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Chapter 11

MORTALITY AND MORBIDITY AMONG ADULTS AND THE ELDERLY

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Handbook of Population and Family Economics. Edited by M.R. Rosenzweig and O. Stark

In Memorial

to

Paul Taubman

(1939–1995)

He served as a friend, mentor, and colleague to all of those whose lives he touched.

1. Introduction

We all die, even if, as Dr. Seuss, we have an immortal corpus. The main issues are when, how, and why. Over the last several decades, health care professionals, demographers, economists and other social scientists have made great strides in their attempts to answer these questions. Interest by economists arises at least in part because of intellectual curiosity about the human condition, because a changing life expectancy affects the costs of pensions, social security, and medical care, and because changing morbidity affects the productivity of labor. Humans are indeed complex beings, and for most our eventual passing will be marked by many clinical events, some acute and others of a more chronic nature. Our medical record thus becomes an important anecdote: it tells not only of the length of our life, but also of the quality with which we lived it. Did we enjoy good health, or were we destined to live many years in pain or in a disabled state? Is it destiny that determines mortality, or do we essentially choose our fate?

The purpose of the chapter is to discuss the questions to be asked of the compiled mortality and morbidity data, paying particular attention to those data sources which are readily available and to those methods which have proven or may prove themselves particularly fruitful. In our summary of key findings from many studies, we will hopefully convince the reader as to the power of many of the newer modeling approaches to expand our understanding of the complicated nexus of mortality determinants. In the course of our survey we must keep in mind the response of a dying Gertrude Stein to the question put to her by Alice B. Tolkas: "Gertrude, what's the answer?", to which Stein's reply was "Alice, what's the question?" (Fuchs, 1992).

Section 2 of our chapter begins with a review of recent trends in mortality and morbidity in the US and in other countries and the possible roles for economic development, gender, ethnicity, migration, nutrition and anthropomorphic factors, government, and the health care system in explaining these trends, particularly in national surveillance data. In order to disentangle the structure of causality that puts into question the interpretation of such associations at the national and international level, we focus in Section 3 on static and dynamic structural models of health based on variants of the original Grossman (1972a, b) health production paradigm. We present these models within a general framework which can be utilized by researchers interested in such structural modeling without reference to particular functional forms chosen for utility and production functions. We also discuss various risk factors that researchers have found to be important determinants of mortality and morbidity at the individual level, including human capital characteristics such as schooling, behavior and lifestyles, and genetics. Section 4 provides a short discussion of the various public-use data sets in the US and elsewhere which are available for mortality and morbidity study and related self-assessed health measurement systems which have been used to fashion informative measures of health status. We turn to statistical and numerical techniques which have been employed or could be implemented to estimate reduced-

form mortality hazard relations and structural models in Section 5, as well as enhancements in specifying and estimating structural dynamic models that are currently proving fruitful for researchers on this and related topics. Section 6 provides concluding remarks. Our focus will be on adults and the elderly.

2. Trends and sources of variations in international and US vital statistics

Throughout the twentieth century, there has been a steady rise in life expectancy throughout the world. We now live in a world where life expectancy averaged 67.2 years (Table 1). In the United States, a person born in 1900 could expect to live an average of 47.3 years. By 1995 that average had increased to 75.9 years (Fig. 1)[1,2]. There are a wide range of life expectancies throughout the world, varying by continent from a weighted average of 56.0 years in Africa to 77.6 years for Australia, and by nation from 37.5 years in Uganda to 79.3 years for Japan.[3]

Worldwide increases in longevity have shifted the age distribution toward older populations, whose morbidity patterns, and the policy implications of such, recently have been documented by the World Bank (1993). The implications of population aging are substantial at the social level (Restrepo and Rozental, 1994) and at the economic level as countries attempt to balance inter-generational transfers. Particularly problematic are the demographic shifts in Japan and Europe, where fertility rates are in decline (Bös and Von Weizsäcker, 1989).

Morbidity also varies substantially throughout the world, ranging from a low of 117 disability-adjusted life years (DALYs)[4] lost per 1000 population in established market economies in 1990 to 575 DALYs lost per 1000 population in sub-Saharan Africa (Word Bank, 1993). The number of DALYs lost worldwide in 1990 was estimated by the World Bank to be 1.36 billion, representing 42 million deaths of newborn children and 80 million deaths for those at age 50.

Fig. 2 shows the distribution of the world's population by national life expectancy based on 66 countries with over five million in population. Were there no country effects one would expect that this distribution would mirror the distribution of individual endowed life expectancies and at a heuristic level, the observed multiple modes in Fig. 2 appear to be inconsistent with a distribution of life expectancies driven by the law of large numbers.

[1] Recent forecasts of age-specific death rates suggest that 2065 life expectancy will increase by an additional 10.5 years (Lee and Carter, 1992).

[2] As noted by many researchers, age distributions based on Census data may overstate the portion of those in the higher age deciles due to systematic overstatement of age by the elderly (Coale and Kisker, 1985).

[3] These figures are based on the *Statistical Abstract of the United States, 1995*. For an exhaustive summary of mortality trends for low mortality countries in earlier periods see, e.g., Condran et al. (1991) and Himes et al. (1994).

[4] We provide a more detailed discussion of this and other measures of morbidity in Section 4.

Table 1
Vital statistics worldwide, 1995[a]

Region	Population[b]	GNP[c]	Life expectancy[d]	Infant mortality[e]
North America	370	17658	75.3	12.7
Canada	27	20840	78.1	6.9
US	253	22550	75.9	8.1
Mexico	90	3051	72.9	27.4
Central America[f]	23	1458	74.5	17.8
South America	278	2532	66.2	47.3
Western Europe[g]	369	18732	77.2	7.0
Eastern Europe	96	4763	72.4	13.9
Asia[h]	3343	2697	64.6	58.5
Africa	476	613	56.0	80.0
Australia	17	16600	77.6	7.3
World[i]	4973 (91%)	4874	67.2	51.5

[a]Weighted (by population) averages for countries of more than five million people, all data are taken from *The Statistical Abstract of the United States, 1995.*
[b]Populations are in millions of persons.
[c]GNPs are expressed in terms of constant 1991 US dollars.
[d]Life expectancy is at birth, in years.
[e]Infant mortality is the rate of deaths before one year of age, per 1000 births.
[f]We have included Cuba here.
[g]We have included the new unified Germany here.
[h]We have included those states comprising the former Soviet Union here.
[i]This sample represents 91% of the world's total population.

Mortality and morbidity clearly vary by country. Can national identity serve as a proxy for the observed variations in life expectancy? Using the US as an example, if we were to approximate our white population by Canada and black population by Africa, we would predict using simple correlations a life expectancy of 75.0 years (Table 2), seemingly consistent with the observed value of 75.9 years. However, as we likewise proxy additional details regarding the ethnic distribution of the population, representing the Hispanic population by Mexico and the Asian by Asia, our prediction begins to diverge, rather than converge. The international data suggest that there are significant determinants of mortality not captured by the simple use of national origin as proxy. We discuss a number of potential determinants below.

2.1. *Level of economic development*

Worldwide data on life expectancy does appear to be strongly correlated with economic development and employment (see, for example, Brenner's (1983) and Wagstaff's (1985) surveys, and Sen (1993)). Fig. 3 displays the strong relationship between life expectancy and per capita gross national product (constant 1991 US$) for the 66

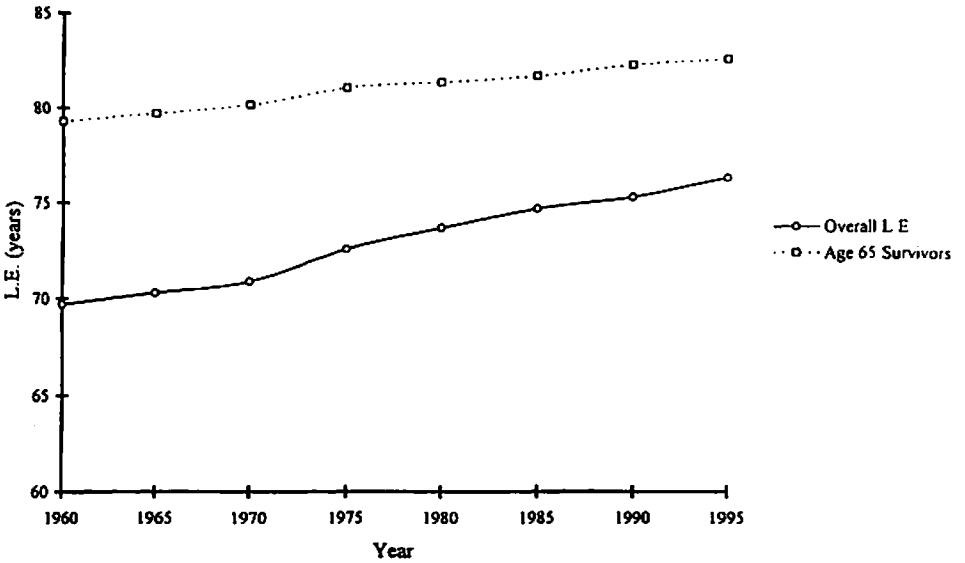

Fig. 1. Evolution of life expectancy by age cohort (US).

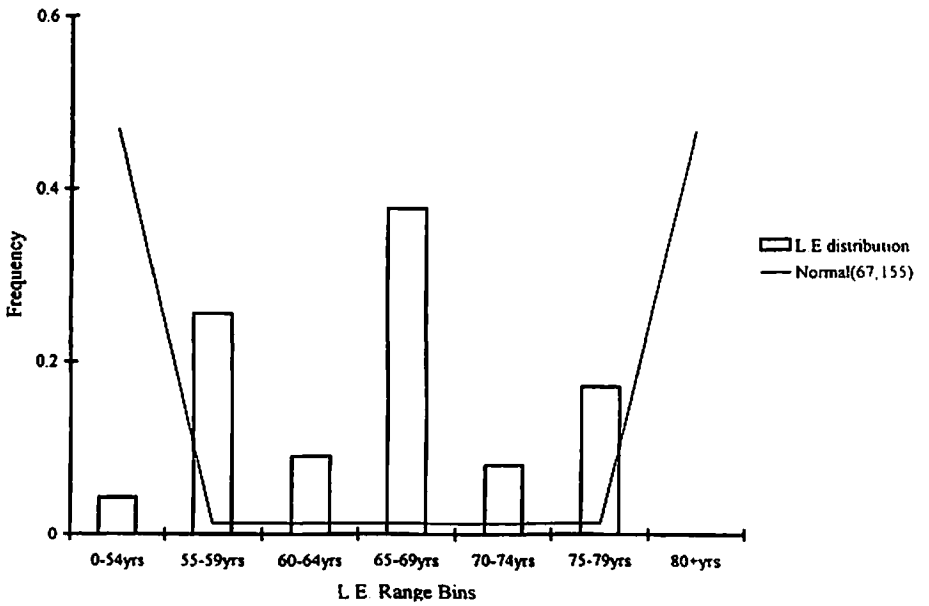

Fig. 2. Distribution of average life expectancy (World 1994).

Table 2
Ethnic group as life expectancy proxy (US 1992)[a]

Ethnic group	Population	National origin (years)	PCI[f]	Economic agent (years)
Black[b]	0.12	56.0	9300	75.1
Hispanic[d]	0.09	72.9	8874	74.5
Asian[e]	0.03	64.6	(n/a)	
White[c]	0.76	78.1	15981	77.4
Interpolate (actual value = 75.9 years)		~74.6		~76.8

[a]All data are taken from *The Statistical Abstract of the United States, 1995.*
[b]Life expectancy approximated by weighted average for Africa for 1995.
[c]Same as above, for Canada.
[d]Same as above, for Mexico.
[e]Same as above, for Asia.
[f]Per capita income by race for 1994 (in constant 1992 US$).

nations of the world with over five million in population, representing 91% of the world population. The data strongly suggest that longevity is an economic good; evidence that life expectancy increases as a country improves its standard of living long has been recognized since the higher income typically associated with development makes possible in part the consumption of goods and services that improve health (Preston, 1976). Recent studies of the most populous country, China, reinforces this assertion (Nolan and Sender, 1992; Knight and Song, 1993), contrary to Sen's (1989) assertion that Chinese reforms have reduced China's ability to provide its population with food and health services. Other reforms in the former Soviet Union and in Eastern Europe have meant more accessibility to mortality data which is showing disturbing trends (Krúmiņš and Zvidriņš, 1992).

The relationship between economic development and mortality and morbidity is overlaid with a mosaic of other competing relationships, such as those between economic development and environmental carcinogens. As reviewed by Knudson (1992), there are known carcinogens in our ambient environment. More aggressive hypotheses concerning the relationships between pollution and cancer are found in the environmental toxicology literature (Ba Loc, 1990; Burney, 1992). The level of economic development may proxy exposure to certain environmental toxins whose proper treatment and disposal in turn requires a relatively high level of economic development.

2.2. *Gender*

The US and international census data also strongly suggest that human life expectancy

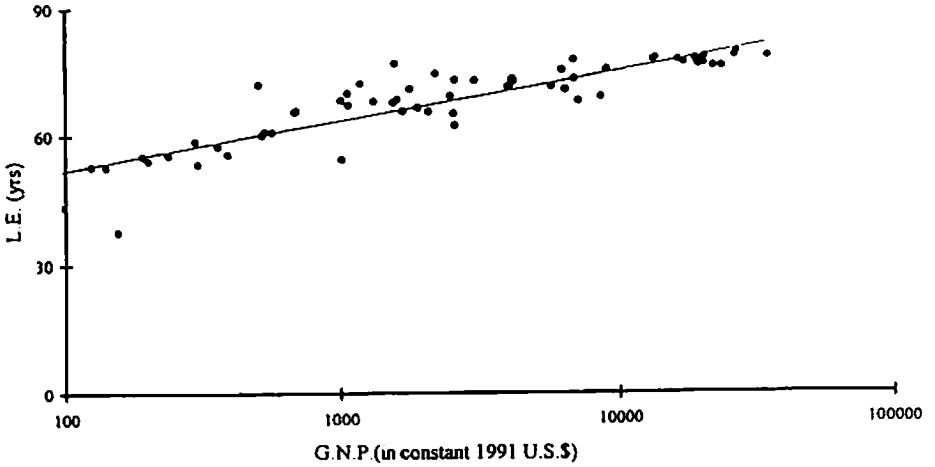

Fig. 3. Life expectancy and gross national product (World 1994).

varies by gender (Zopf, 1992)[5] as it does for other species (Retherford, 1975).[6] As seen in Fig. 4, in the US the discrepancy in life expectancy between men and women has remained fairly constant over the past 35 years, despite a conventional wisdom that the male survival disadvantage is associated with behavior patterns (such as "Type A" behaviors) which have been modified through public education and despite the substantial increase in female labor force participation rates and female adoption of predominately male lifestyle behaviors such as smoking.

There is considerable debate as to whether gender is best viewed as an instrument for behavioral and lifestyle issues, or as a proxy for an unobserved acquired survival advantage. For example, Waldron (1982) investigates the survival advantage for females, and finds that much of the advantage is related to cigarette smoking, so-called "type A behavior", alcohol, and exposure to occupational risks. While alcohol consumption among men has fallen, Waldron (1993, 1994) does not find a corresponding narrowing of sex-differences in survival rates.[7]

[5] In his comprehensive text on mortality, Zopf points out findings of Madigan (1957) which highlighted the biological differences in mortality by gender in his comparison of Roman Catholic male and female celibates living in almost identical conditions. However, Zopf also points to the work of Stolnitz (1956) who found that such factors as high rates of maternal mortality, female infanticide, and the low economic and social status of women in some societies may be significant factors in explaining gender mortality differences.

[6] Anson (1991) notes that variations in age-specific mortality between genders were sufficient to portray both the similarities and differences among 358 human life-tables.

[7] Female-specific studies of mortality have included Wolfe and Haveman (1983), using women from the Panel survey of Income Dynamics, Weatherby et al. (1983), based on life-tables from 38 countries around the world, and more recently, by Kravdal (1994), using the Norwegian Family and Occupational survey which features data on fertility, marriage, education and employment for 4000 women.

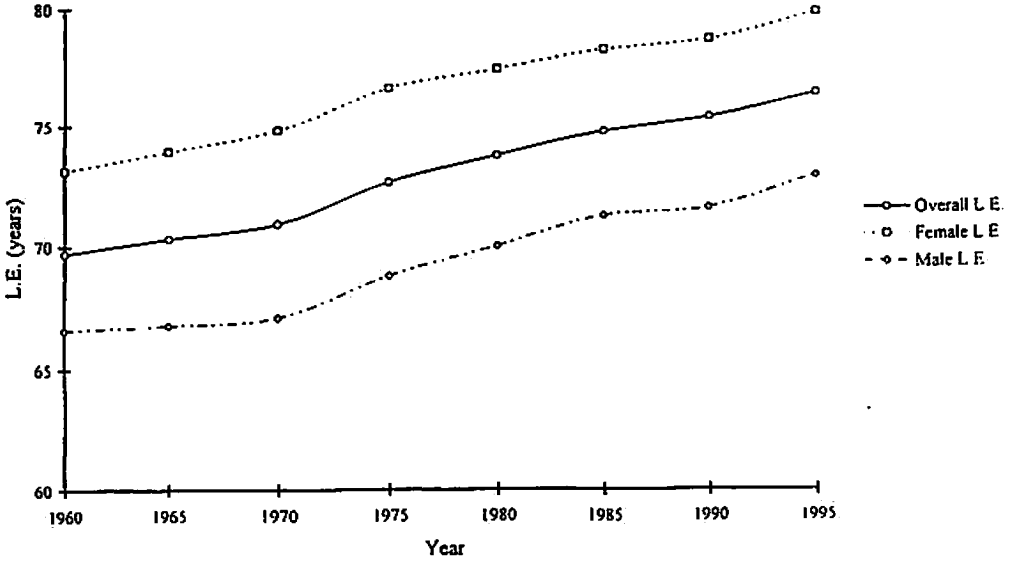

Fig. 4. Evolution of life expectancy by gender (US).

