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Broken Down by Work and Sex: How Our Health Declines

Anne Case and Angus Deaton

6.1 Introduction

The literature contains many examples of the relationship between health and various measures of socioeconomic status, including income, education, and employment. There are undoubtedly multiple causal links between these variables; income and education affect health, and health affects the ability to be educated and the ability to work. There are also third factors that affect both health and socioeconomic status, and that contribute to the correlation between them. Although mortality rates are the gold standard for measuring health status, they are of limited use for investigating the way that health changes over the life cycle, or the interactions between health, work, earnings, and age among the living. Instead, we can use measures of self-reported health status, admittedly imperfect, but certainly informative.

Figure 6.1 uses data from the National Health Interview Survey (NHIS) to plot self-reported health against age; a higher number means worse health, from 1 through 5, and the graph plots averages of these numbers by age. The age profiles of health for both men and women rise with age, although the rate at which health deteriorates with age diminishes sharply

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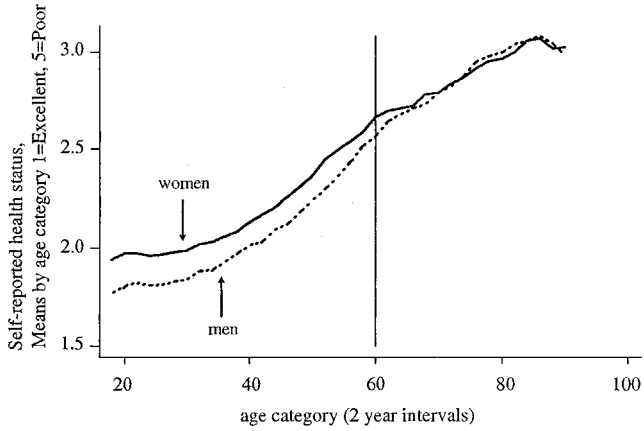

Fig. 6.1 Self-reported health status by age and sex, NHIS 1986–2001

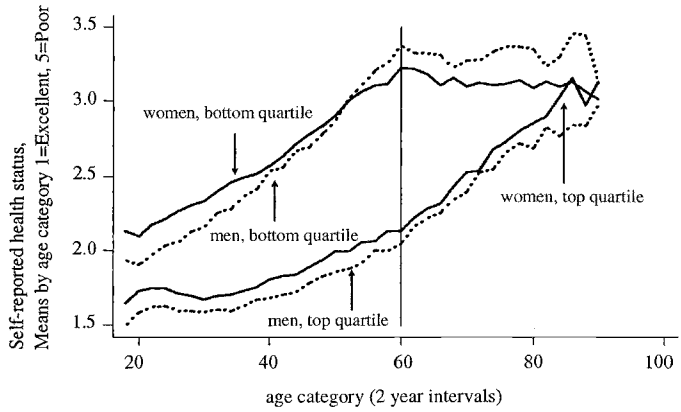

Fig. 6.2 Self-reported health status by age, sex, and income quartile, NHIS 1986–2001

after about age sixty to sixty-five. In spite of their lower mortality rates, women report worse health than men until about age sixty to sixty-five, after which there is convergence; women also make greater use of physicians' services, at least in the West, though most of this is associated with reproductive health (Waldron 1983). As far as we can tell, this pattern of self-reported health status (SRHS) by gender is close to universal around the world; it occurs in our own South African data, in India among the elderly, and in many other surveys around the world; see Sadana and others (2002).

This picture is substantially different if we stratify by income. Figure 6.2 shows age profiles for men and women in the top and bottom quartiles of family income. In the top quartile, the fraction reporting fair or poor

health (which we will refer to here as "poor health") changes little with age until age forty-five, after which it rises steadily. For the same quartile, and at all ages, men are significantly less likely to report poor health than are women, although the differences are not large at any age (1 to 2 percentage points). In the bottom quartile, self-reported health is quite different. It is much worse than in the top quartile, and it deteriorates more rapidly with age. Indeed, at *age twenty,* men in the bottom quartile already report worse health than do men in the top quartile at *age fifty.* The gender pattern in the bottom quartile is also quite different; women report worse health at young ages, but there is a crossover around age fifty, with women reporting better health thereafter. Health in the bottom-income quartile wears out a good deal faster than does health in the top quartile, and, at the bottom, men's health deteriorates more rapidly than women's health.

Although there are clearly other factors at work, including mortality selection conditioned on both sex and self-reported health status, these figures suggest that work, especially low-paid or manual work, exacts a price in terms of health, as may the consumption patterns of poorer people, in terms of tobacco use, obesity, lack of exercise, and so on. If low-paid work is harder on health than is high-paid work, people at the bottom of the income distribution will have both lower health and more rapidly deteriorating health, at least while they are working. Women, who over this period had lower labor force participation than men, would suffer less from the ravages of work, and their health would deteriorate less rapidly. It is this suggestion and its implications that we investigate further in this paper.

6.2 A Theoretical Framework

As the epidemiological literature illustrates only too sharply, it is extremely difficult to untangle the links between work, earnings, health, and education, without some sort of guiding framework. Here we work with a simple intertemporal model of health based on Michael Grossman (1972), whose work is particularly useful in this context because it explicitly analyzes both the level and rate of change of health over the life cycle, something that, for the purposes of this paper, we take to be directly measured by SRHS. We also make use of some of the modifications to Grossman's model introduced by Jana Marja Muurinen (1982). Grossman's analysis has not been widely used for studying the gradient between income and health, perhaps because Grossman himself has emphasized education and not income or wealth, and perhaps because he sees education as making people more efficient at using medical care and other methods of health repair, an interpretation that is antithetical to the explanations favored in the literature on inequalities in health. Even so, Muurinen and Julian Le Grand (1985) have used the Grossman model to interpret the main findings

of the Black report and have shown that the model is in fact well adapted to thinking about these issues.

Muurinen and Le Grand emphasize that people have three kinds of capital: health capital in the form of the health of their bodies, human capital in the form of education, and physical or financial capital in the form of assets. The first of these is more equally distributed across people than the other two; everyone has a body, and most people start life with a healthy body, which deteriorates over time. The rate at which health (body) capital declines with age is partly a biological process over which people have little control, but it is also affected by the extent to which health capital is used in consumption and in work. Manual work is harder on the body than nonmanual work, and some kinds of consumption activity are harder on the body than others. Because the three types of capital are to some extent substitutable in generating earnings, as well as in generating utility from a given level of earnings, people who have less human and financial capital have little choice but to rely more heavily on their health capital. In consequence, through an optimal but heavily constrained choice, poorer and less-educated people will experience a more rapid deterioration of health as they age.

A simple model sharpens and modifies these results and generates explicit predictions about the level and evolution of health over the life cycle. Suppose that there is an instantaneous felicity function $\upsilon(c_t, H_t)$ where t indexes age, c_t is consumption, and H_t is the stock of health. Health is updated according to

$$H_{t+1} = \theta m_t + (1 - \delta_t)H_t, \tag{1}$$

where m_t is the quantity purchased of medical care or other health-promoting activities, θ is the efficiency with which such purchases create health, and δ_t is the rate at which health deteriorates at age t. Equation (1), apparently an innocuous identity, has a number of serious implications, to which we return below.

