

Health Disparities and Medical Technology*

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Abstract. Better-educated people are healthier, but the magnitude of the relationship between health and education varies substantially across groups and over time. We undertake a theoretical and empirical study of how health disparities by education vary according to underlying health characteristics and market forces. Consumer theory suggests that improvements in the productivity of health care will tend to confer the most benefits upon the heaviest health care users. Since richer and more educated patients tend to use the most health care, this suggests that new technologies—by making more diseases treatable, reducing the price of health care, or improving its productivity—will tend to widen disparities in health. On the other hand, by the same reasoning, new technologies that are “timesaving” can contract health disparities if they lower the productivity of patients’ time investments in health. These ideas help us make sense of several empirical patterns. First, compared to healthy people, the chronically ill exhibit wider disparities, but the terminally ill exhibit narrower ones. Second, the advent of complex new HIV technologies increased immune function among HIV patients, but seemed to benefit educated patients disproportionately. In contrast, however, new drugs for hypertension lowered health inequality, by making investments in diet, exercise, and weight control much less important for hypertension control.

A. Introduction

Better-educated people are healthier. This is one of the most robust empirical findings in health. Farrell and Fuchs (1982) argue that this relationship is “one of the strongest generalizations to emerge from empirical research on health in the United States.”

Socioeconomic status in general has been shown to affect mortality and morbidity in a number of studies (Smith, 1999; Marmot, 2000). In particular, the association between education and health is pervasive and quantitatively large (Kitagawa and Hauser, 1973; Feldman et al., 1989; Pappas et al., 1993; Preston and Taubman, 1994; Schoenbaum and Waidmann, 1997). A distinguished theoretical literature has suggested various sources for these correlations. In an early paper, Welch (1970) analyzed the general relationship between education and productive efficiency, but Grossman (1972) was among the first to present an explicit theoretical model linking schooling and health.¹ His hypothesis has been questioned by Fuchs (1982) and others, who suggest that it is primarily unobserved factors such as time preferences that explain both health and education. The empirical work using instrumental variables is not definitive.² Despite this large body of work,

¹ For an earlier discussion of the similarities and differences between education investments and health investments, see Mushkin (1962). Muurinen (1982) provides a later, more general formulation of Grossman’s model.

² Kenkel (1991) finds that health knowledge explains part, but not the majority, of the relationship between schooling and deleterious personal behavior such as smoking and drinking. Berger and Leigh (1989) use a two-stage selection model to demonstrate a causal link between schooling and health. Arkes (2000) uses state-level variation in unemployment rates as an instrument for schooling, to show that schooling has a causal effect on health. Lleras-Muney (2000) uses compulsory schooling laws as an instrument for schooling attainment, to show that schooling lowers mortality. In general, while the literature supports a causal link from schooling to health, it cannot rule out the presence of additional mechanisms.

virtually no research investigates how the relationship between education and health varies with market forces, across time, and across different population groups. This makes it very difficult to understand why health disparities seem to have widened considerably over the past several decades, as shown in Figure 1. The figure plots the difference in death rates (per 1000 people) between high school dropouts and college attendees (people with at least one year of college) for various white male age groups, in 1960 and 1998. The values on the y-axis should be interpreted as the additional deaths (per 1000 people) suffered by high school dropouts relative to college attendees. Over this 38-year period, the absolute gap in death rates among 25-34 year-olds has doubled; it has increased by fifty percent for individuals aged 45 to 64, and it has grown by about a quarter for 35-44 year-olds.

Several simple explanations for this trend are incomplete. From 1960 to 1998, the proportion of high school dropouts fell dramatically, and this may have produced a group of dropouts with lower average ability. However, the widening of disparities can be seen when one uses population groups of fixed sizes (such as quartiles) based on income; since the relative size of each group is fixed, the relative ability of each group ought to remain constant in this analysis. Second, while there has been some general widening of wage and income differentials across education groups, this has not been nearly as widespread as the widening of health disparities. For example, wage differentials between high school graduates and dropouts did not change much over this period, while mortality disparities clearly have widened significantly.

There is great concern about these disparities in the public health community, and the common explanations are restricted access to care or poorer quality of care for the less

educated (Bindman et al., 1995; Andrulis, 1998). This paper attempts to understand these changes using some simple—but important—economic insights about patient behavior. A basic model of health investment in which people differ only by their wages yields some important implications for disparities. One key consequence—which is really a simple application of Roy’s Identity—is that reductions in the price of a good benefit the good’s heaviest consumers the most. Accordingly, reductions in the price of health care or improvements in its productivity benefit the heaviest health care users, who will also tend to be the higher wage, more educated people. As a result, improvements in health care widen disparities. One of the most important and attractive features of this argument is that its implications do not depend on the specific causal link between schooling and health. The only requirement is a positive correlation between schooling and wages.

This simple insight helps generate two important hypotheses concerning the relationship between medical technology and health disparities. First, populations that have access to effective medical treatments—and thus enjoy a higher productivity of health care inputs—are likely to exhibit wider disparities. For example, people with chronic, treatable illnesses may exhibit wider disparities than healthy people who have less need for doctors or drugs. In contrast, an untreatable illness can be a great equalizer by reducing the productivity of all health inputs. In Section C.1, we present some suggestive evidence consistent with these ideas. Second, technological innovation lowers the price of health care and expands health disparities, but technologies that simplify the production of health can sometimes contract them. Simplifying health production disproportionately benefits the less educated, if the educated patients are the ones more likely to engage in complex, time-intensive health investments. For example, the typhoid vaccine made it much easier to prevent typhoid, *without* spending a great deal of time boiling water, washing fresh vegetables,

maintaining a high level of hygiene, and so on. If educated people are more likely to make these kinds of time investments, the advent of the vaccine would have hurt them disproportionately by making redundant these investments. This is the converse of the statement that improvements in input productivity benefit the heaviest users of the input. In Section C.2, we explore these hypotheses by investigating two very different technological breakthroughs in medicine, one of which (Highly Active Antiretroviral Therapy for HIV sufferers) tended to make health care more complex, while the other (beta-blockers in the treatment of hypertension) tended to simplify it. These two examples alone cannot provide a definitive test of a broad hypothesis about health disparities, but they illustrate how these simple theoretical insights can be brought to bear on concrete episodes of technological change in medicine.

B. A Theory of Health Disparities

While the causal links between schooling and health remain unclear, it is nearly certain that more educated people tend to earn higher wages and that this gives them stronger incentives to invest in health. An example helps illustrate the value of this simple assumption. Suppose that person E has stronger incentives to invest in health than person U, and accordingly that E uses 2 units of health care, but U uses only 1. If the price of health care falls by \$1 (or the marginal productivity of health care rises by \$1) E receives a windfall gain of \$2, but U's gain is only \$1. E will parlay her disproportionate monetary advantage into a disproportionate gain in health, because on the margin, she spends a

larger fraction of her income on health.³ This example also illustrates why our results do not depend on the specific causal link between schooling and health. It is driven entirely by the fact that better-educated people invest more in health. It does not matter whether schooling itself makes them better producers, or whether they are better because they are more forward-looking, more able, or because of some other factor.

B.1 A Simple Model of Health Investment

The simplest case assumes that the only way to improve health is to buy units of it at the price π . Increases in health provide the individual with more time for labor, but the returns to health diminish so that labor is given by H^γ , where $\gamma < 1$. In this context, growth in γ is a simple way to think about technological change. If technology improves—i.e., γ increases—the person has more time available for work or play, both of which are valued at the wage w . Faced with this problem, the consumer maximizes income as follows:

$$\max_H wH^\gamma - \pi H \tag{1}$$

The optimal investment in health satisfies:

$$H = \left(\frac{w\gamma}{\pi} \right)^{\frac{1}{1-\gamma}} \tag{2}$$

The gradient in health across wage groups is thus given by:

³ On the margin, health care is equally productive for E and U in equilibrium. Therefore, additional health care consumption will not benefit one person more than the other, to a first-order approximation.

$$\frac{dH}{dw} = \frac{1}{1-\gamma} w^{\frac{\gamma}{1-\gamma}} \left(\frac{\gamma}{\pi}\right)^{\frac{1}{1-\gamma}} \quad (3)$$

This expression has two important implications. First, when health care becomes more expensive (i.e., when π rises), the gradient compresses. Technological innovations that lower the quality-adjusted price of medical care thus expand this gradient. Second, when the technology of health production improves, so that γ rises, the gradient also expands.

