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## A Theory of Health Disparities and Medical Technology

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# A Theory of Health Disparities and Medical Technology\*

Dana P. Goldman and Darius N. Lakdawalla

## Abstract

Better-educated people are healthier, although the sources of this relationship remain unclear. Starting with basic principles of consumer theory, we develop a model of how health disparities are determined that does not depend on the precise causal mechanism. Improvements in the productivity of health care disproportionately benefit the heaviest health care users. Since richer 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 health care productivity—could widen socioeconomic disparities in health. An exception to this rule, however, is a simplifying technology, which can contract health disparities, since richer patients are more likely to invest effort in adhering to complex treatment regimens. We present a few empirical case studies to help illustrate the theoretical results. First, we show that a complicated treatment regimen (antiretroviral therapy for HIV) benefited well-educated patients disproportionately. In contrast, simplifying drugs for hypertension coincided with a contraction in cardiovascular disparities not seen in other diseases. Finally, nationally representative data suggest that there are wider disparities by education among the chronically ill populations—precisely the population one would expect to be the heaviest health care users.

**KEYWORDS:** socioeconomic status, health disparities, technology, medical care

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## A. Introduction

That richer and better-educated people are healthier is one of the most robust empirical findings in health. Socioeconomic status in general has been shown to affect mortality and morbidity in a number of studies (Smith, 1999; Marmot, 2000) and—particularly for education—the relationship is quantitatively large (Kitagawa and Hauser, 1973; Feldman et al., 1989; Pappas et al., 1993; Preston and Taubman, 1994; Schoenbaum and Waidmann, 1997; Case, Lubotsky, and Paxson, 2002; Currie and Stabile, 2003).

Figure 1 provides an example of these empirical regularities using data from Kitagawa and Hauser (1973) and Murphy (1998). It 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. There are two points to be made. First and foremost, there is a large and persistent mortality gap by education between college attendees and high school dropouts. This is the prototypical finding in this literature; namely, there is a strong association between a college degree and good health. Second, the magnitude of this gap varies. According to these data, for instance, 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. Similar expansions in the mortality gradient appear to have taken place in Britain, from 1970 to 1993 (Acheson et al., 1998). While there is still some uncertainty about whether expansions in disparities are robust, these trends pose a challenge to economists interested in understanding the forces that govern health disparities.<sup>1</sup>

Much of the economic literature on this subject has focused on education and individuals' health investment decisions. 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

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<sup>1</sup>It does seem clear though that these trends do not simply reflect changes in the composition of schooling. 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.

linking schooling and health.<sup>2</sup> Fuchs (1982) has argued against these hypotheses by suggesting that it is primarily unobserved factors such as time preferences that explain both health and education. Other researchers have advanced alternative explanations that emphasize restricted access to care or poorer quality of care for the less educated (Bindman et al., 1995; Andrulis, 1998). The empirical work using a variety of instrumental variables methods has not pointed definitively toward a single explanation out of these many alternatives.<sup>3</sup>

Our work is motivated by the need to understand how and why health disparities change in the absence of a single, widely accepted view about the causal relationship between education and health. We present a simple theoretical model that is able to generate some important findings about health disparities, without strong assumptions about the effect of education on allocative efficiency or preferences. In fact, our model is consistent with a variety of different assumptions and models of this type. We then consider a sequence of case studies designed to highlight its testable predictions. The bulk of our findings follow from an application of Roy's Identity: reductions in the price of a good benefit the good's heaviest consumers the most. Accordingly, reductions in the price of health care or (equivalently) improvements in its productivity benefit the heaviest health care users, who will also tend to be the higher wage, higher socioeconomic status individuals. To our knowledge, nearly every model of health, education, and health disparities embeds this assumption; this implies that our results apply quite generically to a wide class of more detailed and specialized models.

Several other hypotheses emerge with regard to medical technology and disease. First, new technologies can expand health disparities by raising the productivity of health care and thus lowering the quality-adjusted price of health. Cutler et al (1998) and Cutler and Huckman (2003) provide empirical evidence that new

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<sup>2</sup> 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.

<sup>3</sup> 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 (2002) 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. For example, Case and Deaton (2003) emphasize the importance of occupation.

technologies raise productivity and lower quality-adjusted prices in the context of heart disease treatment. However, even though medical productivity and disparities are positively related, certain kinds of new technologies can contract disparities: while new technologies will always raise the productivity of *some* health inputs, they can contract disparities if they lower the productivity of others by enough. In particular, innovations that simplify the production of health can lower the productivity of patient effort. For instance, the typhoid vaccine<sup>4</sup> 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 were more likely to make these kinds of effort investments, the advent of the vaccine would have benefited them disproportionately less by making redundant these investments. Therefore, the vaccine might have contracted disparities. Finally, the theory suggests that the effect of a chronic disease on disparities depends on its relationship to the productivity of health investment. For instance, an untreatable illness can be a great equalizer if it reduces the productivity of all health inputs. In contrast, people with chronic, treatable conditions may exhibit wider disparities, because medical care is more productive for these people than for healthy people with fewer medical care needs.

We begin by presenting our theory of health disparities. We then consider 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.

## B. A Theory of Health Disparities

While the precise 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. Of course, we make this assumption *conditional* on initial health status. Unconditionally, poorer people tend to spend more on medical care than richer people because they are sicker, but controlling for health status reverses this result (see Bhattacharya and Lakdawalla, 2005, for some evidence pertaining to the US elderly).<sup>5</sup>

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<sup>4</sup> See, e.g., Griffin (1999) for a general discussion of childhood vaccination against typhoid.