Consumers maximize a life-cycle utility function

$$U = \sum_0^T (1 + \rho)^t \upsilon(c_t, H_t), \tag{2}$$

where ρ is the rate of time preference, and T is the length of life, which is potentially a choice variable, though that is an issue that we do not explore here. The lifetime present-value budget constraint takes the form

$$\sum_0^T \frac{c_t}{(1 + r)^t} + \sum_0^T \frac{p_m m_t}{(1 + r)^t} = A_0 + \sum_0^T \frac{y_t(H_t)}{(1 + r)^t}, \tag{3}$$

where r is the market rate of interest, p_m is the price of health-repair goods, A_0 is initial assets, and $y_t(H_t)$ is earnings, which depends on health. For sim-

plicity, we normalize the price of consumption to unity, and assume that the real interest rate and the price of medical care do not vary with age.

The basic equations of the model can most easily be seen by using the health evolution (equation [1]) to substitute for m_t in the budget constraint (equation [3]), which gives a single integrated constraint that respects both the financial and the health identities. After minor rearrangement, equations (1) and (3) yield

$$\sum_0^T \frac{c_t}{(1+r)^t} + \sum_0^T \frac{H_t}{(1+r)^t}\frac{p_m}{\theta}(r+\delta_t) = A_0 + \sum_0^T \frac{y_t(H_t)}{(1+r)^t} - \frac{p_m}{\theta}(1+r)\left[\frac{H_{T+1}}{(1+r)^{T+1}} - H_0\right]. \tag{4}$$

In this version of the budget constraint, the elements of utility, consumption, and health are multiplied by their respective prices, which, in the case of the health stock in period t, is the discounted present value of its user cost. As usual, user cost is essentially a carrying charge, which is the sum of interest and physical deterioration, multiplied by the effective replacement price, p_m/θ. The right-hand side of equation (4), which represents the value of lifetime resources, includes the valuation of the health stock after death. In consequence, treating equation (4) as a standard intertemporal budget constraint implies that the value of the body is like any other asset, which can be accumulated but which can also be sold to finance consumption. The ability to turn one's body into cash, or allowing m_t to be negative, is clearly not realistic. Even so, the assumption is a convenient starting point, and we shall return later to the (important) consequences of abandoning it and requiring that $m_t \geq 0$, so that the rate of decline of health cannot exceed deterioration. As we shall see, it does not change our basic arguments.

From equation (4), the first-order conditions for consumption and health are

$$\upsilon_{ct} = \lambda\left(\frac{1+\rho}{1+r}\right)^t \tag{5}$$

$$\upsilon_{ht} = \lambda\left(\frac{1+\rho}{1+r}\right)^t\left[\frac{p_m}{\theta}(r+\delta_t) - y_{ht}\right], \tag{6}$$

where subscripts with respect to h and c denote partial derivatives. The Lagrange multiplier λ in equations (5) and (6) is the shadow price of lifetime wealth and is constant over the life cycle. The life-cycle evolution of consumption and health can therefore be conveniently analyzed by examining equations (5) and (6) with λ held constant, a device first used in this context by Adam Wagstaff (1986).

Equation (6) permits derivation of the standard comparative static re-

sults about the *level* of health. Provided that there is diminishing marginal utility of health as well as diminishing marginal productivity of health on earnings, the health stock throughout life will be higher (a) the lower is the price of health repair, p_m, (b) the higher is the efficiency of medical care or other purchases in repairing health, θ, (c) the lower is the *rate* of health *deterioration* δ_t, (d) the lower is the rate of time preference ρ, (e) the higher are initial assets, initial health, or lifetime earnings, and the lower are prices over the lifetime, all of which lower λ through lifetime income effects, and (f) the milder is the effect of diminishing returns to health in either consumption or production.

In Grossman's original model, of which this is a simplified form, the effect of education works to increase the parameter θ, so that health repair is more efficient with the same health inputs, the effective price of health repair is lower, and health is higher throughout the life cycle. This is true even in the "pure investment" version of the model, in which health has no direct effect on utility, so that υ_{ht} is zero, and the health stock is determined through its effects on earnings, by setting the last term on the right-hand side of equation (6) equal to zero. In the pure consumption model, where y_{ht} is zero, or in mixed models with both consumption and earnings effects of health, education will also promote health by lowering λ through the lifetime income effects of higher earnings. Muurinen, in her version of Grossman's model, argues that education works so as to reduce the rate of health deterioration, which lowers the user cost of health and raises its optimal level. Provided that health affects consumption directly, there is also a direct income effect on the health stock through λ, so that both education and income, conditional on education, should promote health, albeit in different ways. Note finally that the correlation between health and the rate of time preference will also generate a correlation between health and education, if more patient people acquire more education. This is the classic "third factor" explanation of the correlation between health and education proposed by Victor Fuchs (1982).

Our main concern in this paper is the way that health declines over the life cycle, as well as with how that decline is affected by education, work, and income. The predictions of the theory come from differentiating equations (5) and (6) with respect to time. We make the assumptions that health and consumption are complementary (or additive) in utility, so that $\upsilon_{ch} \geq 0$, and that the instantaneous felicity functions are concave in health and consumption taken together. Then elementary but tedious algebra shows that, when the rate of interest is equal to the rate of time preference, health will decline over the life cycle if and only if the rate of health deterioration δ_t increases with age. If the rate of interest is not equal to the rate of time preference, there is an additional factor that increases the rate of decline of health with age if the rate of time preference exceeds the rate of interest, and which moderates it if the rate of time preference is less than the rate of

interest. A lower price of health repair, or a higher efficiency of health repair, through education (for example), boosts health throughout the life cycle but accelerates its rate of decline if y_{ht} is positive. At retirement, after which there are no earnings and therefore no effect of health on earnings, there will be a discrete increase in the user cost of health—see the right-hand side of equation (6), where y_{ht} reduces the net user cost—which will generate a corresponding drop in health. Subsequent to retirement, the effect of the increasing rate of deterioration on the user cost will be lower because of the absence of the effect of health on earnings, so that the rate of decline of health should be lower immediately after retirement than it was immediately prior to retirement.

When thinking about how health changes with age in this model, it is important to maintain a clear distinction between the rate of health *deterioration,* which is the quantity δ_t, and the rate at which the stock of health changes, ΔH_t. The two concepts, which sound very much alike, are quite different, but are linked by the identity (1), which can be written in the form

$$\frac{\Delta H_{t+1}}{H_t} = \frac{\theta m_t}{H_t} - \delta_t, \tag{7}$$

so that expenditure on medical care and other health repair offsets, to a greater or lesser extent, the deterioration in health. There is an important question whether equation (7), or equation (1), can be an adequate description of health evolution. In particular, note that equation (7) implies that the technology allows perfect repair of the biological effects of aging, so that it is possible to put a halt to aging and to postpone death for ever. Grossman's model is different from ours, in that m_t is produced using market goods and time, but he assumes constant returns to scale in the technology so that, once again, death can be defeated by sufficiently large amounts of money and time. In a model where time is priced at the market wage, those who can afford to pay for it have the option of eternal life.

Eternal life is more than a hypothetical outcome that, while permitted by the technology, will never actually be chosen. If the rate of biological deterioration is constant, which is perhaps implausible but is hardly impossible (and if the rate of interest is at least as large as the rate of time preference), people will "choose" an infinite life. Otherwise, when the rate of deterioration is increasing with age, people "choose" a finite life, because at some point the cost of medical care is so high, or the unpleasantness of health repair (exercise?) so extreme, that even death is better. Death is not inevitable, but an optimal choice. That the technology exists to make this possible would not be claimed by even the most fanatical proponent of the effectiveness of medical care or of the latest programs of exercise and diet.