Essentially, this is an income effect—if the richer and better educated patients are investing more in health, they will receive a larger windfall from a price reduction.

These results hold even when health is a consumption good that enters the utility function, albeit with one *caveat*. It will continue to be true that richer, more educated people consume more health, and the heaviest health consumers receive the most additional income from a price reduction. There is only one complication: if richer people have a much lower “taste” for health (in terms of relative marginal utilities), they may spend a smaller fraction of their additional income on health, and this could offset the income effect. While this is impossible to rule out theoretically, health is a durable good and will be valued most highly by those with the longest time-horizons, and richer, more educated people tend to live longer (Feldman et al., 1989) and discount the future less heavily (Becker and Mulligan, 1997).

These results are easiest to see in the case of a homogeneous utility function,

$U(c, H) = c^\lambda H^{1-\lambda}$, where the marginal rate of substitution—i.e., the “taste” for health over consumption—is independent of income. The consumer now solves:

$$\begin{aligned} \max c^\lambda H^{1-\lambda} \\ \text{s.t. } c + \pi H \leq wH^\gamma \end{aligned} \quad (4)$$

This problem has the first order condition:

$$\frac{1-\lambda}{\lambda} \frac{c}{H} = \pi - w\gamma H^{\gamma-1} \quad (5)$$

Performing comparative statics on this first order condition yields:

$$\frac{dH}{dw} = H \left(\frac{\gamma + \frac{1-\lambda}{\lambda}}{\frac{1-\lambda}{\lambda} w(1-\gamma) + w\gamma(1-\gamma)} \right) \quad (6)$$

Disparities in health—i.e., $\frac{dH}{dw}$ —widen with price reductions, which increase H . They also widen with increases in the productivity of health technology—i.e., gamma.

B.2 Time Investments in Health

New technologies may not raise the productivity of all health investments. Timesaving innovations can lower the productivity of time invested in health; this can have different effects on disparities. Diabetes provides a constructive example. Patients with type 1 disease must take insulin (either through injection or a pump) to prevent glucose from building up in their blood. The ideal titration can be difficult to achieve. Too much insulin will lower blood sugar below the normal range and can result in acute hypoglycemia and, in rare cases, coma. Persistently high levels raises the likelihood of long-term complications such as kidney failure, neurological problems, eye disease, and cardiovascular disease.

Now consider a new medical technology for diabetics—the development of home monitoring kits to measure blood glucose levels. These devices allow patients to maintain tighter glycemic control, but only if they are willing to monitor blood glucose several times a day by using a small lancet to draw blood from their finger and then processing the sample in a

portable device. The process requires not only a willingness to undergo this procedure several times a day, but also the ability to adjust insulin accordingly once a reading has been obtained. This new technology can (and likely has) increased disparities (Goldman and Smith, 2002).

However, not all diabetes-related innovations need work this way. The development of new oral agents such as metformin have been shown to lower blood sugar without causing acute hypoglycemia in Type 2 diabetes. This drug comes in an extended release form that requires the patient to take the medication once per day. Such drugs require little investment on the part of the patient and might be expected to have different effects on disparities than home monitoring kits.

To model these different types of medical technology, we make health production a function of medical care and the individual's own time. Formally, health is produced at constant returns to scale⁴, using the technology $H = F(t, m)$, where t is time invested in health, and m is medical care purchased at the price π .⁵

Since production is constant returns to scale, the average cost of producing health ($\frac{C}{H}$) is equal to marginal cost (c)—i.e., $C(H; w, \pi) = Hc(w, \pi)$. Increases in input prices will require at least as much expenditure to maintain health, so that that $c_w, c_\pi > 0$. The individual maximizes income according to:

⁴ This restriction is not entirely necessary. Because the scaling of health (H) is arbitrary, this really is tantamount to requiring only non-increasing returns to scale.

⁵Health is used interchangeably with health investment since the model is one-period.

$$\max_H wH^\gamma - Hc(w, \pi) \quad (7)$$

Optimal health investment is now characterized by,

$$H = \left(\frac{\gamma w}{c(w, \pi)} \right)^{\frac{1}{1-\gamma}}, \quad (8)$$

and the gradient in health is given by,

$$\frac{dH}{dw} = \frac{\gamma}{1-\gamma} H^\gamma \frac{c(w, \pi) - wc_w}{c(w, \pi)^2}. \quad (9)$$

Since cost functions are concave in their input prices, $H(c(w, \pi) - wc_w) > 0$. As a result,

increases in the wage always encourage health investment, or $\frac{dH}{dw} > 0$. A useful

reinterpretation of the gradient in health is permitted by Shephard's Lemma, which shows that the derivative of the cost function with respect to wages will equal input demand (t).

Multiplying through by wages yields $wC_w = wt$. Since costs may be written as $wt + \pi m$ —

and noting that ρ_m is the cost share of medical care—we can rewrite $\rho_m = \frac{c - wc_w}{c}$, and

$$\frac{dH}{dw} = \frac{\gamma}{1-\gamma} \frac{H^\gamma}{c} \rho_m \quad (10)$$

Health disparities rise with health investment H and they fall with the marginal cost of health. Improvements in the production of health will increase disparities. In the two input case there is also an additional, sometimes offsetting, force: disparities can also rise if the share of resources spent on medical care goes up. Wage increases have two offsetting effects in this model—they encourage investments in health by making healthy time more valuable, but they also increase the (time) cost of producing health. If the cost share of

medical care rises, the first effect is lessened and the overall effect of wages on health becomes more positive.

Factor-neutral technological will widen health disparities, just as in the single input model. This type of technological change shifts out the function F , but does not change the ratio of marginal products $\frac{F_m}{F_t}$. For example, a new radiotherapy procedure for cancer patients might reduce mortality by 50%, and it may only require half as many treatment sessions. This raises H , lowers marginal costs c , and leaves the cost share of medical spending unchanged, because it does not affect the optimal input ratio. Improvement in the production of health confers the greatest benefits on the high-wage individuals who are using the most health care.

A reduction in the price of medical care—e.g., through technological change—also raises disparities. Clearly, the price reduction lowers marginal costs c and raises health investments H . It has an uncertain effect on ρ_m , and this could in theory offset the other two effects. However, as long as ρ_m is sufficiently bounded away from zero, the effect on cost shares cannot be large enough to dominate. A sufficient condition for a price reduction to raise disparities is that ρ_m be confined to the interval $[\frac{1-\gamma}{2-\gamma}, 1]$.⁶

⁶ When π rises, the maximum possible growth in ρ_m is that which involves no change in

m . This is characterized by: $\frac{d\rho_m}{d\pi} \frac{1}{\rho_m} = \frac{1-\rho_m}{\pi}$. Since $\frac{H^\gamma}{c} = \frac{(\gamma w)^\frac{\gamma}{1-\gamma}}{c^\frac{1}{1-\gamma}}$, Shephard's Lemma

The most important cases relate to factor-biased technological change. Suppose a new treatment makes medical care more productive without affecting the productivity of time, so that $\frac{F_m}{F_t}$ rises. Examples might include a less-invasive and surgical procedure to remove malignant tumors. Since the new technology expands the set of production possibilities, it will lower marginal costs and raise health investment. The share of costs accounted for by medical care will rise. All these forces—price reductions, technological progress, and growth in the cost share of medical care—work to raise disparities in health.

On the other hand, a new technology can fundamentally alter the technology of treatment by simplifying health investments. In this case, F_m rises, but since time investments could actually become less useful, F_t may fall. A vaccine against infectious disease represents a potential example of this effect. In such a case, the new technology can actually compress health disparities. However, in our simplified setting, only timesaving innovations that simplify health care and lower the productivity of time investments can compress health disparities, and only if their timesaving effects offset their price reductions and their increase in the productivity of medical care.

We were able to derive all these results without relying on differences in productivity between educated and uneducated individuals. Adding these differences to the model would merely reinforce the implications we derive. In particular, in this case a reduction in

can be used to show that $\frac{\frac{d}{d\pi} \frac{H^\gamma}{c}}{\frac{H^\gamma}{c}} = \frac{1}{1-\gamma} \frac{\rho_m}{\pi}$. This term is larger than the maximum possible change in ρ_m under the condition in the text.

the productivity of time investments would compress health disparities by even more, since it would affect the high-wage, high-productivity individuals the most.

