Causes, Correlates and Consequences of Death Among Older Adults:

Some Methodological Approaches and Substantive Analyses

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Dedication

Dedicated to Paul Taubman, collaborator, colleague, mentor and friend, 1939-1995.

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1
Introduction

All humans eventually die, but life expectancies differ over time and among different demographic groups. Teasing out the various causes and correlates of death is a challenge, and it is one we take on in this book. A look at the data on mortality is both interesting and suggestive of some possible relationships. In 1900 life expectancies at birth were 46.3 and 48.3 years for men and women respectively, a gender differential of a bit less than 5 percent. Life expectancies for whites then were about 0.3 years longer than that of the whole population, but life expectancies for blacks were only about 33 years for men and women. At age 65, the remaining life expectancies were about 12 and 11 years for whites and blacks respectively.

Fifty years later, life expectancies at birth had grown to 66 and 71 years for males and females respectively. The percentage differential between the sexes was now almost up to 10 percent. The life expectancies of whites were about one year longer than that for the entire population. The big change was for blacks, whose life expectancy had grown to over 60 years with black females living about 5 percent longer than their male counterparts. At age 65 the remaining expected life had increased about two years with much larger percentage gains for blacks. It is likely that the gains for blacks occurred partly from their relative improvement in economic status, though earnings data are not available over this time period and education data are only collected beginning in the 1940 Census in which coverage may have been selective due to prior mortality.

Over the next 20 years (1970-1990) life expectancies at birth increased an additional two years with bigger gains for white women and for blacks of both genders. Even when calculated at age 65, life expectancy grew on average by about one year but with much larger increases for white women. Moreover, there was a 0.4 year decrease for black men.

Life expectancies at birth for the population as a whole grew at an annual rate of about 0.4 years for the period 1900-1950; at about 0.08 per year from 1960 to 1970; accelerated to 0.35 per year from 1970 to 1975; and then slowed down for the 1975 to

1980 years. However, blacks of both genders recorded major gains over the period 1970-1980.

Since then life expectancies at birth have only increased by about a year (in total) for all race and gender groups while at age 65 they have changed little. Thus, from 1900 on, life expectancies grew rapidly until 1950 with blacks (non-whites) and females growing more quickly than whites.

1.1. General Categories of Causes of Differential Life Expectancies among Groups and Over Time: Genetics, Environment and Behaviors

Why are there difference in life expectancies among various demographic groups? Why have these changes in life expectancies occurred? Many reasons are given in the popular and the more scholarly literatures. The myriad of underlying factors can be categorized for our purposes as genetic, environmental, and behavioral. To successfully untangle the causes and correlates of death and morbidity, these factors must be addressed in the analysis. Therefore, it is beneficial to examine them more closely.

Some illnesses and behaviors that lead to early death can be caused by one or more genetic defects. The single gene model, for example, can explain the occurrence of hemophilia, which can be fatal. A polygenic model as in Fisher (1918) also can be used and implemented with data on twins and perhaps other relatives to explain the incidence of and fatality from many other diseases.[1] While genetic differences undoubtedly are of some significance, in this book these variations will be allowed for primarily by subsuming them together with some environmental differences in "frailty" or "unobserved heterogeneity." We explore in some of our analysis how sensitive the estimates are to controls for such frailty.

The environmental category incorporates all other factors that influence age at death but that are not determined by the micro behavior of the individuals (or perhaps the families of such individuals) whose mortality is of interest.[2] This category includes medical technology, accidents, wars, pollution, prices and the rules that govern health insurance. For example, advances in medical care are highly touted as an explanation for changing life expectancies by some. Yet during the period 1975-1990 life expectancies changed little even though new drugs and medical innovations were introduced that changed the environment through behavioral responses to that environment, national health expenditures rose from 8 percent to 12 percent of GNP, and the number of doctors and nursing homes rose noticeably. The explanations thus are much more complicated than just improved medical care and the induced behavioral changes.

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[1] See Behrman, et al. (1980) and Behrman and Taubman (1989) for methods applied to other subject areas. For mortality and death from specific causes using twin samples, see Hrubec and Neel (1981), Kaprio and Koskenvuo (1990), and Floderus, Cederlof, and Friberg (1988).

[2] Some factors that we include in the environmental category reflect behaviors of governments or other entities, but effectively are given from the point of view of individuals.

But, whatever genetic stocks one has and whatever environment one lives in, life precedes death. And an individual's behaviors, as well perhaps as behaviors of others such as other members of the same household, are thought to have major impact on age-specific mortality and therefore life expectancies. Increasing evidence suggests, for example, that there are important associations between mortality on one hand and behaviors related to medical care, diet, exercise, smoking, and drug consumption on the other. Associations among behaviors often has been used to infer causality. But such correlations do not necessarily reflect causality. Instead, for example, both mortality and correlated variables may reflect in part genetic and environmental differences across individuals that affect both mortality and the correlated behavior. In some of our analysis, as noted, we explore to see how sensitive the estimates are to controls for unobserved genetic and environmental heterogeneities.

Distinction among genetic, environmental and behavioral causes is critical for analysis of health and mortality. From the point of view of individuals in the time periods that we analyze,[3] genetic and environmental factors are given — though the latter may change due to wider developments in the society and the economy. Individual behaviors that determine health and mortality, in contrast, are responses to their genetic and environmental endowments. Therefore, to make progress on identifying causality rather than just correlations it is essential to control for behavioral responses to observed and unobserved components of individuals' genetic and environmental endowments.

1.2. Why are the Causes, Correlates and Consequences of Death of Interest?

The causes and the correlates of death are of interest both from the point of view of individuals and of society. It is clear, for instance, that concerns about health, life expectancies and mortality inspire individuals to take actions to change their behaviors as well as society as a whole to try to improve the environment. In part underlying such individual and societal behaviors are observations about correlates of mortality and poor health with a number of genetic, environmental and behavioral factors that are inferred to reflect causality. For example, many people stopped smoking after the Surgeon General's report on this matter (Feldman, et al. 1989), and more recently governments have taken increasing steps to limit smoking behavior and "second-hand" smoke. The extent and use of leisure time, for another example, is thought to have an important impact on health, and therefore mortality. Consumption of health-related goods and services also would seem important. Reverse causality clouds cross-sectional analysis of the relations between use of health-related goods and services on one hand and health and mortality on the other hand. Those who are sicker and have higher probabilities of dying in the near future tend to consume relatively large quantities of health-related goods and services, particularly related to formal medical care. One of our contributions in this book to this literature is successfully accounting for the reverse causality through our estimation techniques.

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[3] Recent medical developments, however, point to increasing future possibilities of interventions to offset an individual's genetic makeup.

Governments, of course, have huge interests in the determinants of mortality for two reasons. One, its impact on the welfare of its citizens. And two, the public costs of many governmental programs such as Social Security, Medicare, and Medicaid in the United States are related to the health and life expectancy of its constituents (though administrators of some of these programs may want shorter life expectancies to reduce "their" expenditures and others may want a long, healthy existence to fulfill "their" mandates). Governments throughout the world have been involved in serious debates about how to best organize the health sector given the prospect, if not the reality, of expanding formal health care costs and aging populations in North America, Europe, and increasingly in Asia and Latin America.

The consequences of death are also important beyond the obvious point of physically ceasing to exist. There are major equity concerns in the design and operation of various public and private policies that are related to the date of death. For example, even though Social Security's benefit schedule is highly progressive (the extra benefit obtained from each extra dollar of covered lifetime earnings declines sharply), Hurd and Shoven (1985) have shown that the implicit rate of return on dollars invested in these funds is essentially the same for low and high earnings people as long as people of the same sociodemographic category who died before receiving benefits are included in the investment pool and differential life spans of beneficiaries are controlled. We doubt that this is the intent of society or the U.S. government. The Hurd and Shoven study is based on older life tables and needs to be updated and expanded. More refined estimates may find that the rate of return actually increases with lifetime earnings; it would be of substantial interest to learn if this occurs.

Moreover, the viability of the Social Security system, private pensions, and annuities depends crucially on the ability of the governmental and private actuaries to make accurate mortality forecasts. If death hazards truly depend on personal characteristics that are malleable — cigarette smoking and education are examples — then the life tables must be updated in ways that will be indicated in our analysis.

1.3. Concerns and Results of This Book

Because of the importance of mortality to individuals and to society, not surprisingly there exists much research in the social sciences and other disciplines that examines trends in life expectancies of various groups of individuals and reasons for differences in such life expectancies. Examples of this research related to the specific substantive topics covered in this book are surveyed at the beginnings of Chapters 2 and 4.

In this book we contribute to this literature by considering selected topics related to the causes, correlates and consequences of adult death in the United States. We examine several methodological approaches that can be used to explore these issues. We first estimate some of the critical parameters in the basic structural relations that determine health of older adults within a basic intertemporal optimizing model of individual behavior since death can be viewed as inadequate health in some critical dimensions. We then focus on estimating hazard functions for mortality for different birth cohorts and gender and race groups that can be interpreted as conditional reduced-form demand relations that are consistent with the same intertemporal optimizing behavior. These topics have important substantive and methodological dimensions that

help to increase not only our knowledge of particular phenomena, but also have broader implications for social science research.

Our investigation into the causes and correlates of death leads to new answers being provided from two data sources — the Dorn Sample of Veterans who served in the military during the period 1917-1940 and the Retirement History Survey which is a random sample of household heads aged 58-63 in 1969. Both these samples contain data on date of death by month and year over long time periods — up to 10 years for the RHS and 26 years for the Dorn sample. These samples are of particular interest for our analysis because of the richness and range of the mortality data and because, particularly for the former, earlier studies have considerably shaped received wisdom about mortality determinants. It is thus of considerable interest to learn if estimates are robust to the application of recently-developed techniques, and particularly to control for unobserved heterogeneity. To examine the consequences of death, we also use the Panel Study of Income Dynamics in a much more limited way to explore some issues related to the returns of the Social Security system to younger cohorts.

Using these samples we apply statistical techniques developed within the last 20 years (and some within the last few years) to examine the probability that a person alive at the beginning of a month will die within that month. We distinguish individuals by characteristics and actions such as education, marital status, occupation, smoking intensity, and income.

These distinctions allow us to answer questions such as: Is there evidence of a positive impact of leisure time on the health of older adults? Is there evidence of a positive impact on the health of older adults of expenditures on health-related consumption of goods and services once there is control for the reverse causality noted above? What are the associations between smoking and the occupational environment and mortality? What are the associations with income? Are there differences between whites and blacks and between men and women? If so, to what extent are these differences related to observed differences in characteristics such as occupation and education?

Our substantive findings about the causes and correlates of death, some of which support previous research, include the following: older adult health is improved with leisure even though "all-or-nothing" retirement decisions may force active adults to have more leisure than they would choose were continuous choices for their time use readily available; once there is control for reverse causality, older adult health is improved significantly with consumption of health-related goods and services — a result contrasting with simple static correlations of morbidity and health care utilization (Agency for Health Care Policy and Research, 1993, Research Findings #16); cigarette smoking is associated with a shorter life span, the more so the more intense the smoking; giving up cigarettes partially offsets the reduction in the life span; more sedentary and more (health) risky occupations are related to shorter life spans; and greater income and being married are both associated with increased life span. Marital associations with mortality are somewhat different for men and women aged 58 to 70. Regional differences are small, but cohort differences are large at least when unobserved heterogeneity is not controlled. We also find a substantial fraction of the observed difference in annual probability of dying between blacks and white is consistent with the observed differences in variables such as Social Security benefits

and marital status, though there are questions as to whether these variables should be treated as exogenous and if their coefficients are unbiased.

In our study of the consequences of death, mortality has a substantial negative impact on rates of return from "investments" in Social Security and that differential mortality reduces the (relatively small) progressivity in these rates of return between whites and blacks, men and women, and poorer and richer people aged 58 to 63 in 1969. Since much of the working life of this age cohort was spent during a period with a relatively low cap on earnings subject to Social Security taxes the variability in their benefits is limited. For younger cohorts which experienced higher earnings caps and greater variations in benefits, we find less progressivity in the Social Security system.

1.4. Methodology

The recently developed econometric tools that we employ have uses in many other economic problems, and we wish to understand and popularize these techniques for future use in this field. We start with a discussion of hazard models and then make allowances in the model for unobserved heterogeneity, which arise from differences in frailty discussed above.

We next look at different methods of estimation. One is Heckman and Singer's (1984) Nonparametric Estimation (NPMLE). The estimator is consistent but the nature of its limiting distribution is as yet not well understood. This estimator has been used in the study of adult health using the Dorn smoking sample by Berhman, et al. (1990). They examine robustness of estimates to functional form, individual heterogeneity, and cohort and time period variations and find that the Heckman-Singer nonparametric methodology is judged best in terms of fit. Another estimator we use is the Maximum Penalized Likelihood Estimator (MPLE). It provides another approach to dealing with unobserved heterogeneity but has been used less widely. It was introduced by Good and Gaskins (1971) and developed by de Montricher, et al. (1975) and Silverman (1982). Huh and Sickles (1984) detail how this model can be modified to handle unobserved variables under different assumptions about temporal and cross-sectional sources of heterogeneity that is uncorrelated with the observed variables.

MPLE may have computational and convergence advantages over NPMLE in finite samples since roughness in the empirical heterogeneity distribution is smoothed from the likelihood by including penalty terms that take into account the degree of roughness or local variability not controlled for by covariates. The samples that we have available for analysis allow us to examine in detail many of these techniques and to determine their robustness. Perhaps most noteworthy, we find that our estimates are fairly robust to the allowance for parametric and nonparametric heterogeneity in the proportional hazard models.

1.5. Organization of This Book

Though the material that is included is often technical, we hope that this book will appeal to a number of different audiences in the social science research community. The detailed work on the estimation of hazard survival function with focus on

socioeconomic and behavioral variables should be of interest to demographers. The use of dynamic household models in a manner in which they are tractable for estimation will be of interest to social scientists with a focus on household behavior. The explorations of the sensitivity of alternative estimation procedures, particularly regarding the alternative approaches to control for unobserved heterogeneities, should be of interest to applied econometricians and other applied social scientists. The substantive results on gender and race differences in mortality determinants should be of widespread interest to social scientists from different disciplines. The material on rates of return to Social Security contributions and mortality also will appeal to a range of social science researchers and policy makers. We have attempted to organize the presentation of material in the book so that readers with different interests easily can focus on the topics of particular interest to them.

Chapter 2 presents a structural model for the optimal choice of health and mortality, which is defined as health falling below a threshold, as a dynamic behavioral problem, given an objective function and intertemporal budget and production function constraints. The implications of this approach for estimation of health behaviors and hazard mortality functions are derived formally. Attention is paid to both the advantages and the limitations of such a formal approach to modeling mortality and related health.

Then it introduces the Retirement History Sample, a random longitudinal sample of 11,000 household heads who were between the ages of 58 and 63 in 1969 and reinterviewed biennially through 1979, who were matched with Social Security records on the month and year of subsequent mortality. Next, this chapter presents estimates of structural parameters for the utility function and the health production function for the model presented earlier in the chapter on the optimal health decisions for older men. These estimates are obtained using instrumental variable methods to attempt to control for behavioral choices. The estimates suggest that the mean marginal utility of health and health-neutral goods are positive, but that of leisure is negative, perhaps due to too much leisure for active persons who retire (given the all-or-nothing nature of many retirement decisions). Both leisure and health-related consumption are estimated to enter positively into the health production function, with elasticities of health with respect to leisure on the order of magnitude of 0.6 to 0.7 and those with respect to health-consumption in the range of 0.01 to 0.02. These are the first estimates available to find that both leisure and health-related consumption have significant positive effects on health, perhaps because the use of panel data and instrumental variable procedures lessens the reverse causality estimation problems that have plagued previous cross-sectional studies (i.e., poor health induced more medical care).

Chapter 3 presents statistical techniques for estimation of hazard functions that are used in the two subsequent chapters to analyze data on age-specific death rates. Both proportional and accelerated-time-to-failure models are considered, with particular emphasis on the role of unobserved heterogeneity. Also, less frequently used simulation and maximum-penalized-likelihood approaches are presented and explored with Monte Carlo experiments.

Chapters 4 and 5, respectively, present estimates of alternative hazard functions for mortality for the Dorn and the Retirement History Sample, based on the models and procedures developed and presented in Chapters 2 and 3.

Chapter 4 first surveys the relevant literature and then introduces the Dorn sample, which consists of approximately 300,000 U.S. veterans who had served in the Armed Forces between 1917 and 1940, who in 1953 held U.S. Government Life Insurance Polices at which time they were surveyed regarding smoking and occupation, and whose subsequent month and year of mortality has been recorded by the Veterans Administration. While several researchers have used this sample to analyze survival, no previous studies have utilized the methodologies used in this book. The robustness of the estimates to sample length, alternative functional forms and unobserved frailty are explored.

Chapter 5 focuses on the effects of covariates rather than the specification of the hazard. Attention is given to the black-white mortality differentials, with differences in marital status, pension income and education consistent with about one fifth of these differentials. Attention also is given to estimates for women, for whom the magnitude of the estimated effect of pension income is smaller than for men.

Chapter 6 presents estimates of rates of return on Social Security. If all individuals in the Retirement History Survey live to age 90, white males would have an annual rate of return of 9.5 percent and black males, who have lower lifetime earnings, would have a rate of return of 10.2 percent. Thus, there is some indication of the redistribution effects that purportedly are intended by the Social Security system. Adjustment for differential mortality by race based on the hazard functions from Chapter 3, however, erodes almost all of the redistribution effects because of the higher age-specific mortality experience of blacks than of whites, for both males and females. Examination of younger cohorts from the Panel Study of Income Dynamics indicates lower rates of return on Social Security contributions with less in the way of redistribution effects.

Chapter 7 presents conclusions.

2

The Optimal Choice of Health and Mortality

In this chapter we use micro economic models of individual behavior in a dynamic context to describe how an individual chooses the optimal level of investment in healthiness and implicitly length of life. This establishes a foundation for the estimation of structural parameters related to health demands for older adult men in Section 2.4 and for the hazard mortality estimates that we present in Chapters 4 and 5. While choice regarding health may be considered commonplace for economists, characterizing the length of life as a choice may seem crude and unreasonable to some. There are, however, numerous examples of people making this choice directly, e.g., suicides, living wills that mandate against extraordinary medical care, the refusal by Christian Scientists of potentially life-preserving medical care, and the shortfall of deaths before personally significant dates with a subsequent spike in death rates afterwards.[4] Moreover, people have cut back on cigarette smoking since the Surgeon General's 1964 report on the morbidity and mortality implications of smoking. They also have to be paid more to work at jobs at which more deaths occur (see Thaler and Rosen, 1975). More to the point, by constantly making choices that affect their health and thus the probability that their health falls irreversibly below some threshold leading to death, individuals constantly are affecting their expected duration of survival — whether or not they characterize these choices as proximate causes of survival.

We approach this subject by assuming that individuals act to maximize an intertemporal utility function subject to an intertemporal budget constraint and a health production function. A utility function is defined as a function, U, that converts various goods and services, and health itself, into a person's utility or well-being. The goods and services that we consider can be divided into three broad categories — goods that have neutral, positive, and negative effects on health. Examples of these are books, medicine, and tobacco, respectively.

The intertemporal budget constraint says that the sum of expenditures on these

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[4] Examples are birthdays, anniversaries, birth of a grandchild.

three types of goods and services over time can not exceed available resources. The health production function indicates how genetic endowments, environment and health-related behaviors are combined to produce a person's health stock. Death occurs when a person's health stock falls below a threshold level, and death is an absorbing state.

2.1. Previous Models

The most widely-used framework for determining health status is the one-period reduced-form demand model originally due to Grossman (1972, 1975) and utilized in numerous studies including several by the authors (e.g., Bartel and Taubman 1986, Behrman and Deolalikar 1987, Behrman and Wolfe 1987a,b, Wolfe and Behrman 1984, 1987, 1992, Sickles and Taubman 1986). A related but distinct framework is provided by Rosenzweig and Schultz (1982) and Wolpin (1984).

The primary differences between the two frameworks are that the latter studies concentrate on obtaining unbiased estimates of the parameters of both the utility function and the health production function that underlie the demand relations, and have examined in greater detail life-cycle utility functions. The Grossman-type models concentrate on estimating static reduced-form equations, sometimes dependent on previous choices so that the demand functions are conditional demand functions as discussed in Pollak (1969, 1970). The endogeneity of some right-side variables in such conditional demand functions can be controlled, at least in principle, by using instruments that affected past choices but are independent of the current disturbance term. Past shocks in market prices, for example, might serve as such instruments. But as in any such instrumental variable estimation, all the past choices that directly affect current health must be observed and instrumented to obtain unbiased estimates of the effects of such choices. If some past choices that were affected by the same instruments as the observed choices are not observed, the instrumented values of the observed choices will represent in part the effects of the unobserved choices.

2.1.1. A one-period model of health demand

In the one-period static model the individual's economic problem is to allocate time to leisure, T_t^L, time to the production of health, T_t^H, and financial resources in order to[5]:

$$(2.1) \qquad \max_{C_t^0, C_t^1, T_t^L, T_t^H} U(C_t^0, T_t^L, H_t)$$

where C^0 is the final consumption good which provides pleasure and C^1 is an intermediate good used to produce health. The individual works for a period of time

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[5] The dual role of women as both child care providers and home care providers, neither of which is formally compensated, has been noted by many as a time allocation which has a significant negative impact on reducing women's health (see, for example, Wolfe and Haveman, 1983).

given by $T^W = T - (T^L - T^H - T^I)$, where T^I is illness time which is a function of the health stock, and given a wage W, has income $Y = W*T^W$ from which C^0 and C^1 are purchased at prices P^0 and P^1, respectively. Let initial wealth be A_0. The individual's budget constraint is then:

$$(2.2) \qquad A_0+[T-T_t^L-T_t^H-T_t^I(H_t)]W_t \geq P_t^0C_t^0+P_t^1C_t^1$$

Income and wealth have served as proxies for many mortality and morbidity factors for adults and the elderly, factors whose independent and dynamic effects can only be disentangled using such a structural formulation. This is because income and wealth provide for more consumption of health related goods, and by way of this, access to higher quality medical care. The importance of pension wealth and its role as an insurer of income for the elderly after retirement has long been recognized (Bodie, 1990). Rendall and Speare (1993), for example, using the Survey of Income and Program Participation (SIPP), note the importance of using the income plus wealth measure of economic well-being for the United States elderly to properly distinguish the most economically vulnerable sub-population, in particular blacks. Health can also affect labor market outcomes by altering tastes and/or the income opportunity curve. Recently, Attanasio and Hoynes (1995), using the SIPP data, point out the many shortcomings of cross-sectional analyses of mortality and wealth due to the differential effects of mortality on asset accumulation and correct for selectivity bias in survival rates. Obviously, income and wealth for adults and the elderly is also influenced by whether they are in or out of the labor force.

The health production function (h_t) is introduced to show how health, H_t, is produced by the consumption of health-related goods (C_t^1), time devoted to health production (T_t^H), and other exogenously determined inputs, X_t, such as human capital characteristics, environmental factors, endowments, and individual specific heterogeneities[6]:

$$(2.3) \qquad H_t = h(C_t^1, T_t^H; X_t)$$

X is a vector of human capital characteristics, endowments and individual specific heterogeneities. It will usually contain such variables as education, nutrition,

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[6] Wagstaff's (1989) excellent survey of the recent British literature of empirical studies on the economics of health point out a number of issues involving how more aggregate system wide production functions can be specified to take account of allocative and technical inefficiency, the former issue having been taken up by Eakin and Kniesner (1988) and the latter by Feldstein (1967). He further points to the potential scope of stochastic frontier approaches for cross-section models (Aigner, Lovell, and Schmidt, 1977) and panel models (Schmidt and Sickles, 1984; Cornwell, Schmidt, and Sickles, 1990). These frontier methods have also been used, along with polynomial-spline regressions to examine the depreciation of physical health using track and road racing data by Fair (1994). One of his more interesting findings is the relatively slow rate of physical depreciation that occurs suggesting a bias in societal perspectives of the elderly's health potential.

occupation and physical activity, marital status, behaviors and lifestyle measures, and genetic factors. Heterogeneities in preferences across different individuals can be accommodated by explicitly conditioning the utility function on X as well.

The solution to the one-period model can be based on the Lagrangian function where the budget constraint is (2.2) and where the health production function is directly substituted into (2.1). The Lagrangian multiplier (λ) is interpreted as the marginal utility of wealth. First-order conditions are:

$$
\begin{aligned}
& U_{C_t^0} - \lambda P_t^0 = 0 \\
& (U_{H_t} - \lambda W_t T_{H_t}^I) h_{C_t^1} - \lambda P_t^1 = 0 \\
& U_{T_t^L} - \lambda W_t = 0 \\
& (U_{H_t} - \lambda W_t T_{H_t}^I) h_{T_{H_t}} - \lambda W_t = 0
\end{aligned}
\tag{2.4}
$$

Note that in a one-period model $T_t^I = f(H_{t-1})$ is a constant so that in this case $T_{H_t}^I = 0$. Using the implicit function theorem these first-order equations are solved for the choice variables $\{C_t^0, C_t^1, T_t^L, T_t^H\}$, after substituting out λ from, e.g. the first equation, in terms of the state variables. Choices of functional forms such as Cobb-Douglas or CES as well as assumptions such as constant returns to scale in the production of health capital provide simplifying expressions for these demand equations. However, as a general rule, choices of flexible forms such as translog or generalized Leontief for the utility or production function may be more easily accommodated by using numerical methods to solve for the demand equations and for related elasticity measures. Dynamics can be imposed on the structure of the one-period model by specifying prices, or any of the nonchoice variables, in terms of some distributed lag of current and past values. However, no structural interpretation can be given to these dynamics since they are not specified as part of the optimizing framework.

One should immediately question a static model of health consumption. Today's exogenous variables affect tomorrow's endogenous choices, and expectations of tomorrow's exogenous variables affect today's endogenous choices. While we can, to a certain extent, overcome some of this concern in the estimation of this model, as when we employ an estimate for those instruments that affected past choices but are independent of the current disturbance term (e.g. past shocks in market prices), for any such estimation all of the past choices that directly affected current health status must be observed in order to obtain unbiased estimates.

2.1.2. Dynamic models of health demand

Two problems associated with alternatives to one-period models are simplifying assumptions usually employed for tractability, namely temporal and intertemporal separability. The first refers to the assumption that arguments of a utility function are

not related to each other within a time period. This problem can be solved by choosing functional forms that model interaction between arguments at each time period. The second problem refers to treating time as a superficial barrier between an argument in time t and the same argument at times t-1 and t+1. This is usually solved by breaking the objective function into smaller time-separable problems which allows dynamic programming techniques to be utilized to solve the maximization problem. The price for mathematical convenience is the treatment of the arguments as time separable. A number of authors have argued for the use of preference structures that incorporate forms of state dependence (e.g., Kydland and Prescott, 1982; Eichenbaum, Hansen, and Singleton, 1988; Hotz, Kydland, and Sedlacek, 1988). Accumulation of assets and retirement pensions such as Social Security that are pegged to past earnings also indicate the important role of dynamics in the timing of decisions. Burtless and Moffit (1984), for example, used the Retirement History Survey to examine the impact of Social Security benefits on labor supply of the aged and found that Social Security has important effects on the exact timing of retirement as well as the amount of labor supplied after retirement. Van de Ven and Van der Gaag (1982) in their panel study of 8000 households in the Netherlands, noted that permanent and transitory components of the income stream must be distinguished in order to properly model the positive long run relationships between the demand for health and permanent income.

Consider a rational individual with perfect information, seeking to maximize the present discounted value of lifetime utility derived from consumption and leisure, subject to constraints of available time and resources.[7] Health for the individual is a capital stock, and the individual values health as it reduces sick time, which correspondingly increases the amount of productive time and leisure time.[8] We first represent the individual's life-cycle maximization problem in terms of the constrained dynamic programming problem where the horizon is long (see, for example, Sargent, 1987; Stokey et al., 1989) as:

$$(2.5)\qquad \max_{C_t^0,C_t^1,T_t^L,T_t^H,A_t} L=\sum_{t=1}^{LE}\beta(x)^{t-1}U\left(C_t^0,T_t^L,H_t\right)+\lambda(A_{t+1}-\gamma_t(A_t+(T-T_t^L-T_t^H-T^I(H_t))W_t-P_t^0C_t^0-P_t^1C_t^1))$$

---

[7] Although the concept of maximization of present discounted value of future utility may appear somewhat questionable in this context, there are confirmations in the literature, most recently by Kenkel (1994), who demonstrates declining demands for health investments by the elderly as they age, consistent with an individual rationally reducing an investment in health as the "pay-off" period diminishes.

[8] If the concept of health is extended to include intellectual and emotional health as well as physical health then one can capture many other important human economic activities, in addition to those we have previously described. Deferring income by investing time in health production in order to advantage oneself of higher wages in the future has similar motives to those utilized in models of the decision-making process for higher education. Further, the substantial time many humans allocate to such activities as fraternization would suggest that human capital should be broadened to include social capital as well (Coleman, 1988) with careful treatment given to distributional heterogeneity that may be present within groups that define intra-individual social norms (Manski, 1993).

where $\beta(X)$ is the per-period discount factor and the per-period intertemporal budget constraint is expressed in terms of the full-wage. A_t are real assets at the beginning of period t, $\gamma_t = (1 + r_t)$, and r_t is the real interest rate. Health capital evolves according to:

$$(2.6) \qquad H_t = h(C_t^1, T_t^H; X_t) + \left(1-\delta_{t-1}(X_{t-1})\right) H_{t-1}$$

where δ is the rate of depreciation. The importance of allowing for unobserved heterogeneity in the discount rate (as well as h and δ) has been recognized by many authors (see, for example, Fuchs, 1982).

Solutions for the life-cycle model of health investment yield the first-order conditions for maximization of constrained life-time utility that take the form of:

$$\begin{aligned}
&U_{C_t^0} - \lambda_t P_t^0 = 0 \\
&(U_{H_t} - \lambda_t W_t T_{H_t}^I) h_{C_t^1} - \lambda_t P_t^1 + \beta(1-\delta_t)\,(U_{H_{t+1}} - \lambda_{t+1} W_{t+1} T_{H_{t+1}}^I) h_{C_t^1} = 0 \\
(2.7)\quad &U_{T_t^L} - \lambda_t W_t = 0 \\
&(U_{H_t} - \lambda_t W_t T_{H_t}^I) h_{T_{H_t}} - \lambda_t W_t + \beta(1-\delta_t)(U_{H_{t+1}} - \lambda_{t+1} W_{t+1} T_{H_{t+1}}^I) h_{T_{H_t}} = 0 \\
&\lambda_t = \beta\gamma_t\, \lambda_{t+1}
\end{aligned}$$

The last equation is the equation of motion for the marginal utility of wealth. Using the implicit function theorem the choice variables $\{C_t^0, C_t^1, T_t^L, T_t^H\}$ can be solved in terms of the state variables and T_t^I after substituting out λ_t from the first first-order condition. Illness time now has a formal role in this model and is solved by inserting H_t into the functional relationship which determines $T^I = f(H_t)$.

The role of future and past wages, prices, rates of time preference, interest rates, endowments and consumer tastes as well as other state variables is now made explicit in the individual's decision model. Once functional forms for utility and production are given, the structural links that are imbedded in the individual's life-cycle decisions as well as the structured role that past and future state variables have on the dynamic demands for consumption and time allocations are specified within the model. Simplifying assumptions on functional forms such as additive separability, and on discount factors and interest rates (for example, it is often assumed that $\gamma_t\beta = 1$ provides more structure on the demand equations that may allow for more transparent analytic interpretations, but in general the derivations must be carried out numerically.

2.1.3. Generalizations of the Grossman-type model

At the time of the Grossman model, there was considerable interest in contrasts between health and other forms of capital. Grossman himself noted the implicit constraint against depletion (negative investment) inherent with health, as opposed to

"pure" capital. Muurinen (1982) generalized Grossman's model, primarily by focusing on the depreciation factor δ, which he noted was likely to be endogenously related to choices made by the individual, and by addressing the issue of length of life as a choice variable by focusing on death as an event associated with subcritical values of health capital, the Grossman death stock, which is implicitly endogenous. By incorporating education into the vector of endowments X, Muurinen established a relationship between education and health, in which education increases as the depreciation rate decreases for health capital. Further, Muurinen was able to clarify the dynamic relationship between wealth and health as well as income and substitution effects in health demand associated with changes in initial wealth. These findings were seen by Muurinen as able to explain the negative income elasticities often reported for health demand. Grossman's (1972a,b) model predicts health to be a normal good, a finding not always found in empirical work on the topic. This may be due to the particular form in which Grossman stated the individual's budget constraint. By assuming identical preferences and allocating time based on income, the value of leisure time for those with lower incomes may be understated. Muurinen points out that this seeming inconsistency may be due to the particular definition of wealth and/or income as permanent or transitory. Moreover, assumptions of temporal separability may cloud the relationships among long run health consumption activities and those which are undertaken towards the end of the life-cycle when health is in decline.

Wolfe (1985) developed an extension to the Grossman model to account for retirement since the original Grossman model did not predict abrupt changes in the time allocation decision between work and leisure. Wolfe noted that in the "pure" model of Grossman, initial levels of health in excess of those whose rates of return were equal to their cost at the margin would disappear, and that a net wealth effect would be obtained by the individual instead, allowing health capital to depreciate over time. In other words, individuals work and defer substantial health investments until such time as the marginal benefits from investing in health equal the opportunity costs of forgone working time. Wolfe includes financial assets such as savings in his model, as a store for pure capital, and treats life expectancy as fixed for computational simplicity. He finds support for the observation that retirement age falls when productivity rises since productive people work harder and thus depreciate their health capital faster, and since productive people have high wages and may have more accumulated assets which allows them to leave the work force sooner.

2.1.4. Endogenous life expectancy

Shortly after the original Grossman (1972) model, Grossman and Benham (1974) began to address the issues of uncertainty which was posed in the original Grossman treatise, when they considered how wages relate to health. In their extended model, a lagged effect of health on wages was introduced. Extended treatments of uncertainty, the importance of which was noted was noted by Grossman, with respect to economic conditions (in particular, future prices) and life expectancy have been pursued by several authors in the context of adult and elderly health. For example, Hamermesh (1984) found that in the Retirement History Survey individuals work more and consume less if they expect to live longer. Hamermesh (1985) also found that

individuals extrapolate their life expectancies as life-tables change and are well informed of levels and changes in the current life-tables, although he noted that the subjective distribution of life expectancies has a larger variance than the actuarial counterpart, with the variance of the subjective distribution decreasing with age. The relative accuracy of subjective life expectancy probabilities also has been noted by Hurd and McGarry (1993) using the Health and Retirement Survey.

A formal treatment of life expectancy as a choice variable has been put forth by Ehrlich and Chuma (1990) who extended the Grossman framework by including the demand for longevity. Using a continuous-time setting, they overcame the paradox of life expectancy as both an endogenous outcome (Grossman, 1972) and as the finite horizon of the discrete-time dynamic programming problem. Ehrlich and Chuma noted that life expectancy cannot be marginalized "myopically", and must be considered as a fully endogenous variable in the life-cycle model. By so doing, they postulate an important economic consideration, that longevity itself is an economic good, as well as the dependence of the demand for longevity upon initial conditions, such as wealth. They also remind that the Fischer effect applies to health demand just like any other commodity and thus that real effects can be brought about by uncertainty about future prices. Finally, they note the importance of heterogeneity in rates of intertemporal substitution (β).

2.1.5. A model of uncertainty in the life-cycle health model

Decision-making under uncertainty characterizes life-cycle models of consumption and thus should characterize life-cycle models of health choice as well. Diamond and Hausman (1984) have examined the effect of two sources of uncertainty for adults workers, physical health and involuntary unemployment, on the timing of retirement using a subset of men aged 45-59 from the National Longitudinal Survey. They find health to be an important determinant of retirement while both private pensions and Social Security, whose effects are strongest at age 62 when benefits first become available, increase the probability of retirement. Anderson, et al. (1986) use a life-cycle, rational expectations model to test the effect of unexpected changes in health on retirement, based on data from the Retirement History Survey, and they show that retirement plans were significantly affected by unexpected health changes. Berger, et al. (1987) derive the relationship between the willingness to pay for health risk changes and the consumer surpluses associated with health changes which occur when there is certainty. They estimate this relationship empirically using survey interview data on 131 people in Denver and Chicago during 1984-1985. Bernheim (1990) has used the Retirement History Survey to test for rationality in expectations of future Social Security benefits following earlier research on the accuracy of such expectations by Bernheim (1988), and work on the accuracy of expectations concerning the timing of retirement by Burtless (1986), Anderson, Burkhauser, and Quinn (1986), Wolpin and Gönül (1987), and Bernheim (1989). He was unable to reject the hypothesis that innovations are unrelated to prior information and that expectations evolve as a random walk. Moreover, he notes that an implication of his findings that responses to new information just before retirement are highly rational, is that individuals recognize the links between labor supply decisions and benefit formulas at the margin, a point raised

by many researchers examining the retirement decision. Uncertainty in the supply of medical care is well documented and noted by Phelps (1992) to be due in large part to the public good aspect of medical information. Information concerning the marginal productivity of medical treatments is underproduced and the extent to which new information diffuses geographically and temporally is highly variable. Uncertainty and the demand for medical care also has been studied by Dardanoni and Wagstaff (1990), who modify Grossman's human capital model of health demand by introducing uncertainty involving illness and therapeutic efficacy. Although they do not pursue this issue empirically, in their comparative statics analysis they derive a Rothschild-Stiglitz increase in uncertainty: given that the average marginal product of medical care is unchanged or reduced as its riskiness increases, there is an increase in its demand. They conclude that health consumption is a normal good and consumers are risk-averse, consistent with the findings of Evans and Viscusi (1993) based on data on nonfatal consumer injuries. Moreover, as the expected therapeutic efficacy increases, Dardanoni and Wagstaff find that demand for medical care is reduced.