That the health technology permits complete repair is a problem for health capital models even apart from the possible choice of eternal life. According to equations (5) and (6), the rate at which health declines over

time depends on the *rate of increase* of δ_t, not on its *level.* Given the identity (7), this implies that medical care or other repair is used fully to offset the level of δ_t; indeed, if δ_t is constant, the health stock is constant, and repair fully offsets deterioration. But deterioration is proportional to the stock of health, so that these models imply that, controlling for the rate of deterioration δ_t and its rate of change, health repair is *higher* for healthier people, because they have more stock to start with and deterioration is proportional to the stock. In Grossman's original work and in several papers since, authors have found a *negative* correlation between the stock of health and medical care, perhaps not surprisingly, given that people tend to seek medical care when they are sick, not when they are well. Of course, these findings may perhaps be attributed to problems with the empirical implementation of the model; as Wagstaff (1993) and Grossman (2000) himself have argued, neither the raw correlation nor the ordinary least squares (OLS) regression of medical care on health can be expected to give the right answer, because of simultaneity through the unobservable components of deterioration, and because health repair involves more than medical care. It is unclear whether there exist feasible methods for correcting these problems and whether an adequate test of the model is possible.

Instead, it is possible that the fundamental problem is not the assumption that people would offset health deterioration if they could, but the assumption that the technology exists that would allow them to do so. If perfect health repair is impossible, we have a very much simpler and more intuitive model of health in which it is the *level* of physical deterioration that determines the rate of decline of health, with only limited offset possible through behavior. In terms of the optimality conditions (5) and (6), the former will still hold, though the latter will not, because, in general, the medical or other technology does not exist to allow the marginal utility of health to be equated to its user cost. One of the issues that we shall examine in our empirical work is whether the rate of health decline in our data is better described by the *level* or by the *rate of change* of the rate of physical deterioration in health.

Even within the Grossman model, there is a source of health decline even when the rate of physical deterioration is constant. This comes from acknowledging something that we have ignored so far, which is that health cannot be sold, because purchases of health-enhancing goods m_t cannot be negative. Suppose that someone is approaching the end of life and in excellent health. According to the basic model (equation [4]), good physical health will be traded in for consumption prior to death, but if this is not possible, maintenance will stop, and health will be allowed to decline at the maximum rate possible, which is the rate of health deterioration δ_t. Hence, during this period at the end of life, and even within the standard Grossman model, the rate of health decline depends on the level of δ_t, not on its

rate of change. However, this cannot be the explanation for health declines later in life, because it implies that during this period there would be no purchases of health-enhancing goods and $m_t = 0$. But this is contradicted by the obvious evidence that purchases of health care rise with age, not the opposite. And for all periods in which $m_t > 0$, we are back to the original analysis, in which the rate of health decline depends on the rate of change of the rate of deterioration, not its level.

A useful extension of the Grossman model, with or without the repair technology, comes from Muurinen and Le Grand's suggestion that people with low education are more likely to work in manual jobs, because non-manual occupations are not open to them. Further, in manual jobs, health deteriorates more rapidly, because the nature of the work makes direct demands on physical health through the amount of exertion required, and because many such jobs carry risks of injury (back problems associated with lifting, for example) or other environmental insults. Similarly, people with high wealth or high wages for their level of education will be better able to avoid such jobs. Those who are lucky enough to be born into wealth are rarely observed performing manual work, even when their intelligence and education equip them for little else. We can model such effects explicitly by extending the dependence of earnings on health to accommodate an additional choice variable that allows people to enhance their earnings at the expense of faster deterioration in health, effectively selling their health capital. If we write earnings as $y_t(H_t, z)$ with a positive partial derivative for z, and compensate by writing the rate of deterioration of the health stock as $\delta_t(z)$, also with a positive partial derivative, then equations (5) through (6) are unchanged (or equation [5] is unchanged if [6] does not hold), but we have the additional condition, directly from the budget constraint, that

$$\frac{\partial y_t}{\partial z} = \frac{p_m}{\theta} \cdot \frac{\partial \delta_t}{\partial z} \cdot H_t, \tag{8}$$

so that the marginal addition to earnings from additional manual work is set equal to the marginal health costs, which is the product of the health stock multiplied by the marginal effect on the user cost. The effect of additional manual labor on earnings is lower at higher levels of education, because professors, unlike construction workers, delivery drivers, or professional boxers, get no increase in earnings by wearing out their bodies more rapidly, so that equation (10) implies that physical effort z and health deterioration are higher among those with lower education. If the health stock is optimally adapted to its user cost, the health stock will be higher among the better educated. If not, and the evolution of the health stock is primarily determined by its rate of physical deterioration, then health will decline more rapidly with age among those with less education. Those with education base their earnings on their human capital, which depreciates slowly

if at all. Those without education sell their bodies, which depreciate more rapidly.

At a fixed level of education, equation (8) also implies that those with more health are less likely to undertake heavy labor to improve their earnings because, with more health, they have more to lose from an increase in its rate of depreciation.

We can also consider a formally identical effect that works through consumption. Suppose that the felicity function contains a second consumption good whose price is paid, not in money, but in the rate of health deterioration. This component includes activities such as smoking, the consumption of junk food, sloth, and cheap risk-taking activities such as unsafe sex, all of which are either low-cost or free, all of which are pleasurable, at least to some, but all of which are paid for out of a higher rate of health deterioration. If the second consumption item is w, say, the additional first-order condition is

$$\upsilon_{wt} = \lambda\left(\frac{1+\rho}{1+r}\right)^t \frac{p_m}{\theta}\frac{\partial \delta_t}{\partial w}\cdot H_t, \tag{9}$$

which, once again, holds whether or not health is optimally adapted to its user cost. The difference between equations (8) and (9) is whether or not health is "sold" directly for utility, or indirectly through the labor market. Holding everything on the right-hand side constant, higher education that changes tastes away from (reduces the marginal utility of) w-goods will reduce their consumption and lower the rate of health deterioration. Of course, education is also likely to increase θ, which will increase the demand for w-goods, because it is now easier to repair their damage, and increase lifetime wealth, which increases demand through income effects. As in the production case, higher health status reduces the consumption of w-goods, because their effects are proportionately more costly for healthier people.

Note again that equations (8) and (9), with their implication for health deterioration, hold whether or not there is a technology that allows full health repair, although their implications for health and its evolution will differ. If the repair technology is less than perfect then, at least beyond some point, the level of health deterioration will show up as an actual decline in health. Manual workers, those with low education or low wealth, will have higher rates of health deterioration, and their physical health will deteriorate more rapidly with age. With full offset possible, there is no such implication. Unless manual work and unhealthy consumption increase the *rate* at which health deterioration *increases* with age, which although possible is far from obvious, they will affect the *level* of health, but not its rate of decline with age. In our empirical analysis we will examine both the level and rate of change of health across different occupations.

6.3 Empirical Evidence

Our data come from the sixteen successive waves of the NHIS from 1986 through 2001. This is a large nationally representative sample of households, whose members are either interviewed directly or, in the case of children, by proxy. There are 1,209,808 people in the sixteen-year sample, though for most of the calculations we work with the subsample of adults aged eighteen to sixty, of which there are 711,765. This provides us with a large enough sample to allow a good deal of disaggregation by age, sex, and occupation. The NHIS is a new cross section in each year so that, although we can track birth cohorts, for example, we cannot follow any particular individual over time.