B.3 Example: Cobb-Douglas Production

A specific example using Cobb-Douglas health production can help illustrate the results of the previous section. We can most easily incorporate diminishing returns to health investment by defining H as “healthy time available for labor” and specifying that $H = m^\alpha t^\beta$, where $\alpha + \beta < 1$. The individual maximizes income:

$$\max_{m,t} wm^\alpha t^\beta - \pi m - wt \quad (11)$$

From the first order conditions, we can derive the input demand functions,

$$\begin{aligned} t &= \left(\frac{w\alpha}{\pi} \right)^{\frac{\alpha}{1-\alpha-\beta}} \beta^{\frac{1-\alpha}{1-\alpha-\beta}}, \\ m &= \left(\frac{w\alpha}{\pi} \right)^{\frac{1-\beta}{1-\alpha-\beta}} \beta^{\frac{\beta}{1-\alpha-\beta}}, \end{aligned} \quad (12)$$

and equilibrium health investment,

$$H = \left(\frac{w\alpha}{\pi} \right)^{\frac{\alpha}{1-\alpha-\beta}} \beta^{\frac{\beta}{1-\alpha-\beta}}. \quad (13)$$

Gradients in health are defined by:

$$\frac{\partial H}{\partial w} = \frac{\alpha}{1-\alpha-\beta} w^{\frac{2\alpha+\beta-1}{1-\alpha-\beta}} \left(\frac{\alpha}{\pi} \right)^{\frac{\alpha}{1-\alpha-\beta}} \beta^{\frac{\beta}{1-\alpha-\beta}} \quad (14)$$

This expression implies all the key results. Disparities fall with the price of medical care, π , and rise with improvements in the productivity of medical care, α and β . In addition,

timesaving innovations that lower β can compress disparities. The only innovations that can compress disparities, therefore, are the timesaving innovations that lower β , the productivity of time spent on health.

C. Empirical Analysis

The basic insight of the preceding analysis is that improvements in the productivity of health care tend to favor its heaviest users. The diversity of health and health care makes it impossible to test this implication comprehensively, but we present some examples of empirical patterns that are made clearer in light of the theory. First, we look at the different impacts of treatable and untreatable illness on disparities. Second, we explore the impact of two rather different technological breakthroughs, both of which improved the productivity of health care, but only one of which simplified its delivery.

C.1 Chronic Illness and Disparities

Individuals with chronic, treatable illnesses are likely to benefit more from medical care than those without any such conditions, but people with untreatable illnesses are likely to benefit less. If so, one would expect that populations with treatable conditions ought to exhibit wider disparities, and that the opposite would hold for those with untreatable illness. This is an important issue, because new technologies that make more diseases treatable are also likely to widen disparities among patient populations.

C.1.1 TREATABLE ILLNESS

We compare disparities in a population with chronic illness to disparities in a population without any. To do so, we use individual-level data from the National Health Interview Surveys (NHIS), an annual survey, conducted every year since 1957, in which individual

respondents are asked various questions about their health, economic, and demographic conditions. We pool the surveys for every year from 1982 to 1996, incorporating year-specific fixed-effects.⁷

To separate the NHIS sample along the lines of chronic illness, we identify five major chronic, treatable illnesses: hypertension, diabetes, asthma, arthritis, and heart disease (defined as the presence of any of the following: ischemic heart disease, heart rhythm disorders, congenital heart disease, or other non-hypertensive diseases of the heart).

Unfortunately, while the NHIS asks respondents about a variety of chronic illnesses, not every respondent is asked about every illness. There are six non-overlapping lists of illnesses; each list is asked of a one-sixth sub-sample. Each individual is thus asked about conditions on one of the six lists. We can identify a set of individuals that definitely have a particular condition, but we cannot identify the complete set of individuals that do not have the condition, because we cannot rule out the possibility that some sick people were not asked about their particular condition. To separate the population along illness lines, therefore, we adopt the following conservative procedure: we define as chronically ill people who definitely report having at least one of the five conditions. All other people in the sample are taken to be the “not chronically ill” population, which *could* include people with one of these five illnesses. This will bias us against finding a difference in health disparities across these populations.

From the NHIS, we will use data on years of schooling, age, sex, and self-reported general health status. General health is measured on a 5-point scale: each individual in the NHIS

⁷ Prior to 1982, the NHIS used a different general health measure. After 1996, it used a different scheme for identifying the chronically ill.

sample is asked whether her health is excellent, very good, good, fair, or poor.⁸ From these variables, we construct a binary variable called *GoodHealth*, which is one if the individual reports good, very good, or excellent health. We will explore whether or not our results are sensitive to restricting this definition to, say, very good or excellent health, or just excellent health. (It does not make sense to expand the definition further, since there are relatively few people who report poor health.) From the years of schooling variables, we construct three dummy variables: *HighSchool*, which is one if the individual has exactly 12 years of schooling; *SomeCollege*, which is one if she has more than 12 years, but less than 16; *College*, which is one if she has at least 16 years of schooling. In general, we estimate the within-year health returns to schooling by regressing *GoodHealth* on age, sex, and our three educational dummies:⁹

$$GoodHealth = \beta_0 + \beta_1 HighSchool + \beta_2 SomeCollege + \beta_3 College + \beta_4 Age + \beta_5 Sex + \varepsilon \quad (15)$$

In most cases, these regressions will be run separately for 5-year age intervals. In these cases, we will enter age linearly. In other cases, data from all age groups will be pooled, and we will use dummy variables for narrow (five years or less) age categories, or age splines.

Table 1 compares health disparities for the chronically ill and healthy populations. Within the “Not Chronically Ill” and “Chronically Ill” panels, each row shows the results of a single regression for the given age group of good health on the three listed education dummies, a

⁸ While there are always questions about whether self-reported health is correlated with objective health, a body of research suggests that it is correlated with outcomes like mortality (Maddox and Douglass, 1973; Ferraro, 1980; Idler and Benyamini, 1997).

⁹ By “within-year,” we mean that year-specific fixed-effects are included in the regression.

linear age term, and a dummy for sex. Within nearly every age group, health disparities are roughly twice as high for the chronically ill than for the healthy. Not surprisingly, the magnitude of the difference is declining with age—one would expect the prevalence of unobserved chronic illness among the healthy population to rise with age. Among the youngest populations, however, disparities are about two and a half times as high for the chronically ill than for the healthy.

To explore the robustness of these results, we must confront two potential problems. First, it is possible that variation in the educational gradient reflects “diagnosis bias.” Suppose that more educated people are diagnosed with chronic illnesses earlier and in milder forms. Among the chronically ill, therefore, the educated would tend to be relatively healthier. To rule out this explanation, we use another measure of health from the NHIS: whether or not an individual reports that he is able to work. The bias with this measure will be in the opposite direction, since educated people will have the most incentive to stay in the labor force, even if they are ill. On the basis of their responses to a series of questions about their ability to work, the NHIS classifies respondents aged 18-69 in one of four categories: unable to work because of a health condition, limited in the kind or amount of work because of a health condition, limited in non-work activities only, and not limited at all by health conditions. We classify an individual in either of the first two categories as being “unable to work” because of a health condition. Once again, we are adopting the conservative procedure of including people who might be only slightly impaired in our “unable to work” category.

Table 2 presents the results of this breakdown. The results are essentially the same as for the chronic illness analysis. At every age and level of schooling, health disparities are greater for those unable to work than those who are. For the younger age groups, the

gradient is about three times as steep for those that are unable to work; for the older age groups, it is nearly twice as steep. It is sensible that the difference between the two groups is more pronounced for the young, because the actual difference in health across groups is probably most pronounced among the young. Among the elderly, even those who pronounce themselves “able to work” may not be in perfectly robust health.

The second problem requires us to rule out the possibility that the ability-schooling gradient is steeper for the chronically ill. To address this problem, we will use an instrument for schooling that we believe is uncorrelated with ability, time preference, or other plausible factors that jointly determine health and schooling. In particular, we use an individual’s quarter of birth, whose use was first suggested by Angrist and Krueger (1991) in the context of estimating the wage returns to schooling. Angrist and Krueger argue that children start school at different ages, depending on their season of birth, but compulsory schooling laws cease to bind at a particular age. As a result, the number of compulsory schooling years varies with a child’s quarter of birth. In particular, children born earlier in the year tend to end up with less educational attainment than those born later in the year.