<sup>5</sup> For example, the elderly respondents in the Medicare Current Beneficiary Survey exhibit a negative relationship between schooling and medical expenditures overall. Including the Survey's detailed controls for disability and health status, however, produces a positive and statistically

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. Under the assumption (discussed below) that poorer and sicker people are more vulnerable to health shocks, 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.<sup>6</sup> 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 more forward-looking, more able, or because of some other factor. This result will obtain for a variety of models that make very different assumptions about the specific effects of education on health production.<sup>7</sup>

We make the standard assumption that health provides an individual with more time for labor and leisure, but at diminishing returns (Grossman, 1972). Define  $H$  as health and  $h$  as health investment.  $H$  increases in a concave fashion with health investment.

Suppose that health investments can take the form of medical care purchased in the market ( $m$ ), and investments by the patient of her own time and effort ( $t$ ). These two inputs are assumed to be complementary, consistent with the empirical finding that better patient adherence improves the effectiveness of treatment (Goldman and Smith, 2002). Under complementarity, richer and more educated patients purchase more medical care and invest more of their own time and effort into producing health. This result is also consistent with the empirical evidence that more educated patients adhere more rigorously to treatment regimens (Goldman and Smith, 2002).

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significant relationship between schooling and medical expenditures. Moreover, this pattern holds true for a population with relatively greater access to public insurance through Medicare.

<sup>6</sup> 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.

<sup>7</sup> A recent and relevant example is the paper by Glied and Lleras-Muney (2003). They argue that educated people are better able to adopt new medical technologies, and that this expands health disparities. This is a specific type of allocative efficiency argument that is nested within our model. Their argument implies that new technologies always raise the productivity and importance of patient effort. In our model too, this expands disparities.

To simplify the exposition, suppose that health production is constant returns to scale and Cobb-Douglas, in the sense that  $h = t^\phi m^{1-\phi}$ , and that  $H(h) = h^\gamma$ . In this case, we can write  $H = m^\alpha t^\beta$ , where  $\alpha + \beta < 1$ . The individual maximizes income:

$$\max_{m,t} wm^\alpha t^\beta - \pi m - wt \quad (1)$$

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

$$\begin{aligned} t &= \left( \frac{w\alpha}{\pi} \right)^{\frac{\alpha}{1-\alpha-\beta}} \beta^{\frac{1-\alpha}{1-\alpha-\beta}}, \\ m &= \left( \frac{w\alpha}{\pi} \right)^{\frac{1-\beta}{1-\alpha-\beta}} \beta^{\frac{\beta}{1-\alpha-\beta}}, \end{aligned} \quad (2)$$

and equilibrium health investment,

$$H = \left( \frac{w\alpha}{\pi} \right)^{\frac{\alpha}{1-\alpha-\beta}} \beta^{\frac{\beta}{1-\alpha-\beta}}. \quad (3)$$

Taking the permanent wage as an index of socioeconomic status, disparities in health are characterized by:

$$\frac{\partial H}{\partial w} = \frac{\alpha}{1-\alpha-\beta} \left( \frac{1}{w} \right) \left( \frac{w\alpha}{1-\alpha-\beta} \right)^{\frac{\alpha}{1-\alpha-\beta}} \beta^{\frac{\beta}{1-\alpha-\beta}} \quad (4)$$

Analyzing and interpreting the expression in equation 4 provides a number of insights into the effects of new technologies on disparities. We will study: (1) The effects of breakthroughs in medical care; (2) Changes in the productivity of patient self-management; and (3) The effects of chronic illness and, more generally, cross-sectional variation in the value of medical care.

Notice that we consider *absolute* disparities in health  $\frac{\partial H}{\partial w}$ , rather than relative or percentage differences in health, such as  $\frac{\partial \ln H}{\partial w}$  or  $\frac{\partial \ln H}{\partial \ln w}$ . There are two reasons for this. First, a simple economic model (such as the one presented here) produces few general results about relative health disparities. There is thus less to be gained by analyzing relative disparities in this framework. Second, while they are not the only way to measure disparities, absolute disparities are important, because they tell us about the size of the difference in lifetime well-being across socioeconomic groups. As noted by Acheson et al (1998) in their report on health

disparities to the British government, absolute disparities may even be more important for welfare, because they internalize the prevalence of illness and health across different socioeconomic groups.

We are considering one of the more basic and general sources of health disparities, namely that richer people value time-extension more than poorer people. This framework and its results could be overlaid on stronger models where, for example, education affects the efficiency of health investment directly, or where preferences for health and education are correlated. The effects we study are present in any model where health produces time, and the value of time is higher for richer people.

### B.1 Innovations in Medical Care

The simplest way to think about a new medical treatment is as a reduction in  $\pi$ , the quality-adjusted price of health investment. When medical technology improves, an individual is able to produce more health investment for the same amount of money. According to equation 4, reducing the price of medical care expands the gradient in health. 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.

One might also think of a breakthrough technology as raising the productivity of medical care,  $\alpha$ , but this has the identical effect of expanding health disparities.

This can be seen by rewriting equation 4 as  $\frac{\partial H}{\partial w} = \frac{\alpha}{1 - \alpha - \beta} \frac{H}{w}$ . Since increases in medical care productivity always raise  $H$ ,<sup>8</sup> it follows that increases in  $\alpha$  raise  $\frac{\partial H}{\partial w}$ .

In either event, better medical care disproportionately benefits the heavier users of medical care—the rich. This basic result also holds for models in which health has value as a consumption good.<sup>9</sup> 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

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<sup>8</sup> This will be true provided we scale  $H$  such that  $H \geq 1$ .

<sup>9</sup> This is the same as a model in which the return to health is concave or the cost of effort spent on health is convex.

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).

## **B.2 Medical Breakthroughs and Patient Self-Management**

Medical breakthroughs have clear and unambiguous effects on the price and productivity of medical care purchased in the marketplace, but their effects on patient self-management are more complicated. While new medical technologies intrinsically raise the productivity of medical care, they may not raise the productivity of patient inputs such as effort, time, or adherence to a treatment regimen.