The survey collects data on SRHS on a scale of 1 to 5, where 1 is "excellent," 2 is "very good," 3 is "good," 4 is "fair," and 5 is "poor," so that bigger numbers always indicate worse health. There is a very substantial literature on the advantages and disadvantages of this measure of nonfatal health; here we simply accept the measure, and our results are conditioned on that acceptance. In most cases we respect the ordinal nature of these data by using appropriate techniques, though we will often show averages based on the nominal 1 through 5 scale.

Family income is collected on a categorical basis, and we assign each person the midpoint of the income range to which they belong and then deflate by the Consumer Price Index (CPI) to bring income to 1982 prices. Education is the number of years of education completed. The survey collects information on whether people are in or out of the labor force, and for those who are working (around three-quarters of the sample) we have two-digit occupational codes. Summary data on education, income, race, and occupation are shown in table 6.1. All the means we present, as well as results from subsequent calculations, use the survey weights in order to describe the national population.

The distribution of men and women across occupations is shown down the columns; apart from the omitted category (new workers, military employees, and those whose status is unknown), the occupational columns would sum to one. A little less than 12 percent of the sample is black, and 51 percent are female. The nonmanual occupations are listed first, from executives to administrative support. Apart from the last, where workers are predominantly female, men and women are more or less equally represented in the nonmanual occupations. We also show ten manual occupations, where there is a great deal of variation in the percentage of workers who are female.

Our starting point is the information in figures 6.1 and 6.2, presented briefly in the introduction. Figure 6.1 shows that average health declines with age and is worse for women than for men but worsens somewhat more

Table 6.1 **Sample means, men and women aged 18–60, NHIS 1986–2001**

	All	Women	Men
Age	37.01	37.04	36.97
Education	13.04	12.98	13.10
Indicator: white	0.817	0.808	0.827
Indicator: black	0.117	0.128	0.106
Log(family income) in $1982	9.993	9.940	10.05
Indicator: Female	0.510	1.000	0.000
Occupation			
Executive	0.122	0.105	0.141
Professional/Specialty	0.121	0.129	0.112
Technician	0.033	0.031	0.034
Sales	0.098	0.096	0.100
Administrative support	0.127	0.198	0.054
Private household services	0.005	0.009	0.001
Protective services fire/police	0.015	0.005	0.025
Service (food, cleaning)	0.092	0.119	0.064
Farming/Fishing	0.021	0.007	0.036
Mechanic	0.031	0.003	0.061
Construction	0.025	0.001	0.050
Precision production	0.026	0.012	0.040
Machine operator	0.059	0.046	0.073
Transportation/Moving	0.037	0.007	0.067
Handler, equip. cleaner	0.033	0.013	0.053
Out of the labor force	0.146	0.211	0.078
No. of observations	711,765	374,700	337,065

Notes: Occupation columns add to less than one because new workers and military employees are omitted. All means are weighted, using individual level sample weights provided by the NHIS.

slowly with age for women than for men. Figure 6.2, which shows the same information for people in the top and bottom quartiles of family income, shows that rich and poor people have very different life-cycle patterns of health. The poor have worse health throughout life, and their health worsens more rapidly with age. Women "age" (in terms of worsening health) less rapidly than do men, but only in the bottom quartile of family income, not in the top quartile. Among the poor, average health stops worsening after age sixty. Although there is undoubtedly some role for health- and income-specific mortality in accounting for these results, the patterns of health change by age, by income groups, by sex, and by retirement age, are consistent with the hypothesis that manual work causes health to decline more rapidly than does professional work. As we saw in section 6.2, with a technology that allows complete health repair, there is no reason to expect such results, even if there are indeed differential rates of deterioration. Yet the existence of the technology is itself implausible, and figure 6.2 might be taken as evidence in that direction.

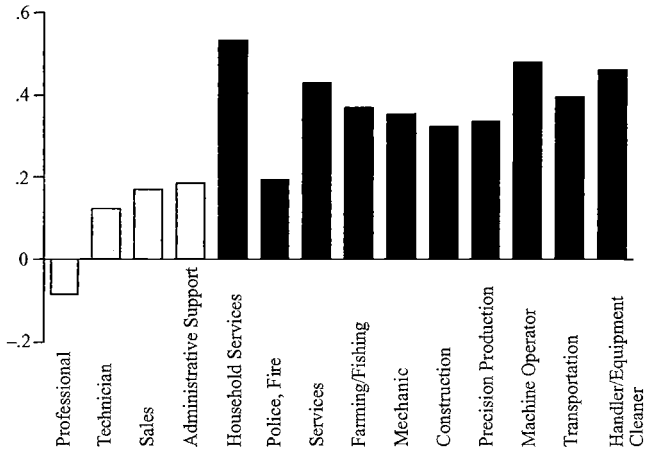

Fig. 6.3 Self-reported health status by occupation

Note: Coefficients on occupation from an ordered probit that includes controls for age, sex, and race. Omitted occupation = executive. The full occupation terms are listed in table 6.2.

A more comprehensive investigation requires that we examine occupational effects on health, on which summary evidence is presented in figure 6.3. Underlying this figure is an ordered probit for those in work in which SRHS is linked to a set of age, sex, race, and occupational dummies. The figure shows the estimated coefficients on the occupational dummies. Those to the right (dark bars) are manual occupations, those to the left (lighter bars) are nonmanual occupations. Consistent with all the theoretical predictions, those employed in manual occupations have worse health than those who work in professional occupations. Police and fire workers are an exception to the general pattern; they are in a manual occupation that carries significant risk of health deterioration, and yet their health status is more like professional than other manual workers. We do not have an explanation for these results, although it is possible that health-based selection into and out of police work and firefighting is sufficiently severe to offset the deterioration associated with the work itself. We can imagine that the same might be true of professional athletes, if we had such data. Selection is important for all of this analysis, and we shall investigate it further below.

Table 6.2 takes the results in figure 6.3 a step further, disaggregating by sex, and also including controls for income and education. The first column shows the results for men and women combined, while the second and third columns show the results by sex. These again come from ordered probits, now run separately for men and for women. The most notable finding here is how similar the results are for men and women. All nonmanual workers are less healthy than executive and administrative workers, with the smallest difference among those in professional and specialty occupa-

Table 6.2 **Self-reported health status and occupation, men and women aged 18–60, NHIS 1986–2001**

	All (1)	Women (2)	Men (3)
Log(household income)	–.192	–.179	–.203
	(.002)	(.003)	(.003)
Education	–.067	–.070	–.065
	(.001)	(.001)	(.001)
Occupation			
Professional/Specialty	.031	.056	.014
	(.006)	(.008)	(.009)
Technician	.085	.089	.080
	(.009)	(.013)	(.013)
Sales	.060	.089	.031
	(.006)	(.009)	(.009)
Administrative support	.069	.062	.386
	(.006)	(.008)	(.010)
Private household services	.145	.165	.141
	(.022)	(.022)	(.098)
Protective services fire/police	.071	.113	.066
	(.013)	(.028)	(.014)
Service (food, cleaning)	.160	.171	.167
	(.007)	(.009)	(.011)
Farming/Fishing	.020	.033	.019
	(.011)	(.024)	(.013)
Mechanic	.155	.139	.150
	(.009)	(.039)	(.010)
Construction	.074	.065	.067
	(.010)	(.061)	(.011)
Precision production	.123	.179	.103
	(.010)	(.019)	(.012)
Machine operator	.201	.253	.177
	(.008)	(.012)	(.010)
Transportation/Moving	.145	.185	.140
	(.009)	(.024)	(.010)
Handler, equip. cleaner	.147	.173	.159
	(.009)	(.019)	(.011)
No. of observations	502,374	243,079	259,295