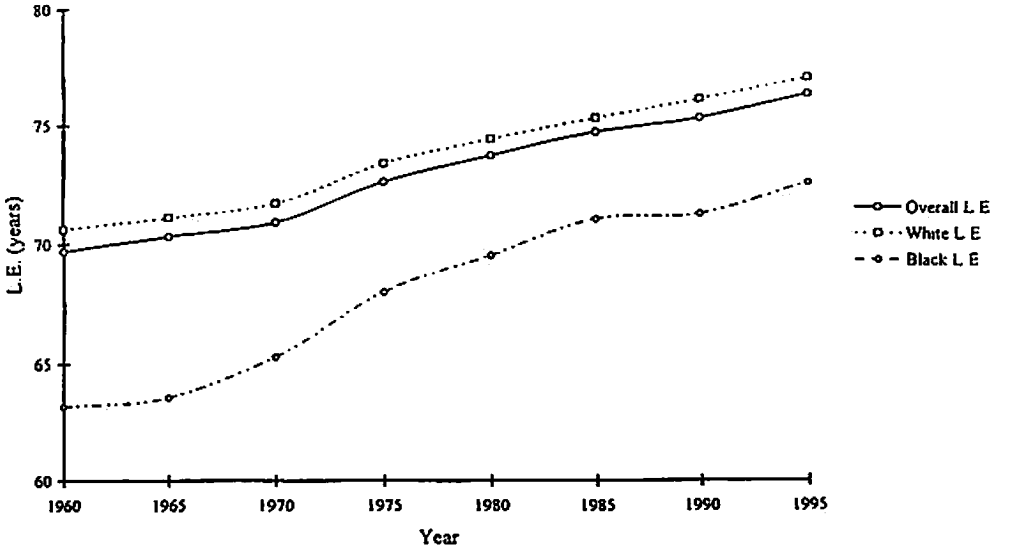

Fig. 5. Evolution of life expectancy by race (US).

2.3. Race

US national trends also suggest a strong source of variation in life expectancy by race. Shown in Fig. 5 is the evolution of life expectancy in the US for blacks and whites. The difference in life expectancy between the two groups narrowed substantially between 1960 and 1995, from 7.4 years to 4.5 years. Were this pattern to continue, the difference would vanish in about 50 years. A more careful review of Fig. 5 suggests that, during the ten years between 1965 and 1975, during which Medicare and Medicaid were introduced and evolved into the institutions they currently represent, the gap narrowed from 7.5 to 5.4 years, whereas during the other 25 years it narrowed by less than one year.

Throughout their lifetime, blacks in the US have higher age-specific death rates than whites. In 1960, for males aged 50 years, the death rate was 9.5 per 1000 persons for whites and 15.6 per 1000 for blacks, whereas the remaining life expectancy at age 55 for whites was 19.5 years and for blacks 18.4 years (Kitagawa and Hauser, 1973). Jaynes and Williams (1989) report that in 1984 remaining years of life at age 65 was an average of 14.8 years for white men and 13.4 for black men. Madans et al. (1986) use the NHANES follow-up to examine the differences by race (as well as gender and those living in poverty) during the 1971–1975 period of deaths during a ten-year follow-up period. They demonstrate strong differences in all of these three dimensions on the ratio of actual to expected deaths. More recently Behrman et al. (1991) show that the death hazard rate is much higher for blacks than for whites in the years covered in the Retirement History Survey (see also, Silver, 1972; Ford and DeStefano, 1991).[8]

Fig. 6 shows black life expectancy and public health care expenditures as a percentage of the gross domestic product. There is a striking correlation between the data, particularly when we consider the lagged effects of public health expenditures on life expectancy. Between 1966, two years after the Civil Rights Act of 1964, and 1976 the percentage of blacks below the poverty line fell from 41.8% to 31.1% (*Current Population Reports*, P60–185), and, with the public sector expanding health care provision to the poor, black life expectancy rose.

Most researchers in public health now disavow race per se as a contributor to mortality. Phillips and Rathwell (1986) review the subject and conclude: "It is now generally accepted in the scientific community as proven that all humans are genetically similar except in terms of susceptibility to a few rare diseases". Cooper et al. (1981), who examine differences in mortality by race and gender, conclude that environmental factors (e.g., nutrition, occupation, and income) underlie the discrepancy in death rates. He states: "...explanations based on biological determinism no longer enjoy consensus support ... there is no evidence that a major proportion of the observed

[8] Fuchs (1992) notes, among other things, the various problems in operationalizing a meaningful poverty measure as well as in linking low income per se with poor health.

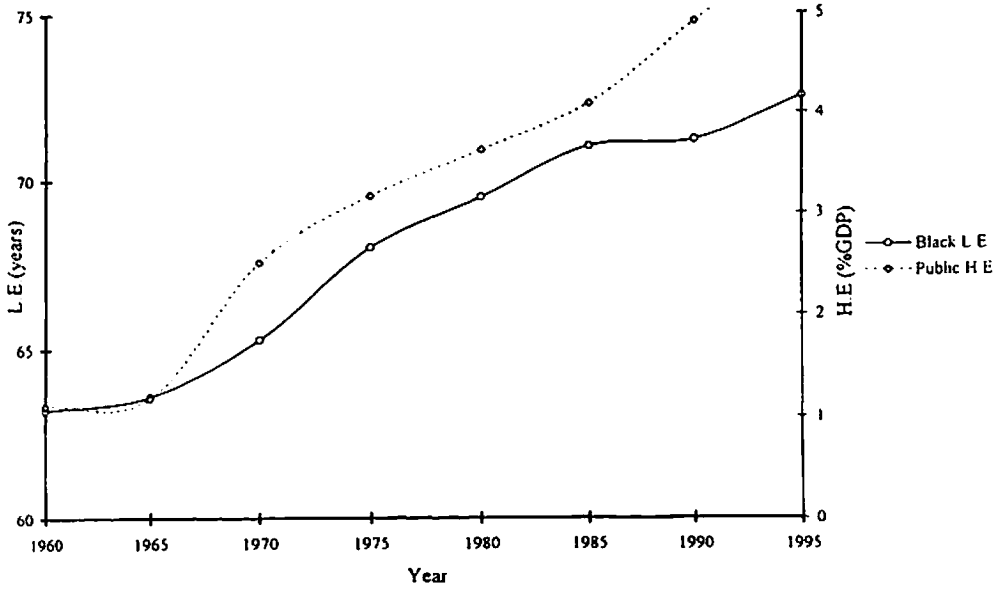

Fig. 6. Evolution of black life expectancy and public health expenditures (US).

racial differentials in health can be explained in population genetics. Environmental forces, namely social conditions, are the root cause".

While it would be naive to preclude the possibility of race as a proxy for unobserved heterogeneities, particularly given our understanding of differences in underlying susceptibilities to certain diseases (e.g., sickle-cell anemia), there is further support within the surveillance data for the race-as-proxy for socioeconomic status (Otten et al., 1990; Rogers, 1992; Menchik, 1993; McCoy et al., 1994; Zick and Smith, 1994). Moreover, differential death rates for blacks and whites based on differential socioeconomic status give rise to peculiar but intuitive mortality patterns for blacks and whites, in particular the crossover of death rates. The crossover is a well-known, recurrent and consistent feature in the US mortality data. Shown in Fig. 7 are annual death rates by race and age cohorts. After 84 years of age, the black cohort has a lower death rate than the white cohort.

2.4. *Infant mortality*

One might dismiss this phenomenon because of a censoring of the black American population, that is the elimination by deaths of the lower tail of the population distribution due to particular traits within the population or due to differential rates of infant mortality which are substantially higher for blacks than for whites.

Differential rates of infant mortality among countries may also explain differential mortality and morbidity for adults and the elderly among countries. Based on the life-

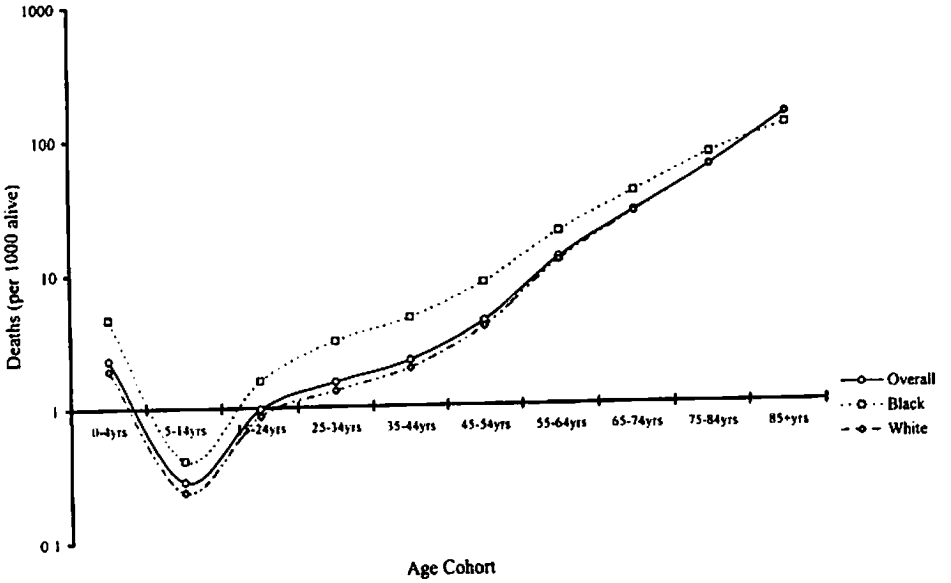

Fig. 7. Annual death rate by age cohort and race (US 1991).

table for the United States for 1991, the overall death rate during the first year of life of 9.3 per 1000 live births was exceeded by the age 55–64 and older cohorts. This death rate crossover occurs much later in life in less developed nations where the infant mortality rate may be more than ten times as high as it is in the United States. Are infant mortality rates related to economic factors? There is a wide consensus to the

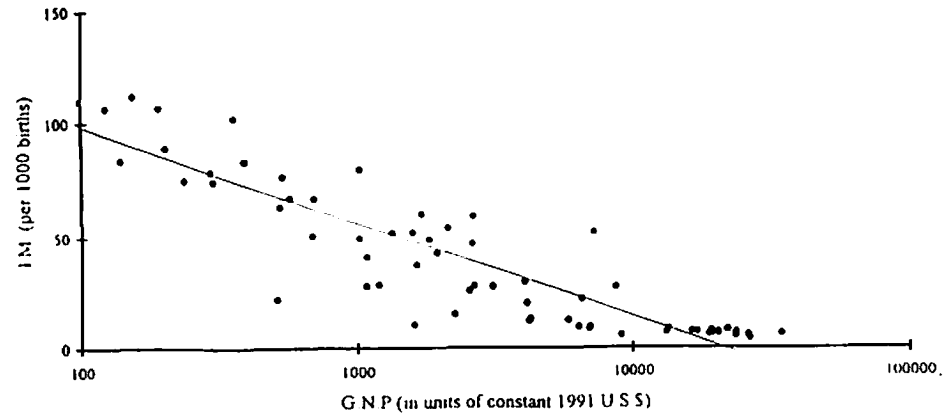

Fig. 8. Infant mortality and per capita gross national product (World 1994).

affirmative on this issue. Fig. 8 relates infant mortality rates and per capita gross national products from the sample of the 66 largest countries in the world, again noted to represent over 91% of the world's population. As was the case earlier with life expectancies, there is a strong and significant negative correlation between the rate of infant mortality and per capita GNP.

Factors which influence infant mortality, in addition to income per se, have been studied extensively. For the US, Corman and Grossman (1985) point to the importance of female education, abortion availability, neonatal intensive care facilities and Medicaid to black neonatal mortality, and note the implications of endogenous censoring of health outcomes among various ethnic groups. Rosenzweig and Schultz (1982a, b) and Thomas et al. (1990) also note the relative importance of maternal education in reducing child mortality in developing countries, although Behrman and Wolfe (1987) found that this effect is reduced when maternal endowments are properly controlled. Akin et al. (1990), Popkin et al. (1993), and their colleagues (Cebu Study Team, 1992) have pursued the estimation of child health production functions in a series of studies from the Philippines and have found important community and environmental effects. Schultz (1993) recently addressed the endogenous selection problem of infant mortality in developing countries, in particular the endogeneity of fertility control (Schultz, Chapter 8, this Handbook). The impact of selective mortality and fertility on estimates for developing countries has also been examined by Pitt and Rosenzweig (1989) and Lee et al. (1995) using parametric and robust estimators.

2.5. *Migration*

There are undoubtedly differential mortality rates within ethnic groups and among countries due to the migration selection of a population. In the US, for example, Diaz-Briquets (1991a, b) presents evidence to support a conventional wisdom that the more socioeconomically integrated Cuban-American population is advantaged in survival against other Hispanic populations. Whereas Puerto Rico is a commonwealth of the United States, with liberal entry and exit between the two, Cuba has had a very different recent immigration history. Earlier patterns were consistent with a selection bias for the "best and brightest" to the United States, and during this period this group was considered an example of successful integration. More recently, migration patterns may have been altered by the relatively large and unusual migration experiences of the late 1970s.

Bradshaw and Liese (1991) note in their analysis that survival data in the Southwestern United States and in Northern Mexican states are consistent with an acquired survival advantage for Mexican-American immigrants to the United States when compared to their particular age–sex cohort remaining in Mexico. However, these findings may be due to selection bias. Not only are there physical hardships associated with the migration from Mexico to the United States, but overall health status appears

to be associated with types of employment common to these individuals. Additionally, the lack of government and employment benefits such as medical or disability insurance may adversely affect less healthy undocumented workers, decreasing their likelihood of remaining in the United States. Overall, Hispanic-Americans remain at a survival disadvantage, possibly due to an excess of early and preventable or manageable deaths (e.g., motor vehicle accidents, complications of diabetes). Censoring of the Hispanic population, which is apparently endowed with survival advantages against many of the later-onset chronic conditions, results in the same type of elderly crossover in death rates as those seen for black Americans.

2.6. *Anthropometric factors*

The secular trends that we have seen in mortality rates seem to be at odds with Malthus' (1798) predictions that "normal" life expectancy was fixed (Fogel, 1994a), although as Fogel points out, the essential point of the Malthusian thesis, that of population pressures against the constraints of available resources, is no less valid today than in Malthus' time. Indeed, the freeing up of constraints by improvements in public-health, sanitation and personal hygiene, advancements in medical technology which restrain certain pathogens (Preston, 1980), and rises in standards of living and concomitant increases in nutrition, appear to have made possible substantial increases in populations and in life expectancies. The issue of how anthropometric factors and nutritional improvements have impacted mortality rates has been taken up by Fogel (1993, 1994a, b, 1995; and Chapter 9, this volume) in a series of recent works to which we now briefly turn.

Fogel (1994b) points out that not only have nutritional advances in their own right altered life expectancies but also the nature of the dynamic between nutrition and infection (Scrimshaw et al., 1968; Preston and van de Walle, 1978) and the changing biological interaction between disease and humans (Fridlizius, 1984; Perenoud, 1984, 1991; Alter and Riley, 1989; Schofield and Reher, 1991).[9] Moreover, identification of the contribution of nutritional advancements per se to mortality hazards is clouded by the interaction, among other things, between nutrition and a set of risk factors including the level of physical activity, climate, disease exposures, and individual susceptibilities or frailties to disease. Since caloric intake must be balanced by the level of physical activity which it supports, a working definition of nutrition net of the claims on it is necessary in order to examine its particular contribution on life expectancy.

In Chapter 9, Fogel provides an extensive discussion of anthropometric indicators (e.g., stature, body mass) of nutritional changes in Europe and the US and their strong

[9] H.O. Lancaster's (1990) and Preston's (1976) comprehensive surveys of world mortality trends suggest that developments in the treatment of infectious diseases with antibacterials such as penicillin, and the development of effective vaccines for childhood viral conditions such as smallpox and polio, underlie much of the gains in life expectancy we have seen this century.

association with morbidity and mortality risk (Heywood, 1983; Waaler, 1984; Martorell, 1985; Osmani, 1992; Fogel, 1993; Fogel et al., 1994).[10] He also points to the ways in which variations in these anthropometric markers allowed for population size and the available food supply to equilibrate and in so doing determine life expectancy. Recent history has also shown the strong similarities between the current experiences of less developed nations and those of developed nations several centuries earlier. Fogel points out the recent debate on the fixity of life-expectancy, estimated to be 85 ± 7 years by Fries (1980) and notes that Fries' assertion that increases in life expectancy are explained by a one-hos-shay type survival curve with most deaths centered around age 85, implies that risk factors such as acute infectious diseases have been crowded out by independent chronic conditions. Compression of mortality, according to Fries (1983, 1989), would thus be possible only by changes in life-style and by medical interventions, at odds with Vaupel's (1991a) findings, using Danish twins, of a much longer life expectancy, only about 30% of the variation of which could be attributed to genetic factors. Vaupel (1991b) argues that the survivor curve will not flatten. Similar findings that argue against survival compression are found in Manton et al. (1993). These issues raised by Fogel and others frame a topical debate whose resolution remains an open research question.