Sickles and Yazbeck (1995) have specified a modified Grossman dynamic programming model in order to evaluate the role of health, consumption, and leisure in life-cycle models with uncertainty and with exogenous wages and exogenous and known life expectancy. They use the framework of the infinite horizon programming problem subject to the usual transversality conditions. Assume that the individual faces exogenous real wages, and that at the beginning of the period realizations of the real wage, W_t , the real interest rate, r_{t-1} and the prices of the two composite consumption goods, P_{t-1}^0, P_{t-1}^1, are known but that future realizations are unknown and random. Abstract from the possibility of addictive goods and assume that the time allocation problem is between work and leisure, the latter to improve health production, that illness time is subsumed within leisure, and that there are no bequests. The individual's economic problem is:

$$(2.15) \qquad E_t\left(\sum_{t=1}^{LE}\beta^{t-1}U(C_t^o,T_t^L,H_t)\right)$$

The per-period intertemporal budget constraint is:

$$(2.16) \qquad A_{t+1}=\gamma_t(A_t + W_tT_t^W-C_t^0-P_t^1C_t^1)$$

where the numeraire price is that C_t^0. The time allocation constraint is:

$$(2.17) \qquad T = T_t^L+T_t^W$$

In order to allow for intertemporal nonseparability, a convenient form for the health capital equation is:

$$(2.18) \qquad H_t = h(C_t^1, T_t^L; X_t) + \alpha a_t$$

Here α measures the importance of past health on current health, and a_t is described below. In this formulation H_t is composed of two parts. The first is current investment which is created using leisure time, health related consumption and exogenous factors which could include endowments/heterogeneity. The second is the stock of past health produced over the life-cycle. In this formulation α measures the rate of technical substitution between current investment in health and the stock of past investment in the production of current health (Hotz, et al., 1988). Alternatively, the accumulation of the stock of health could be modeled the perpetual inventory approach used in the certainty models discussed earlier. In that specification, the level of health stock at time t is an update of period t-1 investment in health plus last period's depreciated health stock. The specification used in (2.21) allows for the possibility that the importance of past health relative to current health, α, may not be unity. The distributed lag specification is in keeping with the Hotz et al. (1988) model and allows for depreciation in health independent of the lagged health effects on current utility. The law of motion for a_t is given by:

$$(2.19) \qquad a_t = (1-\eta)a_{t-1} + H_{t-1}.$$

where η measures the rate of depreciation of the influence of past health on current health. Temporal nonseparability is introduced by including in health a distributed lag of past health investments in addition to the current period's health investment.

The maximization problem is stated in terms the value function at time t:

$$(2.20) \qquad V^t(A_t, a_t, W_t) = \max_{C_t^0, C_t^1, T_t^L} \left(U(C_t^0, T_t^L, H_t) + \beta E_t V^{t+1}(A_{t+1}, a_{t+1}, W_{t+1}) \right)$$

The first-order conditions with respect to C_t^0, C_t^1, and T_t^L are:
Using the envelope theorem and the law of iterated expectations, the Euler equations

$$\begin{aligned} & E_t[U_{C_t^0} - \beta\gamma_t V_A^{t+1}] = 0 \\ (2.21) \qquad & E_t[U_{H_t} h_{C_t^1} - \beta\gamma_t P_t^1 V_A^{t+1} + \beta V_a^{t+1} h_{C_t^1}] = 0 \\ & E_t[U_{T_t^L} + U_{H_t} h_{T_t^L} + \beta V_a^{t+1} h_{T_t^L} - \beta\gamma_t W_t V_A^{t+1}] = 0 \end{aligned}$$

can be rewritten as:

$$
\begin{aligned}
&E_t[U_{C_t^0} - \beta\gamma_t U_{C_{t+1}^0}] = 0 \\
&E_t[U_{H_t} h_{C_t^1} + \beta h_{C_t^1}[\alpha U_{H_{t+1}} + [(1-\eta)][U_{H_{t+1}} - \frac{U_{C_{t+1}^0}}{h_{C_{t+1}^1}}]]] = 0 \\
&E_t[U_{T_t^L} + U_{H_t} h_{T_t^L} - [U_{H_t} - \frac{U_{C_t^0} P_t^1}{h_{C_t^1}}] h_{T_t^L} - W_t U_{C_t^0}] = 0
\end{aligned}
\tag{2.22}
$$

Moreover, if one assumes that expectations are rational, then one-period ahead innovations ($\epsilon_{i,t}$) can be added to the derived Euler equations, where $E_t(\epsilon_{i,t}) = 0$, $\epsilon_{i,t}$, I=1,2,3 is orthogonal to the information set of period t, Ω_t, and where the forecast errors for a given individual are serially uncorrelated. Realizations of future random variables imply that:

$$
\begin{aligned}
&[U_{C_t^0} - \beta\gamma_t U_{C_{t+1}^0}] = \epsilon_{1,t+1} \\
&[U_{H_t} h_{C_t^1} + \beta h_{C_t^1}[\alpha U_{H_{t+1}} + [(1-\eta)][U_{H_{t+1}} - \frac{U_{C_{t+1}^0}}{h_{C_{t+1}^1}}]]] = \epsilon_{2,t+1} \\
&[U_{T_t^L} + U_{H_t} h_{T_t^L} - [U_{H_t} - \frac{U_{C_t^0} P_t^1}{h_{C_t^1}}] h_{T_t^L} - W_t U_{C_t^0}] = \epsilon_{3,t+1}
\end{aligned}
\tag{2.23}
$$

where $\epsilon_{t+1} = (\epsilon_{1,t+1}, \epsilon_{2,t+1}, \epsilon_{3,t+1})$ is the vector of forecast errors at period t. The model's parameters can be estimated by generalized (or simulated) method of moments (Hansen, 1982; McFadden, 1989; Pakes and Pollard, 1989), one of a set of estimators which we discuss in section 5, once functional forms for the utility function and the production function are specified.

The model's parameters can be estimated by generalized (or simulated) method of moments (Hansen, 1982; McFadden, 1989; Pakes and Pollard, 1989), once functional forms for the utility function and the production function are specified. We next formulate the generalized method of moments (gmm) estimator (Hansen, 1982),[9] exploiting in the choice of instruments, the fact that the variables in the information set Ω_t are orthogonal to u_{t+1}. We assume transcendental logarithmic functional forms for both the utility and production functions. The utility function is

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[9] An alternative approach for estimation is to use a nested fixed point (NFXP) algorithm developed by Rust (1988).

$$(2.24)\quad U(C_t^o, T_t^L, H_t)=\zeta_1 lnC_t^0+\zeta_2 lnT_t^L+\zeta_3 lnH_t+\zeta_4 lnC_t^o lnT_t^L+\zeta_5 lnC_t^o lnH_t$$
$$+\zeta_6 lnT_t^L lnH_t+\frac{\zeta_7}{2}(lnC_t^o)^2+\frac{\zeta_8}{2}(lnT_t^L)^2+\frac{\zeta_8}{2}(lnH_t)^2$$

with corresponding marginal utility functions

$$(2.25)\quad U_{C^o}=(\zeta_1+\zeta_4 lnT_t^L+\zeta_5 lnH_t+\zeta_7 lnC_t^o)/C_t^o$$

$$(2.26)\quad U_h=(\zeta_3+\zeta_5 lnC_t^o+\zeta_6 lnT_t^L+\zeta_9 lnH_t)/H_t$$

$$(2.27)\quad U_{T^L}=(\zeta_2+\zeta_4 lnC_t^o+\zeta_6 lnH_t+\zeta_8 lnT_t^L)/T_t^L.$$

The health production function is

$$(2.28)\quad h_t(C_t^1, T_t^L)=\Gamma_1 \ln C_t^1+\Gamma_2 lnT_t^L+\Gamma_3 \ln C_t^1 lnT_t^L+\frac{\Gamma_4}{2}(\ln C_t^1)^2+\frac{\Gamma_5}{2}(lnT_t^L)^2$$

with marginal products

$$(2.29)\quad H_{C^1}=(\Gamma_1+\Gamma_3 lnT_t^L+\Gamma_4 lnk_t C_t^1)/k_t C_t^1$$

$$(2.30)\quad H_T^L=(\Gamma_2+\Gamma_5 \ln T_t^L+\Gamma_3 \ln C_t^1)/T_t^L$$

Next letting the parameter vector be

$$\theta_0=(\zeta_1, \zeta_2, \zeta_3, \zeta_4, \zeta_5, \zeta_6, \zeta_7, \zeta_8, \zeta_9, \Gamma_1, \Gamma_2, \Gamma_3, \Gamma_4, \Gamma_5, \alpha, \eta, \beta),$$

and letting X_{it} be the vector of variables entering the i^{th} individual's first-order conditions in period t, we can express the (1 x 3) system (15)-(17) as $f(X_{it}, \theta_0) = \epsilon_{i,t+1}$. Rationality and its implication that information in Ω_{it} is of no help in forecasting future shocks implies that $E[f(X_{it}, \theta_0)Z_{it}] = 0$, where Z_{it} is a $3xh$ ($h \geq \dim(\theta_o)$) matrix of elements of Ω_{it}. The population orthogonality conditions for the years that the panel data are available can be derived by averaging over time,

$$(2.31) \qquad E\frac{1}{T}\sum_{t=1}^{T}[f(X_{it},\theta_0)Z_{it}]=E[M(X_i,Z_i,\theta_0)],$$

Sample analogues are then constructed by averaging over the random sample of N individuals,

$$(2.32) \qquad O_N(\theta_0)=E\frac{1}{N}\sum_{i=1}^{N}[M(X_i,Z_i,\theta_0)],$$

and gmm estimates of θ_0 are defined as the

$$ARGMIN\,[o_N(\theta_0)\,W_N O_N'(\theta_0)],$$

where W_N is the symmetric positive definite weighting matrix

$$(2.33) \qquad W_N=W_N^*=S_N^{-1}=[\sum_{i=1}^{N}M(X_i,Z_i,\hat{\theta}_0)'M(X_i,Z_i,\hat{\theta}_0)]^{-1},$$

and where consistent first step estimates of θ_0 are based on setting the weighting matrix W_N to the identity matrix.

The asymptotic covariance matrix for the gmm estimator is

$$(2.34) \qquad \Phi=(D_N'S_N^{-1}D_N)^{-1},D_N=\sum_{i=1}^{N}(\partial M(X_i,Z_i,\hat{\theta}_0)/\partial\theta_0).$$

Now that the model is laid out we can estimate it using the data from the Retirement History Survey, but first, a more in depth discussion of the RHS is warranted.

2.2. The Retirement History Survey and Variable Construction

The Retirement History Survey was started in 1969 with about 11,000 men and women. At that time it was a nationwide random sample of heads of households aged 58-63. The sample members were reinterviewed every two years through 1979. We have constructed a longitudinal file from the interviews through 1979. The RHS contains substantial information on the respondents and their spouses, including age, education, wealth, current earnings, pensions, Social Security benefits, earnings covered by Social Security annually for the period 1951-1976, number of children,

current and previous occupation, marital history, spouse's earnings, health status, medical usage, retirement status and plans, nutrition, and some aspects of life style including contact with children.

Advantages of using the RHS for the analysis of this chapter include: (1) it contains substantial information on respondents and spouses; (2) it is a random draw of the population of heads of household aged 58-63 in 1969; and (3) it contains information including indicators of health status that permit the estimation of some critical structural parameters underlying the micro health determinants of older males.[10] Some of the critical variables that we use for the estimates in this chapter are constructed from the raw data in ways that we will define below, and the data on the variables used directly in the estimation is summarized in Table 2.1.

Health status: How to measure health status is a complicated problem. Ideally, a measure of health should reflect the individual's physical and mental well-being using a standardized index. Unfortunately, such indices cannot be constructed from information in most data sets, forcing researchers to rely on subjective and objective qualitative proxies for health status. Subjective measures include information collected from individuals about their own health, while objective measures describe health information collected from a source other than the individual in question. Two typical examples of subjective health measures for the elderly are answers to questions such as: "Is health a reason for your retirement?" or "Would you say that your health is better, same as, or worse than people your age?". A number of problems exist with these measures. First, poor health is a socially acceptable reason for retirement and therefore it is possible that an individual may cite it as a reason for retirement even when it is not. Second, the subjectivity of the answer makes it difficult to compare results among the individuals. Third, some retirement benefits are a function of an individual's well-being which creates an incentive for inaccurate description of one's health status. These problems with the subjective measures lead to biases in estimation but would seem to pull in opposite directions.

Objective measures of health also are not without their problems. The most often used objective measure is mortality information. The fact that an individual died within a follow-up time interval may seem more objective than a self-reported health measure, but may not be an accurate description of the impact of health at the initial time since deaths that occur suddenly from an accident or a disease may have little or no impact on health while the individual was alive. One argument for the use of subjective measures is that they have biases working in opposite directions while objective measures have biases that may not cancel out. Another argument for the use of self-reported measures can be found in the public health literature where subjective ratings by the elderly were found to be highly correlated with their physician's ratings.[11] Maddox and Douglass (1973) compare own and doctor evaluation of health six

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[10] Missing observations for the variables used in our dynamic life-cycle health model reduced the number of complete observations for men to about 5400. The number of women was about 1600. The data requirements to estimate our highly nonlinear dynamic life-cycle model are substantial. Because of this relatively small number of complete observations for women and the large data requirements for consistent estimation of our model, we restrict our analysis in this chapter to men.

[11] See Ferraro (1980) and Mossey and Shapiro (1982).

different times in a fifteen year study. The study begins with 270 noninstitutionalized people aged 60 and over, and by the end of the sample period it drops to 83 people. They find a positive correlation in the two sources of health ratings but with some tendency for people to overstate their health. They also find stability over time, and that self reported health is a better predictor of the future findings of doctors than the reverse.

We combine subjective and objective measures of health in an index constructed along the lines of the Quality of Well-Being index (QWB) developed by Kaplan, et al.[12] The QWB combines four scales that measure mobility, physical activity, social activity, and symptom/problem complexes. In developing the QWB, Kaplan and Anderson (1988) integrate morbidity and mortality, building on a considerable body of theory in economics, psychology, medicine, and public health. Three steps describe the index and its development.

In the first step a comprehensive study was undertaken to enumerate the links between disease and injuries and behavior and role performance. Then three scales were constructed representing related but distinct aspects of daily functioning: Mobility, Physical Activity, and Social Activity. The second step focused on subjective complaints as a important component of a general health measure leading to the development of a fourth index relating to symptom/problem complexes. The third step integrated the three scales and the subjective index into a single index. In constructing this final expression, weights were assigned to the various levels within each scale and among scales based on measured preferences of health states or "quality" judgments from a representative random sample of 866 individuals who were asked to evaluate the relative desirability of specific health conditions. The estimated shadow values of health conditions were found to be quite stable over different stratified subsamples[13]. These shadow values were then used as weights in the QWB index.

The RHS allows us to construct directly the first three scales. The conditions which make up these scales are given in Table 2.2. Detailed information needed to construct the symptom/problem scale is not available in the RHS directly. We thus modify the subjective index by using the evaluation of heads of households' health relative to people their age augmented with death information from the RHS and the Social Security records. The subjective/objective health status variables are discussed at length in Sickles and Taubman (1986, 1997). We assigned to these four health status (health better/same/worse than those the same age and respondent died during the same period) the Kaplan and Anderson symptom/problem weights using a weight of zero for health the same, the group symptom/problem complex weight for health worse, its negative for health better, and assigned an index of 1 (the QWB index ranges from 1 to 2) for those who died during the sample period.

Leisure: A number of alternatives for measuring leisure are possible given the data in the RHS. One is a trichotomous variable indicating whether an individual is retired, semi-retired or still working full-time. Another is to drop the semi-retirement observations and construct a dummy variable of the remaining two outcomes.

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[12] See Kaplan and Bush (1982), Kaplan and Anderson (1988), Anderson, et al. (1989).

[13] A description of the empirical work can be found in Kaplan and Anderson (1988).

However, the most often used index in empirical labor studies of the elderly is the working-full-time/not-working-full-time dichotomy.[14] Sickles and Taubman (1986) test the alternative definitions in a study on health and retirement and chose the last option.[15]

Another way of representing the level of leisure is by looking at the number of hours worked and subtracting it from total available hours. The RHS allows us to construct such a variable since respondents are asked about the hours of work per week in both their part-time or full-time jobs. We construct the leisure variable based on yearly hours of work. Actual hours of work for the elderly carries with it a discreteness that makes implementation of the continuous time-continuous state-space optimization framework used herein problematic. Although alternatives exist to deal with the discrete nature of labor force participation (Mitchell and Fields, 1984; Hotz, Miller, Sanders, and Smith, 1992; Hotz and Miller, 1993) we choose instead to construct a variable that measures the level of *desired* leisure. This is done by first estimating tobit reduced form models for leisure for each year. We then constructed individual estimates of hours worked for each year and refer to this as desired hours. This construction obviates the need to deal with an additional discrete control variable, the no-work state, in the dynamic programming problem and should generate measurement errors that are orthogonal to data not in the contemporaneous information set. The fact remains, however, that the highly nonlinear nature of the leisure/work trade-off for those entering the retirement transition may not be adequately captured in these nonlinear tobit estimates (Burtless and Moffit, 1985)

Major consumption categories: The wealth of variables in the RHS also allows us to construct the nonhealth consumption and health-related consumption variables. The nonhealth consumption variable (net of housing expenditures, food, and clothing) consists of expenditures on gifts, entertainment, charitable organizations, social organizations, transportation, vacations, trips, utilities, and non-food grocery purchases. Health-related expenditures include out-of-pocket doctors bills, hospital stays, prescription drugs, and other medical expenses and co-payments for medical treatment.

Variables for instruments: The set of variables that we use as instruments at time t are contemporaneous values of the hourly wage rate for those who work and the hourly wage on last job for those who are not working, number of people in the household, expected Social Security benefits, education, dummies for currently married, widowed, divorced/separated (excluded category is never married), a dummy for non-white, dummies for longest occupation in the professions or in management, and time dummies for 1969, 1971, 1973, 1975, and 1977. We delete those whose longest occupations are in farming or in the military. Social Security benefits are those one would expect to receive if retirement were to begin in the respective year. They are computed using covered earnings taken from each person's Social Security record,

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[14] Hotz, Miller, Sanders, and Smith (1994) propose an estimator for stochastic dynamic discrete choice models using the method of simulated moments which may prove to be an attractive alternative to our econometric specification. An alternative likelihood based procedure referred to as the conditional choice probability estimator recently has been proposed in a similar context by Hotz and Miller (1993).

[15] The authors found comparable results when they used the alternative definitions. They chose the full-time-work versus less-than-full-time-work because of simplicity and comparability with other studies.

which is part of the RHS, and then replicating the Social Security rules. To do this we first calculated each person's Average Monthly Earnings (AME) by using the respondent's earnings since 1951, which was truncated at the maximum allowable earnings level. The five lowest years of income were dropped and the sum of the remaining incomes was divided by the number of months worked. The resulting AME was then used to compute the Primary Insurance Amount (PIA) based on the tables in the *Social Security Handbook.* These account for inflation and therefore change over the 1969-1979 sample period. Once PIA was computed, the benefits total was determined on the basis of PIA and marital status. By using benefits available rather than those paid to actual retirees, we avoid an obvious selection problem.

2.3. New Estimates of Utility and Health Production Parameters

We estimate the Euler equations presented in Section 2.2 using the data described in Section 2.3. The gmm estimates of the system of Euler equations (2.15)-(2.17) are presented in Table 2.3.[16] The discount factor β is set equal to 0.95, while the real interest rate is equal to the average real 3-year treasury bill rate over each sample period. The number of overidentifying restrictions is h-k=18, where k=dim(θ)=15 and h=number of orthogonality conditions (11 for each of the 3 equations). The Hansen chi-squared test statistic for the overidentifying restrictions is χ^2=1.89 ($\chi^2_{.95,18}$=28.87). The patterns of sample average marginal utilities of health based on equation (2.20) show a rather consistent pattern among cohorts and over time. Estimates in 1969 for the cohorts ages 58-63 are 0.0669, 0.0663, 0.0653, 0.0651, 0.0653, 0.0641, 01, 0.0713, 0.0712, 0.0579 while estimates in 1979 for cohorts of individuals still alive indicate a marginal utility of health of 0.0579, 0.0556, 0.0530, 0.0517, 0.0527, 0.0498, indicating some diminishing returns both between and within cohorts. Sample average estimates (at the median time period) for the marginal utility of a thousand dollar increase in health neutral consumption are 0.0327, 0.0311, 0.0236, 0.0354, 0.0128, 0.0461, for the 58-63 cohorts (standard errors are 0.00214, 0.00212, 0.00258, 0.00489, 0.00248, 0.00703). Comparable average estimates of the marginal utility of a thousand hours of additional annual leisure based on eq. (21) are 0.0210, 0.0236, 0.0251, 0.0188, 0.0267, 0.0181 (standard errors are 0.00094. 0.000869, 0.000929, 0.00158, 0.000795.

Of particular interest are the health production results which are revealed in part by the coefficient estimates of Γ_1 -Γ_5. We analyze the health production results as the cohorts age during the sample period. Tables 2.4 and 2.5 report the health elasticities of desired leisure and health related consumption respectively for each age cohort. Both sets of elasticities are positive across cohorts and time, indicating a significantly positive contribution of desired leisure and health related consumption to better health over the life-cycles represented in the RHS. Our results are in agreement with two-stage least results of Grossman (1972) who shows that the sign of the medical care coefficient is reversed when reverse causality is appropriately dealt with and with Rosenzweig and Schultz (1983a, 1988, 1991) and Grossman and Joyce (1990) in the context of birthweight production. We found that the magnitude of estimated health

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[16] The coefficient on the logarithm of consumption of nonhealth related goods and services was normalized at unity.

elasticities with respect to leisure are rather stable, ranging between 0.59 and 0.073 with some slight upward trend over time and for older cohorts. The health elasticity of health related consumption is between 0.045 and 0.031. The pattern of consumption elasticities indicates some increase over time for each cohort suggesting the increased importance of health related consumption in the production of health as individuals reach the penultimate event.

Finally, the estimate of α, which measures the weight of past health in current utility, is rather small at 0.099 indicating the relatively short-lived memory that agents have of past health when evaluating current preferences. The estimate of η is 0.308 and measures the rate of depreciation of the influence of past health on current utility. The relative importance of past health to current health can be assessed by calculating $\eta/(\alpha+\eta) = 0.757$, which indicates a substantial role for dynamics in the health production function.

2.4. Conclusion

In this chapter we have examined in the context of a structural model how rational individuals choose among various goods, the time path of health, and implicitly the length of life. We assume that individuals wish to maximize their utilities, subject to intertemporal budget and health production function constraints. The choices in part show up in the demand functions for various goods and services and in people's health stocks. We also presented estimates of utility and health production function parameters that utilize the structure outlined in this chapter. When the health stocks of individuals fall below a threshold, people die. We concentrate on using a hazard function model in the next chapter and then do the estimation of the mortality hazard in Chapters 4 and 5.

Table 2.1 Sample Summary Statistics

Variable	Mean	Standard Deviation
Desired Hours/Yr.	4751.6	1561.1
Other Consumption/ Yr. discretionary)	2143.1	3241.6
Out of pocket Health Consumption/ Yr.	326.9	701.9
Health Status Index	1.61	0.28
Hourly Wage	5.01	4.83
Number in the Household	2.35	1.16
Expected S.S. Benefits	1570.8	1610.2
Years of Education	9.75	3.61
Married	0.77	0.42
Widowed	0.15	0.36
Divorced/Separated	0.03	0.17
Black	0.09	0.29
Professional	0.19	0.39
Management	0.16	0.36

Table 2.2 Quality of Well-Being: Mobility, Physical Activity, and Social Activity Scales

Step Number	Index Definition	Weight
	MOBILITY SCALE	
1	No limitations for health reasons	0.0
2	Did not drive a car, health related; did not ride in a car, and/or did not use public transportation, health related; or had or would have used more help than usual for age to use public transportation, health related	-0.062
3	In hospital	-0.090
	PHYSICAL ACTIVITY SCALE	
1	No limitations for health	0.0
2	In wheelchair, moved or controlled movement of wheelchair, moved without help from someone else; or had trouble or did not try to lift, stoop, bend over, or use stairs or inclines, health related; and/or limped, used a cane, or walker, health related; and/or had any other physical limitations in walking, or did not try to walk as far or as fast as others the same age are able, health related	-0.060
3	In wheelchair, did not move or control the movement of wheelchair without help from someone else, or in bed, chair, or couch for most or all of the day, health related	-0.077
	SOCIAL ACTIVITY SCALE	
1	No limitations for health reasons	0.0
2	Limited in other (e.g., recreational) role activity, health related	-0.61
3	Limited in major (primary) role activity, health related	-0.61
4	Performed no major role activity, health related, but did perform self-care activities	-0.61
5	Performed no major role activity, health related, and did not perform or had more help than usual in performance of one or more self-care activities, health related	-0.106

Table 2.3 Parameter Estimates and Standard Errors (Desired Leisure Model)

ζ_2	-0.2171 (0.1027)	Γ_1	-0.1604 (0.0246)
ζ_3	-1.0573 (0.7795)	Γ_2	0.1164 (0.0855)
ζ_4	-0.1108 (0.0136)	Γ_3	0.0051 (0.0045)
ζ_5	0.0231 (0.0034)	Γ_4	0.0405 (0.0214)
ζ_6	-0.0185 (0.0102)	Γ_5	0.2863 (0.1002)
ζ_7	-0.0137 (0.0033)	α	0.0990 (0.0711)
ζ_8	0.1220 (0.0395)	η	0.3077 (0.1212)
ζ_9	1.1473 (0.1482)	$\eta/(\alpha+\eta)$	0.7566 (0.2609)

Table 2.4 **Desired Leisure Elasticity of Health Production** (entries are cohort means with standard deviations in parenthesis)

Age/Year	69	71	73	75	77	79
58	0.6234 (0.0016)					
59	0.5920 (0.0018)					
60	0.5942 (0.0017)	0.6281 (0.0018)				
61	0.5953 (0.0021)	0.5991 (0.0019)				
62	0.5984 (0.0019)	0.6009 (0.0019)	0.6396 (0.0020)			
63	0.6001 (0.0022)	0.6066 (0.0021)	0.6061 (0.0020)			
64		0.6068 (0.0022)	0.6116 (0.0021)	0.6333 (0.0019)		
65		0.6132 (0.0022)	0.6202 (0.0022)	0.6035 (0.0018)		
66			0.6075 (0.0024)	0.6148 (0.0019)	0.6501 (0.0020)	

Age/Year	69	71	73	75	77	79
67			0.6111 (0.0028)	0.6197 (0.0022)	0.6249 (0.0022)	
68				0.5952 (0.0027)	0.6341 (0.0023)	0.6926 (0.0026)
69				0.5961 (0.0030)	0.6421 (0.0026)	0.6648 (0.0028)
70					0.6048 (0.0032)	0.6851 (0.0029)
71					0.6080 (0.0036)	0.6885 (0.0031)
72						0.6518 (0.0040)
73						0.6541 (0.0045)

Table 2.5 **Health-related Consumption Elasticity of Health Production** (entries are cohort means with their standard deviations in parenthesis)

Age/Year	69	71	73	75	77	79
58	0.03865 (0.00058)					
59	0.03810 (0.00051)					
60	0.03759 (0.00051)	0.03926 (0.00065)				
61	0.03940 (0.00053)	0.03988 (0.00057)				
62	0.039203 (0.00054)	0.03844 (0.00059)	0.03399 (0.00066)			
63	0.03900 (0.00058)	0.04025 (0.00060)	0.03474 (0.00047)			
64		0.04010 (0.00057)	0.03338 (0.00047)	0.04183 (0.00067)		
65		0.03695 (0.00059)	0.03080 (0.00048)	0.03984 (0.00055)		

Age/Year	69	71	73	75	77	79
66			0.03106 (0.00046)	0.04074 (0.00055)	0.04048 (0.00072)	
67			0.03119 (0.00050)	0.03888 (0.00055)	0.04042 (0.00062)	
68				0.03928 (0.00052)	0.04087 (0.00061)	0.04372 (0.00077)
69				0.03859 (0.00056)	0.04033 (0.00067)	0.04431 (0.00067)
70					0.04054 (0.00059)	0.04553 (0.00069)
71					0.03955 (0.00069)	0.04492 (0.00075)
72						0.04514 (0.00070)
73						0.04471 (0.00079)

3

Statistical Techniques for Estimation of Hazard Functions

The use of aggregate data to infer group risk factors in mortality and morbidity determination clearly raises questions concerning which variables are endogenous and which are exogenous, a point recently remade by Manski (1993). Although aggregate level studies based on micro level decisions have been undertaken through calibration and simulation [see, for example, Auerbach, et al. (1989) in their study of four OECD countries], estimation of such general equilibrium models is problematic given the enormous data requirements. In the last chapter we outlined a microeconomic model of individual behavior which made explicit the causal links between mortality and morbidity, and which allowed control for the risk factors which give rise to changes in health. The need for such structural modeling of the risk factors which cause variations in health outcomes has been noted by many authors, a recent example being Feinstein (1992) in his survey of health outcomes and socioeconomic status. In these models, the allocation of time and the income it generates and health status are rationally chosen under constraints of scarcity, technology and uncertainty.

One might question the assumption that an increasingly large segment of elderly consumers, those in nursing homes, could be viewd as making the rational choices assumed in economic models of rational choice. Evidence that they do in fact behave in a manner consistent with consumer rationality can be found in Nyman (1989). For a theoretical treatment of an alternative choice problem in which individuals do not have the information processing capacity to compare all feasible allocations but rather adjust allocations myopically, see de Palma et al. (1994). These concepts may be disconcerting at first to the non-economist. If so, consider that the dramatic growth in the relative percentage of the labor force who work part-time or are self-employed suggests that individuals increasingly make decisions to maximize their benefits resulting from their allocation of their time. As for time of death as chosen, consider suicide, living wills, the refusal by Christian Scientists of potentially life-preserving medical care, and the shortfall of deaths before personally significant dates, with a subsequent spike thereafter. Examples are birhtdays, anniversaries, and birth of a

grandchild. Simultaneity between income and death may be seen in the observation that people require higher pay to work in occupations for which more deaths occur (Thaler and Rosen, 1975).

The most widely used set of alternatives to such structured modeling of optimal individual level decision making are based on life-table analyses of mortality. These alternative models utilize an analytical framework in which death is viewed as an event whose occurrence is probabilistic in nature, although individual choices may have contributed to the relative risk of the event of death occurring. For excellent surveys of survival model methods see Kalbfeisch and Prentice (1980), Kiefer (1988), and Lancaster (1990). These analyses require a characterization of the state of the individual as represented by various factors- acquired, environmental, or behavioral- combined with the survival status of the individual at the end of the data reporting period. Typically, a life-table analysis examines an age cohort of individuals, distinguished by a particular risk factor status. Observed age-specific death rates are compared with those expected from all causes using a chi-squared contingency table approach. This is the basic approach pursued by a large body of demographic research during the last four decades (Dorn , 1958; Doll and Hill, 1964; Kitagawa and Hauser, 1973; Rogot et al., 1992). The null hypothesis tested is usually the independence of risk-factor status and rate of death.

Although the life-table has been widely used as a survey prediction of an individual's risk of death, this technique suffers in design in that individuals are not required per se to survive in any consistent manner, as age-specific death rates are calculated independently of each other. We know, however, that there is an important additional structure within the data known as senescence, or increasing death rates with age which, if ignored, can also bias estimates based on structural models of health. This term has also been used by demographers and epidemiologists to apply to a list of concepts which distinguish different kinds of deaths, such as those which are caused by endogenous or exogenous factors and those which are premature instead of senescent (Stoto and Durch, 1993). Use of known senescent trends in the survival data allows the researcher to extend the instantaneous death rates from life-tables to a model of long-term survival which can be estimated from longitudinal and panel data and which in turn allows for the effects of more complicated socioeconomic factors on mortality to be evaluated.

An important shortcoming of survival or hazard function estimates from life-tables concerns the time interval of the observations. Although events are often assumed to evolve in continuous time, data are compiled only periodically and discretely. Right-censoring (or for that matter left-censoring), due to the presence of underlying frailties undetected because of an often arbitrary choice of the data capture interval, may confound the investigator's attempt to properly deal with unobserved heterogeneity in mortality hazards (Heckman and Singer, 1984; Manton, et al., 1986). A similar problem may exist in retrospective analyses of survival rates for the elderly using previous cohort experiences (Thatcher, 1992).

One of our primary concerns in this book is estimating relations for age-specific death rates. During recent years, substantial advances have been made in the methods used to analyze data on age-specific death rates. To put the matter in perspective, in the 1970's Kitagawa and Hauser (1973) used simple cross-classifications and expected versus actual death rates in what was then the state-of-the-art technology for mortality

analysis. Moreover, recently Hrubec and Neal (1981), Madans, et al. (1986a), and Kaprio and Koskenvuo (1990) have used the same technique.[17] However, it is now possible to estimate a variety of hazard rate models, which are more informative.[18] We utilize these methods in the next two chapters.

This chapter is primarily technical background material for the next two chapters and covers many issues that are important in estimating hazard models. Readers who are interested primarily in our substantive results on mortality may wish to skim this chapter or go directly to Chapter 4. We first sketch out the main topics that we cover and then turn to technical details.

3.1. Introduction to Hazard Models

The hazard rate for mortality in a time period is defined as the percentage of people alive at the beginning of a time period who die during the time period. A hazard model describes how death rates vary both over time and across personal characteristics. There are two general classes of hazard models -- the proporational hazard and the accelerated-time-to-failure model.

The proportional hazard model has a base line function that gives the overall relationship of age-specific death rates to age and indicates the cumulative probability of being alive at any age. The model also lets these death rates vary across measured variables that we treat as exogenous, and which in our analysis are person specific, e.g., education. The more and less educated have their own hazard functions but each is proportional to the base line hazard and will terminate at the same age.

We employ at different points two different proportional hazards -- the Cox and the Weibull -- with somewhat different statistical assumptions. The major practical difference is that the Weibull allows one to determine if "unobserved heterogeneity", such as innate robustness, is important.

The accelerated-time-to-death models in general allow a nonproportionate change in the mortality hazard for a change in the covariates, so that, e.g., the less educated may die younger. To estimate this model, a functional form that describes the distribution of age-specific death rates must be specified. This same distribution also must apply to the people who have not died by the end of the sample period or are truncated. Because some people are alive, the average age of death in the sample is a biased estimate of the true average, which can be calculated given that we estimate the parameters that describe the age-specific death rate distribution.

This distribution has a positive relationship to age; hence, we do not use the exponential distribution which implies a constant death rate. We use instead the Weibull, lognormal, loglogistic and generalized gamma.

An important issue considered here and in our empirical work is heterogeneity, which means that a different age-specific death rate distribution applies to various individuals or groups. Above we suggest that it is possible that we may find variables in our data set, e.g., education for which the hazard rates differ. However, there may

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[17] Section 4.1 presents more detail on these and other related studies.

[18] Hazard rate analysis is generally dated to Cox (1972).

be other unmeasured differences in what sometimes is called "frailty". We explore several parametric and non-parametric methods to control for unobserved heterogeneity.

