).

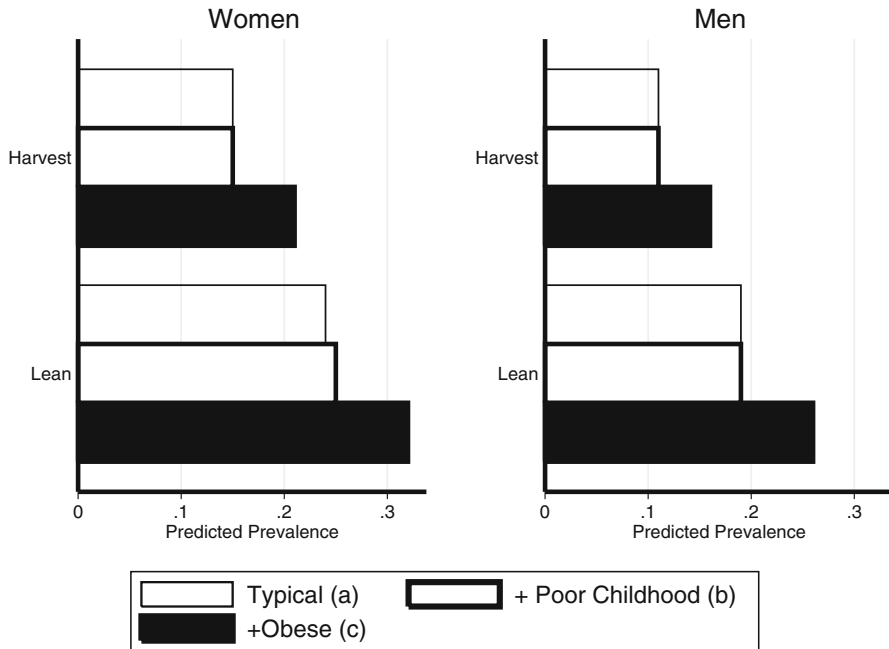


Fig. 4.5 Predicted prevalence of diabetes by exposure period. *Notes:* The predicted probabilities corresponding to the (a) typical respondent (between the ages of 65 and 69 with 7 years of education), (b) plus poor childhood conditions (poor child health, poor child socioeconomic status, low knee height), and (c) plus obesity follow: Women: harvest (0.15, 0.15, 0.21), lean season (0.24, 0.25, 0.32). Men: harvest (0.11, 0.11, 0.16), lean season (0.19, 0.19, 0.26) (*Source:* PREHCO (2007), imputed; all 60- to 74-year-olds born in Puerto Rico who lived in the countryside as children and have no family history of diabetes ($n = 1,457$). Published with permission from *Journal of Gerontology* (McEniry et al., 2008))

predicted probability increased slightly when childhood conditions (e.g., health, knee height, and SES) and obesity were added.

These effects increased when dividing respondents into groups according to either high or low IMR during their birth year.²¹ The probability of heart disease for the average healthy female respondent (i.e., 65–69 years of age with 7 years of education, who never smoked and exercises) in Puerto Rico was of the order of 0.09 for women born during the harvest and 0.18 for women born during the lean period. For men, the figures were 0.06 and 0.13, respectively. Thus, for the average healthy respondent, a shift from the best to the worst exposure category more than doubled the probability of self-reported heart disease for both men and women. The predicted probability of heart disease also increased when poor childhood conditions (e.g., health, knee height, and SES) and adult conditions (e.g., obesity, previous smoker, and does not exercise) were added. For the full model, the probability of heart disease for women (men) was 0.51 (0.42) for those born during

²¹ McEniry (2011b).

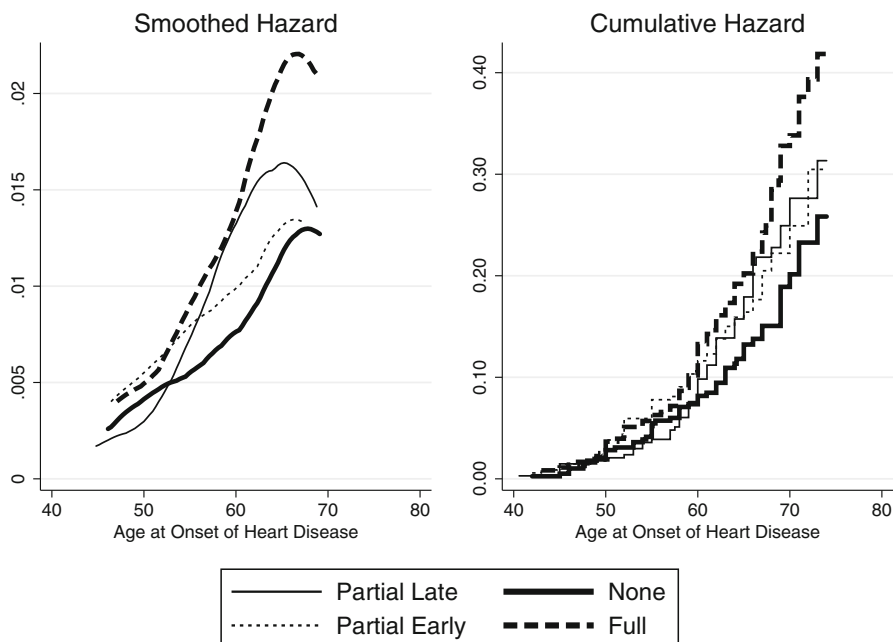


Fig. 4.6 Kaplan Meier hazard estimates by level of exposure during late gestation (Source: PREHCO, 2007, imputed ($n = 1,438$). Just those 60–74 years old born during the late 1920s and early 1940s who said that they lived most of their childhood in rural areas. Published with permission from *Demography* (McEniry & Palloni, 2010))

the harvest and 0.71 (0.62) for those born during the lean period. While both childhood and adult conditions contributed to the increased probability of adult heart disease, adult conditions increased the probability more.

When dividing respondents into groups according to high or low IMR during their birth year, the predicted probabilities for diabetes for the typical healthy female respondent in Puerto Rico were 0.11 for those born during the harvest season and 0.19 for those born during the lean season; for men, the numbers were 0.10 and 0.18, respectively. Thus, for the average healthy respondent, a shift from the best to the worst exposure category increased the probability of self-reported diabetes by about 73 % for women and 80 % for men. When childhood conditions (e.g., health, knee height, and SES) and adult conditions (e.g., obesity, previous smoker, and does not exercise) were added, the predicted probability of diabetes increased; the probability of diabetes for women (men) was 0.34 (0.32) for those born during the harvest and 0.50 (0.48) for those born during the lean period. Childhood conditions had a slightly larger impact on increasing the probability of diabetes than did adult conditions.

Fourth, the hypothesis that *in utero*/early infancy exposure to poor nutrition and infectious diseases may be associated with early onset of adult heart disease was tested using event history analysis (Table 4.7, Source #2). While these early insults are associated with the probability of the occurrence of adult heart disease, they are not associated with the early onset of heart disease. Estimates of the smoothed hazard and cumulative hazard functions by quarter of birth suggest larger differences due to

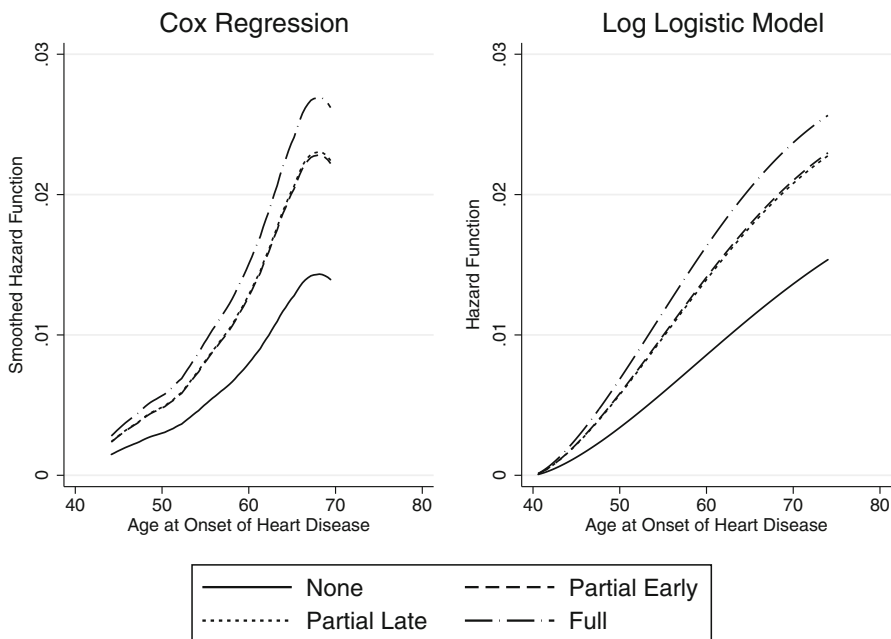


Fig. 4.7 Predicted hazard by level of exposure during late gestation (*Source*: PREHCO (2007), imputed ($n = 1,438$). Just those 60–74 years old born during the late 1920s and early 1940s who said that they lived most of their childhood in rural areas. Published with permission from *Demography* (McEniry & Palloni, 2010))

exposure level after age 60 (Fig. 4.6). The risk of developing heart disease in the group with full exposure during late gestation (fourth quarter) was approximately twice as high as for those within the unexposed category (second quarter) at later ages. The baseline hazard function showed a monotonic increase with age, peaking at around age 66 then decreasing. There were no statistical differences in survival curves by level of exposure for those respondents who lived in urban areas during childhood ($n = 1,128$, results not shown). Figure 4.7 shows that both the Cox and log logistic models produced similar fit with the data. Therefore, it is not surprising that predicted hazards and cumulative hazards from each are very similar, although the log logistic model tended to produce greater hazards at older ages, when the hazard doubled for full exposure (fourth quarter) in contrast with lesser exposed persons (second quarter); hazards of partial exposure appeared closer to those of full exposure.

4.3.1 Discussion²²

Several analyses using season of birth as a marker of *in utero* and early infancy exposures showed a strong association between being born in the lean season and a

²²Some of the text in the following discussion is taken directly from the author's previous publications, including McEniry et al. (2008), McEniry and Palloni (2010), and McEniry (2011b).

higher likelihood of older adult heart disease in Puerto Rico; findings for adult diabetes were less robust. The results consistently suggest the importance of *in utero* and early infancy exposure to poor nutrition and infectious diseases for later adult health. The pattern of exposure and risk of heart disease supports the Barker hypothesis that a more favorable nutritional environment in early gestation sets the fetus on a path of rapid growth which may cause problems if the fetus experiences severe under nutrition during late gestation.²³ Puerto Ricans born at the end of the lean season (who have a higher risk of heart disease) would have experienced early gestation during more favorable circumstances (second quarter, end of the harvest). Puerto Ricans born at the end of the harvest season (lower risk of heart disease) would have experienced early gestation during less favorable circumstances (third quarter) leading to slower growth. It is possible this group would have been less affected by undernourishment during late gestation; these Puerto Ricans, however, most likely experienced better nutrition during late gestation because it was during the harvest season. This result is consistent with several studies which have shown the importance of nutrition *in utero* and adult health status.²⁴ This interpretation does not negate the importance of infectious and inflammatory processes over the life course.²⁵ The synergy between nutrition and infectious disease makes it difficult to disentangle their effects.²⁶

The results help further illuminate associations between early life conditions and older adult health, suggesting that the patterns noted for heart disease and diabetes reflect more than just adult risk behavior and changes in diet during adulthood. The findings have implications for understanding the health of older adults in tip of the iceberg countries such as Costa Rica and Chile, which experienced a mortality decline in the late 1920s and early 1940s due to public health interventions including medical technology that was similar to Puerto Rico's; these countries also produced a large pool of survivors of adverse early life conditions who are now experiencing an increased prevalence of heart disease and diabetes.²⁷ The findings also have implications for understanding what may lie ahead for older adults part of the unique cohorts and born in developing countries during the later portion of the 1930s–1960s.

Detailed discussions of the results are available elsewhere.²⁸ However, a few points are noteworthy. Season of birth can be used to approximate the effects of nutritional deficiencies *in utero* under certain restricted conditions. The difficulty of testing a hypothesis sensitive to community heterogeneity was not a concern in a small and highly homogeneous country such as Puerto Rico, where national and

²³ Barker (2005).

²⁴ Costa (2005), Doblhammer (2004), Gavrilov and Gavrilova (2005), Moore et al. (1999), Prentice and Cole (1994).

²⁵ Finch and Crimmins (2004).

²⁶ Scrimshaw (1968, 1997).

²⁷ Palloni et al. (2005).

²⁸ McEniry et al. (2008), McEniry and Palloni (2010), and McEniry (2009b, 2011b, c).

regional seasonality and economic indicators are highly consistent with each other; in other settings this may not be the case. Using season of birth requires additional historical data to discern the environmental setting in early life. In Puerto Rico, the rainy season coincided with the lean season but in other settings this may not be the case, where seasonality of nutrition based on harvest times may or may not coincide with weather patterns (very dry versus very rainy) which affect the demand for nutrition because of higher exposure to infectious diseases.

Different etiologies of heart disease and diabetes exist in different environments and the physiological mechanisms related to timing.²⁹ The differential effects of the timing of exposure during mid- and late-gestation and early infancy on adult health are largely unknown, although slow growth during early infancy is associated with later heart disease.³⁰ Traits may also vary across cohorts that attenuate the linkages between early exposure and adult health status. Differences in immune functions and exposures to other infections or environmental risks³¹ and differences among cohorts' experiences may influence the effects of season of birth on adult health.

One possible complicating factor is the practice of breastfeeding and weaning. Although no specific data could be obtained to examine the specific variations in breastfeeding in the rural countryside in Puerto Rico during the 1930s and 1940s, the use of bottled formula did not become prevalent until the late 1940s.³² Thus, breastfeeding probably followed traditional norms (nearly universal and of long duration) in the rural countryside prior to the late 1940s. If this was the case and the nutritional status of the mother influences the production as well as the quality of her milk,³³ children whose mothers experienced infection/malnutrition right before their birth and for sometime thereafter could be affected. Therefore, breastfeeding could be a mechanism (not a confounder) through which season of birth affects early nutritional status and growth.

The findings for Puerto Rico are remarkably robust. If anything, the models over-control for traits that, in theory at least, are affected by early exposure, such as height, knee height, BMI, and even diabetes. However, very similar results are found and inferences remain unchanged when these variables are excluded from model estimation. It is clear, then, that estimated effects should be considered the lower bounds of the true estimates because inferences are on the conservative side. Thus, the results suggest that early life conditions have important health ramifications for later adult health among this population of older Puerto Ricans, which can help us better understand the experiences in other countries as part of the observed cross-national morbidity patterns.

²⁹ Godfrey and Barker (2000).

³⁰ Barker, Eriksson, Forsen, and Osmond (2002), Eriksson, Forsen, Tuomilehto, Osmond, and Barker (2001), and Osmond, Barker, Winter, Fall, and Simmonds (1993).

³¹ Moore et al. (1999) and Simondon et al. (2004).

³² Becerra and Smith (1990).

³³ John, Menken, and Chowdhury (1987).

4.4 Conclusions

The results provide conservative estimates of the relative importance of early life nutrition and infectious diseases on older adult diabetes and heart disease. In spite of the potential for underestimation of chronic adult heart disease and diabetes because of self-report, one piece of evidence in support of the conjecture regarding the unique cohorts born in the 1930s–1960s comes from examining morbidity patterns using comprehensive surveys representative of older adults at national, regional or major city levels. While it is not possible to identify the timing or disentangle the synergy between nutrition and infectious diseases, the patterns found support early life nutritional and infectious disease arguments in relation to adult heart disease and diabetes. The results are not modified when adult circumstances such as lifestyle, obesity, living in a more nutritionally rich country or use of medical services are taken into account. There exists the intriguing possibility that over-nutrition in early life may lead to a higher risk for adult heart disease. The cross-national comparisons taken together with the in-depth look at Puerto Rico suggest that pessimistic conjectures regarding the health of older adults in some settings of the developing world may not be far off the mark.

Levels of mortality of older adults in the tip of the iceberg countries should exceed what one would expect; it will be higher than that experienced by populations with equivalent socioeconomic conditions but where the growth of the elderly population attributable to early mortality decline is due to improvements in standards of living.

(Hypothesis #3)

Significant socioeconomic disparities in health status and disability of older adults in tip of the iceberg countries will be found. Keeping everything constant, social and economic disparities will be more salient in areas where the contribution of past mortality decline associated with deployment of novel medical technology is higher.

(Hypothesis #4)

Adapted from Palloni, McEniry, Wong, and Peláez (2006)

5.1 Mortality

Will the levels of mortality of older adults in the unique cohorts of the 1930s–1960s exceed what one would expect given current total levels of mortality (Hypothesis 3)? Will the observed morbidity patterns for adult heart disease and diabetes in the tip of the iceberg countries translate into higher mortality? There are only a small number of countries with available mortality data. However, a few pieces of evidence suggest a partial affirmative to the questions posed.

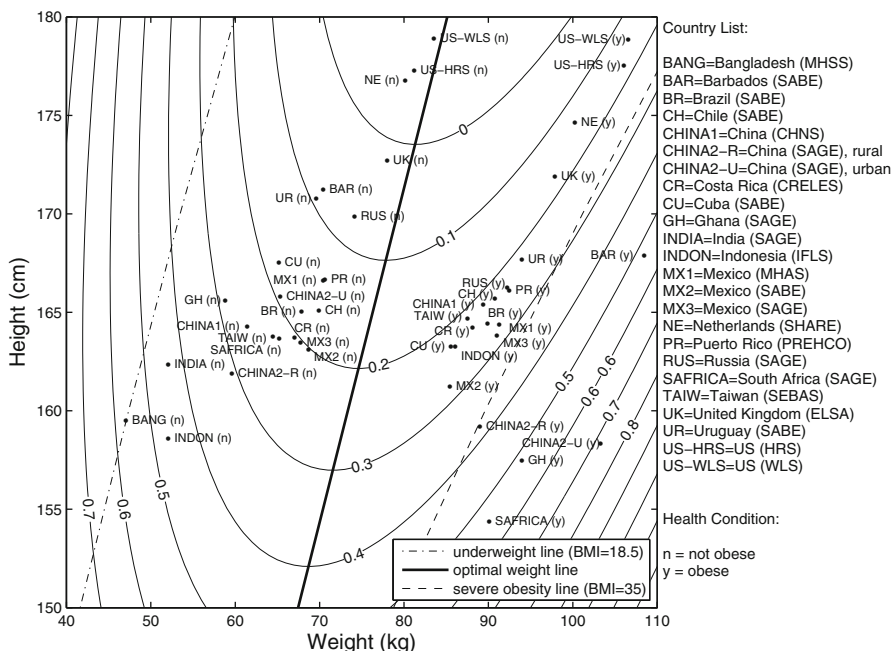


Fig. 5.1 Expected relative risk of mortality for males born during the late 1920s–early 1940s. *Notes:* Average heights and weights from cross national data using RELATE (2013). Expected relative risks are expressed as the $\ln(\text{expected RR mortality})$. After exponentiation, a relative risk of 1.00 means that the risk of death is equal to the overall average risk of death. A relative risk of greater than (less than) 1.00 means that the risk is greater (less) than the average risk for that population. Data on obesity from Bangladesh and India are not shown here because of very small sample sizes (*Source:* Waaler-type surface constructed using Waaler data (1984))

5.1.1 Waaler-Type Surfaces and Observed Mortality Rates

The first piece of evidence comes from examining expected relative risk of mortality using a modified Waaler-type surface based on Waaler mortality data.¹ Figure 5.1 shows mortality risk for non-obese and obese adult males born in the late 1920s through early 1940s. The average height and weight for non-obese and obese men are plotted on the surface. Each curve in the Waaler-type surface shows the expected relative risk of mortality for a particular height-weight combination. Taking a particular height and weight and then decreasing height, the mortality risk increases. Given a particular height, an optimal weight line depicts the point at which mortality risk is minimized.

The figure clearly shows a higher expected relative risk for obese individuals in the countries studied. The lowest mortality risk is found among taller males of normal weight and the highest mortality risk is found among shorter and obese males. The mortality risk for the non-obese groups is closer to the bolded optimal

¹ Waaler (1984).

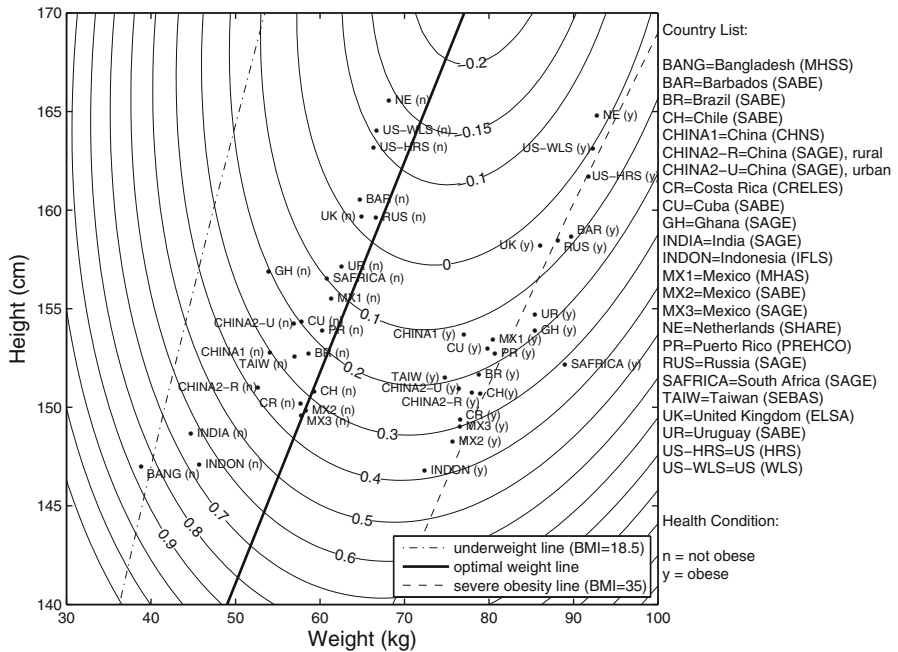


Fig. 5.2 Expected relative risk of mortality for females born during the late 1920s–early 1940s
Notes: Average heights and weights using cross-national data from RELATE (2013). Expected relative risks are expressed as the $\ln(\text{expected RR mortality})$. After exponentiation, a relative risk of 1.00 means that the risk of death is equal to the overall average risk of death. A relative risk of greater than (less than) 1.00 means that the risk is greater (less) than the average risk for that population (*Source:* Waaler-type surface constructed using Waaler data (1984))

weight line in the graph suggesting that their mortality risk is better than the obese groups. Only a few countries approach the underweight line.

The clustering of the countries is consistent for the most part with national income, but it is also consistent with mortality regimes of the early to mid twentieth century: the very early regimes (US-WLS, US-HRS, Netherlands, and England), followed by early regimes (Uruguay), then the mid-to-late demographic regimes (Chile, Puerto Rico, Costa Rica, Taiwan, South Africa, Mexico, and Brazil) and finally the very late demographic regimes (Indonesia, India, rural China-SAGE, and Bangladesh). Exceptions are Barbados which is closer to the earlier regimes, and those living in urban areas in China which are closer to mid-demographic regimes. The surface shows the disadvantage shorter older adults from low- and middle-income countries have compared with adults from high income countries. There are large disparities in mortality risk between non-obese and obese men in some cases. Obese older adult males from South Africa, Ghana, Barbados, and urban China show a particular disadvantage from being obese than non-obese. On the other hand Indonesian obese men showed lower mortality risk than non-obese men.

A modified Waaler surface for females using Waaler data is shown in Fig. 5.2. The general patterns are similar to those of males. Expected relative risk for obese females is consistently higher than for non-obese females, and females in higher

income countries are at an advantage over their counterparts from the developing world even when they are obese. The clustering of countries follows the general pattern of mortality regimes with the similar exception of Barbados and China—the former more closely aligned with the higher income countries and the latter with the mid-paced mortality regimes. South African obese and non-obese females appear to have one of the largest disparities in mortality risk.

The Waaler-type surfaces depicted in Figs. 5.1 and 5.2 show the expected relative risk of mortality based on a particular height and weight but they do not give an indication of the degree to which expected mortality risk translates into actual observed mortality risk. A comparison between expected mortality risk using a Waaler-type surface and actual mortality risk based on country-specific mortality data of adult males born in the late 1920s through early 1940s is depicted in Table 5.1. The Waaler-type surface is generally consistent in showing higher mortality risk for lower-income countries for both those with and without a particular health condition. Interestingly, the difference in mortality risk between those with and without the condition is not large for heart disease and diabetes. In examining overall observed mortality risk, there is a larger difference between those with and without a particular health condition, and the pattern moving from high-income to lower-income countries is less clear. The observed relative mortality risk for those reporting (not reporting) heart disease and diabetes is also generally greater (less) than the expected relative risk of mortality based on the Waaler-type surface. There are a few cases where observed mortality risk is higher for those with a health condition (obesity) such as China and Puerto Rico. Indonesian non-obese males using the Waaler-type surface show a higher mortality risk than obese males. Although not reported here, similar patterns emerged for females using Waaler-type surfaces and actual observed relative risk of mortality.

The estimated excess in relative risk of mortality using Waaler-type surfaces is shown in Table 5.2. Positive numbers indicate instances where the observed relative risk was higher than the expected relative risk; the larger the number the larger the deviation from the average weight for a particular subgroup. Overall, the mid-demographic regimes show a much higher excess of relative risk of mortality for diabetes and heart disease than do the US, Bangladesh, and China. Risk is much higher for those who reported diabetes and heart disease in Costa Rica, Puerto Rico and, especially, Mexico.

A second piece of evidence from a small number of countries comes from comparing estimated mortality rates for older adults who reported heart disease and diabetes. Males in high-income countries (the US and Netherlands) who reported heart disease had lower mortality rates than those from lower-income countries. Costa Rican male mortality rates were lower than those from Puerto Rico and Mexico (Table 5.3). For females who reported heart disease, this same general pattern holds except that Costa Rican and US white females have very similar mortality rates while US black females have a much higher mortality rate. For males who reported diabetes, mortality rates were lowest in the US and similar in the Netherlands, Costa Rica, and Puerto Rico followed by Mexico and Bangladesh. For females who reported diabetes, US black females and Bangladeshi females have the highest mortality rates. Curiously, Puerto Rican and Mexican females have similar mortality rates to US white females in the US-WLS study.

Table 5.1 Expected relative risk and observed relative risk (males born in the late 1920s–early 1940s)

	Waal84		Observed	
	Yes	No	Yes	No
Heart disease				
US-WLS	0.95	0.93	1.42	0.90
US-HRS	0.95	0.94	1.64	0.79
PR	1.09	1.13	1.56	0.89
CR	1.16	1.18	1.58	0.94
Mexico	1.29	1.22	2.64	0.94
China	1.26	1.30	0.67	1.00
Diabetes				
US-WLS	0.99	0.93	1.86	0.86
US-HRS	0.98	0.94	1.64	0.87
PR	1.12	1.12	1.58	0.76
CR	1.17	1.18	2.03	0.74
Mexico	1.23	1.22	1.95	0.87
China	1.20	1.30	0.81	1.02
Bangladesh	1.89	1.84	1.59	0.89
Obesity				
US-WLS	1.15	0.92	1.12	0.84
US-HRS	1.17	0.94	0.99	1.00
PR	1.27	1.14	0.43	1.02
CR	1.25	1.21	1.00	0.95
Mexico	1.25	1.25	0.99	0.97
Indonesia	1.23	1.65	1.17	0.68
China	1.44	1.32	0.26	0.99

Source: McEniry (2010b, Appendix) using surveys with mortality data including US-WLS; US-HRS; PR = Puerto Rico, PREHCO; CR = Costa Rica, CRELES; Mexico = MHAS; China = CHNS; Bangladesh = MHSS; Indonesia = IFLS

Notes: **Expected Relative Risk** is based on Waaler (1984) surfaces (Waal84 data from 1984 and CRELES data). **Observed Relative Risk** is based on observed mortality rates from survey data. A relative risk of 1.00 for a particular health condition means that the risk of death is equal to the overall average risk of death in a particular population of males. A relative risk >1.00 means that the risk is greater than the average risk for that population and a relative risk <1.00 means that the risk is less

Because there are no available mortality rates from other middle-income countries such as Argentina or Uruguay to compare with those of Puerto Rico, Mexico, and Costa Rica, it is difficult to draw conclusions from these data.

In Puerto Rico, historical data permit a closer examination of mortality. In 1963, the death rate from heart disease for males aged 45–64 in Puerto Rico was 202 per 100,000 compared with 679 per 100,000 for men of similar age on the US mainland.² These comparisons are not totally surprising, as the early 1960s was a time when mortality due to heart disease in the US and other developed countries

² García-Palmieri et al. (1970).

Table 5.2 Excess of relative risk of mortality (Waalers) for males born in the late 1920s–early 1940s

	US-HRS	US-WLS	Costa Rica	Puerto Rico	Mexico	Bangladesh ^a	China ^a
Diabetes							
Yes	0.10	0.05	0.23	0.21	0.30	−0.08	0.14
No	0.08	0.01	0.16	0.17	0.19	−0.19	0.11
Heart disease							
Yes	0.14	0.07	0.23	0.21	0.33	–	–
No	0.05	0.02	0.21	0.19	0.21	–	–

Source: McEniry (2010b, Table 6)

Notes: Data based on observed relative risk of mortality and expected relative risk of mortality from a modified Waaler-type surface based on Costa Rican–CRELES data. Excess of relative risk of mortality is a measure of the deviation of the observed relative risk of mortality from the expected relative risk using the modified Waaler surface. Positive numbers indicate instances where the observed relative risk was higher than the expected relative risk; larger numbers indicate a larger deviation

* $p \leq 0.05$; ** $p \leq 0.01$; *** $p \leq 0.001$

^aInformation regarding heart disease was not obtained for the respondents of the 1996 MHSS (Bangladesh). In the case of China, the prevalence of diabetes or heart disease was very small and thus the number of deaths from these conditions is also very small, leading to a possible overestimation

Table 5.3 Age-standardized mortality for those reporting heart disease and diabetes for those born in the late 1920s–early 1940s

	Heart disease		Diabetes	
	Males	Females	Males	Females
US-WLS	0.015	0.019	0.019	0.020
US-HRS Whites	0.014	0.029	0.014	0.035
US-HRS Blacks	0.018	0.048	0.017	0.046
Netherlands	0.019	0.016	0.022	0.007
Costa Rica	0.021	0.026	0.029	0.018
Puerto Rico	0.044	0.039	0.027	0.021
Mexico	0.068	0.061	0.051	0.029
Bangladesh	–	–	0.081	0.056

Source: RELATE (2013), author's calculations

Notes: The overall age-specific mortality rates estimated from survey mortality data (not shown in this table) were compared against 2000 life tables (WHO, 2002) and reported research (Dow, 2011; Zimmer, Kaneda, & Spess, 2007) and were, for the most part, similar and lower than the numbers presented in the above table. For example, the overall mortality rate for a 65 years old in the CRELES study is about 0.01 (Dow 2011); this implies that mortality due to heart disease is approximately two times higher than the overall mortality rate. The Bangladesh survey did not have a question regarding heart disease

was high but then began to seriously decline³; this transition occurred later in Puerto Rico. As can be seen in Table 5.3, for Puerto Rican men born during the late 1920s through early 1940s and interviewed in 2002–2003, the mortality rate

³Harper, Lynch, and Davey Smith (2011).

due to heart disease is now about three times that of the rate for US mainland men. However, mortality from heart disease in Puerto Ricans began a significant decline in the 1980s, slightly later than in the US.⁴ A similar comparison between Costa Rica and the US shows a smaller difference, suggesting that older adults in Costa Rica for some reason are better off than those in Puerto Rico.

5.1.1.1 Discussion

Modified Waaler-type surfaces based on height and weight showed a higher expected relative risk of mortality in low- to middle-income countries than high-income countries; in some poorer countries there were large discrepancies in mortality risk for those with and without a particular health condition. There was a high risk for those with diabetes and heart disease in the mid- and late-mortality regimes of Costa Rica, Puerto Rico, and Mexico using actual mortality data. Age-standardized mortality rates generally showed a similar pattern.

It is not clear the degree to which the expected mortality risks illustrated in the surfaces will translate into actual higher mortality rates at older ages because mortality data from surveys of older adults are not yet available for several of the countries depicted in the surfaces. However, both height and weight drives increased mortality risk. Higher income countries are clearly at an advantage even when average weight is much higher than average weight in poorer countries. The growth of height in the developing world during the early to mid twentieth century was modest due to the stagnant economic conditions,⁵ and early life hypotheses suggest that low height in the form of stunting may lead to overweight or obesity leading then to adult heart disease and diabetes.⁶ The surfaces suggest that historical circumstances place older adults in poorer countries at a considerable disadvantage in terms of mortality risk.