This instrument requires us to confront two issues. First, samples above 100,000 observations are required in order to identify the correlation between quarter of birth and educational attainment with the required precision. Therefore, we pool our data across age groups, and estimate the effect of age as a piecewise linear spline. We are also limited to estimating the effect of years of schooling attained, rather than the effect of three educational dummies, because the instrument lacks the power to identify all three categorical variables. Second, since age affects health, and quarter of birth influences age, this could invalidate the instrument. However, this is unlikely to be a problem here. A

simple regression of good health on sex, education dummies, and single-year age dummies for the population aged 25 to 99 reveals that health significantly declines at adjacent age groups only 3 out of 74 times. In other words, even though health declines with age, it does not decline at the frequency of one quarter; indeed, it does not even decline at the frequency of one year.

Table 3 presents these results. The table shows the coefficients on sex, highest grade attained, and the coefficients on the piecewise linear age spline. The first two columns present the OLS estimates, while the second two present the IV estimates. In both cases, health disparities are about twice as large for the chronically ill. The IV estimates are also consistently larger than the OLS estimates; this is consistent with the findings of many other researchers. Card (1995) reports that nearly all researchers who estimate IV models of the monetary return to schooling find that the IV estimates are larger than the OLS estimates. Lleras-Muney (2001) uses state-level compulsory schooling laws as an instrument for the effect of schooling on mortality and also finds that the IV estimates are considerably and consistently larger than the OLS estimates. This may be because the effect of schooling is larger at lower levels of schooling, where the schooling instruments derive their explanatory power.

C.1.2 UNTREATABLE ILLNESS

It is not straightforward to identify an “untreatable” disease, because many diseases that can be treated often become untreatable at the end of life, and because diseases that seem treatable may not turn out to be (Garber, MaCurdy and McClellan, 1999). The best we can do, it would seem, is to analyze whether health disparities contract for people who are in their last year of life. While people in their last year of life clearly do not know this *ex ante*,

on average they will probably be facing conditions that are less treatable than elderly people who will live longer than one year.

We can perform these comparisons using individual-level data from the Medicare Current Beneficiary Survey (MCBS). The MCBS is a rotating panel survey, available from 1992 through 1998, designed to be representative of the Medicare population in the given year. It contains data on age, sex, education, self-reported health status (also on a scale of one to five), and most importantly, whether the respondent died in the year of the survey. Among the oldest segments of the MCBS population, deaths are common enough for us to construct reasonable samples. For example, pooling the 1992-8 samples, 2085 people over age 80 died during the survey year.

The data we use are summarized in Table 4. Among those in their last year of life, only about 30% report that they are in good health, while the proportion is about twice as high for other respondents. It is also not surprising that men are at greater risk of death, although the gap narrows with age. Finally, those in their last year of life are slightly less educated than other respondents, but these differences are not very large. Using Kolmogorov-Smirnov tests for the equality of distributions, we cannot reject at the 10% level that the distributions are equal for 85-90 year-olds and those over 90. We can reject this hypothesis for 80-85 year-olds, but not at the 5% level. This is consistent with other research that finds mortality gradients narrowing among the very old (Hurd, McFadden, and Merrill, 2001). The rough similarities of the education distribution provide us with some comfort that the ability-schooling distribution does not vary dramatically for those in their last year of life. Table 5 compares health disparities for those in their last year of life

to disparities for other respondents. Disparities among those in their last year of life are about 40 to 50 percent smaller than disparities among other respondents.¹⁰

C.2 Technological Breakthroughs and Disparities

Investigating two different examples of technological change in health care can help clarify the predictions of the model presented above. The effect of a new technology on health disparities depends on the nature of the innovation. An innovation that is not timesaving—in that it does not lower the marginal productivity of time investments—will tend to raise health disparities. However, by simplifying health care, or lowering the relative productivity of patient time, certain technologies can contract health disparities.

Antihypertensive drugs, perhaps the single biggest medical breakthrough of the past fifty years, greatly simplified the treatment of hypertension. Instead of exercising, watching their diet, and controlling their weight, hypertensive patients were able to take two pills in the morning to control their blood pressure. On the other hand, new HIV treatments have greatly improved immune function among HIV patients, but have not simplified its treatment.

C.2.1 BREAKTHROUGHS IN HYPERTENSION TREATMENT: BETA BLOCKERS

Perhaps the most important set of innovations in medical care over the past fifty years occurred in the treatment of heart disease. In 1960, roughly two-thirds of deaths were attributed to heart-related conditions, while by 1986 this had fallen to one-third.¹¹ In

¹⁰ The standard errors for the survivors regression are clustered by respondent, to account for the fact that some people enter this regression twice.

¹¹ 1960 data based on Kitagawa and Hauser (1973). 1986 data based on the 1986 National Mortality Followback Survey (NMFS).

particular, since 1970 there has been a substantial decline in mortality from conditions that are directly linked to hypertension. From 1970 to 1994, mortality from stroke fell by at least 50% across sex and race lines, while mortality for coronary heart disease fell by roughly the same amount (Joint National Committee on Prevention Detection Evaluation and Treatment of High Blood Pressure, 1997).

While the prevalence of uncontrolled hypertension actually rose from 1960 to 1971, it has declined steadily ever since then. During the 1960s, prevalence rose from 30% to 36%, but by 1988, it had declined to 20% (Burt et al., 1995). Much of the decline that began in 1972 was probably the result of new antihypertensive drugs. In 1965, a new drug called propranolol, a member of the class of drugs now called beta-blockers, was introduced in Europe. However, in the US, the FDA was slow to approve this drug. While propranolol was approved for a few minor uses in 1968, it was approved for the treatment of angina only in 1973, and for hypertension during 1976 (Ruwart, 1999). At roughly the same time, in 1967 and 1970, there emerged evidence from two clinical trials that diuretics and vasodilators could also treat high blood pressure effectively (Veterans' Administration Cooperative Study Group on Antihypertensive Agents, 1967; Veterans' Administration Cooperative Study Group on Antihypertensive Agents, 1970). Not coincidentally, mortality from heart disease began to fall from 1973 onwards and continued to fall until the early '90s, when mortality rates reached a plateau at about 50% below their initial level (Joint National Committee on Prevention Detection Evaluation and Treatment of High Blood Pressure, 1997). In terms of mortality reduction, it is possible that these rank as the most significant medical breakthroughs of the past 50 years.

The introduction of new drugs for the treatment of hypertension supplanted the former treatment of diet, exercise, and weight control (occasionally supplemented by the use of

diuretics). The previous treatment regimen placed significant emphasis on the patient's ability to spend time monitoring subtle variations in her health. The advent of the new drugs, however, made these time investments much less important in determining the effectiveness of treatment. As a result, complementarity between time and treatment likely fell, as did the productivity of time investments. This would suggest that these breakthrough drugs would have contracted disparities in the severity of hypertension. Previously, the educated would have been better able to control their blood pressure, but the decreased productivity of time investments would have lessened their advantage. To analyze the effect of antihypertensive drugs, we will use the Framingham Heart Study (FHS). The FHS tracked the health of a cohort of 5209 white men and women, aged 28 to 59 in 1948, and who resided in the town of Framingham, Massachusetts.¹² From 1948 onwards, this cohort received biennial medical exams, which also included interviews about health history and behavior. We will use data from the ninth exam, conducted in 1966, through the nineteenth exam, conducted in 1986. We have chosen only exams that post-date the enactment of Medicare, in order to net out the effects of Medicare on health disparities. We break up this period into two segments: 1966-72, the pre-beta blocker period; and 1980-86, the post-beta blocker period, which begins four years after the initial approval of beta-blockers in 1976.

At baseline, all individuals reported their age and educational attainment, which was broken down into six groups: less than 8th grade, some high school attended, high school

¹² Since the FHS is a local study, it is unable to track migrants from Framingham. However, we study 59 to 69 year-olds, for whom migration is a much less important possibility.

graduate, some college attended, college graduate, and those who attended graduate school, nursing school, art school, music school, or business school. Since they seem to behave similarly empirically, we group together college graduates with those who attended some college, or those who attended some post-high school institution.

