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Health Inequality, Education and Medical Innovation*

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Preliminary version-Comments Welcome

Abstract: Health inequalities across socio-economic groups in the US are large and have been growing. We hypothesize that, as in other, non-health contexts, this pattern occurs because more educated people are better able to take advantage of technological advances in medicine than are the less educated. We test this hypothesis by relating education gradients in mortality to a measure of medical innovation -- the number of active drug ingredients available to treat a disease. We use the Mortality Detail Files and SEER cancer data to estimate consistent causal effects of education on mortality, using compulsory schooling laws in the earlier part of the 20th century as our measure of education. We find that more educated individuals have a larger survival advantage in those diseases where there has been more medical progress. These effects are greater for more recent progress than for older progress, supporting the hypothesis that gradients emerge at the time of innovation. We perform a series of additional analyses to rule out alternative theories.

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Introduction

Socioeconomic disparities in health have increased over the past century in the U.S., the U.K., and continental Europe (Feldman et al., 1989, Pappas et al., 1993, Preston and Elo, 1994, Black Report, 1980, Kunst et al., 2001, Shkolnikov et al., 1998). In the U.S., between 1960 and 1986, the age-adjusted mortality rate for white men who had attended college declined from 5.7 to 2.8 per 1000, while the rate declined only from 9 to 7.6 for those who had not graduated high school (Pappas et al., 1993). Yet, the principal causes of death and disability generating socioeconomic differentials today are quite different from those a century ago (McKeown, 1976; Cutler and Meara, 2002). Over this period, housing, nutrition, and sanitation have improved; the infectious diseases that were the prime causes of death before World War I account for little mortality today; and access to effective medical care for those diseases that remain has become more widespread. Today, gradients are largest for chronic diseases.

Many factors can generate socioeconomic differences in health status at a point in time. What is more difficult to explain is that these differentials have increased and have shifted among diseases during the past century (Carroll, Davey, Smith, and Bennett, 1996). To our knowledge there is no existing research that has systematically investigated the question of why gradients move among diseases and widen over time.

In this paper we propose and test one explanation for this pattern: the gradient moves among diseases because education enhances the ability to exploit technological advances in medicine. For this reason, the gradient increases where and when technological change occurs. The most educated make the best initial use of new information about different aspects of health.

Our hypothesis is an extension to health of Nelson and Phelps' (1966) theory that "the return to education is greater the faster the theoretical level of technology has been advancing (p. 72)." A substantial literature examines this pattern in the labor market (see, for example, Bartel and Sicherman, 1999; Allen, 2001) and in the agricultural sector (Wozniak, 1984). Our

hypothesis is also closely related to the sociological conjecture that socioeconomic status is a "fundamental social cause" of gradients in health (Link, et al., 1998). A fundamental cause is one that involves access to resources that can be used to avoid or minimize risks, influences multiple risk factors, and affects multiple disease outcomes. In this view, more education enables people to better exploit new information and resources.

We test this hypothesis by relating education gradients in mortality to a measure of medical innovation -- the number of active drug ingredients available to treat a disease. We use the Mortality Detail Files and SEER cancer data to estimate consistent causal effects of education on mortality, using compulsory schooling laws in the earlier part of the 20th century as our measure of education. We find that more educated individuals have a larger survival advantage in those diseases where there has been more medical progress. These effects are greater for more recent progress than for older progress, supporting the hypothesis that gradients emerge at the time of innovation. We perform a series of additional analyses to rule out alternative theories.

Relating Outcomes to Progress

Many recent studies of health status differentials focus on gradients associated with education. These studies generally find that education is closely correlated with health status (even controlling for income), and suggest that the relationship is causal.[1] However, this result is still controversial (See Fuchs, 1982), in part because the mechanism(s) by which such a relationship might operate are not well understood. Similarly, the idea that education increases the rate of adoption of innovations is accepted in many contexts, yet it is not obvious that education accelerates diffusion in the context of health.

1 For example see Lleras-Muney (2002b) shows that increases in education induced by compulsory schooling laws lead causally to improvements in health status.

There are several reasons to expect that the relationship between education and health is affected by technological innovation. As in other contexts of diffusion, more educated people are better informed about medical innovation. According to a 1999 National Science Foundation survey, 32% of those with more than a college degree reported they were both very interested and very well informed about new medical discoveries, in contrast to only 14% of those with less than a high school degree.[2] The survey also found that more educated people also have a more positive view of the risks and benefits of innovation: 71% of those with a college degree or higher thought that the benefits of new technologies would strongly outweigh any harmful consequences, in contrast to only 25% of those with less than a high school degree.[3] Greater access to information and more positive valuations of the benefits could lead to the more educated adopting newer medical innovations first. Lleras-Muney and Lichtenberg (2002) find that more educated individuals are more likely to use prescription drugs more recently approved by the FDA.

Medical innovation differs from other kinds of innovation because most medical innovations are prescribed or implemented by medical professionals, not by patients themselves, however educated. As prior research has documented, there are enormous variations in the practice patterns of medical professionals (Skinner et al., 2002; Chandra and Skinner, 2003). In this context, more educated people would have an advantage in the presence of innovation if they were more effective at searching for high quality providers. Consistent with this reasoning, researchers have found that the more educated are more likely to participate in clinical trials, where they would gain access to the newest treatments ().

In the labor market context, more educated people are more adept at implementing new technologies (such as computers) in their early stages, when these technologies are complicated to use. Over time, technologies become refined and are more accessible to less educated people. In

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2 From Science and Engineering Indicators 2000. published by the National Science Foundation. The report can be found at the following website:

the health context, more educated people may be better able to understand and tolerate complex dosing regimes or side effects. In the case of new HIV drugs, for example, complex-dosing regimes contributed to reduced early diffusion of the drugs to less educated groups. As physicians and patients gained expertise with the drugs, they diffused to other populations (Cunningham et al, 2000).

Finally, there are several other mechanisms that could generate a correlation between education and technology adoption, operating through other factors. Education raises income, and income might provide better financial access to quality care. Higher income people are also able to support better-endowed hospitals and more medical specialists. We will try to distinguish our causal hypothesis from these alternatives in the last section of the paper (note that these hypotheses are not exclusive and may all be true to varying extents).

We now provide a formalization of our hypothesis. The following formalization closely follows Nelson and Phelps (1966). This model is only illustrative. It captures the basic features of our hypothesis in a simple fashion and provides guidelines for our empirical approach.