When actual mortality data are available, the results build on previous work⁷ and provide evidence, albeit rather weak, for the conjecture regarding high levels of mortality among regimes where the growth of the elderly population attributable to early mortality decline is due to the unique circumstances of the 1930s–1960s. Cohorts born in mid to late regimes (Puerto Rico, Mexico, Costa Rica) showed a higher excess of relative mortality risk and age-standardized mortality risk for heart disease and diabetes. These were the same cohorts (born during the late 1920s and early 1940s) which were more at risk of having been affected by harsh early childhood experiences while having a high probability of surviving.

It is not surprising there were not large differences in mortality risk for heart disease and diabetes but larger discrepancies for obesity using Waaler-type surfaces. Height may be indirectly related to heart disease and diabetes but directly related to obesity (see Figs. 1.2 and 1.3) thus providing a reasonable explanation for the pattern.

⁴ Palloni, McEniry, Dávila, and García Gurucharri (2005).

⁵ For example, López-Alonso (2007), Godoy, Goodman, Levins, Caram, and Seyfried (2007), and B. Harris (2000).

⁶ Popkin, Horton, and Kim (2001).

⁷ Palloni, McEniry, Wong and Peláez (2007).

The differences in mortality risk between the Waaler-type surfaces and actual observed mortality are also not surprising: observed mortality risk was for the same group of males born in the late 1920s through 1940s for whom average height and weight were calculated for the Waaler-type surfaces. However, the Waaler-type surfaces are an implicit modeling of mortality risk as a function of height and weight whereas the observed relative mortality risk is what really happened. This may explain part of the discrepancies between mortality risk based on Waaler-type surfaces and observed relative risk of mortality. Some of the noted higher mortality risk for those without a particular health condition especially in poorer countries may reflect that these health conditions are more prevalent among a better-off group of adults who may be taller or who may have better access to medical care.⁸ Smaller sample sizes for obese individuals may explain the reason for more extreme values of mortality risk displayed on the Waaler-type surfaces.

To some degree Waaler-type surfaces are a useful descriptive tool. Height reflects, in part, one's past circumstances in terms of nutrition, disease burden, and genetics, and weight reflects more recent circumstances such as lifestyle. However, height and weight by themselves do not adequately capture health. There are many factors which lead to higher mortality risk.

The surfaces based on Waaler data⁹ and on Costa Rican data were used as a standard by which to make cross national comparisons. However, it is important to highlight a couple of points regarding the constructed surfaces. The surfaces based on Waaler data are descriptive of a relatively healthy population and a high income country in the 1980s whereas the Costa Rican-CRELES surface is descriptive of a middle income country in the 2000s and depicts a population many of whom were stunted and exposed to poor early life conditions during the early to mid twentieth century. It may also be that the relationship between such conditions as obesity and mortality have changed since Waaler first produced his data in 1984; thus, the Norwegian data may not prove to be an adequate benchmark or standard by which to make comparisons across countries. The surfaces also require a large amount of data to adequately model height-weight associations with mortality¹⁰—a limitation that is readily apparent with the development of the CRELES surface based on a relatively small number of individuals as compared with the Waaler-type surface.

5.1.2 Case of Puerto Rico

A final piece of evidence comes from the case of Puerto Rico using season of birth to examine early life exposures and older adult health. As described previously, there were consistent and widespread efforts during the late 1920s through the early 1940s

⁸ See Monteiro, Conde, et al. (2004) and Monteiro, Moura, et al. (2004).

⁹ Waaler (1984).

¹⁰ Fogel (2004) and Waaler (1984).

Table 5.4 Comparison of childhood illnesses and SES by cohorts

Variable	All	60–74 years old	75+	p-value
Highly prevalent illnesses in early twentieth century Puerto Rico				
Typhoid fever	3.2	2.5	4.5	0.000
Malaria	5.4	2.8	9.7	0.000
Tuberculosis	0.9	0.5	1.6	0.007
Other serious illnesses				
Rheumatic fever	3.1	2.0	4.8	0.000
Dengue fever	10.1	9.4	11.3	0.001
Chronic bronchitis	4.6	4.7	4.5	0.409
Pneumonia	8.5	8.9	7.9	0.274
Asthma	9.8	10.5	8.6	0.033
Smallpox	21.5	21.4	21.6	0.790
More common illnesses				
Measles	64.8	66.4	61.9	0.058
Chicken pox	73.6	75.3	70.6	0.001
Mumps	54.5	58.4	48.0	0.000
Poor SES				
Mother-no schooling	50.7	41.6	65.9	0.000
Father-no schooling	45.6	36.9	60.1	0.000

Source: PREHCO imputed data, first wave

Notes: Shown are unweighted data. The sample sizes were: all (4,148), 60–74 years old born in the late 1920s through early 1940s (2,603) and the 75 years and older born at the beginning of the twentieth century (1,545). Numbers in the table are expressed as percent and percentages are rounded to the nearest tenth

to improve environmental conditions in Puerto Rico, and these efforts reduced exposure to infectious diseases.¹¹ At the turn of the century, IMR rates were high in Puerto Rico (over 200 per 1,000—see Fig. 1.9); by 1930 overall IMR was still high (slightly above 120 per 1,000) but had been reduced considerably resulting in increasing numbers of survivors of poor early life conditions. Improving environmental conditions due to public health interventions of the early twentieth century produced differences in exposure to disease and it is plausible to believe that the determinants of older adult health based on these early life conditions are different.

Table 5.4 illustrates clear differences between cohorts in exposures to childhood diseases from the PREHCO survey of older adults born in the early to mid twentieth century. Adults born at the beginning of the twentieth century showed a higher prevalence of serious childhood diseases—typhoid fever, malaria, tuberculosis, rheumatic fever, and dengue fever. These children’s parents were likely to have had no formal schooling. In contrast, those born in the 1920s through the early 1940s exhibit a higher prevalence of childhood asthma, chicken pox, and mumps; their parents tended to have more education.

¹¹ Clark (1930).

The results shown in the previous chapter for Puerto Rico suggest a higher likelihood of reporting adult heart disease and diabetes for those born in the lean season during the late 1920s and early 1940s in Puerto Rico. These results fit into a literature describing the importance of early life nutrition and infectious diseases and older adult health¹² and support the idea that the results reflect an increasing pool of survivors of poor early life conditions. Yet, when examining adult mortality among this same cohort, no significant seasonal effects were noted for adult mortality, although there were strong associations between adult heart disease, diabetes, and mortality and between low height and mortality (Table 5.5).¹³

In contrast, for the 75 years and older group, there were no significant seasonal effects on heart disease or diabetes either overall or by residence during childhood (results not shown). However, there were significant seasonal effects on adult mortality and these effects differed based on rural or urban residence during childhood. Overall, the odds of dying were about 1.50–1.61 times higher for those born in the first and third quarter (partial early and late exposure) as compared with the spring quarter (results not shown). For those respondents who lived in rural areas during childhood, the odds of dying were about 1.61–1.68 times higher for those born in the first and third quarter (partial early and late exposure to nutritional deficits and peak season of infectious diseases) as compared with the second quarter (no exposure), even after controlling for other early life and adult factors (Table 5.5). Models including interaction effects showed no significant effects between season of birth and childhood illnesses such as malaria and tuberculosis (results not shown).

Figure 5.3 shows that the predicted probabilities of dying for respondents 75 years and older were highest among those born in the third (partial early exposure) and first (partial late exposure) quarters, especially for males. For women (men) born at the end of the harvest, the probability of dying was 0.17 (0.25) compared with 0.25 (0.35) for those born during the peak season of infectious diseases—an increase of 47 % for women and 40 % for men. For respondents with other poor childhood conditions (e.g., low height, poor childhood health, and childhood SES), however, the probability of death decreased. Women (men) born at the end of the harvest, the probability of dying was 0.08 (0.13) as compared with 0.13(0.20) for those born during the peak of infectious diseases—an increase of 63 % for women and 54 % for men. In contrast, for respondents who also had poor adult conditions (e.g., heart disease, diabetes, obesity, does not exercise, and used to smoke), the probability of dying increased dramatically in several instances: For women (men) born at the end of the harvest, the probability of dying was 0.20 (0.29) compared with 0.30 (0.41) for women (men) born during the peak of infectious diseases—an increase of 50 % for women and 41 % for men.

¹² Barker (1998) and Doblhammer (2004).

¹³ McEniry (2009b).

Table 5.5 Likelihood of dying for PREHCO respondents who lived in rural areas as children

	75+ years old OR (95 % CI)	60–74 years old OR (95 % CI)
Childhood		
Poor childhood health	0.70 (0.43–1.14)	0.52 (0.32–0.82)
Rheumatic fever		1.62 (0.43–6.12)
Low height	1.59 (1.10–2.31)	1.65 (1.03–2.66)
Poor childhood SES	0.80 (0.45–1.39)	1.41 (0.85–2.35)
No exposure (ref) ^a	1.00	1.00
Partial early exposure	1.61 (1.04–2.71)	1.03 (0.63–1.69)
Full exposure	1.54 (0.88–2.68)	0.87 (0.52–1.43)
Partial late exposure	1.68 (1.04–2.71)	0.83 (0.48–1.44)
Adulthood		
Heart disease	1.18 (0.79–1.76)	1.71 (1.11–2.62)
Diabetes	1.83 (1.28–2.63)	2.32 (1.56–3.44)
Underweight	1.51 (0.66–3.45)	1.23 (0.43–3.51)
Normal weight (ref)	1.00	1.00
Overweight	0.58 (0.40–0.83)	0.34 (0.21–0.58)
Obese	0.55 (0.34–0.91)	0.36 (0.22–0.59)
Never smoked (ref)	1.00	1.00
Smoked in past	1.34 (0.91–1.97)	1.57 (1.03–2.39)
Smoke now	2.56 (1.25–5.23)	1.38 (0.77–2.47)
Rigorous exercise	0.52 (0.34–0.81)	0.55 (0.36–0.83)
Log Likelihood ^b	(–510, –502)	(–464, –437)
Chi-square ^b	138–179 (18)	92–112 (19)
Number of observations ^b	892–918	1,485–1,497

Source: McEniry (2009b, 2011c). Models above controlled for gender and age

Notes: 60–74 years old born during the late 1920s through early 1940s; 75+ born at the beginning of the twentieth century

^aSeason of birth and exposure: No exposure (second quarter), partial early (third quarter), full (fourth quarter), partial late (first quarter)

^bThe multiple imputation procedure required us to work with five alternative completed data sets. It was not clear how to calculate conventional statistics, such as chi square, all of which are functions of data-specific log-likelihood functions. As a partial resolution, the range of values for the chosen statistics obtained after estimating models for each of the imputed data sets is presented in this table. Chi-square shows degrees of freedom in parentheses

5.1.2.1 Discussion

The results for the two cohorts (one born prior to the late 1920s and the other born during the late 1920s through the early 1940s) in Puerto Rico may reflect different risks at birth resulting in different pathways to mortality. Both infectious diseases and poor nutrition appear to be important factors affecting the health of PREHCO respondents during their early life, although there were important differences in the timing of effects based on season. Much like individuals in other highly infectious

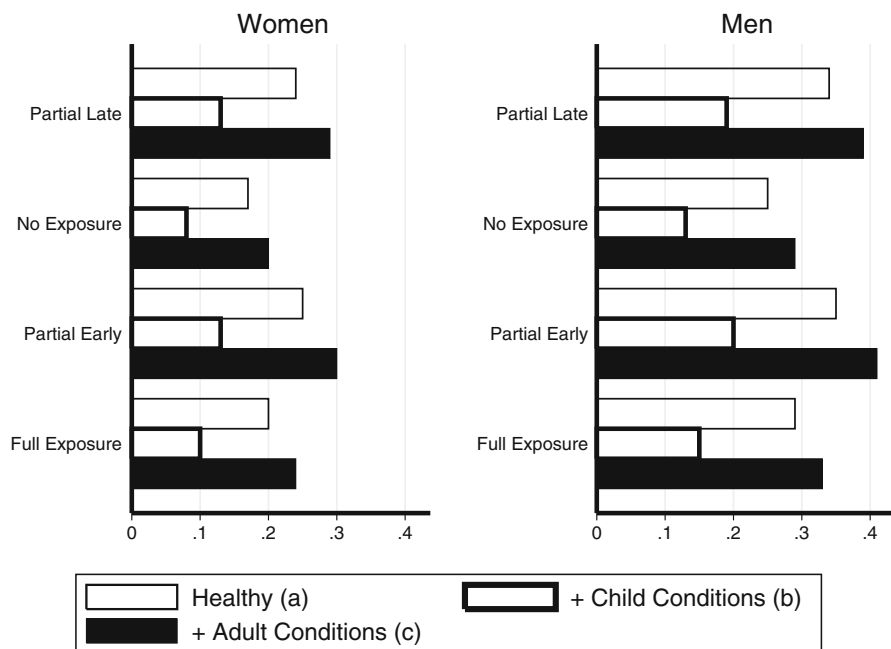


Fig. 5.3 Predicted probability of death by exposure, 75 years and older. *Note:* The predicted probabilities for dying for those respondents who were born prior to the 1920s corresponding to the (a) typical healthy respondent (between the ages of 80 and 84 with 7 years of education and who had never smoked but exercises), (b) plus poor childhood conditions (poor child health, poor child socioeconomic status and low height), (c) plus adult obesity, heart disease, diabetes, previous smoker, and does not exercise: Women: end of the harvest (second quarter)-no exposure (0.17, 0.08, 0.20), end of lean period (fourth quarter)-full exposure (0.20, 0.10, 0.24), peak infectious disease season (first and third quarters) (0.24–0.25, 0.13, 0.29–0.30). Men: end of the harvest-no exposure (0.25, 0.13, 0.29), end of lean period-full exposure (0.29, 0.15, 0.33), peak infectious disease season-partial exposure (0.34–0.35, 0.19–0.20, 0.39–0.41) (*Source:* PREHCO (2007), imputed ($n = 1,545$)). Just those 75+ years old born prior to the 1920s. See McEniry (2009b, 2011c)

disease environments,¹⁴ older Puerto Rican respondents (75+ years, born prior to the late 1920s) were exposed to a more severe mortality regime at birth with higher infectious disease loads. There appeared to be strong seasonal effects on adult mortality in the first and third quarters for those 75 years and older, raising the possibility of the importance of infectious diseases. In the first quarter of the year, temperatures were milder but there was increased risk of exposure to respiratory ailments and the third quarter brought the hurricane season (August–September) with higher exposure to infectious diseases such as dysentery, diarrhea, and malaria¹⁵ for newborns. However, there were no significant interaction effects between season of birth and malaria, tuberculosis or other respiratory childhood illnesses (results not shown). Therefore, it may also be that the effects of poor nutrition and infectious diseases are confounded with the effects of maternal infections acquired *in utero*.

¹⁴ Davey Smith and Lynch (2004).

¹⁵ Monteverde, Noronha, and Palloni (2009).

Because Puerto Rico is a tropical country without the large variation in exposure to sun like northern countries, it is unlikely these results reflect a seasonal effect of deficiency of Vitamin D which has been shown to affect later adult health in other studies.¹⁶ However, there is a tendency for tuberculosis to show strong seasonal patterns with reactivation of the infection peaking during hot summer periods.¹⁷ It may also be the mother's exposure to infection from malaria, hookworm or tuberculosis during the third quarter which affected the unborn baby. There was a high incidence of tuberculosis in urban areas in Puerto Rico due to overcrowding and poor sanitation prior to the 1920s¹⁸ and evidence that the disease struck Puerto Rican adults in their prime with higher mortality rates for women.¹⁹ This, in addition to evidence from other studies supporting a strong seasonal pattern for tuberculosis,²⁰ suggests it may be that the urban born respondents were exposed to tuberculosis infection through their mother either *in utero* or in infancy through breastfeeding.

In contrast, younger respondents (60–74 years) were born during a period of significant mortality decline due to public health interventions including medical technology. As environmental conditions improved, the strong effects of infectious diseases decreased, although seasonal nutritional deficiencies remained. These conditions could partially explain the effects of season of birth on adult heart disease and diabetes in this cohort, and may reflect nutritional effects on adult health. Data for mortality due to heart disease and diabetes were not available; it may be that the all-cause mortality used in the analyses is too broad to show effects based on season of birth.

The 75 years and older group experienced a more severe mortality regime. Mortality rates were higher and fewer infants with low birth weight and fewer children with severe illnesses survived. However, as noted in Table 5.4, this group may have been exposed to more serious illnesses in childhood such as typhoid, malaria, and tuberculosis and this may partially explain the seasonal pattern and suggest the importance of infectious diseases experienced in early life. There is evidence that such serious diseases in childhood have long-term consequences.²¹ However, if early life hypotheses regarding *in utero* exposures have merit and many did not survive *in utero* and early infancy exposures, those who did may be more robust. The younger cohort of 60–74 years olds born in the late 1920s through 1940s includes more individuals who had a higher probability of surviving poor early conditions *in utero*, infancy or childhood. They may have a greater risk of

¹⁶ Tare, Parkington, and Morley (2006).

¹⁷ Thorpe, Frieden, Laserson, Wells, and Khatri (2004).

¹⁸ Clark (1930).

¹⁹ Brenes (2008).

²⁰ Gavrillov and Gavrillova (2005).

²¹ Elo and Preston (1992).

adult heart disease and diabetes due to these circumstances in addition to experiencing the long term consequences of exposure to major infectious diseases.

The results for the 75+ respondents merit further examination to better understand the role of exposure in early life to infectious diseases that impact, for example, adult chronic respiratory conditions and mortality. In terms of addressing the determinants of adult chronic conditions such as heart disease and diabetes, the results from the 60–74 year old respondents provide evidence that poor early life exposures *in utero* or early infancy increase the likelihood of adult heart disease and diabetes which, in turn, strongly impact mortality. Thus, the results provide evidence in support of hypotheses regarding the importance of nutrition²² and infectious diseases in early life²³.

The strong association between low height (first quartile of height) and mortality deserves attention. Low height could reflect stunting and poor nutrition as well as the impact of having to fight infectious diseases in childhood. Analyses using the cross-national data generally do not show low height to be significantly associated with adult heart disease or diabetes. A strong association between height and mortality but not between height and adult heart disease or diabetes may reflect the underestimation of these chronic conditions. However, to some degree the lack of association between height and chronic conditions is not surprising because, as noted in Chap. 1 (Figs. 1.2 and 1.3), stunting as reflected in low height may not be directly associated with adult heart disease or diabetes but may be directly associated with obesity which then can lead to heart disease and diabetes and later mortality. The strong association between low height and mortality noted in Table 5.5 may thus reflect other mechanisms besides nutrition operating in childhood related to the consequences of poor childhood SES.

Admittedly, the study has all the limitations noted in the discussion on measures in Chap. 2 and from selection bias of the survivors. In addition, it is not known the degree to which the case of Puerto Rico is unique or representative of other similar populations. Only replication of the study in similar countries will help determine if the results can be generalized to Latin American and Caribbean populations such as Costa Rica, Chile, and Mexico and to Asian and African populations which experienced similar mortality decline during the late 1920s and early 1940s.

The two relevant pathways to adult mortality originating in early life are relevant in the developing world with the increasing prevalence of heart disease and diabetes among aging populations, in addition to a continued risk of tuberculosis and malaria in some undernourished communities.²⁴ The possible pathway to adult mortality

²² Barker (1998).

²³ Finch and Crimmins (2004), Painter et al. (2006), Jana, Vasishta, Jindal, Khunnu, and Ghosh (1994), Guyatt and Snow (2004), Moore, Cole, Collinson, Poskitt, McGregor, and Prentice (1999), Rayco-Solon, Moore, Fulford, and Prentice (2004), and Ravelli et al. (1998).

²⁴ Murray and López (1996).

through adult heart disease and diabetes and the strong associations between these chronic conditions and mortality for those born during the late 1920s–early 1940s points to the merit of the idea of higher mortality risk for these unique cohorts.

5.2 Health Disparities

Unequal societies tend to have larger health inequalities.²⁵ For the most part, those exposed to poor early life conditions (e.g., poor nutrition, harsh living environments, and infectious diseases such as diarrhea, dysentery, hookworm, and malaria) in developing countries during the early twentieth century but survived those conditions due to public health interventions including medical technology were poor and lived in very unequal societies. Poor early life conditions may also have produced poor childhood health which may have limited an individual's ability over the life course to obtain a higher level of education or income.²⁶ Thus, higher survivorship in early life did not necessarily correlate with improved standards of living during childhood or throughout the life course because of, for example, economic conditions or lack of social mobility. If this were the case, the ramifications of what occurred in the 1930s–1960s would be steeper health inequalities, especially among adults born in the tip of the iceberg countries. If early life conditions are indeed important to older adult health, then a comparison of health status by educational or income level should show increasingly stronger and sharper health disparities moving from very early regimes to very late regimes, controlling for confounding factors especially national income level (Hypothesis 3).

An important caveat to this hypothesis is that this assumes to some degree the pattern observed in the developed world of poorer health being associated with lower levels of education and income. However, puzzling and unexpected patterns of health disparities have appeared in some developing countries,²⁷ so health disparities in the developing world may be different than what has been observed in the developed world. In fact, negative health gradients (i.e., higher prevalence of chronic conditions at lower educational levels) are not always as expected, as illustrated by the prevalence of obesity which was higher among higher educational levels in pre-health transition, low-income countries.²⁸ Thus, the timing of the demographic transition may produce just the opposite of expected patterns of health disparities. In terms of the conjecture for the unique cohorts of the 1930s–1960s, the observation of unexpected health gradients in some countries may suggest they have yet to experience significant mortality decline or to have made a health

²⁵ Wilkinson (1996).

²⁶ For more discussion about health selection see Lundberg (1991) and Palloni, Milesi, White, and Turner (2009).

²⁷ Rosero-Bixby and Dow (2009).

²⁸ Monteiro, Conde, Lu, and Popkin (2004).

transition. This was precisely the case for countries in the very late regimes (severe mortality regimes) of Indonesia, China, and Bangladesh prior to 1945.

If the conjecture regarding the unique cohort of the 1930s–1960s has merit the following is expected:

1. A general pattern of steeper health disparities is expected in the mid-paced (tip of the iceberg) to late regimes across all health outcomes controlling for major confounding factors such as national income.
2. Reversal of the health gradient in the very late regimes where adults with higher levels of educational attainment and/or income show a higher prevalence of chronic conditions.²⁹

In terms of the first expectation, a relevant comparison is older adult health across mortality regimes within similar national income groups to help control for disparities in standard of living. A comparison across mortality regimes within middle-income countries should show steeper health disparities in the tip of the iceberg countries. There may also be differences within mortality regimes of particular note: steeper health disparities in early regimes such as Argentina and Uruguay compared with Cuba which had a very different approach to health care; steeper health disparities within mid-paced (tip of the iceberg) regimes such as Puerto Rico and Chile compared to other mid-paced regimes with either quality primary health care systems (Costa Rica) or large economic improvements in the twentieth century (Taiwan); steeper health disparities within late to very late regimes such as Bangladesh, Indonesia, and India compared to China which implemented a different institutional framework for health care.

International comparisons of health disparities are difficult for a variety of reasons. Other mechanisms such as improved health care systems or changes in health behavior affect health. Those with better socioeconomic conditions may live longer and thus, primarily through longevity, may have a higher prevalence of chronic conditions or frailty. Such might be the case in higher income countries where life has been prolonged by advances in medical technology. Contrasts between higher and lower educational or income levels in these cases may blur the effects of poor early life conditions.

The measurement of income is not standardized across many countries, thus making legitimate comparisons more difficult. Even with years of education, countries may have different thresholds for distinguishing those with higher or lower socioeconomic standing. Furthermore, comparison by education between high-income countries and low- to middle-income countries is difficult because of the large differences in the distribution of years of education. There is unknown measurement error of health outcomes across countries even when survey questions are identical. A comparison of health status by educational and income level is thus not only a very indirect test but one fraught with the difficulty of controlling for confounding factors.

²⁹ Monteiro, Conde, et al. (2004) and Monteiro, Moura, et al. (2004).

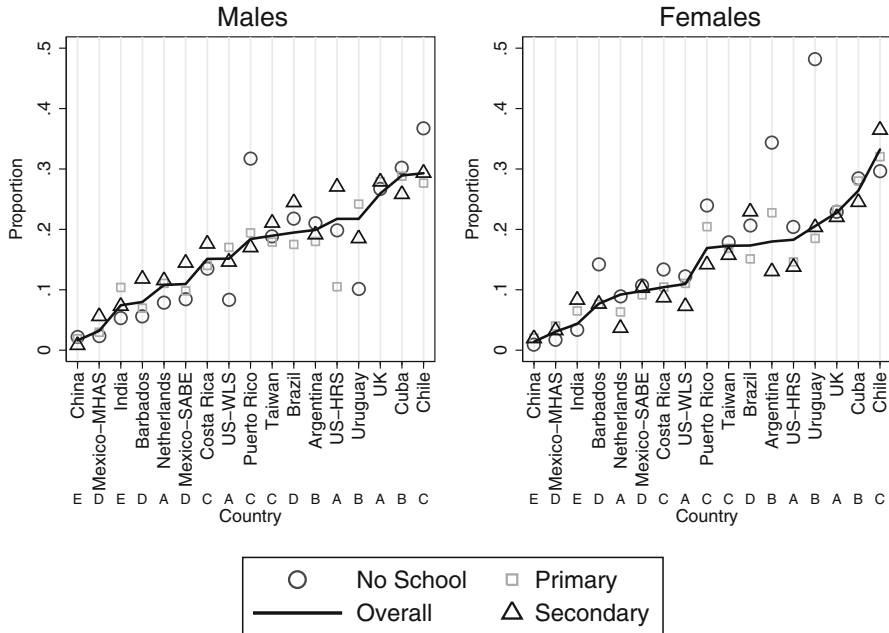


Fig. 5.4 Prevalence of heart disease by educational level. *Notes:* India prevalence is based on self-report data only and not using symptom questions. UK refers to the England-ELSA survey. For developing countries, low = no school, mid = primary school, and high = secondary school or more; for developed countries, low = 0–12 years of education, mid = 13–15 years of education and high = 16 or more years of education. Mortality regimes and corresponding countries: A. Very Early: England, Netherlands, US-HRS, US-WLS; B. Early: Argentina, Cuba, Uruguay; C. Mid: Chile, Costa Rica, Puerto Rico, Taiwan; D. Late: Barbados, Brazil, Mexico-MHAS, Mexico-SABE; E. Very Late: China-CHNS, India (*Source:* McEniry (2010a), imputed, weighted where relevant. Respondents include adults who were born in the late 1920s to early 1940s)

Several analyses were carried out to examine basic patterns of disparities followed by modeling health disparities.³⁰ For respondents born in the late 1920s and early 1940s, the pattern of self-reported heart disease by educational level is mixed both within and across countries (Fig. 5.4). For the most part (except in India, Brazil, and Chile), the gradient is in the expected direction for females—higher prevalence of heart disease at lower educational levels in earlier regimes—but not in the expected direction for males for whom in many countries (Barbados, Netherlands, Mexico, Costa Rica, Taiwan, Brazil, and the US) there is a reversal of expected patterns—those with more education show a higher prevalence of heart disease. With the exception of Chile, the countries with the highest proportion reporting heart disease tend to be from the very early or early mortality regimes. The countries with the lowest proportion reporting heart disease tend to bathe low-income countries

³⁰ McEniry (2010a).

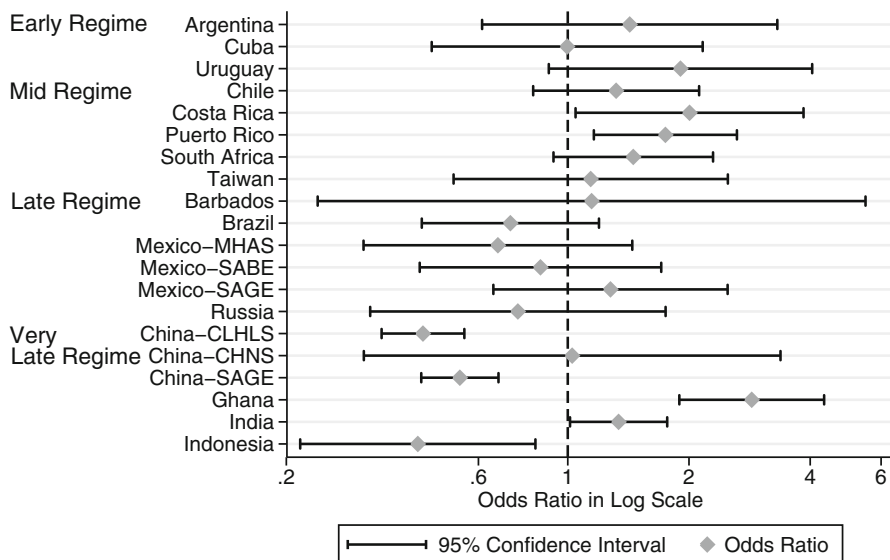


Fig. 5.5 Likelihood of adult heart disease for those with no education. *Notes:* Odds ratios are shown with corresponding 95 % confidence intervals. Reference group is those with at least a secondary school education. Models controlled for age and gender. Older adults born in the late 1920s and early 1940s and are shown in order of mortality regime (*Source:* McEniry (2010a), imputed, weighted where relevant. Respondents are adults born in the late 1920s to early 1940s)

(China, Mexico, and India) followed by a few high income countries (Barbados and the Netherlands). Within countries, the greatest differences in heart disease between the lowest and highest level of education tend to be seen in Argentina, Uruguay, and Puerto Rico.

There are large differences in the distribution of education across countries.³¹ Not surprisingly, earlier regimes such as the US, the Netherlands, and England show a smaller percent of older adults with no formal education compared to the very late regimes of China, India, Bangladesh, and Indonesia; this is especially true for women. Figure 5.5 shows strong disparities with large variations in terms of reporting heart disease for those individuals with no formal education compared with those who attended secondary school and above. Across countries, the odds of reporting heart disease for those with no formal education are anywhere from less than 1 to slightly less than 4 times higher than for those with the highest level of education (secondary and above); however, confidence intervals suggest there is too much variation in the data to discern a clear pattern. A similar pattern emerges when using household per capita income (not shown).³²

³¹ McEniry (2010a).

³² McEniry (2010a).

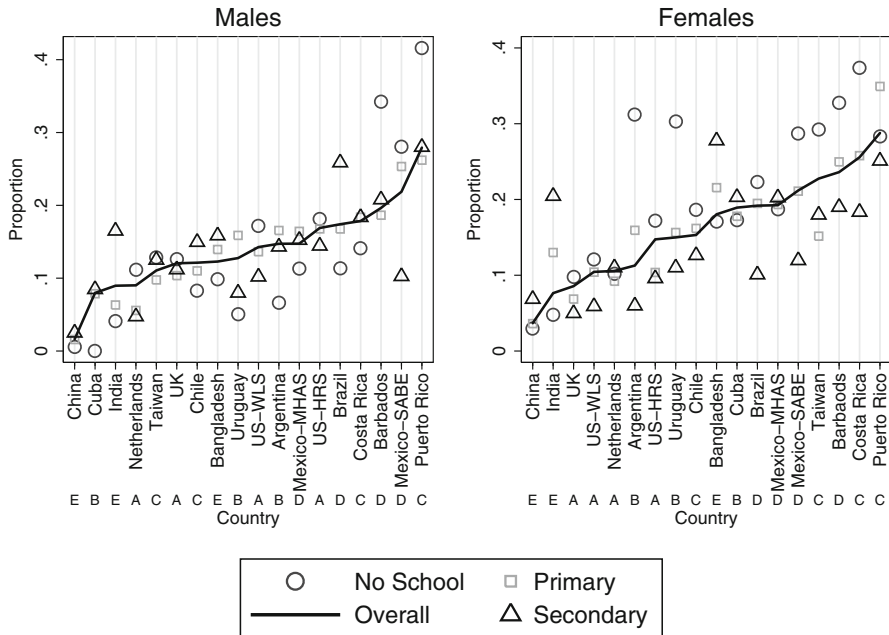


Fig. 5.6 Prevalence of diabetes by educational level. *Notes:* SAGE data are not presented here with the exception of India which are self-report data only. UK refers to the England-ELSA survey. For developing countries low = no school, mid = primary and high = secondary and more; for developed countries low = 0–12 years of education, mid = 13–15 years of education and high = 16 or more years of education. Mortality regimes and corresponding countries: A. Very Early: England, Netherlands, US-HRS, US-WLS; B. Early: Argentina, Cuba, Uruguay; C. Mid: Chile, Costa Rica, Puerto Rico, Taiwan; D. Late: Barbados, Brazil, Mexico-MHAS, Mexico-SABE; E. Very Late: Bangladesh, China-CHNS, India (*Source:* McEniry (2010a), imputed, weighted where relevant. Respondents include adults who were born in the late 1920s to early 1940s)

For diabetes, a much clearer pattern emerges (Fig. 5.6). For the most part, the gradient is in the expected direction for females—higher prevalence of diabetes at lower educational levels in earlier regimes, with the exception of the very late regimes of China, India, and Bangladesh where, as expected,³³ there is a clear reversal of the gradient; for males, the reverse of what is expected occurs in Cuba, Chile, and Brazil. Overall, large educational disparities appear in Barbados and Mexico for males and in Argentina, Uruguay, Taiwan, Barbados, Mexico, and Costa Rica for females.