There are several objective measures of health in the FHS that can be correlated with education: measured high blood pressure, a physician's diagnosis of hypertensive cardiovascular disease (HCVD)--defined as the presence of high blood pressure and an enlarged heart, a physician's diagnosis of any respiratory abnormalities, measured high blood sugar,¹³ and whether the individual's measured height and weight qualify them as obese. Hypertension is diagnosed on the basis of blood pressure readings, and the physician's judgement about whether the patient is being treated for hypertension. The respiratory abnormalities diagnosis is based on a physical examination and X-Rays. The HCVD diagnosis is based on blood pressure readings, X-Rays of the heart, and Electro-Cardiograms (ECG). While this is no longer a commonly used diagnosis category, HCVD is a subset of congestive heart failure, which is a commonly used diagnosis today.

Table 6 summarizes the characteristics of the pre-period and post-period cohorts. By design, the age and sex composition of the pre- and post-cohorts is quite similar. Because the age distribution in the Framingham sample is not exactly uniform, the later cohort is on average one year older than the earlier. Observe the 9 percentage point declines in hypertension and hypertensive cardiovascular disease. Much of this decline has been

¹³ Blood sugar readings in the Framingham Study are "casual" readings, in that they are not taken after a 12-hour fast. Framingham diagnosis protocols specify that such casual readings above 1.5 mg/mL are considered abnormally high.

attributed to the emergence of beta-blockers and other antihypertensives (Joint National Committee on Prevention Detection Evaluation and Treatment of High Blood Pressure, 1997). As we will see shortly, the reductions in high blood sugar and respiratory abnormalities turn out not to be statistically significant, and there is a significant increase in obesity. Therefore, hypertension and related disorders seems to be the only dimension along which the post-period cohort is healthier.

It is also important to understand changes in the educational distribution between the two cohorts. The post-period cohort shows a substantial increase in high school attendance and graduation—there is a shift in the distribution out of “no high school” and into the some high school and high school graduate groups. Presumably, therefore, the average ability of the “no high school” group would have fallen. However, the proportion of people with post-high school education barely moved. If anything, therefore, the gap in quality between the no high school and post-high school groups would have grown. This would bias us against finding that technology contracted health disparities across education groups.

Using these measures of health, along with dummy variables for education groups and age categories, we will estimate the following regression:

$$Health_{it} = \beta_0 + \beta_1 Ed_i * post_{it} + \beta_2 Age_{it} + \varepsilon_{it} \quad (16)$$

Ed_i represents a set of dummy variables for the individual’s education group; these are interacted with an indicator of whether the individual is in the pre- or post-period cohort. These regressions will allow us to investigate whether health disparities changed after the introduction of antihypertensive drugs. If beta-blockers were indeed timesaving, one might expect a lessening of health disparities in conditions related to uncontrolled hypertension.

The results of the regression are shown in Table 7. The coefficients on the interaction terms summarize the change in disparities: when the interactions are positive, disparities compressed, and vice-versa. There is a significant decline in hypertension and HCVD, but no significant declines in any of the other conditions (indeed, there is an increase in obesity). This is useful for our purposes, because we can compare disparities in hypertension to disparities in diseases that did not experience observable innovations in treatment over the same period. The table shows significant compression of the health gradient for hypertensive cardiovascular disease, and statistically insignificant compression of the gradient for hypertension. To understand why the results might be weaker for hypertension itself, observe that the advent of new drugs affects the prevalence of uncontrolled hypertension, but not hypertension itself. A patient on the new drugs is still considered “hypertensive” according to the Framingham diagnosis protocols, even though his disorder might be under control. Uncontrolled hypertension is more likely to result in complications such as HCVD. As a result, the gradient in HCVD may be more heavily influenced by treatment than the gradient in hypertension itself. For all other conditions, disparities expand if they change at all. There is some significant expansion in disparities for respiratory abnormalities and high blood sugar. Disparities in obesity show no clear pattern. This supports our earlier contention that differences in the educational distribution across cohorts will, if anything, lead to a generic expansion in disparities across these cohorts and work against our finding of compression.

The difference in trends across diseases suggests that the compression in disparities is not a generic feature of the pre- and post-cohorts. This provides further evidence against the argument that declines in the relative quality of high school graduates explain our results. It is also worth noting that there is relatively little evidence in the labor economics

literature to support the contention that the relationship between schooling attainment and quality has changed across cohorts (see, e.g., Katz and Murphy, 1992). In particular, changes in the wage premium seem to occur within cohorts rather than between cohorts. One last piece of evidence is that education gradients actually seemed to rise slightly between the 1958-64 period and the 1966-72 period. This is consistent with there being a generic *increase* in health disparities, before the effects of new technologies. This explanation is consistent with the increasing disparities in respiratory abnormalities and in high blood sugar.

C.2.2 BREAKTHROUGHS IN HIV TREATMENT: HIGHLY ACTIVE ANTIRETROVIRAL THERAPY

The treatment of HIV presents a useful example of technological change that did not simplify the delivery of health care and may have even made it more complex. During the mid-1990s, Highly Active Antiretroviral Treatments (HAART) became available for the treatment of HIV. These treatments substantially improved the health status of HIV patients, but they often involved highly complex medication regimens that required substantial patient adherence (Goldman and Smith, 2002). This makes it a perfect candidate for our purposes. Moreover, since it was introduced very rapidly during the mid-1990s, it is possible to identify periods with relatively low and relatively high exposure to HAART.

The data on the HIV positive population come from the HIV Cost and Services Utilization Study (HCSUS). HCSUS employs a multi-stage national probability sample design, described in detail elsewhere (Frankel et al., 1999). The HCSUS sample is representative of the 18 and older HIV positive population, which made at least one visit for regular care

in the contiguous United States in early 1996.¹⁴ Women and patients of private, staff-model HMOs are over-sampled. HCSUS is a panel data set with three rounds of interviews. The first round of 2864 interviews was conducted between January 1996 and April 1997, the period during which HAART were first coming into broad use. The second round of 2466 interviews was conducted between December 1996 and July 1997, and the last round of 2267 interviews was between August 1997 and January 1998. The first wave covers the period prior to the introduction of HAART, and the latter the period post-introduction.

In addition to other covariates, HCSUS collects data on educational attainment, health insurance status (Medicare, Medicaid, Private Insurance, or other Public Insurance), income, and whether or not a respondent was using HAART. Individuals can be placed into one of three groups that differ in the severity of the disease: asymptomatic patients, symptomatic patients, or patients with full-blown AIDS. HCSUS also reports an individual's CD4 T-lymphocyte cell count, a critical measure of the function of the patient's immune system. A depletion of these cells correlates strongly with the worsening of HIV disease and the risk of developing acquired immunodeficiency syndrome-defining opportunistic infection (Schechter, Moulton and Harrison, 1997). Other demographic data on income, sex, race, sexual orientation, exposure route(s), and age are also available.

Table 8 displays the characteristics of the HCSUS population. The average educational make-up of the HCSUS population does not change over time. The population does become sicker, as more people move out of the asymptomatic and symptomatic categories into the

¹⁴ The HCSUS sample does not include patients whose only contact with the health care system was through military, prison, or emergency department facilities.

full-blown AIDS category. People also tend to move out of the uninsured group into the publicly insured groups. However, immune function goes up.

At baseline, only about 25 percent of respondents with AIDS had ever been exposed to HAART treatment. However, just nine months later, over 60 percent of respondents had been exposed. At a minimum, physicians' cost of acquiring information about and prescribing HAART seems to have gone down precipitously during this period, as its dissemination suddenly became widespread. The wide dissemination of HAART in the second follow-up suggests that health disparities among HIV patients would have risen post-baseline.

Moreover, there seems to be little doubt that the adoption of HAART is not equal across education groups. By the second follow-up, approximately 68% of college graduates with AIDS (the final stage of HIV) had been exposed to HAART, while only 55% of high school dropouts with AIDS had been. Adherence to drug therapies is also not equal across groups. During the same follow-up, about 54% of high school dropouts with AIDS reported taking their medicines as prescribed every day over the last seven, while 67% of college graduates reported the same.