Note first from equation 4 the analogous result that increases in  $\beta$ , the productivity of patient effort, expand disparities, and vice-versa.<sup>10</sup> Intuitively, productivity growth benefits the heavier investors. However, the effect of new technologies on the productivity of patient effort may vary from one technology to another.

Diabetes provides a constructive example of a breakthrough that may have lowered the productivity of patient effort. Patients with type 1 diabetes must take insulin (either through injection or an insulin 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. A new technology that makes it less important to monitor blood sugar will make patient effort less important and productive. 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 and alleviates the need for intensive self-monitoring. Since this type of breakthrough makes patient effort less important, it could contract disparities by disproportionately hurting the heavy investors of effort. It is important to emphasize that a breakthrough like this can actually lower the absolute productivity of patient effort, since a given amount of time spent monitoring or managing health actually has a smaller pay-off. As a result, it can contract disparities, if the impact on patient self-management offsets the impact of lower prices and higher productivity of medical care.

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<sup>10</sup> As before, as long as  $H > 1$ , increases in  $\beta$  raise the equilibrium value of  $H$  and the equilibrium value of health disparities in equation 4.

Recent breakthroughs in the treatment of HIV present a rather different example, which we study in our empirical analysis. While the new treatments were extremely effective, they also involved highly complex treatment regimens, involving many pills that had to be taken in a particular sequence and on a highly regimented schedule. Before the breakthroughs, relatively little could be done to improve the condition of an HIV-sufferer, through either medical care or a patient's own efforts. After the breakthroughs, however, much could be done, but the efficacy of the treatment depended on the degree to which a patient could comply accurately with the demanding treatment schedule. As a result, the returns to a patient's own efforts went up with the treatment; this type of breakthrough can be expected to expand disparities, in contrast to the earlier example of diabetes. In this case, the impact on the productivity of patient effort reinforces the effects of cheaper and more productive medical care: all serve to expand disparities.

### **B.3 Chronic Illness and Disparities within Subgroups**

All the analysis above implies that growth in the productivity of health inputs tends to promote health disparities. This has implications for how disparities vary within the population. It is likely that health inputs are more productive for sicker populations, such as the chronically ill, or the elderly. Lichtenberg and Virabhak (2002) find that new drugs confer the greatest benefits upon the chronically ill. If this relationship extends to other kinds of health investments, it suggests that chronic illness would expand disparities, because it makes health care more productive. This is an important implication normatively, since it implies that improvements in early childhood health can limit disparities in adulthood, by limiting the onset of chronic illness later in life.

Clearly, however, much depends on the availability of treatments. A person chronically ill with an untreatable disease will not enjoy more productive health investments. The onset of untreatable chronic illness, therefore, can limit health disparities, as all patients are equally unable to effect improvements in their health, even though some may have greater incentives to do so. In contrast, illnesses with effective treatments can exacerbate disparities if they make health care—such as doctor visits, drugs, or surgical procedures—more productive.

## **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. Using schooling as a measure of the permanent wage  $w$ , we explore

two implications of the model. The first is  $\frac{\partial}{\partial \pi} \frac{\partial h}{\partial w} |_{\beta} > 0$   $\frac{\partial}{\partial \pi} \frac{\partial h}{\partial w} |_{\alpha} > 0$ : technologies that lower the price of health but not the productivity of time expand disparities. As an example of this type of breakthrough, we study the advent of beta-blockers as a treatment for hypertension. The second contends that, holding initial health constant, technologies that lower the productivity of time can sometimes contract disparities. Our case study of this centers on recent breakthroughs in the treatment of HIV.

### **C.1 Technological Breakthroughs and Disparities**

The theory indicates that the effects of a new medical innovation on health disparities will depend in part on whether it is simplifying. New treatments that make patient effort more important (or leave its importance unchanged) will tend to raise health disparities. However, by simplifying health care, some new technologies can contract health disparities. Antihypertensive drugs provide a useful example, because they 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. Investments of patient time were supplanted by the new drugs. On the other hand, new HIV treatments have greatly improved immune function among HIV patients, but have not simplified its treatment. Since these treatments were more difficult to manage than their predecessors, they require additional investments of patient time, effort, and skill. We examine both below, starting with HIV.

### **C.2 Breakthroughs in HIV Treatment: Highly Active Antiretroviral Therapy**

During the mid-1990s, highly active antiretroviral treatments (HAART) to treat HIV became available. These treatments substantially improved the health status of HIV patients, but they involved complex medication regimens requiring strict patient adherence (Goldman and Smith, 2002). HAART involves taking at least three different medications and as many as 24 pills per day, all of which have to be timed with each other and are affected by diet. Non-adherence, which is common, not only impedes treatment, but it can also make the disease worse by breeding viral resistance. 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.

Our data come from the HIV Cost and Services Utilization Study (HCSUS). The 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, who made at least one visit for regular care in the contiguous United States in early 1996.<sup>11</sup> 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 was 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.

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. The HCSUS classified patients into three clinically relevant disease stages: asymptomatic, symptomatic, or AIDS. It also collected self-reported CD4 T-lymphocyte cell counts, 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 1 displays the characteristics of the HCSUS population. The average educational make-up of the HCSUS population does not change over time, even though there is attrition due to mortality. The population does become sicker, as more people move out of the asymptomatic and symptomatic categories into the AIDS category. People also tend to move out of the uninsured group into the publicly insured groups. However, immune function goes up as measured by the CD4 count, perhaps as a result of more effective treatments or because the sicker patients die. At baseline, only about 25 percent of respondents with AIDS had ever used HAART. Just nine months later,<sup>12</sup> 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.

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<sup>11</sup> The HCSUS sample does not include patients whose only contact with the health care system was through military, prison, or emergency department facilities.