In the previous chapter we examined life-cycle models of health demand and estimated the equations using RHS. To see a relation between that and the hazard models, note that the solution to the equilibrium path of individual health stocks in Section 2.2 can be linked to the mortality state by introducing a stochastic rule for observing death. Define the mortality state at time t (M_t) as

$$(3.1) \qquad M_t = \begin{cases} 1, & \textit{if } \epsilon_{it} > H_t \\ 0, & \textit{otherwise} \end{cases}$$

where ϵ_{it} is an individual-specific, time-period-specific threshold for the (local) equilibrium health index. Let the probability that an individual is alive at the beginning of the period be $[1\text{-}F(H_t)]$ and assume that the arrival of shocks, ϵ_{it}, follows a Poisson process. Then the probability that a new value of ϵ_{it} occurs during the period $(t,t+\Delta)$ is $p=\psi\Delta+o(\Delta)$. The hazard of dying during the period is

$$(3.2) \qquad \lambda(t) = \psi[1-F(H_t)],$$

and the survivor function becomes

$$(3.3) \qquad S(t) = \exp(-\psi[1-F(H_t)]t).$$

The choice of ψ and the distribution for the level of the shocks determine the form of the hazard. If the level of shocks is exponential with density $f(\epsilon) = \exp(-\epsilon)$, then $F(H_t) = 1\text{-}\exp(-H^t)$ and for $\psi = \mu t^{\theta-1}$, the hazard of dying at time t is given

$$(3.4) \qquad \lambda(t) = \theta t^{\theta-1} \exp(-H_t),$$

where H_t is given in (3.1) above. Equation (3.4) is the Weibull proportional hazard.

Following Vaupel et al. (1979), Heckman and Singer (1984), Manton et al. (1986), and Vaupel (1988) we can also allow for unobserved heterogeneity in genetic predispositions to death. As pointed out by a number of authors, failure to control for unobserved individual specific frailties can bias downward estimates of duration dependence in mortality hazard models, and in so doing confound the natural ordering between the propensity to die and morbidity states as well as (potentially) the impacts of other covariates. Alter and Riley (1989), for example, using mortality and morbidity

data from British "friendly societies" in the nineteenth century, note that decreases in cohort age-specific mortality rates are observed over time because more frail individuals survived to reach old age due to advances in medical technology, while morbidity increased as these same individuals became more susceptible to non-fatal illnesses.

Treatments for heterogeneity which may be correlated with other covariates have utilized within type transformations of the linear probability model (see, for example, Rosenzweig and Schultz, 1993) or for the alternative duration time model (Olsen and Wolpin, 1983). Instrumental variable estimators can also be used in a natural way to deal with the presence of endogenous choice variables. Because these models are linear, the complications that arise when the mortality state is linked to the covariates by the nonlinear logit or probit transformation can be circumvented and consistent standard errors can be based on a White (1980) type estimator. Fixed effect treatments for logit type specifications (dead/not dead) have been pursued by Chamberlain, (1980, 1983) and for tobit type specifications (censored length of life) by Honoré (1992).

We also investigate in this chapter some estimation procedures that have not been used much in previous work. Two examples are a simulation based method and a maximum penalized likelihood function. The latter is another approach to unobserved heterogeneity. Finally we present some Monte Carlo experiments that examine the small sample properties of some of the little studied estimators. In this chapter we provide formal methods on how to estimate these models and estimation techniques.

3.2. Estimation of Survival Hazard Models with Heterogeneity

Consider first the continuous time duration model in which a nonnegative random variable T, say, time until death, has a density, f(t), and a cumulative distribution, F(t) (Kalbfleisch and Prentice, 1980; Lancaster, 1990). The hazard for T is the conditional density of T given T > t | 0 and is given by:

$$(3.5) \qquad \lambda(t) = f(t|T>t) = \frac{f(t)}{[1-F(t)]} \geq 0.$$

In terms of the integrated hazard, the density and distribution of T are:

$$(3.6) \qquad f(t) = \lambda(t) \exp[-\int_0^t \lambda(\tau)d\tau]$$

$$(3.7) \qquad F(t) = 1-\exp[-\int_0^t \lambda(\tau)d\tau].$$

The distribution associated with realizations on δ is assumed to be independent of the survival time and is functionally independent of the survival distribution. The log likelihood function is:

$$(3.8) \qquad \ln L = \sum_i f(t)(1-\delta) + \sum_i [1-F(t)]\delta$$

Failure to control for unobserved frailities causes a downward bias in duration dependence. Moreover, as is well known misspecifying either the hazard or the frailty distribution leads to inconsistent estimates of the covariate effects. The hazard process being modeled is highly nonlinear and a failure to properly specify the nonlinearity biases coefficient estimates (White, 1980). Either ignoring or improperly specifying the distribution of measurement error in even linear models causes parameter estimates to be inconsistent. It is no surprise that the potential for both problems might put applied researchers in a very uncomfortable position when evaluating results using standard parametric estimators.

Possible solutions to these problems have followed two separate approaches. The first, used by Manton, et al. (1986), assumes a flexible parametric distribution for those frailty differences among individual that enter the hazard multiplicatively. The second, proposed by Heckman and Singer (1984), allows for the distribution of frailty differences to be estimated by a finite support general probability estimator (Keifer and Wolfowitz, 1956). This estimator is consistent and approximated standard errors also can be generated (Heckman and Singer, 1984).

One of the more widely used mortality specifications is the proportional hazard model which expresses the natural logarithm of the conditional hazard of dying as a function of time. The accelerated time to failure model specifies the natural logarithm length of life as a linear function of covariates, $\ln(T) = x\beta + \sigma\epsilon$, where ϵ is a random disturbance and σ is a scale parameter. Failure time can be written as $T = \exp(x\beta)T_0^{\circ}$ where T_0 is an event time drawn from a baseline distribution. Different parametric distributions are available to model unobserved genetic frailities [$\theta(t)$]. Two parametric distributions that are popular to use for $\theta(t)$ are the normal and the inverse Gaussian. The former has an obvious genetic rational and is parsimonious in the first specification of the conditional hazard above. As noted by Manton, et al. (1986, p. 637), the inverse Gaussian provides a mixture that is quite flexible and allows for a very general description of the continuous variability in biological risks and is parsimonious with the latter.

To see how these statistical treatments can be implemented, consider the Weibull proportional hazard mode for individual i:

$$(3.9) \qquad h(t_i|x_i,\theta_i) = \exp(\gamma \ln t_i)\exp(x_i\beta + \theta_i)$$

The log hazard function is given by:

$$(3.10) \qquad \ln h(t_i|x_i, \theta_i) = \gamma lnt_i + x_i\beta + \theta_i,$$

where t_i is the continuous time of a completed spell, x is a vector of exogenous possibly time varying covariates, and where unobserved scalar heterogeneity is θ_i. Censored observations are given by:

$$(3.11) \qquad T_i = \min (t_i, t_c), \quad d_i = I(t_i < t_c),$$

where t_c is the censored time of an incomplete spell and I is an indicator function: $d_i = 1$ if $t_i \leq t_c$ and $d_i = 0$ otherwise. Other specifications of the conditional hazard can be considered, but for pedagogical reasons and due to its widespread use in applied analysis, we use the Weibull proportional hazard to motivate our discussion.

Assuming independence over duration spells, the joint likelihood of duration times and unobserved heterogeneity can be written as:

$$(3.12) \qquad L = \prod_i f(t_i, \theta_i | x_i).$$

where:

$$(3.13) \qquad f(t_i, \theta_i | x_i) = h(t_i, \theta_i) \exp\left(-\int_0^t h(s_i, \theta_i | x_i) ds\right), \text{ if } d_i = 1$$

$$(3.14) \qquad f(t_i, \theta_i | x_i) = \exp\left(-\int_0^t h(s_i, \theta_i | x_i) ds\right), \text{ if } d_i = 0.$$

The joint density is

$$(3.15) \qquad f(t_i, \theta_i | x_i) = g(t_i | x_i, \theta_i) \mu(\theta_i)$$

and the marginal likelihood of duration times $f(t_i, \theta_i | x_i)$, is given by:

$$(3.16) \qquad L = \prod_i \int g(t_i | x_i, \theta_i) d\mu(\theta),$$

The problem is how to control for the unobserved mixing distribution $\mu(\theta)$ (Lancaster, 1979; Lancaster and Nickell, 1980; Heckman and Singer, 1982, 1984). Standard approaches to the estimation of the above equation require a parametric distribution on θ. However, if the density function $\mu(\theta)$ is specified parametrically, then estimation

bias due to an incorrect parameterization of $\mu(\theta)$ is not limited to duration dependence effects but extends to the parameters of included observed variables as well. Moreover, Heckman and Singer (1984) show that the problem of overparamerterization can lead to the observational equivalence of two different sets of distribution.

A class of nonparametric estimators, which can avoid the ad hoc specification of the mixing distribution $\mu(\theta)$ is the nonparametric MLE (Robbins, 1964; Laird, 1978; Lindsay, 1983a,b; Heckman and Singer, 1982, 1984). Heckman and Singer's Nonparametric Maximum Likelihood Estimator (NPMLE) estimator can be used to avoid the ad hoc specification of the mixing distribution $\mu(\theta)$ (Robbins, 1964; Laird, 1978; Lindsay, 1983; Heckman and Singer, 1982, 1984). Basically, this method reduces to the use of a finite support histogram to model $\mu(\theta)$. The EM algorithm (Dempster, et al., 1977) has often been used to solve the likelihood equations. Application to the frailty model is accomplished by treating the sequence of unobservables $\{\theta_i\}$ as missing data. The estimator is consistent for mixing distributions characterized by a finite number of points of support. As a practical matter, the number of these must be small enough for their identification to be empirically feasible. This estimator has been used in the study of adult health using the Dorn smoking sample by Behrman, et al. (1990). They examine robustness of estimates to functional form, individual heterogeneity, and cohort and time period variations, and note that both the Cox model and the Weibull proportional hazard model with no allowance for heterogeneity yield similar coefficients for the smoking variables, although the Heckman-Singer nonparametric methodology is judged best in terms of model fit.

Maximum Penalized Likelihood Estimation (MPLE) provides another approach to dealing with unobserved heterogeneity but has been used less widely. It was introduced by Good and Gaskins (1971) and developed by de Montricher, et al. (1975), and Silverman (1982). They consider the piecewise smooth estimation of an unknown density function by adding a penalty term to the likelihood which penalizes unsmooth estimates. The general form of a penalized log likelihood under random sampling is given by:

$$(3.17) \qquad \log L = \sum_{i=1}^{N} \log f(x_i) - \alpha R f(x),$$

where f(x) is an unknown density, $R\{f(x)\} < \infty$, R is a functional, and α is the smoothing parameter. The choice of α controls the trade-off between smoothness and goodness-of-fit, while the choice of the penalty functional R identifies the type of behavior considered undesirable. For example, if R is defined as the norm of the first derivative, then a penalty functional R will smooth the slope of the density f(x). If R uses the norm of the second derivative, the curvature will be smoothed as well.

Huh and Sickles (1994) detail how this model can be modified to handle unobserved variables under different assumptions about temporal and cross-sectional sources of heterogeneity that is uncorrelated with the observed variables. MPLE may have computational and convergence advantages over NPMLE in finite samples since roughness in the empirical heterogeneity distribution is smoothed from the likelihood by including penalty terms that take into account the degree of roughness or local

variability not controlled for by covariates. MPLE is consistent as $\alpha/\sqrt{n} \to 0$ for bounded α, if the mixing distribution can be characterized by a finite number of supports. The NPMLE and the MPLE converge to the same function for large N since the penalty term becomes negligible as estimates of unobserved heterogeneity become less rough.

Simulation Based Probability Estimators offer another approach to modeling complicated mortality experiences. Monte Carlo approaches to probability calculations are well known in the area of computer simulation and have received recent interest in econometrics (Gourieroux and Monfort, 1992; McFadden, 1989; Pakes and Pollard, 1989). As computing technology advances to handle bigger inputs with shorter processing time, computer intensive statistical methods have been introduced and developed to solve more complicated problems in stochastic process modeling. Simulation methods (Lerman and Manski, 1981; Diggle and Gratton, 1984) have many potential advantages and are seeing increasing use in econometric applications (see, for example, the special issues of the *Journal of Applied Econometrics*, 1994, and the *Review of Economics and Statistics*, 1994). Early approaches were based on frequency or density estimation. For example, the sequence of observations {x} is used to construct an estimate of the true density, $\hat{f}$, and then independent realizations as required are drawn from $\hat{f}$. Construction of $\hat{f}$ is not an easy task and thus it may be desirable to simulate not from $\hat{f}$ itself but from the underlying true structure of the observed data.

Below we outline how simulation based estimation (SIMEST) can be utilized to estimate a hazard model with unobserved heterogeneity. SIMEST is based on axioms that are assumed to govern the data generating process and does not require closed form expressions for the likelihood. The concepts of the simulation based estimation method used herein were introduced by Atkinson, et al. (1983), Diggle and Gratton (1984) and Thompson, et al. (1987) and are summarized in Thompson (1989).

3.2.1. Univariate simulation

Suppose that we wish to estimate only duration dependence (γ) without covariates. Ignoring subscripts for the moments, the hazard function for individual I is:

$$\lambda = t^{\gamma}\exp(\theta), \tag{3.18}$$

where γ is the duration dependence parameter, θ is the unobserved heterogeneity component and t is time until failure.

Suppose that the transition of states follows the Poisson process. According to the Poisson axioms, the probability that failure can occur in the time interval $[0, t_i)$ is:

$$\begin{aligned} &Pr[x(t+\Delta t)=1]= \\ &Pr[x(t)=1]Pr[x(\Delta t)=0]+Pr[x(t)=0]Pr[x(\Delta t)=1)]+0(\Delta t) \end{aligned} \tag{3.19}$$

Let the probability that one failure takes place in [t, (t+Δt)] be λΔt for every t in [0,t) and the probability that more than one failure happens in [t,(t+Δt)] be of order o(Δt), where $\lim_{\Delta t \to o} o(\Delta t)/\Delta t = 0$. Then:

$$(3.20)\quad Pr[x(t+\Delta t)=1] = Pr[x(t)=1](1-\lambda\Delta t) + Pr[x(t)=0](\lambda\Delta t) + 0(\Delta t)$$

$$\frac{Pr[x(t+\Delta t)=1] - Pr[x(t)=1]}{\Delta t} = \lambda(Pr[x(t)=0] - Pr[x(t)=1]) + \frac{0(\Delta t)}{\Delta t}.$$

As $\Delta t \to 0$, $dPr[x(t)=1] / dt = \lambda \{ Pr[x(t)=0] - Pr[x(t)=1] \}$ and thus $Pr[x(t)=1] = \lambda t \exp(-\lambda t)$ and $Pr[x(t)=0] = \exp(-\lambda t)$. The cumulative distribution function for at least one failure on or before t becomes $F(t) = 1 - Pr[x(t)=0] = 1 - \exp(-\lambda t)$.

A common practice is to use maximum likelihood with a parametric specification for the heterogeneity distribution and the probability density function of failure, f(·). An alternative approach is maximum likelihood based on a nonparametric specification of the heterogeneity distribution but with the form of the density function f(·) required. A third approach is to estimate the parameter γ without formally specifying the probability density function.

For the univariate case, we can assume that time to failure for all n individuals is recorded as $t = (t_1 \le t_2 \le ... \le t_n)$. Using this data we can divide the time axis into k bins, the m^{th} of which contains n_m observations. Having an initial value for the parameter γ, the simulation mechanism is employed to generate a large number (N) of simulated failure times $s = (s_1 \le s_2 \le ... \le s_N)$, where $N > n$. The simulation mechanism here is the cumulative distribution function $F(t) = 1 - \exp\{-\lambda(\cdot)f\}$ where $\lambda(\cdot) = t_i^{\gamma}\exp\{\theta_i\}$ is the Weibull proportional hazard. Then a random number u_i, I=1,...,N is generated from the uniform distribution. Using the generated numbers, the simulated time to failure, s_i, can be generated by inverting F(t). Let the number of simulated observations that fall into the m^{th} bin be ν_{km}. The simulated bin probabilities $\hat{P}_{km}(\gamma_0) = \nu_{km} / N$ should approximate the probability the sample observations fall in the same bin, $P_m = n_m / N$, for values of γ_0 close to the truth. The natural criterion function is to minimize the distance between $\hat{P}_{km}(\gamma_0)$ and P_m. This turns out to be Pearson's goodness of fit. Thompson et al. suggest three possible criteria that remain unchanged when, for instance, two cells are combined into a single cell. The goodness of fit is defined as:

$$(3.21)\quad S(\gamma_0) = \sum_{j=1}^{k} \frac{\hat{P}_{kj}(\gamma_0) - P_j}{P_j}$$

where k is the number of bins, and $\hat{P}_{kj}(\gamma_0)$ is the simulated probability of the j-th bin with estimated parameter γ_0. The function is minimized when $\hat{P}_{kj}(\hat{\gamma}) = P_j$, j=1,...,k. Once the criterion function converges to a value $\hat{\gamma}$, confidence intervals for the true value of γ can be derived using bootstrap methods (Thompson, 1989).

3.2.2. Multivariate simulation

Next, suppose that the probability of failure follows the Poisson axioms and is conditional on a set of exogenous variables and duration time. Then the parameter λ of the Poisson process is given by:

$$\lambda = t^{\gamma_i} \exp(x_i \beta_i + \theta_i) \tag{3.22}$$

We wish to estimate the parameters $\delta = (\beta, \gamma)$ by SIMEST. Without loss of generality, we consider the case of one covariate (x) and duration time (t). Let $t = \{t_i(x_i)\}$, I=1,...,n be failure time data conditional on the exogenous variable x_i, I=1,...,n, and let k_1 and k_2 be the number of bins dividing the time axis and the covariate axis, respectively. Let m be the number of repeated simulations. Then simulated time to failure in the time axis is $0 \leq$
$s_{11}(x_1), s_{12}(x_2), ..., s_{1n}(x_n), s_{21}(x_1), s_{22}(x_2), ..., s_{2n}(x_n), ..., s_{m1}(x_1), s_{m2}(x_2), ..., s_{mn}(x_n)$
with the corresponding value of the exogenous variable $x = \{x_i\}$ in the covariate axis. The number of these simulated times and values of a covariate which falls into (l_1, l_2)-th bin is denoted by ν_{l_1, l_2}, where $l_1 = 1,...,k_1$, $l_2 = 1,...,k_2$. If δ_0 is close to the true value, then the simulated bin probability:

$$\hat{P}_{l_1 l_2}(\delta_0) = \frac{\nu_{l_1 l_2}}{n_m} \tag{3.23}$$

should approximate the corresponding portion of data (time and a covariate) in the same bin,

$$P_{l_1 l_2} = \frac{n_{l_1 l_2}}{n} \tag{3.24}$$

A minor modification of the criterion is necessary since the presence of empty bins makes Pearson's goodness of fit criterion uninformative. To prevent this, the modified Pearson goodness of fit is given by:

$$\begin{aligned} S_m(\delta) &= \sum_{l_1=1}^{k_1} \sum_{l_2=1}^{k_2} \frac{(\hat{P}_{l_1 l_2}(\delta) - P_{l_1 l_2})^2}{P_{l_1 l_2}}, \text{ if } \hat{P}_{l_1 l_2}(\delta) = 0 \text{ , } P_{l_1 l_2} \neq 0 \\ &= \sum_{l_1=1}^{k_1} \sum_{l_2=1}^{k_2} \frac{(\hat{P}_{l_1 l_2}(\delta) - P_{l_1 l_2})^2}{\hat{P}_{l_1 l_2}}, \text{ if } \hat{P}_{l_1 l_2}(\delta) \neq 0 \text{ , } P_{l_1 l_2} = 0 \\ &= 0 \text{ otherwise} \end{aligned} \tag{3.25}$$

The modified minimization criterion substitutes the observation probability with the simulated probability when the observed probability of a certain bin is zero. This may be possible since the simulated probability should approximate the observation probability if the estimate of parameter δ is close to the true value. The criterion is also minimized when $P_{\ell_1,\ell_2} = \hat{P}_{\ell_1,\ell_2}(\delta_0)$, $l_1 = 1,...,k_1$, $l_2 = 1,...,k_2$. Once the parameter δ is estimated, confidence intervals for the true value of the parameter δ can be derived. Consistency and asymptotic normality of the simulation based estimator for large N and M are discussed in Lee (1992) and McFadden and Ruud (1992). McFadden (1989) and Pakes and Pollard (1989) prove similar results for alternative simulation estimators when the number of simulations (M) is finite.

McFadden (1989) has pointed out that numerical breakdowns in standard algorithms can be caused by discontinuities in the simulated objective function. Thus kernel-based procedures are often pursued to smooth the discontinuities. Scott's (1979, 1985, 1992) method of average-shifted histograms has been used with success in the hazard model with heterogeneities by Huh and Sickles (1994). Other smoothing techniques for the simulated frequency, maximum simulated likelihood, and simulated method of moments estimators are discussed in McFadden (1989), Stern (1992), McFadden and Ruud (1994), Geweke et al. (1994), and Hajivassiliou et al. (1996).

3.3. Monte Carlo Results

3.3.1. Design of experiments and data generation

We consider the Weibull proportional hazard model

$$h(t_i|x_i,\theta_i) = \exp(\gamma \ln t_i)\exp(x_i\beta+\theta_i)$$

in which observed data are generated as realizations of the stochastic process:

$$h_i = t_i^{\gamma} \exp(\beta_0+x_i\beta + \theta_i),\ i = 1,...,n,$$

where $t_i \geq 0$, $x_i = (x_{1i}, x_2 i)$, $\beta = (\beta_0 \beta_1 \beta_2)$ and where θ_i is an unobserved stochastic process defined on a complete probability space. The heterogeneity parameter, θ_i, need not be i.i.d., but for our experiments we assume that it is. The artificial samples are generated by the following procedures. First, we draw a uniform random variable u_i in the interval [0,1] and generate heterogeneity θ_i according to the implicit function, μ, where:

$$\theta_i = \mu^{-1}(u_i),\ i = 1,...,n$$

and where μ^{-1} is the inverse of an appropriate cumulative probability function. Next, we draw values of two exogenous variables $x_i=(x_{1i}, x_2 i)$ from a standard normal random number generator. Another uniform random number in the interval [0,1] is drawn for the survival function $S_i = (1-F(.))$. We then solve for the implied duration t_i from the survival function with given values of parameters, β and γ. Thus

$$(3.26) \qquad t_i = \exp[(\ln(-\ln S_i) + \ln(\gamma+1) - (\beta_0 + \beta_1 x_{1i} x_{2i} + \theta_i))(1/\gamma+1)]$$

Different mixing distributions for the heterogeneity are drawn to compare the performances of the different estimators. We use the standard normal as our unimodal contamination. In addition, a bivariate normal distribution representing a bimodal heterogeneity distribution, and a multinomial distribution representing multimodal distribution are also employed. Given the duration t_i, (3.43) with true parameter values β_0=0.1, β_1=β_2=γ=1, the right censored times T_r are set to ensure that about 15 percent of observations are censored. We then increase the censoring rate to 20 percent. When left censoring is allowed, censoring times T_l are arbitrarily set to be 1 time-unit since the mean duration of t_i is 3.43 time units. As a result, about 25 percent are censored. Samples of 100, 500 and 1000 are used. These are in the range of sample sizes for the bulk of empirical duration studies though much smaller than most of the samples we use.

The basic logic of the simulation based estimator in this context is rather straightforward. Suppose that sample observations for two covariates and duration variable t are given by the data generation procedure described above. The simulation algorithm in each replication is as follows:

Input initial values for the parameters β_0, β_1, β_2, γ,
Repeat until $t^*_i > 0$, where t^*_i is a simulated time,
Generate θ_i from U(0,1),
Generate a simulated time t^*_i through the survival function,
if $t^*_i < 0$, then discard,
End repeat,
Return t^*_i.

The simulation algorithm can be easily adapted for more complicated models. The method employed to choose the smoothing parameter ϕ of MPLE is the subjective choice method (Bartoszynski, et al., 1981). An alternative is to adapt the cross-validation method by minimizing:

$$CV(\alpha) = n^{-1} \sum_{i=1}^{n} (t_i - t_{\delta\alpha})^2,$$

where $t_{\delta\alpha}$ is the inverse function of the hazard function, h, such as $F^{-1}(\underline{x}_i, h ; \delta, \alpha)$ and where $\underline{x} = (x_1, x_2)$, $\delta = (\beta_1, \beta_2, \gamma)$. However, the evaluation of $CV(\alpha)$ is too computationally burdensome even for pseudodata sets of size 100 because to find a maximum requires no less than: the number of function evaluations times the number of observations times the number of function evaluations with each new α. In a typical case, about 14700 iterations were needed for pseudodata sets of size 100. The adaptation of cross-validatory methods to our model merits further investigation.

3.3.2. Comparisons Among Different Estimators

Typical outcomes of our Monte Carlo experiments (Huh and Sickles 1994) are shown in Table 3.1-3.13. These results are suggestive of some possible discrepancies among the different estimators in different cases but also suggest substantial comparability between them when the underlying stochastic process is not too complicated and has been correctly modeled. Table 3.1 presents results based on the three estimators when there is no censoring and heterogeneity is drawn from a standardized normal distribution. Both the duration parameter and structural parameters are estimated well for all three estimators. We begin with two points of support and add an additional point of support until no directional directives show positive values and no improvement in the likelihood value is shown. Standard deviations from SIMEST are calculated by the bootstrap method with 30 replications. The bin width for SIMEST is based on the expression introduced by Scott (1979) that chooses an optimal bin width by minimizing the integrating mean square error (IMSE) of the multidimensional histogram. In this case with the sample size of 500, the number of bins is six.[19] Samples of size of 1000 and 100 require seven bins and four bins for each dimension, respectively. However, SIMEST continually converges to a local optimum when starting values quite different from the true values are used. For example, when starting values are $(\beta_1, \beta_2, \gamma) = (0.4,0.4,0.4)$, our estimates are (0.532, 0.613, 0.276). Results for SIMEST in Table 3.1 are based on starting values of (0.8,0.8,0.8). For MPLE, the smoothing parameters, α_i, i=1,2, are chosen by the subjective choice method (Bartoszynski, et al., 1981). We start from $\alpha_i = 1.0$ for i=1,2. For the purpose of comparison, $\| h^{(2)}(x) \|$, the norm of the second derivative of the hazard function with respect to $\underline{X}$, is used as the penalty function. After searching for the optimal value of α^* using the starting value of $\alpha = 1.0$, we found the α^* for which the $\{\theta_j\}$, j=1,...,m, do not exhibit significant fluctuations. When α is chosen between 0.6 and 0.4, there are no significant differences in both estimates and values of $\{\theta_j\}$. It is possible to choose the different values for each smoothing parameter, but the same value (0.5) is chosen since we generate the pseudodata for x from the same distribution. Finally, the number of bins to calculate derivatives was 10 in the interval $[\underline{x}_{min}, \underline{x}_{max}]$.

We next assume that there is right censoring after five time-units, which censors about 15 percent of the sample observations. As seen in Table 3.2, NPMLE and MPLE slightly underestimate the true values. However, the degree of underestimation for the structural parameters is greater with NPMLE than MPLE. On the other hand, SIMEST overestimates the duration dependence parameter, but estimates the structural parameters very well.

The principal findings of our experiments are reported in Tables 3.3-3.13. First, both MPLE and NPMLE perform poorly in small samples while SIMEST performs relatively well. As the number of observations increases to 500 and more, both MPLE and NPMLE begin to track the underlying stochastic model, in contrast to SIMEST whose stochastic axioms are at variance with the data generation process and thus should not be expected to perform well asymptotically (see Tables 3.1, 3.3, and 3.4).

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[19] Since the use of five bins produced less biased estimates than that of six bins, we use five bins for the 500 observations experiments.

Second, as we increase the proportion of censored observations NPMLE loses any advantage over MPLE. Of the three methods, SIMEST appears to be most robust. However, when left and right censoring coexist, SIMEST also becomes unstable (see Tables 3.2, 3.5, and 3.6).

Third, Table 3.7 reports how the choice of the smoothing parameter affects parameter estimates from MPLE. Two smoothing parameters are chosen subjectively and are used to estimate MPLE. One is chosen to be 0.2, which is smaller than $\alpha = 0.5$. With this choice of α, estimates tend to be biased downward. The result is expected because the estimates of MPLE should be the same as those of NPMLE if $\phi = 0$. Furthermore, when we select ϕ to be 1.0, which is greater than the best choice of α, parameters are also underestimated due to oversmoothing.

Fourth, the choice of the bin width for SIMEST is quite essential, especially for the multivariate nonlinear function-fitting problem because the estimates become unstable as the chosen bin width differs from the optimal bin width (Table 3.8).

Fifth, Tables 3.9 and 3.10 demonstrate the results when the heterogeneity distribution is drawn from a bimodal and multimodal distribution. MPLE and NPMLE performed well when actual heterogeneity is not unimodal. However, NPMLE has mass points at (0.274, 0.783, 0.823) for a bimodal distribution and shows all negative directional derivatives. For the multimodal distribution, four points of support appear adequate. These results, as well as those with the unimodal distribution, suggest that the mass point method employed by NPMLE has difficulty reflecting the true distribution of heterogeneity and that the choice of optimal supporting points requires further research.

Sixth, we investigated the predictive power of NPMLE, MPLE and SIMEST with different true parameter values and 500 observations. Tables 3.11, 3.12, and 3.13 summarize the results of three different cases. Evidence indicates that these three estimators have substantial predictive power.

This specification of conditional death hazard functions raises the issue of simultaneity. Simultaneity bias may be a problem with right-side covariates, including education, because there may be persistent unobserved heterogeneity (e.g., genetic or family-background environment-related characteristics associated with inherent robustness, ability and motivation) that affect outcomes throughout one's life. Several studies are consistent with the possibilities of such unobserved factors having influence at different points in the life cycle (e.g., Behrman, Hrubec, Taubman, and Wales, 1980; Behrman and Wolfe, 1984, 1987b, 1989; Rosenzweig and Schultz 1983b; Olneck, 1977; Wolfe and Behrman, 1987).

Within the health demand literature, the most common tradition is to emphasize the possible simultaneity bias for labor income. But the same possibility exists for nonlabor income (particularly if brighter people have greater labor market earnings and better investment strategies or simply save more). To control for all such simultaneity with most such data sets is difficult (and almost never done). The methods that usually are used to control for simultaneity, moreover, are not without their limitations since ideal instruments, which are highly correlated with the endogenous variable but orthogonal to the disturbance, are rarely available. If the former condition is not satisfied, measurement error bias may dominate in the estimates. If the orthogonality condition is not satisfied, simultaneity bias still may be a problem.

Although the statistical treatments covered in this chapter are not exhaustive, they do cover most of the structural approaches to modeling mortality and morbidity, and offer up alternatives to univariate approaches that have typically been used in biostatistics. The lack of a controlled experimental setting in survey research on mortality and morbidity pushes the researcher away from univariate methods such as the Kaplan-Meir product limit estimator of simple life tables and toward multivariate techniques such as those outlined above. Moreover, these specifications of mortality and morbidity models can be viewed at a more general level as generic nonlinear models with imbedded measurement error and the estimators as members of the class of maximum likelihood estimators or of simulated method-of-moments estimators (McFadden, 1989).

Table 3.1 Monte Carlo Estimates of Hazard Functions (n=500, uncensored)

	MPLE[a]	SIMEST[b]	NPMLE[c]
γ	.934	1.154	.967
	(11.1)	(72.1)	(13.8)
β_0	3.243	2.877	2.656
	(8.6)	(23.0)	(8.6)
β_1	.988	.983	.971
	(13.2)	(12.6)	(23.7)
β_2	.956	.934	961
	(14.7)	(38.9)	(106.8)

Note: True parameter values are $\gamma = \beta_1 = \beta_2 = 1$.
Values in () denote "t" statistics.
Heterogeneity is specified to be standardized normal.

[a]Smoothing parameters $\alpha_1=\alpha_2=.5$, bin width (BW) = 0.6.
[b]Number of simulated observations (SN) = 50000; number of bins for x_1, x_2, and t is five.
[c]Four points of support were used to identify heterogeneity.

Table 3.2 Monte Carlo Estimates of Hazard Functions (n=500, 15% right censored[a])

	MPLE[b]	SIMEST[c]	NPMLE[d]
γ	.902	1.136	.896
	(7.2)	(30.7)	(6.5)
β_0	7.23	3.57	10.45
	(5.3)	(20.6)	(5.3)
β_1	.912	1.021	.811
	(8.7)	(18.2)	(3.0)
β_2	.899	1.001	.845
	(4.7)	(29.4)	(2.9)

See note in Table 3.1.

[a]78 out of 500 observations are censored.
[b]The smoothing parameter $\alpha = 0.55$.
[c]SN=50000; Number of bins for x_1,x_2, and t is five.
[d]Four points of support were used to identify heterogeneity.

Table 3.3 Monte Carlo Estimates of Hazard Functions (n=100, uncensored)

	MPLE[a]	SIMEST[b]	NPMLE[c]
γ	.656	.823	.753
	(2.2)	(7.7)	(5.2)
β_0	3.653	2.111	3.997
	(22.0)	(10.7)	(20.2)
β_1	.775	1.140	.798
	(2.0)	(11.3)	(1.6)
β_2	.718	.877	.757
	(1.7)	(10.0)	(1.9)

See note in Table 3.1.

[a]The smoothing parameter $\alpha_1 = \alpha_2 = 1.2$.
[b]SN=150000; Number of bins for x_1,x_2, and t is five.
[c]Four points of support were used to identify heterogeneity.

Table 3.4 Monte Carlo Estimates of Hazard Functions (n=1000, uncensored)

	MPLE[a]	SIMEST[b]	NPMLE[c]
γ	.931	.752	1.057
	(13.7)	(8.6)	(30.2)
β_0	8.271	3.997	9.221
	(37.6)	(11.0)	(29.1)
β_1	.956	.812	.937
	(22.2)	(8.9)	(15.1)
β_2	.965	.799	.958
	(11.0)	(11.3)	(9.9)

See note in Table 3.1.

[a]The smoothing parameter $\alpha_1 = \alpha_2 = 0.3$.
[b]SN=100000; Number of bins for x_1,x_2, and t is seven.
[c]Four points of support were used to identify heterogeneity.

Table 3.5 Monte Carlo Estimates of Hazard Functions (n=500, censored[a])

	MPLE[b]	SIMEST[c]	NPMLE[d]
γ	.834	1.165	.842
	(3.5)	(27.1)	(4.0)
β_0	8.611	4.55	12.97
	(4.6)	(13.9)	(5.5)
β_1	.859	1.199	.731
	(3.6)	(28.5)	(2.5)
β_2	.862	1.099	.720
	(4.0)	(29.7)	(2.3)

See note in Table 3.1.

[a]102 of the 500 observations are censored.
[b]The smoothing parameter $\alpha_1 = \alpha_2 = 0.6$.
[c]SN=50000; Number of bins for x_1, x_2, and t is five.
[d]Four points of support were used to identify heterogeneity.

Table 3.6[a] Monte Carlo Estimates of Hazard Functions (n=500, Right and left censored[b])

	MPLE[c]	SIMEST[d]
γ	.697	1.223
	(1.9)	(9.1)
β_0	13.481	8.54
	(3.1)	(15.3)
β_1	.766	1.205
	(2.2)	(12.4)
β_2	.733	1.118
	(2.0)	(10.2)

See note in Table 3.1.

[a]NPMLE is not available.
[b]15% right censored and left censored.
[c]The smoothing parameter $\alpha_1 = \alpha_2 = 1.2$.
[d]SN=50000; Number of bins for x_1, x_2, and t is five.

Table 3.7 Monte Carlo Estimates of Hazard Functions MPLE with Different Smoothing Parameters (n=100, uncensored)

Smoothing parameter	$\alpha = .2$	$\alpha = .5$	$\alpha = 1.0$
γ	.123 (0.7)	.934 (11.1)	.355 (32.3)
β_0	12.005 (3.1)	3.243 (8.6)	5.811 (1.6)
β_1	.661 (1.6)	.988 (13.2)	.602 (1.6)
β_2	.612 (1.3)	.956 (14.7)	.623 (2.3)

See note in Table 3.1.

Table 3.8 Monte Carlo Estimates of Hazard Functions SIMEST with Different Bin Widths (n=500, uncensored)

	Case 1[a]	Case 2[b]	Case 3[c]
γ	.435 (2.5)	1.154 (72.1)	.240 (1.0)
β_0	8.243 (2.8)	2.87 (23.0)	13.566 (4.7)
β_1	.234 (1.3)	.983 (12.6)	.399 (2.0)
β_2	.431 (4.4)	.934 (38.9)	.356 (3.5)

See note in Table 3.1.

[a]SN=50000; Number of bins for x_1, x_2, and t is two.
[b]SN=50000; Number of bins for x_1,x_2, and t is five.
[c]SN=50000; Number of bins $x_1 = x_2 = t = 10$.

Table 3.9 Monte Carlo Estimates of Hazard Functions Bimodal Heterogeneity[a] (n=500, uncensored)

	MPLE[b]	SIMEST[c]	NPMLE[d]
γ	.921	.965	.954
	(5.1)	(21.0)	(7.8)
β_0	5.753	4.547	4.885
	(15.7)	(96.7)	(17.0)
β_1	.923	.976	.928
	(10.6)	(25.7)	(23.8)
β_2	.922	.955	.921
	(13.0)	(12.6)	(43.9)

See note in Table 3.1.