Notes: Coefficients reported are estimates of the health status expected given this occupation, relative to the omitted category of "executive/administrative." Estimates are based on ordered probits that also include a full set of indicator variables for age, survey year, and indicators that race is white or black. The ordered probit in column (1) also includes an indicator for sex. All ordered probits have been weighted using the individual level sampling weights provided by the NHIS. Standard errors appear in parentheses.

tions. Male and female manual workers are typically less healthy on average, and the differences by sex are much smaller than differences across occupations. Compared with figure 6.3, the inclusion of controls for income and education markedly reduces the estimated occupational effects on health for construction workers, and for farmers who are among the least educated and worst-paid groups, and who, conditional on education and income, report no worse health than nonmanual workers. These effects are essentially the same for men and for women. The clearest exception to the similarity is for men who work in administrative support, an occupation in which there are four times as many women as men. While this case might well be attributed to differential selection, such an argument flies in the face of the evidence from other occupations where, in spite of substantial differences in the proportions of men and women, their reported health status is very similar. These results provide prima facie evidence for the existence of occupational specific health effects that operate, at least in part, independently of the personal characteristics of the workers. Note also that to the extent that occupational structure contributes to differences in men's and women's health, the effect comes from the allocation of men and women across occupations, not from differences by sex within them.

Table 6.2 also shows that there are protective effects of income and education on health even when we control for occupational status. Household income is substantially and significantly more protective for men than for women, a standard result in the literature. Years of education are more protective for women than men, and although the difference is significant given the sample size, it is not very large.

As we saw in section 6.2, the existence of *level* effects in health status across occupations is a less effective test of alternative theories than is the existence of differential *rates of change* of health. Figure 6.4 provides evidence on the way that health declines with age during the working life in manual compared with nonmanual occupations. Once again, underlying the results is an ordered probit in which SRHS is linked to a complete set of age, sex, and race indicators, and to education, the logarithm of family income, and an indicator for manual occupation. Education and income are interacted with age, and the manual occupation dummy is interacted with a complete set of age dummies. The figure shows the estimated coefficients on these interactions, so that each point shows the difference in health status between manual and nonmanual workers at that age. The graph shows a rising pattern from left to right, so that the health difference between manual and nonmanual workers is *increasing* with age. Table 6.3 shows the relevant results from separate ordered probit equations for males and females. The interaction coefficients are significantly positive for both, but not significantly different from one another. Older nonmanual workers, whether male or female, suffer a greater self-reported health disadvantage than younger nonmanual workers. It should be noted that these results are affected by

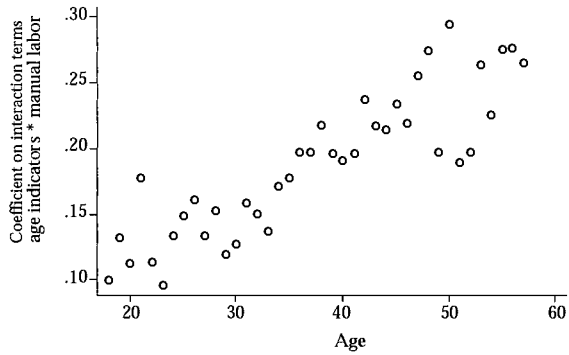

Fig. 6.4 Self-reported health status ordered probit results, manual labor–age indicator interactions, NHIS 1986–2001

Note: Ordered probit estimation included a complete set of age indicators, education, and education interacted with age, log(family income), and log(family income) interacted with age, and controls for sex, race, and manual labor.

Table 6.3 Self-reported health status by manual labor status, men and women aged 18–60, NHIS 1986–2001

	Women	Men
Manual labor	.0557	.0346
	(.0180)	(.0178)
Manual labor × age	.0017	.0013
	(.0005)	(.0005)
Log(household income)	–.0589	–.0902
	(.0093)	(.0101)
Log(household income) × age	–.0036	–.0034
	(.0003)	(.0003)
Education	–.0966	–.0848
	(.0038)	(.0035)
Education × age	.0007	.0005
	(.0001)	(.0001)
Year indicators?	Yes	Yes
Age indicators?	Yes	Yes
Race indicators?	Yes	Yes
No. of observations	243,079	259,295

Notes: Coefficients reported are estimates of the health status expected given this explanatory variable, relative to white-collar employment at age eighteen. Estimates are based on ordered probits that also include a full set of indicator variables for age and year, and indicators that race = white and race = black. All ordered probits weighted using individual sampling weights provided by the NHIS. Standard errors appear in parentheses.

health-specific selection, but because it is the less-healthy workers who are selected out—something on which we present evidence below—the increase in the health differential with age is biased *downward.* Selection cannot explain the upward slope that we see in figure 6.4.

As we argued in the theoretical section, it is hard to reconcile such effects with a story in which a full-repair technology allows people to adjust their health to its user cost. Although manual work causes greater deterioration in the health stock, this is supposed to be offset by repair, so that there is no reason for the health status of manual workers to decline more rapidly with age unless the rate of increase of deterioration with age is itself higher in manual occupations. There is no reason to suppose that this is the case, and indeed, Muurinen and Le Grand (1985) argue that the opposite is likely to be true. They point out that the biological component of health decline is very small among young workers, so that the difference between health deterioration rates of young manual and nonmanual workers is almost entirely attributable to differences in their work. Among older workers, by contrast, there is a large common biological component to health deterioration, so that differences due to the work environment generate a smaller proportional difference in overall health deterioration and thus in the user cost of health. In consequence, the health gradient between manual and nonmanual workers should diminish with age, which is exactly the opposite of what we see in figure 6.4.

As did table 6.2, table 6.3 shows that the effects of income and education on health status are not eliminated by controlling for whether people are manual or nonmanual workers. Income and education are separately protective, and when we allow for interactions with age, the log of family income has a substantially larger effect for men than for women. Although part of the effect of education works through the selection of occupation, there are other protective effects; according to the theory, there are several ways in which education can reduce the user cost of health. There are also effects of both income and education on the *rate* at which health declines with age. The protective effect of income *increases* over the working life, while that of education *decreases.* To account for these, the model with full-repair technology would require that the rate of increase of the depreciation rate be lower at high income and higher at high education. Without full repair, we would require that the *levels* of deterioration respond in the same way.

That health-based selection is indeed important is documented in figure 6.5. This is for men only, and it extends beyond the working years and up to age seventy-five. This is a version of figure 6.2 for men, but now separating those who are in the labor force from those who are not. The latter have much worse health, presumably because poor health is one of the reasons for being out of the labor force. It is the health of those out of the labor force that worsens rapidly with age until around age fifty, and then improves, presumably because more and more people with normal health for

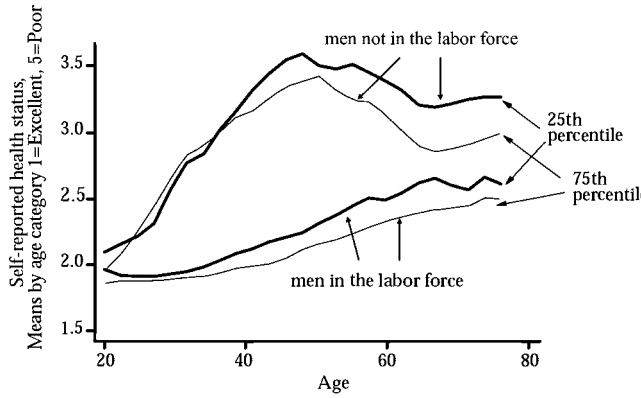

Fig. 6.5 Self-reported health status by age at the 25th and 75th percentile of the income distribution, by labor force status, men, NHIS 1986–2001

their age leave the labor force for normal, non-health-related retirement. Within these two classes, either in or out of the labor force, being at the first versus fourth quartile of income (here taken to be those whose income is within 5 percent of the 25th and 75th percentiles) still affects health in the usual direction, but the effect of income is swamped by the effect of being in or out of the labor force. Figures 6.2 and 6.5 are reconciled by noting that the group of those who are out of the labor force and in the top quartile of income is very small. As a result, we conclude that the gradient of health with respect to income in figure 6.2 is largely driven by causality running from health to income, through health-related participation in the labor force. As figure 6.5 shows, there is still a role for income in conditioning health within each group, and the earlier results of this section show that at least part of this relationship is attributable to the effects of different kinds of work on health, but the major features of figure 6.2 can be accounted for by health-based selection in and out of the labor force.