Figure 5.7 shows the odds ratios and corresponding 95 % confidence intervals for diabetes using models adjusted for age and gender. The observed patterns present a strong case for stronger health disparities in the early to mid-mortality regimes. Although very strong health inequalities appear between the lowest and

³³ Monteiro, Conde, et al. (2004) and Monteiro, Moura, et al. (2004).

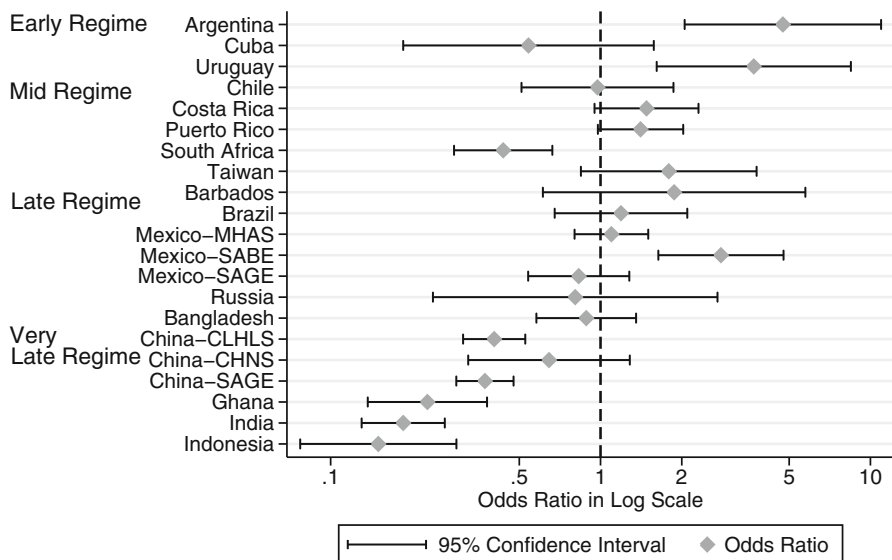


Fig. 5.7 Likelihood of adult diabetes for those with no education. *Notes:* Odds ratios are shown with corresponding 95 % confidence intervals. Reference group is those with at least a secondary school education. Models controlled for age and gender. Older adults born in the late 1920s and early 1940s and shown in order of mortality regime (*Source:* McEniry, 2010a)

highest level of education with large variation between countries, sharper differences exist in countries such as Argentina and Uruguay followed by the mid- to late-mortality regimes of Mexico, Barbados, Taiwan, Costa Rica, and Puerto Rico. These patterns are repeated when using household per capita income (not shown).³⁴

5.2.1 Discussion

Overall, the findings show strong health inequalities across countries and large within-country variation, especially for diabetes. However, those countries hypothesized to have produced a larger pool of survivors of poor early life conditions (the tip of the iceberg countries of Chile, Costa Rica, and Puerto Rico) do not, as a group, show steeper health gradients for heart disease and diabetes when compared with other middle-income countries. A reversal of health disparities occurs in the lower income countries where lower education reduces the likelihood of reporting heart disease and diabetes. Surprisingly, there is a certain

³⁴ McEniry (2010a).

amount of similarity in health disparities across almost all countries whether using education, income or wealth, which is in contrast to what others have noted.³⁵

The findings on health disparities do not suggest a total rejection of the conjecture regarding these unique cohorts of the 1930s–1940s. If social mobility explains the results, the findings may reject the idea of health selection for the unique cohorts of the 1930s–1960s—i.e. that the increasing number of survivors in the tip of the iceberg countries would lead to a larger pool of adults with lower adult SES. On the other hand, the findings do not negate the importance of early life determinants for such adult chronic conditions as heart disease and diabetes.

Underestimation of adult heart disease and diabetes may partially explain the results. As noted in earlier chapters, underestimation of adult heart disease and diabetes may produce similar direction of associations but the magnitude may be very different. If this is the case, then underestimation may indeed be problematic in properly discerning differences in cross-national patterns based on the magnitude of the coefficients.

There are several other possible explanations for the weak evidence regarding sharper health disparities among the unique cohorts of the 1930s–1960s. The health gradient in low- and middle-income countries is complex and conflicting results have emerged.³⁶ In spite of the high levels of inequality experienced in these mid-mortality regimes during the twentieth century, it may be there was sufficient social mobility among those born poor to offset steeper health gradients at older ages. This could have occurred in Puerto Rico where those born poor in urban areas fared better as adults than those born poor in rural areas where conditions were more precarious during the 1930s–1940s. It also could have occurred because migration to the US may have sufficiently improved economic conditions regardless of poverty status during childhood (in the case of Puerto Rico or Mexico). It could also be due to remittances from those who migrated to the US (in the case of Mexico). Finally, it may have occurred due to dramatic economic growth in the 1950s which set countries such as Taiwan on a path to much higher standards of living.

Older adults in the tip of the iceberg countries are not homogenous in terms of the mortality regime they experienced. In the case of Puerto Rico, although in general mortality was declining during the late 1920s through early 1940s, there were regions within the country where high infant and child mortality still prevailed, which may produce results that are hard to interpret. Selection may be an issue. At older ages, the health gradient begins to disappear because of selection effects.³⁷ It may be that SES differentials in the mid-mortality regimes have weakened for this age group due to mortality selection.

The nature of the data and the measures used may contribute to uncertainty. If it were possible to identify a larger pool of survivors of early conditions, then it would

³⁵ Smith (2005).

³⁶ Rosero-Bixby and Dow (2009).

³⁷ Smith (2005).

be possible to be more precise about comparisons, but the sample size for those born during the late 1920s and early 1940s in some countries is small. The SABE survey was conducted in major cities rather than the entire country, so it is not nationally representative. It may be that the smaller sample size in some countries is responsible for some of the very large variation observed. Analyses with income were conducted (results not shown), but income is also notoriously error prone with large proportions of missing data. The measure for wealth used is in a preliminary stage of being tested and developed. It may not be possible to capture the complexity of the health gradient with these simple measures. There may be specific national circumstances among countries in the mid-mortality regimes; this may be more of a country by country story versus a pattern across countries.

It also may be, of course, that the premise on which the conjecture is based is not valid. That is, it may be that the relative importance of early life conditions on older adult health is smaller than anticipated. It may be that health selection in early life plays a smaller, although perhaps non-trivial, role for the unique cohorts of the 1930s–1960s,³⁸ and that the analyses of health disparities presented here were not able to adequately capture this.

5.3 Conclusions

Waalder-type surfaces present suggestive evidence that older adults in low-income countries will have higher mortality risks as they age and as the prevalence of obesity increases. Yet, there is weak evidence for the conjecture that adults born in the tip of the iceberg countries are in any way different in terms of mortality risk due to their survival of poor early life conditions. There are strong health disparities across countries especially for diabetes and a reversal of patterns in health disparities in some poorer countries. However, there is little evidence to suggest the existence of sharper health disparities in the tip of the iceberg countries. Several factors may be in play: Actual mortality data were available in only a handful of countries; comparing mortality regimes within the same economic level would have required a larger sample of countries; and the difficulties of fully addressing confounding factors for health inequalities (for example, social mobility) when measures of such factors in surveys of older adults are often not present.

³⁸ Palloni, Milesi, White, and Turner (2009).

The topic of health determinants for older adults in the developing world, in particular early life determinants, is one of considerable interest and has been recognized as important for public health.¹ The preceding chapters set out with the ambitious task of examining a contrarian and therefore somewhat controversial conjecture regarding the unique cohorts born in the 1930s–1960s that experienced rapid improvements in life expectancy primarily because of public health interventions including medical technology but mostly in the context of stagnant economic conditions. This occurred primarily in low- and middle-income countries. If exposure to poor nutrition and infectious diseases is an important predictor of health at older ages, these unique cohorts may be more susceptible to disease as they age. Of particular interest are those born in the tip of the iceberg countries in the late 1920s through the early 1940s who have already reached the age of at least 60 years. They now live in the areas of the world which are projected to be part of the rising tide of older adults with chronic conditions such as heart disease and diabetes² which, in some instances, may originate in early life.³ These cohorts may shed light on the merit of the conjecture and what lies ahead.

Population surveys of individuals and households from 20 low-, middle-, and high-income countries were compiled and harmonized resulting in data on over 147,000 older adults. Countries were classified into mortality regimes using historical data on life expectancy, infant mortality and GDP per capita. Older adult health was examined across mortality regimes across countries and within groups of countries with similar levels of national income, with a particular focus on tip of the iceberg countries and on adult heart disease and diabetes. An in-depth examination of adult heart disease and diabetes in Puerto Rico (a tip of the iceberg country) was also conducted because rich survey and historical data illustrating conditions in the early twentieth century were available.

¹ Kinsella and He (2009), National Institute on Aging and US Department of State (2007), and National Research Council (2001).

² Murray and Lopez (1996).

³ Barker (1998).

6.1 Factors Across the Life Course and the Wellbeing of Older Adults

The aging process is shaped throughout the entire life course.⁴ The preceding chapters suggest an important way this may be occurring in the unique cohorts of the 1930s–1960s in poorer countries who may have been unduly marked by early life exposure to poor nutrition and infectious diseases. Cross-national comparisons of adult heart disease and diabetes suggest the importance of exposure in early life to poor nutrition and infectious disease, especially for older adults born in the tip of the iceberg countries. On the other hand, the results for mortality and health disparities are less profound:

1. The health profile of obese older adults and those with diabetes in the tip of the iceberg countries appears worse than in developed countries, even after removing the potential effects of current disparities in standards of living (supports Hypothesis #1).
2. Health profiles of those with adult diabetes and heart disease in the tip of the iceberg countries are closely associated with past individual history including nutritional status, early experience with illnesses, and deprivations experienced during early childhood. This occurs even after controlling for adult lifestyle including adult nutritional environment (supports Hypothesis #2).
3. There is insufficient evidence to suggest mortality risk among older adults in the tip of the iceberg countries exceeds what one would expect (does not support Hypothesis #3).
4. Significant socioeconomic disparities in health status exist, but there is little evidence they are worse in the tip of the iceberg countries (does not support Hypothesis #4).

6.1.1 Morbidity: Adult Heart Disease and Diabetes

In regards to Hypotheses #1 and #2, the results suggest that the aging process will be different in the low- and middle-income countries compared with high-income countries in terms of the prevalence of some chronic conditions such as diabetes and obesity and their determinants. Differences in environmental exposures and cohorts may change the magnitude of the relationships between early life conditions and older adult health in the developing world.

One piece of cross-national evidence in support of the idea that the tip of the iceberg cohorts of the 1930s–1960s are more susceptible to the effects of poor early life conditions at older ages lies with diabetes and obesity. The prevalence of diabetes has never been higher in some of the developing countries, including Puerto Rican older adults on the island. In the Latin American and Caribbean

⁴ Kuh and Ben-Shlomo (2004).

regions, mortality due to diabetes has been increasing while mortality due to heart disease has been decreasing.⁵ A cross-national comparison across mortality regimes showed there is a much higher prevalence of adult diabetes and obesity in the tip of the iceberg countries of Puerto Rico, Costa Rica, Chile, Mexico, Brazil, and Barbados compared to the very early regimes of the US and Netherlands and the very late regimes of China, India, Ghana, and Bangladesh. When comparing countries with similar levels of national income, the prevalence of diabetes is higher in the mid- to late-regimes than in other middle-income countries (Argentina and Uruguay). In Barbados, the prevalence of diabetes is higher than in the high-income countries (US, Netherlands, and England). Older adults born during the 1920s through the early 1940s, especially women, show a higher prevalence of diabetes and obesity, both of which are risk factors for adult heart disease, in spite of disparities in standards of living in such high income countries as Barbados and Taiwan and in countries with good primary health care systems such as Costa Rica.

The results for diabetes are conservative because self-report may result in an underestimation of these conditions may be problematic. However, if the prevalence of diabetes is much higher than depicted (see Fig. 4.2), that would only strengthen the association between low early life caloric intake and the prevalence of adult diabetes observed in the figure. Similar results for obesity—a more objective measure of health—also strengthen the case for diabetes. Thus, underestimation in this case does not obscure the potential importance of early life caloric intake for adult diabetes.

The story for adult heart disease is more complicated. The positive association between early life caloric intake and adult heart disease seen in Fig. 4.1 suggest the importance of composition of early life diet (i.e. a diet high in saturated fat) is important than for heart disease because countries with a high caloric intake have a higher prevalence of heart disease. At the same time, multivariate results showed a strong association between later heart disease and being born in a tip of the iceberg country, experiencing low caloric intake, and being born to parents of low SES; this indicates that older adults born in these settings may also be at risk. The association between a diet high in saturated fat and adult heart disease is not surprising; what is surprising is that there the evidence regarding its importance at early ages. Although models controlled for adult caloric intake, it may be that adult caloric intake could not be adequately captured and that it is confounded with early life caloric intake.

6.1.2 Mortality and Health Disparities

In contrast with morbidity, the evidence in support of Hypothesis #3 regarding the susceptibility of older adults born in the tip of the iceberg countries is weaker for mortality but with good reason. There are a smaller number of countries by which

⁵ Palloni, McEniry, Dávila, and García Gurucharri (2005).

to make adequate cross-national comparisons and in most all cases there is no cause-specific mortality data. In their absence, Waaler-type mortality surfaces are helpful descriptive tools to some degree. Mortality data for a few tip of the iceberg countries when compared against the developed world indicate that mortality risk may be higher than expected. However tantalizing these results, more examination awaits as mortality data are made available from surveys in low and middle income countries.

The expected relative risks depicted on the Waaler-type surfaces are related to the contrarian conjecture regarding the historical circumstances of the 1930s–1960s. With improvement in standards of living comes improvement in nutrition which then leads to taller individuals and better health.⁶ Using the surfaces, taller individuals have lower mortality risks even when their average weight is higher than shorter individuals. They are at an advantage as compared with their shorter counterparts. The surfaces thus suggest the importance of economic growth and improvement in standard of living in terms of mortality risk and provide an explanation for the potential negative consequences to health for those born in poor countries during the early to mid twentieth century. The surfaces are not prescriptive, but raise the question of the future impact on adult mortality when survival at young ages is improved primarily due to public health including medical innovation but with continued lack of economic growth or improvement in standard of living. Assuming that early life conditions are important to older adult mortality, will investment in maternal and child health alone, especially in the context of stagnant improvement in living conditions, be sufficient to improve adult mortality risk?

The examination of mortality in two different cohorts of older adults in Puerto Rico—one born at the turn of the century and the other born in the late 1920s through early 1940s—gives some support to the idea that differences exist between cohorts in regards to explaining mortality because of increasing survivorship of infants and children. Both cohorts were exposed to poor early life conditions, but the resulting pathways to mortality may be different. The older cohort born at the beginning of the twentieth century experienced a more severe mortality regime during which many did not survive childhood. This may have led to more robust survivors in terms of infectious diseases, but had consequences in terms of adult mortality. In contrast, the younger cohort had a higher probability of surviving poor early conditions whether *in utero*, infancy or childhood, and if early life hypotheses are to be believed⁷ these circumstances may have led to a higher prevalence of heart disease and diabetes which is then strongly associated with adult mortality.

The evidence for the merit of the conjecture is weak in regards to health disparities (Hypothesis #4). Keeping everything constant, social and economic disparities were not more salient in the tip of the iceberg countries where the contribution of past mortality decline associated with public health and the deployment of

⁶ Fogel (2004).

⁷ Barker (1998).

advanced medical technology is higher. Several analyses using different measures of socioeconomic disparities show that, while there are significant disparities in heart disease, no clear pattern of worsening disparities emerges when comparing the tip of the iceberg countries of Puerto Rico and Costa Rica with the earlier regimes of Argentina and Uruguay (also middle income countries). Diabetes presents a clearer pattern of differences, although a comparison across middle-income countries shows stronger health disparities in the early-mortality regimes of Argentina and Uruguay than it does in the mid- to late-mortality regimes of Puerto Rico, Costa Rica, Mexico, and Brazil suggesting a more complicated story.

Numerous factors may explain these results—prime among them is the difficulty of making cross-national comparisons of health disparities as noted earlier. It may be too early to observe the pattern of health disparities in older adults born prior to 1945 and the 1950s when mortality declined more dramatically. Social mobility may also be an important confounding factor and partially explain the results; the economic circumstances of individuals may have improved, making differences hard to discern. Social mobility may be important in tip of the iceberg countries such as Puerto Rico where migration to the mainland US may have improved individual socioeconomic circumstances.

6.2 Relative Importance of Early Life Conditions

The results presented support the idea that cohort effects of those born in the unique cohorts of the 1930s–1960s will play an important role in older adult health. At least for adult diabetes and heart disease, the results do not contradict the hypothesis that early life experiences for a particular cohort of individuals born in the 1930s–1960s drive adult morbidity differences. Results from other studies suggesting that early life conditions are not a major contributor in countries such as China⁸ do not take into consideration the demographic transition of the 1930s–1960s and the resulting changes in cohorts increasingly characterized by a higher probability of survival of poor early life conditions amidst continued economic stagnation.

The results provide insight into the questions raised in Chap. 1 regarding the relative importance of early life conditions for older adult health.

6.2.1 Critical Period Versus Accumulation of Effects

The results for cross national comparisons of adult heart disease and diabetes suggest the importance of early environment which includes not only nutrition but also exposure to infectious diseases given the synergy between the two.⁹ While

⁸ Popkin, Horton, and Kim (2001).

⁹ Scrimshaw (1968, 1997).

the exact mechanisms cannot be identified, the results do not contradict the possible importance of *in utero*, infancy or early childhood environment on later adult health.¹⁰ While the Barker hypothesis was highlighted in Chap. 1 because it explicitly describes how *in utero* circumstances can lead to adult heart disease and diabetes, it is not the only possible explanation. It may well be that multiple mechanisms operate in early life or that critical periods *in utero* combine with an accumulation of events across the life course to produce the observed results.

The strongest piece of country-specific evidence for the importance of *in utero*/early infancy factors in older adult heart disease and diabetes lies with the case of Puerto Rico—a tip of the iceberg country. Although it is not possible to identify exact mechanisms, broad associations between *in utero* and early life environment and older adult health can be identified which remain consistent after controlling for adult SES, years of education, obesity, smoking, illnesses, caloric intake, poor childhood health, poor childhood SES, and a family member with diabetes. The results from Puerto Rico therefore suggest that a critical period in early life (whether *in utero* or infancy) plays a stronger role than an accumulation of adverse events across the life course and point to the merit of hypotheses regarding the long-term consequences of early life environment on adult health.¹¹

6.2.2 Cohort vs Period Effects

The contrarian conjecture presented in the preceding chapters assumes that cohort effects stemming from exposure to poor early life nutrition and infectious disease in the 1930s–1960s are sufficiently strong to play an important role in older adult health such as heart disease and diabetes. The counterargument points to the strong period changes in adulthood to a diet with higher percentages of saturated fat, sodium and sugar, and a more sedentary lifestyle over the last several years across the world which plays an increasingly important role in determining health.¹² It is likely that the truth lies somewhere between. Nutritional insults *in utero* or early infancy combined with infectious diseases may place some older adults at risk; the consumption of unhealthy foods, being overweight, and smoking at older ages may compound and aggravate what began long ago.¹³ Survivors of poor early life nutrition and infectious disease conditions of the 1930s–1940s may not be able to adapt to rapid nutritional transitions later in life because of physiological changes in early life.

¹⁰ Barker (1998), Finch and Crimmins (2004), and Elo and Preston (1992).

¹¹ Bateson et al. (2004).

¹² Popkin (2006), Murray et al. (2012), Lim et al. (2012), and Basu, Yoffe, Hills, Lustig, and Wagner (2013).

¹³ Elo and Preston (1992), Barker (1995, 2001, 2005), Barker, Eriksson, Forsen, and Osmond (2002), and Barker, Forsén, Utela, Osmond, and Eriksson (2001).

While the results support the idea that a poor early environment reflecting poor nutrition and infectious diseases may play a role in adult diabetes and heart disease, there is less evidence that a rapid transition to enriched nutritional environments at older ages compounds the effects of being exposed to a poor early environment.¹⁴ The morbidity results for adult heart disease and diabetes presented in Chap. 4 in fact suggest neither a compounding nor confounding effect of adult lifestyle including nutrition, smoking, and health status. Thus, from the cross-national data there is little evidence to confirm the importance of either a compounding or confounding effect.

The results, in part, reflect inadequate precision in the use of a broad and crude measure such as country-specific caloric intake. However, although later life adult lifestyle did not appear to compound the effects of early life, the idea that poor early life conditions in some way combine with later adult changes in diet and a more sedentary lifestyle to produce poor older adult health still warrants attention because of the potential importance of period effects over the last 30–40 years in change of diet and sedentary lifestyle. Certainly this is true for the developed world, but increasingly, it is also true for the developing world where traditional diets are changing with already-noted adverse effects on health.¹⁵ However, it may be that not all segments of the population in the developing world have been affected by the consumption of diets higher in saturated fat, sodium and sugar. Individuals living closer to the US border may be more exposed to unhealthy diets from the US. Older adults in the unique cohorts born prior to 1945 may be less inclined to change their dietary habits. Traditional diets may also be less expensive than imported Western diets. It may be that these kinds of changes and exposure will be more important for those born in the unique cohorts during the 1950s–1960s because they will have been exposed to changing diets at earlier ages.

There also may be other important period exposures in recent times besides diet, nutrition, and sedentary lifestyle that affect health. Environmental hazards may be important in explaining health. Exposure to certain chemicals found in food but also furniture, paper, and articles of personal care may be a factor that partially explains the health patterns being observed in certain parts of the world.¹⁶

6.2.3 Mediation of Effects of Early Life

There is evidence from other studies that the effects of poor early life conditions may be mediated through interventions that improve childhood nutrition or family income during childhood, through educational and economic opportunity during young adulthood or through social factors.¹⁷ However, the results for morbidity

¹⁴ Barker (1995, 2001, 2005) and Barker et al. (2001, 2002).

¹⁵ De Schutter (2012).

¹⁶ For example, exposure to endocrine-disrupting chemicals may be a factor in explaining the recent epidemic in obesity in the developed world (WHO & UNEP, 2013).

¹⁷ Almond and Currie (2010).

presented in Chap. 5 give little indication that the effects of early life caloric intake are mediated by later adult conditions or circumstances. It may be that these results simply reflect the more effective types of interventions of nutritional or economic supplements are ones that occur at much younger ages (before the age of 5).¹⁸ It is also doubtful many older adults born in the late 1920s and early 1930s in the developing world had the opportunity for such interventions. Educational and economic opportunity was limited for many born during this period,¹⁹ and this may explain why education did not appear to mediate the effects of early life conditions. The question of inadequate measurement of early life and later life circumstances also may play a role in the results obtained.

There are other factors such as cultural differences in habit and diet in the developing world that may lead to better health outcomes in spite of the more pessimistic outlook of the contrarian conjecture regarding the cohort of older adults of the 1930s–1960s scarred by poor early life conditions. Even though traditional diets are changing around the world with already-noted adverse effects on health,²⁰ cultural differences in habit and diet can yet provide an antidote. This may be particularly true in parts of Asia where the traditional diet tends to be more balanced and moderate than in other parts of the world. Some communities maybe able to maintain such a diet and withstand the introduction of more processed foods higher in saturated fats, sodium and sugar, especially older adults. If this turns out to be the case, the pessimistic viewpoint of either the contrarian conjecture or the argument for strong period effects may not completely materialize in all settings, even if cohorts are heavily characterized by their increasing survival of poor early life conditions. Nevertheless, early reports about diet and risks for heart disease across all ages in China do not present a very optimistic indicator.²¹

6.3 Unintended Consequences and Policy Implications

The preceding chapters go beyond simply espousing the importance of associations between early life conditions and older adult health. They point to questions regarding policy, investments in health, and the long-term consequences of health interventions which will require further investigation and debate. Getting a better grasp on the determinants of the health status of older adults will help guide government and non-profit organizations as they prepare for the increased demand for health services and treatment of disease of an expanding elderly population.

The unique cohorts of the 1930s–1960s present one relevant example that could possibly illuminate the long term consequences on older adult health of

¹⁸ Almond and Currie (2010).

¹⁹ See for example, Clark (1930).

²⁰ De Schutter (2012).

²¹ Yan et al. (2012).

health policies that rapidly improve life at younger ages but without subsequent improvement in standard of living. There are few other examples which focus on the long term consequences of policy on older adult health in the developing world. If the conjecture is proven to have merit these older adults will have a substantial impact on health care systems and costs in the years to come.

It is not yet clear what will happen in the tip of the iceberg countries and if there will there be a tide of adults with poor health due to poor early life conditions. If a large portion of the cohorts born during the 1930s–1960s have been marked by poor early life conditions which increase the burden of older adult diseases, then appropriate types of interventions and programs will be needed for these older adults to support their special needs. The epigenetic basis for disease holds promise in developing future therapeutic approaches to prevent or address disease,²² and there is evidence of effective programs aimed at young children and adults that could potentially mediate the effects of poor early life conditions.²³ However, better clarity is needed regarding the degree to which the effects of early life are amenable to modification through interventions and at which points they will be most effective. Knowing how to best translate research into relevant public policy guidelines, especially in low- and middle-income countries, continues to be important.²⁴

Investment in health is a contentious issue and decisions on those investments by government and non-profit organizations are of necessity made in the context of restricted and limited resources. As population aging becomes an increasingly important concern for policy makers throughout the world, the costs for treatment of older adult heart disease, obesity, and diabetes, which are increasingly common, will become more important. There is already a large investment in nutrition for pregnant women and in early life, and many institutions and non-profit entities are already focused on improving maternal and child health and reducing the effects of chronic malnutrition throughout the world.²⁵ With limited resources, government and non-profit entities will have to ascertain the best investments to adequately balance the demands posed by older adults and the young.

Recent studies show that across the world, life expectancy is generally increasing because of improvements in early life conditions.²⁶ It can be assumed that continued investments in improving maternal and infant health will go a long way toward improving health around the world now and into the future. However, the historical circumstances of the 1930s–1960s and the results presented in the preceding chapters raise the question of whether this approach will be sufficient. Policies focused primarily on investments in improving health primarily through public health including medical technology have benefited many individuals but

²² Waterland (2006).

²³ Almond et al. (2009).

²⁴ See for example Franko, O'Connor, and Morton (2009).

²⁵ For example, WHO (2006).

²⁶ Murray et al. (2012) and Lim et al. (2012).

it may be worthwhile to consider augmenting these policies to also achieve improvement in standard of living over the long run.

Recent studies also show improvement in general life expectancy in the developed world²⁷ with a few noted exceptions.²⁸ It is tempting to assume that the developing world will follow these patterns. However, as presented in the preceding chapters it may very well be that the unique historical circumstances in the developing world will result in very different patterns of life expectancy in these settings.

Within this context, evaluating the impact of historical circumstances and interventions on older adult well being and forecasting the future impact of such interventions are of relevance to policy makers interested in learning from the past to guide the creation of better policy. The natural experiment of the 1930s–1960s is an important case study. It raises an important question regarding the extent to which rapid mortality transitions in early life without economic growth and then later rapid changes in adult diet are a deadly combination leading to unintended consequences in older adult health. Humans exposed to generations of chronic malnutrition may not have evolved quickly enough to adapt rapidly across several generations from chronic malnutrition to a more plentiful and perhaps less healthy diet as they age.²⁹ The influence of public health, medical technology, and standard of living as they impact health continue to require study.³⁰ Understanding the overall impact of exposure to rapidly changing environmental conditions both in early and adult life is important for effective policy.

There are several noted examples of the positive consequences of policies that promote rapid improvement in social achievement and economic growth in the developing world.³¹ In some instances these achievements have come about even with slower economic growth. Among the noted examples are Barbados and Costa Rica. Rapid transitions in sanitation, public health measures, and education due to strong health reforms and public education³² over the long run may have increased the standard of living in Barbados. It is now a high income country. Costa Rica has improved its social indicators including life expectancy.³³ Both are tip of the iceberg countries but they may be exceptions in terms of their achievements because of their smaller size and subsequent ability to make significant improvements that affect the entire population.

The case of the 1930s–1960s is relevant for future generations. There is at least one important region of the world which mirrors some of the early life conditions of the unique cohorts of the 1930s–1960s. While many other countries of the world have seen improvements in IMR with accompanying improvement in nutrition and

²⁷ Floud, Fogel, Harris, and Chul Hong (2011).

²⁸ Crimmins, Preston, and Cohen (2010) and Woolf and Aron (2013).

²⁹ See for example Popkin et al. (2001).

³⁰ See for example, Deaton (2007) and Soares (2007).

³¹ Mehrotra and Jolly (1997).

³² Bishop, Corbin, and Duncan (1997) and West India Royal Commission Report (1945).

³³ Garnier, Grynspan, Hidalgo, Monge, and Trejos (1997).

growth in height, this has not been the case in sub-Saharan Africa. Rapid improvements in IMR primarily due to medical innovations have resulted in little gains in nutritional status. There is a continued high degree of stunting in the population—one of the highest in the world.³⁴ Individuals born under these circumstances may be at higher risk of adult heart disease and diabetes.

6.4 Research Agenda

The use of population studies in this study brings to attention the need for better ways of examining the consequences of early life conditions in general. The next generation of population studies sampling from an increasing number of older adults born in the 1950s and beyond is already planned and in the field.³⁵ Population researchers are working with biomedical researchers to incorporate more biomarkers into population surveys,³⁶ and some work in the developing world has already begun.³⁷ Biomarkers will improve the measurement of adult health outcomes and address concerns regarding the underestimation of adult chronic conditions using self-reported measures. Surveys of older adults now often include information on adult occupation, migration, and lifestyle factors providing a more complete life course history. Linking other types of data (e.g., administrative, historical census vital statistics, or information on nutritional conditions) with survey data holds promise for enhancing analyses of early life conditions.³⁸

Improving the measurement of early life conditions in population-based studies in low- and middle-income countries will be a more difficult task, especially for older adults born in the early to mid twentieth century. More detailed questions on childhood and childhood illnesses are needed, with timing and severity of illnesses. It is difficult to obtain reliable information about illnesses during childhood and reliable historical data such as census or administrative data that may complement survey data. Until such data are available, this compiled, large cross-national dataset across diverse countries provides a lens, albeit a blurry one, through which to view early life conditions and older adult health.

Just as demographers are in the position to interpret social and behavioral factors discovered in population-based studies, biomedical researchers hold the key to illuminating biological aspects of health and providing understanding of different environmental settings and population-specific risk factors. More collaborative research efforts combining population-based surveys with biomedical research

³⁴ Akachi and Canning (2010), and Hult, Tornhammar, et al. (2010).

³⁵ See, for example, the Chinese Health and Retirement Longitudinal Study (CHARLS) at www.charls.ccer.edu.cn/zh-CN and the Longitudinal Aging Study in India (LASI) at www.hsph.harvard.edu/pgda/LASI/about.html

³⁶ Crimmins, Kim, and Vasunilashorn (2010).

³⁷ Brenes (2008), Gurven et al. (2009), and Yan et al. (2012).

³⁸ See for example Almond et al. (2009).

approaches have the potential to illuminate more fully how early life conditions impact older adult health and the degree to which interventions mediate or modify their effects.

There is continued emphasis on the importance of comparative cross-national studies of older adult health across diverse countries to help improve understanding of overall patterns.³⁹ This study developed the most extensive compilation of survey data from major studies of aging in low-, middle-, and high-income countries to date. The data encompass cross-national data but also cross-sectional/panel data on older adults. The health of older adults deserves more scrutiny in some countries such as Taiwan and Barbados which were very poor countries during the early to mid twentieth century and are now high income countries. Regional differences in large countries such as China, India, and Indonesia need to be examined more carefully. Historical data similar to what was obtained for Puerto Rico may help complement survey data and provide better understanding of historical circumstances.⁴⁰

There have not been many comparative cross national surveys that use biomarkers to assess adult health with some exception.⁴¹ As more surveys of older adults in the developing world contain biomarker data, it will be possible to more accurately ascertain the degree to which underestimation of chronic conditions influences the results obtained and to be more confident about cross national longitudinal analyses. A more thorough examination of the ramifications of missing values is needed and the linking of relevant administrative data with survey will strengthen the data. With these inclusions in mind, it will then be possible to take full advantage of panel data to examine health transitions (e.g., good health to poor health; no heart disease to heart disease; no functional difficulty to functional difficulty; co-morbidity) and to more fully address issues around unobserved heterogeneity.