Suppose that the health H of an individual can be modeled as a function of the level of technology A that the individual has access to, education E and other inputs C:

$$H = H(A, E, C)$$

The frontier level of technology is given by T(t), where

$$T(t) = T_0 e^{\lambda t},$$

where T(t) is the level of technology if technology is instantaneously diffused and λ is the exogenous rate of technological progress. Suppose now that the level of technology available to any individual depends on how rapidly individuals adopt new technologies, and that the lag between innovation and adoption is a decreasing function of education, so that

$$A(t) = T(t - w(e)) = T_0 e^{\lambda (t - w(e))},$$

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http://www.nsf.gov/sbe/srs/seind00/access/toc.htm

where w'(e)<0. This key assumption captures the ideas that were presented in the introduction -- that is that the more educated "adopt" new technologies at a faster rate because of better access to information, better use of information, and better capacity to search for better providers and or treatments. This feature can be generated from maximization principles simply by assuming differential costs of technology adoption. Note that this model assumes that individuals have chosen education in previous periods and that technological changes are unanticipated. In this context, we can express the health of the individual as

$$(1) \quad H = H(T_0 e^{\lambda(t-w(e))}, E, C)$$

The derivative of the health production function with respect to education gives us what is known as "the education gradient" in health. It gives the marginal gain in health induced by an additional unit of schooling. In this model it is composed of the effect of education on adoption and of the effect of education on health that is not mediated by technology. For example, the more educated might simply be better at complying with prescribed treatment (Goldman and Smith, 2001). The gradient can be expressed as:

$$(2) \quad \begin{aligned} \frac{\partial H}{\partial e} &= -\lambda w'(e) A \frac{\partial H}{\partial A} + \frac{\partial H}{\partial E} > 0 \\ \frac{\partial H}{\partial e \partial \lambda} &= -w'(e)\left[A \frac{\partial H}{\partial A} + \lambda \frac{\partial A}{\partial \lambda} \frac{\partial H}{\partial A} + \lambda \frac{\partial H}{\partial A \partial \lambda} \right] > 0 \end{aligned}$$

Since w'(e) is negative, the model predicts that health is an increasing function of education and that the rate of return of education is larger the higher the rate of technological change. We test this prediction in data by estimating the disease-specific education gradient and relating the size of the gradient to measures of innovations that proxy for the parameter λ.

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3 ibid.

Empirical Strategy

We are interested in estimating a linear probability model of the probability of dying within 5 years, where education is interacted with progress:

$$(3)\ P(died = 1) = \beta_0 + \beta_1 education + \beta_2 education * progress + X\gamma + e$$

where X includes disease fixed effects, and other individual characteristics. Our model suggests β_2, the interaction between education and progress should be negative (because the outcome is dying rather than positive health), so that the survival advantage of the more educated is larger for diseases with more progress.

To estimate this equation we need to find disease-specific measures of progress. There is no consensus about how to measure either progress or the relative importance of progress (Allen, 2001; Bartel and Sicherman, 1999). We use the number of active ingredients approved by the FDA to treat a particular disease as a proxy for innovation in medical technology. We use the number of active ingredients rather than drug approvals, since the former constitute greater pharmaceutical innovations. Note that the number of drugs to treat a particular disease is an imperfect proxy for innovation: some of the major innovations in medicine in the last decades, such as angioplasty or MRI, are diagnostic or surgical innovations (Fuchs and Sox, 2001).

Data

We use two sources of mortality data for this project: the SEER data, which contains information about cancer mortality conditional on cancer diagnosis 1973-1993; and the Mortality Cause of death data, which contains death rates for all diseases from 1960 to 1990.

Neither the SEER data nor the Mortality data include information on educational status. Instead, we use compulsory schooling laws to measure. Several papers have shown that these

laws had an impact on educational attainment.[4] These laws specified the minimum number of years that a child had to attend school.[5]

Both data sets contain information about state of birth, year of birth, gender and race. We can therefore match individuals to compulsory attendance and child labor laws in place in their state of birth when they were 14 years of age. The implicit number of compulsory years of schooling ranges from 0 to 10 for the cohorts we study. Because compulsory schooling laws were most effective in the first half of the 20th century and they only affected whites (see Lleras-Muney 2002a) we restrict our attention to white cohorts born between 1901 and 1925.

Our cancer data come from the SEER Cancer Incidence Public Use Database collected by the National Cancer Institute. The data contain information on every person diagnosed with cancer from 1973 to 1998 in 9 SEER registries (registries are composed of several counties located in San Francisco, Connecticut, Detroit, Hawaii, Iowa, New Mexico, Seattle, Utah and Atlanta[6]). Information on vital status was recorded for all individuals in the sample as of 1998. These data allow us to look at 5-year mortality, conditional on cancer diagnosis from 1973 to 1993.[7] Summary statistics for the final SEER sample are in Table I. Our sample is relatively old because we exclude people born after 1925: average age at diagnosis for this sample is around 70.[8] About 2/3 of the population died within 5 years of diagnosis, most frequently from cancers of the digestive system, of the respiratory system and of the genital system.

We also use mortality rate data from the Mortality detail causes of death files. We can calculate 4-year mortality by matching mortality counts in the Mortality Detail files with

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4 See Acemoglu and Angrist (1999), Angrist and Krueger (1991), Lleras-Muney (2002b), Margo and Finnegan, (1996) and Schmidt (1996).

5 The data on compulsory attendance and child labor laws were collected from multiple sources (See Lleras-Muney, 2002a, for details). We use only two laws: the age at which a child had to enter school and the age at which he could get a work permit and leave school. The difference between these two variables measures the implicit number of years a child had to attend school.

6 The Seer data include two more registries (San Jose and LA) but we exclude them since data are only available from 1992 to 1998 for these registries.

7 To avoid censoring we drop individuals diagnosed after 1993. We have estimated Cox proportional Hazard models and they yield similar results, but the estimations are very slow because of the large number of observations and explanatory variables.

8 The average age at diagnosis is around 62 in the full SEER data. Our sample is older but not much more.

population data from the Census. We construct death rates by cause of death, gender, cohort and state of birth for three periods, 1960-1963, 1980-1983 and 1990-1993. We aggregate causes of death (ICD 9 codes for 1980 and 1990, ICD 7 codes for 1960) into the 72 broad categories that are commonly used in other mortality data such as the National Health Interview Survey by the CDC. We drop all death rates from unknown or external causes. Summary statistics for this data are in Table II. Not surprisingly, since we are calculating unconditional mortality, the average mortality rate is much lower than in the cancer data, about 3 per thousand. Our sample here is somewhat younger (mean age about 65) because we include data from 1960. The main causes of death in the data are cardiovascular disease, cancer, and respiratory diseases.

Each data set has its own advantages. The mortality data contains deaths from all causes and spans a longer period of time. The cancer data gives us information on cancer mortality *conditional on diagnosis*. The SEER provides individual level data, so it contains more control variables. Furthermore, the match between drugs and diseases is better in the SEER than in the mortality data, since drugs typically treat specific diseases (rather than causes of death) and we know each individual's particular condition. In the mortality data, the match between drugs and causes of death can be imprecise: for example, drugs to control diabetes can reduce death rates from diabetes, but also from heart disease, stroke, kidney failure, etc. The mortality data does not include county identifiers, but we have greater variation in the number of drugs approved by disease and decade.