[a]Heterogeneity is generated by

$$d\mu(\theta) = p(2\pi\theta_1^2)^{-1/2}\exp\frac{-\theta^2}{2\theta_1^2} + (1-p)(2\pi\theta_2^2)^{-1/2}\exp\frac{-(\theta^2)}{2\theta_2^2}d\theta$$

where $p = .5, \theta_1 = 1, \theta_2 = 2.$

[b]The smoothing parameter $\alpha = .65$.
[c]SN=50000; Number of bins for x_1, x_2, and t is five.
[d]Four points of support are used to identify heterogeneity.

Table 3.10 Monte Carlo Estimates of Hazard Functions Multimodal Heterogeneity[a] (n=500, uncensored)

	MPLE[b]	SIMEST[c]	NPMLE[d]
γ	.992	1.002	.986
	(198.4)	(47.7)	(493.0)
β_0	3.43	2.14	3.16
	(24.9)	(142.7)	(197.5)
β_1	.981	.977	.983
	(81.8)	(29.6)	(983.0)
β_2	.966	.978	.968
	(107.3)	(51.5)	(322.7)

See note in Table 3.1.

[a]Heterogeneity is generated from

$d\mu(\theta_i) = p_i$, for i = 1,...,7, where $p_1 = p_3 = p_5 = p_7 = .1$, $p_2 = p_4 = p_6 = 0.2$.

[b]The smoothing parameter $\alpha = 0.3$.
[c]SN=50000; $bin(x_1) = bin(x_2) = bin(t) = 5$.
[d]Four points of support are used to identify heterogeneity.

Table 3.11 The Predictive Power of Estimators (n =500, $\gamma = 2$, $\beta_1 = \beta_2 = 1$)

	MPLE[a]	SIMEST[b]	NPMLE[c]
γ	1.876	2.019	1.941
	(9.5)	(59.4)	(46.2)
β_0	7.453	2.866	5.456
	(29.3)	(86.8)	(26.2)
β_1	.956	.945	.968
	(59.8)	(33.8)	(138.3)
β_2	.947	.965	.976
	(63.1)	(40.2)	(88.7)

See note in Table 3.1.

[a]The smoothing parameter $\alpha = 0.6$.
[b]SN=50000, bin(x_1) = bin(x_2) = bin(t) = 5.
[c]Four points of support are used to identify heterogeneity.

Table 3.12 The Predictive Power of Estimators (n =500, $\gamma = 1$, $\beta_1 = 2$, $\beta_2 = 3$)

	MPLE[a]	SIMEST[b]	NPMLE[c]
γ	.975	.987	.992
	(8.6)	(21.0)	(23.6)
β_0	6.215	3.664	5.757
	(64.0)	(305.3)	(261.7)
β_1	1.831	1.883	1.929
	(28.2)	(94.2)	(275.6)
β_2	2.772	2.688	2.977
	(40.2)	(149.3)	(297.7)

See note in Table 3.1.

[a]The smoothing parameter $\alpha = 0.45$.
[b]SN=50000, bin(x1) = bin(x2) = bin(t) = 5.
[c]Four points of support are used to identify heterogeneity.

Table 3.13 The Predictive Power of Estimators ($n = 500$, $\gamma = .5$, $\beta_1 = \beta_2 = 1$)

	MPLE[a]	SIMEST[b]	NPMLE[c]
γ	.483	.510	.491
	(7.3)	(34.0)	(19.6)
β_0	6.13	2.443	3.545
	(266.5)	(135.7)	(272.7)
β_1	.971	.982	.992
	(80.9)	(70.1)	(330.7)
β_2	.973	.985	.995
	(121.6)	(82.1)	(248.8)

See note in Table 3.1.

[a]The smoothing parameter $\alpha = 0.6$.
[b]SN=50000, $bin(x_1) = bin(x_2) = bin\ (t) = 5$.
[c]Four points of support are used to identify heterogeneity.

4

Mortality Hazard Estimates from the Dorn Sample: Smoking, Occupational Risks, Birth Cohort, Functional Form and Frailty

In this chapter we present estimates of both the proportional and accelerated-time-to-failure hazard models that are discussed in Chapter 3 for the Dorn sample, with emphasis on associations with smoking, occupational risks, birth cohort and unobserved frailty (heterogeneity). The mortality hazard relations that we estimate, as discussed in Section 3.1, can be viewed as production functions or conditional demand relations that come out of the dynamic optimizing behavior in the model presented there. We first survey relevant previous literature and then describe the Dorn sample. We then begin our analysis with no allowance for heterogeneity. We then redo the analysis allowing for parametric and non-parametric heterogeneity, whose significance and importance are examined, and then we use the Maximum Penalized Likelihood functions that also diminish the importance of heterogeneity. These explorations of the impact of unobserved heterogeneity address some possibly important estimation problems since both mortality and the right-side variables may be responding to unobserved heterogeneity.

4.1. Previous Mortality Studies

Mortality studies generally relate the age-specific death rate to some of the variables that influence the supply or demand for health. The underlying assumption is that when one's health becomes too poor, one dies, as is elaborated in the modeling of Chapter 2. Many of these studies use a methodology in which population average death rates for a given age are compared with the death rate of people with a given characteristic such as level of education. Other studies calculate hazard rate models in which the percentage of people who die over a time interval are related to age (at the beginning of the period) and a variety of other variables such as education, marital status, and cigarette smoking, usually motivated by appealing to health/mortality production functions or to reduced-form conditional demands for mortality. Generally such studies

do not consider the possibility that the right-side variables might include choice variables and not be independent of the stochastic term, with biased estimates as a result. Most of these studies take an existing sociodemographic survey and match it to information on date (and sometime cause) of death over some future time interval ranging from 6 months to decades. The completeness of the death record information varies considerably across studies. In the rest of this section we briefly survey the more prominent studies in this literature. A primary distinction among these studies is between those that use life-table methods for organizing and analyzing correlations in the data and those that focus on extensions of life-table methods to more structural approaches to characterizing multiple health states and/or mortality hazards. Another important distinction among these studies is whether they use data at the aggregate or individual level because the latter permits a richer characterization of observed covariates and exploration of the role of unobserved individual heterogeneity ("frailty").

Studies based on individual data. Morris et al. (1953) examined the interrelationship in physical activity and coronary heart disease for drivers, conductors, and guards in the London Transport company in 1949 and 1950. They find the conductors have fewer coronary illnesses and speculate that this may occur because of greater physical activity. Only age is used as a control. They find similar results when comparing postmen and other civil servants. Of course who applied for and was accepted for these jobs is not known, and selectivity bias may be a problem since those in poorer health would tend to select jobs with less physical activity.

Paffenbarger et al. (1966) use Harvard alumni who graduated between 1916-1950 and University of Pennsylvania who graduated between 1931-1940. Each, of course, is a very select population. Date of death was recorded by the Alumni Offices of each university. Given a notice of death, they obtain death certificates, which are used to ascertain cause of death by 1950. Information on sociodemographic information was taken from student health records. They find that heavy cigarette smoking, high blood pressure and obesity are associated with early deaths. These fund raisers' mortality records, unfortunately, are incomplete and outdated. For example, Mr. Lampe, who died in 1969, is only recorded in "Deaths" in the December 1993 issue of the *Pennsylvania Gazette* (p. 46), the alumni magazine of the university. Further, the less successful alumni are less likely to be included in the University death records.

Using the same population Paffenbarger and Williams (1967) examined the death rate from strokes by 1950. They find the same factors given in the previous paragraph increase the chances of death from stroke as well as early parental death (all causes) and non-participation in varsity sports.

Kitagawa and Hauser (1973) matched the 1960 Census forms to the death certificates for those Americans who died during the next four months. For the elderly (65 and older) only a random sample of the death certificates were matched. For those younger than 65, all the death certificates were matched. The study was undertaken in the infancy of the computer age, and Kitagawa and Hauser only used cross classifications — generally one or two ways — as their mode of analysis. They compared their actual death rate estimates in an age group with the number of deaths expected in that group assuming as their null hypothesis that there were no effects of the variable under study. (Rosen and Taubman (1979) show that this technique may impart a bias to their results if the null hypothesis is wrong.) Kitagawa and Hauser

show that for the elderly more educated females have a lower death rate than less educated women, but they find no difference by education level for elderly males. For younger ages, the more educated have lower age-specific death rates for both genders. Many potential important variables were not controlled in their analysis. The sample is no longer available for reanalysis with more modern techniques.

Rosen and Taubman (1979) use the 1973 Exact Match Sample, in which the CPS was matched by Social Security number, name, place and date of birth, and gender to the Social Security records. These records indicate when a person died since old-age benefits are supposed to be terminated and claims are made for partial reimbursement of burial expenses, etc. Subsequently the Social Security records were updated through 1976. They use the 1973-1976 data to study the effects of education and other variables on age-specific death rates. For this time period, the death records are mostly correct for those age 65 and over but much less complete for younger people who were not eligible for old-age benefits. We concentrate here on their findings for the elderly. Using ordinary least squares regressions, they find a statistically significant 23 percent lower death rate for college graduates than the least educated men and strong effects for family income and marital status[20] These findings for men differ from the findings of Kitagawa and Hauser (1973). While the matching methods in these studies differ we expect that the basic reasons for the differences in results are the changes in cigarette smoking behavior since the more educated are less likely to smoke and the institution of Medicare and Medicaid in 1966, which would tend to help the less educated.

Hrubec and Neel (1981) use a sample of white identical and fraternal twins born between 1917 and 1927 in the U.S., both of whom are veterans, to study "early" death rates for the period 1946-1975. They find a slightly lower death rate in this period among fraternal and identical twins when analyzed as individuals than for veterans of the same age. They also find a greater concordance of death rates among identical than among fraternal twins. They include no measured covariates.

Taubman and Rosen (1982) apply more appropriate statistical techniques than earlier studies. They use a sample drawn from the first three waves of the Retirement History Survey, which is a random sample of 58-63 year old household heads (see Section 3.2). Taubman and Rosen used multinomial logit models on data grouped by characteristics in part to explain mortality states using longitudinal data. Their methods are statistically correct if there are not zero cells (Nerlove and Press, 1973) and if the characteristics are independent of random error. These techniques are cumbersome and generate empty cells as the number of characteristics increases. They find own and spouse education and marital status and prior health affect the mortality state. Extended models that allow for continuous covariates, instead of the categorical ones used in their contingency table approach, as well as allow for an explanation of the time in the cell (e.g., survival time) lead naturally to the hazard analysis we pursue in latter chapters.

Madans, et al. (1986b) use the NHANES — National Health and Nutritional Survey — follow-up to examine the differences by race, gender, and living in poverty

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[20] A zero, one dummy variable is the dependent variable. In addition to other problems, the right-side variables are not independent of the disturbance term, and OLS is not guaranteed to reproduce a function limited to that range.

during the 1971-1975 period on deaths during a 10 year follow-up period. They use the ratio of actual to expected deaths (based on census data, which statistical method as noted earlier is subject to bias) and demonstrate strong differences in this ratio due to these three factors.

Sickles and Taubman (1986) use part of the RHS survey to estimate a simultaneous limited dependent variable model of healthiness (including death) and retirement. They find that various measures of income, marital status, and occupation are associated with mortality while controlling for multi-variate random effects.

Paffenbarger et al. (1986) examine data on the same sample of Harvard alumni who graduated between 1916-1950. Besides college records, they used mail questionnaire responses. They have much information on behavior and personal characteristics. As noted, this is a very select sample with incomplete information on the timing of death since they only obtain information on who died from Harvard's alumni office. Presumably these records are incomplete as discussed above. They compare actual with expected deaths rates by age in one or two-way cross-classifications. They find an inverse relationship with physical activity in college even when controlling for cigarette smoking, weight and early death of parents. They also find that smoking and high blood pressure were associated with increased risk of earlier death.

Burtless (1987) analyzes the health status of the elderly using the 1969-1979 survey waves of the RHS's males. He finds that (lifetime) employment in mining, construction, and as a labor operative leads to worse health in old age with the morbidity effects being greater than the mortality effects. He also finds wages and pension coverage matter. He uses highly sophisticated estimation techniques, but does not explore the issues that we cover in this book. Kaplan, et al. (1987) present mortality hazard estimates on tobacco usage and gender for about 4000 people originally living in Alameda County in California (aged at least 60 in 1965 and followed for 17 years) based on Cox's proportional hazard model. They find increased risk associated with being male, smoking, lack of leisure time, abnormal weight to height, and not normally eating breakfast. This is a useful study, but their data are obviously limited in coverage and about 5 percent of their respondents' deaths are unreported.

Jones and Goldblatt (1987) use data from England and find that widows have a 10 percent increased risk of death in the period 1971-1981. They use information on expected and actual death rates.[21] Married and unmarried women in this sample had higher rates if they were in less favorable socio-economic circumstances. The lowest mortality was found among women with a good socio-economic status and who worked part time. In the same sample, occupational mortality differences among men were found with rates highest among laborers, miners, construction, etc. and lowest among professional workers, managers, and electrical workers. Most mortality rates of people employed since 1971 were lower at the start of the survey since some of those with chronic diseases weren't at work due to sickness in 1971. Men actively seeking work in 1971 had higher death rates than those employed by the second half of the study. This was due partially to socio-economic and marital status but not with where one lived in England, e.g., London. Wives of unemployed men also had higher death rates.

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[21] Kaprio, et al. (1987) also indicate an excess mortality of widowers and widows in a four year follow-up in Finland.

Corder and Manton (1987) use the National Long Term Care Survey to study health utilization and mortality for about 20,000 elderly persons over the period 1982-1984.[22] The sample initially contacted 33,000 people from Medicare's Health Insurance Master File[23] who were interviewed by phone to determine if they were disabled. All of the disabled and a subset of the non-disabled (in 1982) were resurveyed in 1984. They examine (1) mortality and disability linkages, (2) risk of institutionalization given their respondents' functional level, (3) mortality levels among the institutionalized, (4) changing patterns of medical care utilization and mortality after a hospital episode, and (5) descriptive statistics of service utilization episodes. Their results include that mortality rapidly increased with the level of disability. However, improvement occurred consistently at all levels of disability. The probability of remaining non-disabled (over a two-year period) is similar for males and females, but institutionally disabled males are more likely to return to nondisabled status than similar females. Females with up to four disabilities are more likely to maintain or to improve their function level than males. Females are more likely to be institutionalized in part because their husbands are more likely to have died. As the disabled age, mortality and institutionalization rates increase, and the likelihood of regaining various functions decreases. Nonmarried people have higher levels of institutionalization than married people and there is no clear education pattern.

Kohl et al. (1988) use a subsample of about 375 males who had answered a mail survey and then were examined physically by doctors. They find that in multiple regressions that treadmill performance is related to physical fitness.

Feldman, et al. (1989) use the NHANES data and the Kitagawa and Hauser study to examine trends in death rates by educational differentials of the elderly for the time periods of 1960 and 1971-84. They did not trace 5 to 10% of the original NHANES survey; hence, the death information is incomplete. Like Taubman and Rosen (1982), they find much sharper declines over time for the more educated in each gender group. These educational effects persist in their proportional hazard models even controlling for such statistically significant risk factors as smoking, weight, hypertension, and high amounts of serum cholesterol. They do not explain why the educational effects persist though economists often argue that the more educated make better decisions and can process new information more effectively.

Ellwood and Kane (1990) use the Panel Survey of Income Dynamics (PSID), a stratified random sample with an over-representation of blacks, to study death rates. The sample began in 1969 and is still on-going with annual surveys. They studied those people whose age was over 65 for at least three years. Ellwood and Kane separate non-responses into various categories including those dead and institutionalized, but there is no information on how complete the death records are.

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22 Manton, writing with several others, has made other notable contributions to the estimation of mortality models and to substantive knowledge on mortality. We use some of the statistical models that he has helped to develop, though we also use some other recent methods. We discuss these developments and other methodological issues in Chapter 3 on estimation. We note here that Manton's work generally has been with small samples or with few covariates.

23 This is a 5 percent random sample of all Medicare patients who are followed over time.

They find that marital status, age, and disability were associated with higher death rates for both men and women though being married had negative (positive) effects on survival for women (men).

Sorlie and Rogot (1990) use individuals in various months in the CPS and the National Death Index for the period 1979-1983 to determine which persons died in the period 1979-1983. The CPS data include which civilians were employed, unemployed, and not in the labor force e.g., houseworkers and students. They also control for age, race, and sex. They compare actual death rates with expected rates in 10 year age intervals. The healthiest — those with lower than expected deaths — are the employed while the least healthy are those unable to work. Causation rather than description is difficult to establish in this sample. Again, there is selectivity bias in that those people who are working have to be healthy enough to work.

Liu, et al. (1990) use the 2123 participants in the Framingham Heart Study to estimate a Cox proportional hazard model over a period spanning 10 years.[24] The participants were initially at least 55 years old. This study makes use of 1929 volunteers who took a battery of eight neuropsychological tests. There were 705 eligible study subjects who refused the neuropsychological testing, but the non-participants had similar characteristics as the volunteers. They find cognitive dispairment is associated with higher neuropsychological hazards, even controlling for age and education. To place this in context with other studies, those with marginal and poor cognitive dispairment have a relative risk of death of 1.37 and 1.66 respectively compared to those without such dispairments. These estimates of relative risks are smaller than those found for identical and fraternal twins discordant with respect to smoking as discussed below.

A subset of studies in this literature have focused particularly on the association of smoking with mortality, a topic that we revisit in our estimates below. We now turn to these studies. Dorn (1958) and other researchers, summarized in Rogot (1974), have used the Dorn sample that we describe in Section 5.2 to study the relation of smoking both to mortality in general and to specific causes of death. They also have used the data to investigate trends. They find immense differences in death rates by smoking status e.g., 14 times as many smokers died of emphysema than nonsmokers in a 16 year time interval. Their statistical methodology is to calculate in each year the cumulative (over time) age-specific death rate by smoking status.

An alternative explanation of the interrelationship of smoking and earlier deaths is that underlying genetic or other factors influence both smoking choices and mortality, and there is no causality of smoking on date of death. See, for example, Fisher (1958).

Kaprio and Koskenvuo (1990) use data on Finnish identical and fraternal twins who are discordant with respect to smoking to study deaths from lung cancer and coronary disease. By definition the identical twins have the same genes; hence, for them only the environment and their behavior, such as smoking, may differ. Since they only use twins who are discordant in smoking behavior, they differ in this behavioral dimension. No other control is used. Their sample consists of all like-sexed pairs born prior to 1958 and with both twins still alive in 1967. Nearly 90 percent of the twins responded to a questionnaire mailed in 1975, which also was used to establish zygosity, i.e.,

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[24] The public use version of the Framingham sample only records date of death within a two year interval. Monthly data were available to Liu, et al.

whether particular pairs of twins are identical or fraternal twins (with validation by biological examination for a subset of twins). The data are for 1278 "current" (in 1975) smokers and 1210 former smokers. Death is recorded in the period 1976-1987. For both identical and fraternal male twins, the current smoker had higher overall death rates than the non-smoking sibling. The relative risks of dying were 13.0 and 2.4 for the identical and fraternal twins. Heavy and moderate smokers had even higher relative risks. There was no excess risk for former smokers. They also find greater risk of coronary and heart disease even though nearly 50 percent of the sample was less than 30 years old.

Floderus, Cederlof, and Friberg (1988) examine the effects of smoking on mortality over a 21 year period beginning in 1961 using data on Swedish twins born between 1886 and 1925. They study differences in mortality related to smoking as of 1961. They find that smokers are more likely to have died by 1982 whether they look within pairs or across individuals. They calculate relative risks of dying over this time period within pairs by using pairs discordant with regard to smoking. Within pairs the relative risk varies from 1.7 to 2.3 for males and females with some trend by cohort which may reflect the type of left censoring we discuss in Section 5.6 below. Relative risks are greater for smokers for death from coronary heart disease. When treated as individuals, the effects of smoking on mortality are much larger.

Studies based on aggregate data. Silver (1972) uses 1959-61 age-adjusted mortality rates by sex and gender for SMSA's and states and finds a negative effect on mortality for education, being married, and income, the last of which is more important for blacks. These variables account for a large share of the observed black excess mortality. A potential problem in this study is that some people do not die in the SMSA that they live in. Now it is possible to obtain place of residence and of death.

Fingerhut and Rosenberg (1982) calculate age-specific death rates and demonstrate that there was a substantial downward shift in age-specific mortality rates among the elderly beginning around 1968, but do not indicate why. Preston (1984) in his presidential address to the Population Association of America notes that this drop in death rates corresponds with the introduction of Medicare and Medicaid, but he offers no strong evidence of causation. These declines in age-specific mortality rates altered the remaining life expectancy of older men and women (Table 1.1). For example, Cutler, et al. (1990) calculate that in 1960 a man aged 65 had an expected remaining life of 12.9 years while in 1990 the corresponding figure was 15.0 years. The comparable numbers for women are 15.9 and 18.9 years. However, much of the gain occurred by 1975.

Hadley (1982) uses county level data for 1968 through the first half of 1972. Mortality data by county of residence is taken from death certificates. Average socioeconomic status of the county is taken from the 1970 Census of Population. He estimates health production functions separately for blacks and whites and men and women. Also, separate functions are estimated for those less than 1, 45 to 64, and greater than 64 years old. He finds that medical resources have a significant negative association with death rates. For example a 10% increase in medical care expenses lowers mortality rates 1.2% and 2.0% for elderly white males and black infant females. In these calculations he has controlled for income, education, cigarette consumption and other factors.

Waldron (1982) provides a summary of differences in male and female morbidity and mortality. She finds that much of the difference in mortality and morbidity is related to cigarette smoking, type A behavior pattern, alcohol, exposure to occupational risks, and reproduction-related diseases, but that women are also more likely to report acute disorders and have more doctor visits.

Doll (1953) using aggregated mortality statistics over a 10 year period finds an increase in bronchial cancer which he tentatively attributes to smoking, atmospheric and other conditions. For similar early studies see Doll and Hill (1952, 1954, and 1956). Doll and Hill (1964) finds in 12 years of British data on 41,000 men and women that cigarette smokers have higher death rates from lung cancer. These differences are like those in Dorn (1959).

Thus, there have been major data sets gathered and new statistical techniques for mortality analysis developed over the previous decades. New findings on associations with smoking, occupational risks, birth cohort, education, income, marital status, race and gender have been of substantial interest. Still most of the previous studies have only begun to tap the potential of the methods and information available. In this and the next chapter, we hope to make advances in these dimensions. In the rest of this chapter we revisit the Dorn sample to explore particular questions related to the mortality associations with smoking, occupational risk, and birth cohorts as well as the sensitivity of the estimates to choice of functional form, length of the sample, and control for unobserved frailty. In Chapter 6 we revisit the RHS to investigate mortality associations with income, marital status, race and gender.

4.2. The Dorn Sample

To estimate the hazard health models presented in Chapter 3, we need to have data on death and on its covariates such as personal characteristics, consumption, and time use. Some data on personal and employment characteristics are available on death certificates, which are now available in the National Death Index. The death certificates have accurate data on date of death. However, the other information tends to have relatively large inaccuracies because it is not supplied by the deceased. Therefore, in our studies of mortality we use two "prospective" samples in which already interviewed people are followed for some period during which some of the interviewees died. In this chapter we use the Dorn sample, and in Chapter 5 we use the Retirement History Survey that we also used in Chapter 2 (and the sample characteristics of which are discussed in Section 2.3). Both of these samples have been used in previous analysis of adult mortality, but not utilizing the approaches that we introduce in Chapter 3 and use in this and the next chapter. As also is discussed in Chapter 3, statistical techniques are available for using data on the people who had not died before the end of the data collection period, and our results appear to be robust even when a majority of the group studied are still alive.

The Dorn sample was initiated by Dorn (1958) and was extended by Kahn (1966), Rogot (1974),[25] Rogot and Murray (1980). Dorn mailed a short questionnaire to

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[25] Rogot is our source for much of the description below.

293,958 U.S. Veterans, who had served in the Armed Forces between 1917 and 1940, and who in December 1953 held U.S. Government Life Insurance Policies. Table 4.1 summarizes data on the distribution across ages in 1954 and response rates. The overall response rate was about 67 percent. Men in this sample were born between 1870 and 1924. The largest group — more than 195,000 — was between 55 and 64 years old in 1954 (and thus born in the 1890's), but there are large sample sizes in all cohorts. The first (1870-1874) and last (1920-1924) quiennium birth cohorts are somewhat smaller.

The questionnaire, which is in Kahn (1966, appendix E), asked how many times a day a person smoked cigarettes, cigars, and/or a pipe both currently and in the past, how long ago he had stopped smoking, his age, and his current occupation and his industry of employment — both recorded at a three digit level. Since then the V.A. (Veterans Administration) has recorded deaths by month and year and by causes. A data tape that records death through 1980 has been assembled.

The sample is not a random draw of deaths of men in a given birth cohort since it only contains those veterans who were still alive and had V.A. insurance in 1954, and who responded to the questionnaire. Moreover, since the military had a minimum health requirement for inductees, the survival rates for veterans probably are slightly higher than for nonveterans (as found for a mostly later birth cohort by Behrman, Hrubec, Taubman, and Wales, 1980). Therefore, inferences to a broader population of individuals should be qualified, though control for unobserved heterogeneity may mitigate any selection bias.

In most of our work we exclude people for whom there is no information on any of the variables studied, leaving a sample of nearly 200,000. Plots of the age-specific death rates for the 200,000 and the full sample are nearly identical. Compared to the relevant population of males, Dorn (1958) and Kahn (1966), however, show that the sample has many fewer unskilled workers than the corresponding white male cohort. Also the proportion of nonwhites in the sample is quite small.

We present estimates below based first on about a 50 percent random subsample drawn from the 200,000 person sample. This sample size was used because it exhausted the memory capacity of a mainframe computer using a statistical method for which there are no sufficient statistics. We then use the same sample disaggregated into those born before 1891 and between 1891 and 1899. These results suggest some cohort differences; hence, we then use the whole set of responses, but we divide the respondents into five-year birth cohorts beginning in 1870 and ending in 1924. The estimates for these narrow birth cohorts give some insight regarding the stability of the parameters and suggest to us that a sample of men aged, say, 60 to 65 years old is not a representative sample of men born 60 to 65 years ago, at least when smoking is studied. This is the problem of left hand censoring since some of those born 60 to 65 years ago have already died. This may prove to be an important problem for prospective samples in general especially since little attention has been paid to left hand censoring and since the previous chapter's simulations indicate some non-robustness when left-hand censoring is important.

Epidemiologists have investigated the accuracy of the V.A.'s information on date of death (see Beebe and Simon, 1969; Cohen, 1953; DeBakey and Beebe, 1952). Details differ as to how accuracy was measured, but basically these researchers took death certificates of men in the appropriate age range, matched them to military records to obtain military serial numbers, and then gave these names and numbers to the V.A.

In the V.A. *population*, roughly 95 percent of the deaths were recorded by the V.A. Many of those not listed had been dishonorably discharged or were in the Army no more than four days during World War I. This high rate of coverage occurs because veterans draw pension benefits that cease at death and other benefits that commence at death (burial plots, a flag, and a burial allowance). In the Dorn sample the incentives to keep in touch with the V.A. were particularly strong since by design all participants had V.A. Life Insurance in force in 1954.

The Dorn death data for 1970-1980 were compiled only recently and have not yet been analyzed for completeness, but the data through 1969 have been examined in detail in this regard. For the Dorn sample, Rogot (1974) reports special efforts were made to check the 75,000 cases who had terminated their V.A. insurance between 1963-69 and he states, "The overall mortality follow-up, with respect to the fact of death and year of death is considered to be almost 100 percent complete."

Some sample characteristics of the Dorn sample are given in Table 5.2, which contains information for all cohorts, and for those people born before 1890 and from 1890 to 1899. The estimates in this table are based on 101,511 people who are randomly drawn from the survey's respondents.

We now describe how we use the Dorn sample's occupation and smoking variables, both of which are based on questions asked in the mid 1950's, and thus are prospective variables in the sense of being ascertained before mortality, but subject to future change (as are those used in other related studies referred to in Section 5.1). We use the information on a person's industry and occupation to assign occupational risks as in the *Underwriters' Handbook* used by the life insurance industry to set insurance premiums. Occupational risk depends on two components: risk and physical activity. The riskiest occupations are jobs such as fire-fighting and police-work, whereas the least risky are jobs such as teaching. The risk index ranges from one to seven as the riskiness of the occupation rises and has a mean of 2.2 with the cohort born before 1890 averaging 2.0 and the 1890-1899 cohort averaging 1.2. Likewise we assign an index for the physical activity of the occupation that ranges from one, for sedentary, to four, for heavy construction jobs using the physical capacity classification in United States Employment Service (1961). The overall mean is 2.0, and there is little difference by birth cohort.

The Dorn sample contains information on smoking in the form of how much tobacco the respondent used, how many years he used it, and the manner in which he used it — all from the perspective of 1954. Since the questionnaire was administered when the respondents' ages differed, we have divided the years smoked variables by "age - 10." These normalized variables have much higher "t" statistics than their non-normalized counterparts in otherwise identical equations. This adjustment, of course, is subject to measurement error since the choice of age 10 is arbitrary, but the results are not very sensitive to the exact age used for the adjustment since most respondents were at least 50 years old at the time this information was gathered. We use two tobacco-use variables. One is the number of (normalized) years of occasional tobacco usage. The second is the number of (normalized) years of regular tobacco usage.

Since there are generally only small differences between the two cohorts shown in Table 4.2, we concentrate here on the overall column. Normalized occasional and regular tobacco usage have averages of about 3 percent and about 50 percent of the time, respectively. Feldman et al. (1989) using the NHANES random sample report

that during the 1971-1975 period about 45 percent and 30 percent of males aged 45 to 64 and 65 to 74 were currently smokers. They do not present data on percent of time spent smoking. As noted earlier, there has been a down-trend in smoking; hence, the Dorn sample drawn from the mid 1950's is not out of line with this random sample.

About 25 to 30 percent of the people resided in each of the three non-western regions in the United States in the mid-1950's while about 20 percent resided in the west then. In the 1960 Census, for comparison, about 16 percent of the males lived in the west, and 22 to 33 percent in the other regions.

The Dorn sample, in contrast to the RHS, covers a non-random sample for whom very little data are available on independent variables, and these data were collected only once. The sample size, however, is huge with 300,000 people who were sent questionnaires and monitored for date of death which corresponds to about 100,000,000 man months of data. Therefore, because of computer constraints (these analyses did not use supercomputing resources),we use only one third of the original sample randomly selected when studying all birth cohorts. However, when we divide up by birth cohort, we can use all the people who responded to the mid-1950 surveys, which appears to be a random draw of those surveyed though not of the U.S. population of the same age.

4.3. Robustness of Estimates Over Time, Functional Form, and with Unobserved Heterogeneity

Three possible problems in earlier mortality studies such as those reviewed in Section 4.1 pertain to the length of samples, to the functional form assumption for the underlying hazard function, and to unobserved heterogeneity in individual frailties. We consider each of these in turn.

Length of Sample. A priori, extending the sample length may both worsen estimates in some respects and improve them in others. It may worsen the estimates because many studies in this genre depend in part on data about covariates, such as whether an individual smokes, that are recorded at the start of or early in the sample, but changes in such covariates over time are not be measured (an exception is Madans, 1987). Extending the sample period also may worsen the estimates because of changes in the underlying parameters due, for instance, to technological developments such as changes in cancer treatment.

On the other hand, extending the sample gives more observations over time, which may lead to greater precision in estimating coefficients. Extending the sample also lessens the magnitude of right censoring, i.e., more people will have died at older ages. In recent studies, right censoring is generally dealt with in one of three ways: making distributional assumptions about the form of the survivor function, using an unobserved baseline hazard and employing a partial likelihood estimator, or using nonparametric techniques. But the results may be sensitive to the specific assumptions that are made in these techniques. Finally, unobserved heterogeneity may cause more of a bias in the estimated duration dependence with samples of shorter duration since those who are more frail tend to die relatively early leaving a sample dominated by those that are less frail. Efforts to control for such unobserved frailty in the mortality context, once again, are relatively few, though Manton, Stallard, and Vaupel (1986) do so.

The impact of extending the sample on mortality estimates, given these conflicting a priori advantages and disadvantages is, of course, an empirical question. To our knowledge, however, there are not many, if any, systematic explorations of the empirical impact of extending a sample's time period.[26]

The first question that we consider is how stable are the results as the sample period lengthens from 1954-1969 to 1954-1980. Table 4.3 presents our Cox partial likelihood estimates for the period 1954-1969 in a sample of 101,511 persons, of whom nearly two-thirds were alive at the start of 1970. We present the results in terms of elasticities calculated at the sample means (except for the dichotomous variables). These results are described in detail in Behrman, Sickles and Taubman (1988). The proportional hazard function for all cohorts indicates a greater association with the hazard of dying: the more one smokes, the riskier one's usual occupation, the less physical activity in one's longest occupation,[27] and residing outside of the west (all variables measured in the mid 1950's). The elasticities are: 0.58 for smoking regularly; 0.21 for smoking occasionally; -0.02 for the occupational activity index; and 0.02 for the occupational risk index. Living in the south rather than elsewhere is associated with an increase of 0.6.

The two cohorts shown in the table yield elasticities with the same signs but with some instability in magnitudes. For example, the occasional tobacco use variable's elasticity is twice as large (though much more imprecisely estimated) in the cohort born before 1891 than in the 1891-1899 birth cohort, who had longer post-questionnaire expected lives during which to alter their smoking behavior. Below in Section 4.4 we present estimates for five-year age cohorts and find more pronounced differences.

Table 4.4 presents the corresponding results for data running through 1980. With the longer sample, there are some differences in coefficient estimates and their "t" statistics. We cannot tell if the coefficient differences reflect greater measurement error for the longer time period since the right-side variables were measured prospectively in the mid 1950's, the greater censoring problem in the shorter period for Table 4.3, or secular changes over time such as improvements in treatments for tobacco-usage related disease. But the combination of such considerations does seem to make some difference.

We first discuss the results for all cohorts and then summarize some of the results for the cohorts born before 1891 and during the period 1891-1899. For all cohorts the estimated impact of occasional tobacco use in Table 4.4 is slightly less than in Table 4.3, though not significantly so. However, the coefficient is estimated more precisely. Physical activity on one's usual occupation now yields an insignificant coefficient. The estimated effect of occupational risk is 40% large (though not significantly so), with a much larger "t" statistic. Those in the most risky occupations have a 16.8 percent higher hazard of dying than those in the least risky occupations. The coefficients on the 1950's residence data (in comparison with those residing in the west then) also

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[26] Alternative statistical methods have been developed to estimate a baseline survivor distribution (what percentage of the sample is alive at a given age) that shifts with covariates. See for example Cox (1972), Kalbfleisch and Prentice (1980), Manton, Stallard and Vaupel (1986), and Vaupel, Manton and Stallard (1979) and the discussion in Chapter 4.

[27] This physical activity result is also found in Moore and Haywood (1988) using the NLS older men.

change from Table 4.3 to Table 4.4. Inhabitants of the south have a much higher hazard in Table 4.4 and those from the northeast have a statistically significant positive coefficient in Table 4.4.

Tables 4.3 and 4.4 also contain results for the cohorts born before 1891 and 1891-1899. In Table 4.4 all coefficient estimates for the separate cohorts have the same sign as in the last column and have generally similar magnitudes. The regular tobacco use variable's coefficient is somewhat smaller in the later born cohort in Table 4.4.

The accelerated-time-to-failure model estimates for the period 1954 through 1969 are given in Table 4.5. For the gamma estimate, which is the most complex functional form and which has the largest log likelihood value, time to death is shorter: the more one smokes, if one lived in the south, the less one's physical activity, and the riskier one's job.[28] The elasticities on regular and occasional smoking frequency are -0.08 and -0.03, respectively, with both statistically significant. Those who lived in the south live about 0.005 shorter lives. The occupational activity variable has a significant elasticity of 0.005 while the occupational risk elasticity is a significant -0.005.

The longer sample findings are given in Table 4.6. These differ somewhat from those for the shorter period. The tobacco-use variables have smaller coefficient estimates in absolute value in Table 4.6 than in Table 4.5, and the differences in the coefficient estimates in these two tables for regular tobacco-use are significant. For regular tobacco use, the elasticities are in the -0.06 to -0.07 range and have "t" statistics in the high 40's. The corresponding estimates in Table 5.5 are about -0.08. The occupational variables, in contrast, have some larger and some smaller coefficients in Table 4.6 than in Table 4.5 with some of the differences statistically significant. The occupational activity variable consistently has smaller coefficient estimates and smaller "t" values in Table 4.6. The occupational risk variable consistently has larger "t" statistics and larger point estimates (in absolute values) in Table 4.6. This may indicate that the advantage of prior exercise atrophies quickly when one stops the activity while the effects of work-related health hazards such as exposure to carcinogens do not vanish as quickly. The regional controls have larger "t" values in two-thirds of the cases.