6.5 Strengths and Limitations

The strengths of the study presented in this book include:

- Presentation of a topic that is of considerable interest in the scientific community;
- Examination of a conjecture about the historical circumstances of the 1930s–1960s and early life conditions that has important policy implications;
- Portrayal of health within a cross national comparative framework of diverse mortality regimes of the early to mid twentieth century;
- Synthesis of a large body of relevant literature;
- Compilation of a large amount of cross-national data based on major surveys of older adults, combined with historical data and an in-depth case study of Puerto Rico;

³⁹ National Research Council (2001).

⁴⁰ See for example Noel, Newby, Ordova, and Tucker (2009) and Tucker, Mattei, Noel et al. (2010).

⁴¹ Kim and Crimmins (2013).

- Examination of validity and underestimation of self-reported chronic conditions and deployment of sophisticated and innovative quantitative techniques such as Waaler-type surfaces to assess mortality risk.

One of the contributions this book has made mentioned above is to have compiled a tremendous amount of available data on individual older adult health across countries based on surveys but also historical data. This type of cross national data on this scale did not previously exist; it now facilitates the ability to conduct cross national comparative studies on aging populations using data from very different surveys. The data collected as part of the book are the types of data with which demographers and sociologists often work. They are by no means perfect. Data limitations cloud the ability to fully examine hypotheses regarding early life conditions and older adult health. The lack of better measures of health throughout the life course, underestimation of chronic conditions, and the heterogeneity of the diverse countries loom in the background. Cross sectional analyses are limited in their ability to make inferences about causality.

Nevertheless, the data are the best available to examine the conjecture. They have been shown to have a certain degree of validity and consistency. Underestimation may not be problematic in reaching conclusions in the case of diabetes because while the magnitude of association may be affected by underestimation, the direction of association with early life conditions does not appear to be. Population-level and individual-level data produced relevant cross national patterns between early life nutritional environmental and older adult health, and the case of Puerto Rico produced remarkably robust results that support the idea of the importance of *in utero*/early life exposures to poor nutrition and infectious diseases.

While older adults born in the tip of the iceberg countries provide insight, the contrarian conjecture is particularly relevant for those born after 1945 and the discovery of antibiotics and other medical innovations when most of the mortality decline in early life occurred in the developing world.⁴² Older adults may be more at risk to some degree given both their early life circumstances and increasing exposure at younger ages to diets higher in saturated fats and sodium. A “purer” test of the conjecture will be to examine improvements in survivorship as a result of antibiotics and other medical innovations for those born after 1945.

6.6 The Future: Tide, Trickle, or Flow

Findings on the importance of early life conditions have significance for high-income countries (e.g., the US, England and Wales, and the Netherlands) in that they provide a lens through which to view how the past may have shaped health at older ages and the mechanisms by which this occurs. The developed world, with its higher standard of living, no longer reflects a population exposed to poor nutrition, severe infectious diseases such as malaria, hookworm, tuberculosis or small pox, or poor socioeconomic conditions in early life. Nevertheless, early life conditions remain

⁴² Preston (1976).

important as cohorts that live through different historical periods continue to age.⁴³ Poor nutrition *in utero*/early infancy may impact at the individual level even though it may not define aggregated cohorts.

Findings on the importance of early life exposures have particular significance in anticipating the health care and policy needs of older adults in low- and middle-income countries who may have been more impacted by their early beginnings than their counterparts in high-income countries. Low- and middle-income countries are projected to experience large increases in the older adult population resulting in an increasing burden from chronic conditions such as heart disease and diabetes. In many instances, increases will occur mostly in the context of lower standards of living and fragile institutional support.⁴⁴ In these countries, poor nutrition for mothers and unborn children and exposure to infectious diseases such as malaria amid lower standards of living continue to be important health concerns.⁴⁵ Poor environmental conditions in early life along with lower standards of living and stagnant economic growth were predominant conditions in much of the developing world during the early twentieth century. These early life conditions, plus overwhelming poverty, made it difficult to properly sustain normal growth and development in children and led to increased susceptibility to disease for a large proportion of the population and made these populations different from those in the developed world with its higher standard of living and economic growth. Investments in education and employment opportunities in early adulthood which resulted in social mobility for those born under poor early life environmental conditions may have mediated the effects of early life conditions in some settings, but this may not have been true in the developing world, in particular for those unique cohorts of the 1930s–1960s born during a different moment in the demographic transition.

Forecasts show a rising tide of older adults with an increasing prevalence of chronic conditions across the world.⁴⁶ The early life conditions of some of these older adults were marked by poor nutritional conditions who survived infant and childhood diseases due to better medical and public health interventions, and some of these adults may have poor diets and/or be smokers. Although this study indicates the possible merit of the contrarian conjecture, the relative importance of early life factors or other factors across the life course as determinants of older adult health such as heart disease and diabetes is not yet clear for the entire membership of the unique cohorts of the 1930s–1960s. It is not yet clear whether the survivors of infant and childhood diseases due to circumstances in the 1930s–1960s will also translate into a tide of adults with poor health at older ages

⁴³ Harper, Lynch, and Davey Smith (2011).

⁴⁴ Barrientos (1997), De Vos and Palloni (2001), Klinsberg (2000), Mesa-Lago (1994), and Palloni (2002).

⁴⁵ Black et al. (2008) and Victora et al. (2008).

⁴⁶ Murray et al. (2012), Lim et al. (2012), and Yan et al. (2012).

or whether this tide will be reduced to a trickle or small flow because of better conditions later in life.

Yet, the data presented here are consistent with an argument based on the contrarian conjecture for the unique cohort of the 1930s–1960s. The data do not dismiss the possibility that rapid mortality transitions in early life in poor countries without economic growth and then later rapid changes in adult diet may be a deadly combination for older adult health. The data provide insight into the long term consequences of public policy and medical interventions at early ages on older adult health across such a diverse group of high-, middle- and low- income countries.

This is the kind of work that is possible now with the available data and it is hoped that this work will motivate future work. The ambitious effort to compile and collect data to examine a conjecture with important ramifications if proven to have merit has thus not been in vain; it has provided a baseline glimpse into the unique cohorts of the 1930s–1960s. As surveys of older adults incorporate more biomarkers to obtain a better sense of adult health status and as relevant administrative and historical data become available it will be possible to further examine the contrarian conjecture—a conjecture that warrants further examination. The conjecture is of importance and relevance to older adult health because it presents a historical and macro explanation to explain the determinants of older adult health in some settings which has not yet been fully explored. A careful examination of the unique cohorts of the 1930s–1960s has the potential for guiding future policy to improve people’s lives.

Appendix A: Sources for Life Expectancy and Infant Mortality

Some life expectancy was identified through extensive bibliographies (Riley, 2005a). It was not always possible to obtain life expectancy or IMR in the early years of the twentieth century for some countries and thus published research studies were used in these cases. In some cases, interpolation was used to bridge the gap between estimates of life expectancy, IMR, or GDP per capita. Shown in the table are references with the relevant years covered. Complete citations are found in the reference section.

Table A.1 Sources for life expectancy and infant mortality

Country	Reference with reference date in parentheses	Years covered
Argentina	Astorga, Berges, and Fitzgerald (2005)	1900–2000
	United Nations (UN) (2002)	1950–2000
	CEPAL/CELADE (2001)	1950–2000
	World Health Organization (WHO) (2002)	2000
Bangladesh	UN (2002)	1950–2000
	Max Planck Institute (2007)	1970s
	Dyson (1997)	Prior to 1950
	WHO (2002)	2000
	Mitchell (2003a)	1900–1960
	Bideau, Desjardins, and Pérez Brignoli (1997)	Early 1900s
Barbados	UN (2002)	1950–2000
	Mitchell (2003b)	1900–1960
	Bishop, Corbin, and Duncan (1997)	Prior to 1950
	WHO (2002)	2000
Brazil	West India Royal Commission Report (1945)	1940s
	Astorga et al. (2005)	1900–2000
	Arriaga (1968)	1900, 1920, 1940, 1950, 1960
	UN (2002)	1950–2000
	CEPAL/CELADE (2001)	1950–2000
Chile	WHO (2002)	2000
	Astorga et al. (2005)	1900–2000
	Arriaga (1968)	1907, 1920, 1930, 1940, 1952, 1960
	UN (2002)	1950–2000
	CEPAL/CELADE (2001)	1950–2000
	WHO (2002)	2000

(continued)

Table A.1 (continued)

Country	Reference with reference date in parentheses	Years covered
China	Max Planck Institute (2007)	1929
	UN (2002)	1950–2000
	Banister (1987)	Prior to 1950
	Coale (1984)	1984
	WHO (2002)	2000
Costa Rica	Astorga et al. (2005)	1900–2000
	Arriaga (1968)	1927, 1940, 1950, 1963
	Rosero-Bixby and Caamaño (1984)	1900, 1910, 1920, 1930, 1940, 1950, 1960, 1970, 1980
	UN (2002)	1950–2000
	CEPAL/CELADE (2001)	1950–2000
Cuba	WHO (2002)	2000
	Astorga et al. (2005)	1900–2000
	UN (2002)	1950–2000
	CEPAL/CELADE (2001)	1950–2000
	WHO (2002)	2000
England and Wales	Max Planck Institute (2007)	1900–2000
	UN (2002)	1950–2000
	WHO (2002)	2000
Ghana	UN (2002)	1950–2000
	Patterson (1981)	1981
	Caldwell (1967)	Prior to 1950
	WHO (2002)	2000
India	Max Planck Institute (2007)	1901, 1911, 1921, 1931, 1941, 1951, 1961, 1971, 1981, 1991, 1995
	UN (2002)	1950–2000
	WHO (2002)	2000
	Dyson (1997)	Prior to 1950
	Mitchell (2003b)	1900–1960
Indonesia	Preston (1976)	1930
	UN (2002)	1950–2000
	Nitisastro (1970)	Prior to 1950
	WHO (2002)	2000
Mexico	Astorga et al. (2005)	1900–2000
	Arriaga (1968)	1900, 1910, 1921, 1930, 1940, 1950, 1960
	UN (2002)	1950–2000
	CEPAL/CELADE (2001)	1950–2000
	Preston (1976, 1980)	1935, 1940
Netherlands	WHO (2002)	2000
	Max Planck Institute (2007)	1900–2000
	UN (2002)	1950–2000
	Preston (1976, 1980)	1931, 1940
	WHO (2002)	2000
	Mitchell (2003c)	1900–1960

(continued)

Table A.1 (continued)

Country	Reference with reference date in parentheses	Years covered
Puerto Rico	Vázquez Calzada, Morales, and Janer (1963)	1900–1985
	Preston (1980)	1976, 1980
	UN (2002)	1950–2000
	WHO (2002)	2000
South Africa	Max Planck Institute (2007)	1920, 1940–1970
Taiwan	UN (2002)	1950–2000
	Van Tonder and Van Eeden (1975)	1975
	WHO (2002)	2000
US	Barclay (1954)	early twentieth century
	Max Planck Institute (2007)	1930–2000
	WHO (2002)	2000
Uruguay	Max Planck Institute (2007)	1900–2000
	UN (2002)	1950–2000
	CEPAL/CELADE (2001)	1950–2005
	Migliónico (2001)	1908–1999
	WHO (2002)	2000

Appendix B: Graphs for Life Expectancy and IMR Across Time and Countries

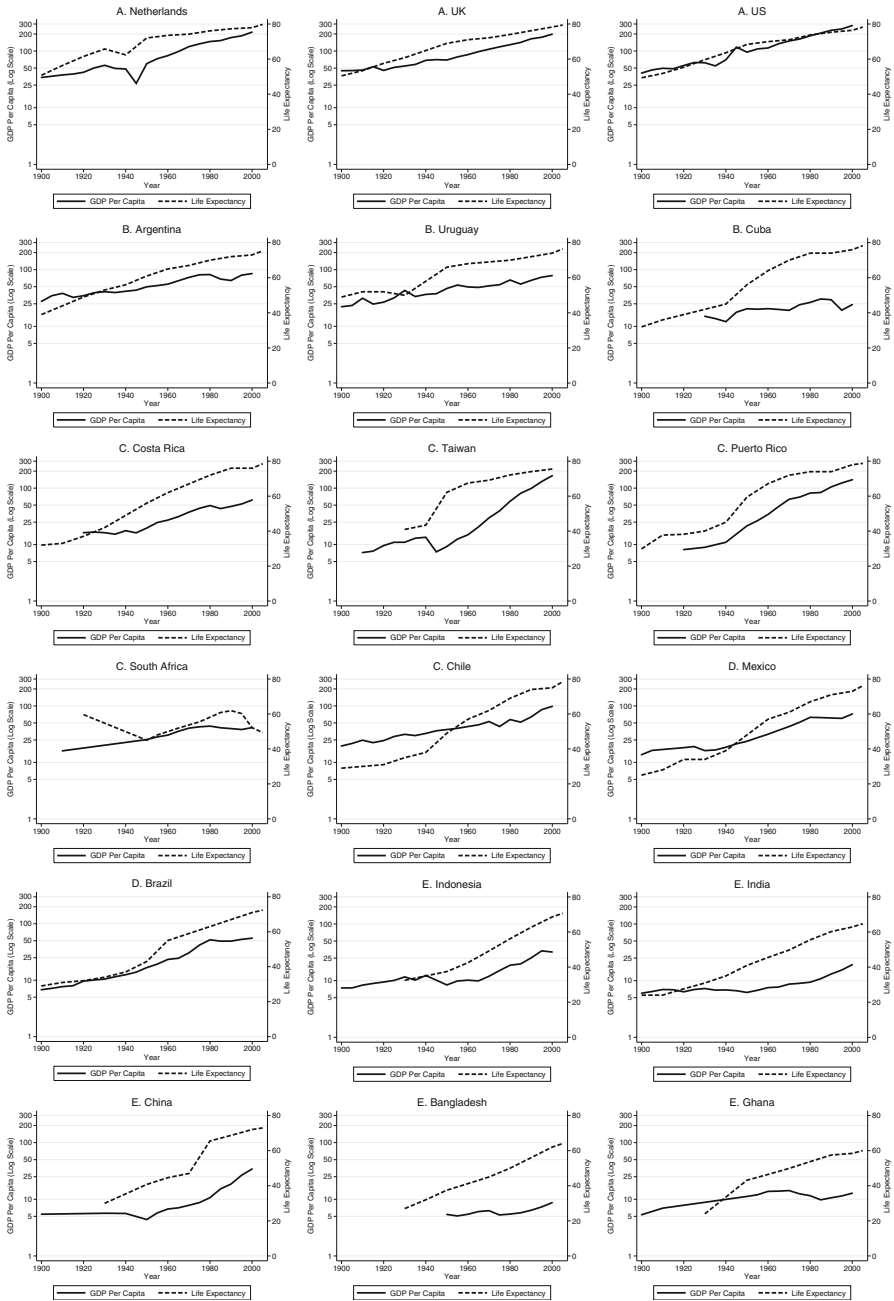


Fig. B.1 Life expectancy vs. In GDP per capita (1990 international dollars) from 1900 to 2000. *Notes:* A Very early regimes, B Early regimes, C Mid-paced regimes, D Late regimes, E Very late regimes. Barbados (*pattern D*) not shown due to incomplete information on GDP per capita (*Source:* Maddison, 2006; Appendix A)

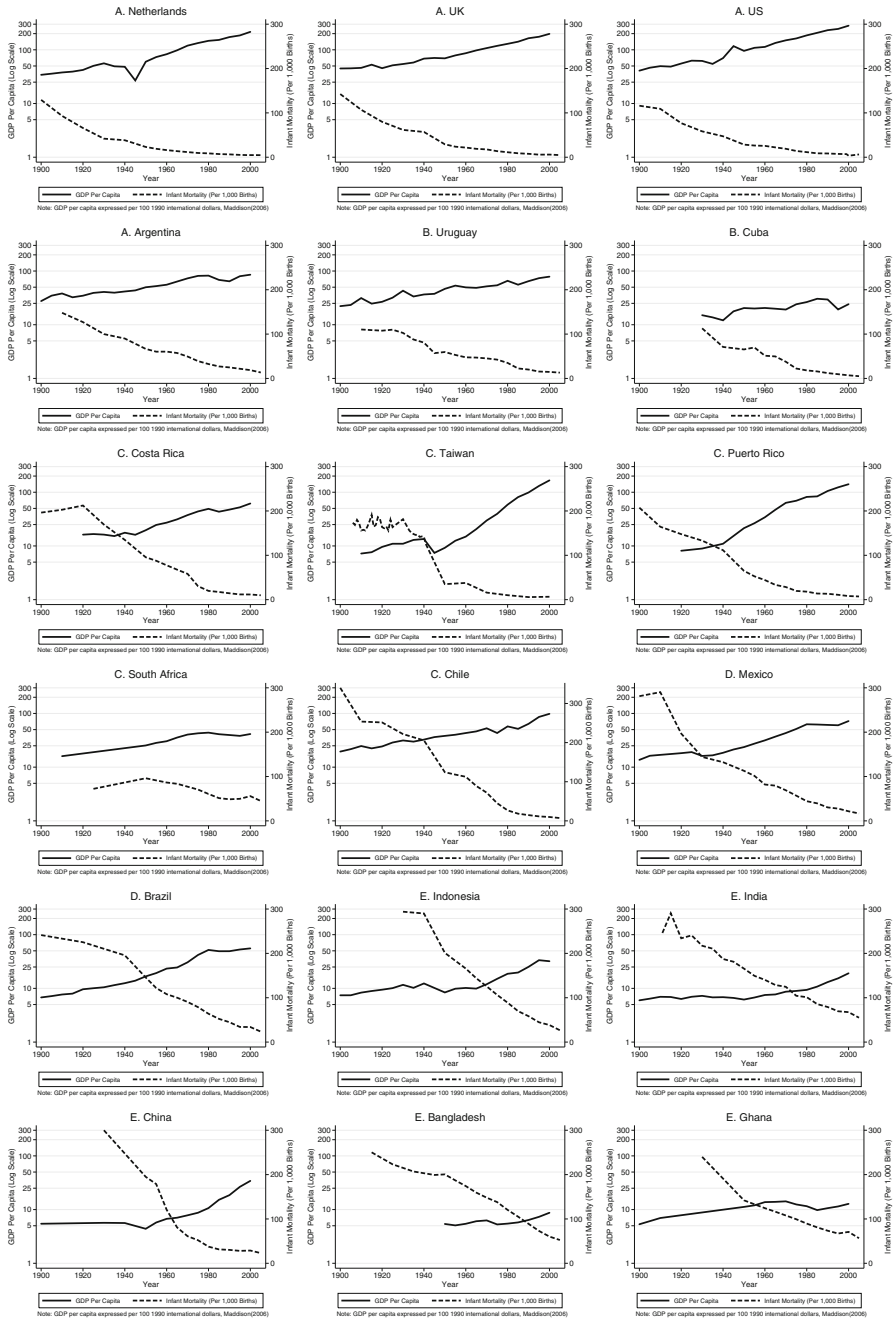


Fig. B.2 IMR vs. ln GDP per capita (1990 international dollars) from 1900 to 2000. Notes: A Very early regimes, B Early regimes, C Mid-paced regimes, D Later regimes, E Very late regimes. Barbados (pattern D) not shown due to incomplete information on GDP per capita (Source: Maddison, 2006; Appendix A)

Table B.1 Growth rate for 60 years and older

	1960	1970	1980	1990	2000	2010	2020	2030	2040	2050
<i>Very early</i>										
Netherlands	2.6	2.1	1.4	1.3	1.1	2.9	2.0	1.5	-0.2	-0.3
England	1.3	1.8	0.3	0.3	0.4	1.8	1.3	1.5	0.3	0.6
US	1.9	1.9	2.0	1.3	1.0	2.6	2.8	1.4	0.7	0.9
<i>Early</i>										
Argentina	4.0	3.3	2.4	2.2	1.7	1.8	1.9	1.7	2.4	1.7
Cuba	3.0	3.9	2.5	2.5	3.3	2.2	2.1	3.1	0.2	0.1
Uruguay	1.5	1.8	1.4	1.7	1.0	0.9	1.4	1.1	1.4	0.9
<i>Mid-paced</i>										
Chile	2.9	2.6	2.2	2.9	3.0	3.5	3.9	2.6	1.2	1.3
Costa Rica	2.9	2.5	3.3	3.0	3.5	4.0	4.9	3.5	2.5	2.2
Puerto Rico	3.1	4.1	5.5	2.8	2.7	2.6	1.8	1.1	1.1	0.9
South Africa	2.4	0.9	1.9	2.8	3.5	2.8	2.5	1.5	1.1	1.9
Taiwan										
<i>Late</i>										
Barbados	1.2	2.2	1.0	0.1	0.5	3.2	4.5	2.4	0.2	-0.2
Brazil	4.0	3.6	2.9	3.3	3.6	3.6	3.9	3.0	2.5	1.6
Mexico	3.7	3.0	2.5	3.5	3.2	3.4	3.9	3.6	2.6	1.5
<i>Very late</i>										
Bangladesh	0.2	2.2	2.4	2.6	3.2	3.3	4.1	3.8	3.1	2.9
China	0.6	2.2	2.8	2.7	2.7	3.2	3.0	3.7	0.7	0.9
Ghana	3.6	2.4	2.2	3.3	3.5	3.5	3.2	3.4	3.6	3.4
India	2.4	2.9	2.9	3.1	3.1	2.8	3.6	3.1	2.8	2.5
Indonesia	0.3	2.8	2.5	3.6	3.6	2.5	3.9	3.7	2.7	1.6

Source: Author's calculations based on United Nations Department of Economic and Social Affairs (2010)

Appendix C: Description of Cross-National Data on Aging Populations

Many of the surveys are or will be longitudinal in nature, and some have or will have linkages to administrative data. Studies with more than two waves of data are: CLHLS, CRELES, ELSA, MHAS, SHARE, HRS, CHNS, IFLS, and WLS (MHAS is conducting a follow-up in 2012 and 2014). Studies with only two waves of data are: PREHCO, and SEBAS. SAGE currently has one wave of data, but will soon have two waves.

Several studies collected biomarkers. These include SEBAS, WLS, SAGE, CHNS, CRELES. Not all of the biomarkers are publicly available.

Table C.1 Description of cross-national data on aging populations as of August 2011

Country	Income level	Selected wave	Sample	Ages	Wave used for health transition
<i>Very early regime</i>					
Netherlands SHARE	High	2004–2005 (wave 1)	2,979	50+	2006–2007 (wave 2)
England ELSA	High	2004–2005 (wave 2)	8,780	50+	2006–2007 (wave 3)
US HRS	High	2000	12,527	60+	2006
US WLS	High	2004–2005	10,317	60+	(Mortality data up to 2009)
<i>Early regime</i>					
Argentina SABE	Upper middle	2000 (wave 1)	1,043	60+	None available
Cuba SABE	Upper middle	2000 (wave 1)	1,905	60+	None available
Uruguay SABE	Upper middle	2000 (wave 1)	1,450	60+	None available
<i>Mid-paced regime</i>					
Chile SABE	Upper middle	2000 (wave 1)	1,301	60+	2005 (wave 2) not yet publicly accessible
Costa Rica CRELES	Upper middle	2003 (wave 1)	2,827	60+	Up to 2008 (wave 2; 3rd wave pending access)
Puerto Rico PREHCO	High	2002–2003 (wave 1)	4,291	60+	2006–2007 (wave 2)
South Africa SAGE	Upper middle	2007–2008 (wave 1)	3,830	50+	Planned for future (wave 2)
Taiwan SEBAS	High	2000 (wave 1)	1,023	50+	2006–2007 (wave 2) not yet publicly available
<i>Late regime</i>					
Barbados SABE	High	2000 (wave 1)	1,508	60+	None available
Brazil SABE	Upper middle	2000 (wave 1)	2,143	60+	(Waves 2 and 3) not yet publicly accessible
Mexico MHAS	Upper middle	2001 (wave 1)	13,463	50+	2003 (wave 2)
Mexico SABE	Upper middle	2000 (wave 1)	1,247	60+	None available
Mexico SAGE	Upper middle	2007–2008 (wave 1)	4,142	50+	Planned for future (wave 2)
Russian Federation SAGE	Upper middle	2007–2008 (wave 1)	4,511	50+	Planned for future (wave 2)

<i>Very late regime</i>								
Bangladesh MHSS	Low	1996 (wave 1)	6,973	36+	Mortality through Dec 2007 for 50+ in 1996			
China CHNS	Lower middle	2000 (wave 5)	6,452	40+	2006 (wave 7)			
China CLHLS	Lower middle	2002 (wave 3)	16,064	60+	2005 (wave 4)			
China SAGE	Lower middle	2007–2008 (wave 1)	13,368	50+	Planned for future (wave 2)			
Ghana SAGE	Low	2007–2008 (wave 1)	4,724	50+	Planned for future (wave 2)			
India SAGE	Lower middle	2007–2008 (wave 1)	7,150	50+	Planned for future (wave 2)			
Indonesia IFLS	Lower middle	2000 (wave 3)	13,260	40+	2007–2008 (wave 4)			
<i>Total</i>			147,278					

Notes: Countries arranged by mortality regime and by World Bank definition of income. Longitudinal data listed above but not publicly accessible are: SEBAS II, Brazil-SABE (waves 2 and 3), Chile-SABE (wave 2), Cuba-SABE (wave 2). Ages shown are the ages at the time of the first survey selected. Not shown in the table are surveys used as pre-tests for the SAGE study: WHO/SAGE's pilot studies for India (492 individuals), Ghana (507 individuals), and Tanzania (525 individuals). While the total sample size in the table is 147,278, the subsample of respondents born before 1960 and whose age could be ascertained for analyses was 143,968. The difference in sample size reflects exclusion of 100 cases born in the 1960s, 2,616 cases where age was missing (2,030 of which are from Mexico-SAGE), and 594 cases from the WLS cohort where the high school graduate was either dead or did not respond to the 2004 survey. Please refer to the following references in parentheses for more detailed information regarding a particular study: CHNS (University of North Carolina-Chapel Hill); CLHLS (Zeng, Vaupel, Zhenyu, Yuzi, & Chunyuan); CRELES (Rosero-Bixby, Fernández, & Dow); ELSA (National Centre for Social Research); HRS (Health and Retirement Study); IFLS (Gertler, Frankenberg, & Karoly); MHAS (Soldo, Wong, Palloni, & Instituto Nacional de Estadística, Geografía e Informática); MHSS (Rahman, Menken, Foster, & Gertler); PREHCO (Puerto Rican Elderly Health Conditions); SABE (SABE); SAGE (World Health Organization); SEBAS (Goldman, Weinstein, Chang, Lin, Chang, Lin, & Wu); SHARE (Mannheim Research Institute for Economics of Ageing); WLS (Herd, Hauser, & Sewell)

Appendix D: Descriptive Statistics on Older Adult Heart Disease and Diabetes

Table D.1 Prevalence of heart disease by age and gender

Regime/country	Ages 50–54 (%)	Ages 55–59 (%)	Ages 60–64 (%)	Ages 65–69 (%)	Ages 70–74 (%)	Ages 75–79 (%)	Ages 80–84 (%)	Ages 85+ (%)
<i>(a) Males</i>								
A. US-HRS			20.6	25.0	34.1	35.6	36.9	39.8
A. Netherlands	3.9	10.6	13.7	21.3	23.4	29.3	17.6	29.8
A. England	12.9	17.8	22.7	27.0	33.2	38.2	41.2	39.6
B. Argentina			18.6	23.2	23.1	21.5	11.3	
B. Cuba			14.7	22.2	20.0	21.4	12.2	31.8
B. Uruguay			10.3	26.5	23.8	29.1	20.4	
C. Chile			21.4	33.4	38.0	29.0	8.1	33.2
C. Costa Rica			11.3	9.8	11.6	14.4	12.2	16.1
C. Puerto Rico			12.7	14.6	18.7	21.4	17.9	28.2
C. South Africa	5.1	6.3	5.8	13.3	9.2	3.0	12.0	0.5
C. Taiwan		5.2	13.7	12.4	19.1	16.6	17.9	
D. Barbados			10.6	7.3	15.7	14.1	14.2	7.7
D. Brazil			15.2	20.0	26.6	24.1	29.1	21.5
D. Mexico-MHAS	1.2	2.4	3.6	2.3	3.6	4.0	4.1	6.6
E. China-CLHLS				17.9	17.7	20.1	18.0	16.6
E. China-SAGE	2.6	4.4	5.4	9.7	14.2	14.3	18.6	23.1
E. Ghana	4.9	11.3	10.7	11.6	13.0	10.0	7.4	10.7
E. India	10.1	14.2	15.9	17.6	26.8	25.9	14.4	25.7
E. Indonesia	4.1	1.9	4.2	5.4	3.7	2.9		
<i>(b) Females</i>								
A. US-HRS			13.9	17.8	23.7	26.7	28.7	35.2
A. Netherlands	2.9	7.5	6.5	7.5	10.1	17.9	11.2	22.4
A. England	10.7	12.6	16.1	21.2	26.1	32.5	37.2	37.5
B. Argentina			17.0	15.6	21.1	16.5	29.8	24.1
B. Cuba			26.3	24.3	28.9	30.1	26.1	31.6
B. Uruguay			17.6	25.0	18.6	30.6	37.0	34.7
C. Chile			31.8	35.2	32.5	41.6	40.9	37.0
C. Costa Rica			8.5	11.0	13.0	16.1	17.3	19.0
C. Puerto Rico			12.8	19.1	20.2	19.7	19.8	28.6
C. South Africa	11.3	9.9	9.4	14.0	6.9	10.4	6.5	8.0
C. Taiwan		8.0	12.1	16.4	27.5	31.3	23.9	
D. Barbados			5.7	7.3	10.2	16.5	17.8	16.2
D. Brazil			14.2	17.7	21.6	23.4	22.4	28.5
D. Mexico-MHAS	3.7	1.7	2.2	3.5	4.5	3.2	4.7	2.1

(continued)

Table D.1 (continued)

Regime/country	Ages 50–54 (%)	Ages 55–59 (%)	Ages 60–64 (%)	Ages 65–69 (%)	Ages 70–74 (%)	Ages 75–79 (%)	Ages 80–84 (%)	Ages 85+ (%)
E. China-CLHLS				21.5	20.2	17.5	18.1	17.0
E. China-SAGE	7.3	8.5	12.0	18.2	18.6	22.7	28.4	20.3
E. Ghana	14.0	15.5	17.7	17.5	17.4	21.1	22.9	12.0
E. India	21.4	19.4	25.2	27.0	22.2	36.2	28.2	22.7
E. Indonesia	3.4	3.5	3.4	2.2	5.8	1.8		

Source: RELATE (2013), weighted where relevant

Note: Highest prevalence is the Russian Federation and SAGE data using the Rose questionnaire (Rose, 1962)

Mortality regimes: A Very early, B Early, C Mid, D Late, E Very late

Not all surveys collected information on 50–59 year old adults

Table D.2 Prevalence of diabetes by age and gender

Regime/Country	Ages 50–54 (%)	Ages 55–59 (%)	Ages 60–64 (%)	Ages 65–69 (%)	Ages 70–74 (%)	Ages 75–79 (%)	Ages 80–84 (%)	Ages 85+ (%)
<i>(a) Males</i>								
A. US-HRS			15.1	17.4	18.6	18.9	15.1	14.1
A. Netherlands	5.1	6.3	7.0	9.6	9.2	15.9	7.6	
A. England	5.9	8.1	7.6	12.3	14.9	15.5	12.5	7.5
B. Argentina			13.9	19.8	10.4	15.5	7.6	
B. Cuba			7.0	8.0	9.5	7.5	2.8	3.8
B. Uruguay			10.7	12.4	15.1	12.1	12.9	
C. Chile			14.6	11.6	8.2	18.8		10.8
C. Costa Rica			15.7	18.7	20.8	18.9	9.6	11.1
C. Puerto Rico			26.8	26.1	32.2	27.3	20.6	22.4
C. South Africa	3.7	5.3	9.4	13.4	5.8	7.8	7.5	10.2
C. Taiwan		10.0	17.2	4.3	11.2	9.9	13.7	
D. Barbados			27.9	14.9	17.0	23.1	12.5	10.0
D. Brazil			12.8	23.0	17.8	17.6	15.0	8.1
D. Mexico-MHAS	9.9	13.2	12.6	16.0	18.2	11.6	15.4	11.4
E. China-CLHLS				13.7	12.7	13.4	11.9	12.3
E. China-SAGE	3.1	3.6	5.4	10.1	8.7	8.2	9.4	4.5
E. Ghana	3.0	1.6	6.6	4.3	3.0	2.3	2.6	1.0
E. India	7.7	9.8	7.0	7.3	10.9	5.1	6.5	3.4
E. Indonesia	4.0	5.2	3.5	3.2	0.6			
<i>(b) Females</i>								
A. US-HRS			12.4	16.0	16.0	15.0	14.4	10.5
A. Netherlands	5.2	5.0	9.8	10.3	10.0	23.7	10.9	12.4
A. England	3.6	4.2	7.3	7.2	10.4	10.7	11.0	8.1
B. Argentina			7.4	12.9	13.1	11.4	9.6	14.6
B. Cuba			17.1	21.1	18.5	23.5	24.7	18.7
B. Uruguay			12.9	14.3	17.9	18.2	7.7	5.7
C. Chile			14.2	14.5	18.4	10.9	14.5	7.9
C. Costa Rica			26.4	23.9	26.4	25.2	19.9	14.6
C. Puerto Rico			28.3	29.8	28.3	31.5	26.3	22.6