The data on active ingredients, the condition(s)[9] they were approved to treat, and their approval dates was given to us by Frank Lichtenberg, and originally were obtained through a FOIA request to the FDA. Unfortunately, not all drugs in our data can be dated: some were invented prior to the creation of the FDA (1938) and there are others for which we could not impute a date of approval.

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9 Note that a single drug can be used to treat many conditions, and a condition can have many drugs to treat it.

We also matched our data (by county or state) with a number of other data sets. See Appendix C for a complete description of the data sources.

Documenting the gradient

Recall that neither data set contains actual years of education. Instead we use the number of years of compulsory schooling applicable to an individual. By including laws in place of education in a model of mortality/health, we are estimating a reduced form equation. The advantage of this method is that we can argue that the effects we measure can be interpreted as causal, assuming that these laws affected mortality only through their effect on education. On the other hand, we are identifying the effect of education for those affected by these laws, i.e. those at the lower end of the distribution of education.

We begin by documenting education gradients in the SEER data. We estimate the model in (3) above without including an interaction, where X includes 47 state of birth dummies, 24 cohort dummies, 8 registry dummies, 2 decade dummies, 4 stage of diagnosis dummies, and dummies for 80 cancer sites and for each site interacted with stage at diagnosis.[10] Since we include state of birth and cohort dummies, the effect of compulsory schooling laws is identified from variations in the laws within states over time. The results are in Panel A of Table III. We find a negative and significant effect of compulsory schooling on mortality for both males and females.

In panel B, we present estimates of the same model using the mortality data. The mortality data is aggregated to cells defined by cause of death, gender, and cohort and state-of-birth. The regression includes gender, age, age squared, year dummies, and dummies for cohort and state of birth. As in the cancer data, we identify the effect of compulsory schooling from variation in the laws within states overtime. Again we find negative and significant effects of

compulsory schooling on mortality for both genders, although these are much smaller in magnitude.

In order to better interpret these effects, we estimate the implied two-sample IV estimates of the effect of education on mortality in Table III. Using the 1960, 1970, 1980 and 1990 censuses we can estimate the first stage, i.e. the effect of compulsory schooling on educational attainment. We find that the effect of one more year of compulsory schooling on education is about 0.05 for both genders. Since the model is exactly identified and provided that we include the same covariates in both estimations, the Two-Sample IV estimate of the effect of education on mortality can be calculated as the ratio of the reduced form equation estimate and the first stage estimate.[11] Using estimates from the first two columns, we find that the TSIV estimate of the effect of education on cancer mortality conditional on diagnosis is somewhere between –0.018 and –0.050 (Panel A). At the means, this coefficient suggests that one more year of compulsory schooling reduces the probability of dying of cancer (in the SEER data) within 5 years of diagnosis by about 7%. For unconditional mortality (Panel B) the implied coefficient is between -0.0006 (female) and –0.002 (male), so one more year of schooling reduces all-cause 4-year mortality by about 36%. Note that in both samples, the effect of education on mortality is greater for men than for women. The effect of education on mortality is greater in the all-cause data than in the cancer data, which suggests that education may reduce the incidence of disease as well as improving survival conditional on disease. The TSIV estimates in the all-cause mortality data are substantially smaller than those found by Elo and Preston (1996) who report that the effect of one more years of schooling on 5-year (all cause) mortality is between 0.02 and 0.05. Since our objective is to look at whether the education gradient is related to progress (rather than to measure

10 Cox proportional hazard models yielded similar results but given that the data are large and that we include many variables, these estimations take a very long times to converge. We therefore present linear probability models instead.
11 This method was used by Dee and Evans (1999).

the gradients themselves), in the remainder of the paper we will present reduced form estimates of the effect of compulsory schooling on outcomes.[12]

The Effect of Progress on the Gradient in Mortality

We relate the education gradients by disease to progress (measured by number of drugs) for that disease. In the SEER data, we estimate equation 3 above, i.e. we regress the probability of dying within 5 years of diagnosis on compulsory schooling and on the interaction between compulsory schooling and the number of drugs.[13] Panel A of Table IV reports the estimated coefficients for the interaction term, for the entire sample and by gender. All coefficients are negative as predicted, but only the estimates for males are significant. The magnitude of the estimate for women is also much smaller. These results are quite robust to the inclusion of covariates (Appendix D): once cancer site dummies are included, adding other covariates has little impact on the magnitude and significance of the interaction estimates. In panel B we report similar aggregate estimates using the Mortality files. All coefficients are negative and significant. Unlike in the cancer data, the estimates of the interaction are quite similar for men and women.

Theme estimated interaction term evaluated at the mean of the data suggest that about 48% of the education gradient in male cancer mortality is mediated by technological innovation, and about 4% and 9% of the gradient in all cause mortality for men and women respectively Overall we find support for the hypothesis that the education gradient is steepest for those diseases where there has been the most progress, although within cancers, this seems to be true only of men.

12 The inclusion of state-of-birth and cohort specific variables (such as infant mortality, number of hospitals per mile and number of doctors per capita in state-of-birth at age 14) did not affect the results. Results available upon request.

13 Results using a non-linear probability model yield similar effects. See appendix X

Is the gradient larger for more recent technological progress?

The steady state relationship between an innovation and the gradient in education may vary by technology. For example, if technological innovation is skill-biased (the more educated have higher returns from a particular innovation) steady state gradients will increase. Our hypothesis, however, suggests that a given technology diffuses over time from the more educated to the less educated. This implies that recent improvements in technology should affect gradients more than older improvements do.

We test the hypothesis that gradients are steeper for newer technologies using the Mortality data, by looking at how the interaction of the gradient with progress varies with the timing of progress. We estimate the following equation:

$$MR_t = \beta_0 + \beta_1 * CSL + \beta_{2t} CSL*\#drugs_t + \beta_{2(t-1)} * CSL*\#drugs_{t-1} + \beta_{2(t-2)} * CSL*\#drugs_{t-2} + X\gamma + e \quad (4)$$

where CSL is the level of compulsory schooling, t is the current decade, t-1 is the previous decade, and t-2 is two decades ago. For each decade we calculate the number of drugs approved by the FDA by disease. The number of drugs approved in 1960 is the proxy of current progress associated with four- year mortality rates in 1960 (1960-1963); approvals in the 1950s are considered lagged progress, etc; and similarly for other years. We do not perform this test with the cancer data because of the shorter panel and small number of drugs by site and decade.

In Table V we present the estimates of β_{2t}, $\beta_{2(t-1)}$, and $\beta_{2(t-2)}$. In all cases we find that the interaction effect for current and lagged approvals is negative and significant, whereas the interaction with approvals two decades ago is positive and significant. This result suggests that the gradients associated with a particular innovation become smaller over time.

Examining Alternative hypothesis

We have documented a robust relationship between education and technology and also find that recent, but not older, innovations increase the education gradient. We interpret these

results as providing evidence to support our hypothesis that the more educated adopt newer technologies first. However, our results are also consistent with a number of alternative hypotheses. In this section we attempt to provide evidence to suggest that these alternative hypotheses do not fully account for our results.