We turn finally to the issue of health selection at the occupational level and investigate whether the estimated rate of health decline with age is indeed biased downward by the fact that people who are less healthy drop out of occupations with high rates of wear and tear. Ideally, we would examine this question using panel data that follow people over time. We cannot do this with the NHIS, but we can match birth cohorts in specific occupations over time and track their size through the successive random population samples in the survey. In particular, we construct occupational birth cohorts by tracking, for example, how many fire and police workers born in 1956 show up in each of the surveys from 1986 through to 2001, and then test whether the number diminishes from one year to the next more rapidly the worse is the average health status of the occupational birth cohort in the first year. Clearly, this technique will only work well if recruitment starts early in the working life and the profession makes no

new hires once its original intake is set. These assumptions are clearly restrictive, but they are the best that we can do given the data available.

Table 6.4 shows the results of the regressions, all of which control for a full set of age, year, and birth cohort dummies. Because of a change in weighting procedures, there is a seam in the series between 1996 and 1997, and this change is omitted from the regressions. The dependent variable is the proportionate change in the number of male workers in the occupation in a given birth cohort. There are 2,210 birth cohort per occupation per year cells for nonmanual workers, and 3,981 for manual workers. Column (1) shows the regression for nonmanual workers with different coefficients on health status for each occupation, and column (2) when the coefficients on

Table 6.4 **Change in labor force participation in given occupations and self-reported health status, reported for men, NHIS 1986–2001**

	White collar workers		Manual workers	
	(1)	(2)	(3)	(4)
Health status × Exec	.051			
	(.051)			
Health status × Prof	.065			
	(.053)			
Health status × Tech	.117			
	(.048)			
Health status × Sales	.055			
	(.049)			
Health status × Admin support	.070			
	(.045)			
Health status		.106		–.122
		(.041)		(.031)
Health status × Protective (fire, police)			–.082	
			(.037)	
Health status × Service (food, cleaning)			–.099	
			(.033)	
Health status × Farming/Fishing			–.101	
			(.034)	
Health status × Mechanic			–.131	
			(.035)	
Health status × Construction			–.079	
			(.036)	
Health status × Precision production			–.104	
			(.036)	
Health status × Machine operators			–.135	
			(.034)	
Health status × Transportation, moving			–.123	
			(.034)	
Health status × Handlers, equip. operators			–.099	
			(.032)	
No. of observations	2,210	2,210	3,981	3,981

Notes: All regressions include year indicators, age indicators, and birth cohort indicators.

health are constrained to be the same across occupations. All estimated coefficients are positive, and only one is significantly different from zero; collectively, the overall effect is positive and significant. While it is unclear why cohort size should rise with poor health, there is certainly no evidence that cohort size *falls* with worse (larger) health status for nonmanual workers. For manual workers, in columns (3) and (4), there is indeed such an effect. Over all the manual occupations, an increase in average health by 0.3, say, equivalent to the effect of about twenty years of normal aging from forty to sixty, would be to remove about 3.6 percent of the age cohort from the workforce. The size of this effect does not vary very much across occupations but is somewhat higher for machine operators and considerably lower for firemen and policemen. Health-based selection appears to be real among manual workers, but even those who remain in the occupation grow less healthy with age, and they do so at a rate that is larger than that for nonmanual workers, among whom there is no evidence of such health-based selection.

6.4 Conclusions

We started from the observation that SRHS worsens with age and that it does so much more rapidly among those at the bottom of the income distribution, who also start their working lives with lower health. Our original suspicion was that, because manual work involves more wear and tear on the body, the health of manual workers would decline more rapidly than that of nonmanual workers, thus offering an explanation for our starting facts. However, the standard health capital model of health, which assumes a technology by which health can be fully repaired, does not predict that health declines more rapidly among those whose work (or consumption) imposes greater demand on their bodies. Instead, people will use medical care or other health-repair mechanisms to offset the physical deterioration. Indeed, if the marginal utility of the health stock is set equal to its user cost, as intertemporal optimality requires, the rate of health decline is not affected by the rate of wear and tear but by the rate of increase with age of the rate of wear and tear. Standard arguments suggest that this rate of increase is likely to be lower, not higher, among manual workers. Yet the data from the NHIS show that the health of manual workers does in fact decline more rapidly during the working years than does the health of nonmanual workers, in spite of the existence of health-based selection out of manual work, which artificially inflates the health of those who remain. We do not find this result at all implausible. Instead, the implausibility lies in the health-repair technology that is routinely assumed in the health economics literature.

Although manual workers have worse health than do nonmanual workers, and although their health declines more rapidly, the major factor accounting for the differences in health and health decline in different parts of the income distribution is whether or not people are in the labor force, a mechanism where causality runs from health to income, not the reverse.

Even so, both income and education have independent protective effects on health for those who are in work, and these effects are reduced but not eliminated by controlling for occupation. With only a few exceptions, we find a marked similarity in all of these results between men and women.

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Comment Daniel McFadden

You might anticipate that a paper with a title as clever as this one will be fun to read, and you would be right. This is a paper about *health capital,* as opposed to physical or skill capital, and its dynamics over the life cycle. Just as the concept of skill or human capital informed the economic analysis of education as a life-cycle decision, the concepts of health capital and life-cycle decision making on health maintenance offer useful insights into the interactions of human biology, medical technology, and individual behavior. Case and Deaton start from a model of health capital introduced

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by Michael Grossman in 1972. Let H denote the stock of health capital, m denote expenditure on health maintenance, θ denote the efficiency of investment, and σ denote a depreciation rate that may increase with age. The equation of motion for H is

$$H_{t+1} = \theta m_t + (1 - \sigma_t)H_t. \tag{1}$$

In this model, health capital is like the stock of water impounded in a reservoir, with an evaporation rate σ that varies with temperature, or age in a health capital interpretation.