<sup>12</sup> The difference between the median interview dates is about nine months between waves one and three.

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

$$CD4_{it} = \beta_0 + \beta_{1t}Sch_i + \beta_{2t}Black_i + \beta_3 I_i + \varepsilon_{it}, \quad (5)$$

where  $Sch_i$  is a set of dummy variables indicating the educational group to which individual  $i$  belongs, and  $I_i$  is a vector of variables measuring the initial health of individual  $i$  at baseline. We are interested in changes over time in the coefficient  $\beta_1$  when initial health is held fixed to the extent permitted by the data. Since HCSUS is a panel data set containing terminally ill patients, we must account for the impact of mortality. To do so, we employ a Tobit model, with the assumption that decedents have CD4 values that are left-censored at  $-1$ , as zero is the lowest possible value that can theoretically be reported by a living individual.<sup>13</sup> That is, the immune function of decedents is assumed worse than the immune function of any living individual.<sup>14</sup>

Identifying the effect of technology on the coefficient  $\beta_t$  requires that there are no other time trends over this short 2-year period that may also influence health disparities. There are two important alternatives to consider. The first is differential diagnosis. 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. A partial solution to this problem is offered by our measures of initial health at baseline: age, age-squared, sex, whether the individual used IV drugs or had gay sex (both of which can affect the virulence of the HIV strain), region of residence, self-assessed health at baseline, and years since diagnosis. Including or excluding these (or various permutations or

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<sup>13</sup> While it is not possible to have literally zero CD4 cells, it is possible for a test to detect no such cells in someone who is extremely ill.

<sup>14</sup> This assumption is reasonable given the clinical progression of HIV. Prior to the introduction of HAART, median CD4 cell counts at the time of death were 10 cells/mL.

combinations of these) variables affects the levels of  $\beta_{1t}$  but it has virtually no effect on its time trend. The other hypothesis is whether or not trends in  $\beta_{1t}$  and  $\beta_{2t}$  are driven by changes in public insurance provision. From our point of view, the effects of private insurance are not problematic, since the decision to purchase (or not purchase) private insurance is essentially a decision to purchase medical care, much like the decision we modeled. However, the provision of public insurance can affect disparities and is outside the model. Therefore, we study the effect of including insurance controls on the change in disparities. We find that, while insurance status has a large effect on health, it does not seem to affect the estimated change in health disparities.

We estimate the Tobit model:

$$CD4_{it} = \beta_0 + \beta_{1t}Sch_i + \beta_{2t}Black_i + \beta_{3t}Health_i + \beta_{4t}Insurance_{it} + \varepsilon_{it}, \quad (6)$$

where  $Health_i$  is a set of correlates for baseline health, and  $Insurance_i$  is a set of insurance variables, and deceased respondents are taken to have a left-censored CD4 value of zero. The estimates for this model are presented in Table 2.<sup>15</sup> The table provides evidence of expansions in socioeconomic inequality over time. At baseline, there is virtually no difference in immune function across education groups, but between baseline and the first follow-up, a substantial gradient emerges between high school dropouts and high school graduates. During the follow-up interviews, high school dropouts have CD4 counts that are on average 50 points lower than high school graduates. However, there does not seem to be any gap between high school graduates and college attendees at any point in the panel. These findings are robust to the inclusion of insurance status, baseline health, and a variety of other covariates. In fact, the inclusion or exclusion of these other regressors does little to alter the basic result that inequality across educational seems to be rising.

We can gain some insight into the nature of HAART's effect by comparing gradients inside and outside the HAART-exposed population, as in Table 3. There are no significant gradients in the population not on HAART, suggesting that patients can do little to improve their own health when not on HAART. This also provides further evidence that our results do not obtain because of different time paths of disease across education groups that are independent of HAART: if the disease progressed differently, we might expect to see differences across

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<sup>15</sup> Since each wave in the table includes all decedents, the small differences across waves are driven largely by missing values for current insurance status (this overwhelms the small amount of attrition for reasons other than mortality).

education groups emerge over time in the HAART-treated population. Selection into HAART status makes it difficult to test this hypothesis directly, but at the very least, it is hard to find evidence of different disease progression across education groups.

While no gradient exists in the untreated population, at any time, a significant gradient is present, even at baseline, in the HAART population. This suggests the importance of differences in adherence to treatment, rather than simply the adoption of treatment. In fact, we cannot rule out the hypothesis that all the gradient that emerges does so as a result of differential adherence, not differential adoption. Under this hypothesis, the gradient in the HAART population multiplied by the prevalence of HAART would be equal to the overall gradient. If true, the CD4 difference between high school dropouts and college attendees would have been:  $59.31 \times (0.24) = 14.23$  at baseline,  $104.53 \times (0.40) = 41.81$  at wave 2, and  $60.66 \times (0.61) = 37.00$  at wave 3. All these are statistically indistinguishable from, and even quantitatively similar to, the overall gradients estimated in Table 2.<sup>16</sup> We are not ruling out the importance of differential adoption of HAART; rather, we are noting that the data cannot reject an explanation that relies entirely on differential adherence.

We investigate the expanding gradient more formally by running a pooled, random effects<sup>17</sup> tobit using all waves of the data, and interacting the education and race variables with the wave of the survey. This allows us to assess whether the expansion in socioeconomic gradients is significant. The results of the pooled model are shown in Table 4: to conserve space, we suppress the coefficients for the variables that are not interacted. The two columns in the table are identical, except that in the first column, the baseline wave is the excluded wave, while in the second wave 2 is the excluded one. This makes it easier to pick out statistically significant changes between any two waves. Note first the significant increase in immune function between Baseline and wave 2. Since HCSUS is a panel of terminally ill patients, one would expect immune function to decline over time, but the new drugs appear to have more than offset this tendency between the first two waves of the survey. The expansion in disparities between high school dropouts and other groups is significant, and it takes place between Baseline and wave 2. There is little change in the education gradient between waves 2 and 3. Moreover, there is some expansion in disparities between high school graduates

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<sup>16</sup> Note also that the unusually high gradient for the HAART population in Wave 2 is not statistically distinguishable from the other waves of HAART users.