2.7. *Public policy*

In his provocative book, Fuchs (1974) indicates that choices made by the government can influence who will die and from what causes by providing resources to pay for treatments in its various insurance programs and in its medical research priorities. Government influences health outcomes by way of public education, regulation of health depreciating activities and public programs (Grossman, 1982a). Governments also influence health outcomes in less obvious ways. Regulatory interventions increase the costs of the firm and thus affect individual income, and may also reduce individual incentives to invest in health by the provision of higher levels of safety (Viscusi, 1994). As noted by Newhouse (1987), about 40% of US expenditure on personal health care is provided by public programs, and for most other countries the figure is even higher.

In the United States, Medicare insures those over the age of 65, although since 1972 it has also covered the costs for those of any age who need kidney dialysis treatment or are disabled (Myers, 1981). Aside from this, most people under the age of 65 and most other diseases are not covered by government provided health care.[11] Many expensive medical procedures, considered somewhat routine, remain experi-

[10] We pursue issues raised in the related literature on the role of nutrition in augmenting human capital in developing countries in Section 3.

[11] Exceptions are people with limited income who are covered by Medicaid if they do not have private insurance and veterans.

mental and thus not covered under Medicare. Only very recently was cardiac transplantation added to the list of available procedures, albeit at a restricted list of medical centers. Since the mid-1970s it has been possible to protect individuals from the effects of hemophilia by providing the missing blood clotting agent. However, these shots, which cost about $20 000 per year, are not covered by Medicare.

Several investigators have explored trends between public health expenditures and life expectancy. For the US, 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. Comparable values for women are 15.9 and 18.9 years, respectively. Much of the gain occurred by 1975, suggesting a one-time innovation in senior care between 1960 and 1975 (Medicare). Fingerhut and Rosenberg (1982) demonstrated a substantial downward shift in age-specific mortality rates among the elderly beginning around 1968, and Preston (1984), in his presidential address to the Population Association of America, asserts that this drop in death rates corresponds with the introduction of the Medicare and Medicaid programs.

The complex relationship between government and social choices is illustrated by the health care financing arrangements of the United Kingdom. In the UK, the National Health Service supposedly provides care from cradle to grave, and yet according to Aaron and Schwartz (1984), many general practitioners in the UK routinely do not tell their patients over the age of 54 that treatments are available for renal failure and other illnesses. Despite evidence of such a failure to treat, life expectancy in the UK is nearly identical to that in the United States. Aaron and Schwartz also reveal what appears to be hospital-specific utilization differences in choices of equipment and medicine as well as note the relatively low share of GNP spent on health care in the United Kingdom.[12]

Vital statistics across the world are quite consistent with the hypothesis that a more socialized system of health care delivery may be consistent with increased life expectancy. Although these comparisons are difficult to make rigorous, paradoxically due in part to our own uniqueness in terms of a private delivery sector, the literature on US–Canadian comparisons supports the hypothesis that the Canadian system may deliver longer life due to a more egalitarian allocation of resources. Infant mortality and preventable deaths underlie much of the survival disadvantage of lower socioeconomic groups. Fuchs and Hahn (1990) have argued that the costs of such public health systems are lower than private systems in place in the US and have attributed this to economies of scale, particularly preferential pricing afforded to large government purchasing agents.

In the United States, the shift towards managed care has been quite dramatic; the percentage of individuals covered by these types of plans (e.g., Health Maintenance Organizations (HMO's)) has risen from 27.3% in 1988 to 61.9% in 1993 (Employee

[12] There is also a bias against treating non-life threatening problems. For example, hip replacements may require a wait of up to one year. Similar problems exist in Canada.

Benefit Research Institute).[13,14] Accompanying this shift has been a substantial drop in hospitalizations; in Southern California, Medicare patients covered under an HMO were hospitalized 56% as much as those remaining on the fee-for-service plan. These types of data are frequently mentioned as outcomes in discussions of life expectancy and cost containment (Bailit et al., 1995), although often neglected are discussions of possible selection biases inherent in an individual freely choosing HMO coverage as opposed to traditional fee-for-service (Clement et al., 1994).

Governments also influence mortality by targeting research expenditures. Most (about 90%) non-drug related research in the US is funded by the National Institutes of Health whose many institutes concentrate on specific diseases. In addition, the National Science Foundation, the Veterans Administration, and the various branches of the Armed Forces also participate in the decisions to allocate health research resources. Open research issues dealing with the political economy of health research resource distribution include whether this research has had an impact on mortality rates, who determines the level and mix of funding, and for what purpose are the choices made. With respect to the National Institutes of Health, Califano (1986: pp. 47–48), former Secretary of Health, Education, and Welfare, characterizes the situation as follows: "...Much of NIH's growth has stemmed from the congressional penchant for diseases-of-the-month and the shrewd assessment of the medical research establishment and its patrons that it would be easier to get money for tragic diseases, dramatized by real-life heart-wrenching cases, than for basic research identified as such...". He then goes on to describe the growth of institutes and their research funds. For example, the budget of the first NIH institute, the Cancer Institute, increased from $400 000 in 1937 to 1.2 billion in 1984. Currently it exceeds $2 billion a year.

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 next section we outline a structured microeconomic framework that provides explicit links between choice variables and those which can be arguably viewed as exogenous to the individual, and which can be used to estimate the parameters that characterize the structural model.

[13] Marder and Zuckerman (1985) have noted substantial geographical and temporal heterogeneity in the optimal scale of medical practices with scale economies evident during the period 1975–1980.

[14] Ashton et al. (1994) have also assessed the effects of aging of the veteran population on utilization rates in the Veterans Affairs medical system between 1980 and 1990. They note that the number of discharges increased by about 7% although it appeared that the utilization rate for older veterans actually declined due to shifts from hospital to ambulatory and long-term care settings.

3. Structural economic models of health

We now wish to outline microeconomic models of individual behavior which make explicit the causal links between mortality and morbidity, and 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.[15] 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.[16] 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 framework for determining health status is the demand model originally due to Grossman (1972a, b, 1975) and utilized in numerous studies (e.g., Wolfe and Behrman, 1984, 1987; Bartel and Taubman, 1986; Behrman and Deolalikar, 1987; Behrman and Wolfe, 1987). A related but distinct framework is provided by Rosenzweig and Schultz (1983a, b, 1985), Rosenzweig and Wolpin (1980, 1988), and Wolpin (1984).

The primary differences between the two frameworks are that the latter studies concentrate on obtaining consistent 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. Grossman-type models often have been used to estimate static reduced-form equations, sometimes dependent on previous choices so that the demand functions are conditional demand functions (Pollak, 1969, 1970). The endogeneity of some of the explanatory variables can be controlled 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. Yet, as in any such instrumental variable estimation, all the past choices that directly affect current health must be observed and instrumented to obtain

[15] One might question the assumption that an increasingly large segment of elderly consumers, those in nursing homes, could be viewed 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).

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

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. Moreover, if the instruments are not sufficiently correlated with the variable being instrumented, then measurement bias may just replace the simultaneity bias (Nelson and Startz, 1990a, b; Bound et al., 1993; Staiger and Stock, 1994).

The models outlined below treat health status as endogenous to the individual's allocation of time between work and leisure, and the individual's allocation among alternative consumption items. This general framework addresses the joint determination of health choices and labor supply decisions by way of both time and consumption allocations. A number of authors have addressed this seemingly obvious link and the selection bias caused by ignoring the endogenous sample-selection of hours worked and wage rates in the health equations (see for example, Bartel and Taubman, 1979, 1986; Passmore et al., 1983; Burkhauser et al., 1986; Mitchell and Butler, 1986). We first outline the one-period model of health, pointing out that certain dynamic issues can be pursued by allowing current prices to be dependent on a distributed lag of past prices. The dynamics that this brings into the model is not specified within the model per se but is rather a statement about the evolution of exogenous variables that is independent of the one-period model's own internal static nature. We then turn to the dynamic model of health production (Grossman, 1972a, b; Grossman and Benham, 1974) and the generalizations suggested by Muurinen (1982) and Wolfe (1985) as well as more general models that address such factors as rational addictions (Becker and Murphy, 1988; Becker et al., 1994), wage endogeneity (Foster and Rosenzweig, 1994), endogenous life expectancy (Ehrlich and Chuma, 1990), uncertainty, and intertemporal nonseparability. In our treatment of economic models of health we will also provide a selected review of studies which have pointed to the importance of key risk factors in determining health outcomes at the individual level.[17,18]

3.1. One-period models of health demand

In the one-period static model the individual's economic problem is to allocate time to leisure, T_i^L, time to the production of health, T_i^H, and financial resources in order to[19]

[17] Newhouse (1987) notes the tension between robust estimators and models, such as those we outline below, which may be highly leveraged on economic assumptions and data requirements, and thus may have questionable robustness properties in empirical implementation. He points out, in particular, that robustness of model estimates is compromised by the substantial skewness of health care expenditures for the elderly.

[18] We do not pursue extended models which deal with bequests, whose treatment could itself be a chapter (see, for example, Skinner, 1985).

[19] 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).

$$\max_{C_t^0, C_t^1, T_t^L, T_t^H} U(C_t^0, T_t^L, H_t), \tag{3.1}$$

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 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 $Y = WT^W$ 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

$$A_0 + [T - T_t^L - T_t^H - T_t^I(H_t)]W_t \geq P_t^0 C_t^0 + P_t^1 C_t^1. \tag{3.2}$$

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 using the Survey of Income and Program Participation (SIPP). Obviously, income and wealth for adults and the elderly is also influenced by whether they are in or out of the labor force. Such labor supply issues are given an exhaustive treatment in Chapter 16 by Hurd and are not pursued in this survey.

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[20]:

[20] 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 Kneiser (1988) and the latter by Feldstein (1967). He further points to the potential scope of stochastic frontier approaches for cross-section models (Aigner et al., 1977) and panel models (Schmidt and Sickles, 1984; Cornwell et al., 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.

$$H_t = h(C_t^I, T_t^H; X_t). \tag{3.3}$$

X_t will usually contain such variables as education, nutrition, occupation and physical activity, marital status, behaviors and lifestyle measures, and genetic factors. Heterogeneities in preferences across individuals can be accommodated by explicitly conditioning the utility function on it as well.

3.1.1. *Health risk factors at the individual level*

3.1.1.1. The role of human capital and other risk factors in adult health. Grossman considered the role of wages and of human capital (i.e., education and nutrition) in the demand for health. Wage increases permit the individual to substitute time devoted to health production which reduces sick time for leisure time, until the marginal utility of leisure time increases to the new wage. Human capital, felt by Grossman to have positive wage effects, was also thought to increase the marginal product of leisure. Human capital is augmented (proxied) by education, as it is often argued that the more educated make better decisions and can process new information more effectively. When income is not controlled, education may also serve as its proxy. Specific discussion of the endogenous role of income is considered shortly. A number of studies have found human capital and its various proxies to have important impacts on adult health. Silver (1972) used 1959–1961 age-adjusted mortality rates by sex and gender for Standard Metropolitan Areas (SMAs), and found an effect upon mortality for educational attainment, marriage, and income. Kitagawa and Hauser (1973) find that among the elderly, more educated females have lower death rates than females less educated, yet there are no similar findings for elderly males. Among those of younger ages, the more educated have lower age-specific death rates for both genders. Many potentially important variables were not controlled for in their analysis, and Rosen and Taubman (1984) have shown that this methodological approach may impart a bias if the null hypothesis is false. Grossman (1975) estimated logit survival models for 1955–1969, and found that survival is positively, and significantly, related to level of education, with each year of education lowering the probability of death by 0.4 percentage points. Further, he finds that ability, earnings, and job satisfaction have significant positive associations. Rosen and Taubman (1984) use the 1973 Exact Match Sample with Social Security mortality records updated through 1976, and examine the effects of education and other variables upon age-specific death rates. Using ordinary least squares regressions, which may be a questionable statistical method, they find a significant 23% lower death rate for college graduates than for those least educated, and correspondingly strong effects for family income. Wagstaff (1986) uses the 1976 Danish Welfare Survey to model health capital as an unobservable variable in a recursive dynamic structural model and in his analysis of life-cycle effects in health production and consumption points out that structural models, such as those discussed in Heckman and MaCurdy (1980), MaCurdy (1981, 1983), and Killingsworth (1983),

are essential in order to disentangle the high correlations between education and real lifetime wealth.

Women's schooling and its impact on adult health has been studied by Wolfe and Behrman (1984) and Behrman and Wolfe (1987) using adult siblings in Nicaragua. They find that control for common family background substantially changes the estimated impact of women's schooling on adult health as well as other outcomes. Strauss et al. (1993) also find strong effects of education on health for adults, consistent with their and others' findings with children. An important additional aspect of the Strauss et al. findings is that there are important gender differences between mortality and morbidity as indicators of well-being in the US and in developing countries. Feldman et al. (1989) use the National Health and Nutritional Survey (NHANES) data and the Kitagawa and Hauser study to examine trends in death rates by educational differentials and gender for the elderly, for the time periods of 1960 and 1971–1984. As with Taubman and Rosen (1982), Feldman et al. find much sharper declines over time for the more educated within each gender group, and these effects persist in their proportional hazard models even after controlling for such additional statistically significant risk factors as smoking, weight, hypertension, and high amounts of serum cholesterol. Land et al. (1994) combine Markov panel regressions with standard increment–decrement life-tables to estimate covariate effects using the Established Populations for Epidemiological Study of the Elderly (Duke University). Their methodology mitigates the standard problems in identifying covariate effects from small longitudinal panels. They find substantial and significant effects of higher education on both total life expectancy and active life expectancy (the period of life free from disability). Using data from the Health Promotion/Disease Prevention Supplement to the 1985 Health Interview Survey, Kenkel (1994) found that schooling improves individuals' choice of health inputs through improving health knowledge.

3.1.1.2. Nutrition. Human capital can also be augmented by better access to and knowledge about nutrition. Although the anthropometric issues taken up by Fogel in Chapter 9, which are largely devoted to the US and Europe, could be viewed as a general treatment of the human capital augmenting role that nutrition takes, we will spend a bit of time discussing how this important dimension of human capital has been treated in the extant literature on nutrition in developing countries. There is a growing literature focusing on developing countries which highlights the impact of anthropometric indicators of health and nutrition (and, to a lesser extent, direct morbidity reports) on productivity, income and wages, and which finds some effects even with efforts to control for reverse causality and unobserved heterogeneity (see, for example, Deolalikar, 1988; Behrman and Deolalikar, 1989; Haddad and Bouls, 1991; Strauss and Thomas, 1992; Schultz and Tansel, 1993; Foster and Rosenzweig, 1994; Strauss and Thomas, 1995). Strauss (1986) has shown links between health and nutrition in Sierra Leone and noted that measurement errors in both variables may cause a downward bias in their estimated effects on labor productivity. Utilizing community-level prices of food as an instrument for nutrition, Deolalikar (1988), Sahn and Al-

derman (1988), and Thomas and Strauss (1995) have found important impacts of nutrition on labor productivity in India, Sri Lanka, and Brazil, respectively. This literature illustrates the problems with looking at the impact of income on mortality/morbidity due to possible reverse causality and unobserved heterogeneity, the former of which might be even more important in developing countries. Many of these studies are surveyed in Behrman (1993) and Behrman and Srinivasan (1995).

3.1.1.3. Occupation. Perhaps no covariate more clearly illustrates the potential reverse causality between explanatory controls and health than does occupation and the reliance of certain occupations on the level of physical activity. Morris et al. (1953) examined the interrelationship between physical activity and coronary heart disease for drivers, conductors, and guards in the London Transport company in 1949 and 1950, and find conductors to have fewer coronary illnesses, speculating that this may be due to greater physical activity. They report similar findings when comparing postmen with other civil servants, admittedly with a possible selection bias for those who applied for and were accepted for positions involving more physical activity. Kitagawa and Hauser (1973) find that mortality among service workers exceeded that for agricultural workers by 80%, 1.37 compared with 0.76. These findings are somewhat specific to white workers, as they report no significant effect for non-white workers, implying that race may be acting as a proxy for occupational differences.

Sickles and Taubman (1986) estimated a model of healthiness (including death) and retirement using the Retirement History Survey and found that occupation (as well as various measures of income) is an important determinant of health. Gustman and Steinmeier (1986) analyze differences in retirement rates between blacks and whites and among more or less physically demanding jobs in the RHS. Black–white differences in retirement rates shrink with age and both health and prior occupation are important in the retirement decision. Jones and Goldblatt (1987) report that women who work outside the home have lower age-specific mortality rates than non-working women, with the lowest mortality rates found among women in relatively good socioeconomic standing who worked part time. Occupational mortality differences among men also were found, with rates the highest among laborers, miners, and construction workers, and lowest among professional workers, managers, and electrical workers. Wives of unemployed men also had death rates which were higher than wives of employed men. Burtless' (1987) study using the Retirement History Survey finds that (lifetime) employment in mining, construction, and as a labor operative, lead to worse health for the elderly, with morbidity effects greater than mortality effects.

Hayward et al. (1989) examine the influences of occupation on the nature and timing of retirement using data from National Longitudinal Survey of Older Men. They use a competing-risk hazard model with three types of exit from the labor force, retirement, disability, and death, and find that substantive complexity and physical demands of various occupations determine retirement decisions. Sorlie and Rogot (1990) estimate relative mortality risk by employment status based on life-tables for over

452 000 records in the National Death Index from 1979–1983. They find that unemployed men have standardized mortality ratios by race from 1.6 to 2.2 times those for the employed, with those unable to work having much higher ratios. Among the elderly, the employed had much lower standardized mortality ratios than those not working. Another longitudinal survey is that of Moore and Hayward (1990), who evaluated 3080 individuals from the National Longitudinal Survey who were 55 years or older. Their hazard function estimates indicate that mortality is reduced by different sets of job factors identified with the longest and most recent occupation.