(continued)

Table D.2 (continued)

Regime/Country	Ages 50–54 (%)	Ages 55–59 (%)	Ages 60–64 (%)	Ages 65–69 (%)	Ages 70–74 (%)	Ages 75–79 (%)	Ages 80–84 (%)	Ages 85+ (%)
C. South Africa	6.6	13.1	10.4	10.2	29.4	8.8	5.2	3.1
C. Taiwan		3.3	19.8	22.2	28.6	27.2	26.9	
D. Barbados			24.5	20.9	25.9	31.6	15.9	20.3
D. Brazil			18.3	20.8	18.6	18.1	17.1	15.1
D. Mexico-MHAS	14.7	16.1	18.2	21.2	19.6	15.7	25.0	9.6
E. China-CLHLS				14.0	14.9	11.5	10.1	11.8
E. China-SAGE	4.2	6.1	7.7	9.4	10.8	10.5	9.3	2.2
E. Ghana	2.9	3.3	6.6	5.0	4.8	4.1	6.3	4.1
E. India	3.8	7.3	3.5	7.2	3.3	16.3	1.8	8.8
E. Indonesia	3.4	4.5	4.5	3.1	0.7			

Source: RELATE (2013), weighted where relevant

Note: Higher prevalence of diabetes among women in some countries

Mortality regimes: A Very early, B Early, C Mid, D Late, E Very late

Not all surveys collected information on 50–59 year old adults

Table D.3 Proportion who are obese for those with and without heart disease

Regime/ country	Heart disease	Ages 50–54 (%)	Ages 55–59 (%)	Ages 60–64 (%)	Ages 65–69 (%)	Ages 70–74 (%)	Ages 75–79 (%)	Ages 80–84 (%)	Ages 85+ (%)
A. US-HRS	Yes			35.1	29.8	24.9	19.7	12.6	7.6
	No			26.8	23.1	20.9	13.0	12.4	7.7
A. Netherlands	Yes		27.1	25.9	21.8	17.6	11.0		
	No	15.9	16.0	16.3	17.9	14.5	18.8	7.4	8.8
A. England	Yes	25.5	36.7	38.1	29.3	29.1	33.1	28.2	19.3
	No	32.4	28.7	29.0	29.0	31.8	25.1	19.3	10.1
A. US-WLS	Yes			39.8	41.2				
	No			29.5	30.8				
B. Cuba	Yes			28.8	29.1	15.3	16.9	9.5	
	No			18.4	12.8	7.6	11.6	5.2	10.4
B. Uruguay	Yes			43.3	31.8	35.5	34.4	41.6	
	No			36.1	31.6	33.1	35.6	34.2	36.2
C. Chile	Yes			46.1	34.7	19.6	18.2	32.8	19.5
	No			38.2	31.4	30.3	33.3	17.5	12.9
C. Costa Rica	Yes			42.0	44.0	19.0	22.6	14.1	9.6
	No			27.8	22.6	24.0	20.5	14.7	7.3
C. Puerto Rico	Yes			42.6	32.4	39.5	23.9	23.0	8.2
	No			30.8	26.7	25.0	26.1	24.2	15.6
C. South Africa	Yes	66.2	63.6	37.8	60.6	41.7	46.0		
	No	45.4	48.4	53.5	50.8	40.0	40.5	39.4	28.1
C. Taiwan	Yes			28.6	10.5	12.7	6.4		
	No		4.4	9.8	9.1	2.7	6.8	6.8	5.7

(continued)

Table D.3 (continued)

Regime/ country	Heart disease	Ages 50–54 (%)	Ages 55–59 (%)	Ages 60–64 (%)	Ages 65–69 (%)	Ages 70–74 (%)	Ages 75–79 (%)	Ages 80–84 (%)	Ages 85+ (%)
D. Barbados	Yes			33.0	38.0	21.7	17.6	7.7	
	No			26.3	31.8	30.1	18.2	13.9	7.7
D. Brazil	Yes			29.5	29.1	26.6	25.3	22.7	21.4
	No			22.6	18.7	22.7	18.4	15.0	13.0
D. Mexico- MHAS	Yes	54.9	19.2	27.7	45.1	22.1	10.2		
	No	26.0	23.3	26.8	18.9	17.7	15.8	11.1	9.0
D. Mexico- SABE	Yes			39.8	22.9	28.8			
	No			36.0	28.6	25.4	20.9	21.5	29.9
D. Mexico- SAGE	Yes		39.3	35.3	27.5	55.7	15.6		
	No		33.2	37.8	30.5	24.1	18.9	19.1	15.7
D. Russia	Yes	56.5	55.5	45.8	47.5	46.4	39.9	31.6	12.1
	No	31.1	38.1	29.1	22.4	17.0	17.0	14.7	
E. China- SAGE	Yes	10.7	14.4	12.0	11.0	11.4	7.7	2.1	
	No	5.0	6.5	5.3	5.3	3.7	5.1	5.8	4.7
E. Ghana	Yes	21.1	7.3	7.6	7.4	4.8	4.5		6.8
	No	12.5	13.6	11.5	9.4	6.5	9.2	4.3	8.9
E. India	Yes	5.4	4.3	2.7	1.5	3.0	0.8	14.4	
	No	3.2	2.2	2.1	2.7	3.1	0.3	3.0	1.1
E. Indonesia	Yes		5.2		8.4				
	No	4.7	3.0	1.7	1.2	1.1		6.7	

Source: RELATE (2013), weighted where relevant

Note: If the sample size for a particular age group and country was less than or equal to 20 individuals, the data point was omitted

Mortality regimes: A Very early, B Early, C Mid, D Late, E Very late

Not all surveys collected information on 50–59 year old adults

Table D.4 Proportion who are obese for those with and without diabetes

Regime/ country	Diabetes	Ages 50–54 (%)	Ages 55–59 (%)	Ages 60–64 (%)	Ages 65–69 (%)	Ages 70–74 (%)	Ages 75–79 (%)	Ages 80–84 (%)	Ages 85+ (%)
A. US-HRS	Yes			49.2	44.0	33.6	24.2	22.8	12.9
	No			24.8	20.6	19.6	13.2	10.8	7.0
A. Netherlands	Yes	56.0	35.4	32.1	22.4		27.7		
	No	13.7	15.9	15.9	18.0	14.1	14.3	5.6	7.6
A. England	Yes	48.9	56.0	69.2	41.8	46.1	31.6	31.8	
	No	30.8	28.2	28.3	27.8	28.8	27.5	21.5	13.2
A. US-WLS	Yes			56.3	58.2				
	No			28.0	28.4				
B. Cuba	Yes			25.6	18.9	13.1	6.7	13.9	
	No			20.0	16.2	8.9	14.5	4.8	7.4
B. Uruguay	Yes			32.5	41.3	34.6	50.9		
	No			37.9	30.3	33.5	32.4	37.2	36.4
C. Chile	Yes			35.4	30.1	30.1	7.7		
	No			42.1	33.6	26.1	30.0	18.3	16.4
C. Costa Rica	Yes			44.8	43.1	26.7	36.8	25.9	18.2
	No			25.4	19.7	22.1	16.4	12.7	6.5

(continued)

Table D.4 (continued)

Regime/ country	Diabetes	Ages	Ages	Ages	Ages	Ages	Ages	Ages	
		50–54 (%)	55–59 (%)	60–64 (%)	65–69 (%)	70–74 (%)	75–79 (%)	80–84 (%)	85+ (%)
C. Puerto Rico	Yes			34.9	36.2	31.3	30.5	31.8	22.1
	No			31.4	24.5	26.3	23.6	21.0	10.9
C. South Africa	Yes	69.6	74.8	67.6	56.4	48.9	36.5		
	No	46.4	46.8	50.2	51.8	37.7	41.3	36.6	24.4
C. Taiwan	Yes			5.0		11.8	5.6		
	No		4.9	13.9	8.7	3.4	6.8	1.5	
D. Barbados	Yes			28.8	52.6	35.6	23.5		
	No			26.2	27.8	27.3	15.9	11.8	6.8
D. Brazil	Yes			32.8	21.9	33.9	21.3	17.1	26.1
	No			22.0	19.8	21.7	19.9	16.9	13.2
D. Mexico- MHAS	Yes	33.2	25.8	29.8	28.1	18.0	16.8	5.3	2.0
	No	25.8	22.7	26.2	17.8	18.1	15.4	13.2	10.4
D. Mexico- SABE	Yes			34.3	31.9	24.1	22.5		
	No			36.9	26.7	25.8	22.9	18.8	28.7
D. Mexico- SAGE	Yes		35.9	47.6	20.9	31.7	36.9	19.1	
	No		33.4	36.6	33.3	27.6	15.9	21.1	13.9
D. Russia	Yes	44.0	78.5	34.9	64.3	60.6	47.1	58.0	
	No	36.0	42.5	31.6	27.7	28.8	31.2	20.1	16.8
E. China- CHNS	Yes					15.4			
	No	3.1	4.5	5.1	5.1	3.1	2.8	1.4	2.5
E. China- SAGE	Yes	16.4	16.8	10.8	5.6	11.0	14.3		
	No	5.3	6.3	6.0	5.7	4.9	5.3	5.4	2.8
E. Ghana	Yes	33.2	32.8	11.2	36.8	21.6	7.4		
	No	12.9	12.9	10.5	7.4	6.6	8.7	3.9	8.5
E. India	Yes	11.9	5.1	10.2	4.8	3.2	1.2		
	No	2.9	2.2	1.9	2.5	2.6	1.2	5.1	0.7
E. Indonesia	Yes	6.4	6.4	9.7					
	No	4.7	2.7	1.7	1.2	0.9		6.7	

Source: RELATE (2013), weighted where relevant

Note: If the sample size for a particular age group and country was less than or equal to 20 individuals, the data point was omitted

Mortality regimes: A Very early, B Early, C Mid, D Late, E Very late

Not all surveys collected information on 50–59 year old adults

Table D.5 Proportion reporting difficulty with at least one functional task for those with and without heart disease

Regime/ country	Heart disease	Ages 50–54 (%)	Ages 55–59 (%)	Ages 60–64 (%)	Ages 65–69 (%)	Ages 70–74 (%)	Ages 75–79 (%)	Ages 80–84 (%)	Ages 85+ (%)
A. US-HRS	Yes			17.2	19.2	25.0	22.8	30.2	37.6
	No			8.9	9.4	12.2	13.2	20.2	28.9
A. Netherlands	Yes		10.3	3.9	14.7	4.3	7.2	35.4	
	No	4.4	2.6	3.8	4.9	9.0	10.7	15.5	34.9
A. England	Yes	16.0	25.5	26.8	25.3	30.3	34.5	46.8	52.9
	No	8.6	11.9	13.5	16.4	20.7	25.7	27.5	41.9
B. Argentina	Yes			17.7	22.4	16.4	28.4		
	No			5.9	8.9	14.3	13.4	30.9	45.9
B. Cuba	Yes			16.3	24.5	17.8	27.8	39.1	40.8
	No			11.2	7.7	11.9	17.8	26.6	37.0
B. Uruguay	Yes			27.8	23.0	16.9	32.3	42.0	34.1
	No			9.5	10.5	8.7	17.6	19.9	16.6
C. Chile	Yes			20.6	23.5	34.8	30.4	40.2	63.6
	No			11.5	11.8	14.4	23.8	29.6	46.7
C. Costa Rica	Yes			9.3	15.5	17.7	20.8	33.0	53.5
	No			10.0	9.0	15.2	20.5	26.4	44.4
C. Puerto Rico	Yes			21.5	16.9	17.8	14.7	16.3	18.9
	No			8.1	8.6	9.6	10.6	10.0	12.4
C. South Africa	Yes	52.4	34.9	30.3	32.9	28.2	57.2		
	No	8.5	15.8	16.3	23.7	24.5	37.8	28.8	33.9
C. Taiwan	Yes				8.8	3.1	3.2		
	No		0.7	5.0	4.1	2.6	5.8	3.9	9.4
D. Barbados	Yes				11.8	5.1	9.8	17.9	
	No			3.6	5.8	9.8	12.1	17.4	23.3
D. Brazil	Yes			14.9	21.3	23.5	28.1	35.1	55.8
	No			14.4	10.7	16.4	22.7	27.6	41.0
D. Mexico- MHAS	Yes	25.4	10.5	23.9	12.8	20.3	42.4	52.6	
	No	4.6	5.8	6.3	9.3	10.2	11.7	18.8	35.5
D. Mexico- SABE	Yes			11.9	21.8	29.3	31.7		
	No			10.1	13.3	13.7	23.3	26.3	51.9
D. Mexico- SAGE	Yes	6.9	42.0	36.5	21.1	34.8	53.9		
	No			16.8	12.8	19.4	27.8	33.3	48.1
D. Russia	Yes	10.6	5.0	12.1	17.2	32.7	32.9	59.0	27.1
	No	0.5	1.3	1.9	9.7	7.1	37.3	43.5	46.4
E. China- CLHLS	Yes				5.8	7.6	11.5	22.8	28.7
	No				3.0	4.7	8.8	13.7	28.9
E. China- SAGE	Yes	0.8	6.1	7.9	11.9	10.8	14.5	22.3	34.7
	No	1.4	2.0	2.0	3.0	4.1	8.0	13.0	22.6
E. Ghana	Yes	22.6	25.5	34.2	39.1	41.0	49.8	42.3	62.4
	No	10.8	15.3	16.0	21.0	26.3	31.6	31.1	45.2
E. India	Yes	24.5	40.2	49.0	50.5	52.7	58.1	56.5	
	No	10.7	14.4	17.8	24.1	29.7	26.7	42.3	45.6
E. Indonesia	Yes		2.0	2.1					
	No	0.5	2.3	3.1	5.7	5.3	8.0	21.0	

Source: RELATE (2013), weighted where relevant

Note: If the sample size for a particular age group and country was less than or equal to 20 individuals, the data point was omitted

Mortality regimes: A Very early, B Early, C Mid, D Late, E Very late

Not all surveys collected information on 50–59 year old adults

Table D.6 Proportion reporting difficulty with at least one functional task for those with and without diabetes

Regime/ country	Diabetes	Ages 50–54 (%)	Ages 55–59 (%)	Ages 60–64 (%)	Ages 65–69 (%)	Ages 70–74 (%)	Ages 75–79 (%)	Ages 80–84 (%)	Ages 85+ (%)
A. US-HRS	Yes			17.4	22.7	25.0	23.9	29.4	42.6
	No			9.2	9.3	13.9	14.6	22.3	30.7
A. Netherlands	Yes	15.3			12.5	3.5	16.5		
	No	4.0	3.5	4.1	5.6	8.8	8.2	17.4	38.1
A. England	Yes	25.4	33.8	37.2	31.9	39.1	33.7	49.8	68.7
	No	8.7	12.8	14.4	17.1	21.3	28.1	33.2	44.0
B. Argentina	Yes			18.3	28.0	18.9	16.0		
	No			6.8	8.3	14.2	15.6	33.3	45.4
B. Cuba	Yes			16.1	15.9	21.0	30.1	47.4	57.4
	No			11.8	10.8	12.0	18.4	25.3	35.1
B. Uruguay	Yes			21.5	20.5	14.2	24.9		
	No			10.8	12.5	9.4	20.7	27.5	23.8
C. Chile	Yes			19.5	22.2	30.5	30.8		
	No			13.0	15.0	19.8	24.6	32.7	50.5
C. Costa Rica	Yes			14.1	10.0	19.7	20.5	34.3	55.5
	No			8.8	9.7	14.0	20.9	26.0	44.9
C. Puerto Rico	Yes			14.9	11.7	13.6	10.7	19.9	20.3
	No			8.0	9.4	10.1	12.1	8.6	12.6
C. South Africa	Yes	17.3	14.1	25.9	37.6	32.0	20.5		
	No	12.3	17.6	16.8	24.2	24.1	42.1	35.9	38.5
C. Taiwan	Yes					6.8	5.6		
	No		1.1	5.4	1.4	1.8	5.1	4.7	
D. Barbados	Yes			4.1	3.3	7.8	20.8	24.3	41.6
	No			3.1	6.6	9.2	8.2	16.1	23.1
D. Brazil	Yes			16.9	14.5	35.3	19.7	37.8	55.6
	No			14.4	12.6	14.2	25.2	27.5	43.0
D. Mexico- MHAS	Yes	12.1	10.3	11.8	11.6	16.9	22.2	24.5	39.6
	No	4.2	5.1	5.9	9.0	8.5	11.2	19.4	36.7
D. Mexico- SABE	Yes			15.5	16.6	19.6	25.5	20.1	
	No			8.7	13.1	14.5	24.0	30.6	54.0
D. Mexico- SAGE	Yes		24.5	22.5	21.9	28.4	35.9	42.2	
	No		8.4	19.6	12.5	21.4	30.4	34.5	53.7
D. Russia	Yes	5.4	4.8	25.0	47.3	60.2	62.4	87.5	
	No	3.1	3.0	8.6	12.1	15.9	32.2	46.9	37.2
E. Bangladesh	Yes	13.9	8.0	30.1	32.4	26.0	44.8	60.3	
	No	4.2	9.5	14.4	20.8	32.1	38.5	42.1	42.6
E. China- CHNS	Yes			4.8		48.1			
	No	5.5	4.1	8.1	10.6	18.2	26.3	52.1	57.5
E. China- CLHLS	Yes				5.1	6.4	10.7	21.9	26.8
	No				3.3	5.1	9.1	14.5	29.1
E. China- SAGE	Yes	0.8	1.4	3.5	6.7	7.5	14.0	14.1	35.7
	No	1.5	2.6	2.6	5.0	5.7	10.1	16.2	24.1

(continued)

Table D.6 (continued)

Regime/ country	Diabetes	Ages	Ages	Ages	Ages	Ages	Ages	Ages	Ages
		50–54 (%)	55–59 (%)	60–64 (%)	65–69 (%)	70–74 (%)	75–79 (%)	80–84 (%)	85+ (%)
E. Ghana	Yes	20.2	27.7	38.7	29.6	35.5			
	No	12.4	16.7	19.6	24.5	28.9	36.5	36.1	50.5
E. India	Yes	17.9	16.6	27.8	31.5	25.2	60.9		
	No	13.6	18.2	24.1	31.6	36.5	33.6	45.5	49.6
E. Indonesia	Yes		4.5	9.0					
	No	0.5	2.2	2.8	5.4	5.0	8.7	21.0	

Source: RELATE (2013), weighted where relevant

Note: If the sample size for a particular age group and country was less than or equal to 20 individuals, the data point was omitted

Mortality regimes: *A* Very early, *B* Early, *C* Mid, *D* Late, *E* Very late

Not all surveys collected information on 50–59 year old adults

References

- Abegunde, D. O., Mathers, C. D., Adam, T., Ortegón, M., & Strong, K. (2007). The burden of costs of chronic diseases in low-income and middle-income countries. *Lancet*, *370*, 1929–1938.
- Aboderin, I., Kalache, A., Ben-Shlomo, Y., Lynch, J. W., Yajnik, C. S., Kuh, D., et al. (2002). *Life course perspectives on coronary heart disease, stroke and diabetes: Key issues and implications for policy and research*. Geneva, Switzerland: World Health Organization.
- Achan, J., Talisuna, A. O., Erhart, A., Yeka, A., Tibenderana, J. K., Balirain, F. N., et al. (2011). Quinine, an old anti-malarial drug in a modern world: Role in the treatment of malaria. *Malaria Journal*, *10*, 144.
- Adair, L. S., Fall, C. H. D., Osmond, C., Stein, A. D., Martorell, R., Ramirez-Zea, M. et al. (2013). Associations of linear growth and relative weight gain during early life with adult health and human capital in countries of low and middle income: Findings from five birth cohort studies. *The Lancet*, *382*(9891), 525–534. Early online publication, 28 March 2013. doi:[10.1016/S0140-6736\(13\)60103-8](https://doi.org/10.1016/S0140-6736(13)60103-8).
- Ahmad, O. B., Boschi-Pinto, C., López, A. D., Murray, C. J. L., Lozano, R., & Inoue, M. (2001). *Age standardization of rates: A new WHO standard* (GPE Discussion Paper Series: No. 31). Geneva, Switzerland: WHO.
- Akachi, Y., & Canning, D. (2010). Health trends in Sub-Saharan Africa: Conflicting evidence from infant mortality rates and adult heights. *Economics and Human Biology*, *8*, 273–288.
- Al-Rimawi, F., & Kharoaf, M. (2011). Analysis of chloramphenicol and its related compound 2-amino-1-(4-nitrophenyl)propane-1,3-diol by reversed-phase high-performance liquid chromatography with UV detection. *Chromatography Research International*, *2011*, 1–6. doi:[10.4061/2011/482308](https://doi.org/10.4061/2011/482308).
- Alexander, C. M., Landsman, P. B., Teutsch, S. M., & Haffner, S. M. (2003). NCEP-defined metabolic syndrome, diabetes, and prevalence of coronary heart disease among NHANES III participants age 50 years and older. *Diabetes*, *52*, 1210–1214.
- Allen, C. H., & Hunt, W. H. (1914). *Annual report to the governor of Puerto Rico*. Washington, DC: Washington Government Printing Office.
- Allender, S., Scarborough, P., O’Flaherty, M., & Capewell, S. (2008). Patterns of coronary heart disease mortality over the 20th century in England and Wales: Possible plateaus in the rate of decline. *BMC Public Health*, *8*, 148.
- Almond, D., Bleakly, H., Ferrie, J., Mazumder, B., Rolf, K., & Troesken, W. (2009, March 12–13). *Healthy, wealthy, and wise? Physical, economic, & cognitive effects of early life conditions on later life outcomes in the US: 1915–2005*. Presented at the National Poverty Center’s conference on the Long-run Impact of Early Life Events II, Ann Arbor, MI.
- Almond, D., & Currie, J. (2010). *Human capital development before age five* (Working Paper No. 15827). Cambridge, MA: National Bureau of Economic Research.
- Almond, D., & Mazumder, B. (2011). *Health capital and the prenatal environment: The effect of Ramadan observance during pregnancy* (Working Paper 2007-22). Chicago: Federal Reserve Bank of Chicago.

- Alter, G. (2004). Height, frailty, and the standard of living: Modelling the effects of diet and disease on declining mortality and increasing height. *Population Studies*, 58(3), 265–279.
- Alter, G., & Riley, J. C. (1989). Frailty, sickness and death. *Population Studies*, 43, 25–46.
- American Diabetes Association. (n.d.). *The genetics of diabetes* [Fact sheet]. <http://www.diabetes.org/genetics.jsp>. Accessed 4 Aug 2008.
- Amos, A. F., McCarty, D. J., & Zimmet, P. (1997). The rising global burden of diabetes and its complications: Estimates and projections to the year 2010. *Diabetic Medicine*, 14(Suppl 5), S1–S85.
- Anderson, B. A., & Silver, B. D. (1986). Infant mortality in the Soviet Union: Regional differences and measurement issues. *Population and Development Review*, 12, 705–738.
- Andrade, F. (2008). *Misreported diabetes: Estimating the 'real' burden of diabetes in Latin America and the Caribbean* (Working Paper). Urbana, IL: University of Illinois-Champaign.
- Antman, K. H. (2001). Introduction: The history of arsenic trioxide in cancer therapy. *The Oncologist*, 6(suppl 2), 1–2.
- Arriaga, E. E. (1968). *New life table for Latin American populations in the nineteenth and twentieth centuries* (Population Monograph Series #3). Berkeley, CA: University of California.
- Arriaga, E. E., & Davis, K. (1969). The pattern of mortality change in Latin America. *Demography*, 6(3), 223–242.
- Ashford, B. K., & Gutiérrez Igaravidez, P. (1911). *Uncinariasis (Hookworm disease) in Porto Rico: A medical and economic problem*. Washington, DC: GPO.
- Astorga, P., Berges, A. R., & Fitzgerald, V. (2005). The standard of living in Latin America during the twentieth century. *The Economic History Review*, LVIII(4), 765–796.
- Ayala, C. J., & Bernabe, R. (2007). *Puerto Rico in the American century: A history since 1898*. Chapel Hill, NC: The University of North Carolina Press.
- Baker, M., Stabile, M., & Deri, C. (2004). What do self-reported, objective, measures of health measure? *Journal of Human Resources*, 39, 1067–1093.
- Banister, J. (1987). *China's changing population*. Stanford, CA: Stanford University Press.
- Banks, J., Marmot, M., Oldfield, Z., & Smith, J. (2006a). Disease and disadvantage in the United States and in England. *Journal of the American Medical Association*, 295(17), 2037–2045.
- Banks, J., Marmot, M., Oldfield, Z., & Smith, J. P. (2006b). *The SES health gradient on both sides of the Atlantic* (Working Paper 12674) (www.nber.org/papers/w12674). Cambridge, MA: National Bureau of Economic Research.
- Barceló, A., Aedo, C., Rajpathak, S., & Robles, S. (2003). The cost of diabetes in Latin America and the Caribbean. *Bulletin of the World Health Organization*, 81, 19–27.
- Barclay, G. W. (1954). *Colonial development and population in Taiwan*. Princeton, NJ: Princeton University Press.
- Barker, D. J. P. (1995). Fetal origins of coronary heart disease. *British Medical Journal*, 311, 171–174.
- Barker, D. J. P. (1998). *Mothers, babies and health in later life* (2nd ed.). Edinburgh, Scotland: Churchill Livingstone.
- Barker, D. J. P. (2001). The malnourished baby and infant. *British Medical Bulletin*, 60, 69–88.
- Barker, D. J. P. (2002). Commentary: Components in the interpretation of the high mortality in the country of Finnmark. *International Journal of Epidemiology*, 31, 309–310.
- Barker, D. J. P. (2005). The developmental origins of coronary heart disease. In M. Marmot & P. Elliott (Eds.), *Coronary heart disease epidemiology: From aetiology to public health* (pp. 547–567). Oxford, UK: Oxford University Press.
- Barker, D. J. P., Eriksson, J. G., Forsen, T., & Osmond, C. (2002). Fetal origins of adult disease: Strength of effects and biological basis. *International Journal of Epidemiology*, 31, 1235–1239.
- Barker, D. J. P., Forsén, T., Uutela, A., Osmond, C., & Eriksson, J. G. (2001). Size at birth and resilience to effects of poor living conditions in adult life: Longitudinal study. *British Medical Journal*, 323, 1273–1277.
- Barker, D. J. P., & Osmond, C. (1986). Infant mortality, childhood nutrition and ischaemic heart disease in England and Wales. *Lancet*, 1, 1077–1081.

- Barker, D. J. P., Thornburg, K. L., Osmond, C., Kajantie, E., & Eriksson, J. G. (2010). Beyond birthweight: The maternal and placental origins of chronic disease. *Journal of Developmental Origins of Health and Disease*, 1(6), 360–364.
- Barreto, S. M., Miranda, J. J., Fiqueroa, J. P., Schmidt, M. I., Munoz, S., Kuri-Morales, P. P., et al. (2012). Epidemiology in Latin America and the Caribbean: Current situation and challenges. *International Journal of Epidemiology*, 41(2), 557–571. doi:10.1093/ije/dys017.
- Barrientos, A. (1997). The changing face of pensions in Latin America: Design and prospects of individual capitalization pension plans. *Social Policy & Administration*, 31(4), 336–353.
- Bary, H. V. (1923). *Child welfare in the insular possessions of the United States, Part I, Porto Rico* (Bureau Publication 127). Washington, DC: US Department of Labor, Children's Bureau.
- Basu, S., Yoffe, P., Hills, N., & Lustig, R. H., with Wagner, B. (Ed.). (2013). The relationship of sugar to population-level diabetes prevalence: An econometric analysis of repeated cross-sectional data. *PLoS ONE*, 8(2), e57873. doi:10.1371/journal.pone.0057873
- Bateson, P., Barker, D., Clutton-Brock, T., Deb, D., D'Udine, B., Foley, R. A., et al. (2004). Developmental plasticity and human health. *Nature*, 430, 419–421.
- Batty, G. D., Horta, B. L., Davey Smith, G., Barros, F. C., & Victora, C. (2009). Early life diarrhoea and later blood pressure in a developing country: The 1982 Pelotas (Brazil) birth cohort study. *Journal of Epidemiology and Community Health*, 63, 163–165.
- Beaglehole, R., & Yach, D. (2003). Globalisation and the prevention and control of non-communicable disease: The neglected chronic diseases of adults. *Lancet*, 362, 903–908.
- Becerra, J. E., & Smith, J. C. (1990). Breastfeeding patterns in Puerto Rico. *American Journal of Public Health*, 80, 694–697.
- Beckett, M., Weinstein, M., Goldman, N., & Yu-Hsuan, L. (2000). Do health interview surveys yield reliable data on chronic illness among older respondents? *American Journal of Epidemiology*, 151(3), 315–323.
- Beinart, W., & Dubow, S. (Eds.). (1995). *Segregation and apartheid in twentieth century South Africa*. London: Routledge.
- Belaval, J. S. (1945). Reasons for the decline of mortality from puerperal causes in Puerto Rico during the decade 1933–1943. *Porto Rico Journal of Public Health and Tropical Medicine*, 20, 515–523.
- Beltrán-Sánchez, H., Crimmins, E. M., Teruel, G. M., & Thomas, D. (2011). Links between childhood and adult social circumstances and obesity and hypertension in the Mexican population. *Journal of Aging and Health*, 23, 1141. doi:10.1177/0898264311422255.
- Bengtsson, T., & Lindstrom, M. (2000). Childhood misery and disease in later life: The effects on mortality in old age of hazards experienced in early life, Southern Sweden, 1760–1894. *Population Studies*, 54(3), 263–277.
- Bengtsson, T., & Lindstrom, M. (2003). Airborne infectious diseases during infancy and mortality in later life in southern Sweden, 1766–1894. *International Journal of Epidemiology*, 32, 286–294.
- Bengtsson, T., & Mineau, G. P. (2009). Early life effects on socio-economic performance and mortality in later life: A full life-course approach using contemporary and historical sources. *Social Science & Medicine*, 68(9), 1561–1564.
- Bergman, H., Ferrucci, L., Guralnik, J., Hogan, D. B., Hummel, S., Karunanathan, S., et al. (2007). Frailty: An emerging research and clinical paradigm—Issues and controversies. *Journal of Gerontology*, 62A(7), 731–737.
- Bideau, A., Desjardins, B., & Pérez Brignoli, H. (1997). *Infant and child mortality in the past*. Oxford, UK: Clarendon.
- Birn, A. (2005). Uruguay on the world stage: How child health became an international priority. *American Journal of Public Health*, 95(9), 1506–1517.
- Bishop, M. D., Corbin, R., & Duncan, N. (1997). Barbados: Social development in a small island state. In S. Mehrotra & R. Jolly (Eds.), *Development with a human face: Experiences in social achievement and economic growth* (pp. 323–354). Oxford, UK: Clarendon.
- Black, R. E., Allen, L. H., Bhutta, Z. A., Caulfield, L. E., de Onis, M., Ezzati, M., et al. (2008). Maternal and child undernutrition: Global and regional exposures and health consequences. *Lancet*, 371, 243–260.