<sup>17</sup> The random effects structure accounts for the possibility that the observations on a single individual could be correlated across waves.

and college attendees, but it is not sustained through the end of the panel. In any event, the benefits of the new technology seemed to accrue disproportionately to graduates of high school.

While the results are not shown, we also ran specifications where we interacted public, private, and lack of insurance with the survey waves. Interestingly, we found no evidence to suggest that gradients across insurance status changed over time. This is consistent with the earlier finding that differences in adherence seem more important than differences in adoption; insurance status might affect who gets the treatment, but it may have less impact on how well someone uses it.

Our findings are summarized in Figure 2. From the pooled tobit regression, we calculate predicted CD4+ counts for every respondent in the sample. We then average these predicted values within education groups and waves of the survey to calculate the extent to which disparities changed for actual respondents in the HCSUS. Figure 2 displays the results.<sup>18</sup> At baseline, the difference between high school dropouts and college attendees was less than 15 CD4+ points, but in subsequent waves, it expanded to be nearly 80 points. To provide some context, at HCSUS baseline the average CD4+ count of people diagnosed by a physician with end-stage AIDS was 176, but was 399 for people who were simply symptomatic HIV+, and 465 for asymptomatic HIV+. The disparity is more than one-third the distance between AIDS and symptomatic HIV, and about one-quarter the distance between end-stage AIDS and asymptomatic disease.

### **C.3 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.<sup>19</sup> In 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%, 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).

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<sup>18</sup> These predicted disparities are more reliable than those estimated from the interaction terms in the tobit model, because magnitudes of interaction terms are not readily interpretable in such models (Ai and Norton, 2003).

<sup>19</sup> 1960 data are based on Kitagawa and Hauser (1973). 1986 data are based on the 1986 National Mortality Followback Survey (NMFS).

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, 1970). Not coincidentally perhaps, mortality from heart disease began to fall from 1973 onwards and continued to fall until the early 1990s, 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 emergence of beta-blockers and in part also the clinical validation of diuretics provided a new pharmaceutical therapy for hypertension that supplanted earlier treatments, which placed less emphasis on drugs and more on diet, exercise, and weight control. Prior to the advent of the drugs, relatively few hypertensive patients received any active pharmaceutical treatment from their physicians (Carruthers, 2002). The advent of the new drugs, however, made these effort investments less important.

To analyze the effect of antihypertensive drugs, we 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.<sup>20</sup> From 1948 onwards, this cohort received biennial medical exams, which also included interviews about health history and behavior. We 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.

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<sup>20</sup> 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 much less frequent.

At baseline, all individuals reported their age and educational attainment, which was broken down into six groups: less than 8<sup>th</sup> grade, some high school attended, high school 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. We choose measures that are clearly linked to hypertensive disease, as well as several other conditions that presumably were unaffected by new anti-hypertensive treatment as a pseudo-control group. Measures related to hypertension include: high blood pressure itself, and a physician's diagnosis of hypertensive cardiovascular disease (HCVD)—defined as the presence of high blood pressure and an enlarged heart. The pseudo-control measures include: a physician's diagnosis of any respiratory abnormalities; and elevated blood sugar.<sup>21</sup> As an additional control, we use the individual's self-report of whether he smoked cigarettes during the past two years, to assess the importance of changes in smoking behavior around this period of time.

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-eardiograms (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 5 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. Most striking are the 9 percentage point declines in hypertension and hypertensive cardiovascular disease. Much of this decline has been 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

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<sup>21</sup> 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. The medical evidence shows that treating hypertensives with beta-blockers has no medically significant effects on high blood sugar (Majumdar, 1999).

abnormalities turn out not to be statistically significant, but the decline in smoking (as is well known) was statistically and substantively large. However, we will show that these declines were not skewed towards the less educated groups, at least to the extent that the Framingham data can detect such a pattern.

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, 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 estimate the following regression via OLS:

$$Health_{it} = \beta_0 + \beta_1 Ed_i * post_{it} + \beta_2 Age_{it} + \varepsilon_{it} \quad (7)$$

$Ed_i$  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 allow us to investigate whether health disparities changed after the introduction of antihypertensive drugs. If beta-blockers were indeed simplifying, one might expect a lessening of health disparities in conditions related to uncontrolled hypertension.

The results of the regression are shown in Table 6. 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, and no significant declines in any of the other diseases. This is useful for our purposes, because we can compare disparities in hypertension to disparities in diseases that did not experience observable reductions 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 the stability of the gradient in hypertension itself, it is important to note 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, on the other hand, will manifest itself in complications such as HCVD. Therefore, we expect the gradient in HCVD to be more affected by treatment than the gradient in hypertension itself.

The contraction in HCVD disparities is notable. 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. Thus, our findings are not generic to all diseases in the Framingham cohort, but seem confined to complications of hypertension.<sup>22</sup> This makes it less likely that our results are driven by changes in the generosity of public insurance, or other public policies designed to favor the health of the poor.<sup>23</sup>

As shown in Table 5, rates of smoking fell dramatically over this period. If less educated people benefited disproportionately from this decline, our results could be spurious. Table 6 presents two regressions that suggest otherwise. First, in the right-most column of the table, we show that gradients in smoking rates did not change significantly, in spite of the dramatic decline in mean smoking rates. Over this particular period of time and in this sample, smoking reduction does not seem to be skewed towards any particular education group. Second, we show that the gradient in hypertensive cardiovascular disease fell even for non-smokers. If smoking created a spurious effect, one would expect the compression to be less pronounced among non-smokers. In fact, there is no statistical difference in compression between all people and non-smokers: the point-estimates of compression are actually somewhat higher for non-smokers than for the entire population. Both these results suggest that the compressed gradient is being driven by something other than smoking decline.

