

Health Inequality, Education and Medical Innovation *

Sherry Glied
Department of Health Policy and Management
Mailman School of Public Health

Adriana Lleras-Muney
Department of Economics
Princeton University

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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.

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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 with high educational attainment declined from 5.7 to 2.8 per 1000, while the rate declined only from 9 to 7.6 for those with low educational attainment (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 economics 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 research in demography that argues that differential knowledge and diffusion of knowledge were at the root of differences in infant mortality rates in the early XX century US (Preston and Haines, 1991); as well as 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 recently approved to treat a disease. We use the Mortality Detail Files and Surveillance, Epidemiology, and End Result (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 a proxy for 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.

Why would Education gradients be related 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

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

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.

There are several mechanisms through which the relationship between education and health could be 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 a high school degree or less.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 47% of high school graduates and 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. As an example, when analyzing the effects of the 1964 Surgeon General's Report, Meara (2001) concludes "the response to knowledge plays a more important role than knowledge itself in creating differential health behavior."

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.⁴ Consistent with this reasoning,

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

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

³ *ibid.*

⁴ Bradley (1991) reviews the literature on prescription behavior by physicians. He finds that there is wide variation in the prescription behavior of doctors, and in the rate at which doctors start prescribing new drugs.

researchers have found that people of higher socioeconomic status, such as the more educated, are more likely to participate in clinical trials, where they would gain access to the newest treatments (Sateren et al 2002). If this is the mechanism through which innovation affects education gradients, we would expect to see less effect of innovation on the gradient when the general quality of providers is greater. We test this mechanism below.

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. The same may also be true for some health innovations. New innovations may be simpler or more complex than those they replace at introduction, which would lead to shifts in the gradient. Over time, these technologies (which may vary in their complexity at introduction) tend to become simpler and more refined and, hence, more accessible to less educated people. In 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). In our context, this mechanism suggests that the effects of innovation on the gradient will be greatest soon after the introduction of an innovation.

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 (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

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

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. 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 directly 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:

$$(2) \quad \frac{\partial H}{\partial e} = -\lambda w'(e)A \frac{\partial H}{\partial A} + \frac{\partial H}{\partial E} > 0$$

$$\frac{\partial H}{\partial e \partial \lambda} = -w'(e) \left[A \frac{\partial H}{\partial A} + \lambda \frac{\partial A}{\partial \lambda} \frac{\partial H}{\partial A} + \lambda \frac{\partial H}{\partial A \partial \lambda} \right] > 0$$

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.⁵ 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 .

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:

$$(3) \quad P(\text{died} = 1) = \beta_0 + \beta_1 \text{education} + \beta_2 \text{education} * \text{progress} + \beta_3 \text{progress} + X\gamma + e .$$

In this specification, X includes disease fixed effects, and other individual characteristics. We expect that β_1 will be negative, reflecting the steady-state survival advantage of the more educated and that β_3 will be negative, since progress improves survival. Our discussion further suggests that 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.

Mortality 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.

⁵ The second derivative above assumes that H is additively separable, and that changes in the rate of innovation only affect the marginal productivity of education through shorter adoption lags.

Neither the SEER data nor the Mortality data include information on educational status. Instead, we use compulsory schooling laws to measure educational attainment. Several papers have shown that these laws had an impact on educational attainment, more recently Moretti and Lochner (2003).⁶ These laws specified the minimum number of years that a child had to attend school. The implicit number of compulsory years of schooling ranged from 0 to 10 for the cohorts we study.⁷

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. 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 specific counties located in San Francisco, Connecticut, Detroit, Hawaii, Iowa, New Mexico, Seattle, Utah and Atlanta⁸). The National Cancer Institute suggests this is a nationally representative sample of patients.⁹ Even if individuals leave these counties, they are followed by the registry to establish their status as of the last year of the data, in our data up to 1998. Information on vital status was recorded for all individuals in the sample as of 1998. These data allow us to look at 5-year

⁶ Also see Acemoglu and Angrist (1999), Angrist and Krueger (1991), Lleras-Muney (2002b), Margo and Finnegan, (1996) and Schmidt (1996), Goldin and Katz (2003).