Is this an apt model for health capital? To begin a discussion of this issue, note first that equation (1) is an oversimplified model of the dynamics of a capital stock, even for water in a reservoir. The reservoir has finite capacity, and water added beyond capacity is spilled. There may be natural replenishment, seasonal in nature, in addition to budgeted water replacement. Because evaporation is proportional to surface area and stock is proportional to volume, the *rate* of depreciation depends on the geometry of the reservoir, and is often lower when stocks are high. Figure 6C.1 illustrates a cylindrical reservoir, where the amount of depreciation $D_t = \sigma_t H_t$ is independent of the stock, so the rate of depreciation is inversely proportional to the stock. Other cases in the figure are a reservoir with a triangular cross section, where D_t is proportional to the square root of H_t, and a flask-shaped reservoir, where D_t *rises* when H_t falls. Only when the reservoir is composed of a series of small vessels, so that storage and evaporation are both proportional to the number of vessels filled, is it correct to describe the dynamics of the capital stock using equation (1). This case corresponds to a capital stock that is an aggregate of many smaller units that have independent failure probabilities, such as light bulbs, or perhaps cells in the body. However, as the reservoir example illustrates, it may not describe well the evolution of a single system, such as a human body, where there may be a complex interaction between component failures and system failures.

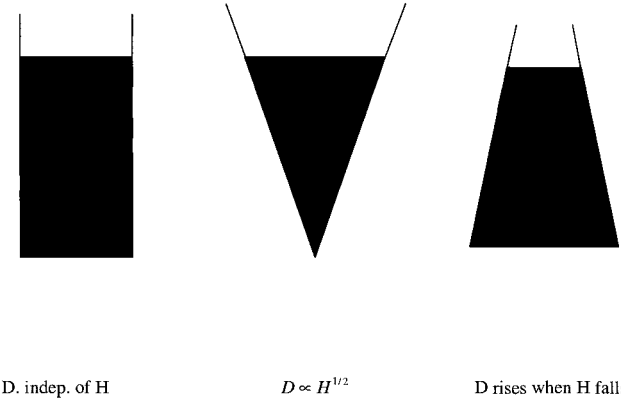

Fig. 6C.1 Models of Depreciation

While the water reservoir analogy to health capital should not be stretched too far, it does suggest the possibilities that (a) early in life the body's self-repair and replenishment mechanisms are usually adequate to maintain the stock near capacity, (b) with age natural replenishment diminishes and more budgeted investment is needed to maintain the stock, and (c) the technology of depreciation may induce losses that are not proportional to stock and are relatively larger when the stock is small, old, and worn. This analogy provides a simple explanation as to why budgeted health investments can be low when we are young and health capital is high, and can rise sharply as we age and the remaining stock of health capital diminishes.

Other models of physical aging may be better analogs of biological aging. Automobiles are reputedly designed so the power train will usually operate for 70,000 miles, with sharply increasing hazard rates thereafter. Optional equipment is often less durable. Life can be prolonged by preventative maintenance and behavior (e.g., avoid wear and tear by driving only to church on Sundays), but repair frequency and costs rise with age, and it is sometimes easier to work around flaws than to fix them. When the power train fails, the machine is scrapped.

Is this a useful analogy for biological capital? Selection has designed us to stay healthy through our productive and reproductive years and is indifferent to our survival thereafter. Preventative maintenance is important, and hard use hurts, but hazard rates for various failures are largely beyond our control. We work around the failures of some body parts and repair others, until the power train fails and it's "goodbye, Charlie."

The automobile depreciation example suggests that it may be fruitful to look to failure-time models for description of the dynamics of health capital. There is ample precedent for this in both physical and biological applications, and in the epidemiology of aging. Multiple hazard models such as accelerated failure-time models with hazard rates influenced by exposure to various risk factors and by preventative and restorative investments may work to describe the evolution of health capital. If we are lucky in choosing our models, a one-dimensional index of health capital may suffice, perhaps a comprehensive analog of the activities of daily living (ADL) index we might call a "capacity for life-cycle living" (CLCL) index.

Life-cycle models readily accommodate exogenous mortality hazard. Do they remain tractable with the introduction of other health hazards, perhaps summarized in a single CLCL index, with hazard rates that are determined endogenously through preventative medical expenditures and behavior, with some failures requiring expensive repairs? If we are less lucky, health capital may be fundamentally multidimensional, requiring more work to identify and measure its components, but also inviting new analysis; for example, are mental, cardiovascular, and skeletal capital complements or substitutes, and can our portfolio of health capital stocks be rebalanced through the life cycle to minimize risk?

When considering life-cycle models with health capital, a few questions

arise that parallel issues that appear in the dynamics of financial capital. To what extent do the precautionary motives to maintain "buffer stocks" of assets also operate on health capital? Is there any analog of the bequest motive in management of financial capital? We know that optimal life-cycle consumption jumps at retirement due to regime change in the consumption of leisure. Does this regime change also affect the productivity of health capital; for example, are leisure and health capital substitutes or complements? Can we as a result expect to see structural breaks in medical expenditures at the time of retirement?

Case and Deaton use SRHS as an indicator for health capital and show in a time series of cross sections that bad SRHS is associated with hard work, low income, and low education. Work and income associations strengthen with age; however, the reverse is true for education. There are sex differences, with females reporting somewhat worse health than males until old age, where the paths cross. They conclude that these patterns are inconsistent with the simple Grossman model for health capital and that the model of medical technology implicit in Grossman is wrong. I agree. The technologies implicit in survival models and some models of physical capital facing multiple hazards provide more plausible alternative starting points for life-cycle models with health capital.

How good is SRHS as an indicator of health capital? In analysis of AHEAD data, I find that an indicator for Poor/Fair SRHS (hereafter, P/F-SRHS) is predictive of future incidence of health conditions and of mortality; see Adams and others (2003). The Good/Very Good/Excellent gradient is not predictive. This may be a reporting effect, or if SRHS is a good indicator of health capital, may reflect sharply diminishing productivity of health capital above a threshold.

Baker, Stabile, and Deri (2001) find in Canada overreporting of health impairments among the unemployed, using medical records as a benchmark, a justification effect. Thus, SRHS may be susceptible to justification effects. In AHEAD, I find that P/F-SRHS is strongly associated with clinical depression and with a dwelling rated Poor/Fair, even with statistical control for overall socioeconomic status. This again suggests that reporting effects may influence SRHS.

Sex differences in SRHS may be largely due to plumbing differences and to the incidence and detection of problems that arise during the female reproductive years. In addition, my internist tells me that he sees sex-linked cultural differences, with females complaining more freely about health problems, males suppressing them. This may also be a factor in SRHS reports.

In Adams and others (2003a,b, 2003), probit models are estimated for prevalence and for incidence of P/F-SRHS, given objective health status indicators and a variety of measures of socioeconomic status. Tables 6C.1 and 6C.2 reproduce these models for the AHEAD panel. Consider first the explanation of prevalence in table 6C.1. For both females and males,