#### D. Conclusions

Simply by improving the productivity of health care, new technologies can widen disparities across socioeconomic groups. However, new treatments that simplify the production of health and reduce the importance of patient effort work in the opposite direction. Regardless of what one believes about the precise causal relationship between education and health, the future course of health disparities will be determined by the interplay between chronic illness growth and the nature of technological change.

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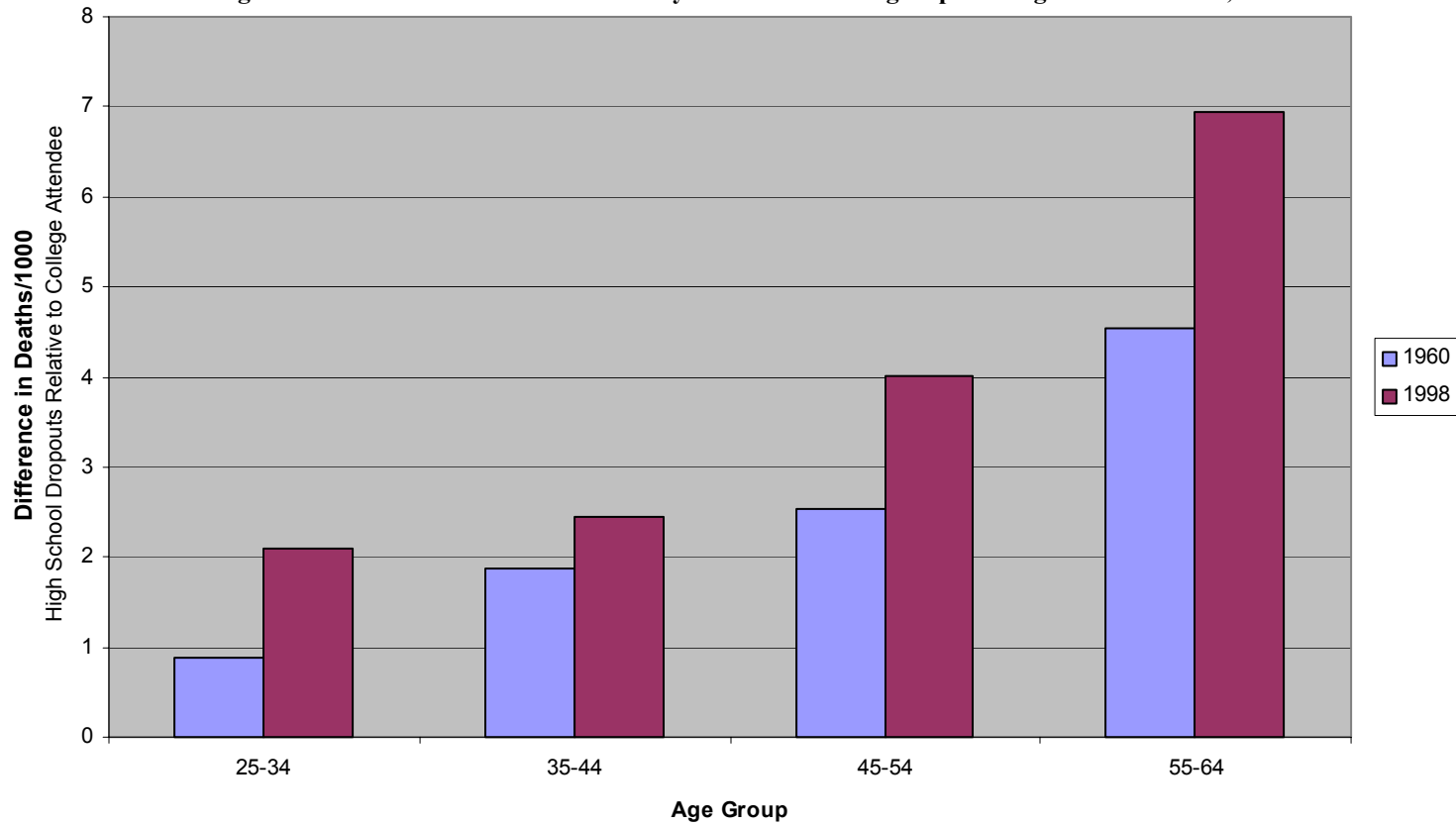
<sup>22</sup>This also provides further evidence that declines in the relative quality of high school graduates cannot explain our results. Ultimately, this is not so surprising, since there is relatively little evidence in the labor economics literature to support the contention that the relationship 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.

<sup>23</sup> Unfortunately, the Framingham data do not include information about insurance status.