⁷ 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.

⁸ 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.

⁹ This is how the National Cancer Institute describes a registry on their web site “Geographic areas were selected for inclusion in the SEER Program based on their ability to operate and maintain a high quality population-based cancer reporting system and for their epidemiologically significant population subgroups. The population covered by [SEER is comparable to the general US population](#) with regard to measures of poverty and education. The SEER population tends to be somewhat more urban and has a higher proportion of foreign-born persons than the general US population.”

mortality, conditional on cancer diagnosis from 1973 to 1993.¹⁰ We exclude individuals that died from external causes. 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 70.¹¹ 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 rate by matching mortality counts in the Mortality Detail files with 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 (state of birth is not available in the death certificates from 1963 until 1979. We are therefore limited to looking at 4 years death rates—rather than the standard 5, and we also cannot obtain rates for the 1970s). 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

¹⁰ 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.

¹¹ The average age at diagnosis is around 62 in the full SEER data. Our sample is older but not much more.

conditional on diagnosis. The SEER provides individual level data, so it contains more control variables.

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.

Measure of progress

To estimate the main equation of interest, equation (3) 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. For notational simplicity, we refer to active ingredients as drugs below.

Drugs are a limited measure of innovation. Many drugs are subsequently used to treat diseases that they were not originally approved for and these innovations would not be captured in our data. Furthermore, non-drug related innovations are not captured at all (except to the extent that they occur concurrently with drug innovation) and 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).

For the all-cause mortality data, we match mortality rates with the number of drugs approved in the last five years (so, for example, 1960-63 mortality is matched to the number of drugs approved between 1955 and 1960). We also repeat these analyses using stocks of drugs.

In the cancer data we use the stock rather than the flow of drugs. Very few new cancer drugs are approved in any given year, and many of these are used for several diseases, so there is little variation in our data. This reflects the nature of innovation in cancer chemotherapy, which often occurs through the use of novel combinations of existing drugs or through new modes of

delivery of existing drugs.¹² The number of new combinations of existing drugs is likely to be correlated with the stock of available drugs.

The data on active ingredients, the condition(s)¹³ they were approved to treat, and their FDA 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 may have been invented prior to the creation of the FDA (1938), there are others for which we could not impute a date of approval. There are about 80,000 different drugs in the US today if we define drugs using the National Drug Code (NDC) assigned by the FDA. However we count only about 2,000 unique active ingredients. Although prescribed medicines can contain one or more active ingredients, more than $\frac{3}{4}$ of the drugs are single-ingredient drugs. We match the active ingredient data to our sample using ICD9 condition codes. 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.

We conduct two tests to assess the usefulness of drugs as a measure of progress. First, we examine the correlation between the stock of cancer drugs by cancer subtype and the change in the 5-year survival rate conditional on cancer diagnosis, a direct measure of cancer progress. The correlation between number of drugs for cancer treatment and increases in the 5-year survival by cancer subtype is positive, about 0.3.

Next, we examine the direct correlation between drugs and mortality in our data. For both cancer and all-cause mortality data we estimate the following equation:

$$(4) P(died = 1) = \lambda_0 + \lambda_1 progress + X\gamma + e$$

¹² http://www.cancer.org/docroot/CRI/content/CRI_2_6x_the_history_of_cancer_72.asp?sitearea=CRI.

¹³ Note that a single drug can be used to treat many conditions, and a condition can have many drugs to treat it.

Where X includes cohort dummies, state-of-birth dummies, gender, age, age squared, year and disease dummies (for cancer we also include marital status, and Hispanic, but we do not include site dummies given that there is a unique progress measure for each site). As expected, we find that the stock of drugs (cancer) and flow of drugs (all cause mortality) have a negative and significant effect on the probability of dying in both samples (see Appendix D).