We will investigate changes over time in CD4 disparities across education groups. Consider the basic model:

$$CD4_{it} = \beta_0 + \beta_1 Sch_i + \varepsilon_{it}, \quad (17)$$

where Sch_i is a set of dummy variables indicating the educational group to which individual i belongs. Identifying the effect of technology on the coefficient β_1 requires that there are no other time trends over this short 2-year period that may also influence health

disparities. There are three important alternatives to consider. Since the HCSUS is a true panel data set, the first issue is differential mortality, which will probably work to contract health disparities, if its effects are as usual. Indeed, among very old populations, uneducated people are less likely to die than more educated people (Hurd, McFadden, and Merrill, 2001). Similar patterns can be observed for other risk factors: among the elderly, having smoked in the past is positively correlated with survival, even though it is negatively correlated with survival for the young.¹⁵ This is because the average uneducated decedent is likely to be healthier than the average educated one. Differential mortality lowers the average health of the less educated by relatively more and would thus contract health disparities. In any event, the fact that the educational composition of the HCSUS population barely changes over time is evidence that differential mortality is not quantitatively significant. The second issue—differential diagnosis—is more difficult to sign. Suppose that more educated people are diagnosed with HIV earlier. Since the HCSUS is a sample of people who are being treated for (and thus know they have) HIV, the educated people may be in an earlier stage of the disease. Therefore, the time path of the disease’s natural progression may differ across education groups. To assess the importance of this, we include various correlates of the individual’s health at baseline: age, age-squared, race, sex, whether the individual used IV drugs or had gay sex, region of residence, self-assessed health at baseline, and years since diagnosing. Including or excluding these (or various permutations or combinations of these) variables affects the levels of β_{it} but it has virtually no effect on its time trend. The final hypothesis we

¹⁵ Based on authors’ analysis of the Medicare Current Beneficiary Survey (MCBS) data.

investigate is whether or not trends in β_{1t} are driven by differential access to insurance.

For example, since HAART is an expensive therapy, its adoption could be governed entirely by insurance status. We include HCSUS measures of insurance to see if these change trends in β_{1t} , but they also seem to have little effect.

In sum, we estimate the model:

$$CD4_{it} = \beta_0 + \beta_{1t}Sch_i + \beta_{2t}Health_i + \beta_{3t}Insurance_{it} + \varepsilon_{it}, \quad (18)$$

where $Health_i$ is a set of correlates for baseline health, and $Insurance_{it}$ is a set of insurance variables. The estimates for this model are presented in Table 9. At the bottom of the table, we can see the effect of HAART in the (albeit insignificant) growth of the constant term. While HAART drugs reduce mortality, they also have the direct effect of boosting immune function. Clinically, they have been found to boost CD4 readings, but the improvement does not seem to have been evenly distributed. In particular, there has been an expansion in inequality between high school dropouts and college attendees, as seen in the top line of the table. At baseline, there was no statistically significant difference in immune function between high school dropouts and college attendees. In the follow-up periods, however, a substantial gradient emerges, equal to about one-third the size of the total growth in the constant term over the three waves of the survey. At baseline, there was no significant gap between high school graduates and college attendees, and no gap emerges after the advent of HAART, whose effects seem to be felt the least by high school dropouts.

Our results are robust to the inclusion of insurance status, region, various demographic characteristics, along with log income and income category (not shown in the table). As

mentioned earlier, the inclusion of these variables has little effect on the trend in β_{it} , although the levels of the coefficients are affected. For example, estimating the simplest model of CD4 on the schooling dummies yields the following series of coefficients on the high school dropout variable: -23.5, -68.7, and -52.7. The standard errors are slightly smaller in this specification, although the baseline coefficient continues to be insignificant. The expansion in disparities between high school dropouts and other education groups seems to be statistically significant. We estimated a pooled regression, with all three waves of the survey, where all coefficients were fully interacted with dummies for the wave of the survey. Standard errors were clustered by respondent. The magnitudes of the coefficients were quite similar to the magnitudes in Table 9. The difference in the high school dropout coefficient had an associated t-value of 1.97 between baseline and follow-up 1, and 1.86 between baseline and follow-up 2. There is a statistically significant difference at the 95% level in the first case and at the 90% level in the second case.

D. Conclusions

We have presented some simple ideas that can help us make sense of how and why health disparities vary. The theory predicts that most technological innovations in health care, by lowering the price of health, will expand health disparities. However, certain innovations can contract health disparities, if they simplify the production of health and reduce the importance of time investments. In accordance with these predictions, the development of new HIV drugs that involved complicated medication regimens widened health disparities, while the development of hypertension drugs that lessened the need to undertake diet control, weight control, and exercise had the opposite effect.

This paper suggests the importance of an empirical research agenda designed to understand how health disparities vary across the population and over time. We have identified a few important dimensions along which the returns vary, but there are likely to be many other dimensions that require further empirical and theoretical investigation. In addition, this paper has relied on advances in technology as a source of change over time in health disparities. While we have taken these types of innovations as exogenous, it is possible that health disparities themselves have a role to play in the development of technological change. Timesaving technologies may be more likely to arise when large numbers of uneducated people suffer from a disease; conversely, timesaving technologies are less likely when a disease is confined to the educated or the rich. Alternatively, economy-wide growth in schooling may encourage certain types of technological change that involve more own-time investments. Growth in schooling raises the payoff to developing such technologies. Future work could examine the theoretical linkage between the growth in the educated population and the incentives for patient-intensive technology.

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Figure 1: Growth in differential mortality across education groups among white US males, 1960-98.