A- Geographical variation in treatment quality

Gradients in health outcomes might emerge not because of differences in the behavior of patients, but rather because of variations in the quality of care available (Skinner et al. 2002; Chandra and Skinner, 2003). If rich educated neighborhoods have access to high quality providers who adopt new technologies earlier, while disadvantaged, uneducated neighborhoods have access mostly to poor quality providers who adopt innovations later, we would find a significant effect for the interaction between education and innovation. Under this scenario, however, the interaction effect would be completely driven by endogenous co-location of educated patients and high quality providers. How can we distinguish our theory from the geographic inequalities theory?

First, we note that our cancer results are robust to the inclusion of county fixed effects. In these specifications, we are identifying our interaction effects using within county variation. It is, of course, very plausible that there are also provider quality differences within counties, but we would also expect to see substantial variation in quality across counties. We find, however, that controlling for county fixed effects has no impact on our estimates (see Appendix C) of the interaction effect, although the county fixed effects themselves are, as expected, highly significant. This suggests that the pure geographical hypothesis does not explain our interaction results for cancer.

To further explore the issue, we add additional time-varying county covariates in the cancer models.[14] In Panel A of table VI, we add county level mean education and log of per capita personal income. While county income has a significant negative effect on mortality, inclusion of these additional variables has no effect on the interaction coefficient.

Next, we include a proxy for the quality of care available in each county. For the geographic theory above to explain our results across disease types, it must be the case that the quality of treatment of *all* diseases is higher is rich counties than in poor counties. Therefore, we can use cardiovascular (CV) mortality rates of whites in each county and year as a proxy for the general quality of care. We estimate a model where education (denoted CSL for compulsory school), drugs and CV mortality (denoted CVMR) are fully interacted:

$$MR_t = \beta_0 + \beta_1 * CSL + \beta_2 * CVMR + \beta_3 CLS*\#drugs_t + \beta_4 * CVMR*\#drugs + \beta_5 * CSL*\#drugs * CVMR + X\gamma + e \quad (5)$$

The results are in Panel B, Table VI. For men, we find that indeed cancer mortality is higher in areas where CV mortality is higher. We also find that the effect of education interacted with drugs remains negative and significant. Interestingly, the triple interaction between education, drugs and CV mortality is positive and significant. This suggests that the cancer survival advantage of the more educated in the presence of cancer specific progress is reduced when cardiovascular mortality is high. This result is consistent with the idea that individuals who have poor care, are in worse health when and if they get cancer.

Finally, in Panel C of Table VI, we use the number of medical specialist in the county as an alternative measure of the quality of care available, and again estimate the fully interacted model. Again, focusing on significant coefficients for men, we find that counties with more specialists have higher mortality (which may reflect endogenous location decisions) and that the presence of specialists enhances the benefits of progress for all. The interaction between education and drugs is still negative and significant, but the triple interaction with specialists is

14 We don't have county identifiers for all our mortality cause of death files.

positive. This suggests that the benefits of education occur, in part, through more effective searching. When all providers are of higher quality, searching for a good provider matters less.

B- Education and income

It is likely that our estimated effect of education is capturing more than the direct advantages of education. More educated individuals will also have higher permanent income. Suppose that new technologies are expensive, but their financial price falls over time. In this scenario, richer, more educated individuals "purchase" technology first, and poorer less educated individuals purchase it later. We would find that the more educated benefit more from recent progress, but the effect is due to permanent income, not education, per se. This problem is difficult to solve since it is almost impossible to disentangle education and permanent income in survey data.

We have shown (Table IV, panel A) that our results are robust to controls for annual income in the county. This, however, is a very noisy measure of individuals' lifetime resources. We consider two alternative strategies that both rely on the role of income in reducing the effect of financial barriers to care. Note that the income effects of education should be strongest where financial costs are largest, but the direct effects of education need not vary with financial cost. First, we compare cancer patients 65 and over who are Medicare-eligible and those under 65. Medicare patients face fewer financial barriers to cancer care since Medicare covers most related medical expenditures, *including most chemotherapy drugs*.[15] In table VII, we estimate the cancer mortality model for individuals over and under 65.[16] If education primarily acts a proxy for income, we expect the coefficients on the interactions to be greater for those under age 65. In fact, we find the opposite: there is no interaction effect among young males, but there is a significant

15 Medicare always covered anti-cancer drugs if administered intravenously (as most chemotherapy drugs are) in an outpatient setting. Beginning in 1993, Medicare also covered these drugs if they were taken orally and self-administered. See http://cms.hhs.gov/media/press/testimony.asp?Counter=612

interaction for those above 65. As in all other regression with cancer, we find no significant interaction effects for women.

As a second test, we examine how variations in the price of medical care affect these results. Following Chernew, Cutler, and Keenan (2002), we obtained average Medicare expenditures per beneficiary (excluding home health care expenditures) by state and year (MEB). Skinner (2000) has shown that the differences in these expenditures across regions are largely uncorrelated with health outcomes. We estimated a fully interacted model as in equation 5, where MEB replaces CVMR. We hypothesize that higher costs reduce survival, they matter less to the more educated, and they retard the effects of progress less for the more educated (and higher income). The results (Panel B, Table VII) indicate that the interaction is still negative and significant for men. Interestingly, we find that the triple interaction is positive and significant for men, suggesting that the adoption advantage of the more educated in *smaller* where prices are high. Again, this finding suggests that education does not operate entirely through income.

Conclusion

Studies of technological diffusion consistently point to education as a factor that increases the diffusion rate (Hall and xx, REVIEW). We find evidence that this pattern also holds true in the context of health services. More educated people appear to benefit from the development of new health care technologies more rapidly than do less educated people.

Diffusion of medical technologies to patients is mediated by patterns of diffusion among providers. We find some evidence that suggests that the advantages of more educated people are smaller where providers are more uniformly knowledgeable. This result implies that variations in the quality of medical care pose greater hazards to less educated than to more educated patients. Yet our results also suggest that neither geographic variation nor income. alone, explains the

16 Since Medicare does not cover most other drugs, we do not perform this test with the all mortality data.

advantage of educated people in the presence of technological innovation. Education also appears to have a direct, causal, impact on the benefit of innovation.

Over time, we find, the benefits of a given technology do diffuse to less educated people. In an era of accelerating technological innovation, however, this pattern can generate ever-widening gradients in overall health. This prediction is consistent with the experience of the post-1950 period, a period of substantial innovation in the treatment of disease and of widening gradients in health.

References

Acemoglu, Daron and Joshua Angrist, "How Large are the Social Returns to Education? Evidence from Compulsory schooling Laws," NBER Working Paper No. W7444, December 1999

Allen, Steven G., "Technology and the Wage Structure." Journal of Labor Economics, Vo. 19, No. 2: 440-483.