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.¹⁴ 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, and dummies for 80 cancer site. Importantly we control for 4 stage of diagnosis dummies, and for each site interacted with stage at diagnosis.¹⁵ 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 all-cause mortality data. The mortality data is aggregated to cells defined by cause of death, gender, cohort and state-of-

¹⁴ This assumption is tested in Lleras-Muney (2003).

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.

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 of a year of schooling 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.¹⁶ 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 education 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%. 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 year 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 the gradients themselves), in the remainder of the paper we will present reduced form estimates of the effect of one more year of

¹⁵ 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.

¹⁶ This method was used by Dee and Evans (1999).

compulsory schooling on outcomes.¹⁷ We also compare this to gradients using actual education further below.

Two results are worth pointing out. In both samples, the effect of education on mortality is greater for men than for women. This is a commonly found result in the literature (for example see Elo and Preston, 1996). Secondly, 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.

Our results are consistent with Lleras-Muney and Lichtenberg (2002), who find that more educated individuals are more likely to use prescription drugs more recently approved by the FDA. In the estimates here, however, the number of drugs is used as a proxy measure to capture innovation more broadly, since drug innovation might be correlated with broader innovation for a disease, for example with better understanding of disease causes and prevention. Drug innovation may also expand the availability of novel therapeutic combinations of new and existing drugs.

The Effect of Progress on the Gradient in Mortality

We now 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.¹⁸ Panel A of Table IV reports the estimated coefficients for the interaction term, for the entire sample and by gender. Male coefficients are negative as predicted but not females. We suspect that this lack of robustness may be due to unmeasured characteristics of some cancers that are likely to be correlated with education and that may also be related to the efficacy of therapy (Kaufmann et al., 2003), in

¹⁷ 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.

¹⁸ Results using a non-linear probability model yield similar effects. See appendix X

particular cancers of the reproductive organs. The incidence and severity of cancers of the reproductive system is correlated with whether a woman has ever been pregnant and with her age at first birth (Constantino, et al., 1999; Riman et al., 2002). Age at first birth, in turn, is correlated with education (Martin, 2000). Therefore we repeat the analysis for females excluding cancers of the female reproductive and we find the expected result. We therefore restrict all following cancer regressions for women to non-reproductive organ cancers.

These results are quite robust to the inclusion of covariates (Appendix E): once cancer site dummies are included, adding other covariates, including stage of cancer dummies, has little impact on the magnitude and significance of the interaction estimates.

In panel B we report similar aggregate estimates using the Mortality files. We estimate the interactions using two specifications. First, we interact education with the number of drugs approved in the previous five years for the disease (we include the main effect of the number of drugs approved). All coefficients, irrespective of the specification, are negative and significant. The estimates of the interaction are quite similar for men and women.

Secondly, we estimate equation (3) using the total number of drugs approved by disease as of 1990 (not the flow) and interact it with education. We do this because date of approval is missing for several drugs and was sometimes imputed, but more importantly we want to test whether using the stock rather than the flow of drugs results in any bias, given that we are using stocks in our cancer estimations. We find that the interaction with total number of drugs is also negative and significant, and it is smaller as one might expect given that this is a noisier measure of innovation.

As a test of the robustness of these results, we examined the effect on the interaction between the gradient and progress if we randomly matching drugs to diseases (rather than matching drugs to the disease for which they are indicated). We randomly matched drugs to diseases 200 times in each set of data, re-estimated equation 3 for each random match, and

compared our correctly-matched estimate with these random matches. For the all-cause mortality we randomized the match only within years. The descriptive statistics for the estimated interactions are in Appendix F. As expected, in both the SEER and the mortality data, we cannot reject that the estimates from the random match is significantly different from 0. Additionally, the point estimate of correctly-matched interaction term (Table IV) is outside the 95th percent confidence interval of the simulated interactions.