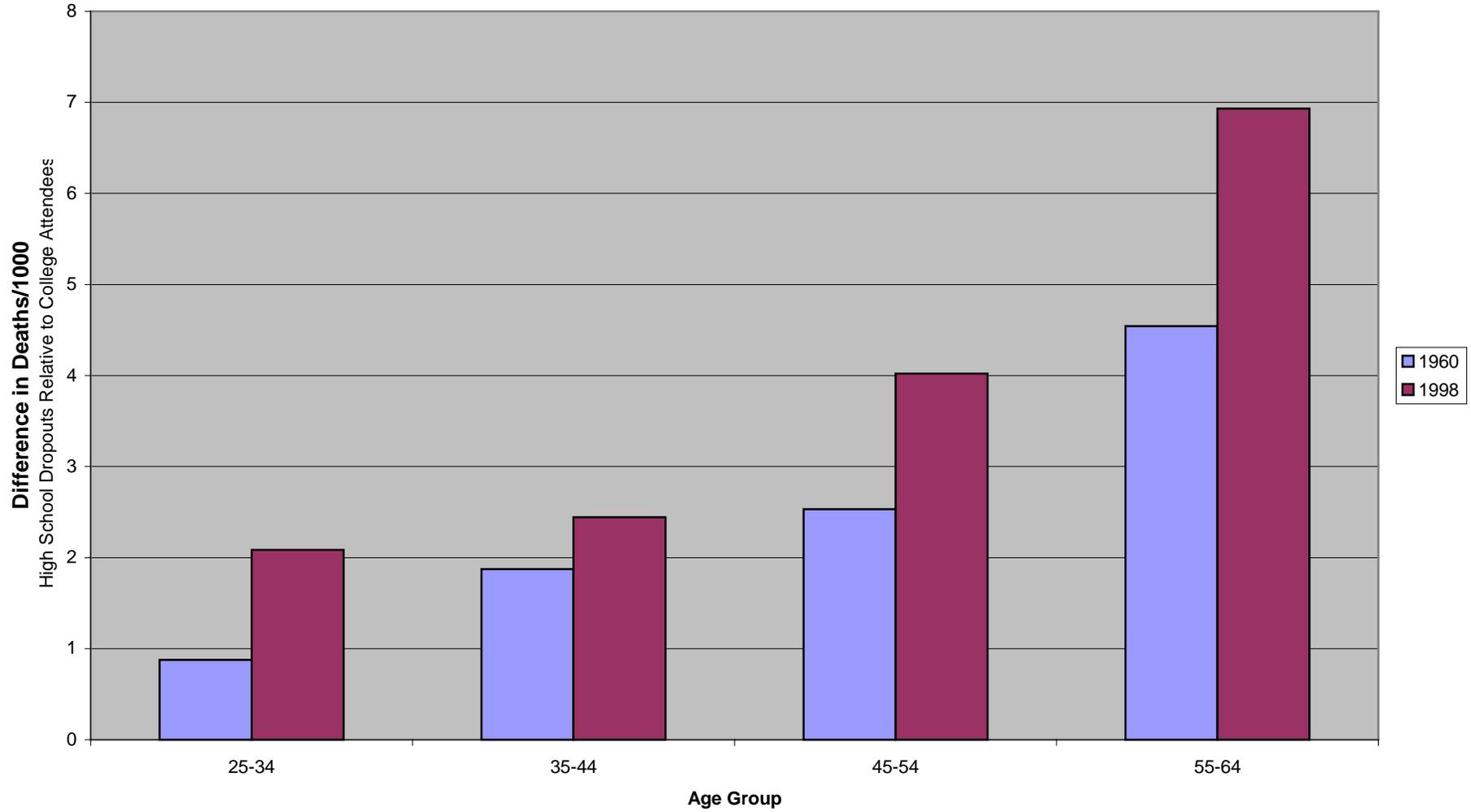


Table 1: Health Disparities by Age and Chronic Illness Status.^a

Age Group	Not Chronically Ill					Chronically Ill				
	High School	Some College	College	# Obs	R ²	High School	Some College	College	# Obs	R ²
25-34	0.074 (35.69)	0.093 (44.87)	0.108 (54.23)	244,664	0.03	0.188 (13.81)	0.233 (16.32)	0.319 (23.55)	11,384	0.07
35-44	0.101 (38.94)	0.123 (47.01)	0.144 (57.87)	212,428	0.03	0.239 (22.82)	0.308 (27.13)	0.398 (37.01)	18,540	0.09
45-54	0.123 (40.69)	0.150 (46.8)	0.172 (57.52)	139,868	0.04	0.270 (34.79)	0.342 (35.88)	0.436 (49.01)	26,345	0.11
55-59	0.131 (28.5)	0.162 (31.37)	0.180 (37.85)	53,389	0.04	0.255 (28.1)	0.303 (24.51)	0.406 (35.05)	17,696	0.09
60-64	0.134 (27.9)	0.167 (30.12)	0.181 (34.41)	46,594	0.04	0.238 (29.04)	0.304 (26.29)	0.368 (33.22)	21,540	0.08
65-69	0.109 (21.57)	0.142 (23.46)	0.158 (27.82)	39,536	0.03	0.199 (25.26)	0.258 (23.14)	0.315 (28.64)	23,209	0.06
70-74	0.110 (19.4)	0.148 (20.99)	0.147 (20.71)	31,991	0.03	0.163 (18.32)	0.216 (17.03)	0.251 (20.07)	19,278	0.04
75+	0.074 (13.9)	0.105 (14.97)	0.088 (13.35)	42,412	0.02	0.107 (14.22)	0.165 (15.84)	0.127 (12.57)	31,179	0.02

Note: Table shows the difference in the probability of reporting good health between the listed education group and those with less than a high school education, using a linear probability model. Regressions are run separately for each age group and illness status, and also control for gender and year. The chronically ill are those who report having either: hypertension, diabetes, asthma, arthritis, or heart disease. Data are from the 1982-1996 NHIS.

^aAbsolute values of T-statistics appear below coefficients.

Table 2: Health Disparities by Age and Work-Limitation Status.^a

Age Group	Unable to work due to health condition					Able to work				
	High School	Some College	College	# Obs	R ²	High School	Some College	College	# Obs	R ²
25-34	0.162 (14.65)	0.210 (16.49)	0.302 (21.87)	44,869	0.07	0.055 (28.12)	0.071 (36.08)	0.081 (42.73)	195,427	0.01
35-44	0.159 (16.46)	0.225 (20.28)	0.306 (25.95)	36,632	0.12	0.077 (31.62)	0.095 (38.76)	0.108 (45.85)	182,371	0.02
45-54	0.193 (23.03)	0.266 (24.24)	0.326 (27.25)	33,195	0.13	0.103 (35.21)	0.127 (41.85)	0.141 (49.17)	125,005	0.03
55-59	0.176 (17.13)	0.218 (14.33)	0.259 (15.66)	23,509	0.12	0.115 (26.19)	0.143 (30.17)	0.156 (35.44)	44,472	0.03
60-64	0.168 (18.11)	0.219 (15.59)	0.271 (18.48)	37,179	0.08	0.109 (24.41)	0.140 (28.14)	0.145 (30.64)	28,733	0.03
65-69	0.151 (16.67)	0.211 (15.19)	0.241 (16.64)	48,720	0.06	0.095 (20.44)	0.122 (22.35)	0.133 (26.12)	12,827	0.02

Note: Table shows the difference in the probability of reporting good health between the listed education group and those with less than a high school education, using a linear probability model. Regressions are run separately for each age group and work-limitation category, and all contain dummies for gender and year, and a linear term for age. Data are from the 1982-1996 NHIS.

^aAbsolute values of T-statistics appear below coefficients.

Table 3: Instrumental Variables Estimates of Health Disparities by Illness Status.

	OLS		Instrumental Variables		Education Instruments ^a	
	Chronic	Healthy	Chronic	Healthy	Chronic	Healthy
Female	0.005 *** 1.86	-0.001 -1.16	0.017 ** 2.10	0.001 0.38	-0.305 * -17.61	-0.182 * -27.07
Highest Grade Attained	0.033 * 94.42	0.015 * 113.78	0.072 * 2.96	0.022 * 3.06		
Age, 25 to 34	-0.008 * -7.97	-0.002 * -15.94	-0.008 * -7.86	-0.002 * -9.19	0.014 *** 1.79	0.027 * 17.18
Age, 35 to 44	-0.007 * -9.67	-0.002 * -15.10	-0.005 * -2.85	-0.002 * -13.42	-0.061 * -11.14	-0.008 * -5.25
Age, 45 to 54	-0.005 * -7.47	-0.002 * -12.90	-0.002 -0.84	-0.002 * -3.23	-0.087 * -17.46	-0.076 * -40.95
Age, 55 to 59	-0.007 * -4.88	-0.004 * -6.75	-0.005 * -2.61	-0.003 * -5.06	-0.049 * -5.08	-0.050 * -10.47
Age, 60 to 64	0.005 * 3.46	-0.002 * -2.56	0.007 * 3.59	-0.001 *** -1.71	-0.052 * -5.31	-0.055 * -9.64
Age, 65 to 69	0.003 ** 2.10	-0.004 * -4.74	0.005 ** 2.55	-0.003 * -3.27	-0.041 * -4.27	-0.080 * -12.79
Age, 70 to 74	-0.001 -0.60	-0.005 * -6.08	0.001 0.65	-0.005 * -4.12	-0.054 * -6.01	-0.094 * -15.15
Age, 75+	0.001 1.25	-0.003 * -7.58	0.003 ** 1.96	-0.003 * -4.49	-0.063 * -16.25	-0.063 * -21.94
Birth Quarter 2					0.013 0.55	0.029 * 3.02
Birth Quarter 3					0.106 * 4.47	0.120 * 12.91
Birth Quarter 4					0.118 * 4.85	0.063 * 6.52
Constant	0.55 * 16.99	0.81 * 187.43	0.07 0.22	0.72 * 8.02	12.26 * 48.28	12.19 * 237.90
R-Squared	0.082	0.058	0.006	0.052	0.073	0.060
Observations	169171	810882	169061	810421	169061	810421

*Significant at 99% level.

**Significant at 95% level.

***Significant at 90% level.

Note: All Regressions Include Year Dummies.

^aJoint F-statistics for the Quarter of Birth Instruments were 12.23 (3, 169941) for the Chronically ill, and 64.00 (3, 814125) for the Healthy.

Table 4: Characteristics of MCBS Respondents Over Age 80, by Mortality Status.

	Age Group					
	80-85		85-90		90+	
	Survivors	Last Year of Life	Survivors	Last Year of Life	Survivors	Last Year of Life
Good Health	0.61 (0.49)	0.33 (0.47)	0.59 (0.49)	0.37 (0.48)	0.56 (0.5)	0.35 (0.48)
Male	0.35 (0.48)	0.45 (0.5)	0.30 (0.46)	0.37 (0.48)	0.23 (0.42)	0.27 (0.45)
Less than HS	0.48 (0.5)	0.50 (0.5)	0.53 (0.5)	0.58 (0.49)	0.58 (0.49)	0.60 (0.49)
High School	0.28 (0.45)	0.30 (0.46)	0.23 (0.42)	0.22 (0.42)	0.18 (0.39)	0.19 (0.4)
Some College	0.25 (0.43)	0.20 (0.4)	0.24 (0.43)	0.20 (0.4)	0.23 (0.42)	0.21 (0.4)
Observations	12,498	1,064	7,051	1,002	3,557	967

Note: Numbers in parentheses are standard deviations.