Angrist, Joshua D. and Alan B. Krueger, "Does Compulsory School Attendance Affect Schooling and Earnings?," Quarterly Journal of Economics, November 1991

Bartel, Ann P. and Nachum Sicherman, "Technological Change and Wages: An Interindustry Analysis," The Journal of Political Economy, April 1999: 285-325.

Black, D, Morris J, Smith C, Townsend P. (1980) Inequalities in health: Report of a Research Working Group (The Black Report) Department of Health and Social Security

Chernew, M., Cutler, D., Keenan, P. S. (2002). Rising health care costs and the decline in insurance coverage. National Bureau of Economic Research, Cambridge, MA. Draft paper, November 2002.

Chandra, Amitabh and Jonathan Skinner, "Geography and Racial Health Disparities," NBER Working Paper No. w9513 , February 2003

Cunningham WE, Markson LE, Andersen RM, Crystal SH, Fleishman JA, Golin C, Gifford A, Liu HH, Nakazono TT, Morton SC, Bozzette SA, Shapiro MF, Wenger NS. Prevalence and Predictors of Highly Active Antiretroviral Therapy Use in Persons with HIV Infection in the U.S. Journal of Acquired Immune Deficiency Syndromes, Vol. 25, No. 2, October 2000, pp. 115-123.

Cutler, David and Ellen Meara, "Changes in the Age Distribution of Mortality Over the 20th Century," NBER Working Paper No. w8556, October 2001

Elo, Irma T. and Samuel H. Preston, "Educational Differentials in Mortality: United States, 1979-85," Social Science and Medicine 42(1), 1996

Feldman J. D. Makuc, J. Kleinman and J. Cornoni-Huntley, "National Trends in Educational Differences in Mortality," American Journal of Epidemiology, 1989

Fuchs, Victor R., "Time Preference and Health: An Exploratory Study," in Victor Fuchs, Ed., *Economic Aspects of Health*, Chicago: The University of Chicago Press, 1982

Fuchs, Victor R., and Harold Sox. 2002. "Physicians's Views Of the Relative Importance of Thirty Medical Innovations," Health Affairs, September/October 2001. Volume 20, Number 5.

Goldman, D.P and J.P. Smith. 2002. "Can Patient Self-Management Help Explain the SES Health Gradient?" Proceeding of the National Academy of Sciences of the United States of America 99(16): 10929-10934.

Link, Bruce G. et at, "Social Epidemiology and the Fundamental Cause Concept: on the Structuring of effective Cancer Screens by Socioeconomic Status," The Milkbank Quarterly Vol. 76, No. 3, 1998

Lleras-Muney, Adriana, "Were State Laws on Compulsory Education Effective? An analysis from 1915 to 1939," Journal of Law and Economics, Vol. XLV(2), October 2002a

Lleras-Muney, Adriana "The Relationship between Education and Adult Mortality in the U.S." NBER Working Paper #8986, May 2002b

Lleras-Muney, Adriana and Frank Lichtenberg, "The Effect of Education on Medical Technology Adoption: Are the More Educated More Likely to Use New Drugs," NBER Working Paper No. w9185, September 2002

Margo, Robert A. and T. Aldrich Finegan, "Compulsory Schooling Legislation and School Attendance in Turn-of-The Century America: A 'Natural Experiment'," Economics Letters, October 1996

McKeown, Thomas, The modern rise of Population, New York: Academic Press, 1976
Nelson, Richard R. and Edmund S. Phelps, "Investment in Humans, Technological Diffusion and Economics Growth," American Economic Review, 1966

Pappas, Gregory, Susan Queen, Wolber Hadden and Grail Fisher, "The Increasing Disparity in Mortality Between Socioeconomic Groups in the United States, 1960 and 1986," The New England Journal of Medicine, 1993

Schmidt, Stefanie, "School Quality, Compulsory Education Laws, and the Growth of American High School Attendance, 1915-1935," MIT Ph.D. Dissertation 1996

Skinner, Jonahtan and John E. Wennberg , "How Much is Enough? Efficiency and Medicare Spending in the Last Six Months of Life." The Changing Hospital Industry: Comparing Not-for-Profit and For-Profit Institutions, Cutler, David M., pp. 169-193, (Chicago: The University of Chicago, 2000).

Sorvillo, F. et al. 1999. "Use of Protease Inhibitors Among Persons with Aids in Los Angeles County." AIDS Care 11(2): 147-155.

Wozniak, Gregory D., "The Adoption of Interrelated Innovations: A Human Capital Approach," The Review of Economics and Statistics, Vol. 66, No. 1. (Feb., 1984), pp. 70-79.

Appendix A: 81 cancer sites and number of drugs

Code	Cancer Site Name	# of white persons born in the 48 states between 1901 and 1925 in our sample	Number of drugs (active ingredients)	Number of drugs approved 1973-1993 (active ingredients)
20010	Lip	2,270	12	7
20020	Tongue	3,728	12	7
20030	Salivary gland	1,270	12	7
20040	Floor of mouth	2,282	12	7
20050	Gum & other mouth	3,204	12	7
20060	Nasopharynx	624	12	7
20070	Tonsil	1,813	12	7
20080	Oropharynx	576	12	7
20090	Hypopharynx	2,093	12	7
20100	Other buccal cavity and pharynx	734	12	7
21010	Esophagus	6,765	0	0
21020	Stomach	13,397	1	1
21030	Small intestine	1,691	0	0
21041	Cecum	14,406	8	3
21042	Appendix	350	8	3
21043	Ascending colon	8,512	8	3
21044	Hepatic flexure	2,798	8	3
21045	Transverse colon	6,451	8	3
21046	Splenic flexure	2,488	8	3
21047	Descending colon	4,917	8	3
21048	Sigmoid colon	23,206	8	3
21049	Large intestine, NOS	2,993	8	3
21051	Rectosigmoid junction	9,649	8	3
21052	Rectum	18,013	8	3
21060	Anus, anal canal & anorectum	1,424	0	0
21071	Liver	3,640	0	0
21072	Intrahepatic bile duct	535	0	0
21080	Gallbladder	2,349	0	0
21090	Other biliary	2,312	6	4
21100	Pancreas	19,281	12	8
21110	Retroperitoneum	550	0	0
21120	Peritoneum, omentum & mesentery	369	0	0
21130	Other digestive organs	524	0	0
22010	Nasal cavity, middle ear & accessory sinuses	996	0	0
22020	Larynx	8,529	4	1
22030	Lung and bronchus	113,844	23	10
22050	Pleura	1,586	0	0
22060	Trachea, mediastinum & other respiratory organs	329	0	0
23000	Bones & joints	585	11	6
24000	Soft tissue (including heart)	2,143	20	5