As we discussed previously, drugs are not the only form of medical innovation. We repeated the estimation in table IV using increases in the 5-year survival rate as a measure of progress. Again we find that the interaction between compulsory education and the progress measure is negative and significant.¹⁹

Overall we find support for the hypothesis that the education gradient is steepest for those diseases where there has been the most progress.

Illustrative Cases

Our empirical results show a robust relationship between education gradients and rates of technological progress. We next document the relationship with respect to specific diseases, comparing cases where we know from alternative sources whether there has been progress or not.

There has been enormous progress in the treatment of cardiovascular disease since 1970. Cutler and McClellan (2001) estimate that the value of the benefits associated with new technologies in this area considerably outweigh treatment costs. By contrast, they find little improvement in cancer survival (although there has been progress in the treatment of some types of cancer). In Figures 1a and 1b, we compare changes in gradients (estimated from the all-cause mortality data) in cardiovascular disease mortality and cancer mortality against time and the number of new drugs available to treat these diseases. We find, in both cases, that the education

gradient tends to increase with the number of drugs available for the treatment of the disease. We also find that the education gradient in cardiovascular disease has increased much more rapidly over time than has the gradient in cancer, consistent with the greater progress in treatment of cardiovascular disease that has previously been documented.

Among cancers, progress has varied by subtype of disease. In particular, there has been very little progress in the treatment of pancreatic cancer. Even today, the overall 5-year survival rate is less than 1%, and most people with this disease die within one year.²⁰ By contrast, colon cancer is quite treatable, using a combination of surgery and chemotherapy. There has also been considerable innovation in the treatment of this disease over time. Most recently, in 1990, a large clinical trial documented substantial benefits from a new form of post-surgical chemotherapy that combined two existing agents.²¹ Note that this type of innovation is not captured by changes in the flow of drugs, but may be proxied by the stock of available drugs. Figures 2a and 2b show the education gradients in survival for men and women diagnosed with pancreatic and colon cancer. The graphs show little change in the gradient for pancreatic cancer (indeed, the gradient for women actually fell over time) and a steep increase in the education gradient in colon cancer, consistent with our hypothesis that gradients increase with technological progress.

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.

¹⁹ See table 11 in Glied and Lleras-Muney (2002) for these results.

²⁰ See www.nci.nih.gov/cancerinfo/pdq/adulttreatment

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:

$$(4) MR_t = \beta_0 + \beta_1 * CSL + \sum_{i=0}^n \beta_{2t-i} CSL * \# drugs_{t-i} + X\gamma + e$$

where CSL is the level of compulsory schooling. We estimate the interaction for the current drugs, and for t-1, the previous 5 years. The number of drugs approved between 1955 and 1960 is the proxy of current progress associated with four- year mortality rates in 1960 (1960-1963); approvals between 1950 and 1955 are considered lagged progress. 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} , and $\beta_{2(t-1)}$. The interaction effect for current drug approvals is negative and significant. Interactions associated with earlier approvals are generally smaller than the effect of the most recent drugs. This result suggests that the gradients associated with a particular innovation are largest immediately after the innovation is introduced.

Examining Alternative Mechanisms

We have documented a robust relationship between education and technology and also find that recent innovations increase the education gradient most. We interpret these results as providing some evidence to support our hypothesis that the more educated adopt newer technologies first.

We next examine alternative reasons that education might affect the gradient.

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;

²¹ See www.nci.nih.gov/cancerinfo/pdq/adulttreatment

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.

To distinguish these theories 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 highly significant as one might expect. 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.²² 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.