The accelerated-time-to-failure estimates are presented in Tables 4.7 and 4.8 for the two age cohorts born before 1900 for the periods through 1969 and 1980 respectively. Here we use only the gamma baseline failure time, our preferred distribution on the basis of the earlier results. For both time periods, tobacco usage is important, but the results are stronger for the later-born cohort in both tables. The absolute magnitudes of the coefficients are greater and, in three of the four cases, the t-statistics are larger for the longer period.

For the longer time period, the occupation risk index has significant coefficient estimates and about the same elasticity for both cohorts. In the shorter time period, it has a significant coefficient estimate for only the older cohort with a similar elasticity as in Table 4.8. The occupational activity index has a significant coefficient estimate only for the younger cohort in both time periods; this estimate is much larger once

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[28] Of course the signs of the elasticities are reversed since the theoretical concept has changed from the age-specific death rate to how long you will survive. Most of the same results hold for the other baseline hazard distributions, but not all (e.g., the Northeast regional effects are significantly nonzero for the Weibull and lognormal cases).

again for the shorter time period for which measurement error and parametric changes are likely to be less important.

Sensitivity to Functional Form Choices. We now consider how sensitive our results are to choices regarding functional forms. We examine various specifications for the accelerated- time-to-failure model. The Cox and Weibull proportional hazard models differ in their theoretical form from the Weibull accelerated hazard by an estimated scale factor; hence, we can also compare these estimates.

Consider first the results for the period 1954-1969 in Table 4.5 and those for 1954-1980 in Table 4.6. In both cases, for a given sample length, the patterns across functional forms are quite similar. All four baseline hazards fit the data about equally well in terms of the maximized values of the log likelihood function, with the gamma model slightly the best fitting and the lognormal the worst. For the tobacco variables, the point estimates and "t" statistics are very similar in the four columns. The occupational and regional coefficient estimates have much greater variation, though the signs of the significant estimates are the same across the estimates.

A comparison of the Cox proportional hazard (Table 4.3 and 4.4, column 3) and the Weibull accelerated-time-to-failure models (Tables 4.5 and 4.6, column 1) indicates that, as expected, coefficient signs are reversed and the coefficients in the Cox model equal those in the Weibull model divided by the estimated scale parameter (0.128 in Table 4.5 and 0.109 in Table 4.6). That the covariate estimates across different functional forms for the death hazard are comparable despite the substantial differences in the highly nonlinear functional forms for the accelerated-time-to-failure and proportional hazard models is of interest. However, the age profiles for the logarithm of the hazard (relative to which our estimated covariate effects are calibrated) are fairly flat. The nonlinear covariate estimates are evaluated at the mean of the sample, but there may be little nonlinearity at this point in the sample space; thus, our finding of robustness across different functional forms may be an artifact of the pattern of our sample death rates.

Unobserved Heterogeneity in Frailty. The previous results assume no individual differences in unobserved frailty. Manton, Stallard, and Vaupel (1986) argue that one can expect heterogeneity in a mortality hazard model. On a priori grounds the presence of such heterogeneity seems likely since critical variables, such as genetic stocks, are not observed. Heterogeneity can be modeled as following a particular parameterization as in Manton, Stallard, and Vaupel (1986) or a nonparametric distribution as in Heckman and Singer (1984). We use both methods. To make these calculations we use a NEC SX-2 supercomputer.

To investigate the importance of heterogeneity, we use a Weibull *proportional* hazard model whose derivation is presented in Section 3.6 In Table 4.9 we present estimates for the pre-1890 cohort. The first column presents the model estimated with no allowance for heterogeneity. This column has elasticities similar in sign and magnitude to those obtained with the Cox proportional hazard model in column 1 of Table 4.4, though the coefficient estimate on the occupational risk index is much larger (and statistically significant) in Table 4.9 than in Table 4.4.

The Heckman-Singer nonparametric model using two points of support,[29] given in column 2, fits the data a bit better than do the estimates in column 1. The coefficients on the variables that were significant in column 1 remain significant with those on regular tobacco use and on the ln of duration increasing significantly.

The estimates assuming that heterogeneity follows a normal distribution are given in column 3.[30] This estimator fits the data slightly worse than the Heckman-Singer model and slightly better than the model with no allowance for heterogeneity. The "t" statistics tend to be lower with that on the "occasional tobacco" use parameter falling to 1.75. The coefficients are very similar to those in the first two columns though a little closer to the ones in the first column.

Table 4.10 contains the 1891-1899 cohort Weibull proportional hazard results. These highlight differences obtained in very large samples between specifications with and without heterogeneity. The model without heterogeneity yields parameter estimates somewhat different from those obtained with the Cox model (Table 4.4). In Table 4.10 the occupational activity and especially the occupational risk indices have much bigger impacts (in absolute value), but the regional and tobacco-use variables have smaller effects (some significantly so). Based on the second column in Table 4.10, the allowance for nonparametric heterogeneity improves the fit slightly, but it does not alter the overall impression as to the magnitude of the impact of the coefficients. For example, the regular tobacco-use coefficient estimate goes from 0.45 to 0.48 and the occupational activity index's coefficient estimate goes from -0.084 to -0.098.

Comparison of Tables 4.9 and 4.10 shows that the occupational activity index has an insignificant coefficient estimate for the older cohort, but a significant one for the younger group. The occupational risk index has a somewhat larger coefficient in the older cohort, though not significantly so. The smoking effects are somewhat larger for the older cohort (significantly so for regular use) when heterogeneity is controlled.

We also use the procedure proposed by Lancaster (1985), detailed in Section 3.2 to test for unobserved heterogeneity. The test is based on generalized residuals (Cox and Snell, 1968) and is a special case of the information matrix test for misspecification proposed by White (1982) and expanded upon in the context of duration models by Chesher (1984). We consider here only frailty differences among individuals that effect the constant term, not the entire set of parameters as in the tests of White and Chesher.

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[29] We have one estimate using three points of support for this cohort. Most parameter estimates are very similar to those in Table 5.9, but the coefficient on regular tobacco use rises sharply to 0.58. The third support increases CPU time on the NEC SX-2 supercomputer used in these analyses by about 50 percent. For a sample of about 13,000 observations, the running time is about 4.5 hours.

[30] Although distributions other than the normal can be specified in the CTM program (Yi, et al., 1986), the extreme computational demands implied by the relatively large number of covariates and sample observations mean that only a limited number of parametric heterogeneity distributions could be considered. These included the lognormal and gamma. However, due to convergence problems and CPU constraints, the only parametric heterogeneity distribution on which we report estimates is the normal. Although the normal is not a flexible distribution, its ubiquity in applied work and justification therein provides us with some rationale for its selection for this study.

Computational constraints limited the use of this test to the first cohort of the Dorn sample, in which we have approximately 13,000 individuals. The results are summarized in Table 4.3.

Although results from the three classes of models suggest the presence of heterogeneity, the evidence seems to indicate that modeling of unobserved heterogeneity directly in a proportional hazard setting may not be as important as allowing covariates to affect the hazard as in the highly nonlinear gamma accelerated-time-to-failure model. Comparisons of alternative methods to reduce the impact of distributional assumptions about the form of heterogeneity have generally been made in the context of proportional hazard models with few covariates and limited sample sizes. These last results suggest that renewed focus might be placed on generalizing the specification of the conditional hazard, as well as on limiting the distributional impacts of unobserved heterogeneity.

4.4. More Detailed Cohort Effects

In Section 4.3 we allow for cohort effects for about half of the respondents. By using all 200,000 who responded in the mid 1950's, we can break up the full sample into five-year cohorts and obtain reasonable sample sizes for all five-year cohorts starting with those born between 1875 and 1879 up to the cohort that runs through 1924. For those alive in 1954 the oldest cohort was in their eighties while the youngest was in their thirties.

Our estimates in Table 4.11 do not indicate that improvements in health technology and more enlightened preventive health measures were adopted more frequently by younger cohorts than older ones. However it does appear that heavy tobacco use (which has a long latency period) increases the hazard of dying for younger cohorts more than for older ones. Data on level of tobacco use should be the most accurate for the older cohorts, so this result would not seem to reflect greater measurement error for older cohorts. Therefore the cohort differences may reflect left-hand censoring problems — see the simulation results in Section 3.7 — and suggest that some results may be unstable when the sample is surveyed after age 50 or 55.

Furthermore, the cohort patterns trend less when the maximum penalized likelihood estimator is used than with the Cox partial likelihood estimator. This latter finding suggests that the spurious heterogeneity findings associated with cohort effects is not completely ameliorated by analyzing separate cohorts alone and that unobservable frailty still needs to be adequately controlled for in estimation. Note that in the 1954-1980 data set the censored observations range from 2 percent in the 1875-79 cohort to 87 percent in the 1920-1924 cohort and, not surprisingly, decrease with age.

In the maximum likelihood estimates in Table 4.11, in the 1875-79 cohort, the only significant coefficients are living in the South in 1954. These people have a smaller hazard of dying than those in the West.

In the 1880-84 cohort both the occasional and regular tobacco use (normalized by Age-10) variables have significant effects that increase the hazard, though the occasional smoking effect may be greater because in this age cohort many of the regular smokers died earlier. There are no significant regional effects.

Throughout the rest of Table 4.11, the effect of occasional smoking increases the hazard, though this coefficient is only significant for cohorts born up to 1895-99. After this birth period, censoring exceeds 40 percent and relatively few people are in the sample.

The regular smoking effects are much more pronounced and are statistically significant in each cohort beginning with the 1880 group. For each successive cohort through the 1910-14 group, each normalized year of regular smoking causes greater increases in the hazard of dying. The two youngest cohorts, where the censoring rate is 85 to 90 percent, still have highly significant coefficients and substantial effects on the hazard. Recall that the cigarette information was collected during the mid 1950's and covers a longer portion of a person's lifetime the older he is though our normalization of dividing by (Age-10) makes the numbers in each cohort more comparable. Nevertheless, the effects of cigarette smoking are generally more harmful in the cohorts born more recently. Note also that the implied standard errors, which vary by cohort size and percent censored, remain fairly small even for the 1920-1924 cohort. Given that each age cohort only covers five birth years, it seems unlikely that data on the independent variables only measured in the mid 1950's explains the sharp trends in the effects of regular smoking in the Cox hazard. However, it is possible that heterogeneity in frailty is correlated with smoking. Conceptually controls for selectivity are straightforward. We could include the inverse of the Mills ratio as an independent variable, e.g. Heckman (1979), but we have too little information in the sample or on the population to do so. Since we have not done so, we may have an omitted variable bias.

Next consider the 1954 region of residence effects. With the exception of the 1875, 1905, and 1920 cohorts, those living in the South have higher hazards than those in the West, but the positive coefficients are only significant in the cohorts of 1885-1899. Similar conclusions hold for those in the Northeast except that the 1905 cohort has a positive but insignificant coefficient. Most of the North Central coefficients are positive, but only the 1885 and 1895 birth cohorts are significant. Overall, the regional differences seem to be greatest for the 1895-99 cohort (ignoring the small first cohort) many of whom would have retired around the time of the survey.

Increases in the physical activity of one's job only has a significant effect for those in the 1895-99, 1905-09, and 1915-19 cohorts. For this group the hazard is reduced 5 percent when moving from sedentary to heavy activity. The life insurance premium index is more important and has significant and similar positive effects for the 1885-99 cohorts. The difference between the most and least risky job adds about 10 percent to the hazard.

Table 4.12 contains results for the Cox model for people who never smoked prior to the mid 1950's. The only significant coefficients are for the South in the 1890 and 1895 cohorts, the Northeast in the 1890 cohort, and the North Central in the 1895 cohort. All of these coefficients indicate increased hazards of 7 to 14 percent as compared to those of the same age living in the West.

Table 4.13 contains results for those who smoked occasionally. Only the 1890-94 and 1895-99 cohorts contain large samples. In these cohorts the impact of normalized tobacco usage is 13 and 7 percent (not significant), which are marginally smaller than the effects calculated in Table 4.11 for the whole sample. There are regional residence

effects with those in the South and Northeast (in the 1890 cohort) having noticeably elevated hazards. The risk index is important for those in the 1895-99 cohort.

Table 4.14 is for regular smokers. The percent of the time they smoked regularly has highly significant effects beginning with the 1880-84 cohort, which has an elasticity of 0.34. In general, the more recent the birth cohort the more the hazard of dying shifts up. The big jump occurs in the 1900-04 cohort, which was about 50 to 55 years old at the survey date and which has an elasticity of 0.85. The largest elasticity is 0.99 for the 1915-19 cohort. Those from the South and Northeast have significantly higher hazards in the 1885-99 cohorts as do those in the North Central in the 1885 cohort. Physical activity on the job reduces the hazard for the 1890-94 cohort but raises it for the 1905-09 cohort. Higher life insurance risk increases the age-specific death rate for the 1890-94 and 1895-99 cohorts.

4.5. Maximum Penalized Likelihood Estimators

At this point we present some estimates based on the Maximum Penalized Likelihood method. Note that the MPLE results control for unobserved heterogeneity while the results using the Cox model have no explicit controls for unobserved heterogeneity, which can vary by cohort.

In Table 4.15 the (normalized) years smoked occasionally raises the hazard of dying by about 20 percent, which is statistically significant for the birth cohorts beginning with the 1880-84 cohort (except for the 1905-09 cohort). There is no marked trend across cohorts. Heavy smoking increases the hazard by 50 to 80 percent with the larger shifts for the more recently born cohorts. The geographical differences in place of residence in the mid 1950's are small and generally insignificant. Greater physical activity leads to lower hazards, which are significant and of noticeable size for the four successive cohorts beginning in 1885 birth cohort, who were mostly at work in the mid 1950's. The life insurance assessment of greater occupational risk shifts the hazard up significantly except for the birth cohorts of 1870-74, 1880-84, 1905-09, and 1920-24. The increase is generally about 0.03 with no obvious trend across cohorts.

Compared with Table 4.11 the results in Table 4.15, in which the impact of unobserved heterogeneity is reduced, show much less of a trend for the effects of heavy smoking but still have somewhat bigger impacts in the older birth cohorts. The coefficients of the job characteristics are raised in absolute value.

For non-smokers there are major differences in the size of insignificant variables and standard errors, but no difference in the sign of significant variables. More MPLE than Cox estimates are statistically significant (12 versus 10) including those in some smaller birth cohorts. The activity variable tends to have larger coefficients (in absolute value) in the MPLE estimators. When the occupational risk variable is significant, the new estimates are much larger while the geographic differences are muted.

For light smokers, the size of coefficients and standard errors alter but the signs of significant variables are unchanged. The variable for years of light smoking is significant for all the birth cohorts between 1880 and 1924 except the 1905-1914 ones. Most of the elasticities are about 0.2 though it is about 0.38 in the 1880 cohort and 0.11 in the 1900 cohort, indicating that the hazard is more elevated when significant than in

Table 4.13. The MPLE estimates of the number of normalized years of light smoking are much larger than the Cox estimates. The coefficients on job physical activity and risk tend to indicate bigger impacts when we use the MPLE estimator. The geographic differences are now more pronounced.

For heavy smokers considered alone, signs on significant variables (in both Tables 4.14 and 4.18) remain unchanged, but the size and the standard errors of the coefficients are altered noticeably. The number of significant coefficients in Table 4.18 is 30 while in Table 4.14 it is 25. The more years one smokes heavily has substantially larger coefficients in Table 4.18 for most cohorts. Both the occupational physical activity and risk variables have bigger impacts in the MPLE results.

The coefficients on years of heavy smoking are significant for all cohorts from 1880-1920 in Table 4.18. The elasticities in 1880 are about 0.6 and increase for nearly every cohort (except the youngest) to about 0.8. The difference is statistically significant. The increase in the hazards over birth cohorts are less pronounced in Table 4.18 than in Table 4.14 which went from 0.34 to 0.99. It is difficult to believe that such a trend as in Table 4.18 over narrow cohorts is due to changes in medical technology that affects primarily the elderly. Instead it seems likely that the people alive in the mid 1950's and over the age of, say, 40 or 45 are already a "selected" population and that the effects of smoking have already weeded out the most frail portion of the younger smokers.

In the prior section we have examined the robustness of mortality hazard analysis with respect to assumptions on functional form of the hazard and to the allowance of heterogeneity using parametric and non-parametric representations. Generally we have found our estimates to be robust to other explorations. But here a major shift in our estimation strategy has resulted in major differences in the coefficient estimates.

The maximum penalized likelihood estimates generally show much less of a trend in the effects of smoking by birth cohort than the maximum likelihood estimates. The differences by five-year birth cohort appear to be too steady and pronounced to be explained by changes in medical technology, but may be explained by left-side truncation with an omitted selectivity variable correlated with the smoking variable.

4.6. Starting the Analysis Later

An explanation of the trends on the cigarette usage variables in the prior section is that left censoring has left us with a non-random sample of hardier people still alive in the mid 1950's. In this section we pretend that the sample began in 1964, delete all people who died prior to 1964 and estimate a model for the Cox proportional hazard.

The results are presented in Table 4.19. In the 1870-74 and the 1875-79 cohorts, which have few people, the smoking coefficients are statistically insignificant. Beginning with the next cohort the normalized years of heavy smoking variable has a coefficient of 0.37 which is significant. As we move across birth cohorts, the same coefficient is 0.37, 0.47, 0.54, 0.82, 0.67, 0.97, 0.96 and 0.87. Again we are finding a strong upwards trend in this coefficient as we move to younger people. In comparison with the results in Table 4.11, which uses the whole sample period, the coefficients now are about the same or somewhat larger, but only for the cohorts born most recently do the elasticities jump and reach a level of close to one.

We leave for future research the solution of the left-censoring problem. One possible solution would be to start another sample at an earlier age. This, however, involves a trade-off since one would have to wait much longer for a sufficient number of people to have died, or trade-off left-censoring for right-censoring problems. Finally we note that major instability in the coefficients occur to those over 50 years old when the sample began.

4.7. Conclusion

In this chapter we have used the Dorn sample, which covers veterans who were born during the period 1870 through 1924 and who had V.A. life insurance at the end of 1953, to estimate both proportional hazard and accelerated-time-to-death models.

When we examine these data for all cohorts, we find that heavy smoking (normalized by Age-10) is highly significant and reduces the time to death by a noticeable and statistically significant amount. Less frequent smoking also has a significant but smaller impact. We also find some impact of occupational physical activity, occupational riskiness, and geographic location.

We have examined the robustness of all our cohort estimates to functional form, unobserved heterogeneity, and time period variations. Cox and Weibull proportional hazard models with no allowance for unobserved heterogeneity yield very similar coefficients for the smoking and for the other variables except for the occupational risk variable. The accelerated-time-to-failure models generally yield similar coefficients on the observed variables, and the Weibull version of this model — adjusted for the estimated scale parameter — is similar to the Cox proportional hazard model that has the same theoretical parameters.

Variation in the time period studied has several effects. Lengthening the time period covered from 1954-1969 to 1954-1980 tends to make the estimated coefficients more precise, but also tends to reduce the elasticities of the smoking variables. The first effect probably occurs because we have many more observations (man months) and because far fewer observations are censored (alive at the end of the sample period). The second effect may occur because the mid 1950 data on smoking, geographic location, and occupational characteristics may be more outdated as time passes. This suggests that it may be worthwhile to update information in prospective studies as in Madans, et al. (1986a) though this is expensive, and the Dorn sample certainly gives sharp estimates over a 25-year span with no updating.

A comparison of the estimates of the Weibull proportional hazard model without and with allowance for parametric and non-parametric unobserved heterogeneity indicates that the allowance for such heterogeneity allows us to fit the data better. The Heckman-Singer non-parametric methodology fits the data best, but the improvement in fit is not huge, the coefficients on the measured variables are changed by modest amounts, and the extra computer costs are large. Modelling unobserved heterogeneity as being normally distributed achieves much of the same improvement at a lower cost.

The results for five-year birth cohorts, however, reveal some important differences. The elasticity of heavy smoking is much smaller for cohorts born before 1900. We believe this reflects left censoring with the more frail smokers dying before the sample commenced. This receives rough confirmation from Table 4.19 when the death data

are restricted to the period 1964-1980. Stronger confirmation is given in Tables 4.15-4.18 that are based on the Maximum Penalized Likelihood estimation technique. By giving less weight to outliers, this method reduces the impact of unobserved heterogeneity. The smoking elasticities have much less of a trend by cohort and overall are not as large. This reduction is also found in all the cohort estimates when we allow explicitly for parametric or non-parametric heterogeneity.

If the cohorts born in 1900 and later are more representative since fewer deaths exacerbate the frailty problem, the increase in the hazard with heavy smoking may be closer to 100 percent than the 50 to 60 percent estimate obtained in the whole sample.

Table 4.1 Age and Response Distribution, Dorn Sample

Age in 1954	Responders 1954	Responders 1957	No Reply	Total
30-34	7,421	43	2,148	9,612
35-44	16,735	7,156	4,037	27,928
45-54	10,317	1,242	2,232	13,791
55-64	137,820	26,579	31,468	195,867
65-74	25,002	13,683	5,603	44,288
75-84	1,525	523	424	2,472
30-84	198,820	49,226	45,912	293,958

Source: Rogot (1974: 192)

Table 4.2 Dorn Sample: Summary Statistics for Different Cohorts

Variable	Cohort Born Before 1890		Cohort Born 1890-1899		All Cohorts[a]	
	Mean	Standard Deviation	Mean	Standard Deviation	Mean	Standard Deviation
Smoking						
Proportion of Years Used Tobacco Occasionally[a]	.0330	.142	.0318	.143	.0307	.138
Proportion of Years Used Tobacco Regularly[a]	.495	.327	.546	.353	.543	.343
Occupation						
Activity Index	2.17	.848	2.13	.830	2.19	.841
Risk Index	2.01	.158	1.19	.156	2.20	.170
Region						
South	.258	.438	.240	.425	.249	.430
Northeast	.300	.458	.307	.461	.290	.452
North Central	.264	.441	.289	.452	.271	.442
Number of Observations	12,822		69,991		101,511	
% Censored						
(Through '69)	40.4		63.7		65.5	
(Through '80)	8.5		22.9		33.3	

[a]Includes those born after 1899.

Note: Number of years of occasional and regular tobacco use is divided by age at survey date minus 10 years.

Table 4.3 Dorn Sample, 1954-1969 Cox Partial Likelihood Proportional Hazard Model

Variable	Cohort Born before 1891 Estimate (t-statistic)	Cohort Born 1891-1899 Estimate (t-statistic)	All Cohorts[a] Estimate (t-statistic)
Smoking			
Proportion of Years Used Tobacco Occasionally	0.532 (.562)	.287 (5.78)	.212 (4.44)
Proportion of Years Used Tobacco Regularly	.454 (11.68)	.624 (30.3)	.577 (32.8)
Occupation			
Activity Index	-.013 (-.26)	-.022 (-2.21)	-0.021 (-2.43)
Risk Index	.0013 (.135)	.014 (2.69)	.020 (4.71)
Region			
South	.057 (1.63)	.078 (3.87)	.055 (3.30)
Northeast	.014 (.404)	.077 (3.96)	.027 (1.65)
North Central	.034 (.969)	.051 (2.58)	.0088 (.534)
Log Likelihood	-69998	-278404	-387619
X^2	192.8	1522.8	2113.2

[a]Includes people born in the period 1900-1924 in addition to those born before 1900.

Table 4.3A Specification Error Tests

Model	Score-statistic
Cox's Proportional Hazard (Partial Likelihood)	-15.9
Nonparametric Maximum Likelihood Estimate with Weibull Hazard (Two Points of Support)	-11.1
Gamma Accelerated Hazard	-2.61

Table 4.4 Dorn Sample, 1954-1980 Cox Partial Likelihood Proportional Hazard Model

Variable	Born before 1891 Estimate (t-statistic)	Born 1891-1899 Estimate (t-statistic)	All Cohorts[a] Estimate (t-statistic)
Smoking			
Proportion of Years Used Tobacco Occasionally	.171 (2.34)	.189 (5.46)	.187 (6.14)
Proportion of Years Used Tobacco Regularly	.425 (13.6)	.550 (38.8)	.564 (45.0)
Occupation			
Activity Index	-.0014 (-.099)	-.0062 (-.86)	-.0087 (-1.42)
Risk Index	.012 (1.59)	.016 (4.28)	.028 (8.83)
Region			
South	.063 (2.24)	.090 (6.28)	.070 (5.73)
Northeast	.014 (.52)	.060 (4.35)	.026 (2.19)
North Central	.059 (2.10)	.028 (2.04)	.0072 (.60)
Log Likelihood	-101289	-530670	-716098
χ^2	203.9	1699.5	2329.5

[a]Includes people born in the period 1900-1924 in addition to those born before 1900.

Table 4.5 Dorn Sample, 1954-1969 Accelerated-Time-to-Failure Models, All Cohorts

Variable	Weibull Estimate (t-statistic)	Lognormal Estimate (t-statistic)	Loglogistic Estimate (t-statistic)	Gamma Estimate (t-statistic)
Intercept	5.20 (1750.)	5.15 (1666.)	5.14 (1718.)	5.15 (1572.)
Smoking				
Proportion of Years Used Tobacco Occasionally	-.032 (-5.73)	-.029 (-5.22)	-.033 (5.99)	-.029 (-5.07)
Proportion of Years Used Tobacco Regularly	-.080 (-35.0)	-.081 (-35.2)	-.083 (-36.8)	-0.081 (-35.0)
Occupation				
Activity Index	.003 (2.78)	.0044 (3.85)	.0037 (3.33)	.0047 (4.00)
Risk Index	-.002 (-4.34)	-.0048 (-8.28)	-.0031 (-5.46)	-.0054 (-8.99)
Region				
South	-.007 (-3.43)	-.0055 (-2.43)	-.0073 (-3.31)	-.0051 (-2.20)
Northeast	-.004 (-2.02)	.00002 (.03)	-.0042 (-1.98)	.00113 (.51)
North Central	-.003 (-1.35)	.0015 (.67)	-.0023 (-1.08)	.00250 (1.11)
Scale	.128 (228.7)	.186 (247.3)	.104 (226.)	.192 (150.1)
Shape	—	—	—	-.122 (-6.58)
Log Likelihood	-204263	-201959	-202457	-201931
χ^2	1724.	1572.	1778.	1855.

Table 4.6 Dorn Sample, 1954-1980 Accelerated-Time-to-Failure Models, All Cohorts

Variable	Weibull Estimate (t-statistic)	Lognormal Estimate (t-statistic)	Loglogistic Estimate (t-statistic)	Gamma Estimate (t-statistic)
Intercept	5.15 (2899.)	5.10 (2473.)	5.10 (2576.)	5.13 (2599.)
Smoking				
Proportion of Years Used Tobacco Moderately	-.021 (-6.44)	-.021 (-5.55)	-.023 (-6.33)	-.022 (-6.22)
Proportion of Years Used Tobacco Heavily	-.064 (-46.5)	-.071 (-46.0)	-.071 (-48.1)	-.068 (-47.0)
Occupation				
Activity Index	.0011 (1.63)	.0027 (3.39)	.0020 (2.70)	.0016 (2.25)
Risk Index	-.003 (-8.65)	-.0056 (-14.2)	-.0040 (-10.3)	-.0038 (-10.3)
Region				
South	-.008 (-5.98)	-.0055 (-3.53)	-.007 (-4.68)	-.0074 (-5.18)
Northeast	-.003 (-2.43)	.0015 (1.0)	-.0022 (-1.55)	-.0021 (-1.5)
North Central	-.001 (-.846)	.0035 (2.29)	.0001 (.073)	.00016 (.12)
Scale	.109 (321.6)	.146 (357.3)	.082 (361.)	.126 (229.3)
Shape	—	—	—	.545 (29.2)
Log Likelihood	-338697	-339291	-338209	-337936
χ^2	2532.	2227.	2601.	2789.

Table 4.7 Dorn Sample, 1954-1969, Accelerated-Time-to-Failure Model, Gamma Baseline Failure Time, Two Cohorts

Variable	Cohort Born Before 1891 Estimate (t-statistic)	Cohort Born 1891-1899 Estimate (t-statistic)
Intercept	5.03 (1090.)	4.97 (1435.)
Smoking		
Proportion of Years Used Tobacco Occasionally	-.012 (-1.65)	-.015 (-3.06)
Proportion of Years Used Tobacco Regularly	-.029 (-9.23)	-.043 (-21.3)
Occupation		
Activity Index	.0016 (1.05)	.0042 (3.99)
Risk Index	-.0016 (-1.97)	-.0006 (-1.11)
Region		
South	-.00069 (-.236)	-.0058 (-2.74)
Northeast	.00052 (.18)	-.00447 (-2.19)
North Central	-.0054 (-1.86)	-.00296 (-1.45)
Scale	.11 (85.4)	.174 (171.2)
Shape	-1.79 (-37.7)	-2.36 (-74.9)
Log Likelihood	-37930	-137900
χ^2	82.5	334.0

Table 4.8 Dorn Sample, 1954-1980, Accelerated-Time-to-Failure Model, Gamma Baseline Failure Time, Two Cohorts

Variable	Cohort Born Before 1891 Estimate (t-statistic)	Cohort Born 1891-1899 Estimate (t-statistic)
Intercept	5.09 (1271.)	5.09 (2064.)
Smoking		
Proportion of Years Used Tobacco Occasionally	-.015 (-1.64)	-.023 (-5.76)
Proportion of Years Used Tobacco Regularly	-.041 (-14.3)	-.064 (-39.5)
Occupation		
Activity Index	.0011 (.77)	.0019 (2.23)
Risk Index	-.0017 (-2.24)	-.0015 (-3.39)
Region		
South	-.045 (-1.64)	-.010 (-5.72)
Northeast	-.00066 (-.245)	-.0078 (-4.76)
North Central	-.0056 (-2.06)	-.0042 (-2.55)
Scale	.10 (150.4)	.137 (262.8)
Shape	-.29 (-9.44)	-.141 (-8.30)
Log Likelihood	-52402	-245401
χ^2	119.	527.

Table 4.9 Weibull Proportional Hazard with and without Unobserved Heterogeneity Dorn Sample, 1954-1980, Cohort Born Before 1891

	No Heterogeneity	Nonparametric Heterogeneity[a]	Normal Heterogeneity
Variable	Estimate (t-statistic)	Estimate (t-statistic)	Estimate (t-statistic)
Smoking			
Proportion of Years Used Tobacco Occasionally	.180 (2.74)	.196 (2.51)	.150 (1.75)
Proportion of Years Used Tobacco Regularly	.458 (15.9)	.542 (15.7)	.424 (3.21)
Occupation			
Activity Index	.00435 (0.08)	-.00777 (0.12)	-.0380 (0.61)
Risk Index	.0975 (2.03)	.123 (2.12)	.134 (1.95)
Region			
South	.0668 (2.69)	.0775 (2.54)	.0501 (1.54)
Northeast	.0189 (0.76)	.0207 (0.69)	.008 (0.29)
North Central	.0695 (2.79)	.0759 (2.5)	.0593 (1.77)
In Duration	9.87 (112.2)	11.29 (117.9)	.93 (97.5)
Log Likelihood	-52019	-50427.	-50662.
χ^2	129	147	139

[a]With two points of support.

Table 4.10 Weibull Proportional Hazard with and without Unobserved Heterogeneity Dorn Sample, 1954-1980, Cohort Born (1891-1899)

	No Heterogeneity	Nonparametric Heterogeneity
Variable	Estimate (t-statistic)	Estimate (t-statistic)
Smoking		
Proportion of Years Used Tobacco Occasionally	0.164 (4.40)	0.158 (3.74)
Proportion of Years Used Tobacco Regularly	0.448 (28.7)	0.475 (26.7)
Occupation		
Activity Index	-0.084 (2.73)	-0.098 (-2.95)
Risk Index	0.073 (2.73)	0.094 (3.15)
Region		
South	0.040 (2.67)	0.035 (1.92)
Northeast	0.028 (1.93)	0.013 (0.80)
North Central	0.009 (0.58)	0.0027 (0.17)
In Duration	7.01 (202.)	8.07 (212.)
Log Likelihood	-247002.	-241613.
X^2	501.	519.