3.1.1.4. Marital status. Marital and family status may be related to age-specific mortality for a variety of reasons, both positive (caring and companionship) and negative (stress and anxiety). Some of these effects may persist (habit formation) after the marriage has ended from death or divorce. Overall, the life-tables suggest the marriage effect to be positive, attributed to single persons leading a more dissolute lifestyle, to selection biases of persons in chronic ill health less likely to marry or stay married, and to early death due to grief on the part of widows or widowers. One might also envision negative influences due to family effects such as psychological and physical abuse or environmental hazards such as second-hand cigarette smoke. In the United States, Kitagawa and Hauser (1973) report that age-adjusted death rates maintained a consistent differential between married persons and their unmarried counterparts. Carefully correcting the data for reporting inconsistencies, they report that the mortality rate for unmarried white females was 17% higher than for married white females, and the rate for unmarried white males was 52% higher than for those married. The greater longevity and lower morbidity of married men is particularly well recognized. As Fuchs (1974) points out, "...In all developed countries, the unmarried have significantly higher death rates than the married and this differential is much greater for males than females: on the average unmarried males ages 45–54 in developed countries have double the death rate of their married counterparts. For females the marital status differential is only 30 percent...". The marriage benefit for males is, however, usually exceeded by the gender effect (Gove, 1973).

Taubman and Rosen (1982) analyze a sample drawn from the Retirement History Survey (RHS), and with models and methods similar to those discussed in Burtless (1987) based on self-reported health status, find that marital status significantly affects mortality, within and between periods, after controlling for education, family income, use of medical resources, and previous health. Their multinomial logit regressions were performed on compiled data (contingency tables), and inferences remain valid so long as the characteristics remain independent of all but random error (Goodman, 1968), criteria difficult to maintain as the number of characteristics increases. Jones and Goldblatt (1987) found that English widows had a 10% increased risk of death during the period 1971–1981, when comparing expected and actual death rates. Kaprio et al. (1987) report similar findings for both widows and widowers from Finland.

Ellwood and Kane (1990) have used the Panel Survey of Income Dynamics (PSID)

to examine mortality over a 35-year simulated time span for a cohort of 65-year olds. Their comprehensive study points to a number of conclusions, among them that not being married, as well as age and disability, are associated with higher death rates for males, and that being married also has positive effects on survival for women. Smith and Waitzman (1994) have rigorously evaluated the interaction among marital status, poverty and mortality, using the NHANES 25–74-year-olds from 1971–1975 who were successfully followed between 1982–1984, applying both additive and multiplicative relative risk models. They find support for the interactive nature of these risk factors for males and less so for females. A recent study which has found negative effects of marital status on the health of women due to domestic violence can be found in Tauchen et al. (1991).

Factors such as these have been labeled socioeconomic risk factors in the demographics literature, a term which, while providing a convenient encompassing taxonomy to differentiate these risk factors from the endowment related risk factors to which we next turn, has often clouded discussion on the causal links that may determine socioeconomic risk factors themselves. Given this caveat, however, there is a growing body of evidence from studies of age-standardized mortality ratios that points to either a lack of improvement in the relative mortality experience for those of low socioeconomic status (for the US experience see, e.g., Duleep, 1986, 1989) or a widening of socioeconomic differences in mortality (see, e.g., for the US and England, Rogot et al. (1992a, b), Pappas et al. (1993), Christenson and Johnson (1995).

3.1.1.5. Behavioral, lifestyle, genetic factors. We next focus attention on individual behavioral and lifestyle and related genetic factors which are associated with an elevated risk of mortality. For example, alcoholics are at increased risk of motor vehicle accidents and liver diseases such as cirrhosis and chronic active hepatitis, obesity predisposes many to diseases of the circulatory system, and AIDS is now the leading cause of death for young urban males in this country.[21] Fuchs (1974) provides a simple and forceful argument for the importance of lifestyle in health outcomes. He notes that Nevada has a similar physical environment to Utah. Mormons are prevalent in Utah and are admonished not to consume alcohol, coffee, or tobacco. Utah has uniformly lower age-specific mortality rates than in Nevada.

The extent to which certain lifestyle and behavioral risk factors can be modified is, however, unclear. Li et al. (1994) note the compelling evidence for a genetic marker which predisposes the individual to alcoholism. Obesity, well known in the medical literature to predispose the individual to heart disease, hypertension, diabetes and the like, all significant contributors to mortality, is known to be accompanied by genetic markers as well (Grilo and Pogue-Guile, 1991; Stunkard, 1991). With regard to male homosexuality, there is considerable debate in the literature. Hamer et al. (1993) published findings of a genetic marker for male homosexuality, although these findings have recently been challenged. Indeed, there is growing skepticism regarding this par-

[21] Waldron (1993, 1994) notes that the AIDS mortality gender gap is likely to narrow.

ticular genetic-behavior link, voiced by Billings and Beckwith (1993), Byne (1994), and Greenspan (1995). Moreover, current transmission mechanisms for AIDS may be driven as much by heterosexual behavior and by IV drug use as by homosexual behavior.

Smoking behavior has long been associated with increased mortality hazards. Doll (1953) and Doll and Hill (1952, 1964) used aggregated mortality statistics to find an increase in bronchial cancer which was attributed in part to cigarette smoking. In 12 years of British data on 41 000 men and women, cigarette smokers were found to have higher death rates from lung cancer. Similar findings during this period were by Dorn (1958). With the cooperation of the Veterans Administration, Dorn mailed a short questionnaire on smoking habits to 294 000 US veterans, selected in December 1953 as holders of US government life insurance policies who served in the armed forces between 1917 and 1940, and the response rate was an overwhelming 200 000 in 1954 and another 49 000 to a second mailing in January 1957. Dorn found substantial differences in death rates by smoking status, including 14 times as many smokers dying from emphysema as compared with nonsmokers during one 16-year interval. Additional studies using the original Dorn (1958) that have corroborated his findings include Kahn (1966), Rogot (1974), Rogot and Murray (1980), and Behrman et al. (1988, 1990). Smoking behavior was also studied by Paffenberger et al. (1966) using Harvard alumni who graduated between 1916–1950, and those from the University of Pennsylvania who graduated between 1931–1940, both admittedly highly selected populations. They found that heavy cigarette smoking, as well as high blood pressure and obesity, were associated with early deaths due to coronary heart disease. Using the same population Paffenberger and Williams (1967) examined the death rate from strokes, and found the same factors, as well as early parental death and nonparticipation in varsity sports, associated with higher probabilities of death from stroke. Kaplan et al. (1987), evaluate mortality hazards for tobacco usage and gender, for approximately 4000 people originally living in Alameda County, California. The sample members were at least 60 years of age in 1965, and were followed for 17 years.[22] Using Cox's proportional hazard model, they find an increased risk of early death from smoking.[23] Ford and DeStefano (1991) use a proportional hazard model on the NHANES data set for 13 164 persons aged 40–77, evaluating heart disease and overall mortality, controlling for diabetes mellitus. They find age, male gender, current smoking, hypertension, and inactivity associated with occupational status are significant mortality risk factors. Obesity was also significantly associated with mortality due to coronary heart disease. Manton et al. (1993) provide a summary of the medical literature on mortality from heart attacks, which account for approximately 1/3 of all deaths and whose frequency has declined from 250 (per 100 000) for men in 1968 to 110 (per 100 000) in 1988. They find evidence of reduced risk of heart

[22] About 5% of their respondents' deaths are unreported.

[23] They also found increased risk associated with being male, lack of leisure time, abnormal weight to height, and not normally eating breakfast.

attack for those who cease smoking, reduce serum cholesterol and high blood pressure, exercise more, and have a more ideal body weight.

Although smoking appears to be a well-documented mortality risk factor, modifications in such behavior, as presumably with other risk factors which result in premature death, has serious implications for actuarially-based pension systems such as Social Security. Shoven et al. (1988) have concluded that the precarious state of fundability of the Social Security System would be exacerbated if premature deaths by those who pay into the system but whose benefits are cut short by early death were to be reduced.

Genetic factors, which recently have been addressed in the growing twins literature, have confounding effects on health by way of their effect on behaviors and lifestyles. For example, observed earlier deaths of smokers could occur because underlying genetic factors influence both smoking choices and mortality, in which case the true causality between smoking per se and date of death may be questioned (Fisher, 1958). Similar genetic predispositions or endowments may affect schooling and/or income and, through such channels, morbidity and mortality. Genetic endowments were found to be significant determinants of returns to schooling in Behrman et al. (1980) and several recent twins studies (Behrman et al., 1994, 1995; Miller et al., 1995) also indicate such effects, even when controlling for random measurement error in schooling, a key variable in the Ashenfelter and Krueger (1994) study of a sample of twins who attended a convention in Twinsberg, Ohio. This latter study found that measurement errors, and not endowments, are the key to explaining downward biases in the economic returns to education.

Ideally, a study to test the hypothesis that a risk factor such as smoking serves as a proxy for unobserved heterogeneities would involve stratification of a large and randomly divided sample of individuals who were either instructed to smoke or not to smoke. Although such a protocol obviously could not be institutionally approved, there remains a group of more recent studies that may serve as a reasonable substitute, involving identical twins smoking discordant. Hrubec and Neel (1981) analyze a sample of white identical and fraternal twins born between 1917 and 1927 in the US, both of whom are veterans, and study rates of "early" death for the period 1946–1975. They find slightly lower death rates among fraternal and identical twins, as compared to all veterans of the same age, and they also find a greater concordance of death rates among identical than among fraternal twins, suggesting a role for genetic endowments. Notably, they include no measured covariates in their analysis. Floderus et al. (1988) study Swedish twins born between 1886 and 1925. Using pairs discordant with respect to smoking, both still alive in 1960, the relative risk of mortality over a 21-year period beginning in 1961 ranges from 1.7 to 2.3 for males and females, respectively, with trend by cohort which may reflect the type of left censoring we discussed above in the Dorn sample. Smokers' relative risks for death from coronary heart disease are also found to be higher. Kaprio and Koskenvuo (1990) use data on Finnish identical and fraternal twins to study deaths from lung cancer and coronary disease. In

a sample of 1278 smokers in 1975 and 1210 "former smokers" like-sex pairs born prior to 1958, with deaths recorded during the period 1976–1987, they find that the relative increase in risk of dying for the smoking twin was 13 in the case of identical twins and 2.4 for fraternal ones. Heavy smokers have even higher relative risks, whereas a former smoker had no excess mortality risk. The results of Akerman and Fischbein (1991), who also study Swedish twins indicate that twins are a population selected for risks for lower birth weight and birth complications, which results have implications for the health/developmental literature. Further, one must carefully distinguish between shared frailties and individual frailties. McGue et al. (1992) and Vaupel et al. (1992) show with Danish twins that a hypothesis of shared acquired longevity is inferior to one of moderate heredity with multivariate frailties, and Hougaard et al. (1992) note that the dependence of shared frailties using the Danish twins data is quite small.

Our review of the important risk factors entering the vector X in Eq. (3.3) has pointed to the often subtle role that such factors have in determining health outcomes as well as to the difficulty in treating all of these risk factors as purely exogenously determined. Our pursuit of formal derivations of the demand equations for alternative single period and multiple period structural models, however, does not focus on the determination of variables in X. More general treatments of the variables in X can be generated by rather straightforward modifications of the models we discuss below.

3.1.2. Demand equations for the one-period model

The solution to the one-period model can be based on the Lagrangian function where the budget constraint is Eq. (3.2) and where the health production function is directly substituted into Eq. (3.1). The Lagrangian multiplier (λ) is interpreted as the marginal utility of wealth. First-order conditions are:

$$\begin{aligned}
&U_{C_t^0} - \lambda P_t^0 = 0,\\
&(U_{H_t} - \lambda W_t T_{H_t}^1) h_{C_t^1} - \lambda P_t^1 = 0,\\
&U_{T_t^L} - \lambda W_t = 0,\\
&(U_{H_t} - \lambda W_t T_{H_t}^1) h_{T_{H_t}} - \lambda W_t = 0.
\end{aligned} \tag{3.4}$$

Note that in a one-period model $T_t^1 = f(H_{t-1})$ is a constant so that in this case $T_{H_t}^1 = 0$. 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 based on 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.

3.2. *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 et al., 1988; Hotz et al., 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.[24] 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.[25] 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

$$\max_{C_t^0, C_t^1, T_t^L, T_t^H} L = \sum_{t=1}^{LE} \beta(x)^{t-1} U(C_t^0, T_t^L, H_t) + \lambda(A_{t+1} - \gamma_t(A_t + (T - T_t^L - T_t^H - T^I(H_t))W_t - P_t^0 C_t^0 - P_t^1 C_t^1)), \tag{3.5}$$

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

$$H_t = h(C_t^I, T_t^H; X_t) + (1 - \delta_{t-1}(X_{t-1}))H_{t-1}, \tag{3.6}$$

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 lifetime utility that take the form of

$$\begin{aligned}
&U_{C_t^0} - \lambda_t P_t^0 = 0,\\
&(U_{H_t} - \lambda_t W_t T_{H_t}^I) h_{C_t^I} - \lambda_t P_t^I + \beta(1 - \delta_t)(U_{H_{t+1}} - \lambda_{t+1} W_{t+1} T_{H_{t+1}}^I) h_{C_t^I} = 0,\\
&U_{T_t^L} - \lambda_t W_t = 0,\\
&(U_{H_t} - \lambda_t W_t T_{H_t}^I) h_{T_{H_t}} - \lambda_t W_t + \beta(1 - \delta_t)(U_{H_{t+1}} - \lambda_{t+1} W_{t+1} T_{H_{t+1}}^I) h_{T_{H_t}} = 0,\\
&\lambda_t = \beta \gamma_t \lambda_{t+1}.
\end{aligned} \tag{3.7}$$

[24] 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.

[25] 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).

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.

3.2.1. 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.

3.2.2. Rational addictions

One may question the assumption that the rate of depreciation of health capital is exogenous since there are several choices we make, particularly the consumption of cigarettes and the excessive consumption of alcohol, which are known to negatively impact health status (see, e.g., Muurinen, 1982). In part to address this inconsistency, Becker and Murphy (1988) and Becker et al. (1991) formalized the theory of rational addictions, wherein individuals consume items such as tobacco, alcohol, or narcotics, whose future marginal utility is increased by current consumption. This model recently has been applied to cigarette smoking by Becker et al. (1994) who note that the long-run elasticity of demand with respect to price is high, and that the revenue increases to government from increased excise taxes on cigarettes may be smaller than thought, and by Chaloupka (1991, 1995). Recent applications to alcohol addiction can be found in Grossman et al. (1995).

Grossman (1972) partitioned consumption goods into those which produced health and those which did not. However, in the rational addictions model, a good such as cigarettes is considered a consumption good and a negative input into the health production function. Rational individuals smoke because they receive pleasure, despite the adverse health effects which can often be assumed known. The Grossman model can be modified to account for consumption of goods such as tobacco and alcohol, with adverse health effects by including a rationally addictive composite good, C^2, at price P^2, in addition to the composite consumption good, C^0, and the intermediate composite good, C^1. Consumption of the addictive good is accumulated into a stock of historical consumption (N_t) which is depreciated each period by a factor η_t. C^2 is a "good" in the utility function and a "bad" in the production of health.

We can incorporate these more general phenomena into the dynamic programming problem by restating Eq. (3.5) as

$$\max_{C_t^0, C_t^1, C_t^2, T_t^L, T_t^H} L = \sum_{t=1}^{LE} \beta(x)^{t-1} U(C_t^0, C_t^2, T_t^L, H_t, N_t)$$
$$+\lambda(A_{t+1} - \gamma_t(A_t + (T - T_t^L - T_t^H - T^I(H_t))W_t$$
$$-P_t^0 C_t^0 - P_t^1 C_t^1 - P_t^2 C_t^2)), \tag{3.8}$$

where the health stock is

$$H_t = h(C_t^I, C_t^2, T_t^H; X_t) + [1 - \delta_{t-1}(X_{t-1})]H_{t-1}, \tag{3.9}$$

the stock of addictive capital (see, for example, Chaloupka, 1991) is

$$N_t = C_t^2 + (1 - \eta_{t-1})N_{t-1}, \tag{3.10}$$

and the per-period intertemporal budget constraint is modified to account for the addition of the consumption of the addictive good (C_t^2). The resulting first-order equations from this dynamic program are

$$\begin{aligned}
&U_{C_t^0} - \lambda_t P_t^0 = 0,\\
&(U_{H_t} - \lambda_t W_t T_H^I) h_{C_t^1} - \lambda_t P_t^1 + \beta(1 - \delta_t)(U_{H_{t+1}} - \lambda_{t+1} W_{t+1} T_{H_{t+1}}^I) h_{C_t^1} = 0,\\
&U_{C_t^2} + U_{N_t} + (U_{H_t} - \lambda_t W_t T_H^I) h_{C_t^2} + \beta(1 - \eta_t) U_{N_{t+1}}\\
&\quad + \beta(1 - \delta_t)(U_{H_{t+1}} - \lambda_{t+1} W_{t+1} T_{H_{t+1}}^I) h_{C_t^2}^2 - \lambda_t P_t^2 = 0,\\
&U_{T_t^L} - \lambda_t W_t = 0,\\
&(U_{H_t} - \lambda_t W_t T_{H_t}^I) h_{T_t^H} - \lambda_t W_t + \beta(1 - \delta_t)(U_{H_{t+1}} - \lambda_{t+1} W_{t+1} T_{H_{t+1}}^I) h_{T_t^H} = 0,\\
&\lambda_t = \beta \gamma_t \lambda_{t+1}.
\end{aligned} \tag{3.11}$$

Solutions for the demand equations are obtained in the same fashion as above, albeit with the additional equation for the demand for addictive capital.