- Blackwell, D. L., Hayward, M. D., & Crimmins, E. M. (2001). Does childhood health affect chronic morbidity in later life? *Social Science & Medicine*, 52, 1269–1284.
- Bourgeois-Pichat, J. (1952). An analysis of infant mortality. *Population Bulletin*, 2, 1–14.
- Brenes, G. (2008). *The effect of early life events on the burden of diabetes mellitus among Costa Rican elderly: Estimates and projections*. Ph.D. dissertation, University of Wisconsin, Madison, WI.
- Brenes-Camacho, G., & Palloni, A. (2011, January 13–15). *Malaria survivors during early life, health at old age, and stroke mortality in Costa Rica*. Prepared for the seminar on Lifespan Extension and the Biology of Changing Cause-of-Death Profiles: Evolutionary and Epidemiological Perspectives, organized by the IUSSP Scientific Panel on Evolutionary Perspectives in Demography, Rauschholzhausen, Germany.
- Buckles, K., & Hungerman, D. M. (2008). *Season of birth and later outcomes: Old questions, new answers* (Working Paper 14573). Cambridge, MA: National Bureau of Economic Research.
- Bureau of Vital Statistics. (1926). Vital statistics in Porto Rico. *Porto Rico Review of Public Health and Tropical Medicine*, 2(4), 15–26.
- Caldwell, J. C. (1967). Population change. In W. Birmingham, I. Neustadt, & E. N. Omaboe (Eds.), *A study of contemporary Ghana* (pp. 78–110). London: George Allen & Unwin Ltd.
- Campbell, C. (1997). Public health efforts in China before 1949 and their effects on mortality: The case of Beijing. *Social Science History*, 21(2), 179–218.
- Campbell, C., & Lee, J. (2009). Long-term mortality consequences of childhood family context in Liaoning, China, 1749–1909. *Social Science & Medicine*, 68(9), 1641–1648.
- Case, A., & Paxson, C. (2010). Causes and consequences of early-life health. *Demography*, 47(Suppl), S65–S85.
- Ceesay, S. M., Prentice, A. M., Cole, T. J., Foord, F., Weaver, L. T., Poskitt, E. M., et al. (1997). Effects on birth weight and perinatal mortality of maternal dietary supplements in rural Gambia: 5 year randomized controlled trial. *British Medical Journal*, 315, 786–790.
- Centers for Disease Control and Prevention (CDC). (2001). *Achievements in public health, 1900–1999: Decline in deaths from heart disease and stroke – United States, 1900–1999*. <http://www.cdc.gov/mmwr/preview/mmwrhtml/mm4830a1.htm>. Accessed 2011.
- Centers for Disease Control and Prevention (CDC). (2011). *Protein*. <http://www.cdc.gov/nutrition/everyone/basics/protein.html>. Accessed 7 Feb 2012.
- Centers for Disease Control and Prevention (CDC). (2012). *Defining overweight and obesity*. <http://www.cdc.gov/obesity/adult/defining.html>. Accessed 2013.
- Central Intelligence Agency. (2012). *The world Factbook*. <https://www.cia.gov/library/publications/the-world-factbook/fields/2172.html>. Accessed Jan 2012.
- CEPAL/CELADE. (2001). *Latin America: Life tables 1950–2025*. Santiago, Chile: CELADE – Population Division.
- Clark, V. S. (1930). *Porto Rico and its problems*. Washington, DC: The Brookings Institution.
- Coale, A. J. (1984). *Rapid population change in China, 1952–1982*. Washington, DC: National Academy Press.
- Cole, T. J. (1993). Seasonal effects on physical growth and development. In S. J. Ulijaszek & S. S. Strickland (Eds.), *Seasonality and human ecology* (pp. 89–106). Cambridge, UK: Cambridge University Press.
- Coleman, P. G., Ruth, J.-E., & O’Hanlon, A. (2004). *Ageing and development: Theories and research*. London: A Hodder Arnold Publication.
- Cook, D. H., Axtmayer, J. H., & Dalmau, L. M. (1943). Nutritional studies of foodstuffs used in the Puerto Rican diet: A comparative study of the nutritive value of three diets of frequent use in Puerto Rico. *The Puerto Rico Journal of Public Health and Tropical Medicine*, 16(1), 3–13.
- Costa, D. L. (2002). Changing chronic disease rates and long-term declines in functional limitation among older men. *Demography*, 39(1), 119–137.
- Costa, D. L. (2005). Becoming oldest-old: Evidence from historical US data. *Genus*, LXI(1), 125–161.

- Cowie, C. C., Rust, K. F., Byrd-Holt, D. D., Eberhardt, M. S., Flegal, K. M., Engelgau, M. M., et al. (2006). Prevalence of diabetes and impaired fasting glucose in adults in the US population. *Diabetes Care*, 29(6), 1263–1268.
- Crimmins, E. M., Alley, D., Reynolds, S. L., Johnston, M., et al. (2005). Changes in biological markers of health: Older Americans in the 1990s. *Journal of Gerontology: Medical Sciences*, 60A(11), 1409–1413.
- Crimmins, E. M., & Finch, C. E. (2006). Infection, inflammation, height and longevity. *Proceedings of the National Academy of Sciences of the United States of America*, 103(2), 498–503.
- Crimmins, E., Kim, J. K., & Vasunilashorn, S. (2010). New approaches to understanding trends and differences in population health and mortality. *Demography*, 47(Suppl), S41–S64.
- Crimmins, E. M., Preston, S. H., & Cohen, B. (2010). *International differences in mortality at older ages*. Washington, DC: The National Academies Press.
- Crofton, J. (1959). Chemotherapy of pulmonary tuberculosis. *British Medical Journal*, 1(5138), 1610–1614.
- Cutler, D., Deaton, A., & Lleras-Muney, A. (2006). The determinants of mortality. *Journal of Economic Perspectives*, 20(3), 97–120.
- Cutler, D. M., & Meara, E. (2001). Changes in the age distribution of mortality over the 20th century (Working Paper 8556). Cambridge, MA: National Bureau of Economic Research.
- Cutts, F. T. (1993). *Module 7: Measles*. Geneva, Switzerland: World Health Organization.
- Daengsvang, S. (1932). An epidemiological study of hookworm disease in a rural coastal plain and a city area of Porto Rico. *Porto Rico Journal of Public Health and Tropical Medicine*, 7, 359–376.
- Davey Smith, G., Greenwood, D., Gunnell, R. D., Sweetnam, P., Yarnell, J., & Elwood, P. (2001). Leg length, insulin resistance, and coronary heart disease risk: The Caerphilly study. *Journal of Epidemiology and Community Health*, 55, 867–872.
- Davey Smith, G., Hart, C., Blane, D., & Hole, D. (1998). Adverse socioeconomic conditions in childhood and cause specific adult mortality: Prospective observational study. *British Medical Journal*, 316, 1631–1635.
- Davey Smith, G., Hart, C., Upton, M., Hole, D., Gillis, C., Watt, G., et al. (2000). Height and risk of death among men and women: Aetiological implications of associations with cardio-respiratory disease and cancer mortality. *Journal of Epidemiology and Community Health*, 54, 97–103.
- Davey Smith, G., & Lynch, J. (2004). Life course approaches to socioeconomic differentials in health. In D. Kuh & Y. Ben-Shlomo (Eds.), *A life course approach to chronic disease epidemiology* (pp. 77–115). Oxford, UK: Oxford University Press.
- Davis, D., Campbell, C. D., & Lee, J. Z. (2009, May 2). *Birth circumstances and adult health and mortality in northeast China, 1749–1909*. Paper presented at the annual meeting of the Population Association of America, Detroit, MI.
- Davis-Kean, P. E. (2005). The influence of parent education and family income on child achievement: The indirect role of parental expectations and the home environment. *Journal of Family Psychology*, 19(2), 294–304.
- Dawber, T. R., Moore, F. E., & Mann, G. V. (1957). Coronary heart disease in the Framingham study. *American Journal of Public Health*, 47(4 part 2), 4–24.
- Day, C. (2001). The rising tide of type 2 diabetes. *The British Journal of Diabetes and Vascular Disease*, 1(1), 37–43.
- De Schutter, O. (2012). *The right to an adequate diet: The agriculture-food-health nexus*. Report presented at the 19th session of the United Nations Human Rights Council.
- De Vos, S., & Palloni, A. (2001). *Living arrangements of elderly people around the World. Living arrangements of older persons around the world*. Unpublished manuscript prepared for 2005 United Nations publication.
- Deaton, A. (2007). Global patterns of income and health: Facts, interpretations, and policies. *WIDER Annual Lecture*, 10.
- Delaney, L., McGovern, M., & Smith, J. P. (2009). *From Angela's ashes to the Celtic tiger: Early life conditions and adult health in Ireland* (Discussion Paper 4548). San Francisco: Institute for the Study of Labor.

- Diaz-Briquets, S. (1981). Determinants of mortality transition in developing countries before and after the second World War: Some evidence from Cuba. *Population Studies*, 353(3), 399–411.
- Doblhammer, G. (2003). Commentary: Infectious disease during infancy and mortality in later life. *International Journal of Epidemiology*, 32, 294–295.
- Doblhammer, G. (2004). *The late life legacy of very early life*. Berlin, Germany: Springer.
- Dow, W. H. (2011, December 5). *Socioeconomic risk factors for adult mortality in a middle income country: The Costa Rican longitudinal mortality study*. Presented at the Population Studies Center of the University of Michigan, Ann Arbor, MI.
- Dyson, T. (1997). Infant and child mortality in the Indian subcontinent, 1881–1947. In A. Bideau, B. Desjardina, & H. Pérez Brignoli (Eds.), *Infant and child mortality in the past* (pp. 109–134). Oxford, UK: Clarendon.
- Dziadek, M. (2006). Genomic imprinting and epigenetic programming of fetal development. In D. M. Hodgson & C. L. Coe (Eds.), *Perinatal programming: Early life determinants of adult health and disease* (pp. 289–307). London: Taylor & Francis Medical Books.
- Earle, W. C. (1925). Malaria surveys in Porto Rico. *Porto Rico Review of Public Health and Tropical Medicine*, 1, 12–18.
- Elo, I. T., Martikainen, P., & Myrskylä, M. (2010). *Early life conditions and cause-specific mortality in Finland* (Working Paper 10-04). Philadelphia: University of Pennsylvania Population Aging Research Center.
- Elo, I. T., & Preston, S. H. (1992). Effects of early-life conditions on adult mortality: A review. *Population Index*, 58(2), 186–212.
- Eriksson, J. G., Forsen, T., Tuomilehto, J., Osmond, C., & Barker, D. J. P. (2001). Early growth and coronary heart disease in later life: Longitudinal study. *British Medical Journal*, 322(7292), 949–953.
- Eriksson, J. G., Kajantie, E., Osmond, C., Thornburg, K., & Barker, D. J. P. (2010). Boys live dangerously in the womb. *American Journal of Human Biology*, 22(3), 330–335. doi:10.1002/ajhb.20995.
- Eveleth, P. B., & Tanner, J. M. (1990). *Worldwide variation in human growth*. Cambridge, UK: Cambridge University Press.
- Fall, C. H. D., & Sachdev, H. S. (2006). Developmental origins of health and disease: Implications for developing countries. In P. Gluckman & M. Hanson (Eds.), *Developmental origins of health and disease* (pp. 456–471). Cambridge, UK: Cambridge University Press.
- Farley, J. (2004). *To cast out disease: A history of the international health division of the Rockefeller foundation (1913–1951)*. Oxford, UK: Oxford University Press.
- Fernós Isern, A. (1925). Infant mortality in Porto Rico. *Porto Rico Review of Public Health and Tropical Medicine*, 1(1), 3–10.
- Fernós Isern, A. (1928). Infantile morbidity and mortality in Porto Rico. *Porto Rico Review of Public Health and Tropical Medicine*, 3, 461–467.
- Fernós Isern, A. (1932). *Report of the commissioner of health of Porto Rico for the fiscal year ending June 30, 1931, to the governor of Porto Rico*. San Juan, Puerto Rico: Bureau of Supplies, Printing and Transportation.
- Fernós Isern, A. (1933). *Report of the commissioner of health of Puerto Rico for the fiscal year ending June 30, 1932, to the governor of Puerto Rico*. San Juan, Puerto Rico: Bureau of Supplies, Printing and Transportation.
- Fernós Isern, A. (1946). *Report of the Commissioner of health to the Hon. Governor of Puerto Rico for the fiscal year 1944–45*. San Juan, Puerto Rico: Bureau of Supplies, Printing and Transportation.
- Fernós Isern, A. (n.d.). *Report of the Commissioner of health to the Hon. Governor of Puerto Rico for the fiscal year 1942–43*. San Juan, Puerto Rico: Bureau of Supplies, Printing and Transportation.
- Fernós Isern, A., & Rodríguez Pastor, J. (1930). A survey of infant mortality in Porto Rico. *Porto Rico Review of Public Health and Tropical Medicine*, 6, 151–193.
- Ferrie, J., Rolf, K., & Troesken, W. (2009). . . . *Healthy, wealthy, and wise? Physical, economic and cognitive effects of early life conditions on later life outcomes in the US, 1915–2005*. Unpublished manuscript.

- Finch, C. E., & Crimmins, E. M. (2004). Inflammatory exposure and historical changes in human life-spans. *Science*, *305*, 1736–1739.
- Flegal, K. M., Ezzati, T. M., Harris, M. I., Haynes, S. G., Juarez, R. Z., Knowler, W. C., et al. (1991). Prevalence of diabetes in Mexican Americans, Cubans, and Puerto Ricans from the Hispanic health and nutrition examination survey, 1982–1984. *Diabetes Care*, *14*, 628–638.
- Floud, R., Fogel, R. W., Harris, B., & Chul Hong, S. (2011). *The changing body: Health, nutrition, and human development in the western world since 1700*. Cambridge, UK: Cambridge University Press.
- Floud, R., Wachter, K., & Gregory, A. (1990). *Height, health and history: Nutritional status in the United Kingdom, 1750–1980*. Cambridge, UK: Cambridge University Press.
- Fogel, R. (2004). *The escape from hunger and premature death, 1700–2100: Europe, America and the third world*. Cambridge, UK: Cambridge University Press.
- Food and Agriculture Organization of the United Nations. (1946). *World food survey*. Washington, DC: United Nations.
- Food and Agriculture Organization of the United Nations. (1984). *Agroclimatological data for Africa*. Rome, Italy: Food and Agriculture Organization of the United Nations.
- Food and Agriculture Organization of the United Nations. (1985). *Agroclimatological data for Latin America*. Rome, Italy: Food and Agriculture Organization of the United Nations.
- Food and Agriculture Organization of the United Nations. (1987). *Agroclimatological data for Asia*. Rome, Italy: Food and Agriculture Organization of the United Nations.
- Food and Agriculture Organization of the United Nations. (2004). *The state of food insecurity in the world*. Rome, Italy: Food and Agriculture Organization of the United Nations.
- Food and Agriculture Organization: Statistics Division. (2010). *Food balance sheets*. <http://faostat.fao.org/site/368/default.aspx#ancor>. Accessed Jan 2012.
- Ford, E. S., Ajani, U. A., Croft, J. B., Critchley, J. A., Labarthe, D. R., Kottke, T. E., et al. (2007). Explaining the decrease in US deaths from coronary disease, 1980–2000. *The New England Journal of Medicine*, *356*(23), 2388–2398.
- Ford, E. S., & Giles, W. G. (2003). Changes in prevalence of nonfatal coronary heart disease in the United States from 1971–1994. *Ethnicity & Disease*, *13*, 85–93.
- Forrester, T. (2006). Developmental origins of health and disease: Implications for primary intervention for cardiovascular and metabolic disease. In P. Gluckman & M. Hanson (Eds.), *Developmental origins of health and disease* (pp. 436–445). Cambridge, UK: Cambridge University Press.
- Forsdahl, A. (1978). Living conditions in childhood and subsequent development of risk factors for arteriosclerotic heart disease. *Journal of Epidemiology and Community Health*, *49*, 10–15.
- Franko, K. L., O'Connor, K. C., & Morton, S. M. B. (2009). The economics of developmental origins of health and disease: Modeling the benefit of a healthy start to life. In M. Ross (Ed.), *The economics of developmental origins of health and disease* (pp. 21–28). Basel, Switzerland: Karger.
- Gagnon, A., & Mazan, R. (2006). Influences of early life conditions on old age mortality in old Québec. *PSC Discussion Papers Series*, *20*(5), 1–19.
- Galley, C., & Wood, R. (1999). On the distribution of deaths during the first year of life. *Population: An English Selection*, *11*, 35–59.
- Gamble, D. R. (1980). The epidemiology of insulin dependent diabetes, with particular reference to the relationship of virus infection to its etiology. *Epidemiologic Reviews*, *20*(2), 49–70.
- García-Palmieri, M. R., Costas, R., Cruz-Vidal, M., Cortés-Alicea, M., Colón, A. A., Feliberti, M., et al. (1970). Risk factors and of coronary heart disease in Puerto Rico. *Circulation*, *42*, 541–549.
- Gardiner, H. M. (2007). Early environmental influences on vascular development. *Early Human Development*, *83*(12), 819–823.
- Garnier, L., Grynspan, R., Hidalgo, R., Monge, G., & Trejos, J. D. (1997). Costa Rica: Social development and heterodox adjustment. In S. Mehrotra & R. Jolly (Eds.), *Development with a human face: Experiences in social achievement and economic growth* (pp. 355–383). Oxford, UK: Clarendon.

- Garrido Morales, E. (1935). *Report of the commissioner of health of Puerto Rico to the governor of Puerto Rico for the fiscal year ending June 30, 1935*. San Juan, Puerto Rico: Bureau of Supplies, Printing and Transportation.
- Garrido Morales, E. (1936). *Report of the commissioner of health to the Hon. Governor of Puerto Rico for the fiscal year 1935–36*. San Juan, Puerto Rico: Bureau of Supplies, Printing and Transportation.
- Garrido Morales, E. (1937). *Report of the commissioner of health to the Hon. Governor of Puerto Rico for the fiscal year 1933–34*. San Juan, Puerto Rico: Bureau of Supplies, Printing and Transportation.
- Garrido Morales, E. (1939). *Report of the commissioner of health to the Hon. Governor of Puerto Rico for the fiscal year 1937–38*. San Juan, Puerto Rico: Bureau of Supplies, Printing and Transportation.
- Garrido Morales, E. (1940). *Report of the commissioner of health to the Hon. Governor of Puerto Rico for the fiscal year 1938–39*. San Juan, Puerto Rico: Bureau of Supplies, Printing and Transportation.
- Garrido Morales, E. (1941). *Report of the commissioner of health to the Hon. Governor of Puerto Rico for the fiscal year 1939–40*. San Juan, Puerto Rico: Bureau of Supplies, Printing and Transportation.
- Gavrilov, L. A., & Gavrilova, N. S. (2005, March 31–April 2). *Mortality of centenarians: A study based on the social security administration death master file*. Paper presented at the annual meeting of the Population Association of America, Philadelphia.
- Gavrilov, L. A., & Gavrilova, N. S. (2011). Season of birth and exceptional longevity: Comparative study of American centenarians, their siblings, and spouses. *Journal of Aging Research*. doi:10.4061/2011/104616.
- Gavrilov, L. A., & Gavrilova, N. S. (2012). Biodemography of exceptional longevity: Early-life and mid-life predictors of human longevity. *Biodemography and Social Biology*, 58(1), 14–39.
- Gayer, A. D., Homan, P. T., & James, E. K. (1938). *The sugar economy of Puerto Rico*. New York: Columbia University Press.
- Gertler, P., Frankenberg, E., & Karoly, L. (2000–2007). *Indonesia Family Life Surveys (IFLS)*. Los Angeles, CA: University of California-Los Angeles [distributor]. <http://www.rand.org/labor/FLS/IFLS.html>, <http://www.rand.org/labor/FLS/IFLS/access2.html>. Accessed 27 July 2007.
- Gilles, H. M., & Ball, P. A. J. (Eds.). (1991). *Human parasitic diseases* (Vol. 4). Amsterdam: Elsevier.
- Gluckman, E., & Hanson, M. (2006). *Developmental origins of health and disease*. Cambridge, UK: Cambridge University Press.
- Godfrey, K. M., & Barker, D. J. P. (2000). Fetal nutrition and adult disease. *The American Journal of Clinical Nutrition*, 71(Suppl), 1344S–1352S.
- Godoy, R. A., Goodman, E., Levins, R., Caram, M., & Seyfried, C. (2007). Adult male height in an American colony: Puerto Rico and the USA mainland compared, 1886–1955. *Economics and Human Biology*, 5, 82–99.
- Goldman, N., Lin, I., Weinstein, M., & Lin, Y. (2003). Evaluating the quality of self-reports of hypertension and diabetes. *Journal of Clinical Epidemiology*, 56, 148–154.
- Goldman, N., Weinstein, M., Chang, M.-C., Lin, H.-S., Chang, Y.-L., Lin, S.-J., Wu, S.-I. (2000). *Social Environment and Biomarkers of Aging Study (SEBAS) in Taiwan, 2000 and 2006* (ICPSR03792-v5). Ann Arbor, MI: Inter-university Consortium for Political and Social Research [distributor], <http://www.icpsr.umich.edu/icpsrweb/NACDA/studies/03792/version/5>. Accessed 12 Jan 2007.
- González, D. A., Goncalves, H. D., & Victora, C. G. (2009, November 19–22). *Effects of seasonality of birth on asthma and pneumonia in childhood and adult life in a birth cohort in southern Brazil*. Poster presented at DOHaD, Santiago, Chile.
- Gordon, T. (1964). *Glucose tolerance of adults: United States—1960–1962* (Public Health Service Publication No. 1000—Series 11, No. 2). Washington, DC: US Government Printing Office.

- Gordon, T., & Garst, C. C. (1965). *Coronary heart disease in adults: United States—1960–1962* (Public Health Service Publication Series No. 1000—Series, No. 10). Washington, DC: US Government Printing Office.
- Greenland, S., & Robins, J. (1994). Invited commentary: Ecologic studies—Biases, misconceptions, and counterexamples. *American Journal of Epidemiology*, *139*(8), 747–760.
- Gregg, E. W., Cadwell, B. L., Cheng, Y. J., Cowie, C. C., Williams, D. E., Geiss, L., et al. (2004). Trends in the prevalence and ratio of diagnosed to undiagnosed diabetes according to obesity levels in the US. *Diabetes Care*, *27*(12), 2806–2812.
- Gruber, J., & Wise, D. (1998). Social security and retirement: An international comparison. *American Economic Review*, *88*(2), 158–163.
- Guerrant, R. L., Carneiro-Filho, B. A., & Dillingham, R. A. (2003). Cholera, diarrhea, and oral rehydration therapy: Triumph and indictment. *Clinical Infectious Diseases*, *37*, 398–405.
- Guerrant, R. L., Lima, A. A. M., & Davidson, F. (2000). Micronutrients and infection: Interactions and implications with enteric and other infections and future priorities. *Journal of Infectious Diseases*, *182*(Suppl 1), S134–S138.
- Guha, S. (2001). *Health and population in South Asia*. Delhi, India: Permanent Black.
- Gunnell, D. J., Davey Smith, G., Holly, J. M. P., & Frankel, S. (1998). Leg length and risk of cancer in the Boyd Orr cohort. *British Medical Journal*, *317*(14/11), 1350–1351.
- Gurven, M., Kaplan, H., Winking, J., Rodríguez, D. E., Vasunilashorn, S., Kim, J. K., et al. (2009). Inflammation and infection do not promote arterial aging and cardiovascular disease risk factors among lean horticulturalists. *PLoS One*, *4*(8), e6590.
- Guyatt, H. L., & Snow, R. W. (2004). Impact of malaria during pregnancy on Low birth weight in Sub-Saharan Africa. *Clinical Microbiology Reviews*, *17*(4), 760–769.
- Gwatkin, D. R. (1980). Indications of change in developing country mortality trends. The end of an era? *Population and Development Review*, *6*(4), 615–644.
- Haas, S. A. (2007). The long-term effects of poor childhood health. *Demography*, *44*(1), 113–135.
- Haas, S. A. (2008). Trajectories of functional health: The ‘long arm’ of childhood health and socioeconomic factors. *Social Science & Medicine*, *66*(4), 849–861.
- Hadden, W. C., & Harris, M. I. (1987). *Prevalence of diagnosed diabetes, undiagnosed diabetes, and impaired glucose tolerance in adults 20–74 years of age* (Data from the National Health Survey Series 11, No. 237). Hyattsville, MD: US Department of Health and Human Services.
- Harper, S., Lynch, J., & Davey Smith, G. (2011). Social determinants and the decline of cardiovascular diseases: Understanding the links. *Annual Review of Public Health*, *32*, 39–69.
- Harris, B. (2000). Height and nutrition. In K. Kiple & K. C. Ornelas (Eds.), *The Cambridge world history of food* (pp. 1427–1438). New York/Cambridge, UK: Cambridge University Press.
- Harris, M. I., Flegal, K. M., Cowie, C. C., Eberhardt, M. S., Goldstein, D. E., Little, R. R., et al. (1998). Prevalence of diabetes, impaired fasting glucose, and impaired glucose tolerance in US adults. *Diabetes Care*, *21*(4), 518–524.
- Hauser, R. M., Tsai, S. L., & Sewell, W. H. (1983). A model of stratification with response error in social and psychological variables. *Sociology of Education*, *56*(1), 20–46.
- Hauspie, R. C., & Pagezy, H. (1989). Longitudinal study of growth of African babies: An analysis of seasonal variations in the average growth rate and the effects of infectious diseases on individual and average growth patterns. *Acta Paediatrica Supplement*, *350*, 37–43.
- Hawkesworth, S., et al. (2009, November 19–22). *Blood pressure and kidney function at 4.5 years of age in the offspring of the MINIMat trials: Effect of maternal food and multiple micronutrient supplementation*. Poster presented at DOHaD, Santiago, Chile.
- Hayward, M. D., & Gorman, B. K. (2004). The long arm of childhood: The influence of early-life social conditions on men’s mortality. *Demography*, *41*(1), 87–107.
- He, W., Muenchrath, M. N., & Kowal, P. (2012). *Shades of gray: A cross-country study of health and well-being of the older populations in SAGE countries, 2007–2010*. Washington, DC: US Census Bureau, US government Printing Office.
- Health and Retirement Study, Public Use Dataset. (2000–2006). *Produced and distributed by the University of Michigan with funding from the National Institute on Aging* (grant Number

- NIA U01AG009740), Ann Arbor, MI. <http://hrsonline.isr.umich.edu/>. Accessed 01 July 2008. Last accessed 2012.
- Heijmans, B. T., Tobi, E. W., Lumey, L. H., & Slagboom, P. E. (2009). The epigenome: Archive of the prenatal environment. *Epigenetics*, 4(8), 526–531.
- Heijmans, B. T., Tobi, E. W., Stein, A. D., Putter, H., Blauw, G. J., Susser, E. S., Slagboom, P. E., et al. (2008). Persistent epigenetic differences associated with prenatal exposure to famine in humans. *Proceedings of the National Academy of Sciences of the United States of America*, 105(44), 17046–17049.
- Herd, P., Hauser, R. M., Herd, P., & Sewell, W. H. *Wisconsin Longitudinal Study (WLS) [graduates, siblings, and spouses]. 1957–2005 Version 12.27 [machine-readable data file]*. Madison, WI: University of Wisconsin-Madison, WLS [distributor]. <http://www.ssc.wisc.edu/wlsresearch/>. Accessed 01 July 2012.
- Hertzman, C. (1994). The lifelong impact of childhood experiences: A population health perspective. *Daedalus*, 123(4), 167–180.
- Hill, R. B. (1926). The amount and distribution of hookworm infestation in Porto Rico. *Porto Rico Review of Public Health and Tropical Medicine*, 2(3), 3–8.
- Hinman, E. H. (1966). *World eradication of infectious diseases*. Springfield, IL: Charles C. Thomas.
- Horiuchi, S., & Preston, S. H. (1988). Age-specific growth rates: The legacy of past population dynamics. *Demography*, 25(3), 429–440.
- Howard, H. H. (1928). Hookworm disease and hookworm infestation in Porto Rico. *Porto Rico Review of Public Health and Tropical Medicine*, 4(6), 239–246.
- Huang, C., & Elo, I. T. (2009). Mortality of the oldest old Chinese: The role of early-life nutritional status, socio-economic conditions, and sibling sex-composition. *Population Studies*, 63(1), 7–20.
- Huang, C., Soldo, B., & Elo, I. (2011). Do early-life conditions predict functional health status in adulthood? The case of Mexico. *Social Science & Medicine*, 72(1), 100–107.
- Hull, T. H. (2008). Conflict and collaboration in public health: The Rockefeller foundation and the Dutch colonial government in Indonesia. In M. Lewis & K. L. MacPherson (Eds.), *Public health in Asia and the Pacific: Routledge advances in Asia-Pacific studies* (pp. 139–152). New York: Routledge.
- Hult, M., Tornhammar, P., Ueda, P., Chima, C., & Edstedt Bonamy, A.-K. (2010). Hypertension, diabetes and overweight: Looming legacies of the Biafran famine. *Pediatric Research, Suppl 1*, 8–9.
- Hunger Notes. (2011). *2011 world hunger and poverty facts and statistics*. <http://www.worldhunger.org/articles/Learn/world%20hunger%20facts%202002.htm>. Accessed 2011.
- Huxley, R. (2006). Early nutritional determinants of coronary artery disease: A question of timing? *The American Journal of Clinical Nutrition*, 84, 271–272.
- Huxley, R., Neil, A., & Collins, R. (2002). Unraveling the fetal origins hypothesis: Is there really an inverse relationship between birth weight and subsequent blood pressure? *Lancet*, 360, 659–665.
- International Labor Organization. (2010). *ISCO, International standard classification of occupations*. <http://www.ilo.org/public/english/bureau/stat/isco/index.htm>. Accessed 2012.
- Jana, N., Vasishta, K., Jindal, S. K., Khunnu, B., & Ghosh, K. (1994). Perinatal outcome in pregnancies complicated by pulmonary tuberculosis. *International Journal of Gynecology & Obstetrics*, 44, 119–124.
- Jayachandran, S., Lleras-Muney, A., & Smith, K. V. (2009). *Modern medicine and the 20th century decline in mortality: Evidence of the impact of sulfa drugs* (Working Paper 15089). Cambridge, MA: National Bureau of Economic Research.
- Jensen, R. T., & Miller, N. H. (2011). *A revealed preference approach to measuring hunger and undernutrition* (Working Paper 16555). Cambridge, MA: National Bureau of Economic Research.
- Jiménez de la Jara, J., & Bossert, T. (1995). Chile's health sector reform: Lessons from four reform periods. *Health Policy*, 32, 155–166.
- John, L., & Firth, D. (2005, February 28–March 5). *Water, watersheds, forests and poverty reduction: A Caribbean perspective*. Paper presented at 17th Commonwealth Forestry Conference, Kuala Lumpur, Malaysia.

- John, A. M., Menken, J. A., & Chowdhury, A. K. M. A. (1987). The effects of breastfeeding and nutrition on fecundability in rural Bangladesh: A hazards-model analysis. *Population Studies*, 41, 433–446.
- Johnson, R. C., & Schoeni, R. F. (2007). *The influence of early-life events on human capital, health status, and labor market outcomes over the life course* (Working Paper Series). Berkeley, CA: Institute for Research on Labor and Employment, UC Berkeley.
- Johnson, R. C., & Schoeni, R. F. (2011). Early-life origins of adult disease: National longitudinal population-based study of the United States. *American Journal of Public Health*, 101, 2317–2324.
- Joseph, K. S., & Kramer, M. S. (1996). Review of the evidence of fetal and early childhood antecedents of adult chronic disease. *Epidemiologic Review*, 18(2), 158–174.
- Kannisto, V., Christensen, K., & Vaupel, J. W. (1997). No increased mortality in later life for cohorts born during famine. *American Journal of Epidemiology*, 145, 987–994.
- Katz, S., & Akpom, C. A. (1976). Index of ADL. *Medical Care*, 14, 116–118.
- Kevan, S. M. (1979). Season of life—Season of death. *Social Science & Medicine*, 13(D), 227–232.
- Khan, M. R. (1984). Bangladesh. In H. Schubnell (Ed.), *Population policies in Asian countries: Contemporary targets, measures, and effects* (pp. 40–50). Lübeck, Germany: Dräger Foundation.
- Kim, J. K., & Crimmins, E. M. (2013, August 26–31). *Cross-country comparison of changes in health in US, England, Mexico, Taiwan, and Indonesia*. Paper to be presented at the XXVII IUSSP International Population Conference, Busan, Republic of Korea.
- King, H., Aubert, R. E., & Herman, W. H. (1998). Global burden of diabetes, 1995–2025. *Diabetes Care*, 141, 1414–1431.
- Kinsella, K., & He, W. (2009). *An aging world: 2008* (US Census Bureau, International Population Reports, P95/09-1). Washington, DC: US Government Printing Office.
- Kinsella, K., & Velkoff, V. (2001). *An aging world. US Bureau of the census*. Washington, DC: US Government Printing Office.
- Klinsberg, B. (2000). *América Latina: una región en riesgo, pobreza, inequidad e institucionalidad social* [Latin America: A region in risk, poverty, inequity, and social institutionalism]. Washington, DC: InterAmerican Development Bank.
- Kohler, I. V., & Soldo, B. J. (2005). Childhood predictors of late-life diabetes: The case of Mexico. *Social Biology*, 52(3/4), 113–131.
- Kuder, G. F., & Richardson, M. W. (1937). The theory of the estimation of test reliability. *Psychometrika*, 2, 151–160.
- Kuh, D. L. (1999). *A life course approach to women's health*. Oxford, UK: Oxford University Press.
- Kuh, D., & Ben-Shlomo, Y. (Eds.). (2004). *A life course approach to chronic disease epidemiology*. Oxford, UK: Oxford University Press.
- Kuh, D., Power, C., Blane, D., & Bartley, M. (2004). Socioeconomic pathways between childhood and adult health. In D. Kuh & Y. Ben-Shlomo (Eds.), *A life course approach to chronic disease epidemiology* (pp. 371–395). Oxford, UK: Oxford University Press.
- Kuh, D., & Wadsworth, M. (1989). Parental height: Childhood environment and subsequent adult height in a national birth cohort. *International Journal of Epidemiology*, 18(3), 663–668.
- Lannoy, J. L. (1963). *Los niveles de vida en América Latina*. Bogotá, Colombia: Oficina Internacional de Investigaciones Sociales de Feres.
- Leeder, S., Raymond, S., Greenberg, H., Liu, H., & Esson, K. (2004). *A race against time: The challenge of cardiovascular disease in developing economies*. New York: Columbia University.
- Leitch, I. (1951). Growth and health. *British Journal of Nutrition*, 5, 142–151.
- Leon, D. A., & Davey Smith, G. (2000). Infant mortality, stomach cancer, stroke and coronary heart disease: Ecological analysis. *British Medical Journal*, 320, 1705–1756.
- Leon, D. A., Davey Smith, G., Shipley, M., & Strachan, D. (1995). Adult height and mortality in London: Early life, socioeconomic confounding, or shrinkage? *Journal of Epidemiology and Community Health*, 49, 5–9.