Table 6C.1 Prevalence of P/F-SRHS, AHEAD wave 1

	Females		Males	
Variable	Coefficient	*t*-statistic	Coefficient	*t*-statistic
One	–1.046	–4.18	–0.440	–1.21
Age since 70	0.002	1.85	0.004	3.62
Age since 80	–0.004	–3.31	–0.010	–5.20
Lowest quartile wealth	0.059	0.83	0.195	2.13
Highest quartile wealth	–0.215	–2.85	0.035	0.39
Lowest quartile income	0.036	0.51	–0.031	–0.33
Highest quartile income	–0.049	–0.67	–0.071	–0.80
High school	–0.149	–2.37	–0.127	–1.64
College	–0.308	–3.38	–0.239	–2.36
P/F neighborhood	0.121	1.44	0.233	2.06
P/F dwelling	0.368	4.51	0.460	4.33
Never married	–0.301	–1.81	0.216	1.06
Widow	–0.090	–1.47	–0.170	–1.77
Divorced/Separated	–0.046	–0.35	–0.129	–0.69
Mother death age	–0.001	–0.62	–0.002	–0.75
Father death age	–0.002	–1.12	–0.002	–0.69
Ever smoke	0.031	0.49	0.020	0.24
Cancer	0.207	2.74	0.295	3.34
Heart disease	0.0651	11.33	0.585	8.61
Stroke	0.347	3.47	0.405	3.55
Lung disease	0.609	7.19	0.747	8.32
Diabetes	0.547	6.32	0.253	2.58
High blood pressure	0.234	4.26	0.034	0.49
Arthritis	0.322	5.26	0.289	3.41
Incontinence	0.177	2.86	0.188	1.86
Fall	0.100	1.13	–0.064	–0.41
Hip fracture	0.144	1.35	–0.189	–0.96
Proxy respondent	0.257	2.19	0.306	2.77
Cognitive impairment	0.206	2.91	0.339	3.78
Psychiatric disease	0.016	0.20	–0.001	0.00
Depression	0.915	10.05	0.814	5.37
BMI	–0.005	–0.77	–0.032	–3.31
Smoker now	0.217	2.10	0.099	0.92
No. of ADL impairments	0.189	6.59	0.154	3.93
No. of IADL impairments	0.077	2.09	0.134	2.93
Likelihood	–1,449.1		–945.3	
	Count	Percent	Count	Percent
Observation				
Negative	2,079	66.06	1,351	66.65
Positive	1,068	33.94	676	33.35

Table 6C.2 Incidence of P/F-SRHS, AHEAD wave 1 to wave 3

	Females		Males	
Variable	Coefficient	*t*-statistic	Coefficient	*t*-statistic
One	–2.163	–1.71	–6.543	–3.78
Log months between waves	0.738	1.91	1.769	3.32
Age since 70	–0.001	–0.55	0.001	0.64
Age since 80	–0.001	–0.50	–0.005	–1.50
Lowest quartile wealth	0.066	0.72	–0.002	–0.02
Highest quartile wealth	–0.065	–0.61	0.123	0.88
Lowest quartile income	0.189	2.07	0.222	1.61
Highest quartile income	0.017	0.16	–0.018	–0.13
High school	–0.082	–0.99	–0.133	–1.14
College	–0.083	–0.61	0.096	0.57
P/F neighborhood	–0.022	–0.21	–0.088	–0.59
P/F dwelling	0.041	0.41	0.328	2.22
Never married	0.264	1.10	–0.170	–0.55
Widow	–0.173	–2.11	–0.375	–2.61
Divorced/Separated	–0.074	–0.42	–0.009	–0.03
Mother death age	–0.001	–0.61	0.000	0.05
Father death age	0.001	0.37	0.008	2.46
Ever smoke	0.076	0.90	0.102	0.82
Preexisting impairments				
Cancer	–0.015	–0.14	0.054	0.44
Heart disease	0.134	1.80	0.092	0.91
Stroke	–0.119	–1.08	0.116	0.77
Lung disease	0.259	2.49	0.496	3.90
Diabetes	0.018	0.19	–0.031	–0.23
High blood pressure	0.024	0.30	0.301	2.82
Arthritis	0.146	1.81	0.268	2.12
Incontinence	–0.032	–0.36	–0.156	–1.12
Fall	0.094	0.94	0.015	0.08
Hip fracture	0.384	2.73	0.042	0.15
Proxy respondent	–0.063	–0.38	0.028	0.14
Cognitive impairment	0.065	0.68	0.168	1.27
Psychiatric disease	0.196	1.90	–0.239	–1.53
Depression	0.266	2.67	0.042	0.21
Smoker now	0.001	0.01	0.155	0.90
High BMI	–0.010	–0.89	–0.036	–1.85
Low BMI	–0.158	–0.75	0.188	0.74
No. of ADL impairments	0.000	0.00	0.073	1.29
No. of IADL impairments	–0.053	–1.17	–0.078	–1.21
New impairments				
Cancer	0.260	1.27	0.566	2.49
Heart disease	0.247	2.42	0.511	3.58
Stroke	0.463	2.56	–0.070	–0.30
Lung disease	0.564	2.64	0.074	0.31
Diabetes	0.071	0.68	0.147	0.51
High blood pressure	0.143	0.82	0.309	1.27
Arthritis	0.210	1.82	0.062	0.38

Table 6C.2 (continued)

	Females		Males	
Variable	Coefficient	*t*-statistic	Coefficient	*t*-statistic
Incontinence	0.092	1.00	0.076	0.56
Fall	0.019	0.19	0.152	0.85
Hip fracture	–0.330	–1.60	0.216	0.43
Proxy respondent	–0.058	–0.40	0.035	0.18
Cognitive impairment	–0.040	–0.35	–0.033	–0.21
Psychiatric disease	0.103	0.69	0.272	0.92
Depression	0.300	1.94	0.089	0.37
BMI better	0.041	0.43	0.166	1.28
BMI worse	–0.120	–1.29	0.234	1.72
Smoke now	0.277	1.22	0.041	0.15
No. of ADL impairments	0.214	6.78	0.141	2.74
No. of IADL impairments	–0.049	–1.29	0.046	0.69
Likelihood	–822.73		–451.73	
	Count	Percent	Count	Percent
Observation				
Negative	420	25.81	244	26.24
Positive	1,207	74.19	686	73.76

prevalence of cancer, heart disease, stroke, lung disease, diabetes, arthritis, cognitive impairment, depression, ADL limitations, and IADL limitations are associated with P/F-SRHS. In addition, high blood pressure for females and low body mass index (BMI) for males are associated with P/F-SRHS. Thus, P/F-SRHS appears to correlate well with objective health impairments. The very strong association of depression and P/F-SRHS may reflect, in addition, a perception or reporting effect that could reduce the reliability of SRHS as an overall measure of health capital. Socioeconomic variables show some association with SRHS, with the prevalence of P/F-SRHS higher when socioeconomic status (SES) is lower for both females and males. The measurement problem is to disentangle true links between SES and health capital coming out of life-cycle planning and use of medical technology, and spurious associations arising from reporting effects.

Table 6C.2 is a model for incidence of new P/F-SRHS between waves 1 and 3 of the AHEAD panel, a period of about five years, as a function of preexisting SES and health status, and of incidence of new objective health impairments between the waves. In this model, at least some spurious reporting effects are controlled, and the model can be interpreted more plausibly as giving structural, causal effects of incidence of new objective health conditions or changes in SES. The pattern that emerges is that for both females and males some preexisting chronic conditions, such as lung disease,

and some new acute conditions, such as cancer, heart disease, and ADL impairment, appear to induce a transition into P/F-SRHS. In addition, for females, preexisting hip fracture, depression, and new stroke, induce P/F-SRHS, while for males, preexisting high blood pressure and arthritis induce P/F-SRHS. The fact that all serious health impairments are not significantly related to incidence of P/F-SRHS is probably a consequence of the modest frequency with which various detailed impairments appear over a five-year period. There are weak impacts of SES on incidence of P/F-SRHS, notable primarily because these impacts are absent for incidence of most objective health impairments. The good news to be drawn from these models is that SRHS relates strongly to objective health conditions, meeting the one criterion for a measure of health capital that it be predictive for objectively measured health status. The bad news is that the strong dependence of P/F-SRHS on depression, and to a lesser extent on SES, may reflect reporting effects as well as the interdependence of health and financial status that life-cycle decisions on health investment would induce. Careful measurement and analysis will be needed to isolate reporting effects and construct health capital variables that capture the real health-wealth interactions embedded in the dynamics of life-cycle behavior.

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