Table 4.11 Cox Models with Dorn Data (Five-Year Cohorts) From 1870-1924 (t statistics in Parentheses)

	Smoking		Region			Occupation	
	Proportion of Years Used Tobacco Occasionally	Proportion of Years Used Tobacco Regularly	South	Northeast	North Central	Activity Index	Risk Index
1870-74	.2833	.0706	.1399	.1483	-.0225	.0125	-.0267
[N=265, Censored=4]	(.55)	(.29)	(.74)	(.78)	(-.11)	(1.0)	(-.33)
1875-79	-.2650	.0265	-.2026	-.0745	.0036	-.0126	-.0306
[N=1038, Censored=23]	(-.86)	(.22)	(-2.0)	(-.75)	(.04)	(-.21)	(-1.02)
1880-84	.4957	.3075	.0293	.0171	-.0354	-.0192	-.0059
[N=2902, Censored=97]	(3.3)	(4.4)	(.49)	(.34)	(-.59)	(-.64)	(-.30)
1885-89	.0996	.4409	.0862	.0582	.0752	-.0011	.0133
[N=21329, Censored=2055]	(1.7)	(22.0)	(4.3)	(2.9)	(3.8)	(-.11)	(1.3)
1890-94	.1670	.5097	.0711	.0486	.0230	-.0062	.0167
[N=65389, Censored=13699]	(5.6)	(51.0)	(7.1)	(4.9)	(2.3)	(-.62)	(4.2)
1895-99	.1414	.5692	.0937	.0529	.0475	-.0163	.0190
[N=74981, Censored=25386]	(3.5)	(56.9)	(4.7)	(5.3)	(4.8)	(-1.6)	(4.8)

Table 4.11 Cox Models with Dorn Data (Five-Year Cohorts) From 1870-1924 (t statistics in Parentheses) (Continued)

	Smoking		Region			Occupation	
	Proportion of Years Used Tobacco Occasionally	Proportion of Years Used Tobacco Regularly	South	Northeast	North Central	Activity Index	Risk Index
1900-04	.1769	.8521	.0761	.0660	.0152	.0223	.0100
[N=8253, Censored=3755]	(1.3)	(17.0)	(1.5)	(1.7)	(.30)	(.74)	(1.0)
1905-09	-.1456	.7623	-.0314	.0955	.0199	.1192	-.0251
[N=2022, Censored=1357]	(-.36)	(5.4)	(-.31)	(.87)	(.17)	(2.0)	(-1.3)
1910-14	.5398	.9711	.0371	.1081	.0192	-.0047	-.0012
[N=4743, Censored=3728]	(1.9)	(8.1)	(.46)	(1.2)	(.21)	(-.12)	(-.06)
1915-19	.201	.9693	.0790	.0338	.0676	-.0494	-0150
[N=13538, Censored=11425]	(.88)	(12.1)	(1.3)	(.56)	(1.1)	(-1.6)	(-1.5)
1920-24	.2138	.7572	-.0872	-.1888	-.1068	-.0400	-.0229
[N=8668, Censored=7489]	(.61)	(6.3)	(-1.1)	(-2.4)	(-1.3)	(-1.0)	(-1.1)

Table 4.12 Cox Models with Dorn Data (Five-Year Cohorts) From 1870 To 1924 (t statistics in Parentheses)

NON-SMOKERS

	Region			Occupation	
	South	Northeast	North Central	Activity Index	Risk Index
1870-74	.144	.2009	.2635	.0730	-.0384
[N=66, Censored=1]	(.41)	(.57)	(.63)	(.23)	(-0.2)
1875-79	-.3164	.0526	.1265	.0566	-.0724
[N=240, Censored=4]	(-1.6)	(.28)	(.63)	(.47)	(-1.0)
1880-84	.0299	.0472	-.0122	-.1390	.0287
[N=597, Censored=33]	(.25)	(.39)	(-.09)	(-2.0)	(.72)
1885-89	.0399	.0024	.0403	.0270	.0301
[N=4412, Censored=607]	(.80)	(.05)	(.81)	(-1.4)	(3.0)
1890-94	.0924	.1415	.0587	-.0050	-.0004
[N=13059, Censored=3733]	(3.1)	(4.7)	(2.0)	(-.25)	(-.04)
1895-99	.0690	.0560	.0723	-.0333	.0198
[N=14031, Censored=5974]	(1.7)	(1.9)	(2.4)	(-1.7)	(2.0)
1900-04	.1965	.2095	.1643	.0583	-.0037
[N=1145, Censored=682]	(1.4)	(1.5)	(1.2)	(.83)	(-.09)
1905-09	-.2250	-.3697	-.1384	.1200	-.1538
[N=306, Censored=230]	(-.78)	(-1.1)	(-.42)	(.71)	(-2.2)
1910-14	.0532	.5776	.5970	-.0093	.0643
[N=762, Censored=654]	(.20)	(2.1)	(2.1)	(-.07)	(1.3)
1915-19	-.0037	.0360	-.2977	-.1123	.0122
[N=1774, Censored=1592]	(-.02)	(.17)	(1.4)	(-1.1)	(.24)
1920-24	.2201	.5445	.1090	.0174	-.0896
[N=895, Censored=796]	(.76)	(2.0)	(.34)	(.11)	(-1.3)

Table 4.13 Cox Models with Dorn Data (Five-Year Cohorts) From 1870 To 1924 (t statistics in Parentheses)

LIGHT-SMOKERS

	Smoking	Region			Occupation	
	Proportion of Years Used Tobacco Occasionally	South	Northeast	North Central	Activity Index	Risk Index
1870-74	.4493	.2296	.0899	.0291	-.0662	.0115
[N=87,Censored=2]	(.83)	(.72)	(.29)	(.08)	(-.23)	(.08)
1875-79	-.3746	-.3231	-.0764	.1336	-.0281	-.0511
[N=303, Censored=8]	(-1.2)	(-1.8)	(-.42)	(.74)	(-.28)	(-.85)
1880-84	.4543	.0108	.0434	-.0369	-.1047	.0082
[N=767, Censored=35]	(2.8)	(1.0)	(.43)	(-.31)	(-1.7)	(.21)
1885-89	.0740	.0429	.0042	.0296	-.0354	.0740
[N=5760,Censored=798]	(1.2)	(1.1)	(.11)	(.74)	(-1.8)	(1.2)
1890-94	.1279	.0917	.1337	.0441	-.0147	.0099
[N=17274, Censored=4867]	(4.3)	(3.1)	(4.5)	(1.5)	(-1.5)	(.99)
1895-99	.0692	.0720	.0414	.0423	-.0218	.0224
[N=18589, Censored=7974]	(1.7)	(2.4)	(1.4)	(1.4)	(-2.2)	(2.2)

Table 4.13 Cox Models with Dorn Data (Five-Year Cohorts) From 1870 To 1924 (t statistics in Parentheses) (Continued)

LIGHT-SMOKERS

	Smoking	Region			Occupation	
	Proportion of Years Used Tobacco Occasionally	South	Northeast	North Central	Activity Index	Risk Index
1900-04	.0394	.0787	.1167	.0421	.0226	-.0263
[N=1621, Censored=974]	(.28)	(.66)	(1.1)	(.35)	(.38)	(-.88)
1905-09	-.2976	-.1761	-.2073	.0242	-.0292	-.0393
[N=432, Censored=331]	(-.73)	(-.677)	(-.72)	(.09)	(-.20)	(-.79)
1910-14	.3945	.1399	.4351	.4461	-.0682	.0405
[N=1095, Censored=934]	(1.3)	(.66)	(2.0)	(1.9)	(-.62)	(1.0)
1915-19	.0271	.0804	.0445	-.3144	-.1911	.0394
[N=2580, Censored=2314]	(.11)	(.54)	(.26)	(-1.7)	(-2.4)	(1.0)
1920-24	-.1748	.1742	.2318	.0165	.1503	-.1234
[N=1286, Censored=1151]	(-.47)	(.73)	(1.0)	(.06)	(1.2)	(-2.1)

Table 4.14 Cox Models with Dorn Data (Five-Year Cohorts) From 1870 To 1924 (t statistics in Parentheses)

HEAVY-SMOKERS

	Smoking	Region			Occupation	
	Proportion of Years Used Tobacco Regularly	South	Northeast	North Central	Activity Index	Risk Index
1870-74	.1289	.1276	.1879	.0205	.0474	-.0414
[N=244, Censored=3]	(.52)	(.64)	(1.0)	(.09)	(.34)	(-.52)
1875-79	.0346	-.1963	-.0490	-.0090	.0172	-.0028
[N=975, Censored=19]	(.29)	(-2.0)	(-.54)	(-.09)	(.29)	(-.09)
1880-84	.3410	.0312	.0143	-.0320	-.0205	-.0037
[N=2732, Censored=95]	(4.9)	(.52)	(.24)	(-.53)	(-.68)	(-.19)
1885-89	.4280	.0885	.0615	.0808	.0034	.0104
[N=19981, Censored=1864]	(21.4)	(4.4)	(3.1)	(4.0)	(.34)	(1.0)
1890-94	.5094	0702	.0451	.0250	-.0032	.0151
[N=61174, Censored=12565]	(50.9)	(3.5)	(4.5)	(2.5)	(-.32)	(3.8)
1895-99	.5594	.0947	.0565	.0534	-.0184	.0187
[N=70423, Censored=23386]	(28.0)	(4.7)	(5.7)	(5.3)	(-1.8)	(4.7)

Table 4.14 Cox Models with Dorn Data (Five-Year Cohorts) From 1870 To 1924 (t statistics in Parentheses) (Continued)

HEAVY-SMOKERS

	Smoking	Region			Occupation	
	Proportion of Years Used Tobacco Regularly	South	Northeast	North Central	Activity Index	Risk Index
1900-04	.8463	.0886	.0732	.0273	.0278	.0127
[N=7777, Censored=3463]	(14.1)	(1.8)	(1.8)	(.55)	(.93)	(1.3)
1905-09	.7424	-.0282	.0950	.0015	.1414	-.0358
[N=1896, Censored=12566]	(5.3)	(-.28)	(.86)	(.01)	(2.4)	(-1.8)
1910-14	.9606	.0250	.1097	.0156	.0047	-.0012
[N=4410, Censored=3448]	(8.0)	(.31)	(1.2)	(.16)	(.12)	(-.06)
1915-19	.9866	.0708	.0316	.0818	-.0356	-.0203
[N=12732, Censored=10703]	(11.0)	(1.2)	(.45)	(1.4)	(-1.2)	(-2.0)
1920-24	.7476	-.0929	-.1701	-.1070	-.0589	-.0167
[N=8277, Censored=7134]	(6.2)	(-1.2)	(-2.1)	(-1.3)	(-1.2)	(-.84)

Table 4.15 Maximum Penalized Likelihood Estimation with Dorn Data From 1870 To 1924 (t statistics in Parentheses)

	Smoking		Region			Occupation	
	Proportion of Years Used Tobacco Occasionally	Proportion of Years Used Tobacco Regularly	South	Northeast	North Central	Activity Index	Risk Index
1. 1870-74	.3435	.4370	.0459	-.0323	.0433	-.0557	.0401
	(.82)	(.87)	(2.3)	(-.80)	(.62)	(-.46)	(.40)
2. 1875-79	.1054	.2543	-.0123	.0524	.0143	-.0984	.0213
	(.88)	(1.3)	(-.62)	(1.3)	(.72)	(-1.6)	(2.4)
3. 1880-84	.2134	.5866	.0034	.0213	.0093	-.1003	-.0002
	(2.1)	(19.6)	(.17)	(.71)	(.47)	(-1.7)	(-.01)
4. 1885-89	.2481	.6031	.0073	.0084	.0105	-.0982	.0325
	(3.1)	(20.1)	(.91)	(1.4)	(1.5)	(-2.5)	(4.6)
5. 1890-94	.2200	.6321	.0104	.0099	.0066	-.0883	.0235
	(3.7)	(31.6)	(1.5)	(5.0)	(6.6)	(-4.4)	(11.7)
6. 1895-99	.2611	.6999	.0189	.0119	.0154	-.0829	.0589
	(4.4)	(35.0)	(2.7)	(11.9)	(15.4)	(-4.1)	(58.9)

Table 4.15 Maximum Penalized Likelihood Estimation with Dorn Data From 1870 To 1924 (t statistics in Parentheses) (Continued)

	Smoking		Region			Occupation	
	Proportion of Years Used Tobacco Occasionally	Proportion of Years Used Tobacco Regularly	South	Northeast	North Central	Activity Index	Risk Index
7. 1900-04	.1873	.7321	.0178	.0123	.0079	-.0421	.0232
	(3.1)	(36.6)	(2.0)	(1.5)	(1.3)	(-2.1)	(77.3)
8. 1905-09	.1002	.7231	.0395	.0233	.0128	-.0024	.0021
	(1.3)	(12.1)	(2.0)	(1.2)	(.64)	(-.03)	(.11)
9. 1910-14	.1824	.7421	.0335	.0212	.0131	-.0175	.0312
	(2.3)	(12.4)	(1.7)	(1.1)	(.66)	(-.44)	(3.1)
10. 1915-19	.2110	.8002	.0232	.0176	.0100	-.0182	.0271
	(4.2)	(80.0)	(2.3)	(2.09)	(1.1)	(-1.8)	(3.4)
11. 1920-24	.2088	.7832	.0088	.0100	.0083	-.0266	.0099
	(3.0)	(39.2)	(.88)	(1.0)	(.83)	(-1.3)	(.99)

Table 4.16 Maximum Penalized Likelihood Estimation With Dorn Data From 1870 To 1924 (t statistics in Parentheses)

NON-SMOKERS

	Region			Occupation	
	South	Northeast	North Central	Activity Index	Risk Index
1. 1870-74	.1200	.2720	.0832	.0773	-.0690
	(.24)	(.76)	(.31)	(.30)	(-.29)
2. 1875-79	-.0378	.0722	.0744	-.0376	.0300
	(-1.3)	(1.2)	(1.2)	(-.54)	(1.5)
3. 1880-84	.0039	.0254	.0103	-.1121	.0017
	(.20)	(.85)	(.52)	(-1.4)	(.17)
4. 1885-89	.0081	.0096	.0128	-.1200	.0608
	(1.0)	(1.4)	(1.6)	(-2.4)	(6.1)
5. 1890-94	.0132	.0122	.0086	-.0923	.0213
	(1.9)	(1.7)	(1.2)	(-9.2)	(10.7)
6. 1895-99	.0218	.0200	.0187	-.0843	.0678
	(3.1)	(3.3)	(3.1)	(-4.2)	(67.8)
7. 1900-04	.0173	.0121	.0067	-.0323	.0399
	(1.7)	(1.3)	(1.1)	(-1.6)	(4.0)
8. 1905-09	-.0218	.0388	.0225	-.0021	-.0033
	(-.36)	(.97)	(.45)	(-.11)	(-.08)
9. 1910-14	.0586	.0632	.0432	-.0301	.0481
	(1.5)	(2.1)	(1.4)	(-.75)	(1.6)
10. 1915-19	.0332	.0212	.0192	-.0201	-.0310
	(1.7)	(2.1)	(1.9)	(-1.0)	(-1.0)
11. 1920-24	.0102	.0133	.0103	-.0123	-.0065
	(1.0)	(.67)	(.52)	(-.62)	(-.33)

Table 4.17 Maximum Penalized Likelihood Estimation With Dorn Data 1870 To 1924 (t statistics in Parentheses)

LIGHT-SMOKERS

	Smoking	Region			Occupation	
	Proportion of Years Used Tobacco Occasionally	South	Northeast	North Central	Activity Index	Risk Index
1. 1870-74	.4280 (.63)	.1004 (.50)	.0423 (.30)	-.1189 (-.48)	-.0058 (-.04)	.1023 (1.0)
2. 1875-79	.0868 (.33)	-.1343 (-.42)	.0087 (.11)	.0175 (.15)	-.0012 (-.01)	.0757 (.51)
3. 1880-84	.3782 (3.4)	.0534 (1.1)	.0468 (1.2)	.0288 (.72)	-.0554 (-2.8)	-.0012 (-.12)
4. 1885-89	.2103 (2.3)	.0082 (1.0)	.0101 (1.1)	.0117 (1.5)	-.0911 (-2.3)	.0434 (4.3)
5. 1890-94	.2356 (3.4)	.0119 (2.0)	.0100 (2.0)	.0107 (1.8)	-.0890 (-2.2)	.032 (16.1)
6. 1895-99	.2103 (5.3)	.0136 (2.3)	.0176 (2.9)	.0096 (2.4)	-.0643 (-2.1)	.0854 (14.2)

Table 4.17 Maximum Penalized Likelihood Estimation With Dorn Data 1870 To 1924 (t statistics in Parentheses)

LIGHT-SMOKERS

	Smoking	Region			Occupation	
	Proportion of Years Used Tobacco Occasionally	South	Northeast	North Central	Activity Index	Risk Index
7. 1900-04	.1102	.0099	.0087	.0056	-.0305	.0250
	(2.2)	(.50)	(.87)	(.70)	(-1.0)	(1.3)
8. 1905-09	.0984	.0108	.0398	-.0039	-.0095	.0012
	(1.1)	(.27)	(.80)	(-.08)	(-.11)	(.06)
9. 1910-14	.1223	.0262	.0279	.0378	-.0301	.0332
	(1.4)	(1.3)	(1.4)	(.95)	(-.60)	(1.7)
10. 1915-19	.2203	.0310	.0198	.0123	-.0210	.0339
	(5.5)	(3.1)	(2.2)	(1.8)	(-3.5)	(17.0)
11. 1920-24	.1854	.0063	.0074	.0063	-.0123	.0109
	(.10)	(.63)	(.74)	(.63)	(-.41)	(1.1)

Table 4.18 Maximum Penalized Likelihood Estimation With Dorn Data From 1870 To 1924 (t statistics in Parentheses)

	HEAVY-SMOKERS					
	Smoking	Region			Occupation	
	Proportion of Years Used Tobacco Regularly	South	Northeast	North Central	Activity Index	Risk Index
1. 1870-74	.4653 (.91)	.0535 (1.8)	-.0266 (-.89)	.0575 (1.2)	-.0654 (-.65)	-.0097 (-.07)
2. 1875-79	.2913 (1.5)	-.0124 (-.41)	.0176 (.59)	-.0025 (-.08)	-.0766 (-1.3)	.0200 (1.0)
3. 1880-84	.5919 (19.7)	.0048 (.24)	.0242 (.81)	.0078 (.39)	-.1018 (-1.5)	-.0011 (-.06)
4. 1885-89	.6203 (20.7)	.0081 (.81)	.0089 (1.4)	.0100 (1.3)	-.0722 (-1.8)	.0299 (3.7)
5. 1890-94	.6757 (33.8)	.0112 (1.4)	.0102 (1.7)	.0078 (2.0)	-.0612 (-3.1)	.0232 (11.6)
6. 1895-99	.7066 (35.3)	.0195 (2.8)	.0121 (6.1)	.0149 (14.9)	-.0830 (-4.2)	.0600 (30.0)

Table 4.18 Maximum Penalized Likelihood Estimation With Dorn Data From 1870 To 1924 (t statistics in Parentheses) (Continued)

	HEAVY-SMOKERS					
	Smoking	Region			Occupation	
	Proportion of Years Used Tobacco Regularly	South	Northeast	North Central	Activity Index	Risk Index
7. 1900-04	.7588 (37.9)	.0191 (2.1)	.0133 (1.5)	.0102 (1.7)	-.0407 (-2.0)	.0242 (8.1)
8. 1905-09	.7497 (12.5)	.0402 (2.0)	.0243 (1.2)	.0128 (.64)	-.0026 (-.04)	.0023 (.12)
9. 1910-14	.8015 (13.4)	.0337 (1.7)	.0236 (1.2)	.0141 (.71)	-.0177 (-.44)	.0333 (3.3)
10. 1915-19	.8211 (82.1)	.0230 (2.3)	.0178 (1.8)	.0111 (1.1)	-.0168 (-1.7)	.0283 (3.5)
11. 1920-24	.7623 (38.1)	.0087 (.44)	.0100 (1.0)	.0093 (.93)	-.0213 (-1.1)	.0112 (1.1)

Table 4.19 Cox Models from 1964-1980 (t statistics in Parentheses)

	Smoking		Region			Occupation	
	Proportion of Years Used Tobacco Occasionally	Proportion of Years Used Tobacco Regularly	South	Northeast	North Central	Activity Index	Risk Index
1870-74	.2833	.0706	.1399	.1483	-.0225	.0125	-.0267
[N=265, Censored=4]	(.56)	(.29)	(.74)	(.78)	(-.11)	(.10)	(-.33)
1875-79	-.9481	-.2679	-.1811	-.1210	-.0063	-.1834	-.0062
[N=376, Censored=23]	(-1.8)	(-1.3)	(-1.1)	(-.76)	(-.04)	(-1.7)	(-.12)
1880-84	.6292	.3705	-.0081	.0437	-.1109	-.0103	-.0152
[N=1429, Censored=97]	(2.9)	(3.7)	(-.10)	(.55)	(-1.4)	(-.21)	(-.76)
1885-89	.0509	.3686	.1277	.0724	.1027	.0122	.0163
[N=13729, Censored=2055]	(.72)	(12.3)	(4.3)	(2.4)	(3.4)	(1.2)	(1.6)
1890-94	.1488	.4699	.0773	.0447	.0214	.0030	.0200
[N=49069, Censored=13699]	(3.7)	(23.5)	(3.9)	(2.2)	(1.1)	(.30)	(5.0)
1895-99	.0946	.5359	.0843	.0430	.0355	-.0139	.0206
[N=61369, Censored=25386]	(2.4)	(26.8)	(4.2)	(2.2)	(1.7)	(-1.4)	(5.2)

Table 4.19 Cox Models from 1964-1980 (t statistics in Parentheses) (Continued)

	Smoking		Region			Occupation	
	Proportion of Years Used Tobacco Occasionally	Proportion of Years Used Tobacco Regularly	South	Northeast	North Central	Activity Index	Risk Index
1900-04	.1110	.8164	.0346	.0637	.0048	.0321	.0192
[N=7139, Censored=3755]	(.69)	(13.6)	(.69)	(1.3)	(1.0)	(1.1)	(1.9)
1905-09	-.4094	.6658	-.0338	.1421	-.0050	.1662	-.0242
[N=1856, Censored=1357	(-.85)	(4.4)	(-.31)	(1.1)	(-.04)	(2.8)	(-1.2)
1910-14	.7916	.9695	-.0376	.0683	.0194	-.0204	.0050
[N=4554, Censored=3728]	(2.7)	(7.5)	(-.42)	(.68)	(.19)	(-.41)	(.25)
1915-19	.1798	.9633	.1013	.0683	.0793	-.0439	-.0113
[N=13183, Censored=11425]	(.72)	(10.7)	(1.7)	(.98)	(1.1)	(-1.5)	(-1.1)
1920-24	.1304	.8735	-.1185	-.2102	-.0714	-.0181	-.0154
[N=8478, Censored=7489]	(.33)	(6.7)	(-1.5)	(-2.3)	(-.79)	(-.36)	(-.77)

Figure 4.1

Dorn Sample
All Ages
Proportional Hazard

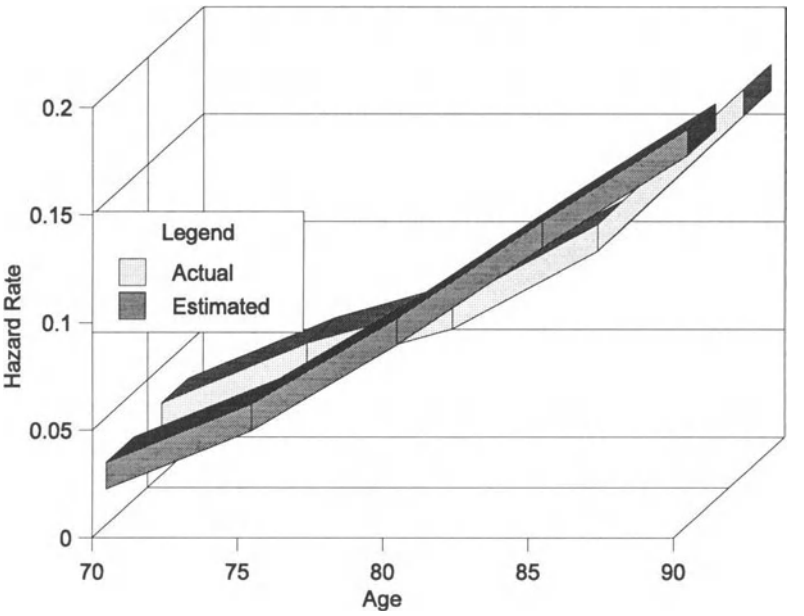
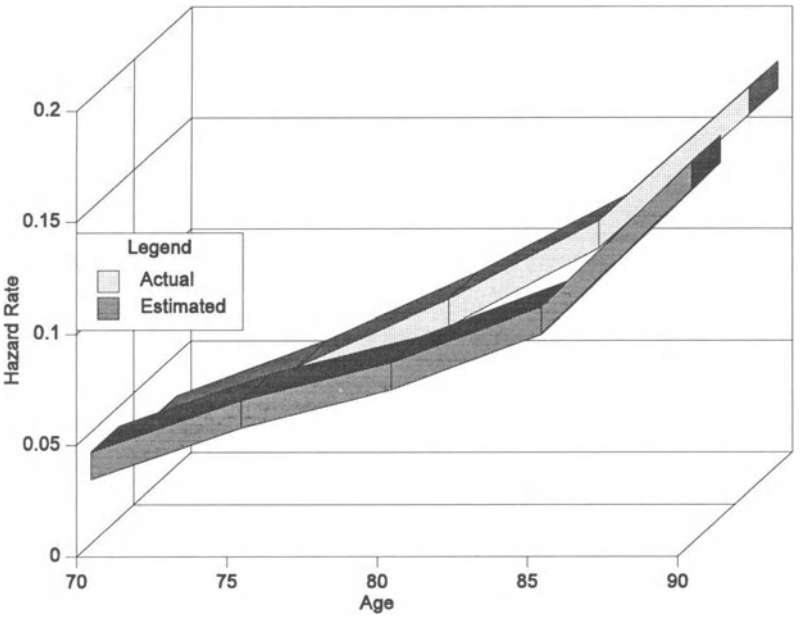


Figure 4.2

Dorn Sample,
All Ages, Hazard Functions,
Time-to-Failure Gamma Function



5

Mortality Hazard Estimates From the Retirement History Survey: Education, Pensions and Marital Status and Black-White and Gender Differences

In this chapter we continue our estimation of adult mortality hazard functions. Here we use the Retirement History Survey (RHS), which covers heads of households aged 58-63 in 1969 and for whom death records are taken from Social Security records for the period 1969-1979 (though the data for 1978 and 1979 are incomplete because of late posting of the death data).[31] Section 2.2 provides more detail about this data source. As discussed in Section 2.3, we use these data to estimate mortality hazard functions that can be interpreted as conditional demand functions or dynamic decision rules in which the right-side variables reflect decisions and the state of the world up to the time of the data collection.[32]

The RHS has both shortcomings and advantages for this analysis in comparison with the Dorn sample that we analyze in Chapter 4. Among the relative shortcomings, the RHS sample is much smaller and does not include nearly as wide range of ages, so it does not permit cohort analysis of the types that we present in Sections 4.3 and 4.4, nor some of the other data-intensive sensitivity analysis that we undertake for the Dorn sample. Because the RHS is a random sample of 58 to 63 year old heads of household in 1969, we also may face the same type of left-censoring problem suggested by the Dorn sample results in Section 4.6. That is, the RHS may not be a random sample of the 1906-1911 birth cohort and previous deaths may be correlated with some of our independent variables. Further right censoring is much more important for the RHS — almost 80 percent — than for the Dorn sample. Among the relative advantages of the RHS is that it is more recent and therefore of more interest for current problems, includes some interesting variables that are not in the Dorn sample (e.g., education, marital status, spouse work status, number of dependents, Social Security benefits,

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[31] Data on deaths through 1981 have been collected, but the Census Bureau has refused to link the new information to the RHS because of legal constraints.

[32] Also see Section 2.3 for discussion of possible estimation problems.

Supplemental Security Income, pension income), and includes both whites and blacks and males and females — which permits some interesting comparisons by race and gender below. Like the Dorn sample, the matching of the RHS data with death records is very good.[33]

We begin this chapter with estimation of mortality hazard functions for men, then exploration of black-white differences for men, and finally consider estimates for women.

5.1. Mortality Hazard Function Results for Men

Table 5.1 contains proportional hazard model estimates for all men in the RHS for the period 1969-1977. If the spouse works, one has a noticeably higher chance of dying. Presumably this does *not* reflect the benefits of higher family income. It is possible that a wife at work cannot urge a husband to see a doctor, keep him out of bars and away from unhealthy consumption, or provide regular meals. However, it is also possible that having a chronically-ill husband induces a wife to work, thereby biasing this coefficient due to reverse causality. Thus, we also present equations omitting this variable. The changes in the other coefficients in some cases differ a fair amount with and without the working spouse variable. For example, changes in absolute magnitude of more than 20 percent result in the coefficient estimates for being black, married, a widower, number of dependents, and longest occupation professional.

In column 1, which includes the spouse working variable, blacks have a 20 percent (statistically significant) higher risk of dying. This type of result is a common finding for blacks except among the very old. With this specification race enters into the hazard only in an additive form. In Section 5.2, as noted, we allow possible black-white differentials in all the coefficient estimates.

The marital status variables indicate that married and divorced/separated men have lower hazards than single males, while widowers have higher rates. The married differential is the largest (in absolute value) and suggests that those who are never married (the omitted category) are about 2.5 times more at risk of dying than those who are married. This relative risk factor may be important because men who are chronically ill at younger ages don't marry or because a wife contributes to a husband's health stock by encouraging him to see a doctor, urging him to smoke less, increasing family income, and providing nursing care. Note that the married effect is closer to zero when spouse working is omitted as in column 2, which is consistent with all the above explanations.

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[33] For the time period covered by the RHS Social Security death records are nearly 100 percent accurate (Duleep 1986). Examination of the more limited data on death provided primarily by widows in the RHS provides few contradictions with Social Security death records.

The increased hazard for widowers is also consistent with the above explanations since the deceased wife cannot perform the nagging and nursing functions though lost income may be made up by life insurance.[34] In addition, grief may play a role.

The more unusual result is the strong decrease in the hazard for divorced and separated men. In morbidity studies, such as Rosen and Taubman (1979), this group is not better off than single men. These men no longer get benefits from a spouse. Of course, they had not been precluded from marrying originally because of a chronic health problem, though they may not have remarried because of more recent diseases. However, health is generally thought of as a stock, which could have increased enough previously while married that on average a divorced man's stock in 1969 was above that of single men.

Education has an estimated negative effect on the mortality hazard. But this effect is very imprecisely estimated and *not* significantly nonzero even at the 25 percent level. Since both education and occupation are included in this analysis, education may not be significant because its effects are operating through occupation.

Perhaps what is most interesting is that income is strongly inversely associated with mortality. An additional $1,000 of Social Security or pension benefits lowers the hazard by 10 to 20 percent. Social Security benefits are a function of an average, generally calculated over 20-25 years of monthly earnings. As shown in Chapter 6, the Social Security's transformation of average earnings is progressive. Pension benefits are a function of the type of plan used. In "defined contribution" plans, payments are based on both lifetime contributions, which are tied to wages in each year accumulated and the pension's earnings; "defined benefit" plans, however, are often based on earnings in the last one to five years.[35] Although it is possible that chronic health problems affect earnings throughout a person's lifetime, it is difficult to believe that our findings on the two-income sources represent the common effect of poor health on income given the huge differences in the way earnings are translated into Social Security and pension benefits. Supplemental Security Income has an estimated impact that is about five times as large as that of Social Security benefits. This may reflect that Supplemental Security Income goes to the poor.

The estimates for the coefficients of dummy variables for the longest occupation being either professional or management all are negative.[36] But all of these are fairly imprecisely estimated with only that for professional in column one being significantly nonzero even at the 10 percent level.

Finally, one is about ten percent more likely to die if one has an additional current dependent. This may reflect stress or the reduction in income available for one's own health needs. Perhaps such dependents require time and emotional resources that could be used to lengthen the husband's life. Alternatively, "dependents" may be more likely

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[34] However, this is unlikely since for many wives life insurance is not sufficient to cover the costs of replacing their services. In 1985 the average amount of adult women life insurance was $21,000 (American Council of Life Insurance, 1986).

[35] Defined benefit pensions are either proportional to the earnings base used or are progressive when a company integrates its pension plan with Social Security benefits (as many do).

[36] We omit the physical activity transformation which is highly collinear with the dummies used.

to stay at home to provide care for a sick parent, and this morbidity experience leads to a higher age-specific death rate.

In Table 5.2 we examine the accelerated-time-to-failure specifications under the same four alternative distributional assumptions explored in Section 4.3. Again, as in Table 5.1, estimates with and without the working spouse variable are included. A working spouse has a negative effect on the age at death of 3 percent with the gamma distribution, with estimates of up to 5 percent in the other specifications. This variable is statistically significant for all four distributional assumptions. As for the estimates in Table 5.1, some of the estimates change 20 percent or more depending on whether or not working spouse is included.

The gamma function fits the data best, but the coefficients are robust to functional form differences. The Weibull model in Table 5.2 yields coefficients, adjusted for sign and division by its scale coefficient, similar to those in Table 5.1 as in theory it is supposed to. Since the coefficient estimates are similar using the various functional forms in Table 5.2 and since the Weibull in Table 5.2 is similar to the corresponding equation in 5.1 (with scaling and sign reversal), the comments made above about Table 5.1 generally apply. However, the longest occupation having been professional or management in Table 5.2 tend to be statistically significant with increases in life span of about 0.7 percent, though they are not significant in the proportional hazard models in Table 5.1.

In Figure 5.1, we present the actual and estimated hazards using the gamma distribution.[37] The model fits the data very closely with an R^2 of 0.981.

Thus far we have shown that even over a period as short as 10 years we find a number of strong associations of age differences in death rates with observed variables for men using either proportional hazard functions or accelerated-time-to-failure. Our results seem to be fairly robust to functional form and statistical assumptions even though a large proportion of the initial sample is still alive (censored) by the end of the sample period. In the next section, we try to determine how well these variables can account for the observed differences in black/white differences. We remind the reader that the Dorn sample considered in Chapter 4 can not be used for this purpose since race is not included in the data set and few blacks served in the army in between World Wars I and II.

5.2. Black-White Mortality Inequalities for Males

Racial inequality, particularly between blacks and whites, has long been of major concern in the United States. This inequality may take a number of forms such as with regard to schooling, housing, health, employment options, and income.[38] The National Academy of Science report on *Blacks and American Society* edited by Jaynes and

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[37] Figure 5.2 displays the observed and estimated hazards for the proportional hazard model. The fit is good with an $R^2 = 0.88$.

[38] For example, see Shulman (1987), Kahn and Sherer (1988) Andrisani (1977), Welch (1973), Smith (1984), Orazem (1987), Darity (1982), Ashenfelter (1977), Freeman (1973), Smith and Welch (1977), and Welch (1974).

Williams (1989) reviews the recent status of black Americans. Jaynes and Williams (p. 6) summarize their "main findings on the status of blacks in America in the late 1980's succinctly:

- By almost all aggregate statistical measures — incomes and living standards; health and life expectancy; educational, occupational, and residential opportunities; political and social participation — the well-being of both blacks and whites has advanced greatly over the past five decades.
- By almost all the same indicators, blacks remain substantially behind whites."

Economists concerned with racial inequalities in the United States have concentrated on the nature of such inequalities in income, though they also have examined other differences. A major focus has been to try to understand to what extent such inequalities have been due to average differences in observed characteristics thought to underlie an outcome (e.g., in the schooling underlying wage rates) and to what extent they are due to differences in the effects of those characteristics. Or, to put the question slightly differently, how much of the existing black-white differences in an outcome of interest would disappear if blacks had the same observed characteristics as do whites?

Throughout much of their lifespan, blacks have higher age-specific death rates than whites in the United States (though there may be a cross-over at later ages). For example, the annual death rate for males aged 50 in 1960 was about 9.5 and 15.6 per 1000 for whites and blacks respectively. Kitagawa and Hauser (1973, p. 103) indicate that at about the same time, the remaining life expectancy at age 55 for white and black males was 19.5 and 18.4 years respectively. Jaynes and Williams (1989, p. 427) report that between 1900 and 1984 the expected remaining years of life at age 65 increased from 11.5 to 14.8 for white men and from 10.4 to 13.4 for black men. Also, as shown in Figure 5.2 and Table 5.3, the death hazard rate is much higher for blacks than whites in the years covered in the RHS.

Over the life cycle, whites and blacks face substantially different environments and have major differences in education, earnings, occupation and marital status, all of which variables have been found to be related to morbidity and mortality in studies by Behrman, Sickles and Taubman (1988), Sickles and Taubman (1986), Rosen and Taubman (1982), Kitagawa and Hauser (1973), Madans et al. (1986b), and Sorlie and Rogot (1990). While such observed characteristics may account substantially for black-white mortality differentials, there also may be major causes that are not observed in most socioeconomic data sets. Jaynes and Williams (1989, p. 425), for example, suggest that such factors may be quite important: "Black adults reach age 65 with life histories of disproportionate prevalence of acute and chronic disease, illness, and disability. They have had poorer quality of health care from conception and birth, continuing exposure to greater and more severe environmental risk factors, and the stress of prejudice and discrimination (Cooper, et al., 1981). Cohort data for cause-specific mortality and morbidity over the past four decades suggest the presence of accumulated deficits across the early years of the life course. These deficits place black older people at greater risk for morbidity and mortality than whites of comparable ages."

We first consider mortality hazard data for black males and for white males, smoothed versions of which are given in Figure 5.2 and Table 5.3.[39] It is evident that the hazard is higher for blacks at every age covered. Thus, in our limited age range, there is no cross-over in hazard rates for blacks and whites.

Our estimated conditional mortality demand relations for blacks and whites are presented in Tables 5.4 and 5.5. For each racial group, we have carried out the analysis with and without corrections for unobserved heterogeneity.

In columns 1 and 2 of Table 5.4, we present the results for black men assuming a Weibull and loglogistic accelerated-time-to-failure model. The estimates yield coefficients comparable to the previous estimates in the proportional hazard models. The statistically significant variables are for marital status and pension income. The marital status estimates indicate that those who are married or divorced/separated have significantly less probability of dying in the given age range than those males who are never married or who are widowed. Again the pension effect presumably indicates the advantages of higher income or related characteristics rather than occupation per se since longest occupation is included as two dichotomous variables for professional and management. Yet in some instances group health insurance is provided to retired employees, which may be correlated with occupation. However, Social Security benefits, Supplemental Security Income, education, occupation, and number of children are not statistically significant. We have reestimated the equations and included a self-assessed measure of health as of 1968, a variable that is highly correlated with physician evaluations in this age range. The coefficients (not shown), including those for marital status, are nearly the same as those shown in Table 5.4.

Columns 3-7 present various proportional hazard estimates with and without treatments for heterogeneity. The numerical results are very similar to those in columns 1 and 2. Coefficient estimates are very robust though significance levels change and the duration estimate is insignificant using the Nonparametric Maximum Likelihood Estimator (NPMLE). The estimates with no heterogeneity and a loglogistic accelerated hazard fit the data best in terms of the maximized value of the log likelihood function. We again find strong negative associations of the hazard with pension income and of being married or divorced/separate. These associations again persist even if we control for self-assessed health status (in results not shown).

Table 5.5 contains the corresponding results for white males. The greater significance levels than in Table 5.4 partly reflect the approximate tenfold greater sample size. Coefficient estimates again differ little across the various models. As was found with black men, the introduction of non-parametric or parametric heterogeneity yields a small improvement in fit, similar parameter estimates, and changed significance levels (smaller in column 4). For the estimates for white men, all three marital categories have highly significant coefficient estimates with widowers having an increased hazard relative to being never married. Increased pension income significantly reduces the hazard. Other variables tend to have expected signs, but are not statistically significant at conventional levels. Comparison of Tables 5.4 and 5.5 indicates that the coefficients significant in both tables are usually larger in absolute

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[39] The figure is limited to people aged 60 through 66 (even though our data include ages 58-73) in part because of the smaller sample sizes for other ages (arising from the age and panel structure of the RHS) and in part because of the incomplete information on death after 1977.

value for blacks than for whites though the coefficients for being a widower are larger for whites than for blacks (and only significant for the former). The duration estimates, however, are similar for the two groups.

We have examined several specifications (not presented) in which data from the two racial groups are pooled. These models were estimated using the Cox partial likelihood, the Weibull accelerated hazard, and the NPMLE model. We examine models in which selected regressors, including the constant term, are allowed to differ between the two groups, although we have not considered a model in which race interacted with all covariates due to computational constraints. The coefficients typically lie between the estimates for blacks and whites. In all pooled models, however, we reject the null hypothesis that the coefficients are the same for blacks and whites at the 99 percent level.

Table 5.6 uses the white Cox partial likelihood estimates from Table 5.5 to assess the association of the racial differences in the means with the hazard rate differentials. We use the white rather than the black hazard because the former is more precisely estimated with its ten times larger sample. The big differences in Table 5.6 come from marital status, pension income, and education (whose coefficients are not statistically significant and whose linear and square terms largely offset each other). Overall the white hazard would be about 19 percent higher if whites had blacks' observed characteristics. Approximately the same results would be found in the other proportional hazards given the robustness of the coefficients.[40]

The actual differences in the hazards are given in Table 5.6 for ages 60-66. The differentials range from 35 to 15 percent with some instability arising from small subsamples, especially for blacks. The average differential is about 34 percent. The differential in the median time period is about 25 percent. Differences in the observed characteristics are associated with between 60 and 80 percent of the difference in the observed hazard rates.