Appendix A (continued): Cancer sites and number of drugs

Code	Cancer Site Name	# of white persons born in the 48 states between 1901 and 1925 in our sample	Number of drugs (active ingredients)	Number of drugs approved 1973-1993 (active ingredients)
25010	Melanomas-skin	9,760	22	8
25020	Other non-epithelial skin	902	1	1
26000	Breast	77,573	48	10
27010	Cervix	6,608	12	5
27020	Corpus	22,993	1	0
27030	Uterus, NOS	324	8	3
27040	Ovary	12,375	25	7
27050	Vagina	719	0	0
27060	Vulva	1,996	1	1
27070	Other female genital organs	560	0	0
28010	Prostate	74,951	34	10
28020	Testis	295	14	7
28030	Penis	690	2	1
28040	Other male genital organs	162	0	0
29010	Bladder	30,684	16	4
29020	Kidney and Renal pelvis	13,013	15	4
29030	Ureter	1,409	0	0
29040	Other urinary organs	680	0	0
30000	Eye & orbut	1,029	1	0
31010	Brain	7,864	8	3
31040	Other nervous system	273	8	3
32010	Thyroid	2,608	7	2
32020	Other endocrine (include. Thymus)	474	13	6
33011	Hodgkin's Disease-Nodal	1,664	27	6
33012	Extranodal	44	2	0
33041	Non- Hodgkin's Lymphomas--Nodal	14,916	38	9
33042	Extranodal	4,420	40	9
34000	Multiple myeloma	7,921	20	3
35011	Acute lymphocytic leukimia	442	22	2
35012	Chronic lymphocytic	6,158	20	2
35013	Other lymphocytic	231	14	1
35021	Acute granulocytic	4,024	15	4
35022	Chronic granulocytic	2,128	15	2
35023	Other granulocytic	450	4	1
35031	Acute monocytic Leukemia	299	3	0
35032	Chronic monocytic leukimia	30	1	0
35033	Other monocytic leukimia	40	1	0
35041	Other acute leukimia	933	3	1
35042	Other chronic	56	2	0
35043	Aleukemic, subacute, and NOS	1,174	12	2
37000	Ill defined and unspecified sites	19,860	22	9

Appendix B: Mortality cause of death diseases and number of drugs

Recode	Disease name	Death rate in sample (unweighted)	Number of active ingredients approved
10	Shigellosis and amebiases	0.00016	12
20	Other Intestinal infections	0.00020	24
40	Tuberculosis of respiratory system	0.00026	24
50	Other tuberculosis	0.00011	6
60	Whooping Cough	0.00003	2
70	Streptococca; sore throat, scarlatina and erysipelas	0.00011	21
80	Meningococcal infection	0.00008	8
90	Septicemia	0.00162	55
100	Acute poliomyelitis	0.00005	0
110	Measles	0.00005	0
120	Viral Hepatitis	0.00018	7
130	Syphillis	0.00014	16
140	All other infections	0.00038	919
160	Neoplasms-lip, oral cavity and pharynx	0.00061	
170	Neoplasms-digestive system	0.00752	37
180	Neoplasm-respiratory system	0.00829	33
190	Neoplasms-breast	0.00284	30
200	Neoplasms-genital organs	0.00455	65
210	Neoplasms-urinary organs	0.00165	30
220	Neoplasms-unspecified site	0.00339	99
230	Leukemia	0.00125	63
240	Other malignant neoplasms of lymphatic tissues	0.00178	118
250	Begin neoplasms	0.00055	92
260	Diabetes	0.00441	10
270	Nutritional deficiencies	0.00048	31
280	Anemias	0.00044	55
290	Meningitis	0.00012	45
320	Rheumatic fever and rheumatic heart disease	0.00068	30
330	Hypertensive heart disease	0.00161	0
340	Hypertensive heart and renal disease	0.00032	0
360	Acute myocardial infarction	0.02587	32
370	Other forms of ischemic heart disease	0.00037	2
380	Angina pectoris	0.00504	49
390	Old myocardial infection, chronic heart disease	0.02484	6
400	Other diseases of endocardium	0.00162	10
410	All other forms of heart disease	0.01338	227
420	Hypertension with or without renal disease	0.00076	129
440	Intracerebral and other intracranial hemorrhage	0.00158	0
450	Cerebral Thrombosis	0.00172	16
460	Cerebral embolism	0.00020	1
470	All other late effects of cerebrovascular disease	0.00857	15
480	Atheosclerosis	0.00181	7

Appendix B continued: Mortality cause of death diseases and number of drugs

Recode	Disease name	Death rate in sample (unweighted)	Number of active ingredients approved
490	Other disease of arteries, arterioles and capillaries	0.00208	66
500	Acute bronchitis and bronchiolitis	0.00023	2
520	Pneumonia	0.00576	283
530	Influenza	0.00028	5
550	Bronchitis, chronic and unspecified	0.00053	102
560	Emphysema	0.00172	21
570	Asthma	0.00035	99
580	other chronic obstructive pulmonary diseases	0.00561	27
590	Ulcer of stomach and duodenum	0.00065	83
600	Apendicitis	0.00013	0
610	Hernia	0.00062	9
620	Chronic liver disease and cirrhosis	0.00134	12
630	Cholelithiasis and other disorders of the gallbladder	0.00038	6
650	Acute glomerulonephritis and nephrotic syndrome	0.00012	16
660	Chronic glomerulonephritis and nephrotic syndrome	0.00028	4
670	Renal failure	0.00194	10
680	Infections of the kidney	0.00028	5
690	Hyperplasia of prostate	0.00036	7

Appendix C: Sources and Bibliography of data

I-Sources

Compulsory Schooling Laws: Multiple sources. For a detailed description of these laws and to find the sources from which they were compiled, please see Lleras-Muney, 2002a.

Mean education: average education levels in cohort, gender, and county. This measure of education can be calculated from the census Summary Tape Files from 1970, 1980 and 1990. We match individuals to education by decade, i.e. individuals diagnosed in the 1970s are matched to the average education in their cohort, gender, and county, calculated from the 1970 census.

Effect of compulsory schooling on education: Ipums data: 1960 General Sample, 1970 Form 2 State Sample, 1980 1% Metro Sample and 1990 1% Metro Sample.

Log of per capita personal income: is available for each county every year starting from 1975 onwards. Source: Bureau Of Health Professions Area Resource File

Mortality rates: for all whites by county and year. There are no missing values for this variable. Source: Compress Mortality Files (CMF).

The number of MD specialists: only available for years 85, 90 and 94, so we imputed the missing values within county and year. Source: Bureau Of Health Professions Area Resource File

Prices of Medical Care: we use state level data by year. This variable contains the average medical expenses per Medicare Beneficiary (excluding home health care). Source: Centers for Medicare and Medicaid Services.

II-Data Bibliography

National Center for Health Statistics. Data File Documentations, Mortality Data, Multiple Cause-of-Death Public Use Data Files, (multiple years) (machine readable data file and documentation, CD-ROM Series), National Center for Health Statistics, Hyattsville, Maryland.

National Center for Health Statistics (1968-1988). Data File Documentations, Compressed Mortality Files, 2000 (machine readable data file and documentation, CD-ROM Series, Series Number 20. No. 2A), National Center for Health Statistics, Hyattsville, Maryland.