We next incorporate two endogeneities noted by Grossman, Muurinen, and Wolfe and others, but not yet formally addressed: endogenous wages and endogenous life expectancy.

3.2.3. *Wage endogeneity*

Foster and Rosenzweig (1994), in their analysis of moral hazard in the labor market for Philippino agricultural workers, recently assumed that wages were a known function of health capital (assumed in their study to be a measure of body mass), terms of employment, in particular the type of contract, and unobservable worker effort. Wage

endogeneity can be brought into the Grossman-type framework by defining a wage function whose arguments are job-specific human capital (E), health capital, endowments, and individual-specific heterogeneity. First, define the stock of job-specific human capital as

$$E_t = T_t^{\mathrm{W}} + (1-\nu_{t-1}(X_{t-1}))E_{t-1}, \tag{3.12}$$

where ν_{t-1} is the depreciation of last period's stock of work experience which could in principal be allowed to vary at the individual level.

Assume that the wage W_t is positive throughout life and given by

$$W_t = W(E_t, H_t; X_t). \tag{3.13}$$

Human capital is incorporated into the vector of endowments X through such variables as education and nutrition. Solutions to the individual's dynamic programming problem modified by endogenous wages of this form are:

$$\begin{aligned}
&U_{C_t^0} - \lambda_t P_t^0 = 0,\\
&(U_{H_t} - \lambda_t W_t T_{H_t}^{\mathrm{I}})h_{C_t^1} - \lambda_t P_t^1 + \lambda_t T_t^{\mathrm{W}}(W_{H_t} - W_{E_t} T_{H_t}^{\mathrm{I}})h_{C_t^1}\\
&\quad + \beta(1-\delta_t)(U_{H_{t+1}} - \lambda_{t+1} W_{t+1} T_{H_{t+1}}^{\mathrm{I}})h_{C_t^1}\\
&\quad + \beta\lambda_{t+1} T_{t+1}^{\mathrm{W}}(1-\delta_t) W_{H_{t+1}} h_{C_t^1} - \beta\lambda_{t+1} T_{t+1}^{\mathrm{W}}(1-\nu_t) W_{E_{t+1}} T_{H_t}^{\mathrm{I}} h_{C_t^1} = 0,\\
&U_{C_t^2} + U_{N_t} + (U_{H_t} - \lambda_t W_t T_{H_t}^{\mathrm{I}})h_{C_t^2} - \lambda_t P_t^2 + \lambda_t T_{W_t}(W_{H_t} - W_{E_t} T_{H_t}^{\mathrm{I}})h_{C_t^2}\\
&\quad + \beta(1-\nu_t)U_{N_t} + \beta(1-\delta_t)(U_{H_{t+1}} - \lambda_{t+1} W_{t+1} T_{H_{t+1}}^{\mathrm{I}})h_{C_t^2}\\
&\quad + \beta(1-\delta_t)\lambda_{t+1} T_{t+1}^{\mathrm{W}} W_{H_{t+1}} h_{C_t^2} - \beta(1-\nu_t)\lambda_{t+1} T_{t+1}^{\mathrm{W}} W_{E_{t+1}} T_{H_t}^{\mathrm{I}} h_{C_t^2} = 0,\\
&U_{T_t^{\mathrm{L}}} - \lambda_t(W_t + T_t^{\mathrm{W}} W_{E_{t+1}}) - \beta(1-\nu_t)\lambda_{t+1} T_{t+1}^{\mathrm{W}} W_{E_{t+1}} = 0,\\
&(U_{H_t} - \lambda_t W_t T_{H_t}^{\mathrm{I}})h_{T_{H_t}} + \lambda_t T_t^{\mathrm{W}}(W_{H_t} - W_{E_t} T_{H_t}^{\mathrm{I}})h_{T_t^{\mathrm{H}}} - \lambda_t T_t^{\mathrm{W}} W_{E_t}\\
&\quad + \beta(1-\delta_t)(U_{H_{t+1}} - \lambda_{t+1} W_{t+1} T_{H_{t+1}}^{\mathrm{I}})h_{T_t^{\mathrm{H}}}\\
&\quad + \beta\lambda_{t+1} T_{t+1}^{\mathrm{W}}[(1-\delta_t) W_{H_{t+1}}(1-\nu_t) W_{E_{t+1}} T_{H_t}^{\mathrm{I}})h_{T_t^{\mathrm{H}}}] - \beta\lambda_{t+1} T_{t+1}^{\mathrm{W}}(1-\nu_t) W_{E_{t+1}} = 0,\\
&\lambda_t = \beta\gamma_t\lambda_{t+1}.
\end{aligned} \tag{3.14}$$

The same algorithm for solving the control variables and the marginal utility of wealth can be used here as in the above problems. The only difference is that the endogeneity of the wage now has a formal link to the solutions of the structural model by way of the functional form chosen for the wage Eq. (3.13).

3.2.4. 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 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).

3.2.5. 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 adult 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 et al. (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^0_{t-1}, P^1_{t-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 to maximize

$$E_t\left(\sum_{t=1}^{LE}\beta^{t-1}U(C_t^0, T_t^L, H_t)\right). \tag{3.15}$$

The per-period intertemporal budget constraint is

$$A_{t+1} = \gamma_t(A_t + W_t T_t^W - C_t^0 - P_t^1 C_t^1), \tag{3.16}$$

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

$$T = T_t^L + T_t^W. \tag{3.17}$$

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

$$H_t = h(C_t^1, T_t^L; X_t) + \alpha a_t. \tag{3.18}$$

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 by 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 Eq. (3.18) 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

$$a_t = (1-\eta)a_{t-1} + H_{t-1}, \tag{3.19}$$

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 of the value function at time t:

$$V^t(A_t, a_t, W_t) = \max_{C_t^0, C_t^1, T_t^L} (U(C_t^0, T_t^L, H_t) + \beta E_t V^{t+1}(A_{t+1}, a_{t+1}, W_{t+1})). \tag{3.20}$$

The first-order conditions with respect to C_t^0, C_t^1, and T_t^L are

$$\begin{aligned} &E_t[U_{C_t^0} - \beta\gamma_t V_A^{t+1}] = 0, \\ &E_t[U_{H_t} h_{C_t^1} - \beta\gamma_t P_t^1 V_A^{t+1} + \beta V_a^{t+1} h_{C_t^1}] = 0, \\ &E_t[U_{T_t^L} + U_{H_t} h_{T_t^L} + \beta V_a^{t+1} h_{T_t^L} - \beta\gamma_t W_t V_A^{t+1}] = 0. \end{aligned} \tag{3.21}$$

Using the envelope theorem and the law of iterated expectations, the Euler equations can be rewritten as

$$\begin{aligned} &E_t[U_{C_t^0} - \beta\gamma_t U_{C_{t+1}^0}] = 0, \\ &E_t\left[U_{H_t} h_{C_t^1} - U_{C_t^0} P_t^1 + \beta h_{C_t^1}\left[\alpha U_{H_{t+1}} - [(1-\eta)]\left[U_{H_{t+1}} - \frac{U_{C_{t+1}^0} P_{t+1}^1}{h_{C_{t+1}^1}}\right]\right]\right] = 0, \\ &E_t\left[U_{T_t^L} + \frac{U_{C_t^0} P_t^1 h_{T_t^L}}{h_{C_t^1}} - W_t U_{C_t^0}\right] = 0. \end{aligned} \tag{3.22}$$

Moreover, if one assumes that expectations are rational, then one-period ahead innovations ($\varepsilon_{i,t}$) can be added to the derived Euler equations, where $E_t(\varepsilon_{i,t}) = 0$, $\varepsilon_{i,t}$, $i = 1,2,3$, 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

$$\begin{aligned} &[U_{C_t^0} - \beta\gamma_t U_{C_{t+1}^0}] = \varepsilon_{1,t+1}, \\ &\left[U_{H_t} h_{C_t^1} - U_{C_t^0} P_t^1 + \beta h_{C_t^1}\left[\alpha U_{H_{t+1}} - [(1-\eta)]\left[U_{H_{t+1}} - \frac{U_{C_{t+1}^0} P_{t+1}^1}{h_{C_{t+1}^1}}\right]\right]\right] = \varepsilon_{2,t+1}, \\ &\left[U_{T_t^L} + \frac{U_{C_t^0} P_t^1 h_{T_t^L}}{h_{C_t^1}} - W_t U_{C_t^0}\right] = \varepsilon_{3,t+1}. \end{aligned} \tag{3.23}$$

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.

3.3. Death as an exhaustive state

The economic model of health wherein life is not explicitly modeled as a choice variable can be used to express death as an exhaustive state for which the accumulated health stock fails to exceed a critical value H^* (Grossman, 1972a, b; Muurinen, 1982). Although such a mortality expression is conceptually linked to structural models they are typically estimated in isolation of that structure, that is, they are estimated as unrestricted quasi-reduced-forms. Rosenzweig and Schultz (1982b, 1983b) have argued that estimation of the structural model is valuable in its own right as well as for purposes of providing a set of restricted quasi-reduced-form estimates that formally link the structural parameters to the coefficients of the determinants of the mortality equation. This allows a much richer menu of ex ante questions to be addressed concerning the role and timing of economic factors on life expectancy.

The solution to the equilibrium path of individual health stocks can be linked to mortality by introducing a stochastic rule for observing death. Define a mortality state for individual i at time t as

$$\begin{aligned} M_{it} &= 1 \quad \text{if } H_{it} < H_{it}^* \ , \\ &= 0 \quad \text{otherwise,} \end{aligned} \tag{3.24}$$

where H_{it}^* is an individual and time-specific threshold for the equilibrium health index. In general the mortality state will be a function of prices, income, and endowments/heterogeneity. Let the probability that an individual is alive at the beginning of the period be $[1 - F(H_{it})]$ where $F(\cdot)$ is the distribution function and assume that the mortality threshold is a shock whose arrival time follows a Poisson process. Then the probability that a new value of H_{it}^* occurs during the period $(t, t + \Delta)$ is $P = \psi\Delta + o(\Delta)$.

The hazard of dying during the period is

$$\lambda_i(t) = \psi[1 - F(H_{it})] \tag{3.25}$$

and the survivor function becomes

$$S_i(t) = \exp\big(-\psi[1 - F(H_{it})]t\big). \tag{3.26}$$

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

$$\lambda_i(t) = \theta t^{\theta - 1} \exp(-H_{it}) . \tag{3.27}$$

Death defined in this fashion has several interpretations. In the health capital model, death might be defined as an infrathreshold value for any one of the several separable components to human capital, broadening the model to include issues such as preventable accidents caused by insufficient investment in health-education augmenting human capital and crime victimization as chosen on the basis of inadequate investment in social capital.

4. Sources of data on mortality and morbidity and health measurement systems

In our discussion of international mortality trends and risk factors we have pointed to particular studies, and often to particular data sets, used by researchers to study adult mortality and morbidity in countries other than the United States. In most of the industrialized countries, the health care delivery system is overseen by the federal government. Medical records data are thus centralized and are often used to conduct longitudinal mortality analyses. The earliest large-scale analyses were often conducted in Continental Europe. The United Kingdom is the source of many of the first working hypotheses concerning relationships between health and economic factors. Scandinavian countries are also the source of much data, including twins data which are crucial to many explorations of hypotheses involving the role of inherited mortality frailty. Canada frequently provides data of interest for North America. In this section, we wish to be more specific about the sources of death and mortality data, particularly those that exists in the US, in part because of the substantial body of literature that has been based on such data, including more rigorous attempts to construct better measures of quality of life, and in part because the survey instruments often used allow researchers to explore the life-cycle issues and formal structural models that we outlined in Section 3.

4.1. Mortality and morbidity data

The death certificate remains the official record of death and, since 1978, all US death certificates are maintained in a computerized file known as the "National Death Index". Decedents are identified by name, an identification number if available, overt demographic variables such as gender, assumed (or provided by family members) race, ethnic background, and one socioeconomic descriptor of occupation. Recently, the National Center for Health Statistics has examined the accuracy of the death certificate in the National Mortality Followback Study, calculating an effective matching rate ranging from 76% for veteran status to 86.6% for racial identity (Poe et al., 1993).

Kitagawa and Hauser (1973), in their important review of mortality differentials in the US, used the 1960 Matched Records Study of 340 000 deaths occurring over a four-month period in 1960, a collaborative effort of the Bureau of the Census, the Na-

tional Center for Health Statistics, and the Population Research Center of the University of Chicago. Kliss et al. (1979) examined the general issue of accuracy of matching procedures based upon Social Security number, name, race, sex, and gender, and estimate 80–90% accuracy, with change of surname for married women the principal source of error.

More recently, Sorlie and Rogot (1990) and Sorlie et al. (1992) match deaths records with those from the Current Population Survey (CPS), which is obtained monthly and has a significant amount of demographic and economic information, to match over 452 000 death records from selected months during 1979–1983. It is important to note that Census-based matched panels may not be complete. For example, it is generally agreed that there is a Census undercount of the poor and, particularly, the undocumented Hispanic population (Tienda and Ortiz (1986); see also Ericksen and Kadane (1985) and Schirm and Preston (1987) on generic undercount problems with the Census)).

Another data source for mortality studies is the Social Security files, which must accurately track deaths in order to stop payment of certain benefits and to commence with payment of others, such as widow and surviving-child benefits. Rosen and Taubman (1984) report these data to be accurate for deaths among those over 65, and incomplete for those younger. Duleep (1986) showed that by combining several Social Security administration sources of mortality information complete death reporting may be achieved.[26]

Several other more detailed surveys of demographic, socioeconomic and behavioral issues have been matched to deaths records or Social Security files. The Retirement History Survey is a random sample of about 11 000 heads of households age 58–63, first taken in 1969 and followed up every second year through 1979. Comparable surveys such as the National Health and Nutrition Survey (NHANES), which includes the National Health Epidemiologic Follow-Up Study (NLMS) and the National Longitudinal Mortality Study (NLMS), rely upon self-assessment and survey to provide deaths data (Madans et al., 1986). Unfortunately, recent privacy legislation compromises all Social Security number-based matching protocols.

A more selected, and yet extensive, panel of records on date and cause of death are kept by the Veterans Administration (VA). Studies by DeBakey and Beebe (1952), Beebe and Simon (1969) and Rogot (1974) have each concluded that the VA's death records are nearly 100% complete. It is essential to remember that the sample of veterans is highly selected, for health, intelligence, occupation, and other potentially important mortality factors.[27]

One of the more important panels for econometric evaluation is the Panel Study of

[26] Still one reads of occasional fraud such as a son cashing a dead parent's check.

[27] Interestingly, World War I data are less selected than subsequent military cohorts since various health effects could not be detected then, the US IQ test was only developed during the war, and the Army was mostly drafted.

Income Dynamics (PSID) which was begun in 1968 and annually follows an original sample of 5000 households, with an oversampling of the poor, nonwhites, and elderly. This panel is felt to be representative of US households, but until recently could not be used to study longitudinal patterns of aging since records were dropped when persons died or left the sample for other reasons. This problem has recently been addressed by the provision of a nonresponse sample for all people surveyed as well as information on the reason for nonresponse (Ellwood and Kane, 1990).

A follow-up to the Retirement History Survey, ended in 1979, is the Health and Retirement Study (HRS) which is detailed in Juster and Suzman (1995). The survey is be based on 13 500 individuals and 8000 families whose head of household is 51–61 in 1993 and provides information on labor force participation and pensions, health conditions and health status, family structure and mobility, and economic status, along with detailed information on expectations. These data are to be linked to the National Death Index, Medicare files, and Social Security earnings as well as to employer health insurance benefit plans and pensions. The first two annual waves have been collected and the third is in progress. The HRS, which already has been used to examine intergenerational transfers by McGarry and Schoeni (1994) and wealth differentials by ethnicity by Smith (1995a), among others, promises to be an exciting and comprehensive data source for new and seasoned researchers in the field of aging research.

The Asset and Health Dynamics Survey of the Oldest Old (AHEAD) is a new panel of heads-of-households born in 1923 or before as well as their spouses or partners and in its current early release form contains 7911 of the eventual 8224 respondents. The AHEAD collects detailed information on income, wealth, and health status, as well as information on the children of the respondents, such as schooling, income, family structure and financial transfers and has recently been used by McGarry and Schoeni (1995) in their study of intergenerational transfers. Smith (1995b) has recently used these last two new data sets to explore wealth inequality and savings incentives for different racial and ethnic groups and their implications on retirement behavior. The PAID, HRS, and AHEAD panels are collected through the Institute for Survey Research (ISR) at the University of Michigan. The ISR works with various governmental agencies in other data collection activities, including the Labor Department's Quality of Employment Survey and the Monitoring the Future survey.

The Survey of Income and Program Participation (SIPP) is one of the largest and most comprehensive panels of Census Data in the United States and is distinguished, in part, because of the detail of data collected on participation in government programs. The data collected include demographic identification, work hours and earnings, and participation in such programs as Social Security, Worker's Compensation, Aid to Families with Dependent Children (AFDC), Women, Infants and Children (WIC). The SIPP data are commonly the source of empirical welfare analyses, often on topics dealing with the elderly and the indigent. Several thousand individuals are interviewed by CPS field representatives each month on a rotating topic of interest. For example,

the March data are for income and are cited frequently. Del Bene and Vaughan (1992) note the improvements that could be made to these analyses were records from SIPP matched with Medicare files from the Health Care Financing Administration.