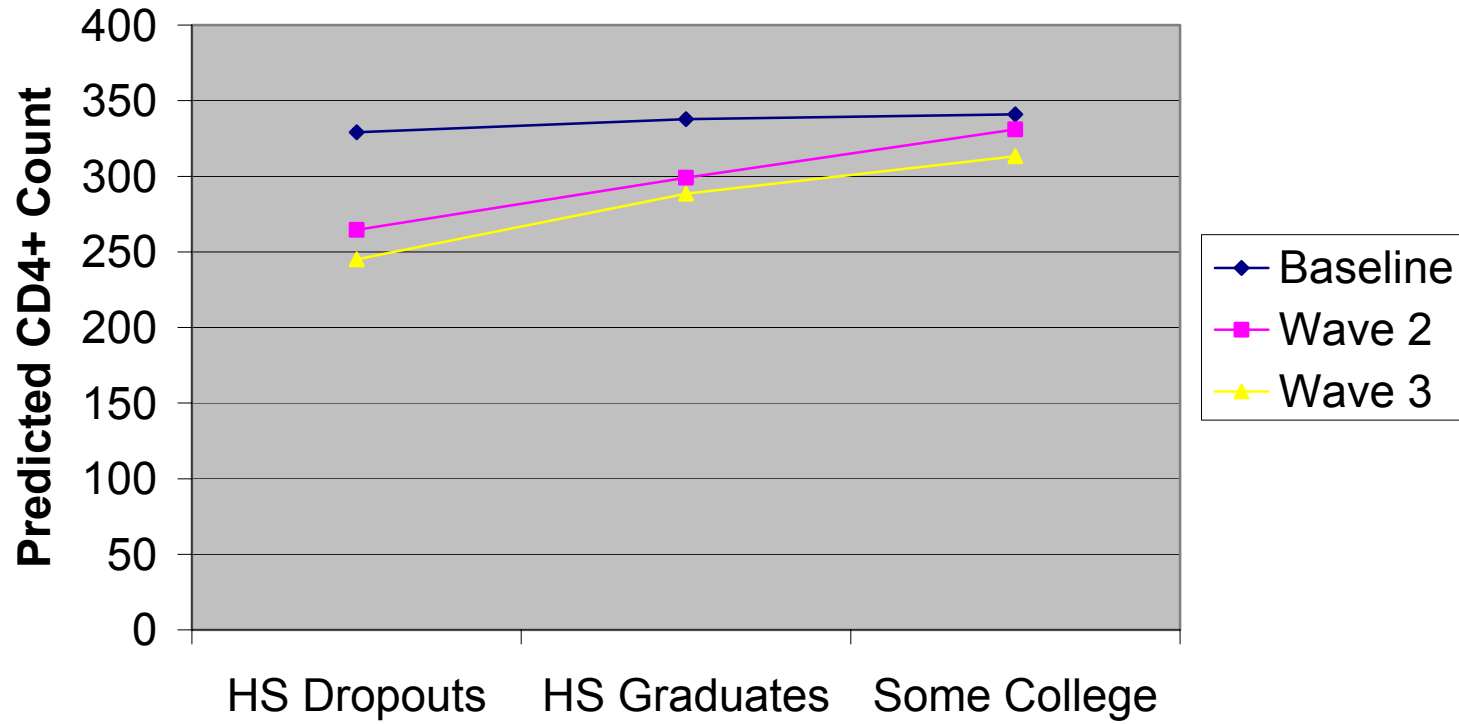
We have presented evidence that new medical breakthroughs have systematic and predictable effects on health disparities. Breakthroughs that lower the price or raise the productivity of medical care expand health disparities. Those that simplify health care and make patient effort less important can have the effect of compressing health disparities. We have presented empirical examples on each side of this divide: complex new treatments for HIV appear to have increased disparities among HIV+ individuals, while pharmaceutical breakthroughs in the treatment of hypertension made self-management less important and coincided with a contraction in disparities for complications of hypertension. Both these findings suggest that the ultimate path of disparities will be heavily influenced by the nature of technological change.

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. Simplifying technologies may be more likely to arise when large numbers of poor people suffer from a disease, and vice-versa. Future work should examine the theoretical linkage between the growth in the overall standard of living and the incentives for patient-intensive technology; these are issues not only of interest for economic theory but also health policy.

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



**Figure 2: Growth in Predicted CD4+ Disparities, based on HCSUS Tobit Estimation.**



**Table 1: Characteristics of the HCSUS Population.**

	Baseline	Wave 2	Wave 3
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 <sup>a</sup> (0.43)	0.40 <sup>b</sup> (0.49)	0.61 <sup>c</sup> (0.49)
Observations	2864	2466	2267

Note: Standard deviations appear in parentheses below means.

<sup>a</sup>Based on 2828 observations.

<sup>b</sup>Based on 2405 observations.

<sup>c</sup>Based on 2216 observations.

**Table 2: Effect of HAART on Health Disparities among HIV Patients**

Variable	CD4+ lymphocyte count (cells per mm <sup>3</sup> )		
	Baseline	Wave 2	Wave 3
Years of schooling (excluded=13 years or more):			
Less than 12 years	-4.24 (13.71)	-47.03 (17.69)***	-45.30 (18.29)**
12 years	5.79 (12.85)	-11.34 (16.72)	5.84 (16.97)
Age	-13.07 (4.35)***	-5.05 (5.43)	-4.65 (5.55)
Age squared/1000	146.88 (53.16)***	49.02 (65.63)	41.16 (65.93)
Black	20.55 (11.86)*	-4.09 (15.50)	-15.94 (16.05)
Female	47.66 (13.99)***	59.72 (18.16)***	47.04 (19.00)**
Used intravenous drugs	11.74 (15.65)	-4.84 (20.23)	-23.76 (21.10)
Had sex with men (0 if female)	-1.52 (13.82)	16.99 (18.15)	19.23 (18.66)
Region (excluded=Midwest):			
Northeast	52.77 (18.50)***	19.80 (24.76)	15.88 (25.60)
West	81.75 (17.10)***	73.00 (22.96)***	74.23 (23.57)***
South	29.83 (17.46)*	84.36 (23.22)***	88.75 (23.99)***
Insurance (excluded=None):			
Medicaid	-69.65 (14.70)***	261.85 (18.40)***	412.56 (19.41)***
Medicare	-84.30 (23.26)***	197.36 (29.05)***	319.10 (26.66)***
Private Insurance	-53.40 (15.12)***	254.19 (19.20)***	390.63 (19.76)***
Medicaid and Medicare	-106.20 (18.31)***	239.39 (22.06)***	353.00 (21.92)***
Self-reported baseline health (excluded=Poor):			
Excellent/Very Good	105.00 (15.56)***	145.64 (20.39)***	173.21 (20.72)***
Good	87.01 (13.68)***	133.55 (17.77)***	113.29 (18.02)***
Fair	28.01 (12.68)**	35.39 (16.54)**	36.01 (16.89)**
Years Since Diagnosed with HIV	-1.85 (1.72)	-5.46 (2.35)**	-10.84 (2.43)***
Observations	2457	2468	2414
Decedents (Left-Censored)	0	397	596