In this section we have explored inequalities in mortality between black and white older adult males in the United States. We have estimated hazard functions separately for blacks and whites. The equations have different coefficients by race. Within a race, the equations are robust to changes in specification including allowance for heterogeneity. Replacing whites' means by blacks' means in the proportional hazard for whites would raise the white hazard rate by about 19 percent, a noticeable amount that is consistent with most of the inequalities in the observed mortality hazards. Such observed characteristics — particularly those related to marital status, pension income, and education — thus capture most of the black-white mortality differences among older men. The factors emphasized by Jaynes and Williams (1989, p. 425) in the quotation given earlier — including poorer quality health care, greater exposure to environmental risk factors and the stress of prejudice and discrimination — if important, apparently largely work through these observed characteristics. If there were movements towards convergence in regard to such observed socioeconomic characteristics and their covariates, therefore, there probably would be a reduction in older adult male black-white mortality rate inequalities.

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[40] However, the white hazard would be about 11 percent higher if whites had blacks' observed characteristics rather than the 18 percent figure when differences in pension income are based on the 1975 figures.

5.3. Mortality Hazard Model for Women

The RHS also sampled female heads of household, and the Social Security files also contain information on their dates of death. While some data on wives of male respondents were collected, Social Security numbers were not obtained. Hence, date of death is not usually known. Since the Census definition generally defines the head of the household as male if there is a couple, there are only about 35 percent as many female household heads as male household heads in the RHS (about 2700 females versus about 7900). In addition women have several years longer expected remaining life span at about age 60. During this period 338 females, 12 percent, died. These factors tend to diminish the significance of the coefficients that we estimate. They also imply that our data on females are not necessarily representative of all females of the age range included even in the initial year of the survey since females who are household heads may be a selected set of females.

Table 5.7 gives summary statistics in 1969 for female heads of households. In comparison with male heads of households (see Table 3.1), the racial composition is about the same, mean schooling is slightly greater, current or past marriage is much less likely (only 3.5 percent of these women were currently married and 36 percent were never married), having dependents is less than a fifth as common, longest occupation being professional is somewhat less common and being a manager is only about a third as common, and mean Social Security benefits are only a sixth and mean pension income is only two fifths, but mean Supplementary Security Income is almost four times as large (though much smaller than mean Social Security benefits and pension income).

Since our examination of men in the RHS indicates that our results are fairly stable to functional form and heterogeneity specifications, we restrict our analysis here to the Cox proportional hazard model. Table 5.8 contains our estimates for all women who were heads of household. We only allow for black-white differences by an additive dummy as in Section 5.1 (where one equals being black) because of the much smaller sample size for women.

Despite the smaller sample size, we find that a number of variables have large and statistically significant estimates. All the included marital status variables (never married is excluded) have coefficients of about -2.3. Black women have a hazard rate about 80 percent more than non-black females. Social Security benefits, Supplemental Security Income and pension income all are associated with lower hazard rates, though for the last of these the point estimate is very imprecise. The pension elasticity is about 20 times as large (in absolute value) as the Social Security benefit elasticity even though we include some control for occupations and for education.[41]

Now we compare the hazard results for those obtained for men in Table 5.1. There are too few married women (because of the sample design) to make a valid comparison for this group. Only 75 of the 2748 women heads of households in our sample are married. However, comparisons in the other marital status groups are appropriate and suggest large differences. Among men the widowers had a 70 percent higher death rate

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[41] We have also run the equation for non-black women. The results are given in Table 5.8A. The marital status and income variables change by about 10 percent e.g., from -2.0 to -2.2, but the patterns and significance levels are similar.

than the never married. Widows, who are about one half of the female-headed households, have a much lower death rate than never married women. The coefficient on the divorced and separated females is -2.5 while in Table 5.1 it is -1.5 for men. It is often argued that currently and formerly married men benefit from the nursing and nagging behavior of their wives. These results suggest that either men don't perform these activities to the same degree (quality) for wives or women are less responsive to such treatment.

The other major difference is the elasticity of the pension income variable, which measures -2.6 for women but -0.12 for men. To get a pension, one (or one's former spouse) would have to have worked, usually for a significant time at least for the people in this sample who had a defined benefit plan. Many women born around 1910 would not have worked long enough to have been vested in such pensions. Also some may not have worked nor married because of poor health. In addition some who qualified for a pension could have had better health insurance before and after retiring though we don't have this information for individuals. Moreover, the extra post retirement income could let them buy better health care. Note also that number of dependent children is not significant here though it is in Table 5.1.

5.4. Conclusion

In this chapter we have used the males and females in the RHS, a national random sample of heads of household in the United States aged 58 to 63 in 1969, to estimate hazard models for dying during the period 1969-1979. We have estimated equations for all male and separately for blacks and whites and for all female heads of household. We find that various measures of income, marital status, occupation, and, for males, number of dependents and wife working are significantly related to mortality. Strikingly, we find no significant associations with education once we control for these other characteristics. We also ask how much higher would the white hazard rate be if their hazard was evaluated at the average characteristics of black men. We find that the increase in the so calculated hazard rate would be much higher and explain more than half of the currently observed differences in the hazard rates. We find many similar results for females though some marital status, presence of dependents, and some income impacts differ.

Table 5.1 RHS Sample for Males: Proportional Hazard Model (Cox's Partial Likelihood)

Variable	Proportional Hazard Estimate (t-statistics)	
	—	—
	(—)	(—)
Spouse working	.960	—
	(16.6)	(—)
Black	.211	.165
	(2.53)	(1.99)
Married	-2.42	-1.74
	(-29.2)	(-24.3)
Widowed	.695	.952
	(11.9)	(17.3)
Divorced/separated	-1.46	-1.46
	(-6.71)	(-6.70)
Education	-.0066	-.0075
	(-.884)	(-1.01)
Social Security benefits (thousands)	-.238	-.229
	(-13.4)	(-13.0)
Supplemental Security Income (thousands)[a]	-1.25	-1.47
	(-4.50)	(-5.14)
Number of dependents	.110	.137
	(3.30)	(4.15)
Pension income (thousands)	-.117	-.128
	(-6.15)	(-6.4)
Longest occupation		
Professional	-.121	-.092
	(-1.69)	(-1.2)
Management	-.0909	-.09
	(-1.27)	(1.35)
LogL	-13533.2	-13663.6

[a]Measured in 1975.

Table 5.2 RHS Sample: Accelerated-Time-to-Failure Models

Variable	Weibull Estimate (t-statistic)		Lognormal Estimate (t-statistic)		Loglogistic Estimate (t-statistic)		Gamma Estimate (t-statistic)	
Intercept	6.75 (1525)	6.74 (1456)	6.72 (1544.7)	6.72 (1522)	6.72 (1497.)	6.72 (1463.)	6.71 (1283.)	6.70 (1344.)
Spouse working	-.0512 (-17.5)	—	-.0345 (-11.6)	—	-.0410 (-13.3)	—	-.0261 (-7.70)	—
Black	-.0112 (-2.69)	-.00930 (-2.14)	-.0124 (-2.98)	-.0114 (-2.69)	-.0133 (-3.09)	-.0124 (-2.80)	-.0118 (-2.91)	-.0102 (-2.58)
Married	.124 (26.8)	.0914 (22.1)	.105 (27.9)	.0837 (26.6)	.112 (26.9)	.0855 (24.4)	.0984 (26.5)	.0829 (29.9)
Widowed	-.0354 (-12.1)	-.0509 (-17.1)	-.0369 (-11.8)	-.0478 (-15.6)	-.0371 (-12.0)	-.0517 (-17.1)	-.0353 (-10.9)	-.0384 (-11.2)
Divorced/ separated	.0742 (6.78)	.0781 (6.79)	.0755 (8.93)	.0765 (8.95)	.0745 (7.96)	.0769 (7.97)	.0766 (9.94)	.0779 (10.9)
Education	.000331 (.895)	.00394 (.985)	.000352 (.951)	.000367 (.973)	.000381 (1.00)	.000435 (1.12)	.00031 (.858)	.000213 (.609)
Social Security benefits (thousands)	.0124 (14.1)	.0127 (13.8)	.0123 (16.0)	.0122 (15.6)	.0132 (15.8)	.0132 (15.2)	.0118 (16.4)	.012 (16.7)
Supplemental Security Income (thousands)	.0652 (4.63)	(.0811) (5.32)	.0559 (5.70)	.0605 (5.98)	.0624 (5.01)	.0715 (5.40)	.0518 (5.95)	.0488 (6.03)
Number of dependents	-.00589 (-3.56)	-.00747 (-4.32)	-.00557 (-2.87)	-.00660 (-3.36)	-.00527 (-2.76)	-.00662 (-3.47)	-.00536 (-2.68)	-.00572 (-2.82)
Pension income (thousands)	.00614 (6.46)	.00720 (6.80)	.00615 (7.16)	.00539 (6.72)	.00639 (6.86)	.00639 (6.51)	.00572 (7.33)	.00491 (7.01)
Longest occupation:								
Professional	.00683 (1.91)	.00536 (1.42)	.00683 (2.01)	.00679 (1.97)	.00731 (1.98)	.00746 (2.05)	.00639 (1.97)	.00611 (1.96)
Management	.00431 (1.12)	.00515 (1.37)	.00781 (2.25)	.00819 (2.32)	.00706 (1.98)	.00753 (2.06)	.00853 (2.54)	.00894 (2.75)
Scale	.0499 (49.7)	.0524 (49.3)	.0697 (53.1)	.0712 (52.9)	.0394 (49.3)	.0407 (49.0)	.0761 (40.3)	.0802 (51.4)
Shape	—	—	—	—	—	—	.466 (4.07)	-.912 (-8.74)
Log L	387.0	239.9	501.24	431.56	455.24	364.64	510.5	475.41

Table 5.3 Black and White Male Hazard Rates

Age Difference	White Hazard Rate	Black Hazard Rate	Percentage
60	.0091	.0133	46.2
61	.0106	.0164	54.7
62	.0150	.0173	15.3
63	.0204	.0254	24.5
64	.0270	.0355	31.5
65	.0321	.0418	30.2
66	.0324	.0443	36.7

Table 5.4 Hazard Estimates for Black Men

Variable	Gompertz Accelerated Hazard (NPMLE)	Weibull Accelerated Hazard Frailty	LogLogistic Proportional Hazard (MPLE)	Cox[a] Proportional Hazard Frailty	Weibull Proportional Hazard Normal	Weibull Proportional Hazard	Proportional Hazard Inverse Gaussian
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Intercept	-0.764	.277	-.456	0.906	0.383	.840	1.54
	(0.85)	(0.41)	(6.11)	(0.20)	(1.21)	(0.92)	(2.83)
Education	-.0727	.0836	-0.068	0.079	0.096	-.138	.0711
	(1.08)	(1.20)	(1.06)	(1.16)	(1.17)	(1.21)	(1.11)
Education^2	0.002	.0023	0.003	0.002	0.003	.0102	.00181
	(0.47)	(0.51)	(0.60)	(0.46)	(0.50)	(0.71)	(0.43)
Married	-2.630	2.725	-2.536	2.796	3.191	2.777	-2.622
	(10.7)	(11.3)	(10.7)	(5.32)	(11.2)	(11.4)	(11.05)
Widowed	0.016	.0517	0.053	0.009	0.007	.0333	.00994
	(0.03)	(0.12)	(0.31)	(0.05)	(0.03)	(0.52)	(0.06)
Divorced/ Separated	-2.556	2.68	-2.491	2.721	3.128	2.718	2.55
	(4.84)	(4.91)	(4.85)	(3.85)	(5.33)	(4.66)	(4.97)
Longest Occupation: Professional	0.356	.287	0.263	0.379	0.400	.377	.344
	(1.47)	(1.25)	(1.02)	(1.32)	(1.16)	(1.36)	(1.32)
Longest Occupation: Management	0.671	.8500	.5850	.702	0.765	.705	.650
	(1.30)	(1.65)	(1.48)	(1.35)	(1.22)	(1.44)	(1.63)

Table 5.4 Hazard Estimates for Black Men (Continued)

Variable	Gompertz Accelerated Hazard (NPMLE)	Weibull Accelerated Hazard Frailty	LogLogistic Proportional Hazard (MPLE)	Cox[a] Proportional Hazard Frailty	Weibull Proportional Hazard Normal	Weibull Proportional Hazard	Proportional Hazard Inverse Gaussian
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Pension Income (1000$)	-0.331 (2.50)	.330 (2.58)	-0.330 (2.53)	0.355 (2.66)	0.394 (3.12)	.348 (2.61)	.327 (2.43)
Expected Social Security Benefits (1000$)	0.020 (0.31)	.0714 (0.28)	-0.008 (0.13)	0.032 (0.60)	.0492 (0.81)	.0610 (0.91)	.0455 (0.78)
Number of Dependent Children	0.028 (0.42)	.0216 (0.35)	0.009 (0.15)	0.012 (0.12)	0.005 (0.04)	.0582 (0.22)	.0250 (0.39)
Supplemental Security Income (1000$)	-0.798 (1.35)	.785 (1.38)	-0.743 (1.28)	0.797 (0.99)	0.803 (0.97)	-.797 (1.38)	.758 (1.29)
ln Duration	.559 (5.07)	.511 (4.60)	.436 (4.56)	.385 (1.32)	.587 (4.51)	.520 (4.11)	.402 (2.97)
lnL	399.40	389.98	400.4	395.83	392.43	397.2	404.8
X	286.2	305.4	287.2	293.7	300.6	296.6	275.4

Sample Size is 692; t-statistics are in parenthesis.

[a]See footnote 5.

Table 5.5 Hazard Estimates for White Men

Variable	Weibull Accelerated Hazard	Loglogistic Accelerated Hazard (NPMLE)	Cox[a] Proportional Hazard Frailty	Gompartz Weibull Proportional Hazard (MPLE)	Weibull Proportional Hazard Normal Frailty	Weibull Proportional Hazard	Proportional Hazard Inverse Gaussian
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Intercept	-2.129	1.63	-1.79	-2.366	-1.979	-1.88	-2.757
	(8.74)	(6.59)	(64.5)	(2.19)	(10.7)	(7.15)	(15.1)
Education	-0.026	-.004	0.045	0.015	0.004	.0221	-.0193
	(0.85)	(0.16)	(1.36)	(0.47)	(0.10)	(0.79)	(0.75)
Education^2	-0.0012	.00031	-0.002	0.0004	0.0001	.000031	.0009
	(0.85)	(0.34)	(0.98)	(0.23)	(0.06)	(0.45)	(0.55)
Married	-1.738	-1.740	-1.687	-1.796	-2.005	-1.872	-1.955
	(23.0)	(22.6)	(21.8)	(16.3)	(22.7)	(20.7)	(19.9)
Widowed	1.228	1.333	1.172	1.350	1.530	1.441	1.395
	(22.4)	(24.0)	(20.0)	(16.6)	(21.1)	(19.3)	(20.2)
Divorced/ Separated	1.241	-1.331	-1.284	-1.297	-1.499	-1.531	-1.426
	(5.20)	(5.47)	(5.35)	(4.94)	(5.45)	(5.21)	(5.05)
Professional	-0.098	-.131	-0.119	-0.107	-0.132	-0.122	-.123
	(1.32)	(1.66)	(1.56)	(1.34)	(1.45)	(1.47)	(1.52)
Management	-0.104	-.141	-0.130	-0.117	-0.153	-.151	-.129
	(1.49)	(1.92)	(1.77)	(1.53)	(1.72)	(1.62)	(1.77)

Table 5.5 Hazard Estimates for White Men (Continued)

Variable	Weibull Accelerated Hazard	Loglogistic Accelerated Hazard (NPMLE)	Cox[a] Proportional Hazard Frailty	Gompartz Weibull Proportional Hazard (MPLE)	Weibull Proportional Hazard Normal Frailty	Weibull Proportional Hazard	Proportional Hazard Inverse Gaussian
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Pension Income (1000$)	-0.186 (7.10)	-.184 (7.12)	-0.179 (7.01)	-0.186 (8.42)	-0.205 (8.6)	-.199 (8.19)	-.201 (7.81)
Expected Social Security Benefits (1000$)	0.028 (1.59)	.0295 (1.62)	-0.016 (0.82)	0.030 (1.64)	0.030 (1.50)	0.0281 (1.49)	.0533 (1.63)
Number of Dependent Children	0.024 (0.42)	-.059 (.93)	0.040 (0.72)	-0.029 (0.46)	-0.046 (0.67)	-0.0441 (0.98)	-.0488 (0.88)
Supplemental Security Income (1000$)	0.099 (0.48)	.466 (0.38)	0.174 (0.84)	0.001 (0.003)	0.037 (0.16)	0.0227 (0.92)	.00202 (0.12)
ln Duration	.552 (15.6)	.543 (14.9)	.435 (8.72)	.359 (5.36)	.532 (12.5)	.521 (11.8)	0.413 (13.2)
lnL	3815.5	3772.5	3820.4	3814.4	3808.0	3813.2	3836.1
χ^2	2214.1	2300.1	2219.0	2216.3	2229.1	2218.8	2173.1

Sample Size is 7223; t-statistics are in parenthesis.

[b]See footnote 5.

Table 5.6 Differences in Racial Means of Selected Variables for Males and Impacts on the Hazard

Variable	Black-White Means	Percentage Effect on Log Hazard[a]
Married in 1969	-.0597	10.1
Widower in 1969	.0296	3.7
Divorced/Separated in 1969	.0571	-7.3
Education	-3.24	-14.6
(Education)^2	-52.5	10.5
Longest Occupation Professional	-.0756	0.9
Longest Occupation Manager	-.151	2.0
Expected Social Security Benefits in 1973	-.279	0.5
Pension Income	-.442	7.9
Dependent Children in 1973	.256	1.0
Supplemental Security Income in 1975	.188	3.3

[a]Using Cox partial likelihood estimates for whites in Table 6.2.

Table 5.7 RHS Survey Means and Standard Deviations in 1969 for Female Heads of Households

	Mean	Standard Deviation
White	0.861	0.346
Married	0.035	0.183
Widowed	0.482	0.499
Divorced/Separated	0.126	0.331
Education	10.10	.362
Social Security benefits (thousands)[a]	0.207	0.439
Supplementary Security Income (thousands)	0.098	0.320
Dependents	0.026	0.113
Pension income[b] (thousands)	0.524	1.701
Professional	0.140	0.347
Management	0.064	0.244
Number of observations	2748	
Percentage censored	87.7	

[a]As of 1969.

[b]Actually received.

Table 5.8 Female Heads of Household Cox Proportional Hazard

Variable	Coefficient	t-statistics
Married	-2.3	-4.0
Widowed	-2.4	-14.8
Divorced/Separated	-2.3	-7.8
Education	-0.0	-0.5
Education^2	0.0	0.2
Professional	-0.4	-1.8
Management	-0.1	-0.3
Pension income	-2.6	-2.7
Social Security benefit	-0.1	-2.1
CSUPC	-0.4	-0.7
Supplemental Security Income	-0.4	-1.2
Log likelihood =		-2350.4

Table 5.8A Cox Models: RHS White Females

Variable	Coefficient	t-statistics
Married	-1.9	-3.3
Widowed	-2.2	-12.1
Divorced/Separated	-2.0	-6.0
Education	-0.1	-1.0
Education^2	0.0	0.7
Professional	-0.4	-1.8
Management	-0.1	-0.3
Pension income	-2.5	-2.6
Social Security benefit	-0.2	-2.5
CSUPC	-0.2	-0.3
Supplemental Security Income	-0.0	-0.1
Log likelihood =		-1797.9

Figure 5.1

Dorn Sample,
All Ages, Hazard Functions,
Time-to-Failure Gamma Function

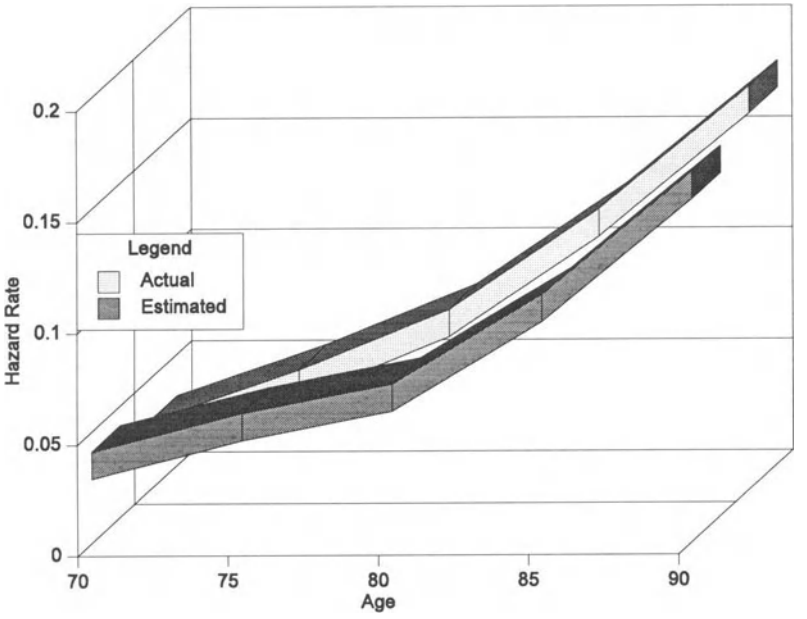
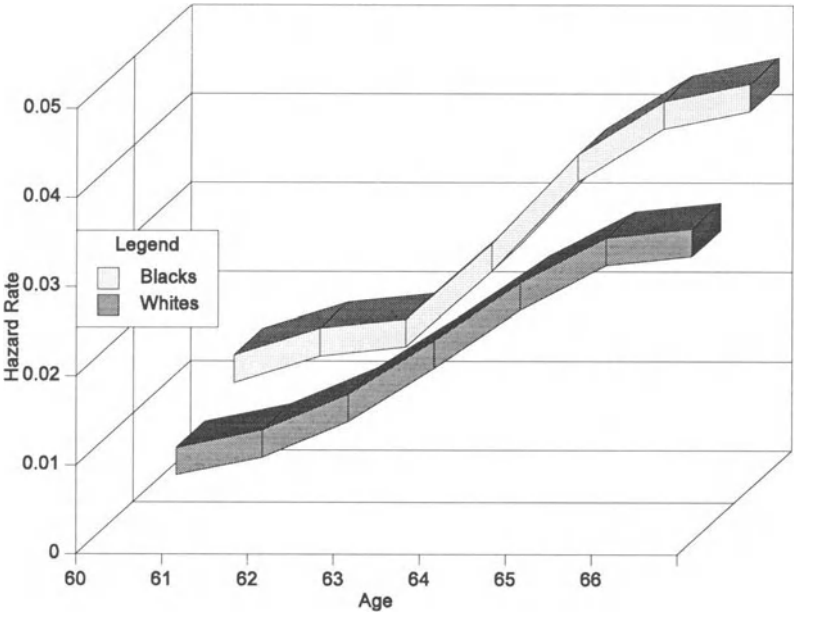


Figure 5.2

Hazard Functions for Blacks and Whites



6

Private Rates of Return on Social Security and Their Relation to Mortality for Groups Defined by Socioeconomic Characteristics

In the previous chapters we have looked for causes of differential death rates among various groups. These differences can have effects on public policy issues. In this chapter we consider the implications of differential death rates on the variation in expected private rates of return on individual and group "investments" in the Social Security System. These investments take the form of Social Security taxes paid (by employer and employee) while the returns take the form of old-age benefits received by the individual and sometimes a current or former spouse. We use the Retirement History Survey again (as in Chapters 2 and 5), which has been matched to Social Security records for each respondent. While the RHS is a random sample of household heads aged 58 to 63 in 1969, it may not be typical of other cohorts because of the rapid increase in the maximum taxable ceiling on covered wages, much of which occurred after 1970. However, the results are indicative of the impact of differential death rates on the rates of return to Social Security.

Social Security benefits are tied to lifetime covered earnings, to length of service, and to marital status. In terms of immediate pre-retirement pay, Social Security benefits average about 57 percent for low-income and 42 percent for middle-income workers (Myers, 1981).

Similarly Shoven, Sundberg, and Bunker (1989) demonstrate that one of the big "taxes" on cigarette smokers is the reduction in their rate of return from the Social Security system. These authors' estimates on the relationship of smoking to death rates are taken from an early study which is based on crude sociodemographic or economic differences. Better estimates would be useful in setting an optimal benefit pay-out schedule and also may affect private choices on smoking. Moreover, estimates by socioeconomic characteristics may result in more informed choices by individuals and life insurance companies on the use and design of insurance policies.

The old-age tax and benefit part of the Social Security System is sometimes thought of as a forced annuity contribution. As with privately purchased annuities, payments,

which begin at a particular age, are guaranteed to individuals and sometimes surviving spouses for their remaining lifetimes.

The Social Security System, however, differs from private annuities,[42] in several important respects. Private annuities generally pay fixed amounts per time period (though some adjust for inflation). Social Security adjusts payments for annual inflation but currently curtails or "taxes" payments a person is entitled to (based on age and average earnings) by 33.3 percent for each dollar of *earnings* above a certain amount which varies with the CPI (Consumer Price Index).[43] Currently this tax is waived if one is at least 70 years old.

Social Security also differs from a private annuity in that an individual's annual benefits are not proportional to taxes (or premiums) that individual paid into the system. Instead Social Security's annual benefits, at least without considering differential life expectancies, are structured to be progressive with successively smaller steps in benefits as average lifetime earnings increase.[44]

Also the Social Security System differs from private annuities in that it provides divorced women, who had been married at least 10 years (after 1977),[45] the right to the higher of either a percentage of the former husband's benefits or benefits based on own earnings. Widows also have been covered by a similar provision since 1956. For both groups the percentage of husband's benefits that can be obtained has varied over time, e.g., 82.5 percent during the period 1956-1971 and (for widows) 100 percent thereafter. A similar provision also applies to currently married couples, both age eligible, except that there is no minimum time to be married and the applicable percentage is 50 percent of the benefits of the higher-paid spouse. Economies of scale provide a rationale for this lower percentage.[46] Below we calculate how important these provisions are for married couples and for female heads of household, who by Census Bureau definition do not include married women with a non-institutionalized husband present. While these provisions are justified on "equity" grounds, they make the system different form a pure forced saving plan.

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[42] Until recently the rate of return on private annuities was small, e.g., a 2 percent nominal rate. More recently, Keogh's, IRA's and supplemental pension plans have been paying rates comparable to those offered on equally risky assets on a pre-tax basis while these annuities allow you to defer taxes. Much more money is invested now in these tax-deferred annuities.

[43] This is called the "earnings tax." The earnings are also subject to Social Security, income, and payroll taxes. Private annuities are not subject to the earnings tax though the interest component is subject to the income tax. In our sample period, the minimum age to waive the earnings tax was 72 and the earnings tax rate was 50 percent for amounts above about $5,000 (with annual variation).

[44] Average lifetime earnings are calculated as the primary insurance amount (PIA). For example, 1990 the formula for PIA was based on a 20 year average monthly covered wage received from 1951-1990, but excluding from the average the years of lowest earnings.

[45] Eligibility required 20 years of marriage beginning in 1956, and there was no eligibility before then. The widow's benefit also began in 1956.

[46] These percentages apply at age 65. Actuarially reduced benefits are given if the woman retires early, which can be as early as 60 for widows and 62 for other women.

Of course, the Social Security tax system in itself is progressive since the tax is only levied on wages and self-employed earnings up to a ceiling. This ceiling has changed over time. For example in the mid 1960's more than 50 percent of white male workers were at the ceiling, while by the early 1990's less than 5 percent of this group were at this maximum.

In this chapter we use available information to estimate in more detail the expected private rates of return to the Social Security retirement pension system for various demographic groups in the RHS. We make calculations that clarify the importance of differential death rates and the provisions for current and former spouse.

6.1. Prior Research on Returns to Social Security System

Prior research by Hurd and Shoven (1985) examined the pattern of estimated rates of return (r) for the Social Security System by assuming that people could be divided into categories by the level of their lifetime income, race, and sex for the purpose to calculating these returns. They used the RHS and the then-available information on death rates by these demographic categories. They found that the differences in death rates were large enough that the rate of return for the various groups was about the same for low and high earnings people despite the progressive benefit and regressive Social Security tax schedule.

Boskin and Puffert (1988) surveyed the existing literature on rates of returns and provide new estimates that incorporate differences in mortality only by age and fertility. They find substantial variation in their estimates of the rate of return by birth cohort and by means of financing. All of the estimates in the literature assume that the incidence of employer Social Security contributions is entirely on employees.

6.2. Estimated Rates of Return to Investments in Social Security

The rate of return on annuity contracts issued by a firm to the survivors each of T periods among N demographic groups is the rate of return r that solves the following equation:

$$(6.1) \qquad \Sigma_{j,t} \frac{(P_{j,t}B_{j,t} - P_{j,t}C_{j,t})}{(1+r)^t} = 0 \; j=1...N, \; t=1...T$$

where $P_{j,t}$ is the percentage of people in the j^{th} demographic group who still are alive in the t^{th} year, $B_{j,t}$ is the dollar amount of benefits paid to each surviving individual in the

j^{th} group in the t^{th} year,[47] and $C_{j,t}$ is the premium paid by each surviving individual in the t^{th} year.[48]

The Social Security System and individuals could make a corresponding calculations by substituting their own expectations of P_j's and with individuals using their (and their spouses') Social Security taxes. While the Social Security benefit schedule is progressive, Social Security's rate of return paid to a group with given characteristics, such as race and gender, also depends on the P_j's and the C_j's. However, different (private) r's result for individuals than for the Social Security System because in making their calculations individuals only include Social Security taxes paid by themselves and perhaps by spouses while Social Security would use both employee and employer contributions to calculate C_j.

6.3. Analysis of RHS Data

The RHS data on (household head) *respondents* (but not spouses) have been linked to Social Security records extending annually back to 1951 and also with the total of Social Security taxes paid earlier than 1951. This linkage permits accurate estimation of each respondent's primary insurance amount (PIA) since by 1979 the youngest person in the RHS was 68 years old and nearly all years of work experience and taxes paid are included in the records. Thus, we have a record of covered wages for each year, and we can calculate for each respondent the taxes due in each year.[49]

We further assume that the employer's matching contribution is included in the employee's tax base, C_t. This represents the upper bound on employee true contributions to the Social Security System, under the assumption that the true incidence of the employer tax contribution is borne entirely by employees through lower wages (dollar for dollar) than would have been received without employer Social Security contributions. This assumption yields a lower bound on the private rates of return to employees from Social Security contributions and is a maintained assumption in the literature on rates of return to Social Security contributions. If, however, part of the incidence of the employer Social Security tax contribution is not on the employee, the true rate of return to the employee accordingly will be higher. As long as the average incidence on employees of the employer Social Security tax contribution is the same among the groups that we consider (i.e., designated by race or gender), the

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47 We assume, for simplicity that retired people don't work.

48 For all years after 1950, we have exact figures on taxes paid in each year through 1979. We have the total of taxes paid prior to 1951 and we interpolated these data to obtain annual figures. To estimate pre-1951 earnings, we selected heads of household born in 1904-13 from the CPS-IRS-SSA exact match study. For these individuals, we regressed 1937-50 earnings on 1951-76 earnings (separate regressions by race/gender). Using these coefficients, we get an estimate of pre-1951 earnings for each RHS head of household.

49 These results assume that the people in the RHS in 1969 are a random sample of the people in their birth cohort. The results in Chapter 5 on the Dorn sample suggest that there is selective mortality before age 58. However, since mortality rates are sufficiently low for this age group this should not greatly affect our results.

patterns of expected rates of return among different types of workers and the changes in those patters with different morality assumptions are not affected by what assumption is made regarding the incidence of the employer Social Security tax contribution.

In our benefit stream for males, in some calculations we include those benefits received by a current or a former wife that were based on husband's PIA if claimed in lieu of her own benefits.[50] We also calculate the rate of return for some men ignoring the current or ex-wife's benefits. Because the RHS only contains the Social Security records for respondents (household heads), we do not have Social Security taxes paid by spouses. Thus, r will be overstated in the first set of calculations (inclusive of spouse benefits) and will be understated in the second (exclusive of spouse benefits). Hurd and Shoven used the spouse benefit inclusive procedure in their analysis and thus overstate married men's rate of return.[51]

We first assume that everyone lived to 90. Then to begin our analysis of the importance of mortality differences, we use the standard mortality tables. Next we project survival probabilities for various sociodemographic groups using the results for the RHS from Chapter 5. We do so using our proportional hazard model estimates and extrapolating survival rates back to the time the person entered the labor market and forward to age 90.[52]

We calculate taxes paid based on the respondent's actual covered earnings, adjusted to constant dollars using the CPI, and actual Old Age Social Insurance (OASI) tax provisions. Benefits are also in real terms; hence, our rate of return is in constant dollars. We calculate benefits based on earnings histories and Social Security provisions. These calculations are done using actual receipts for the period through 1979 after which we assume benefits are constant in real dollars as provided by law. We assume the earnings tax rate on the elderly is zero.

Consider first Table 6.1. If individuals in the RHS were to live to age 90, and if we do not account for taxes paid by early decedents, white males would have an annual

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50 We use the 1977 rule for divorcees that the marriage had to last at least 10 years. For female heads of household who were widowed or divorced, we calculate that 30 and 37 percent respectively were receiving benefits based on their own PIA. In this calculation we assume the benefits are not based on one's own history if they exceed by 5 percent or more what we calculate is due based on Social Security earnings' records.

51 We can match by age and education the married women who do not draw the 50 percent of husband's benefits to the widows in 1969 who draw their (82.5 percent) benefits. This gives us an estimate of the extra taxes paid (or investments made) by a couple. Problems with such a procedure include that some widowed women fall into the interval between the 50 and 82.5 percent differential, that some women who expected to be widows may have deliberately altered their labor force behavior, and those widowed young may have rejoined the labor force. The proportion of women heads of households receiving old-age benefits that were based on former husband's PIA was 60 to 70 percent. About 14 percent of widows and divorcees of both races would have had between 50 percent and 67 percent of actual benefits if they had used their own primary insurance amount.

52 Some members of the sample did not have twenty years of covered earnings after 1950. For these individuals we estimated the labor force experience missing from the twenty years necessary to use the standard PIA formula. We based earlier earnings on Social Security taxes paid by that individual before 1951.

rate of return of 9.5 percent and black males, who have lower lifetime earnings, a corresponding return of 10.2 percent. Thus, there is some indication of the intended progressivity.

Using standard mortality tables and including taxes paid by early decedents, the white and black male rates of return fall to 8.3 and 8.9 percent respectively — numbers comparable to those in Hurd and Shoven who also used the RHS and standard mortality tables. If we did not include the taxes paid by early decedents, the estimated rate of returns for white and black males would be 8.6 and 9.4 percent. Thus, even using standard life tables, progressivity has been eroded a bit by differential death rates with roughly one-third of the erosion arising from taxes paid by "like" early decedents.

If all white women heads of household in the sample in 1969 lived to 90, the rate of return (without including taxes paid by early decedents) is 10.0 percent. For black women, the comparable rate of return is 11.1 percent. Using standard mortality tables, the rates of return for white and black women are 8.7 and 9.2 percent respectively. Thus, mortality differentials reduce the yields substantially and nearly eliminate differences in rates of return by race for women.

Usually in deciding on the desirability of an investment, a person examines not only the average expected return but also the asset's riskiness. Since the system is indexed against inflation,[53] the major risks to an individual are a collapse in the Social Security System or an early death. Up to now, every time there was a crisis in the Social Security System, the government found a solution — mostly by raising tax rates or the ceiling on taxable earnings though it has enacted an increase in the age one can start to draw benefits. Moreover, when in the 1970's the indexation of benefits was done improperly and the price adjusted benefits rose by an unexpected and unintended amount, the law was rewritten four years later to solve the technical problem, but benefits were *not* rolled back to the intended level (Myers, 1981). Thus, history indicates the government will take some action to prevent a collapse.

The risk associated with early mortality is real. One does not get a penny back if one dies when too young. To examine these risks we calculate variances in rates of return. These variances across individuals are calculated by drawing random death dates from distributions determined by standard mortality tables. A death date is drawn for each male head in the RHS and for the wife whenever a wife was present at the beginning of the survey. Calculated individual benefit streams are then adjusted to account for these death dates and household rates of return are calculated. The variances of these rates of return are given under two assumptions:

1. Those who died before collecting any benefits are included. Technically, the rate of return does not exist for these households. However, with a vector of costs, C, bounded away from zero and a vector of benefits, B, approaching the zero vector, the rate of return approaches -1. Thus, a value of -1 is assigned to all those individuals.

2. Only those who lived long enough to collect positive net benefits in at least one year are included. These individuals can still have negative rates of return

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[53] While the inflation adjustment only comes into effect when the annual inflation rate exceeds 3 percent, the one time recently when this rate was less than 3 percent, Congress adjusted benefits anyway.

if they died soon after drawing first benefits, but all individuals with r = -1 are eliminated.