Steven Ruggles and Matthew Sobek et al. Integrated Public Use Microdata Series: Version 3.0 Minneapolis: Historical Census Projects, University of Minnesota, 2003. web site: http://www.ipums.org.

Surveillance, Epidemiology, and End Results (SEER) Program (www.seer.cancer.gov) Public-Use Data (1973-1998), National Cancer Institute, DCCPS, Surveillance Research Program, Cancer Statistics Branch, released April 2003,based on the November 2000 submission.

U.S. Dept. of Commerce, Bureau of the Census. CENSUS OF POPULATION AND HOUSING, 1970 [UNITED STATES]: SUMMARY STATISTIC FILE 4A -- HOUSING [FOURTH COUNT] [Computer file]. ICPSR version. Washington, DC: U.S. Dept. of Commerce, Bureau of the Census [producer], 197?. Ann Arbor, MI: Inter-university Consortium for Political and Social Research [distributor], 2001.

U.S. Dept. of Commerce, Bureau of the Census. CENSUS OF POPULATION AND HOUSING, 1980 [UNITED STATES]: SUMMARY TAPE FILE 3A [Computer file]. ICPSR version. Washington, DC: U.S. Dept. of Commerce, Bureau of the Census [producer], 1982. Ann Arbor, MI: Inter-university Consortium for Political and Social Research [distributor], 1999

.S. Dept. of Commerce, Bureau of the Census. CENSUS OF POPULATION ND HOUSING, 1990 [UNITED STATES]: SUMMARY TAPE FILE 3A [PUERTO RICO] [Computer file]. Washington, DC: U.S. Dept. of Commerce, Bureau of the Census [producer], 1993. Ann Arbor, MI: Inter-university Consortium for Political and Social Research [distributor], 1999.

U.S. Dept. of Health and Human Services, Health Resources and Services Administration, Bureau of Health Professions. BUREAU OF HEALTH PROFESSIONS AREA RESOURCE FILE, 1940-1994: [UNITED STATES] [Computer file]. Rockville, MD: U.S. Dept. of Health and Human Services, Office of Data Analysis and Management [producer], 1995. Ann Arbor, MI: Inter-university Consortium for Political and Social Research [distributor], 1995.

Appendix D: Robustness checks in the SEER

Compulsory school*number of drugs	All	Male	Female
Basic controls(*)	-52***	-65***	-65
(*10^5)	(20)	(4.2)	(42)
Add site dummies and site*stage	-2.5	-14**	0.25
dummies (*10^5)	(7.8)	(6.1)	(4.9)
Add education and log income	-2.8	-14**	0.20
(*10^5)	(7.5)	(6.1)	(4.9)
Add County of residence dummies	-2.7	-14**	-0.042
(*10^5)	(7.6)	(6.1)	(5.1)

Standard errors (in parenthesis) clustered by site.
(*) Basic controls include married, Hispanic, diagnosis year, age, and age squared, cohort dummies, state-of-birth dummies, and stage dummies.

Table I: SEER Summary Statistics

Variable	Obs	Mean	Std. Dev.	Min	Max
Years of compulsory school	625958	6.939	1.065	0	10
Mean education in cohort, gender and registry	625958	10.956	1.026	4.944	14.500
Per capita personal income in county and year	625958	9.494	0.470	7.696	10.553
Female=1	625958	0.468	0.499	0	1
Age at Diagnosis	625958	69.397	7.991	47	92
Hispanic=1	625958	0.020	0.142	0	1
Married=1	625958	0.640	0.480	0	1
Died within 5 year of diagnosis=1	625958	0.634	0.482	0	1
Year of diagnosis	625958	11.747	5.726	1	21
Number of drugs approved by FDA by site	625958	21.17	14.39	0	48
Cancer Site (Broad categories)					
Bones and joints	625958	0.001	0.031	0	1
Brain and other nervous system	625958	0.124	0.329	0	1
Breast	625958	0.234	0.424	0	1
Digestive system	625958	0.005	0.070	0	1
Endocrine system	625958	0.002	0.041	0	1
Eye and orbit	625958	0.194	0.396	0	1
Genital system	625958	0.026	0.158	0	1
Leukemia	625958	0.034	0.180	0	1
Lymphomas	625958	0.030	0.170	0	1
Buccal cavity and pharynx	625958	0.013	0.112	0	1
Multiple Myeloma	625958	0.013	0.113	0	1
Ill-defined and unspecified sites	625958	0.032	0.175	0	1
Respiratory system	625958	0.200	0.400	0	1
Skin	625958	0.017	0.129	0	1
Soft tissue	625958	0.003	0.058	0	1
Urinary system	625958	0.073	0.260	0	1

TABLE II: Mortality Cause of Death files
Summary statistics

	Obs	Mean	Std. Dev.	Min	Max
4 year mortality rate	264218	0.0033	0.0081	0.0000	0.2286
Years of Compulsory schooling	264218	6.6346	1.3529	0	10
Year of death	264218	1978.267	11.9623	1960	1990
Number of drugs	264218	65.26	136.61	0	919
Age	264218	65.6147	13.6298	35	89
Broad causes of death					
Infectious diseases	264218	0.072	0.259	0	1
Cancer	264218	0.241	0.428	0	1
Diabetes	264218	0.027	0.162	0	1
Cardiovascular Diseases	264218	0.324	0.468	0	1
Respiratory diseases	264218	0.126	0.332	0	1
Digestive diseases	264218	0.094	0.291	0	1
Genito-Urinary diseases	264218	0.061	0.240	0	1

Death rates calculated at the gender, year, state-of-birth, year of birth level for each disease (broad recode-see Appendix). Sample of whites born between 1901 and 1925 in the 48 states.

Table III: The effect of education on the probability of dying

SEER data and Mortality cause of death data

	Effect of compulsory school on the probability of dying in 4/5 years	Effect of compulsory school on education 1960/70/80/90 Census	TSIV Effect of education on the probability of dying in 4/5 years[3]	Effect of one more year of compulsory school on mortality at the mean
Panel A: SEER[1]				Mean mortality: 0.634
All	-0.002*** (6.9e-04)	0.0463*** (0.0077)	-0.0432** (0.0165)	7%
Males	-0.002** (9.4e-04)	0.0404* (0.0088)	-0.0495* (0.0256)	
Females	-0.003*** (0.001)	0.0529*** (0.0092)	-0.0189*** (0.0033)	
Panel B: Mortality[2]				Mean mortality: 0.0033
All	-5.5e-05*** (1.1e-05)	0.0463*** (0.0077)	-0.0012*** (0.0003)	36%
Males	-8.2e-05*** (1.66e-05)	0.0404* (0.0088)	-0.0020** (0.0006)	
Females	-3.11e-05*** (1.3.e-05)	0.0529*** (0.0092)	-0.00059*** (0.00004)	

Notes: Standard errors in parentheses.