Table 5: Determinants of Good Health among MCBS Respondents, by Mortality Status.

	Survivors	Last Year of Life
High School	0.08 * (0.01)	0.04 (0.03)
Some College	0.15 * (0.01)	0.09 * (0.03)
Age 80-85	0.03 ** (0.01)	-0.03 (0.03)
Age 85-90	0.02 (0.02)	0.02 (0.03)
Male	0.03 * (0.01)	0.05 * (0.02)
R-Squared	0.018	0.009
Observations	15888	2085

Note: Robust Standard Errors, clustered by respondent, in parentheses.

*Significant at 95% level.

**Significant at 90% level.

Table 6: Characteristics of pre- and post-Beta Blocker Cohorts in the Framingham Heart Study.

	Pre-Beta Blockers 1966-72		Post-Beta Blockers 1980-86	
	Mean	Std Dev.	Mean	Std Dev.
Age	66.2	3.8	67.5	3.5
Male	43.2%	0.50	41.7%	0.49
Never Attended High School	31.5%	0.46	16.5%	0.37
Attended High School	15.2%	0.36	12.5%	0.33
Graduated from High School	24.1%	0.43	41.0%	0.49
Attended College	29.2%	0.45	29.9%	0.46
Hypertension	62.8%	0.48	53.2%	0.50
HCVD	34.5%	0.48	25.9%	0.44
Respiratory Disorders	13.3%	0.34	9.1%	0.29
High Blood Sugar	10.2%	0.30	6.1%	0.24
Obesity	17.6%	0.38	23.6%	0.42
Person-Years	4496		4121	

Note: The pre-Beta Blockers group includes all respondents aged 59 to 69 in 1966. The post-Beta Blockers group includes all respondents aged 59 to 69 in 1980. High Blood Sugar is defined as a blood glucose reading above 1.5 mg/mL.

Table 7: Effects of Beta-Blockers on Health Disparities in Heart Disease.

	Hypertension	Hypertensive CVD	Any Respir. Disorders	High Blood Sugar (>1.5mg/mL)	Obesity
Some HS	-0.02 [0.03]	-0.10 [0.03]***	0.04 [0.03]	0.02 [0.02]	-0.07 [0.03]**
HS Grad	-0.05 [0.03]*	-0.11 [0.03]***	-0.03 [0.02]	0.03 [0.02]**	-0.10 [0.03]***
Some College	-0.07 [0.03]***	-0.11 [0.03]***	-0.04 [0.02]**	0.02 [0.01]	-0.12 [0.03]***
Post-Beta Blockers	-0.14 [0.03]***	-0.14 [0.03]***	-0.02 [0.02]	-0.02 [0.02]	0.07 [0.04]*
Some HS*	0.04	0.11	-0.07	-0.03	-0.01
Post-Beta Blockers	[0.05]	[0.05]**	[0.03]*	[0.03]	[0.05]
HS Grad*	0.06	0.08	-0.01	-0.03	0.04
Post-Beta Blockers	[0.04]	[0.04]**	[0.03]	[0.02]	[0.05]
Some College*	0.03	0.03	-0.02	-0.05	-0.01
Post-Beta Blockers	[0.04]	[0.04]	[0.03]	[0.02]**	[0.04]
Age	0.01 [0.04]	0.13 [0.04]***	-0.03 [0.03]	0.06 [0.02]**	0.00 [0.04]
Age-Squared	0.00 [0.00]	-0.00 [0.00]***	0.00 [0.00]	-0.00 [0.00]**	-0.00 [0.00]
Male	-0.03 [0.02]**	-0.03 [0.01]**	0.09 [0.01]***	0.03 [0.01]***	-0.03 [0.02]**
Constant	0.14 [1.46]	-4.18 [1.40]***	0.82 [0.95]	-2.18 [0.79]***	0.30 [1.25]
Observations	8212	8078	8105	7401	7753
R-squared	0.02	0.02	0.03	0.02	0.02
Unique Individuals	2454	2454	2454	2424	2441

Robust standard errors clustered by individual appear in brackets.

*Significant at 10%; **Significant at 5%; ***Significant at 1%.

Note: The pre-Beta Blockers cohort is aged 59 to 69 in 1966, and followed from 1966-72.

The post-Beta Blockers cohort is aged 59 to 69 in 1980, and followed from 1980-86.

Table 8: Characteristics of the HCSUS Population.

	Baseline	Follow-Up 1	Follow-Up 2
Less than High School	0.25 (0.43)	0.25 (0.43)	0.25 (0.43)
High School Degree	0.27 (0.45)	0.27 (0.45)	0.28 (0.45)
Some College	0.28 (0.45)	0.28 (0.45)	0.28 (0.45)
College Graduate	0.19 (0.39)	0.20 (0.40)	0.20 (0.40)
Black	0.33 (0.47)	0.33 (0.47)	0.32 (0.47)
Female	0.23 (0.42)	0.23 (0.42)	0.23 (0.42)
Medicaid	0.44 (0.50)	0.46 (0.50)	0.47 (0.50)
Medicare	0.19 (0.39)	0.22 (0.41)	0.25 (0.43)
Private Insurance	0.35 (0.48)	0.35 (0.48)	0.34 (0.47)
No Insurance	0.20 (0.4)	0.18 (0.38)	0.16 (0.36)
Asymptomatic	0.10 (0.31)	0.06 (0.24)	0.05 (0.21)
Symptomatic	0.51 (0.5)	0.52 (0.50)	0.51 (0.50)
AIDS	0.38 (0.49)	0.41 (0.49)	0.44 (0.50)
Immune Function (CD4)	315 (254)	351 (280)	373 (260)
On HAART	0.24 ^a (0.43)	0.40 ^b (0.49)	0.61 ^c (0.49)
Observations	2864	2466	2267

Note: Standard deviations appear in parentheses below means.

^aBased on 2828 observations.

Table 9: Effect of HAART on Health Disparities among HIV Patients

Variable	CD4+ lymphocyte count (cells per mm ³)		
	Baseline	FU1	FU2
Years of schooling (excluded=13 years or more):			
Less than 12 years	-19.06 (19.11)	-66.81 (27.35)**	-54.60 (17.89)***
12 years	10.58 (19.38)	-20.03 (29.15)	22.06 (22.31)
Age	-8.53 (3.84)**	-13.00 (11.86)	-9.43 (5.21)*
Age squared/1000	91.43 (49.60)*	137.92 (131.00)	89.70 (66.81)
Black	12.42 (12.88)	23.31 (26.32)	14.18 (13.22)
Female	47.84 (13.78)***	47.46 (18.77)**	26.63 (16.91)
Used intravenous drugs	-1.62 (12.19)	4.51 (18.43)	-1.21 (17.25)
Had sex with men (0 if female)	-17.37 (11.20)	-4.40 (25.44)	-16.25 (15.81)
Region (excluded=Midwest):			
Northeast	63.88 (25.85)**	52.81 (41.66)	39.65 (21.56)*
West	102.62 (27.96)***	77.79 (42.53)*	79.13 (20.27)***
South	46.76 (28.23)	52.76 (44.90)	66.17 (22.56)***
Self-reported baseline health (excluded=Poor):			
Excellent/Very Good	105.84 (17.98)***	108.77 (19.67)***	89.40 (22.14)***
Good	94.40 (20.51)***	111.96 (22.97)***	86.92 (25.25)***
Fair	28.86 (13.83)**	26.07 (26.28)	24.85 (19.22)
Years since diagnosed	-2.47 (2.07)	0.87 (2.46)	-3.73 (2.51)
Insurance (excluded=None):			
Medicaid	-62.60 (17.28)***	-64.60 (35.03)*	-68.37 (30.79)**
Medicare	-76.24 (27.29)***	-119.51 (30.15)***	-131.72 (26.56)***
Private Insurance	-35.13 (23.99)	-51.32 (20.10)**	-37.55 (23.57)
Medicaid and Medicare	-96.82 (19.18)***	-77.33 (28.67)***	-109.15 (42.56)**
Constant	458.88 (73.89)***	603.90 (214.56)***	605.46 (105.48)***
Observations	2192	1855	1628
R-squared	0.08	0.06	0.09

Robust standard errors in parentheses

* significant at 10%; ** significant at 5%; *** significant at 1%