The geographic co-location hypothesis suggests that controlling for differences in the quality of local health care providers should erase the advantage of more educated people. A related hypothesis suggests that the mechanism by which more educated people take advantage of new health technologies is by searching for better health providers who will have access to such technologies. If part of the advantage of educated people comes from better search for high quality providers, then we expect that this advantage will be smaller when the overall quality of care is greater. We consider two proxies for the quality of care available in each county: the

quality of care for other diseases and the number of medical specialists. If county level quality of care, correlated with education differences, is the explanation of our education findings, inclusion of quality indicators (and of interactions between quality indicators and progress) should eliminate our estimated effect. If the mechanism by which more educated people obtain new technologies is search, however, our estimated baseline effect should remain after inclusion of quality indicators and the coefficient of a triple interaction between quality, education, and progress should be positive.

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 in 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 (lack of) quality of care. Although CV mortality also captures differences in health habits such as smoking, as implemented here, with controls for county and year dummies, this measure should also capture in CV mortality that are due to changes in the quality of care. We estimate a model where education (denoted CSL for compulsory school), drugs and CV mortality (denoted CVMR) are fully interacted:

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

The results are in Panel B, Table VI. We find that the effect of education interacted with drugs remains negative and significant for men.²³ We also find that indeed cancer mortality is higher in areas where CV mortality is higher (either because underlying health habits are worse or because the quality of care is worse). Contrary to our hypothesis, the triple interaction between education, drugs and CV mortality (a marker of low quality) is positive and significant. This suggests that the cancer survival advantage of the more educated in the presence of cancer

²² We don't have county identifiers for all our mortality cause of death files.

²³ In a model excluding the triple interaction term, it is also negative and significant for women.

specific progress is reduced when cardiovascular mortality is high. This may reflect poorer overall health status in these areas.

Next, in Panel C of Table VII, 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. The interaction between education and drugs remains negative and significant for men²⁴. Among 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. We also find that, for men, the triple interaction with specialists is positive. This suggests that one mechanism through which education improves health is more effective searching. When all providers are of high quality, skill in searching for a good provider may matter 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 income, not education, per se. This problem is difficult to solve since it is almost impossible to disentangle education and 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’ 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 who have relatively

uniform insurance and those under 65, whose insurance coverage tends to vary with income. Medicare patients face fewer financial barriers to cancer care since Medicare covers most related medical expenditures, *including most chemotherapy drugs*.²⁵ In table VII, we estimate the cancer mortality model for individuals over and under 65.²⁶ 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 interaction for those above 65. Coefficients for both age groups are negative for women, but neither is significant.

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. Chernew, Cutler, and Keenan show that expenditures are correlated with uninsurance in the population under 65. We estimate a model that includes medical expenditures, expenditures interacted with progress,²⁵ and expenditures interacted with education. We hypothesize that higher costs reduce access to care and hence survival, that they reduce the benefits of progress (by limiting access), and that they matter less to the more educated (and higher income). The results (Panel B, Table VII) indicate that the interaction between progress and education is still negative and significant for both men and women. We find no other significant effects of prices. Again, this finding is consistent with our hypothesis that the effect of education does not operate entirely through income.

²⁴ In a model excluding the triple interaction term, it is also negative and significant for women.

²⁵ 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>

²⁶ Since Medicare does not cover most other drugs, we do not perform this test with the all mortality data.

Conclusion

Studies of technological diffusion consistently point to education as a factor that increases the diffusion rate (Hall and Khan, 2003). 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 advantage of educated people in the presence of technological innovation. Education also appears to have a direct effect that enhances the benefits of innovation.

We find that the effect of a technology on the mortality gradient is greatest immediately after the introduction of a new technology, suggesting that technologies may eventually 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.