- Lim, S. S., Vos, T., Flaxman, A. D., Danaei, G., Shibuya, K., Adair-Rohani, H., et al. (2012). A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: A systematic analysis for the global burden of disease study 2010. *The Lancet*, 380(9859), 2224–2260.
- Lin, I., Schaeffer, N. A., Seltzer, J. A., & Tuschen, K. L. (2004). Divorced parents' qualitative and quantitative reports of children's living arrangements. *Journal of Marriage and the Family*, 66, 385–397.
- López-Alonso, M. (2007). Growth with inequality: Living standards in Mexico, 1850–1950. *Journal of Latin American Studies*, 39, 81–105.
- López-Alonso, M., & Porras Condey, R. (2003). The ups and downs of Mexican economic growth: The biological standard of living and inequality, 1870–1950. *Economics and Human Biology*, 1, 169–186.
- Lundberg, O. (1991). Childhood living conditions, health status, and social mobility: A contribution to the health selection debate. *European Sociological Review*, 7(2), 149–162.
- MacPherson, K. L. (2008). Invisible borders: Hong Kong, China and the imperatives of public health. In M. Lewis & K. L. MacPherson (Eds.), *Public health in Asia and the Pacific: Routledge advances in Asia-Pacific studies* (pp. 10–54). New York: Routledge.
- Maddison, A. (2006). *The world economy*. Paris: OECD Publishing.
- Mannheim Research Institute for Economics of Ageing (MEA). (2004–2006). Survey of Health, Ageing and Retirement in Europe (SHARE). Research Data Center [distributor]. www.share-project.org/. Accessed 12 May 2011.
- Manton, K. G., & Singer, B. (1994). *What's the fuss about compression of mortality?* New York: Springer.
- Marin, C. M., Sequer, J. L., Bern, C., Freedman, D. S., Guillermo Lescano, A., Benavente, L. E., et al. (1996). Seasonal change in nutritional status among young children in an urban shanty town in Peru. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 90(4), 442–445.
- Marmot, M., & Elliott, P. (Eds.). (2005). *Coronary heart disease epidemiology: From aetiology to public health*. Oxford, UK: Oxford University Press.
- Martorell, R., Stein, A., & Schroeder, D. (2001). Early nutrition and later adiposity. *Journal of Nutrition*, 131, 874S–880S.
- Maurer, J. (2010). Height, education and later-life cognition in Latin America and the Caribbean. *Economics and Human Biology*, 8(2), 168–176.
- Max Planck Institute for Demographic Research and Department of Demography. (2007). *Human life table database* [Online]. Berkeley, CA: University of California-Berkeley. <http://www.lifetable.de/>. Accessed 2009.
- Mazumder, B., Almond, D., Park, K., Crimmins, E. M., & Finch, C. E. (2009). Lingering prenatal effects of the 1918 influenza pandemic on cardiovascular disease. *Journal of Developmental Origins of Health and Disease*, 1(1), 26–34. doi:10.1017/S2040174409990031.
- Mazzeo, V. (1993). *Mortalidad infantil en la ciudad de Buenos Aires (1856–1986)* [Infant mortality in the city of Buenos Aires (1856–1986)]. Buenos Aires, Argentina: Centro Editor de América Latina.
- McDowell, I. (2006). *Measuring health: A guide to rating scales and questionnaires*. New York: Oxford University Press, Inc.
- McEniry, M. (2008, October 23–26). *Determinants of health status among the elderly population in Latin America and the Caribbean and Asia: A comparison of past mortality regimes*. Paper presented at the 33rd annual meeting of the Social Science History Association (SSHA), Miami, FL.
- McEniry, M. (2009a). *Infant mortality during the 1920s–1940s in Puerto Rico and the health of older Puerto Rican adults* (Working Paper #2009-03). Madison, WI: Center for Demography & Ecology, University of Wisconsin-Madison.
- McEniry, M. (2009b, November 19–22). Early life exposures and adult mortality in Puerto Rico. Abstract published as part of the bi-annual meeting of the International Society for Developmental

- Origins of Health and Disease, Santiago, Chile. *Journal of Developmental Origins of Health and Disease*, 1(Suppl 1), S16.
- McEniry, M. (2009c). *Mortality decline in the twentieth century, early life conditions and the health of aging populations in the developing world* (Working Paper #2009-04). Madison, WI: Center for Demography & Ecology, University of Wisconsin.
- McEniry, M. (2010a). *Early life conditions and health disparities among aging populations in Latin America, the Caribbean, Asia and Africa* (Working Paper #2010-02). Madison, WI: Center for Demography & Ecology, University of Wisconsin-Madison.
- McEniry, M. (2010b). *The health transition and mortality among older adults in Latin America, the Caribbean and Asia* (Working Paper #2010-01). Madison, WI: Center for Demography & Ecology, University of Wisconsin-Madison.
- McEniry, M. (2011a, March 30–April 2). *Early life conditions, adult disability and mortality among aging populations in developing countries*. Presented at the Population Association of America's annual meeting, Washington, DC.
- McEniry, M. (2011b). Infant mortality, season of birth and the health of older Puerto Rican adults. *Social Science & Medicine*, 72(6), 1004–1015.
- McEniry, M. (2011c, November 19–22). *Early life exposures and adult mortality in Puerto Rico*. Unpublished manuscript. Presented at the annual conference of the International Society for Developmental Origins of Health and Disease (DOHaD), Santiago, Chile.
- McEniry, M. (2012). Early-life conditions and older adult health in low- and middle-income countries: A review. *Journal of Developmental Origins of Health and Disease*, 4(1), 10–29. doi: <http://dx.doi.org/10.1017/S2040174412000499>
- McEniry, M., Moen, S., & McDermott, J. (2013). Methods report on the compilation of the RELATE cross-national data on older adults from 20 low, middle and high income countries. Ann Arbor, MI: University of Michigan.
- McEniry, M., & Palloni, A. (2010). Early life exposures and the occurrence and timing of heart disease among the older adult Puerto Rican population. *Demography*, 47(1), 23–43.
- McEniry, M., Palloni, A., Dávila, A. L., & García, G. A. (2008). Early life exposure and its effects on the health of older Puerto Rican adults. *Journal of Gerontology: Psychological Sciences and Social Sciences*, 63B(6), S337–S348.
- Mehrotra, S., & Jolly, R. (Eds.). (1997). *Development with a human face: Experiences in social achievement and economic growth*. Oxford, UK: Clarendon.
- Meisel, A., & Vega, M. (2007). *La calidad de vida biológica en Colombia, Antropometría histórica, 1870–2009* [Quality of biological life in Colombia, historical anthropometry, 1870–2009]. Cartagena, Colombia: Centro de Estudios Económicos Regionales (CEER).
- Mesa-Lago, C. (1994). *Changing social security in Latin America: Toward alleviating the costs of economic reform*. Boulder, CO/London: Lynne Rienner Publishers, Inc.
- Migliónico, A. (2001). *República oriental del Uruguay: Tablas abreviadas de mortalidad por sexo y edad total del país, 1908 a 1999* [Republic of Uruguay: Abbreviated mortality tables by gender and age, 1908–1999]. Montevideo, Uruguay: Ministerio de Salud Pública.
- Military Vaccine Agency. (2005). *Diphtheria vaccination program questions and answers*. Office of the Army Surgeon General, US Army.
- Mitchell, B. R. (2003a). *International historical statistics: Africa, Asia & Oceania, 1750–2000* (4th ed.). New York: Palgrave Macmillan.
- Mitchell, B. R. (2003b). *International historical statistics: The Americas, 1750–2000* (5th ed.). New York: Palgrave Macmillan.
- Mitchell, B. R. (2003c). *International historical statistics: Europe, 1750–2000* (5th ed.). New York: Palgrave Macmillan.
- Monteiro, C. A., Conde, W. L., Lu, B., & Popkin, B. M. (2004). Obesity and inequities in health in the developing world. *International Journal of Obesity*, 28, 1181–1186.
- Monteiro, C. A., Moura, E. C., Conde, W. L., & Popkin, B. M. (2004). Socioeconomic status and obesity in adult populations of developing countries: A review. *Bulletin of the World Health Organization*, 82(12), 940–946.

- Monteverde, M., Noronha, K., & Palloni, A. (2009). Effect of early conditions on disability among elderly in Latin America and the Caribbean. *Population Studies*, 63, 21–35.
- Moore, S. E., Cole, T. J., Collinson, A. C., Poskitt, E. M. E., McGregor, I. A., & Prentice, A. M. (1999). Prenatal or early postnatal events predict infectious deaths in young adulthood in rural Africa. *International Journal of Epidemiology*, 28, 1088–1095.
- Moore, S. E., Fulford, A. J., Streatfield, P. K., Persson, L. A., & Prentice, A. M. (2004). Comparative analysis of patterns of survival by season of birth in rural Bangladeshi and Gambian populations. *International Journal of Epidemiology*, 33, 137–143.
- Morales Otero, P., & Pérez, M. A. (1939). Health and socio-economic studies in Puerto Rico. *Porto Rico Journal of Public Health and Tropical Medicine*, 14, 201–289.
- Morales Otero, P., Pérez, M. A., Ramírez Santos, R., Espino, R., & Marrero, M. (1937). Health and socio-economic studies in Puerto Rico: Health and socio-economic conditions on a sugar cane plantation. *Porto Rico Journal of Public Health and Tropical Medicine*, 12, 405–490.
- Morales Otero, P., Pérez, M. A., Ramírez Santos, R., Espino, R., & Marrero, M. (1939). Health and socio-economic studies in Puerto Rico: Health and socio-economic conditions in the tobacco, coffee and fruit regions. *Porto Rico Journal of Public Health and Tropical Medicine*, 14, 201–281.
- Morley, D. (1973). *Paediatric priorities in the developing world*. London: Butterworth's.
- Mountin, J. W., Pennell, E. H., & Flook, E. (1937). *Illness and medical care in Puerto Rico* (Public Health Bulletin No. 237). Washington, DC: United States Government Printing Office.
- MRC Lifecourse Epidemiology Unit. (2011). *Cohorts*. <http://www.mrc.soton.ac.uk/index.asp?page=245>. Accessed Aug 2011.
- Muñoz-Tuduri, M., & García-Moro, C. (2008). Season of birth affects short- and long-term survival. *American Journal of Physical Anthropology*, 135, 462–468.
- Murray, C. J. L., Ezzati, M., Flaxman, A. D., Lim, S., Lozano, R., Michaud, C., et al. (2012). GBD 2010: A multi-investigator collaboration for global comparative descriptive epidemiology. *The Lancet*, 380(9859), 2055–2058.
- Murray, C. J. L., & López, A. D. (Eds.). (1996). *Global health statistics: Global burden of disease and injury series* (Vol. 2). Boston: Harvard School of Public Health.
- Nagi, S. Z. (1976). An epidemiology of disability among adults in the United States. *The Milbank Memorial Fund Quarterly. Health and Society*, 54(4), 439–467.
- National Heart, Lung, and Blood Institute. (2008). *Puerto Rico Heart Health Program (PRHHP)*. <https://biolincc.nhlbi.nih.gov/studies/prhhp/>. Accessed 2011.
- National Centre for Social Research (NatCen), University College London, & Institute for Fiscal Studies (IFS) (2004–2006). *English Longitudinal Study of Ageing (ELSA)*. Economic and Social Data Service [distributor]. www.ifs.org.uk/ELSA, www.esds.ac.uk/longitudinal/access/elsa/15050.asp. Accessed 29 Mar 2011.
- National Institute on Aging (NIA). (2009). *BSR review committee report*. Washington, DC: National Institute on Aging.
- National Institute on Aging & US Department of State. (2007). *Why population aging matters: A global perspective*. Washington, DC: National Institute on Aging.
- National Research Council. (2001). *Preparing for an aging world: The case for cross-national research*. Panel on a Research Agenda and New Data for an Aging World, Committee on Population and Committee on National Statistics, Division of Behavioral and Social Sciences and Education. Washington, DC: National Academy Press.
- Newnham, J. P., & Ross, M. G. (Eds.). (2009). *Early life origins of human health and disease*. Basel, Switzerland: Karger.
- Nickel, J. C. (2005). Management of urinary tract infections: Historical perspective and current strategies: Part 2—Modern management. *The Journal of Urology*, 173, 27–32.
- Nikolic, I. A., Stanciole, A. E., & Zaydman, M. (2011). *Chronic emergency: Why NCDs matter* (HNP Discussion Paper). Washington, DC: The International Bank for Reconstruction and Development/The World Bank.
- Nitisastro, W. (1970). *Population trends in Indonesia*. Ithaca, NY: Cornell University Press.

- Noel, S. E., Newby, P. K., Ordova, J. M., & Tucker, K. L. (2009). A Traditional Rice and Beans Pattern is Associated with Metabolic Syndrome in Puerto Rican Older Adults. *The Journal of Nutrition, 139*, 1360–1367.
- O’Rand, A. M., & Hamil-Luker, J. (2005). Processes of cumulative adversity: Childhood disadvantage and increased risk of heart attack across the life course. *Journal of Gerontology: Social Sciences, 60B*, S117–S124.
- Oppen, J., & Vaupel, W. (2002). Broken limits to life expectancy. *Science, 296*, 1029–1031.
- Omran, A. R. (1971). The epidemiologic transition: A theory of the epidemiology of population change. *The Milbank Memorial Fund Quarterly, 49*(4), 509–537.
- Ortiz, P. N. (1927). *Report of the commissioner of health of Porto Rico to the governor of Porto Rico for the fiscal year ending June 30, 1926*. San Juan, Puerto Rico: Bureau of Supplies, Printing and Transportation.
- Ortiz, P. N. (1929). *Report of the commissioner of health of Porto Rico to the governor of Porto Rico for the fiscal year ending June 30, 1927*. San Juan, Puerto Rico: Bureau of Supplies, Printing and Transportation.
- Ortiz, P. N. (1931). *Report of the commissioner of health of Porto Rico for the fiscal year ending 1930 to the governor of Porto Rico*. San Juan, Puerto Rico: Bureau of Supplies, Printing and Transportation.
- Osmond, C., & Barker, D. J. P. (2000). Fetal, infant, and childhood growth are predictors of coronary heart disease, diabetes and hypertension in adult Men and women. *Environmental Health Perspectives, 108*(Suppl. 3), 545–553.
- Osmond, C., Barker, D. J. P., Winter, P. D., Fall, C. H. D., & Simmonds, S. J. (1993). Early growth and death from cardiovascular disease in women. *British Medical Journal, 307*, 1519–1524.
- Otero-Rodríguez, A., León-Muñoz, L. M., Banegas, J. R., Guallar-Castillón, P., Rodríguez-Artalejo, F., & Regidor, E. (2011). Life-course socioeconomic position and change in quality of life among older adults: Evidence for the role of a critical period, accumulation of exposure and social mobility. *Journal of Epidemiology and Community Health, 65*(11), 964–971.
- Painter, R. C., de Rooij, S. R., Bossuyt, P. M., Simmer, T. A., Osmond, C., & Barker, D. J. P. (2006). Early onset of coronary artery disease after prenatal exposure to the Dutch famine. *The American Journal of Clinical Nutrition, 84*, 322–327.
- Palloni, A. (1981). Mortality in Latin America: Emerging patterns. *Population and Development Review, 7*(4), 623–649.
- Palloni, A. (2002). Living arrangements of older persons [Special Issue]. *Population Bulletin of the United Nations, 42*(43), 54–110.
- Palloni, A. (2006). Luck, wallets, and the enduring effects of childhood health. *Demography, 43*(4), 587–615.
- Palloni, A. (2011, June 14). *Mortality and longevity in Latin America: Progress and setbacks in the last 200 years*. Presented at the Center for Demography and Ecology Summer Seminar, Madison, WI.
- Palloni, A., McEniry, M., Guend, H., Davila, A. L., Garcia, A., Mattei, H., et al. (2004). *Health among Puerto Ricans: Analysis of a new data set* (Working paper 2004–11). Madison, WI: Center for Demography and Ecology, University of Wisconsin-Madison.
- Palloni, A., McEniry, M., Dávila, A. L., & García Gurucharri, A. (2005). The influence of early conditions on health status among elderly Puerto Ricans. *Social Biology, 52*(3/4), 132–164.
- Palloni, A., McEniry, M., Wong, R., & Peláez, M. (2006). The tide to come: Elderly health in Latin America and the Caribbean. *Journal of Aging and Health, 18*(2), 180–206.
- Palloni, A., McEniry, M., Wong, R., & Peláez, M. (2007, August 31–September 2). *Ageing in Latin America and the Caribbean: Implications of past mortality* (pp. 253–284). Proceedings of the United Nations Expert Group Meeting on Social and Economic Implications of Changing Population Age Structures, Mexico City, Mexico.
- Palloni, A., Milesi, C., White, R. G., & Turner, A. (2009). Early childhood health, reproduction of economic inequalities and the persistence of health and mortality differentials. *Social Science & Medicine, 68*(9), 1574–1582.
- Palloni, A., Noronha, K., & McEniry, M. (2009). *Roadblocks for sustained improvements in life expectancy in Latin American and the Caribbean* (Working Paper 2009-02). Madison, WI: Center for Demography and Ecology, University of Wisconsin-Madison.

- Palloni, A., & Pinto-Aguirre, G. (2004, April 1–3). *One-hundred years of mortality in Latin America and the Caribbean: The fragile path from hunger to longevity*. Paper presented at the Population Association of America Meetings, Boston, MA.
- Palloni, A., Pinto-Aguirre, G., & Peláez, M. (2002). Demographic and health conditions of ageing in Latin America and the Caribbean. *International Journal of Epidemiology*, *31*, 762–771. doi:10.1093/ije/31.4.762.
- Palloni, A., & Wyrick, R. (1981). Mortality decline in Latin America: Changes in the structures of causes of deaths, 1950–1975. *Social Biology*, *28*(3–4), 187–216.
- Palmer, S. (2003). *From popular medicine to medical populism: Doctors, healers and public power in Costa Rica, 1800–1940*. Durham, NC: Duke University Press.
- Pardoko, H. (1984). Indonesia. In H. Schubnell (Ed.), *Population policies in Asian countries: Contemporary targets, measures, and effects* (pp. 51–67). Lübeck, Germany: Dräger Foundation.
- Patterson, K. D. (1979). Health in urban Ghana: The case of Accra 1900–1940. *Social Science & Medicine*, *138*, 251–268.
- Patterson, K. D. (1981). *Health in colonial Ghana: Disease, medicine and socio-economic change 1900–1955*. Waltham, MA: Crossroads Press.
- Paul, H. H. (1964). *The control of diseases* (2nd ed.). Baltimore: Williams & Wilkins.
- Payne, G. C., Berríos, M. B., & Martínez Rivera, E. (1929). Heights and weights of children in three communities of Porto Rico. *Porto Rico Journal of Public Health and Tropical Medicine*, *5*(1), 344–363.
- Peck, M. N., & Lundberg, O. (1995). Short stature as an effect of economic and social conditions in childhood. *Social Science & Medicine*, *41*(5), 733–738.
- Pembrey, M. E., Bygren, L. O., Kaati, G., Edvinsson, S., Northstone, K., Sjöström, M., et al. (2006). Sex-specific, male-line transgenerational responses in humans. *European Journal of Human Genetics*, *14*(2), 159–166.
- Pérez, M. A. (1926). Vital statistics in Porto Rico. *Porto Rico Review of Public Health and Tropical Medicine*, *1*(9), 3–8.
- Pérez, M. A. (1941). Health and socio-economic studies in Puerto Rico. *Porto Rico Journal of Public Health and Tropical Medicine*, *16*, 547–615.
- Perloff, H. S. (1952). The United States and the economic development of Puerto Rico. *Journal of Economic History*, *12*(1), 45–59.
- Phelps, E. B. (1928). Diarrhea and enteritis in Porto Rico: An epidemiological study. *Porto Rico Review of Public Health and Tropical Medicine*, *3*(9), 345–357.
- Pickles, A., Maughan, B., & Wadsworth, M. (2007). *Epidemiological methods in lifecourse research*. Berkshire, UK: Open University Press. eScholarID:4d140.
- Pluijm, S. M., Bardage, C., Nikula, S., Blumstein, T., Jylhä, M., Minicuci, N., et al. (2005). A harmonized measure of activities of daily living was a reliable and valid instrument for comparing disability in older people across countries. *Journal of Clinical Epidemiology*, *58*(10), 1015–1023.
- Popkin, B. M. (2006). Global nutrition dynamics: The world is shifting rapidly toward a diet linked with noncommunicable diseases (NCDs). *American Journal of Clinical Nutrition*, *84*, 289–298.
- Popkin, B. M. (1998). The nutrition transition and its health implications in lower-income countries. *Public Health Nutrition*, *1*(1), 5–21.
- Popkin, B. M., Horton, S., & Kim, S. (2001). *The nutritional transition and diet-related chronic diseases in Asia: Implications for prevention* (Food Consumption and Nutrition Division Discussion Paper No. 105). Washington, DC: International Food Policy Research Institute.
- Power, C., Kuh, D., & Morton, S. (2013). From developmental origins of adult disease to life course research on adult disease and aging: Insights from birth cohort studies. *Annual Review of Public Health*, *34*, 7–28.
- Prentice, A. M. (2009). Regional case studies-Africa. In S. C. Kalhan, A. M. Prentice, & C. S. Yajnik (Eds.), *Emerging societies-coexistence of childhood malnutrition and obesity*. Nestle

- Nutrition Workshop Series Pediatric Program* (Vol. 63, pp. 33–46). Basel, Switzerland: Karger Medical and Scientific Publishers.
- Prentice, A. M., & Cole, T. J. (1994). Seasonal changes in growth and energy status in the third world. *Proceedings of the Nutrition Society*, 53, 509–519.
- Pressat, R. (1961). *Demographic analysis: Methods, results, applications*. Chicago: Aldine-Atherton.
- Preston, S. (1976). *Mortality patterns in national populations with special reference to recorded causes of death*. New York: Academic.
- Preston, S. (1980). Causes and consequences of mortality change in less developed countries in the twentieth century. In R. Easterlin (Ed.), *Population and economic change in developing countries* (pp. 289–360). Chicago: University of Chicago Press.
- Preston, S. H., Hill, M. E., & Drevenstedt, G. L. (1998). Childhood conditions that predict survival to advanced ages among African-Americans. *Social Science and Medicine*, 47(9), 1231–1246.
- Prokhorov, B. B. (2005–2009). *The public health in Russia during the last 100 years (1897–1997)*. <http://www.rus-stat.ru/eng/index.php?vid=1&year=2000&id=58>. Accessed 2011.
- Puerto Rican Elderly: Health Conditions (PREHCO). (2007). *Waves 1–2, 2003–2007. Principal Investigators: Alberto Palloni and Ana Luisa Dávila*. Madison, WI: Data and Information Services Center, University of Wisconsin-Madison [distributor]. www.disc.wisc.edu/archive/prehco/. Accessed 17 July 2007.
- Raghunathan, T. E., Reiter, J. P., & Rubin, D. B. (2003). Multiple imputation for disclosure limitation. *Journal of Official Statistics*, 19, 1–16.
- Raghunathan, T. E., Solenberger, P. W., & Van Hoewyk, J. (2007). *IVEware: Imputation and variance estimation* [Computer software]. www.isr.umich.edu/src/smp/ive/. Accessed 31 July 2007.
- Rahman, O., Menken, J., Foster, A., & Gertler, P. (1999). *Matlab [Bangladesh] Health and Socioeconomic Survey (MHSS), 1996* (ICPSR02705-v5). Ann Arbor, MI: Inter-university Consortium for Political and Social Research [distributor]. <http://www.rand.org/labor/FLS/MHSS.html>, <http://www.icpsr.umich.edu/icpsrweb/NACDA/studies/02705/version/5>. Accessed 25 Apr 2008.
- Ramasubban, R. (2008). History of public health in modern India: 1857–2005. In M. Lewis & K. L. MacPherson (Eds.), *Public health in Asia and the pacific: Routledge advances in Asia-pacific studies* (pp. 87–105). New York: Routledge Taylor & Francis Group.
- Ranis, G., & Kosack, S. (2004). *Growth and human development in Cuba's transition* (Institute for Cuban and Cuban-American studies). Miami, FL: University of Miami.
- Rasmussen, K. M. (2001). The “fetal origins” hypothesis: Challenges and opportunities for maternal and child nutrition. *Annual Review of Nutrition*, 21, 73–95.
- Ravelli, A. C., van der Meulen, J. H., Michels, R. P., Osmond, C., Barker, D. J., Hales, C. N., & Bleker, O. P. (1998). Glucose tolerance in adults after prenatal exposure to the Dutch famine. *Lancet*, 351, 173–177.
- Rayco-Solon, P., Moore, S., Fulford, A. J., & Prentice, A. M. (2004). Fifty-year mortality trends in three rural African villages. *Tropical Medicine & International Health*, 9(11), 1151–1160.
- Reddy, K. S. (2009). Regional case studies-India. In S. C. Kalhan, A. M. Prentice, & C. S. Yajnik (Eds.), *Emerging societies-coexistence of childhood malnutrition and obesity. Nestle Nutrition Workshop Series Pediatric Program* (Vol. 63, pp. 15–24). Basel, Switzerland: Karger Medical and Scientific Publishers.
- Regidor, E., Guitiérrez-Fisac, J. L., Calle, M. E., Navarro, P., & Domínguez, V. (2002). Infant mortality at time of birth and cause-specific adult mortality among residents of the region of Madrid born elsewhere in Spain. *International Journal of Epidemiology*, 31, 368–374.
- Research on Early Life and Aging: Trends and Effects (RELATE) Principal Investigator: Mary C McEniry. (2013). Produced and distributed by the University of Michigan: Institute for Social Research, ICPSR [distributor]. Release date: June 12, 2013. <http://www.icpsr.umich.edu/icpsrweb/DSDR/studies/34241>.

- Richards, A. A., Fulford, A. J., & Prentice, A. M. (2009, November 19–22). *Birth weight, season of birth and postnatal growth do not predict levels of low-grade systemic inflammation in Gambian adults*. Poster presented at DOHaD, Santiago, Chile.
- Rigau Pérez, J. G. (2000). La Salud en Puerto Rico en el Siglo XX [Health in Puerto Rico in the twentieth century]. *Puerto Rican Health Sciences Journal*, 19(4), 357–368.
- Riley, J. C. (2005a). *Bibliography of works providing estimates of life expectancy at birth and estimates of the beginning period of health transitions in countries with a population in 2000 of at least 400,000*. <http://www.lifetable.de/RileyBib.htm>. Accessed 2009.
- Riley, J. C. (2005b). The timing and pace of health transitions around the world. *Population and Development Review*, 31(4), 741–764.
- Riley, J. (2008). *Low income, social growth and good health: A history of twelve countries*. New York: Milbank Memorial Fund.
- Riosmena, F., & Wong, R. (2008, April 17–19). *Health selectivity and SES-health gradients in Mexico-US migration and return: A bi-national perspective on older adults*. Paper presented at the annual meeting of the Population Association of America, New Orleans, LA.
- Robinson, U., & Suárez, R. M. (1947). The nutritional status of a Puerto Rican Rural Community in relation to its dietary intake. *Porto Rico Journal of Public Health and Tropical Medicine*, 23(1), 100–134. Table 15.
- Rockwood, K., & Mitnitski, A. (2007). Frailty in relation to the accumulation of deficits. *Journal of Gerontology*, 62A(7), 722–727.
- Rodríguez de Romo, A. C., & Rodríguez de Pérez, M. E. R. (1998). Historia de la salud pública en México: siglos XIX y XX [History of public health in Mexico: 19th and 20th century]. *História, Ciências, Saúde-Manguinhos*, 5(2), 293–310.
- Rosario, J. C. (1930). The Porto Rican peasant and his historical antecedents. In V.S. Clark (Au.), *Porto Rico and its problems* (Appendix A). Washington, DC: The Brookings Institution.
- Rose, G. A. (1962). The diagnosis of ischaemic heart pain and intermittent claudication in field surveys. *Bulletin of the World Health Organization*, 27, 645–658.
- Rose, G., Reid, D. D., Hamilton, P. J. S., McCartney, P., Keen, H., & Jarrett, R. J. (1977). Myocardial ischaemia, risk factors and death from coronary heart disease. *Lancet*, 309(8003), 105–109.
- Roseboom, T. J., van der Meulen, J. H., Osmond, C., Barker, D. J., Ravelli, A. C., Schroeder-Tanka, J. M., et al. (2000). Coronary heart disease after prenatal exposure to the Dutch famine, 1944–45. *Heart*, 84, 595–598.
- Rosero-Bixby, L. (1991). Socioeconomic development, health interventions and mortality decline in Costa Rica. *Scandinavian Journal of Social Medicine Supplement*, 46, 33–42.
- Rosero-Bixby, L., & Caamaño, H. (1984). *Tablas de vida de Costa Rica*. San José, Costa Rica: Asociación Demográfica Costarricense.
- Rosero-Bixby, L., & Dow, W. H. (2009). Surprising SES gradients in mortality, health, and biomarkers in a Latin American population of adults. *Journal of Gerontology: Social Sciences*, 64B(1), 105–117.
- Rosero-Bixby, L., Fernández, X., & Dow, W.H. (2003). *CRELES: Costa Rican Longevity and Healthy Aging Study, 2005 (Costa Rica Estudio de Longevidad y Envejecimiento Saludable)* (ICPSR26681-v2). Ann Arbor, MI: Inter-university Consortium for Political and Social Research; Costa Rica: The Central American Population Center [distributor]. www.icpsr.umich.edu/icpsrweb/NACDA/studies/26681/version/2. Accessed 09 July 2007.
- Royston, P. (2004). Multiple imputation of missing values. *The Stata Journal*, 4(3), 227–241.
- Royston, P. (2006). *ICE: Stata module for multiple imputation of missing values*. Chestnut Hill, MA: Statistical Software Components, Boston College Department of Economics.
- Rubin, D. (1987). *Multiple imputation for nonresponse in surveys*. New York: Wiley.
- SABE. (2003). *Salud y Bienestar en el Adulto Mayor, SABE, version No. 1, restricted circulation data set*. Produced and distributed by the Pan American Health Organization (PAHO) and the Center for Demography and Health of Aging (CDHA) with the support of the National Institute of Aging, R03 AG15673.