4.2. Measurement systems for health status

Arguably, there is no more active area of epidemiology currently underway than the reliable measurement of individual health status, underlying the emergent discipline of outcomes research which attempts to evaluate and compare empirically the results of various clinical interventions. There are objective health measurement systems proven to have prognostic capabilities for hospitalized patients. One such triage system is known as the Acute Physiology and Chronic Health Evaluation (APACHE) scoring system. Our interest instead lies in a reliable system for the measurement of health status for individuals who are not necessarily hospitalized and/or critically ill and one that lends itself to economic modeling. Health status measures are often developed by self-assessment, involving both objective questions as to functional status as well as subjective appraisals of perceived health status relative to others. Ferraro (1980) and Mossey and Shapiro (1982) have shown that subjective responses are highly correlated with their physicians' rating and the latter indicate that these subjective responses can actually surpass seemingly more objective findings, in terms of ability to predict and explain behaviors. Gafni and Birch (1991) provide adjustment algorithms for utility–health outcome measures detailed in Torrance (1986), which are based on the axioms of Von Neumann–Morgenstern utility theory, to correct for different valuations of health outcomes for different individuals or groups of individuals. These adjustments are meant to provide an equity neutral index of quality adjusted life-years based on individuals' preferences instead of more arbitrarily chosen weighting schemes. Idler (1992) argues that self-reported health status relative to others does not vary significantly with particular wordings of different survey instruments. Self-reported health measures in the United States and internationally have been found to have high degrees of reliability and validity by a variety of researchers. As pointed out by Strauss et al. (1993) these include the RAND health insurance study (Stewart et al., 1978; Ware et al., 1980), the RAND Medical Outcomes Study (Stewart et al., 1988), WHO surveys in Korea, Malaysia, and the Philippines (Andrews et al., 1986) and the ASEAN surveys in Indonesia, Malaysia, the Philippines, and Singapore (Ju and Jones, 1989).

4.2.1. Economic considerations in quality of life

Mortality studies which focus solely on the quantity of life in terms of years do not take into consideration the quality with which those years were lived. There are medical examples where quality adjustments appear warranted. Elderly individuals may

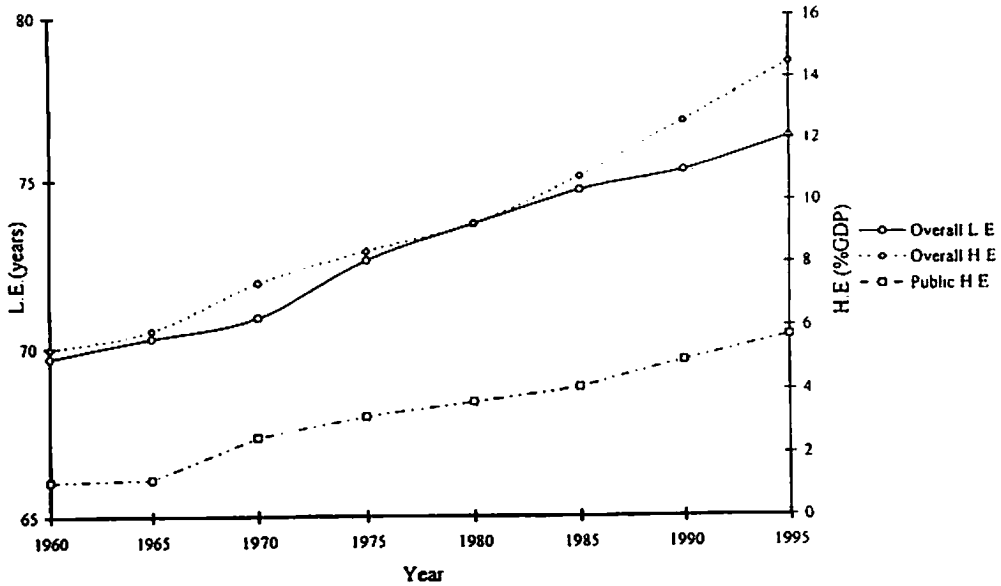

Fig. 9. Evolution of life expectancy and health expenditures (US).

elect to refuse life-prolonging therapies which are associated with severe toxicities or additional disabilities. While there are countervailing economic considerations in the case of what are referred to as "refusing medical advice" or "living wills", it would appear that a model of health in which individuals make choices in maximizing utility should account for the quality of the years of life as well as the quantity of such.

The importance of quantifying the quality with which periods of life are enjoyed, in addition to its intuitive appeal, can be motivated with recent US data. Shown in Fig. 9 are the evolution of overall life expectancy and overall health expenditures as a percentage of gross domestic product. Clearly, from 1960 until 1985, increases in health expenditures were met with increases in life expectancy. Since 1985, however, expenditures have increased much faster than life expectancy, and one might question whether these health expenditures have been valuable in terms of an increased quality of life.

Perhaps the first encounter most investigators have with quality/quantity issues concerns the reporting in the vital statistics of the "Years of (Potential) Life Lost" (YLL), which adjusts the condition-specific distribution of deaths to account for the age of the individual (Murray, 1994). Deaths past a certain average age expectancy, which is now taken to be about 75 years of age, are not counted as a loss under this measure. A more traditional economic measure, the "Years of Productive Life Lost"

Table 3
Causes of death and adjusted impact (US 1991)

Condition	Deaths		Expected years lost			Productive years lost			PDV earnings lost		
	Total	(%)	Total	(%)	Per death	Total	(%)	Per death	Total	(%)	Per death
Heart disease	721	(33)	3355	(24)	4.7	1340	(17)	1.9	32	(19)	90
Cancers	515	(24)	4275	(31)	8.3	1880	(24)	3.7	30	(18)	111
Cerebrovascular	144	(7)	535	(4)	3.7	223	(3)	1.6	4	(2)	72
Accidents, injuries	89	(4)	2800	(20)	31.5	2120	(27)	23.8	27	(16)	447
Chronic pulmonary	91	(4)	380	(3)	4.2	105	(1)	1.1			
Acute pulmonary	78	(4)	315	(2)	4.0	175	(2)	2.2			
Diabetes mellitus	49	(2)	365	(3)	7.5	165	(2)	3.4			
Hepatic, cirrhosis	25	(1)	418	(3)	16.7	233	(3)	9.3			
Suicide	31	(1)	938	(7)	30.2	678	(9)	21.9			
Homicide, legal	27	(1)	1153	(8)	43.5	893	(11)	33.7			
All others	400	(19)	(–684)	(–5)	(–1.7)	63	(1)	0.2	79	(45)	211
Totals	2170		13850		6.4	7875		3.6	172		153

(YPLL), is similar to the YLL with the exception that the productive (retirement) age cut-off age is 65 years.

Shown in Table 3 are the impacts of death by cause from the US life-table from 1991, where age-cohort deaths were collected at the age interval midpoint and adjusted impacts accordingly interpolated. Heart disease and cancer trade places between deaths and years of expected life lost, and perhaps most notably, whereas accidents and injuries accounted for only 4% of all deaths, they represented the leading cause of years of productive life lost.

4.2.2. Self-assessed health and mortality

Idler (1992, 1995), in her recent comprehensive reviews of the literature on self-assessed health status and mortality, proposes that subjective responses to questions soliciting self ratings of individual health status and health relative to others be complemented with "objective" controls for acute and chronic conditions, symptoms, functional status, physiology, and use of medical care.[28] Idler reviews the literature for 16 recent studies worldwide involving 49 516 subjects, followed for an (weighted) average of 6.3 years (Table 4). Self-assessed health is analyzed against mortality by computation of adjusted-odds mortality ratios, using methods of logistic regression or Cox or Weibull proportional hazards, with the superior response category receiving an odds ratio of 1.00. In Table 4, we have extracted from these studies their subjective response categories, source of objective findings, analysis technique and adjusted-odds mortality ratios by response category. Compiling weighted averages by converting response categories to a more comparable system of self-assessed health status as "excellent" "good" "fair" and "poor", we can compute the following survey estimate of the adjusted-odds mortality ratios by self-rated health status:

Self-rated health status	Adjusted-odds mortality ratios
Excellent	1.00
Good	1.82 ± 0.4
Fair	2.06 ± 0.6
Poor	2.87 ± 1.3

(16 studies, $n = 49\,516$)

Results appear to be quite consistent among studies and with respect to minor variations in the wording of the responses. Several other health status systems have been evaluated, as reviewed by Torrance (1986), by Donaldson et al. (1988), Nord (1992), and Murray (1994). Distinguishing these is a level of subjectivity associated with the

[28] Sole reliance on simple subjective health variables in the case of arthritis diagnosis was noted by Butler et al. (1987) to induce a systematic measurement bias towards better health for nonworking individuals.

Table 4
Self-rated health status and relationship with mortality (Idler, 1995)[a]

Authors	Year	Cohort	*n*	F/U (years)	Analysis	Objective and controls		Adjusted-odds ratios		
								Good	Fair	Poor
Mossey and Shapiro	1982	E Canada	3128	6	LogReg	MD, self	Socio	1.4	2.0	2.8
Kaplan and Camacho	1983	A US	6928	9	LogReg	Self	Socio, psych			1.9
Kaplan et al.	1988	E Israel	1078	5	CoxPH	Self	Age, sex	1.1	1.9	2.1
Jagger and Clarke	1988	E England	1203	5	CoxPH	Self	Socio		1.3	2.1
Idler et al.	1990	E US	5909	4	LogReg	Int, self	Socio, health	1.9	2.5	4.0
Idler and Angel	1990	A US	6440	13	CoxPH	MD	Socio, health	1.9	2.2	2.8
Idler and Kasl	1991	E US	2812	4	Cox, LR	Int, self	Socio, econo	2.8	3.4	4.9
Ho	1991	E Hong Kong	1054	3	LogReg	Self	Socio, health			2.0*
Wannamethee et al.	1991	A England	7725	4	LogReg	Self	Socio, health		1.4	
Wolinsky and Johnson	1992	A US	5151	4	LogReg	Self	Socio, health		1.9	2.2
Shahtahmasebi et al.	1992	E Wales	534	8	Weibull	Self	Socio, psych	1.5	2.0	2.4
Pijls et al.	1993	A Netherlands	783	5	CoxPH	MD, self	Socio, health	1.3	2.4	5.4
Tsuji et al.	1994	A Japan	2651	3	CoxPH	Self	Socio		2.2	3.1
McCallum et al.	1994	A Australia	1050	7	CoxPH	Self	Socio, psych	1.3	1.7	2.4
Schoenfeld et al.	1994	A US	1192	3	LogReg	Int, self	Socio, health	1.4	2.0	2.8
Dasbach et al.	1994	A US	1878	8	CoxPH	MD, self	Age, sex			1.7*
Weighted averages (standard deviations)			49516	6				1.8(0.4)	2.1(0.5)	2.8(1.3)

[a]Cohort age A = adults, E = elderly; objective Int = interviewer; (*) conversion "health relative to others" same = fair, worse = poor.

Table 5
Rosser and Watts disability–distress classification

Index	Disability
1	No disability
2	Slight social disability
3	Severe social disability and/or slight impairment of performance at work. Able to do all household work except very heavy tasks
4	Choice of work or performance at work severely limited. Housewives and old people able to do light housework only, but able to go out shopping
5	Unable to undertake any paid employment. Unable to continue any education. Old people confined to home except for escorted outings and short walks and unable to do shopping. Housewives only able to perform a few simple tasks
6	Confined to chair or to wheelchair or able to move around the home only with support from an assistant
7	Confined to bed
8	Unconscious

(Self-valuations) Disability index	Level of distress			
	None	Mild	Moderate	Severe
1	1.000	0.995	0.990	0.967
2	0.990	0.986	0.973	0.932
3	0.980	0.972	0.956	0.912
4	0.964	0.956	0.942	0.870
5	0.946	0.935	0.900	0.700
6	0.875	0.845	0.680	0.000
7	0.677	0.564	0.000	(−1.486)
8	(−1.028)			

Source: Kind et al. (1982).

weights of functional status of the respondents. Perhaps one of the more interesting systems is the Rosser–Watts system (Table 5) which is discussed in Kind et al. (1982). For this system two of the (disability, distress) states, namely (unconscious,...) and (confined to bed, severe distress), are associated with a lower utility than death itself. Apparent contradictions between lifetime utility maximization and the deliberate choice of death by suicide or a living will may be rationalized within the Rosser–Watts system.

There are a number of examples of the use of health status measurement in the study of the determinants of mortality using the Retirement History Survey. Hausman and Wise (1985) study the effect of health, earnings, and Social Security payments on the retirement hazard. Their results indicate that increases in Social Security benefits between 1969 and 1975 resulted in a 3–5% increase in the probability of male retirement for those aged 62–66, while higher real earnings reduced the probability of re-

tirement. Incentive effects of benefit cuts were also found in Fields and Mitchell (1984). Anderson and Burkhauser (1985) treat mortality and retirement as a jointly determined process and estimate the bivariate interaction effects of health and retirement. They find that health and retirement are jointly determined and that the use in empirical analysis of self-assessment measures of health instead of more objective measures exaggerates the influence of health on labor withdrawal and underestimates the importance of wages. Bazzoli (1985) focuses on the effect of a variety of health variables including a new self-evaluation of health status measures which she develops. She finds that previous studies had overestimated the effect of health on retirement and that pension and Social Security income have a larger impact on retirement than previously reported. Burtless (1987) analyzes the health status of 8131 males from the 1969–1979 waves of the RHS. Respondents were asked three self-reported questions as to health status: handicap or physical disability, relative health compared to others their own age, and the extent to which working was limited by health. Categorical responses to these three questions were ranked, and two multiperiod probit models of death were estimated by a maximum likelihood method (Butler and Moffitt, 1982). Burtless evaluates and estimates two models of death – as a continuation of subjectively reported health status below that of the lowest level reported by the respondents, and as an objective determination from deaths records. Each model is parametric for a normal distribution fit so that cumulative values (cross-sections) are consistent with the categorical responses as characteristic of an underlying continuum of health status. Burtless' findings are sensitive to the precise models of health status and death applied, particularly the relationship between occupational effects and mortality. Bound (1991) looks at the sensitivity of labor supply models to different health measures. He develops and estimates a model that incorporates self-reported measures of health and an objective measure (mortality) and finds that both measures result in biases but that the biases are in opposite directions.

In order to adjust the years of life to account for the health status, one can employ the disability- (or quality-) adjusted life-year (DALY), which relies on a determination of the health status measured for the individual during the year. Such a disability adjusted life-year measure was recently used by the World Bank (1993) in their comprehensive international study of shifts in mortality patterns and their policy implications. The quantity of life can then be defined as the sum of these year-states. Adjustments of years of life to DALY are not without ethical controversy, as reviewed by Busschbach et al. (1993) who note the work of Harris (1987) and Donaldson et al. (1988). The latter two studies contend that these adjustments result in evaluation of health care outcomes extremely unfavorable to the elderly population.

Developed by Robert Kaplan and John Anderson,[29] the Quality of Well-Being (QWB) index combines four measurement scales: mobility, physical activity, social activity, and symptom/problem complexes, building upon a body of theory in econom-

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

Table 6
Quality of well-being index (Kaplan and Anderson, 1988)

Step number	Index definition	Weight
Mobility		
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, 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		
1	No limitations for health	0.0
2	In wheelchair, controlled movement of wheelchair without assistance; had trouble to lift, stoop, or use stairs or inclines; or limped, used a cane or had any other limitation in walking, health related	–0.060
3	In wheelchair, did not move or control the 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		
1	No limitations for health reasons	0.0
2	Limited in other (e.g. recreational) role activity, health related	-0.061
3	Limited in major (primary) role activity, health related	-0.061
4	Performed no major role activity, health related, but did perform self-care activities	-0.061
5	Performed no major role activity, and did not perform or had more help than usual in performance of one or more self-care activities, health related	–0.106

ics, psychology, medicine, and public health.[30] In order to develop QWB, they evaluated the effects of disease and injuries upon behavior and role performance, constructing scales representing three aspects of daily functioning: mobility, physical activity, and social activity (Table 6), including subjective complaints by adding a symptom/problem complex. QWB reports a value between 0 and 1, defined as

$$QWB = 1 + CPX + MOB + PAC + SAC, \tag{4.1}$$

where *CPX* is individual's "score" on the Symptom/Problem scale, *MOB* is the Mobility scale, *PAC* is the Physical Activity scale, and *SAC* the Social Activity scale. Weights were estimated by data from a survey of at least eight measured days for each of 1025 subjects,[31] and these estimates have subsequently been tested across subsam-

[30] Rust (1990) analyzed the explanatory power of up to 75 health-related questions in the RHS and found that such health constraints on mobility as well as the subjective/objective four state health polytomy used by Sickles and Taubman were most relevant in explaining mortality hazard and the labor/leisure trade-off.

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

ples of the population. Sickles and Yazbeck (1995) augmented the QWB index with mortality information in the RHS in estimating the dynamic model life-cycle, Eqs. (3.15)–(3.19).

4.2.3. Aging, disability, and health status

The health status measures that we have discussed above attempt to deal in particular ways with the quality of life as mortality approaches. In attempting to quantify the quality of life in the later years, health measurement systems generally result in an index of health that is declining in age. The presence of a disability, which may also result in the placement of the individual in a nursing home, typically plays a relatively large role in weighing down health index measures. Moreover, the presence of an individual in a nursing home may also put in question the extent to which such individuals may be able to make decisions in accordance with the rational decision-making assumptions pursued in the economic models of health-augmenting allocations. A recent report by the Institute of Medicine (1990) pointed out that there was substantial scope for reducing disability in the elderly by better utilization of existing medical knowledge while Wolfe and Haveman (1990) and Stoto and Durch (1993) have noted that disability is not only becoming a more accepted condition but that better financial status has permitted an increasing number of elderly to identify themselves as disabled.