(1) Regressions include age at diagnosis, age at diagnosis squared, 47 state of birth dummies, 24 cohort dummies, 80 cancer site dummies, county of residence dummies, stage of cancer at diagnosis dummies, and site*stage interaction dummies. Sample consists of whites born in the 48 states between 1901 and 1925 for whom education could be imputed.

(2) Sample consists of whites born in the 48 states between 1901 and 1925 for whom education could be imputed.

(3) Standard errors for the TSIV estimates were calculated using the Delta method.

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

Table IV: Is the Effect of education on mortality larger for diseases with more progress?

SEER data and Mortality cause of death data

	Compulsory school * number of drugs	Compulsory school
Panel A: SEER[1]		
All	-2.7	-205.67
($*10^5$)	(7.6)	(193.5)
Males	-14**	91.71
($*10^5$)	(6.1)	(167.5)
Females	-0.042	-318.46
($*10^5$)	(5.1)	(197.72)
Panel B: Mortality[2]		
All	-4.46***	-264**
($*10^7$)	(0.060)	(116)
Males	-4.59***	-523***
($*10^7$)	(0.923)	(177)
Females	-4.22***	-39.3
($*10^7$)	(0.709)	(135)

Notes: Standard errors (in parentheses) are clustered at the disease level.

(1) Regressions include age at diagnosis, age at diagnosis squared, 47 state of birth dummies, 24 cohort dummies, 80 cancer site dummies, county of residence dummies, stage of cancer at diagnosis dummies, and site*stage interaction dummies. Sample consists of whites born in the 48 states between 1901 and 1925 for whom education could be imputed.

(2) Sample consists of whites born in the 48 states between 1901 and 1925 for whom education could be imputed.

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

Table V: Is the Effect of education on mortality larger for diseases with more RECENT progress?

Mortality cause of death data

	All	Males	Females
Compulsory school ($*10^5$)	-0.16	-0.405**	0.0552
	(0.1240)	(0.1900)	(0.1450)
Compulsory school*number of drugs in decade ($*10^5$)	-0.0212**	-0.0198*	-0.0234**
	(0.0074)	(0.0114)	(0.0086)
Compulsory school*number of drugs in past decade ($*10^5$)	-0.0627***	-0.06***	-0.0655***
	(0.0071)	(0.0109)	(0.0082)
Compulsory school*number of drugs two decades ago ($*10^5$)	0.0865***	0.0743***	0.101***
	(0.0144)	(0.0219)	(0.0168)

Notes: Standard errors (in parentheses) are clustered at the disease level. Sample consists of whites born in the 48 states between 1901 and 1925 for whom education could be imputed.

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

Table VI: Individual education versus geographic disparities
SEER cancer data

	All	Males	Females
Baseline: Compulsory school*number of drugs	-2.7e-05	-1.4e-04**	-4.2e-05
	(7.6e-05)	(6.1e-05)	(5.1e-05)
Panel A: Add county education and income(*)			
Compulsory school*number of drugs	-2.9e-05	-1.5e-04***	-8.9e-07
	(7.6e-05)	(6.0e-05)	(5.0e-05)
Mean education in registry, gender, cohort	-0.003	-9.4e-04	-2.4e-04
	(0.002)	(0.002)	(0.002)
Log of per capita personal income	-0.058***	-0.042**	-0.080***
	(0.023)	(0.021)	(0.025)
Panel B: cardiovascular mortality(*)			
Compulsory school*number of drugs	-0.00027	-0.00065**	-0.000051
	(0.00021)	(0.00026)	(0.000180)
Cardiovascular white mortality rate in county (CMR)	-3.58	27.11*	-29.797**
	(11.14)	(15.87)	(15.205)
CMR*compulsory school	1.15	-2.25	3.39
	(1.43)	(2.12)	(2.08)
CMR*number of drugs	0.225	-0.661	0.804**
	(0.338)	(0.514)	(0.385)
Compulsory school*CMR*number of drugs	0.013	0.164**	-0.0812
	(0.056)	(0.075)	(0.0545)
Panel C: number of specialists(*)			
Compulsory school*number of drugs	-2.3e-04***	-3.9e-04***	-1.3e-04
	(9.8e-05)	(1.2e-04)	(1.1e-04)
# of MD specialists in county, year	0.016***	0.023***	0.008
	(0.004)	(0.004)	(0.005)
# of specialists*compulsory school	-0.001**	-0.001	-0.001*
	(5.0e-04)	(6.9e-04)	(5.8e-04)
# of specialists *number of drugs	-2.7e-04*	-3.3e-04**	-1.7e-04
	(1.4e-04)	(1.6e-04)	(1.4e-04)
Compulsory school*# of specialists *number of drugs	3.8e-05**	4.7e-05*	2.3e-05
	(1.7e-05)	(2.4e-05)	(1.9e-05)

Notes: Standard errors (in parentheses) are clustered at the disease level. Regressions include age at diagnosis, age at diagnosis squared, 47 state of birth dummies, 24 cohort dummies, 80 cancer site dummies, county of residence dummies, stage of cancer at diagnosis dummies, and site*stage interaction dummies. Sample consists of whites born in the 48 states between 1901 and 1925 for whom education could be imputed. (*) See data Appendix for the sources of these data

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

Table VII: Is the education of education operating only through income?

SEER cancer data

	All	Males	Females
Baseline	-2.7e-05 (7.6e-05)	-1.4e-04** (6.1e-05)	-4.2e-05 (5.1e-05)
Panel A: Medicare versus non Medicare population			
Medicare (ages 65 and above) Compulsory school* drugs	-7.8e-05 (7.8e-05)	-1.9e-04*** (5.6e-05)	-2.6e-05 (3.5e-05)
Non-Medicare (age below 65) Compulsory school* drugs	9.2e-06 (6.6e-05)	1.8e-04 (2.2e-04)	-7.0e-05 (5.6e-05)
Panel B: prices of medical care Compulsory school*number of drugs	-1.1e-05 (1.9e-04)	-4.8e-04*** (1.9e-04)	2.1e-04** (1.0e-04)
Medicare Expenditure (ME)	-1.3e-05 (1.9e-05)	6.2e-06 (2.8e-05)	-2.0e-05 (2.0e-05)
ME*compulsory school	2.4e-06 (1.7e-06)	-8.1e-07 (2.1e-06)	3.9e-06* (2.2e-06)
ME *number of drugs	1.5e-07 (5.8e-07)	-1.2e-06* (6.9e-07)	8.8e-07** (4.2e-07)
Compulsory school*ME *number of drugs	-3.1e-08 (5.7e-08)	1.3e-07* (7.5e-08)	-1.2e-07*** (4.2e-08)

Notes: Standard errors (in parentheses) are clustered at the disease level. Regressions include average education and per capita personal income ion county of residence, age at diagnosis, age at diagnosis squared, 47 state of birth dummies, 24 cohort dummies, 80 cancer site dummies, county of residence dummies, stage of cancer at diagnosis dummies, and site*stage interaction dummies. Sample consists of whites born in the 48 states between 1901 and 1925 for whom education could be imputed.

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