- Scarano, F. A., & Curtis White, K. J. (2007). A window into the past: Household composition and distribution in Puerto Rico, 1910 and 1920. *Caribbean Studies*, 35(2), 115–154.
- Scazufca, M., Menezes, P. R., Araya, R., Di Rienzo, V. D., Almeida, O. P., Gunnell, D., et al. (2008). Risk factors across the life course and dementia in a Brazilian population: Results from the Sao Paulo Ageing & Health Study (SPAH). *International Journal of Epidemiology*, 37, 879–890.
- Schmidhuber, J., & Shetty, S. (2005, April 21–22). *The nutrition transition to 2030: Why developing countries are likely to bear the major burden*. Plenary paper presented at the 97th Seminar of the European Association of Agricultural Economists, University of Reading, Reading, UK.
- Schmidt, I., Jorgensen, M., & Michaelsen, K. (1995). Height of conscripts in Europe: Is postneonatal mortality a predictor? *Annals of Human Biology*, 22(1), 57–67.
- Schooling, C. M., Jiang, C., Lam, T. H., Zhang, W., Cheng, K. K., & Leung, G. M. (2011). Parental death during childhood and adult cardiovascular risk in a developing country: The Guangzhou biobank cohort study. *PLoS One*, 6(5), e19675. doi:10.1371/journal.pone.0019675.
- Schroeder, D. G., Martorell, R., & Flores, R. (1999). Infant and child growth and fatness and fat distribution in Guatemalan adults. *American Journal of Epidemiology*, 149(2), 177–185.
- Scrimshaw, N. S. (1968). *Interaction of nutrition and infection*. New York: World Health Organization.
- Scrimshaw, N. S. (1997). Nutrition and health from womb to tomb. *Food and Nutrition Bulletin*, 18(1), 1–19.
- Shlaes, D. M., Projan, S. J., & Edwards, J. E., Jr. (2004). Antibiotic discovery: State of the state. *ASM News*, 70(6), 275–281.
- Sichieri, R., Siqueira, K. S., & Moura, A. S. (2000). Obesity and abdominal fatness associated with undernutrition early in life in a survey in Rio de Janeiro. *International Journal of Obesity*, 24, 614–618.
- Simondon, K. B., Elguero, E., Marra, A., Diallo, A., Aaby, P., & Simondon, F. (2004). Quarter of birth is not associated with risk of early adult death in rural Senegal. *International Journal of Epidemiology*, 33, 130–136.
- Smith, J. P. (2005). *Unraveling the SES health connection* (IFS Working Papers W04/02). London: Institute for Fiscal Studies.
- Smith, L. A., Branch, L. G., & Scherr, P. A. (1990). Short-term variability of measures of physical function in older people. *Journal of American Geriatric Society*, 38, 993–998.
- Soares, R. R. (2007). On the determinants of mortality reductions in the developing world. *Population and Development Review*, 33(2), 247–287.
- Stanner, S. A., Bulmer, K., Andr es, C., Lantseva, O. E., Borodina, V., Poteen, V. V., et al. (1997). Does malnutrition in utero determine diabetes and coronary heart disease in adulthood? Results from the Leningrad siege study, a cross sectional study. *British Medical Journal*, 315, 1342–1348.
- Stein, A. D., Wang, M., Martorell, R., Norris, S. A., Adair, L. S., Bas, I., et al. (2010). Growth patterns in early childhood and final attained stature: Data from five birth cohorts from Low- and middle-income countries. *American Journal of Human Biology*, 22, 353–359.
- Steward, J. H., Manners, R. A., Wolf, E. R., Padilla Seda, E., Mintz, S. W., & Scheele, R. L. (1956). *The people of Puerto Rico: A study in social anthropology*. Champaign, IL: University of Illinois Press.
- Stump, T. E., Clark, D. O., Johnson, R. J., & Wolinsky, F. D. (1977). The structure of health status among Hispanic, African American, and white older adults [Special Issue]. *Journals of Gerontology*, 52B, 49–60.
- Soares, R.R. (2007). On the determinants of mortality reductions in the developing world. *Population and Development Review*, 33(2), 247–287.
- Soldo, B.J., Wong, R., Palloni, A., & Instituto Nacional de Estadística, Geografía e Informática (INEGI). Mexican Health and Aging Study (MHAS). (2013). Mexican Health and Aging Study [distributor]. www.mhas.pop.upenn.edu/english/home.htm; new website as of 2013 is: www.mhasweb.org. Accessed 29 May 2008.

- Suárez, R. M. (1943). *Studies of the nutritional problem of Puerto Rico: 1. Vitamin A deficiency in relation to dark adaptation and ocular manifestations* (p. 65, table 1). From the Department of Clinical Medicine of the School of Tropical Medicine and the Mimiya Hospital. *Porto Rico Journal of Public Health and Tropical Medicine*, 19, 62–80.
- Svensson, P., Broström, G., & Oris, M. (2004, November 18–21). *Early-life conditions and social mobility in nineteenth century Belgium and Sweden*. Paper presented at the Social Science History Association meeting, Chicago.
- Tare, M., Parkington, H. C., & Morley, R. (2006). Vitamin D in pregnancy and offspring health. In M. E. Wintour & J. A. Owens (Eds.), *Early life origins of health and disease* (pp. 195–203). New York: Springer Science + Business Media.
- Thorpe, L. E., Frieden, T. R., Laserson, K. F., Wells, C., & Khatri, G. R. (2004). Seasonality of tuberculosis in India: Is it real and what does it tell us? *Lancet*, 364(9445), 1613–1614.
- Todar, K. (2008). Pathogenic *E. coli* (p. 1). *Todar's online textbook of bacteriology*. <http://www.textbookofbacteriology.net/e.coli.html>. Accessed Aug 2011.
- Tomkins, A. (1993). Environment, season and infection. In S. J. Ulijaszek & S. S. Strickland (Eds.), *Seasonality and human ecology* (pp. 123–134). New York: Cambridge University Press.
- Toro, H. (2008). Inequality in Puerto Rico. In *ReVista: Harvard review of Latin America*. http://dev.drclas.harvard.edu/files/revista_sp08_web.pdf. Accessed Jan 2012.
- Trowbridge, F., & Newton, L. H. (1979). Seasonal changes in malnutrition and diarrheal disease among preschool children in El Salvador. *The American Journal of Tropical Medicine and Hygiene*, 28(11), 136–141.
- Trowell, H., & Burkitt, D. (1985). *Western diseases: Their causes and prevention*. London: Edward Arnold.
- Tucker, K. L., Mattei, J., Noel, S. E., Collado, B. M., Mendez, J., Nelson, J., et al. (2010). The Boston Puerto Rican health study, a longitudinal cohort study on health disparities in Puerto Rican adults: Challenges and opportunities. *BMC Public Health*, 10, 107.
- Ubink-Veltmaat, L. J., Bilo, H. J. G., Groenier, K. H., Houweling, S. T., Rischen, R. O., & Meyboom-de Jong, B. (2003). Prevalence, incidence and mortality of type 2 diabetes mellitus revisited: A prospective population-based study in the Netherlands. *European Journal of Epidemiology*, 18(8), 793–800.
- United Nations. (2002). *World population ageing 1950–2050*. New York: United Nations.
- United Nations Department of Economic and Social Affairs. (2010). *Population division, population estimates and projections section: Population*. http://esa.un.org/unpd/wpp/unpp/panel_population.htm. Accessed 2010.
- United Nations Development Program. (2002). *Human development report*. New York: United Nations.
- United Nations Educational, Scientific and Cultural Organization (UNESCO). (1953). *Progress in literacy in various countries: A preliminary statistical study of available census data since 1900*. Paris: United Nations Educational, Scientific and Cultural Organization.
- UNESCO. (1965). *Statistical yearbook*. Paris: United Nations Educational, Scientific and Cultural Organization.
- UNESCO. (1977). *Statistics of educational attainment and illiteracy, 1945–1974* (UNESCO Statistical Reports and Studies, No. 22). Paris: United Nations Education, Scientific and Cultural Organization.
- United Nations Statistical Office & the Department of Economic and Social Affairs. (1973). *Statistical yearbook* (24th Issue). New York: United Nations Publications.
- United Nations Statistical Office & The Department of Economic and Social Affairs. (1960). *Statistical yearbook* (12th Issue). New York: United Nations Publications.
- United Nations Statistical Office & The Department of Economic and Social Affairs. (1962). *Statistical yearbook* (14th Issue). New York: United Nations Publications.
- United States Census Bureau. (1932). *Outlying territories and possessions. Number and distribution of inhabitant composition and characteristics of the population, occupations, unemployment and agriculture*. Washington, DC: US Government Printing Office.

- United States Environmental Protection Agency. (2010). *Basic information about E. coli O157:H7 in drinking water*. <http://water.epa.gov/drink/contaminants/basicinformation/ecoli.cfm#three>. Accessed 2011.
- University of North Carolina-Chapel Hill, The Carolina Population Center, National Institute of Nutrition and Food Safety, & Chinese Center for Disease Control and Prevention. *China Health and Nutrition Survey (CHNS) (2000–2006)*. Chapel Hill, NC: University of North Carolina [distributor]. www.cpc.unc.edu/projects/china/data. Accessed 11 Feb 2010.
- Van Buren, S., Boshuizen, H., & Knook, D. (1999). Multiple imputation of missing blood pressure covariates in survival analysis. *Statistics in Medicine*, 18, 681–694.
- van den Berg, G. J., Doblhammer, G., & Christensen, K. (2009). Exogenous determinants of early-life conditions, and mortality later in life. *Social Science & Medicine*, 68, 1591–1598.
- van den Berg, G. J., Lindeboom, M., & Portrait, F. (2006). *Early childhood conditions and longevity: The effect of food shortages, infectious diseases and economic conditions in the 19th century in the Netherlands*. Unpublished manuscript. Amsterdam: Free University.
- Van Ewijk, R. J. G., Painter, R. C., & Roseboom, T. J. (2013). Associations of prenatal exposure to Ramadan with small stature and thinness in adulthood: Results from a large Indonesian population-based study. *American Journal of Epidemiology*, 177(8), 729–736. doi:10.1093/aje/kwt023.
- Van Tonder, J. L., & Van Eeden, I. J. (1975). *Abridged life tables for all the population groups in the Republic of South Africa (1921–1970)*. Pretoria, South Africa: Institute for Sociological Demographic and Criminological Research.
- Vaupel, J., Manton, K., & Stallard, E. (1979). The impact of heterogeneity in individual frailty on the dynamics of mortality. *Demography*, 16, 439–454.
- Vázquez Calzada, J. L. (1988). *La población de Puerto Rico y su trayectoria histórica* [The population of Puerto Rico and its historical trajectory]. Río Piedras, Puerto Rico: Escuela Graduada de Salud Pública, Recinto de Ciencias Médicas, Universidad de Puerto Rico.
- Vázquez Calzada, J. L., Morales, N. R., & Janer, J. L. (1963). *Tablas de vida abreviadas para Puerto Rico: 1894–1961*. San Juan, Puerto Rico: Universidad de Puerto Rico.
- Vázquez Calzada, J. L., & Rivera Acevedo, S. (1982). The seasonal pattern of live births in Puerto Rico. *Puerto Rico Health Sciences Journal*, 1(4), 167–171.
- Veena, S. R., Krishnaveni, G. V., Wills, A. K., Kurpad, A. V., Muthayya, S., Hill, J. C., Karat, S. C., Nagarajaiah, K. K., Fall, C. H. D., & Srinivasan, K. (2009, November 19–22). *Association of birthweight and head circumference at birth to cognitive performance in 9–10 year old children in South India: Prospective birth cohort study*. Poster presented at DOHaD, Santiago, Chile.
- Victora, C. G., Adair, L., Fall, C., Hallal, P. C., Martorell, R., Richter, L., et al. (2008). Maternal and child undernutrition: Consequences for adult health and human capital. *Lancet*, 371, 340–357.
- Waalder, H. T. (1984). Height, weight, and mortality: The Norwegian experience. *Acta Medica Scandinavica*, 679(Suppl), 1–56.
- Wadsworth, M., Hardy, R., Paul, A., Marshall, S., & Cole, T. (2002). Leg and trunk length at 43 years in relation to childhood health, diet and family circumstances: Evidence from the 1946 national birth cohort. *International Journal of Epidemiology*, 31, 383–390.
- Wadsworth, M., & Kuh, D. (1997). Childhood influences on adult health: A review of recent work from the British 1946 national birth cohort study, the MRC National Survey of Health and Development. *Paediatric and Perinatal Epidemiology*, 11, 2–20.
- Walker, J. B., & Kerridge, D. (1961). *Diabetes in an English community: A study of its incidence and natural history*. Leicester, UK: Leicester University Press.
- Waterland, R. A. (2006). Critical experiments to determine if early nutritional influences on epigenetic mechanisms cause metabolic imprinting in humans. In M. E. Wintour & J. A. Owens (Eds.), *Early life origins of health and disease* (pp. 195–203). New York: Springer Science + Business Media.

- Wegman, M. E., Fernández Marchante, R., & Kramer, M. (1942). Infant mortality and infant feeding in Puerto Rico. *Porto Rico Journal of Public Health and Tropical Medicine*, 17, 228–245.
- Weinstein, M., & Goldman, N. (2003). *Social environment and biomarkers of aging study (SEBAS) in Taiwan, 2000* (ICPSR version). Washington, DC/Princeton, NJ: Maxine Weinstein, Georgetown University, Graduate School of Arts and Sciences, Center for Population and Health/Noreen Goldman, Princeton University, Office of Population Research [producers], 2000. Ann Arbor, MI: Inter-university Consortium for Political and Social Research [distributor], 2003.
- Wen, M., & Gu, D. (2011). The effects of childhood, adult, and community socioeconomic conditions on health and mortality among older adults in china. *Demography*, 48(1), 153–181.
- West India Royal Commission. (1945). *West India Royal Commission report*. London: His Majesty's Stationery Office.
- Whitelaw, E., & Garrick, D. (2006). Epigenetic mechanisms. In P. Gluckman & M. Hanson (Eds.), *Developmental origins of health and disease* (pp. 62–74). New York: Cambridge University Press.
- Wickrama, K. A. S., Conger, R. D., & Abraham, W. T. (2005). Early adversity and later health: The intergenerational transmission of adversity through mental disorder and physical illness. *Journal of Gerontology: Social Sciences*, 60B, S125–S129.
- Widjojo, N. (1970). *Population trends in Indonesia*. Ithaca, NY: Cornell University Press.
- Wilkerson, H. L. C., & Krall, L. P. (1947). Diabetes in a New England town; study of 3,516 persons in Oxford, Mass. *JAMA: The Journal of the American Medical Association*, 135, 209–216.
- Wilkinson, R. G. (1996). *Unhealthy societies: The afflictions of inequality*. London: Routledge.
- Wilkinson, R. G., & Marmot, M. (Eds.). (2003). *Social determinants of health: The solid facts* (2nd ed.). Copenhagen, Denmark: World Health Organization.
- Wolf, B. (1947). Studies on infant mortality. *British Journal of Social Medicine*, 2, 73–125.
- Wolf, S. H., & Aron, L. (Eds.). (2013). *US health in international perspective: Shorter lives, poorer health*. Washington, DC: National Academies Press.
- World Bank. (2011). *How we classify countries*. <http://data.worldbank.org/about/country-classifications>. Accessed 2008.
- World Bank. (2012). *GINI index*. <http://data.worldbank.org/indicator/SI.POV.GINI/countries?display=default>. Accessed 2012.
- World Health Organization (WHO). (2000a). *Obesity: Preventing and managing the global epidemic. Report of a WHO consultation*. Geneva, Switzerland: World Health Organization.
- World Health Organization (WHO). (2000b). *The world health report 2000. Health systems: Improving performance*. Geneva, Switzerland: World Health Organization.
- World Health Organization (2002). *World mortality I 2000: Life expectancies for 191 countries*.
- World Health Organization (WHO). (2005). *Preventing chronic disease: A vital investment: WHO global report*. Geneva, Switzerland: World Health Organization.
- World Health Organization (WHO). (2006). *Promoting optimal fetal development: Report of a technical consultation*. Geneva, Switzerland: WHO.
- World Health Organization (WHO). (2008). *Fact sheet No. 312: Diabetes*. <http://www.who.int/mediacentre/factsheets/fs312/en/>. Accessed 5 Jan 2010.
- World Health Organization (WHO). (2011a). *Initiative for vaccine research (IVR): Hookworm disease*, paragraph 3. http://www.who.int/vaccine_research/diseases/soa_parasitic/en/index2.html. Accessed 2009.
- World Health Organization (WHO). (2011b). *Healthy life expectancy (HALE) at birth (years)*. http://data.un.org/Data.aspx?q=lifexpectancy&d=WHO&f=MEASURE_CODE%3AWHOSIS_000002. Accessed 2011.
- World Health Organization (WHO). (2012). *Nutrition: Micronutrient deficiencies*. <http://www.who.int/nutrition/topics/vad/en/>. Accessed Sept 2012.
- World Health Organization (WHO), & United Nations Environment Programme (UNEP). (2013). *State of the science of endocrine disrupting chemicals – 2012*. Geneva, Switzerland: WHO Press.

- World Health Organization (WHO) (2007–2008). SAGE: WHO Study on global AGEing and adult health (SAGE). World Health Organization [distributor]. <http://www.who.int/healthinfo/systems/sage/en/>. Accessed 2011.
- Wray, L. A., Herzog, A. R., & Park, D. C. (1996, November). *Physical health, mental health, and function among older adults*. Presented at the annual meetings of the Gerontological Society of America, Washington, DC.
- Wray, L. A., & Lynch, J. W. (1998, November). *The role of cognitive ability in links between disease severity and functional ability in middle-aged adults*. Presented at the annual meetings of the Gerontological Society of America, Philadelphia.
- Wu, G., Bazer, F. W., Cudd, T. A., Meininger, C. J., & Spencer, T. E. (2004). Maternal nutrition and fetal development. *The Journal of Nutrition*, *134*, 2169–2172.
- Xizhe, P., & Zhigang, G. (Eds.). (2000). *The changing population of China*. Oxford, UK: Blackwell Publishers.
- Xu, T., Zhang, Z.-x., Han, S.-m., Xiao, X.-h., Gong, X.-m., Chen, X., et al. (2009). Relationship between perinatal characteristics and later activities of daily living in Chinese elderly people. *Chinese Medical Journal*, *122*(9), 1015–1019.
- Yajnik, C. S., & Dshmkh, U. S. (2008). Maternal nutrition, intrauterine programming and consequential risks in the offspring. *Reviews in Endocrine & Metabolic Disorders*, *9*(3), 203–211.
- Yan, S., Li, J., Li, S., Zhang, B., Du, S., Gordon-Larsen, P., et al. (2012). The expanding burden of cardiometabolic risk in China: The China health and nutrition survey. *Obesity Reviews*, *13*(9), 810–821.
- Zhang, Z., Gu, D., & Hayward, M. D. (2010). Childhood nutritional deprivation and cognitive impairment among older Chinese people. *Social Science & Medicine*. doi:10.1016/j.socscimed.2010.05.013.
- Zeng, Y., Gu, D., & Land, K. (2007). Healthy longevity at the oldest-old ages in China. *Demography*, *44*(3), 497–518.
- Zeng, Yi, Vaupel, J. W., Zhenyu, X., Yuzi, L., & Chunyuan, Z. (2002–2005). *Chinese Longitudinal Healthy Longevity Survey (CLHLS), 1998–2005* (ICPSR24901-v2). Center for the Study of Aging and Human Development: Duke University. Ann Arbor: Inter-university Consortium for Political and Social Research [distributor]. http://centerforaging.duke.edu/index.php?option=com_content&view=article&id=115&Itemid=152. Accessed 19 June 2009.
- Zimmer, Z., Kaneda, T., & Spess, L. (2007). An examination of urban versus rural mortality in China using community and individual data. *Journal of Gerontology, Social Sciences*, *62B*(5), S349–S357.

Index

A

- Accumulation, 9, 10, 157–158
- Adult
 - diet, 8, 72, 162, 167
 - lifestyle, 9–11, 49, 52, 54, 61, 62, 64, 79–80, 98–101, 106, 112, 114, 116, 117, 154, 159
 - drink, 80, 99, 100
 - exercise, 9, 11, 49, 79, 80, 89, 91, 98–101, 141, 142
 - smoking, 9, 11, 49, 61, 65, 79, 88, 89, 91, 98–100, 107–109, 111–113, 115–117, 120, 124, 125, 140–142, 158, 159, 166
- Almond, D., 5, 9, 10, 23, 32, 54, 57, 59, 159–161, 163
- Antibiotics, 12, 13, 17, 18, 21, 22, 25, 40, 41, 51–53, 69, 70, 73, 84, 93, 96, 99, 103, 165

B

- Banks, J., 59, 92
- Barker, D.J.P., 1, 5–7, 10, 12, 25, 34, 37, 49, 50, 56, 57, 120, 127, 128, 140, 144, 153, 156, 158, 159
- Barreto, S., 47
- Bengtsson, T., 5, 9, 54, 57
- Biomarker, 55, 60, 61, 64, 81, 91, 92, 163, 164, 167, 177
- Brenes, G., 6, 55, 56, 59, 91, 92, 143, 163

C

- Caloric intake, 15, 20, 23, 24, 49, 59, 61, 62, 65, 69, 72, 78, 101, 108, 110–117, 119–121, 155, 158–160

- Chronic condition, 1, 3, 4, 6, 10, 12, 36, 46, 47, 51, 52, 54, 59, 60, 62, 64, 69, 82, 87–92, 104–106, 120, 144–146, 151, 153, 154, 163–166
- diabetes, 1, 47, 81, 106, 131, 153
- heart disease, 1, 47, 80, 106, 131, 153
- underestimation, 60–62, 64, 81, 87–92, 101, 120, 129, 144, 151, 155, 163–165
- Clark, D.O., 82
- Clark, V.S., 27–29, 31–33, 45, 56, 83, 88, 121, 122, 139, 160
- Cohort effects, 49, 157, 158
- Compound, 10, 25, 32, 34, 50, 117, 120, 158, 159
- Confound, 7, 10, 25, 26, 50, 52, 58, 65, 78, 111, 113, 115, 117, 119, 128, 142, 145, 146, 152, 155, 157, 159
- Contrarian conjecture, 47–68, 104, 156, 158, 160, 165–167
- Costa, D.L., 5, 12, 57, 58, 127
- Crimmins, E.M., 5, 6, 12, 25, 47, 54, 55, 57, 127, 144, 158, 162–164
- Critical period, 10, 53, 117, 157–158

D

- Davey Smith, G., 5, 9, 10, 52, 57, 95, 118, 136, 142, 166
- Demographic regime, 53, 109, 133, 134.
See also Mortality, regimes
- Demographic transition, 1, 11, 46, 52, 56, 106, 113, 118, 145, 157, 166
- Developed country(ies), 2, 11–13, 47, 51, 105, 106, 111, 115, 119, 120, 135, 147, 149, 154
- Developing country(ies), 1–4, 13, 15, 18, 20, 23, 24, 26, 27, 46, 47, 58, 63, 73, 78, 106, 111, 117, 118, 120, 127, 145, 147, 149, 154

Diabetes, 1, 47, 81, 106, 131, 153
 Doblhammer, G., 5, 9, 57, 58, 95, 127, 140
 Drink, 80, 99, 100

E

Early life, 1, 47, 71, 106, 137, 153
 Effects
 accumulation, 9, 10, 157–158
 cohort, 49, 157, 158
 compound, 10, 25, 32, 34, 50, 117, 120, 158, 159
 confound, 7, 10, 25, 26, 50, 52, 58, 65, 78, 111, 113, 115, 117, 119, 128, 142, 145, 146, 152, 155, 157, 159
 critical period, 10, 53, 117, 157–158
 mediate, 10, 11, 32, 159–161, 164, 166
 period, 158–160
 Elo, I.T., 1, 5, 6, 9, 10, 55, 56, 59, 117, 143, 158
 Environmental, 4, 8, 9, 32, 34, 35, 37, 39, 42, 45, 49, 55, 58, 110, 113, 128, 139, 143, 154, 159, 162, 163, 165, 166
 Environmental conditions, 8, 9, 32, 34, 42, 45, 49, 55, 139, 143, 162, 166
 Exercise, 9, 11, 49, 79, 80, 89, 91, 98–101, 141, 142

F

FAO. *See* Food and Agriculture Organization (FAO)
 Floud, R., 5, 47, 66, 162
 Food and Agriculture Organization (FAO), 9, 15, 31, 48, 57, 59, 65, 78, 110, 113
 Functionality, 6, 54, 62, 65, 81, 82, 87–89–91, 101, 103, 112, 114–116, 120

G

Gluckman, E., 5
 Growth rate, 2, 3, 24, 30, 63, 76, 176
 Gruber, J., 60

H

Harmonized, 61, 79, 80, 82, 90, 95, 112, 114, 116, 153
 Harmonization, 61
 Harris, B., 47, 49, 137, 162
 Health care, 3, 9, 11, 13, 28, 48, 49, 52, 59, 60, 90, 92, 120, 146, 155, 161, 166
 Health selection, 50, 145, 151, 152
 Heart disease, 1, 47, 80, 106, 131, 153

Heterogeneity, 61, 127, 164, 165
 He, W., 1, 3, 92, 153
 High income, 1, 2, 15, 17, 52, 55, 69, 72, 73, 78, 89, 95, 104, 106, 109, 112, 114, 116, 117, 133, 134, 138, 146, 148, 153–155, 162, 164–166
 Hookworm, 13, 21, 27, 31, 34, 36, 39, 41, 51, 53, 143, 145, 165
 Hypotheses (“hypothesis”), 6, 8, 11, 25, 46, 47, 49, 51–55, 60, 61, 68, 105, 121, 122, 125, 127, 131, 137, 143–145, 150, 154–158, 165

I

IMR. *See* Infant mortality rates (IMR)
 Incidence, 3, 57, 59, 143
 Infant mortality rates (IMR), 17, 18, 20–23, 34–36, 40–42, 57, 63, 64, 66, 69, 70, 73–78, 122, 124, 125, 139, 162, 163, 169, 174–76
 Infection, 3, 6–8, 12, 22–24, 30, 34, 36–39, 51, 53, 56, 58, 128, 142, 143
 Infectious disease, 1, 3–8, 10, 12, 13, 23, 25, 27, 30, 32, 34–38, 46, 49, 52, 53, 55, 57–59–61, 64–66, 78, 79, 85, 86, 95, 115, 117, 119, 121, 125, 127–129, 139–145, 153, 154, 156–159, 165, 166
 Intervention, 1, 10, 12, 13, 17, 21–24, 38, 40, 41, 45, 46, 49, 51, 52, 70, 77, 127, 139, 143, 145, 153, 159–162, 164, 166, 167
In utero, 5–10, 25, 37, 53, 55–58, 86, 117, 119, 120, 125–127, 142–144, 156, 158, 165, 166

K

Kinsella, K., 1–3, 153
 Kuh, D., 4–6, 9, 10, 83, 87, 120, 154

L

Life expectancy, 1, 11–14, 16, 17, 19–21, 27, 45, 47–50, 60, 63, 64, 67, 69, 70, 73–78, 153, 161, 162, 169–171, 173–176
 Limitations, 68–73, 78, 81, 101, 119, 120, 138, 144, 164–165
 Low birth weight, 7, 36–38, 58, 119, 143
 Low income, 17, 20, 21, 52, 61, 73, 78, 89, 90, 95, 97, 98, 100, 101, 113, 145, 152, 167

M

Malaria, 3, 6, 13, 18, 21, 27, 32, 34–40, 42, 43, 51, 66, 87, 88, 139, 140, 142–145, 165, 166
 Marmot, M., 7, 10, 50, 56, 119
 Mazumder, B., 57, 59
 McEniry, 6, 20, 43, 49, 52, 55–58, 61, 63–67, 71, 73, 77, 78, 80–83, 85–88, 98, 117, 118, 121–127, 135–137, 140–142, 147–150, 155
 Mediate, 10, 11, 32, 159–161, 164, 166
 Medical innovation, 12, 13, 17, 24, 45, 46, 53, 156, 163, 165
 Medical technology, 1, 2, 17, 21, 23, 24, 40, 46, 49, 51–53, 127, 143, 145, 146, 153, 157, 161, 162
 Middle income, 1–5, 8, 17, 23, 25, 45, 47, 49, 50, 52, 54, 60, 67, 68, 73, 78, 83, 87, 89, 92, 95, 105, 106, 115, 118, 119, 121, 133, 135, 137, 138, 146, 150, 151, 153–155–157, 161, 163, 166
 Monteiro, C.A., 4, 6, 138, 145, 146, 149
 Mortality, 1, 49, 69, 105, 131, 153
 regimes, 73 (*see also* Demographic regime)
 timing, pace of and reason for mortality decline, 11, 14, 16–18, 24, 46, 52, 63, 73, 77, 105
 Murray, C.J.L., 1, 3, 47, 50, 144, 153, 158, 161, 166

N

Nutrition
 1930s–1960s, 24–26
 mortality regimes, 14, 16, 19
 Nutritional status, 12, 22, 23, 33, 42, 44, 49, 51, 56, 57, 59, 64, 86, 95, 105, 128, 154, 163

O

Obesity, 1, 4, 7, 61, 62, 66, 81–83, 86, 87, 89–91, 101, 104, 106–108, 111, 113, 115, 117, 120, 123–125, 129, 132, 134, 135, 137, 138, 140, 142, 144, 145, 152, 154, 155, 158, 159, 161
 Omran, A.R., 11, 63

P

Palloni, 2, 3, 5, 6, 11, 17, 24–26, 49–52, 55, 56, 63, 66, 67, 73, 78, 83, 85, 87, 88, 105, 117, 118, 122, 123, 125–127, 137, 142, 143, 145, 152, 155, 166

Parental SES, 64, 65, 78, 83–85, 111, 113, 115, 117
 Period effects, 158–160
 Policy, 2, 4, 9, 45, 46, 160–164, 166, 167
 Pool of survivors, 53, 106, 127, 140, 150, 151
 Poor childhood health, 6, 50, 59, 66, 83, 117, 122, 140, 141, 145, 158
 Poor childhood SES, 5, 66, 141, 144, 158
 Poor early life conditions, 1, 2, 5, 10–13, 24, 25, 45, 46, 49, 50, 52, 54, 77, 106, 138–140, 145, 146, 150, 152, 154, 156, 157, 159–161
 Popkin, B.M., 4, 6–10, 25, 50, 54, 119, 137, 145, 157, 158, 162
 Population surveys, 56, 70, 153, 163
 Preston, S., 1, 5, 6, 9, 11, 14, 17, 18, 24, 47, 50, 51, 59, 63, 64, 77, 117, 143, 158, 162, 165, 170, 171
 Prevalence, 9, 36, 40, 51, 52, 59–62, 64, 65, 68, 81, 86–92, 101, 103–106, 110, 111, 113, 117–121, 123, 127, 136, 139, 144–147, 149, 152, 154–156, 166, 179–181
 Public health intervention, 1, 13, 17, 21–24, 40, 41, 45, 49, 51, 52, 77, 127, 139, 143, 145, 153, 166

R

Reduction (in disease, exposure), 1, 12, 13, 24, 25, 38–45, 49, 64, 73, 76
 RELATE, 69–71, 80, 85, 89, 94, 95, 97, 98, 100, 102, 103, 107–110, 112–114, 116, 132, 133, 136, 180, 181, 183–185, 187
 Relative risk, 66, 67, 83, 132–138, 156
 Riley, J., 1
 Riley, J.C., 17, 21, 22, 49, 75, 169
 Rose, G.A., 64, 81, 90, 103, 107, 114, 118, 180
 Rural, 1, 17, 21, 22, 27–33, 35–38, 55, 56, 62, 64, 71–73, 78, 83–87, 94–96, 98, 101, 104, 108, 111, 112, 114–116, 125, 126, 128, 133, 140, 141, 151

S

Sanitation, 2, 12, 13, 18, 21, 38, 44, 51, 143, 162
 Saturated fat, 1, 4, 8–10, 46, 50, 117, 119, 120, 155, 158–160, 165
 Schoeni, R.F., 5
 Scrimshaw, N.S., 51, 127, 157
 Season of birth, 57, 58, 64–66, 78, 85, 95, 101, 121, 122, 126–128, 138, 140–144

- Sedentary lifestyle, 1, 9, 10, 25, 46, 50, 72, 117, 158, 159
- Self-reported health, 54, 59, 65, 80–82, 96, 97, 101, 103, 112, 114–116
- Smith, J.P., 12, 18, 59, 82, 128, 151
- Smoking, 9, 11, 49, 61, 65, 79, 88, 89, 91, 98–100, 107–109, 111–113–115–117, 120, 124, 125, 140–142, 158, 159, 166
- Standard of living, 2, 3, 11, 17, 22, 27–28, 44–46, 48, 51, 52, 77, 95, 146, 156, 161, 162, 165, 166
- Susceptible, 1, 37, 46, 153, 154
- Synergistic, 6, 23
- Synergy, 25, 51, 127, 129, 157
- T**
- Tide to come, 46
- Timing, pace of and reason for mortality decline, 14, 16, 46, 52, 63
- Tip of the iceberg, 24–26, 45, 46, 51, 52, 61, 63–64, 68, 73–78, 105, 117, 119, 127, 131, 145, 146, 150–158, 161, 162, 165
- Tuberculosis, 3, 28, 34, 35, 37, 38, 88, 139, 140, 142–144, 165
- U**
- Underestimation, 60–62, 64, 81, 87–92, 101, 120, 129, 144, 151, 155, 163–165
- Unintended consequences, 2, 11, 45, 160–163
- Unique cohorts, 24–26, 46, 47, 49, 51–53, 68, 105, 113, 117, 127, 129, 131, 145, 146, 151–154, 157, 159, 160, 162, 166, 167
- Unique cohorts of the 1930s–1960s, 24–26, 46, 47, 68, 105, 131, 145, 151, 152, 154, 157, 160, 162, 166, 167
- United Nations, 2, 3, 23, 31, 48, 57, 63, 65, 169, 176
- Unobserved heterogeneity, 164
- Urban, 1, 17, 21, 22, 26–28, 30, 33, 37–40, 51, 55, 56, 62, 64, 69, 71–73, 76, 78, 83, 87, 88, 93–95, 98, 101, 108, 115, 126, 133, 140, 143, 151
- V**
- Validity, 55, 56, 61, 64, 69–104, 165
- Vaupel, J.W., 6, 47
- W**
- Waaler-type surfaces, 66, 67, 83, 104, 132–138, 152, 156, 165
- WHO. *See* World Health Organization (WHO)
- Wise, D., 60
- World Bank, 2, 48
- World Health Organization (WHO), 3, 4, 11, 30, 36, 47, 48, 56, 66, 80, 103, 110, 113, 136, 159, 161, 169–171, 178