A number of studies have focused on issues such as these. Poterba and Summers (1987), using the NHIS, indicate that, between 1963 and 1982, the percentage of the elderly in nursing homes rose sharply for each age group 65–74, 75–84, and 85-and-older. For men aged 65–74, those residing in a nursing home rose from 0.68% to 1.23%, and for women 85+, from 17.5% in 1963 to 22.7% in 1982, although as pointed out by Garber and MaCurdy (1990) since the duration of stay for the high-risk elderly is relatively short, this segment of the elderly population may not be the heaviest user of long-term care. Another measure of wellness being demand for medical care, Poterba and Summers were able to show that Medicare expenditures rise sharply with age, slightly higher for women. For those aged 65–74, there was a 10% increase in restricted-activity days between 1961 and 1980, with no increase for those over 74. Multinomial analysis revealed remaining life expectancy to be significantly related to a restricted activity level. The financing of elderly long-term care is, of course, an issue seen by many as an example of market failure (see, for example Pauly, 1986; Gravelle and Taylor, 1989; Wolfe, 1993).

Manton et al. (1993) use the National Long Term Care Survey to study health utilization and mortality for about 20 000 elderly persons over the period 1982–1984. This sample initially contacted 33 000 people from Medicare's Health Insurance Master File, in order to determine if they were disabled in 1982. All of the disabled, and a subset of the non-disabled, were resurveyed in 1984. Corder and Manton examine (1) mortality and disability linkages, (2) death rates among the institutionalized, and (3)

changes in patterns of mortality after a hospital stay. They find mortality rapidly increases with the level of disability and as the disabled age. Crimmins and Pramaggiore (1988) look at patterns of change in health and labor force participation between 1969 and 1981 and found no indication of improved health between 1970 and 1980 among men aged 56–64, but did find evidence of deteriorating health among the retired. Crimmins et al. (1989) update and extend the work of Poterba and Summers (1987). Using the NHIS and various Census sources, they find that for females age 65 years, 17.0 years of the expected remaining life of 18.4 years were spent outside any institution, that for 15.3 years of these years they could perform their major activity, and that for 9.3 of these years they had no long-term disability. At age 85, more than 65% of women's expected remaining life would be spent in a non-institutionalized status.

There has also been work on the implications of the disability system for labor force participation. Parsons (1980a, b) used a model of a worker's decision process under uncertainty to study the decline in male labor force participation in the US. He found evidence to confirm the hypothesis that labor force withdrawal was induced by the rapid expansion of welfare alternatives to work, principally the Social Security disability program. Fenn and Vlachonikolis (1986) develop a model of the labor force participation for the disabled using maximum likelihood techniques with a switching regression model and corrections for self-selection. Their data are taken from the 1976 Social and Community Planning Research unit in Oxford. Labor force participation varies by age, disability, and income in reduced-form equations estimated with logit methods. Holden (1988) studied the effects of ill health and employment in physically demanding occupations in the year before receipt of benefits. In the New Beneficiary Survey, employment in physically demanding jobs is associated with lower probability of work during retirement and having a work-limiting health condition decreased the probability of work. Sickles and Taubman (1986) use part of the Retirement History Survey to estimate a model of healthiness and retirement. They do not use a life-cycle model. They find a trade off in retirement between Social Security benefits and wage rates and a sharp decline in retirement before age 62, the earliest date non-disabled men are eligible for Social Security benefits. They also find that health is worse for those whose longest occupation is unskilled labor.

Liu et al. (1990) analyze 2123 participants in the Framingham sample who are 55 years or older and estimate Cox proportional hazard models over a period spanning ten years. These volunteers took a battery of eight neuropsychological tests, and Liu et al. find cognitive disparity associated with higher neuropsychological hazards, controlling for age and education. Those with marginal and poor cognitive skills had a relative risk of death of 1.37 and 1.66 respectively compared to those with no impairment in cognitive skills. Ellwood and Kane (1990) study death rates in the Panel Survey of Income Dynamics, a stratified random sample begun in 1969, with an overrepresentation of blacks. Focusing upon those people whose age was over 65 for at least three years, Ellwood and Kane find that disability, as well as marital status and age, is associated with higher death rates for both men and women, with the effect

reversed for woman (marriage was negative for survival). Chirikos and Nestel (1991) study the extent to which occupation and impaired health explain variations in the functional capacity of older men to delay retirement. They use a competing risk framework of retirement, work disability, and death. The parameters of the hazard rate models are estimated with panel data covering a period of 17 years from the National Longitudinal Survey (NLS) of Labor Market Experience of Older Men. They find that health-related physical conditions play an important role in determining the ability of male workers to delay retirement.

The disincentive effects of programs such as the Social Security Disability Insurance program on labor force participation of older men has been studied by Bound and Waidman (1992) who extended the mostly cross-sectional studies of Leonard (1979), Parsons (1980a, b), Haveman and Wolfe (1984), Slade (1984), and Haveman et al. (1991). Building on his earlier work on identifying exogenous cross-sectional variations in replacement rates (Bound, 1989; Bound and Waidman, 1991), Bound examined these causal links using the National Health Interview Survey and found that between 1949 and 1987, as the program was substantially expanded, 25–33% of the 19.9% who dropped out of the labor force moved into the disability program. Recently, Waidmann et al. (1995) have pursued this line of research and have found, again with the National Health Interview Survey, that self-reported health limitation responses and labor force participation are jointly determined.

5. Statistical and numerical techniques

This section outlines developments in statistical and numerical treatments of mortality and morbidity modeling. The two broad classes of models are those which examine the duration of life or competing risks therein and those which model the healthiness of an individual.

5.1. Life-tables and mortality hazards

Life-table analyses of mortality are based upon 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.[32] 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

[32] For excellent surveys of survival model methods see Kalbfeisch and Prentice (1980), Kiefer (1988), and T. Lancaster (1990).

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., 1992b). 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 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[33] which, if ignored, can also bias estimates based on structural models of health. 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).

Assume that time is continuously distributed and that individuals at time t are at instantaneous hazard of death $h(t)$. Certainty of death requires that the hazard function integrate to 1, and senescence requires that the hazard function be increasing. Denote by $s(t)$, the survival function at time t, that proportion of the population surviving until time t, which is equal to 1 minus the integral of the hazard function from time 0 until time t. Let t_i be event time for individual i and assume that life-table data are to be compiled over the interval $(t_i, t_i + \Delta t_i)$. A life-table constructed for this interval is expected to report a death rate $D(t)$ equal to the expectation of $h(t)$ over $(t_i, t_i + \Delta t_i)$, multiplied by Δt_i:

$$D(t_i, t_i + \Delta t_i) = \int_{t_i}^{\Delta t_i} h(t)\, dt \tag{5.1}$$

and the observed survival fraction at $t_i + \Delta t_i$ should represent $s(t_i + \Delta t_i)$. Estimates of the survival function (often observed to be exponential and/or logarithmic) and the

[33] 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).

hazard function can be gotten from the Kaplan–Meier product limit estimator (Kaplan and Meier, 1958).

Aggregate data are not sufficient to test the null hypothesis of independence of risk factor status and risk of death, and cross-sectional analyses extend the analytical techniques of the demographer to the study of chosen behaviors. Recently, Foster (1991) develops a model wherein autocorrelation in cohort mortality rates can be used to estimate the extent of heterogeneity among the population, without need for strong assumptions of the distributional form of such heterogeneities, or of the hazard function itself.

Life-table analysis is distinguished by the fact that the data are inherently cross-sectional; a collection of anecdotes, if you will, of ordered pairs of state and survival status. With a null hypothesis of independence between risk factor status and risk of death, life-table analyses make a critical assumption of separability of the population into risk factor cohorts. Heterogeneities which may undoubtedly underlie particular survival advantages must remain consistently distributed within the surviving population, a questionable proposition. Human beings and their existences are complex phenomena, and exhaustive models are problematic. Inference may thus be compromised by bias associated with the classification system, and thus one cannot preclude risk status as an instrument or proxy for unobserved heterogeneities. As an example of the limitations of cross-sectional data to provide inference, consider the possible relationship between income and life expectancy. In the life-tables ordered pairs of data such as income status and life expectancy are analyzed. One causative hypothesis might be that those with higher incomes are less constrained in choices reflected in increased survival. However, the same ordered pairs of data would necessarily be consistent with the converse hypothesis that those endowed for longer survival have a choice advantage in intertemporal substitution reflected in higher socioeconomic status. Further, these same data would also be consistent with a non-causation hypothesis that socioeconomic status and life expectancy are proxies for underlying genetic endowments. Goldman (1993) has recently demonstrated the importance of this point. Using a straightforward "matching" model for marriage, she shows that cross-sectional data cannot be used to draw inference about the marriage pattern of reduced mortality as it is either reflective of a selection bias or a causal factor (e.g., a care giving factor). She proposes that the many studies which have so proceeded may be methodologically flawed.

5.2. *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; T. Lancaster, 1990). The hazard for T is the conditional density of T given $T > t$ and is given by

$$\lambda(t) = f(t|T > t) = \frac{f(t)}{[1 - F(t)]} \geq 0. \tag{5.2}$$

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

$$f(t) = \lambda(t) \exp\left[-\int_0^t \lambda(\tau)\, d\tau\right], \tag{5.3}$$

$$F(t) = 1 - \exp\left[-\int_0^t \lambda(\tau)\, d\tau\right]. \tag{5.4}$$

Let $\delta = 1$ if the duration is right-censored and $\delta = 0$ otherwise. 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

$$\ln L = \sum_i f(t)(1-\delta) + \sum_i [1 - F(t)]\delta. \tag{5.5}$$

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 specification of mortality (see, for example, Rosenzweig and Schultz, 1983b) 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).

5.2.1. Proportional hazard model

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 of length of life as a linear function of covariates, $\ln(T) = x\beta + \sigma\varepsilon$, where ε is a random disturbance and σ is a scale parameter. Failure time can be written as $T = \exp(x\beta)T_0^{\sigma}$ where T_0 is an event time drawn from a baseline distribution. Different parametric distributions are available to model unobserved genetic frailties $[\theta(t)]$. Also available are semiparametric estimators in which $\theta(t)$ is either factored out of the likelihood using Cox's partial likelihood (Cox, 1972) or is estimated by a finite support density estimator (Heckman and Singer, 1984). For parametric approaches, the normal distribution has an obvious genetic rationale and is parsimonious. The inverse Gaussian provides a quite flexible mixture (Manton et al., 1986), and allows for a very general description of biological risks.

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

$$h(t_i \mid x_i, \theta_i) = \exp(\gamma \ln t_i) \exp(x_i\beta + \theta_i). \tag{5.6}$$

The log hazard function is given by

$$\ln h(t_i \mid x_i, \theta_i) = \gamma \ln t_i + x_i\beta + \theta_i, \tag{5.7}$$

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

$$T_i = \min(t_i, t_c), \quad d_i = I(t_i < t_c), \tag{5.8}$$

where t_c is the censored time of an incomplete spell and I is an indicator function: $d_i = 1$ if $t_i < t_c$ and $d_i = 0$ otherwise.

Assuming independence over duration spells, the joint likelihood of duration times and unobserved heterogeneity is

$$L = \prod_i f(t_i, \theta_i \mid x_i), \tag{5.9}$$

where

$$f(t_i, \theta_i \mid x_i) = h(t_i, \theta_i) \exp\left[\int_0^{t} h(s_i, \theta_i \mid x_i)\, ds\right], \quad \text{if } d_i = 1, \tag{5.10}$$

$$f(t_i, \theta_i \mid x_i) = \exp\left[\int_0^{t} h(s_i, \theta_i \mid x_i)\, ds\right], \quad \text{if } d_i = 0. \tag{5.11}$$

The joint density is

$$f(t_i, \theta_i \mid x_i) = g(t_i \mid x_i, \theta_i)\mu(\theta_i) \tag{5.12}$$

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

$$L = \prod_i \int_0^t g(t_i \mid x_i, \theta_i)\mu(\theta_i)\, d\theta_i. \tag{5.13}$$

This likelihood function is a common form of the statistical mixture model. Control for $\mu(\theta)$ parametrically requires a distribution for θ. However, an incorrect parameterization of $\mu(\theta)$ leads to estimation bias in both duration dependence effects and the parameters of included variables (Heckman and Singer, 1984).

5.2.2. *Competing risk model*

The use of single cause duration models for mortality modeling implicitly aggregates all causes of death into a single cause measure and thus may impart a misspecification due to differential risk factor effects for different types of diseases which ultimately cause death. Given the dynamic cause of death structure in the US (Manton and Stallard, 1982) this aggregation is problematic. Recently, Stoto and Durch (1993) have pointed out that age-adjusted death rates for 14 of the 15 leading causes of death in the US for 1950–1990 indicate that trends in particular competing risks of death are substantially different. The single cause duration model extends to competing risk models for cause of death (Cox, 1962; Tsiatis, 1975; Basu and Ghosh, 1978; Yashin et al., 1986). Consider two competing risks, each with a Weibull proportional hazard of mortality. Let t_1 be failure time were the first risk factor the only one present and let t_2 be failure time were the second risk factor the only one present. The density function associated with t_j, $j = 1,2$, takes the form

$$\begin{aligned} f(t_{ji}, \theta_i \mid x_i) &= h(t_{ji}, \theta_i)\exp\left[-\int_0^{t_{ji}} h(s, \theta_i \mid x_i)\, ds\right], && \text{if } t_{ji} < t_{jc}, \\ &= \exp\left[-\int_0^{t_{jc}} h(s, \theta_i \mid x_i)\, ds\right], && \text{otherwise.} \end{aligned} \tag{5.14}$$

Assume for the moment that mortality is due to risk factor 1 and that correlation between conditional proportional hazards for t_1 and t_2 is due to shared individual specific frailties, i.e. θ_i is common to the specifications of the two hazard functions for individual i. We observe a completed spell for t_1 (t_1^*) and calculate the contribution to the likelihood as $\Pr[t_2 > t_1^*, t_1^*]$, where

$$f(t_{1i}, t_{2i}, \theta_i \mid x_{1i}, x_{2i}) = g(t_{1i} \mid x_{1i}, \theta_i)\mu(\theta_i)g(t_{2i} \mid x_{2i}, \theta_i)\mu(\theta_i). \tag{5.15}$$

If the correlation between the risk factors is due to the shared frailty θ_i then $\Pr[t_2 > t_1^*, t_1^*]$ is calculated by first integrating out the heterogeneity, either by specifying the heterogeneity distribution parametrically or by using a finite support histogram method of Heckman and Singer, and then calculating the probability of observ-

gram method of Heckman and Singer, and then calculating the probability of observing t_1^*. Heckman and Honoré (1989) and Han and Hausman (1990) have shown that even if x_1 and x_2 are identical, the competing risk model is identified so long as at least two covariates are continuous. Heckman and Taber (1994) have recently generalized these identifiability conditions. These results are quite important in light of the non-identifiability results of Cox (1962) and Tsiatis (1975) when covariates are not used.[34]

5.2.3. Semi-nonparametric method

Heckman and Singer's *nonparametric maximum likelihood estimator (NPMLE)* can be used to avoid the ad hoc specification of the mixing distribution $\mu(\theta)$ (Robbins, 1964; Laird, 1978; Heckman and Singer, 1982, 1984; Lindsay, 1983). 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 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 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 examine the piecewise smooth estimation of an unknown density function after adding a term which penalizes unsmooth estimates:

$$\log L = \sum_{i=1}^{N} \log f(x_i) - \alpha R f(x), \tag{5.16}$$

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.

Huh and Sickles (1994) detail how this model can be modified to handle unobserved variables under different assumptions about temporal and cross-sectional

[34] An alternative to the competing risks model is the grade-of-membership (GOM) methodology put forth by Berkman et al. (1989) and their colleagues in which fuzzy-set topology is used to catagorize heterogeneous individual health characteristics and mortality risks.

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 $\alpha/\sqrt{n} \rightarrow 0$ for bounded α, if the mixing distribution can be characterized by a finite number of points of support. 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.

Huh and Sickles compared the NPMLE and MPLE using Monte Carlo experiments. They find some discrepancies among the estimators over different experiments but estimates are similar when the underlying stochastic process is not too complicated and has been correctly modeled. Average point estimates display downward bias for both NPMLE and for MPLE. While downward bias in the duration dependence term is expected when heterogeneity is ignored and the data exhibit positive duration dependence, the source of the downward bias in the covariate effects is unclear. As the censoring rates became larger, both NPMLE and MPLE continue to underestimate the true parameter values, but NPMLE has a bigger bias. Since censoring increases the number of observations in the tails of the distribution, MPLE benefits substantially from the smoothing function of the penalty term.

5.2.4. Simulation methods

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 (McFadden, 1989; Pakes and Pollard, 1989; Gourieroux and Monfort, 1992). 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 data generating process (DGP) itself.

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).

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

$$\lambda = t^{\gamma} \exp(\theta), \tag{5.17}$$

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

$$\Pr[x(t+\Delta t)=1] = \Pr[x(t)=1]\Pr[x(\Delta t)=0] + \Pr[x(t)=0]\Pr[x(\Delta t)=1)] + o(\Delta t). \tag{5.18}$$

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

$$\Pr[x(t+\Delta t)=1] = \Pr[x(t)=1](1-\lambda \Delta t) + \Pr[x(t)=0](\lambda \Delta t) + o(\Delta t), \tag{5.19}$$

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

As $\Delta t \to 0$, $d \Pr[x(t)=1]/dt = \lambda\{\Pr[x(t)=0] - \Pr[x(t)=1]\}$ 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, $f(\cdot)$. An alternative approach is maximum likelihood based on a nonparametric specification of the heterogeneity distribution but with the form of the density function $f(\cdot)$ required. A third approach is to estimate the parameter γ without formally specifying the probability density function.