Finally, the implicit assumptions on spouse's benefits used in the calculations of population rates of return are maintained (i.e., the wife, if present, collects on the husband's earnings record, and in this section no wives are collecting on the earnings records of husbands who died before they began to draw benefits). Our calculations are

	White σ^2	Non-White σ^2
All	.200	.277
Only those who lived to collect benefits	.0040	.0039

Two results stand out. First, almost all of the variance comes from premature death.[54] Second, the variance is about the same for whites and non-whites *conditional* on their living to draw some benefits. However, the higher incidence of premature deaths among non-whites leads them to have a substantially higher overall (unconditional) variance.

To get an estimate of the proportions of variance due to differential mortality and differential earnings/benefits streams given lifespan, we have calculated the variances for all RHS male-headed households assuming husband and wife (if applicable) exactly attain their life expectancies as of the date when taxes were first paid. Two assumptions are made on the presence of a wife:

1. wife present if married or divorced/separated at time of initial survey.
2. wife present as in 1 and also if husband is widower in 1969.

Assumption 2 should be more accurate since few of the 1969 widows would have been widowed before paying Social Security taxes and not have remarried. The differences between assumptions 1 and 2 are trivial. Thus, for this sample differences in earnings and the PIA contribute only about one third of the variation in the rate of return on Social Security investments.

Presence of wife	White σ^2	Non-White σ^2
Assumption 1	.000539	.000687
Assumption 2	.000521	.000643

[54] Again, defined as deaths before drawing first benefits.

6.4. Differences by Socioeconomic Characteristics

Now we discuss more detailed calculations that tell how the expected private rates of return to Social Security vary by gender, race, education and marital status and how important is the provision that allows one to base benefits on current or former spouse's PIA.

In Table 6.2 we present our estimates based on actual benefits received by women, who were heads of household, in various groups defined by race and marital status in 1969. We again begin by assuming that all these women live to age 90. Overall white women have an average rate of return of 11.6 percent while non-white women have a rate of return of 13.3 percent. In both groups the rate of return falls with education.

Interestingly for white women the range of variation in rate of return is only from 12.2 percent for those who did not graduate from high school to 11.1 percent for those who went to college. For black women the range is from 13.8 percent for those who did not graduate from eighth grade to 12.9 percent for those who did graduate from high school. Thus, there is limited progressivity for black women even excluding the impact of differential death rates. The lack of noticeable progressivity for women occurs because of: their lower lifetime earnings, the smaller percentage whose earnings exceed the taxable maximum, the heavy reliance (60 to 70 percent) on their former spouse's PIA, and the importance of capping earnings as explained below. The difference between having a primary education and at least graduating high school indicates some limited progressivity.

When we drop the assumption that every woman lives to 90 and substitute the standard life table survival probabilities (and include taxes paid by early decedents), the estimated rate of return for all white women falls to 10.5 percent with slightly more variation in the rate of return by education level than above. For non-white women the estimated rate of return is 12.0 percent with a slightly smaller variation by education level. Note that the difference between the races is a point to a point and a half while under the first life expectancy assumption it is close to two points.

We also present in Table 6.2 the calculated rate of return for the live-to-age-90 and standard mortality assumptions for female heads of household who in 1969 were divorced, widowed, or never married. Using the live-to-age-90 assumption, the white women widows have a rate of return of about 13.2 percent while the divorced and never married have a rate of return of 11.5 and 9.6 percent.[55] For non-white women the estimated rates of return are 14.7, 12.9, and 10.5 percent for widowed, divorced, and never married, all of which are higher than for white women. By construction mortality differences play no role here. Instead variations arise because of differences in labor force participation and earnings of both the women and their former husbands (if previously married).

Using standard mortality assumptions, the returns for widowed, divorced, and never married white women are 12.2, 10.5, and 8.4 percent respectively. For non-white women, the estimated rates of return are 13.1, 11.2, and 8.6 percent for widows, divorced, and never married. These exceed the rates of return for white women by less than a point while the previous calculations always indicated larger differences.

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[55] We have not included in C_j the Social Security taxes paid by former spouses. We have included benefits based on former husbands' PIA.

Comparing Table 6.2 to 6.6 below, for the same marital group women received higher rates of return than men. This reflects both progressivity in the system and the women's benefits being based largely on the former husband's earnings while in these calculations in Table 6.2 the costs are based solely on the women's taxes paid.

We now consider in some detail the impact of the provision that says (age eligible) currently or formerly married women can draw a benefit that is the higher of one based on their husband's or their own history. Table 6.3 presents the percentages for female heads of household whose benefits were based solely on their own earnings' history. These women account for about 55 percent of the women defined to be female heads of household of all races in the RHS.[56] In terms of education this percentage increases slightly for whites but decreases slightly for non-whites. Divorced and separated women have slightly greater reliance on their own earnings' history than widows in each race.[57]

In Table 6.4, we present the rates of return that all these women would have received based on just their history of own earnings and taxes paid. The estimates in this table are lower than in Table 6.2 by from one to more than three points. The differentials are greater for the less educated and for widows. While these calculations indicate that formerly married women (for at least 10 years for divorcees) would have improved their economic status greatly by using the provision that allows them to base their benefits on former husbands' PIA, several caveats are in order. Social Security's tax rates are related to overall benefits paid. Thus, some unknown part of husband's taxes should have been included in the cost base, i.e., we have overstated these rates of return. Moreover, the existence of this either/or provision could have altered women's labor force behavior and rates of return, though this possibility may be less important for widows.

In Table 6.4A, we present the rates of return for those women (heads of household) who actually draw old-age benefits based on their own PIA. These mostly wealthier women have slightly greater rates of return than those shown in Table 6.4 though it should be noted that sample sizes are small in some instances, and there clearly is some selectivity since husband's earnings are not used.

In Table 6.5, we present the corresponding calculations for males. If we assume that everyone lives to 90, the real rate of return for whites and non-whites are 9.5 and 10.2 percent. These are each about one percentage point less than the estimates for women heads of household and indicate some progressivity.

The results by marital status require some explanation when we include the wife's benefits.[58] Women on average were 3.5 years younger than their husbands. We

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[56] Recall that female heads of household do not include currently married women with non-institutionalized husbands.

[57] Never married obviously rely on their own earnings.

[58] Some idea of the magnitude of this bias from not including taxes paid by married women can be obtained from available data on widowed women who as head of household in 1969 were matched to Social Security earnings files, and who were drawing Social Security benefits based on their former husbands' earnings. Including taxes paid as calculated from the widows' records on earnings in covered employment, the rate of return using standard mortality is estimated at 8.2 and 9.2 percent for whites and non-whites. Thus, the either/or provision adds substantially to the r for married men even when

assume that the current (or former) wife retires at the (actual) age of 62 and draws 37.5 percent of her husband's benefit as based on his PIA. Since this is approximately an actuarially adjusted value of the 50 percent rate if she had retired at age 65, this age of retirement assumption should not affect the estimated average rate of return.

Based on own benefits only and using the live-to-age-90 assumption, the estimated real rate of return for males hardly varies by marital status (ignoring spousal benefits) even though married men have higher earnings. When we include current or former wife's benefits (but not her taxes), the estimated rates of return rise by about three points. The largest difference in rates of return across education levels is 0.3 and 0.2 for non-whites and for whites respectively.[59]

By construction these results do not reflect mortality differences. An explanation is that for most of the time these men worked, the taxable cap imposed on Social Security earnings was quite low relative to average earnings, e.g., in 1964 more than half of white male workers reached the cap. Thus, while average earnings in the population vary substantially by marital status, the numbers used in calculating the PIA varied far less.

When the standard mortality tables are used, and we include taxes paid by early decedents, we obtain lower returns and a somewhat different pattern. The rate of return for non-whites falls more, and this tends to reduce the progressivity of the Social Security System. However, for non-whites the difference in the estimated rate of return between the least and most educated groups is 0.5 while in the live-to-90 calculations it is 0.3.

In Table 6.6, we calculate rates of return for males by marital status using marital-specific life tables but without varying taxes paid by early decedents or benefits by marital status. Note that the non-single categories include — unless otherwise noted — benefits of current or former spouse paid on the basis of the male's earnings. Also note that in this table we do not include in the investment costs any Social Security taxes paid by the women. Married, divorced, and separated men whose wives draw benefits based on the husband's earnings receive the highest rate of return, 9.3 and 10.0 for whites and blacks respectively. Never married and widowed men receive a rate of return of about three to four points lower. Essentially this difference reflects the fact that married men with age-qualified wives receive an old-age benefit of 150 percent. These estimates are a bit higher than in Hurd and Shoven presumably because we use different life tables in which life expectancies are longer.

In Table 6.7 we adjust the life tables using our proportional hazard results. We include taxes paid and benefits received by marital status. Compared with Table 6.6 rates of return are now generally raised a bit for married and divorced men. For example for white widowed men, the estimate is raised from 6.0 to 6.2 percent.

In Table 6.8 we present the results for males by education level and race. We find only small differences in rates of return by education level despite large differences in average earnings. The rate of return is about half a point higher for non-whites than whites. In comparison with Table 6.5 which uses standard life tables, we find small

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"wasted" investments of spouse are accounted for.

[59] Note that we use slightly different educational levels for whites and non-whites to have adequate sample sizes for non-whites.

changes for whites and most non-whites. There is, however, a large reduction in the rate of return for the least educated non-whites.

If whites had the average black characteristics and life table, their rate of return would drop slightly to 8.3 percent as shown in Table 6.9. If we also give whites the same tax and benefit streams, the rate of return would be about the same as blacks received using the standard life tables.

6.5 Results from Two Cohorts in the PSID

RHS was a cohort born in 1906 to 1911. Substantial changes in the Social Security System have been legislated in eligibility, contributions and benefit levels (Myers, 1981). Therefore it is of interest to look at cohorts born more recently than those in the RHS.

We consider two cohorts from the 1988 Panel Study of Income Dynamics (PSID): those born in 1929-33 and therefore 55-59 years old in 1988, and those born in 1942-1946 and therefore 42-46 years old in 1988. The procedures we use to calculate real Social Security rates of return from PSID data are followed as close as possible to those we use with the RHS data. The most significant difference is that we do not have the actual Social Security earnings records for the PSID sample. Therefore, we extrapolate lifetime earnings profiles from the 21 available years of data. To do this, we assume that real earnings grow at an annual rate of 1.5 percent. However, due to lifecycle effects, this rate is not constant over the typical working life. Therefore, Mincer's (1974) estimates of the age/earnings profile are adapted to create profiles with an average growth rate of 1.5 percent.

Returns are calculated for the same subgroups as we use in the RHS. We do not compute the rate of return when there are less than ten people in a subgroup but do include these individuals in the calculations of overall returns for the age/race cohorts.

Heads are assumed to retire at age 65 and, as in the RHS, wives are assumed to be three years younger and retire contemporaneously with the heads. Taxes include both worker and employer contributions. Both taxes and benefits for male heads include payments for and to both husband and wife. Computations for female heads do not include taxes or benefits of former husbands. Benefits of female heads are determined three ways: 1) benefits based solely on their own earnings records, 2) benefits based on own or matched former spouses earnings, whichever was higher, and 3) benefits based on own records when they exceed estimated benefits from spouses records and equal to zero when collecting on former spouses earnings. In all cases, taxes are those paid by female heads and their employers only. Mortality estimates are based on the 1987 life tables from *Vital Statistics of the United States*. Mortality is adjusted according to marital status and education by the race-specific proportional hazard estimates.

Consider the rates of return for males in Tables 6.10 and 6.11. These tables give Social Security rates of return, first under the assumption that all sample members live until age 90 and second under the assumption that mortality conforms to our estimated relations from the RHS. Generally the estimated rate of return is much lower in the two later born PSID cohorts than in the RHS cohort regardless of what mortality assumptions are made. Whites born 1929-1933 using the RHS estimated mortality

profiles have real rates of return around 3.5 percent. Black males of the same cohort have a slightly lower rate of return though they would have had slightly higher rates of returns if everyone lived to age 90. For the cohort born during 1942-1946, estimated rates of return are lower still — for whites 2.3 percent and for blacks 2.2 percent. Within a cohort for each race, there are minor differences in the expected returns by educational level or marital status.

In Tables 6.12 and 6.13 we present results for females. As discussed in Section 6.4, these are more extensive because women, much more frequently than men, opt to use former spouses earnings in the calculation of their benefits. We distinguish between divorced/separated and widows because divorced/separated women are eligible for using former spouse's benefits only if they were married at least ten years. If the benefits from former spouses earnings are included in the calculations the real rates of return are slightly higher for females than those we discussed above for males. However, these rates of return are still significantly lower than those obtained for females from the RHS using the earlier cohort. Racial mortality differentials either substantially eliminate or reverse the differential rates of return between white and black women.

6.6 Conclusion

In this chapter we have examined the rate of return on taxes paid to Social Security by individuals and groups and the impact of differential death rates on the estimated rate of return using respondents in the Retirement History Survey.

We have calculated the returns assuming (1) everyone in the sample lives to age 90, (2) every respondent is subject to the standard death hazards, and (3) the hazards are shifted proportionally by martial status or education as we estimate in Chapter 6. We also have considered the importance of including in the investment base the taxes paid by people in a group who died before becoming eligible for old-age benefits.

We find that if every 1969 respondent lived to age 90, the Social Security System would yield the members of the RHS real rates of return for non-white and white males of 10.2 and 9.5 percent and females of 10.0 and 11.1 percent. Several comments about these results are of interest. First, males with lower lifetime earnings would have had higher rates of return though the differences are not huge. Second, the level of the estimates is quite high. The average real rate of return on U.S. Treasury notes has generally been about zero though longer term Treasury securities have had real returns closer to 5 percent.

Of course everyone does not live to age 90. When we incorporate standard life tables and include taxes paid by others, we obtain estimated rates of return of 8.3 to 9.2 percent with the differences by race and gender of the same order of magnitude. Allowance for this differential mortality has reduced substantially the estimated rate of return and differences in the rates of return between high and low earnings groups. Using our proportional hazard results, there is only a small additional effect on the estimated rate of return.

We also have examined results for marital and education groups. Once we use standard life tables and adjust for taxes paid by early decedents, there is only minor variation by education level given race and gender. The differences by marital status

are much larger with never-married men having the lowest rate once we allow for extra benefits paid to current or former spouse.

While the RHS is a random sample of household heads 58 to 63 years old in 1969, the high rates of return may not be applicable to younger cohorts as we find in the PSID. This issue is discussed in Thompson (1983) which provides a summary of the literature. Because of their age and the periodic expansion in occupational coverage, the people in the RHS have not paid as large a share of their earnings to Social Security as have subsequent cohorts.

Appendix 6.A

Procedures used for calculating mortality adjusted rates of return from Social Security from RHS-SSA data

This appendix provides documentation of the procedures and assumptions employed in the calculation of Social Security rates of return. General references include the *Social Security Bulletin's Annual Statistical Supplement* and Myers (1981), which were used to ascertain the every-changing Social Security tax and benefit formulas, and *Vital Statistics of the United States*, which was the source of mortality tables. The base year for the mortality tables is 1978.

For the purposes of calculating the average monthly wage (AMW) required to determine benefit amounts, the total number of eligible years after 1950 are calculated by age and gender. This number of years is adjusted downward for any individuals who died prior to receipt of benefits. Since RHS deaths are recorded in alternate years, the working assumption is, for example, that an individual alive in 1969 but deceased in 1971 who had no 1970 income is treated as deceased in 1970. If 1970 income is positive, the person is not treated as deceased until 1971.

Following the Social Security benefit formulas, the five lowest income years are dropped and the AMW is calculated by summing the remaining years of income and dividing by the number of eligible months minus 60 (corresponding to dropping the five lowest earnings years).

The Primary Insurance Amounts (PIAs) for 1968-1990 are adjusted to account for mid-year benefit formula changes in some years. Thus, benefits follow those actually received year-by-year by the 1906-1911 birth cohorts. All benefits and taxes are converted to real 1989 dollars and real benefits are assumed to remain constant after 1990.

The 1951-1974 OASI taxes are calculated from the SSA earnings records. These taxes include those paid by the individuals as well as employers. Contributions from multiple employers are included even when the individual has attained the maximum taxable earnings. Since our data only allows us to determine Social Security retirement benefits, the disability insurance and Medicare portions of the taxes are *omitted.* The alternative minimum PIA is also calculated.

Because the RHS-SSA data contains earnings only for 1951-1974, we need an estimate of 1937-1950 taxes annually in order to calculate rates of return. Social Security records have only the sum for this period. We approximate by using results from this program to run regressions of 1937-1950 earnings on 1951-1974 earnings and interpolate over the life cycle. The estimates are done separately for white and non-white males.

Actual Social Security benefits are calculated by allowing for the alternative minimum PIA.[60] Benefits are calculated for wives and adjusted for expected mortality as reflected in life tables for white and non-white females published in the *Statistical Abstract of the United States*. It is assumed that wives are three years younger than their husbands.

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[60] Note that minimum PIA is sufficiently low that few observations are affected.

Table 6.1 Social Security Percentage Rates of Return for Males and Females

	WHITE	NON-WHITE
Males		
Everyone Not Dead at Survey Date Lives to 90 and then Dies[a]	9.5	10.2
Using Standard Mortality Tables for Those Alive in 1969[a]	8.6	9.4
Using Standard Mortality Tables	8.3	8.9
Females		
Everyone Not Dead at Survey Date Lives to 90 and then Dies[a]	10.0	11.1
Using Standard Mortality Tables for Those Alive in 1969[a]	8.7	9.2
Using Standard Mortality Tables	8.8	9.2

[a]This does not include in costs the taxes paid by people in the j^{th} group who died before receiving benefits.

Table 6.2 Social Security Rates of Return for Female Heads Including Own or Husband's Benefits, Whichever is Greater

WHITE		NON-WHITE	
Live until 90	With standard mortality tables	Live until 90	With standard mortality tables
11.6%	10.5%	13.3%	12.0%
0-11 years education		0-7 years education	
12.2%	11.1%	13.8%	12.2%
12 years education		8-11 years education	
11.2%	10.2%	13.2%	11.5%
13+ years education		12+ years education	
11.1%	10.0%	12.9%	11.2%
Widowed		Widowed	
13.2%	12.2%	14.7%	13.1%
Divorced/Separated		Divorced/Separated	
11.5%	10.5%	12.9%	11.3%
Never Married		Never Married	
9.6%	8.4%	10.5%	8.6%

Table 6.3 Probability of Female Heads of Household Receiving Benefits Based on *Own* Earnings Record[a]

WHITE	NON-WHITE
All	All
.559	.565
0-11 years education	0-7 years education
.548	.589
12 years education	8-11 years education
.540	.558
13+ years education	12+ years education
.616	.500
Widowed	Widowed
.293	.367
Divorced/Separated	Divorced/Separated
.346	.479
Never Married	Never Married
1.000	1.000

[a]Sample includes 2605 white and 404 non-white women.

Table 6.4 Hypothetical Social Security Rates of Return for All Female Heads of Household in 1969 (RHS): Based Only on Own Earnings' History and Own Taxes Paid

WHITE		NON-WHITE	
Live until age 90	With standard mortality tables	Live until age 90	With standard mortality tables
0-11 years education		0-7 years education	
10.2%	9.0	11.3%	9.2
12 years education		8-11 years education	
9.8%	8.5	11.1%	9.2
13+ years education		12+ years education	
10.0%	8.7	11.1%	9.2
Widowed		Widowed	
10.3%	9.1	11.4%	9.5
Divorced/Separated		Divorced/Separated	
10.3%	9.0	10.9%	9.0
Never Married		Never Married	
9.6%	8.4	10.5%	8.6

Table 6.4A Social Security Rates of Return for Female Heads who Actually Received Benefits Based on Own PIA and Paid Own Taxes

WHITE		NON-WHITE	
Live until age 90	With standard mortality tables	Live until age 90	With standard mortality tables
All		All	
9.7%	8.5	10.6%	90%
0-11 years education		0-7 years education	
9.7%	8.5	10.8%	9.1
12 years education		8-11 years education	
9.7%	8.5	10.9%	9.1
13+ years education		12+ years education	
9.8%	8.6	1.0%	8.1
Widowed		Widowed	
9.8%	8.6	10.7%	9.0
Divorced/Separated		Divorced/Separated	
9.7%	8.6	10.7%	9.0
Never Married		Never Married	
9.6%	8.4	10.5%	8.6

Table 6.5 Social Security Rates of Return for Males

WHITE		NON-WHITE	
Live until age 90	With standard mortality tables	Live until age 90	With standard mortality tables
0-11 years education		0-7 years education	
9.5%	8.5	10.4%	9.3
12 years education		8-11 years education	
9.2%	8.2	10.1%	8.7
13+ years education		12+ years education	
9.3%	8.3	10.1%	8.8
Widowers		Widowers	
8.7%	6.3	9.6%	6.5
Married		Married	
Only husband's benefits			
8.5%	6.0	9.3%	6.1
Including wife's benefits			
9.8%	9.2	10.5%	9.9
Divorced/Separated		Divorced/Separated	
Only husband's benefits			
8.7%	6.3	9.5%	6.4
Including ex-wife's benefits			
10.0%	9.3	10.8%	10.1
Never Married		Never Married	
8.6%	6.1	9.4%	6.4

Table 6.6 Social Security Rates of Returns for Males

	WHITE MALES	NON-WHITE MALES
Never Married	6.1%	6.2%
Married	9.3%	10.0%
Widower	6.0%	6.2%
Divorced/Separated		
including benefits to ex-spouse[a]	9.2%	10.0%
excluding benefits to ex-spouse[a]	6.1%	6.3%

[a]Taxes paid by women not included. Women's eligibility based on being married 10 years.

Table 6.7 Rates of Return to Social Security by Marital Status for Males Allowing both Mortality and Tax/Benefit Streams to Vary by Marital Status

	WHITE MALES	NON-WHITE MALES
Married including spouse's benefits	9.2%	9.9%
Widower	6.2%	6.4%
Divorced/Separated		
With ex-wife's benefits	9.3%	10.0%
Only husband's benefits	6.3%	6.5%
Never Married	6.1%	6.3%

Table 6.8 Social Security Rates of Return by Race and Education, Where Education's Effects on Both Mortality Rates and Tax/Benefit Streams are Accounted For

	WHITE MALES			NON-WHITE MALES		
Mean Education			10.1 years			6.9 years
	Education Class	Mean Education Within Class	Rate of Return	Education Class	Mean Educaiton Within Class	Rate of Return
Low	0-11 years	7.8 years	8.5%	0-7 years	4.2	8.9%
Middle	12 years	12.0 years	8.2%	8-11 years	8.8	8.8%
High	> 12 years	15.0 years	8.3%	≥ 12 years	13.2	8.9%

Percentage of Males by Education Class

	White		Black	
Low	0-11	58.4%	0-7	56.0%
Middle	12	23.1%	8-11	28.8%
High	> 12	18.5%	≥ 12	15.1%

Table 6.9 Retirement History Survey Rates of Returns

	WHITE MALES
Rate of Return Using Life Table if Had Black Characteristics	8.3
If Had Black Observable Characteristics and Tax and Benefit Streams	9.0

Table 6.10 Social Security Real Rates of Return 1988 PSID Male Heads Aged 55-59 in 1988

White	Live until age 90**	Without mortality**	n
All*	4.37%	3.40%	220
Married	4.37%	3.38%	203
Education < 12 years	4.55%	3.66%	53
Education = 12 years	4.66%	3.51%	81
Education > 12 years	4.21%	3.21%	86

*Includes 8 divorced/separated, 1 never married, and 8 widowed.

**Includes taxes and benefits of spouse/former spouse.

Black	Live until age 90**	Without mortality**	n
All*	4.67%	3.27%	64
Married	4.65%	3.23%	51
Education < 8 years	4.75%	3.38%	28
8 ≤ Education ≤ 1 years	4.71%	3.22%	17
Education ≥ 12 years	4.47%	3.20%	19

*Includes 5 divorced/separated, 3 never married, and 5 widowed.

**Includes taxes and benefits of spouse/former spouse.

Table 6.11 Social Security Real Rates of Return 1988 PSID Male Heads Aged 42-46 in 1988

White	Live until age 90**	Without mortality**	n
All*	3.28%	2.26%	311
Married	3.30%	2.29%	279
Divorced/Separated	3.12%	1.98%	28
Education < 12 years	3.72%	2.74%	40
Education = 12 years	3.45%	2.47%	107
Education > 12 years	3.14%	2.11%	164

*Includes 4 never married.

**Includes estimated taxes and benefits of spouse/former spouse.

Black	Live until age 90**	Without mortality**	n
All*	3.58%	2.15%	83
Married	3.55%	2.08%	65
Divorced/Separated	3.77%	2.15%	14
8 ≤ Education ≤ 1 years	3.92%	2.55%	33
Education ≥ 12 years	3.34%	1.89%	46

*Includes 2 never married, and 2 widowed, and 4 without education < 8 years.

**Includes taxes and benefits of spouse/former spouse.

Table 6.12 Social Security Real Rates of Return 1988 PSID Female Heads Aged 55-59 in 1988

White		Live until age 90**	Without mortality**	n
All*	1	4.96%	4.07%	60
	2	5.47%	4.61%	
	3	4.40%	3.48%	
Divorced/Separated	1	4.81%	3.92%	29
	2	5.29%	4.43%	
	3	3.98%	3.04%	
Widowed	1	5.48%	4.58%	26
	2	6.19%	5.34%	
	3	5.15%	4.23%	
Education < 12 years	1	5.73%	4.87%	19
	2	6.48%	5.67%	
	3	4.89%	3.99%	
Education = 12 years	1	5.02%	4.13%	21
	2	5.76%	4.91%	
	3	4.38%	3.45%	
Education > 12 years	1	4.58%	3.67%	20
	2	4.76%	3.87%	
	3	4.22%	3.29%	

*Includes 5 never married.

Table 6.13 Social Security Real Rates of Return 1988 PSID Female Heads Aged 55-59 in 1988

Black		Live until age 90**	Without mortality**	n
All*	1	5.52%	4.18%	82
	2	6.28%	4.98%	
	3	4.52%	3.11%	
Divorced/Separated	1	5.44%	4.10%	50
	2	6.30%	5.01%	
	3	4.69%	3.29%	
Widowed	1	5.54%	4.17%	25
	2	6.20%	4.88%	
	3	3.88%	2.40%	
Education < 8 years	1	6.54%	5.24%	11
	2	7.88%	6.66%	
	3	5.07%	3.66%	
8 ≤ Education ≤ 12 years	1	6.03%	4.73%	36
	2	7.26%	6.04%	
	3	4.27%	2.87%	
Education ≥ 12 years	1	5.17%	3.78%	35
	2	5.52%	4.16%	
	3	4.57%	3.14%	

*Includes 7 never married.

Table 6.14 Social Security Real Rates of Return 1988 PSID Female Heads Aged 42-46 in 1988

White		Live until age 90**	Without mortality**	n
All*	1	3.72%	2.82%	40
	2	3.84%	2.95%	
	3	3.36%	2.45%	
Divorced/Separated	1	3.65%	2.76%	25
	2	3.79%	2.90%	
	3	3.17%	2.25%	
Education = 12 years	1	4.03%	3.17%	15
	2	4.16%	3.31%	
	3	3.68%	2.80%	
Education > 12 years	1	3.41%	2.49%	19
	2	3.55%	2.64%	
	3	3.05%	2.11%	

*Includes 7 never married, 8 widowed, and 6 education < 12 years.

1: Head's taxes and benefits based on own earnings record.
2: Head's taxes and actual benefits (i.e., own benefits or former husband's estimated benefits, whichever higher).
3: Head's taxes and own benefits only if not receiving.

Table 6.15 Social Security Real Rates of Return 1988 PSID Female Heads Aged 42-46 in 1988

Black		Live until age 90**	Without mortality**	n
All*	1	4.10%	2.74%	63
	2	4.29%	2.94%	
	3	3.95%	2.57%	
Divorced/Separated	1	4.08%	2.70%	37
	2	4.28%	2.91%	
	3	3.93%	2.54%	
Never Married	1	4.00%	2.65%	19
8 ≤ Education ≤ 12 years	1	4.58%	3.25%	18
	2	5.08%	3.79%	
	3	4.43%	3.09%	
Education ≥ 12 years	1	3.96%	2.57%	41
	2	4.05%	2.68%	
	3	3.81%	2.41%	

*Includes seven widowed, and four with education < 8 years.

1: Head's taxes and benefits based on own earnings record.
2: Head's taxes and actual benefits (i.e., own benefits or former husband's estimated benefits, whichever higher).
3: Head's taxes and own benefits only if not receiving.

7
Conclusion

In this book we have developed models of health or of its absence (morbidity) and estimated relations implied by these models. We have used both static and dynamic utility-maximization models subject to both budget constraints and health production functions.

The constrained maximization of the utility function yields demand relations for health (and consumption functions), which can be estimated given the appropriate data. If the model is identified, the health production function also can be estimated. However, data necessary for such estimation are often not available or are poorly approximated.

For those still alive, healthiness can be measured "objectively" by clinical examinations or "subjectively" by introspection. Longitudinal studies indicate that both types of measure have information, much of which overlaps but some of which is distinctive to the measure, for predicting subsequent mortality. Following earlier work by Grossman (1974) and Sickles and Yazbeck (1991), we combine various objective and subjective measures of people's healthiness and estimate a health and consumption demand model. Another variable that is difficult to measure, and that we ignore, is the "price" of health. In contrast to these data problems, our data on the extreme form of the absence of health, the date of death, is measured quite accurately in the two samples that we use — the Dorn and the RHS (Retirement History Survey).

We link death to health by assuming that when one's health falls below some critical level the person dies. Since one's health is determined in part by consumption and other choices that act directly or indirectly through the health production function, the reduced-form death relation depends on all predetermined variables that affect the decision of the individual and the household of which s/he is a member. Some of the variables that we use on the right side of our mortality equations are also chosen by the individual or the household and may not be independent of the disturbance term in the relations that we estimate. It is possible,

for example, that those whose marriage proposal is accepted at time t are healthier than those who did not propose or whose offer was refused, and that this healthiness persists over time. We estimate the annual persistence rate of past health to be 0.9 (i.e., an annual depreciation rate of 0.1). Even with this much persistence, the correlation between health status at the age of marriage and at the age at which we observe such individuals some decades later is small so that the simultaneity bias problem should not be horrendous. For some right-side variables for which the assumption of independence of the disturbance term in our mortality relations is more problematic, such as whether the spouse is working, we also present estimates with and without the variable and find that the estimates of the coefficients of other included variables are fairly robust to this change.

We employ different estimation techniques for the health and mortality equations. For the health system, we use the generalized method of moments estimator. We devote much more attention to the estimation of the mortality hazard equation since there is much less available evidence regarding how robust such estimates are to various alternatives. We use both Cox and Weibull proportional hazard models and also various accelerated-time-to-failure models. For the latter models we make use of four alternative functional forms for the distribution of observed and unobserved date of death. Our results indicate that our estimates are robust across the various models and specification.

We have also allowed for the possibility of individual differences in frailty or heterogeneity using both parametric and non-parametric estimators. We find that the coefficient estimates on observed variables are changed by only small amounts by allowing for unobserved heterogeneity, except possibly in our separate cohort estimates that are summarized below.

While many of our models are estimated using standard partial or maximum likelihood techniques, we also use two methods that have not been extensively employed in economics. These are the Maximum Penalized Likelihood Estimator (MPLE) and the Nonparametric Maximum Likelihood Estimators (NPMLE). The MPLE modifies the likelihood function by giving less weight to (penalizing) outliers and thus reduces the influence of heterogeneity. The NPMLE incorporates heterogeneity explicitly by classifying each observation in one group or point of support. We have mostly used two points of support for the NPMLE, although we have estimated a few models with three points of support, which adds substantially to computer costs. We also used a parametric (normal) estimate of heterogeneity. This fits the data better than when we do not allow for heterogeneity and worse than in estimates based on non-parametric methods. The parameters of the observed variables, as noted above, change when we incorporate heterogeneity but not by huge amounts.

To obtain a "feel" for the properties of these not-often-used estimators, we conducted some "Monte Carlo" studies on small samples ranging up to 100 observations. Neither estimator dominates over all conditions studied, though the MPLE seems to be somewhat better on the whole.

We have used the various estimators to examine date of death in the Dorn and RHS samples. The first sample obtains month and year of death from the VA's records, which are nearly 100 percent accurate (Beebe and Simon, 1969). The

second sample obtains month and year of death from Social Security records, which also are now considered nearly 100 percent accurate (Duleep, 1986), for the period 1969-1977 with less accurate data for 1978 and 1979.

The Dorn sample covers some 300,000 (mostly white) males who served in the Armed Forces between the World Wars. Data were collected in the mid 1950's on current and past tobacco usage and current occupation. Age and 1954 residence are also known. Monthly date of death has been collected through 1980 from V.A. records. The occupation data have been used to construct measures of physical activity and life riskiness (via a life insurer's handbook). We find that the more one smokes the younger one dies. However, those who had stopped smoking have the same life span as non-smokers. Occupation characteristics also have important associations with mortality. Mortality is higher for those in occupations with less physical activity and more risk.

We have analyzed the data for several time periods and cohorts as defined narrowly over five-year birth intervals. The longer time period studied yields more precise estimates but also results in lower estimated effects of smoking. Perhaps this is because some people stopped smoking after the mid 1950's as indicated in Feldman, el al. (1989) and stopping smoking helps to extend expected lives. The birth cohorts studied date back to 1870 and end in 1924. The effect of smoking is not constant over birth cohorts. A strong trend exists with a much greater reduction in life expectancy for the more recently born people even though our retrospective data cover a smaller fraction of their life span. We think this occurs because by 1954 some people had already died, and those born before the turn of century who survived to be in the sample are the hardier individuals. The effects of this left censoring remain to be resolved fully. However, our estimates using the MPLE, which is the only method we have used to control for heterogeneity in the five-year-cohort studies, indicate smaller trends than if there is not control for heterogeneity.

The RHS is a nationwide random sample of heads of households who were aged 58 to 63 in 1969. By Census definition a head of household is a male if present. Hence, the sample has about 7,900 usable male respondents and about 2,500 usable female respondents. While some information is collected on wives of the male respondents, their dates of death are not recorded since they are not linked to the Social Security records. Hence, for women we can only study mortality hazard functions for those who are household heads most of whom are non-married. The RHS collected extensive socioeconomic information in 1969 and subsequently every second year through 1979. We have used some of the information on marital status, dependents, health, race, gender, income sources and occupation. Since Social Security benefits depend on one's whole history of working, including choices made in the 1970's, to minimize simultaneity problems we used the benefits that would have been available in 1969 based on appended records from Social Security. We do not have the information to calculate pensions due as of 1969. For one thing, we have a personal lack of knowledge of a company's pension plan. For another, some plans depend on earnings in the last or last few years of work, which were unknown in 1969, and the "expected" pension data collected then were inaccurate; hence, we used "actual" pensions collected.

Our estimates indicate that greater Social Security benefits, pension benefits and

asset income are associated with death at older ages. Married men live 10 percent longer than those men who are never married, while divorced and widowed men live 8 and -5 percent longer respectively. The marital differentials are smaller for women though we have very little data on currently married women. Blacks tend to die younger than whites and have a higher death rate at each of the limited ages covered in the RHS. Much of the differential would be eliminated if blacks and whites had the same averages for the socioeconomic variables studied.

We further use the mortality information to study the impact of differential death rates on the rates of return on investments made (taxes paid) on earnings taxed by Social Security. According to the mandated Social Security benefit schedule, the ratio of benefits paid to lifetime earnings decreases as earnings increase. Because Social Security taxes paid are proportional to (annual) earnings up to a ceiling, this payment pattern indicates that the rate of return on taxes paid should decrease as earnings increase. We find this pattern for both genders when we do not allow for mortality differences. Once we impose the observed mortality differences and allow one's benefits to cease at death, differentials in rates of return are substantially reduced, and the Social Security System redistributes little income across income classes, education groups, gender and races.

We also find evidence of two problems that will need to be explored in future research. The first is that a sample of people who are drawn from a birth cohort subject to prior mortality may be "selected" with implications for obtaining unbiased estimates of effects of independent variables. We indicate and implement some ways to surmount this problem.

Second, it is generally accepted that more information about the length of life (i.e. less right censoring of the mortality distribution) provides researchers with a better empirical vehicle for examining covariate effects on mortality and morbidity. In one of our samples mortality information was initially available only through 1969, which we first analyzed based on individual characteristics reported in the 1950's. Subsequently, mortality information became available through 1980. Covariate estimates from alternative specifications of the mortality hazard are robust for a given censoring date but not for different censoring dates. We think that these changes are not only due to reduced censoring but also to changes in covariates such as smoking and occupation that occurred after the data on these covariates were collected. Less censored mortality data may not have as great an advantage as often is assumed if covariates (measured at some base period and not remeasured) become less relevant in effecting the death hazard.

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