**Table 3: Change in Health Disparities by HAART Status.**

Variable	CD4+ lymphocytes (cells/mm <sup>3</sup> )			
	Baseline	Wave 2	Wave 3	
Years of schooling (excluded=13+ years):				
On HAART	Less than 12 years	-59.31 (27.20)**	-104.53 (28.03)***	-60.66 (20.12)***
	12 years	6.08 (19.94)	-33.46 (23.87)	-13.71 (17.83)
Observations		591	873	1151
Years of schooling (excluded=13+ years):				
No HAART	Less than 12 years	-1.81 (15.80)	-8.33 (20.74)	5.42 (29.49)
	12 years	10.77 (15.65)	1.30 (20.54)	35.58 (27.61)
Observations		1845	1159	643

Standard errors in parentheses

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

Note: These are the results of a Tobit Regression, in which decedents are considered to have left-censored CD4 values of minus 1.

Note: These are the results of a Tobit Regression, in which decedents are considered to have left-censored CD4 values of minus 1.

**Table 4: Pooled Tobit Analysis of Disparities Among HIV Patients.**

Variable	CD4+ lymphocyte count (cells per mm3)	
	(1)	(2)
Baseline		-15.20 (7.52)**
Wave 2	15.20 (7.52)**	
Wave 3	8.38 (7.69)	-6.82 (7.64)
Less than 12 Years Schooling	2.97 (14.99)	-59.42 (15.13)***
Less than 12 Years Schooling*Baseline		62.39 (12.34)***
Less than 12 Years Schooling*Wave 2	-62.39 (12.34)***	
Less than 12 Years Schooling*Wave 3	-59.37 (12.77)***	3.02 (12.84)
12 Years Schooling	5.36 (14.55)	-25.72 (14.78)*
12 Years Schooling*Baseline		31.07 (12.39)**
12 Years Schooling*Wave 2	-31.07 (12.39)**	
12 years Schooling*Wave 3	-16.59 (12.61)	14.48 (12.82)
Black	15.20 (13.14)	1.85 (13.32)
Black*Baseline		13.35 (11.10)
Black*Wave 2	-13.35 (11.10)	
Black*Wave 3	-35.01 (11.49)***	-21.67 (11.54)*
Total Person-Years	7339	7339
Total Person-Years for Decedents	2762	2762

Standard errors in parentheses

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

Note: Decedents are considered to have left-censored CD4 counts of -1.

All regressions also include: dummies for region (Northeast, West, South, Midwest), age, age-squared, whether the respondent ever used IV drugs or had gay sex with men, years since diagnosed with HIV, and dummies for self-reported baseline health (poor, fair, good, or excellent/very good), female, dummies for insurance status (None, Medicaid, Medicare, Private Insurance, Medicaid and Medicare), and a constant term. Specifications (1) and (2) differ in the wave of the

**Table 5: 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
Smoked	50.3%	0.50	19.9%	0.40
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. An individual is said to have smoked if he reported smoking cigarettes within the past two years.

**Table 6: Effects of Beta-Blockers on Health Disparities in Heart Disease.**

	Hypertensive CVD			Any Resp. Disorders	High Bld Sug (>1.5 mg/mL)	Smoked
	Hypertens.	All People	Non-Smokers			
Some HS	-0.02 [0.03]	-0.1 [0.03]***	-0.13 [0.05]***	0.04 [0.03]	0.02 [0.02]	0.06 [0.03]**
HS Grad	-0.05 [0.03]*	-0.11 [0.03]***	-0.15 [0.04]***	-0.03 [0.02]	0.03 [0.02]**	0.01 [0.03]
Some College	-0.07 [0.03]***	-0.11 [0.03]***	-0.19 [0.04]***	-0.04 [0.02]**	0.02 [0.01]	-0.01 [0.02]
Post-Beta Blockers	-0.14 [0.03]***	-0.14 [0.03]***	-0.29 [0.04]***	-0.02 [0.02]	-0.02 [0.02]	-0.31 [0.03]***
Some HS*	0.04 [0.05]	0.11 [0.05]**	0.14 [0.06]**	-0.07 [0.03]*	-0.03 [0.03]	-0.06 [0.05]
HS Grad*	0.06 [0.04]	0.08 [0.04]**	0.13 [0.05]**	-0.01 [0.03]	-0.03 [0.02]	0 [0.04]
Some College*	0.03 [0.04]	0.03 [0.04]	0.1 [0.05]**	-0.02 [0.03]	-0.05 [0.02]**	-0.03 [0.04]
Age	0.01 [0.04]	0.13 [0.04]***	0.25 [0.06]***	-0.03 [0.03]	0.06 [0.02]**	0.03 [0.05]
Age-Squared	0 [0.00]	0 [0.00]***	0 [0.00]***	0 [0.00]	0 [0.00]**	0 [0.00]
Male	-0.03 [0.02]**	-0.03 [0.01]**	-0.02 [0.02]	0.09 [0.01]***	0.03 [0.01]***	0.12 [0.02]***
Constant	0.14 [1.46]	-4.18 [1.40]***	-8.11 [2.04]***	0.82 [0.95]	-2.18 [0.79]***	-0.96 [1.70]
Observations	8212	8078	4258	8105	7401	6837
Unique Individuals	2454	2454	1804	2454	2424	2392
R-squared	0.02	0.02	0.06	0.03	0.02	0.12

Robust standard errors 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.

Smokers are defined as people who have smoked within the last two years.

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