First note that time to failure for all n individuals is recorded as $t = (t_1 \leq t_2 \leq \cdots \leq t_n)$. Using this data we can divide the time axis into k bins, the mth 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 $s = (s_1 \leq s_2 \leq \cdots \leq s_N)$, where $N > n$. The simulation mechanism here is the cumulative distribution function $F(t) = 1 - \exp\{-\lambda(\cdot)t\}$ where $\lambda(\cdot) = t_i^{\gamma} \exp\{\theta_i\}$ is the Weibull proportional hazard. Then a random number u_i, $i = 1, \ldots, N$, 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 mth bin be ν_{km}. Then the simulated probability becomes $\hat{P}_{km}(\gamma_0) = \nu_{km}/N$. If the probability that the data fall in the corresponding bin is $P_m = n_m/N$, 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

$$S(\gamma_0) = \sum_{j=1}^{k} \frac{\hat{P}_{kj}(\gamma_0) - P_j}{P_j}, \tag{5.20}$$

where k is the number of bins, and $\hat{P}_{kj}(\gamma_0)$ is the simulated probability of the jth bin with estimated parameter γ_0. The function is minimized when $\hat{P}_{kj}(\hat{\gamma}_0) = P_j$, $j = 1, \ldots, k$. 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).

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

$$\lambda = t^{\gamma_1} \exp(x_i\beta_i + \theta_i). \tag{5.21}$$

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 $t = \{t_i(x_i)\}$, $i = 1, \ldots, n$, be failure time data conditional on the exogenous variable x_i, $i = 1, \ldots, n$, 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 $0 \le s_{11}(x_1), s_{12}(x_2), \ldots, s_{1n}(x_n), s_{21}(x_1), s_{22}(x_2), \ldots, s_{2n}(x_n), \ldots, s_{m1}(x_1), s_{m2}(x_2), \ldots, s_{mn}(x_n)$ 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 the (l_1, l_2)th bin is denoted by ν_{l_1,l_2}, where $l_1 = 1, \ldots, k_1$, $l_2 = 1, \ldots, k_2$. If δ_0 is close to the true value, then the simulated bin probability

$$\hat{P}_{l_1l_2}(\delta_0) = \frac{\nu_{l_1l_2}}{n_m} \tag{5.22}$$

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

$$P_{l_1l_2} = \frac{n_{l_1l_2}}{n}. \tag{5.23}$$

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

$$
\begin{aligned}
S_m(\delta) &= \sum_{l_1=1}^{k_1}\sum_{l_2=1}^{k_2} \frac{(\hat{P}_{l_1 l_2}(\delta) - P_{l_1 l_2})^2}{P_{l_1 l_2}}, \quad \text{if } \hat{P}_{l_1 l_2}(\delta) = 0,\ P_{l_1, l_2} \neq 0, \\
&= \sum_{l_1=1}^{k_1}\sum_{l_2=1}^{k_2} \frac{(\hat{P}_{l_1 l_2}(\delta) - P_{l_1 l_2})^2}{\hat{P}_{l_1 l_2}}, \quad \text{if } \hat{P}_{l_1 l_2}(\delta) \neq 0,\ P_{l_1, l_2} = 0, \\
&= 0, \qquad \text{otherwise.}
\end{aligned} \tag{5.24}
$$

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 $P_{l_1, l_2} = \hat{P}_{l_1, l_2}(\delta_0)$, $l_1 = 1, \ldots, k_1$, $l_2 = 1, \ldots, k_2$.

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).

Conditions for consistency and asymptotic normality of the simulation-based estimator are shown in Lerman and Manski (1981). McFadden (1989) and Pakes and Pollard (1988) prove similar results for alternative simulation estimators for finite numbers of simulations.

5.3. Estimation of models of morbidity with heterogeneity/classical MLE

We next turn to *classical maximum likelihood estimators*. As an example we choose the simultaneous static model of morbidity which can be linked to labor force participation or to the labor supply decision based on a model considered in Lee (1982), Sickles and Taubman (1986), Burtless (1987) and Sickles (1989). The model is an extension of the single equation limited dependent variable model of Heckman (1976). The longitudinal nature of the data set is accommodated by using a conventional error components specification in which heterogeneity among individuals is modeled as a random effect. There are two equations in the system: the first models the unobservable health stock while the second links the observed health status to the retirement decision. The system can be written as

$$y_{it}^{*(1)} = x_{it}^{(1)}\beta_1 + \varepsilon_{it}^{(1)},$$
$$y_{it}^{*(2)} = \gamma_2 y_{it}^{(1)} + x_{it}^{(2)}\beta_2 + \varepsilon_{it}^{(2)}, \tag{5.25}$$

for $i = 1, \ldots, N$; $t = 1, \ldots, T$,

$$\varepsilon_{it}^{(j)} = \mu_i^{(j)} + \nu_{it}^{(j)}, \quad j = 1,2, \tag{5.26}$$

and where

$$\begin{aligned} E[\varepsilon_{it}^{(j)}\varepsilon_{ks}^{(l)}] &= \sigma_\mu^{(j)} + \sigma_\nu^{(j)}, && j = l,\ i = k,\ t = s, \\ &= \sigma_\mu^{(j)}, && j = l,\ i = k,\ t \neq s, \\ &= \sigma_{\nu^{(1)}}\sigma_{\nu^{(2)}}, && j \neq l,\ i = k,\ t = s, \\ &= 0, && \text{elsewhere.} \end{aligned} \tag{5.27}$$

Here $x_{it}^{(1)}$ and $x_{it}^{(2)}$ are $(1 \times k_1)$ and $(1 \times k_2)$ vectors of exogenous variables, β_1 and β_2 are conformable vectors of structural coefficients, γ_2 is a scalar, $j_{it}^{*(1)}$ and $j_{it}^{*(2)}$ are the latent variables for healthiness and leisure, respectively, and $y_{it}^{(1)}$ and $y_{it}^{(2)}$ are their observed counterparts; $\varepsilon_{it}^{(1)}$ and $\varepsilon_{it}^{(2)}$ are the structural disturbances that are decomposed by the rule in Eq. (5.26). The latent variables are linked to the observed indicators by the following rules:

$$y_{it}^{(1)} = j, \quad \text{if } A_{j-1}^{(1)} - x_1\beta_1 < \varepsilon_{it}^{(1)} \leq A_j^{(1)} - x_1\beta_1, \quad j = 1, \ldots, J_1, \tag{5.28}$$

with $A_0^{(1)}$, $A_{J_1}^{(1)}$, normalized at $-\infty$ and $+\infty$, respectively, and

$$y_{it}^{(2)} = j, \quad \text{if } A_{j-1}^{(2)} - x_2\beta_2 < \varepsilon_{it}^{(2)} \leq A_j^{(2)} - x_2\beta_2, \quad j = 1, \ldots, J_2, \tag{5.29}$$

with $A_0^{(2)}$, $A_{J_2}^{(1)}$, normalized at $-\infty$ and $+\infty$, respectively.

The data Sickles and Taubman analyzed was a panel of five biennial waves of about 800 males drawn from the approximately 11 000 individuals in the Retirement History Survey. A computational issue arises when implementing maximum likelihood since calculation of the joint probabilities of observing differing configurations of health–retirement states for the same individual over the T time periods of health–retirement states is problematic if the number of time periods is large. Numerical methods for handling the ten-period integration problem are available (Clark, 1964) but are both computationally burdensome and have an approximation error that is difficult to bound. Although moment based alternatives to maximum likelihood exist (McFadden, 1989; Pakes and Pollard, 1989) the direct evaluation of multidimensional integral is practical due to the particular structure of the correlation pattern of distur-

bances implied by the variance components model, a point which was originally noted by Butler and Moffitt (1982) and applied to the univariate probit model. Computational details for utilizing Gaussian quadrature techniques can be found in Butler and Moffitt (1982) and in Sickles and Taubman (1986).

Because computation of the joint probabilities is problematic when different correlation patterns and/or limited dependent variable structures are used, a more attractive alternative to classical maximum likelihood is *simulated maximum likelihood* (Albright et al., 1977), Hajivassiliou and McFadden (1990), Geweke (1991), Keane (1994), and Geweke et al. (1994) have demonstrated the appeal of this estimator using the GHK probability simulator in the multinomial probit model. Semiparametric maximum likelihood alternatives to exact maximum likelihood of the simultaneous latent variable model have been pursued by Ichimura (1993), Klein and Spady (1993), Lee (1995), and have been pursued empirically by Lee et al. (1995) in their structural equations semi-parametric study of family allocations and child health.

5.4. *Alternative estimators for structural dynamic models of health*

Generalized method of moments (Hansen, 1982) or *simulated method of moments* (McFadden, 1989; Pakes and Pollard, 1989) are natural approaches to estimate the dynamic model in Section 3.2.5. Recently, Haveman et al. (1994) utilized the former estimator to investigate a three-equation simultaneous model of health, work-time, and wages using panel data from the PSID. To proceed with estimation, one first selects a parametric form for the utility and production function which is specified in terms of a set of parameters (β). Letting X_{it} be the vector of variables entering the ith individual's (G) first-order conditions in period t, the $(1 \times G)$ system can be expressed as $f(X_{it}, \beta) = \varepsilon_{i,t+1}$. Rationality and its implication that information in Ω_{it} is of no help in forecasting future shocks implies that $\mathrm{E}[f(X_{it}, \beta) \otimes Z_{it}] = 0$, where Z_{it} is a $(1 \times h)$ 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

$$\mathrm{E}\left(\frac{1}{T}\sum_{t=1}^{T}[f(X_{it}, \beta) \otimes Z_{it}]\right) = \mathrm{E}[M(X_i, Z_i, \beta)]. \tag{5.30}$$

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

$$O_N(\beta) = \mathrm{E}\left(\frac{1}{N}\sum_{i=1}^{N}[M(X_i, Z_i, \beta)]\right) \tag{5.31}$$

and generalized method of moments estimates of β are defined as the

$$\text{argmin}[O_N(\beta)W_N O'_N(\beta)], \tag{5.32}$$

where W_N is the symmetric positive definite weighting matrix

$$W_N = W_N^* = S_N^{-1} = \left(\sum_{i=1}^{N} M(X_i, Z_i, \hat{\beta})' M(X_i, Z_i, \hat{\beta}) \right)^{-1} \tag{5.33}$$

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

The asymptotic covariance matrix for the gmm estimator is

$$\Phi = (D_N' S_N^{-1} D_N)^{-1}, \quad D_N = \sum_{i=1}^{N} (\partial M(X_i, Z_i, \hat{\beta})/\partial \beta). \tag{5.34}$$

This approach has great appeal as well as considerable overlap with the estimation theory behind the simulated score estimator based on distributional instead of moment assumptions (McFadden and Ruud, 1994).

There are a rich set of extended model specifications and empirical approaches that have been pursued and can be utilized to deal with particular aspects of matching the theoretical models of health with empirical methodologies. As noted above, individual specific heterogeneity can be included directly in the production or utility function, either of which can be specified in terms of a set of observable individual specific variables or unobservable frailties/heterogeneities. Moreover, other types of latent structures can be dealt with in the context of this estimator by utilizing a finite draw from the heterogeneity or latent structure distribution and averaging the moments, essentially integrating out the heterogeneity or latent structure from the Euler equations (McFadden, 1989; Pakes and Pollard, 1989). This is computationally intensive but promises to be more widely used as computing cycles become increasingly less expensive. McFadden and Ruud (1994) recently extended the simulated method of moments to more general cases by utilizing simulated bias corrections. Distributional assumptions can also be the basis for estimation, either using exact maximum likelihood or simulated maximum likelihood. Geweke et al. (1994) have pursued alternative sampling algorithms for the simulated maximum likelihood estimator as well as the kernel-smoothed frequency estimator considered in Huh and Sickles (1994) and elsewhere. The Geweke et al. results point to the advantage of Gibbs sampling in the simulation exercise. Moreover, because such key variables as leisure choice are often constrained or are categorical for adults and the elderly due to retirement or various states of labor force status, dynamic discrete methods are often employed as opposed to Kuhn–Tucker constraints on leisure or an explicit construction of virtual wage rates that correspond to the observed limit observations (Wales and Woodland, 1983; Lee

and Pitt, 1986; Ransom, 1987; and Hurd, 1990, Chapter 16, this volume, with respect to retirement specifically). In different contexts, exact and approximate solutions for the dynamic discrete model have been explored by, among others, Gotz and McCall (1984), Miller (1984), Wolpin (1987), Pakes (1987), Rust (1987, 1989), Eckstein and Wolpin (1989), Berkovec and Stern (1991), Hotz and Miller (1993), Hotz et al. (1994), Keane (1994), and Stern (1994).

A particularly appealing approach to estimating the discrete-state/discrete-time case of the dynamic programming model outlined in Section 3 has been put forth by Rust (1989, 1990, 1995). With estimates of the Markov transition probability densities for the stochastic laws of motion as inputs, Rust is able to estimate the underlying utility structure using his nested fixed point maximum likelihood algorithm (Rust, 1989). Rust analyses a model in which the control variables are consumption, a trichotomous work decision (full time, part time, retired), and a Social Security decision, with health status, among other variables, as a state variable, and with the augmented mortality data supplied by Taubman and utilized by Sickles and Taubman (1986) in their retirement-health model. A formal treatment of unobserved heterogeneity is not undertaken although reduced-form proxies for "worker beliefs" are included in his analyses. His findings are intuitive and offer an important computationally-intensive alternative to standard closed form approaches to dynamic optimization found in the literature. Backward recursion is the main method for solving this problem. When there is a finite number of states, the value function represents a finite list of options for each given state. One then maximizes the value function in each period starting from the last period and works backward through each prior period. A problem with this method is the total number of computing operations required. The number of operations depends on the number of time periods, states, and actions. The bulk of computations occurs in the evaluation of conditional expectations of the value function for each possible combination of state, action, and time. Rust implements a method which exploits the special sparsity structure of the transition probability matrix to speed up the procedure which is described in detail in Rust (1989, 1995). The solution algorithm is modified somewhat when the planning horizon is infinite. There are two main methods for numerical solutions in this case: successive approximation and policy iteration. These methods take advantage of the fact that the solution to the infinite horizon problem can be found by computing a fixed point of the Bellman operator.

Successive approximation, also referred to as value function iteration, starts with an arbitrary guess for a solution of Bellman's equation and the Bellman operator is simply iterated. By the contraction mapping theorem, this algorithm will find a fixed point and thus a solution to the problem. An initial guess of the Bellman equation equal to zero is equivalent to solving an approximate finite-horizon problem by backward induction. It is important to note that successive approximation converges linearly and is thus a relatively slow procedure.

The policy iteration method starts by choosing an arbitrary initial policy function which is used to update the value function in the following period. In infinite horizon

problems, one is interested only in the stationary optimal policy of the problem. In the policy iteration, each time a new policy function is computed a new value function is computed as if that policy were used forever. Then, with this new value function, one derives a new policy function and again computes the value function. This procedure is repeated until convergence of the policy function, and thus the value function.

A final procedure that we discuss is related to the work of Rust but utilizes computational methods to approximate the Bellman equation using continuous time models. This avoids the necessity of deriving explicit Euler equations and may allow for a richer menu of stochastic assumptions concerning, among others, heterogeneity and discrete control variates. Judd (1994, 1995) and others have pursued this approach based on orthogonal polynomial approximations to the Bellman equation. Instead of discretization methods used for solving problems of continuous state problems, the value function is approximated with continuous functions. The only restrictions that enter into the value function are imposed through the state and control variables. In this method, one considers a finitely parameterizable collection of functions, where the functional form could be a linear combination of orthogonal polynomials. The value function $V(x)$ is approximated by $\hat{v}(X;\alpha)$ where α is a vector of coefficients. Once the basic functional forms are determined, e.g., a linear combination of orthogonal polynomials, splines, or neural networks, one focuses on finding the coefficients α so that the function $\hat{v}(X;\alpha)$ approximately satisfies the Bellman equation by choosing a residual function to estimate coefficients. For more detail on these procedures see Judd and Solnick (1994) and Judd (1995). Keane and Wolpin (1994) have recently also pursued simulation approaches to solve the dynamic discrete choice model using a combination of Monte Carlo integration and regression based interpolations as alternatives to the polynomial approximations suggested in Bellman et al. (1963). These and other alternatives, such as those proposed by Judd (1995) which utilize orthogonal polynomials or other series approximations for Bellman's equation, appear particularly attractive for future research on health using highly structured models which consider such issues as heterogeneity, discrete state and control variables, and constraints.

Although the statistical and numerical treatments covered in this section are not exhaustive, they do cover most of the generic 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–Meier product limit estimator of simple life-tables and toward the multivariate techniques we have outlined.

6. Comments and conclusions

This chapter has pointed to the converging paths that demographic, epidemiologic,

biological, and socio-economic paradigms must take in order for the study of mortality and morbidity of adults and the elderly to bear fruitful results. It is clear that central to the study of such a complex issue is the availability of data monitoring systems that provide accurate information on disease etiology and specific causes-of-death as well as the plethora of economic, biological, lifestyle and behavioral, and other risk factors whose relevance to mortality and morbidity outcomes we have discussed in this chapter. Grossman's (1982b) commentary on the width of scope for theoretical work to guide future empirical study on the topic of mortality and morbidity of adults and the elderly (Gravelle, 1984) appears just as relevant today as it was a decade ago, augmented with the additional comment that the scope for emerging empirical and computational methods to implement the theoretical advances is just as wide.

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