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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
<u>Cancer Site (Broad categories)</u>					
Bones and joints	625958	0.00	0.03	0	1
Brain and other nervous system	625958	0.01	0.11	0	1
Breast	625958	0.12	0.33	0	1
Digestive system	625958	0.23	0.42	0	1
Endocrine system	625958	0.00	0.07	0	1
Eye and orbit	625958	0.00	0.04	0	1
Genital system	625958	0.20	0.40	0	1
Leukemia	625958	0.03	0.16	0	1
Lymphomas	625958	0.03	0.18	0	1
Buccal cavity and pharynx	625958	0.03	0.17	0	1
Multiple Myeloma	625958	0.01	0.11	0	1
Ill-defined and unspecified sites	625958	0.03	0.17	0	1
Respiratory system	625958	0.20	0.40	0	1
Skin	625958	0.02	0.13	0	1
Soft tissue	625958	0.00	0.06	0	1
Urinary system	625958	0.07	0.26	0	1

TABLE II: Mortality Cause of Death files
Summary statistics

	Obs	Mean	Std. Dev.	Min	Max
4 year mortality rate	259206	0.0034	0.0082	0.0000	0.2286
Years of Compulsory schooling	259206	6.6319	1.3564	0	10
Year of death	259206	1978.323	11.9371	1960	1990
Number of drugs	259206	32.5775	40.7364	0	230
Age	259206	65.6985	13.5874	35	89
<u>Broad causes of death</u>					
Infectious diseases	259206	0.0735	0.2609	0	1
Cancer	259206	0.2459	0.4306	0	1
Diabetes	259206	0.0275	0.1634	0	1
Cardiovascular Diseases	259206	0.3298	0.4701	0	1
Respiratory diseases	259206	0.1285	0.3346	0	1
Digestive diseases	259206	0.0953	0.2937	0	1
Genito-Urinary diseases	259206	0.0627	0.2424	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 education on mortality at the mean
Panel A:				Mean mortality:
SEER⁽¹⁾				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)	-.0567 *** (0.0033)	
Panel B:				Mean mortality:
Mortality⁽²⁾				0.0033
All	-5.4e-05*** (1.1e-05)	0.0463*** (0.0077)	-0.0012*** (0.0003)	36%
Males	-8.0e-05*** (1.66e-05)	0.0404* (0.0088)	-0.0020** (0.0006)	
Females	-3.10e-05** (1.3.e-05)	0.0529*** (0.0092)	-0.00059*** (0.00004)	

Notes: Standard errors in parentheses. Standard errors clustered at the cancer site level for cancer regressions.

(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.

(2) Sample consists of whites born in the 48 states between 1901 and

(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 (*10 ⁵)	-2.7 (7.6)	-205.67 (193.5)
Males (*10 ⁵)	-14** (6.1)	91.71 (167.5)
Females (*10 ⁵)	-0.042 (5.1)	-318.46 (197.72)
Females, excluding cancers of the reproductive system (*10 ⁵)	-22.84** (11.33)	49.95 (206.74)
Panel B: Mortality⁽²⁾		
Use total number of drugs in existence approved in past 5 years (regressions include main effect)		
All	-2.2e-05*** (2.2e-06)	5.7e-06 (1.3e-05)
Males	-2.0e-05*** (3.3e-06)	-2.8e-05 (2.0e-05)
Females	-2.4e-05*** (2.6e-06)	3.6e-05** (1.6e-05)
Use total number of drugs in existence in 1990		
All	-3.2e-06*** (2.0e-07)	4.8e-05*** (1.3e-05)
Males	-3.5e-06*** (3.1e-07)	3.4e-05* (2.0e-05)
Females	-2.8e-06*** (2.4e-07)	5.9e-05*** (1.5e-05)

Notes: Standard errors in parentheses. Clustered at the cancer site level for cancer regressions.

(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.

(2) Sample consists of whites born in the 48 states between 1901 and 1925.

* 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
t= 1			
Compulsory school	5.7e-06 (1.3e-05)	-2.8e-05 (2.0e-05)	3.6e-05** (1.6e-05)
Compulsory school*number of drugs in past 5 years	-2.2e-05*** (2.2e-06)	-2.0e-05*** (3.3e-06)	-2.4e-05*** (2.6e-06)
t= 1, 2			
Compulsory school	1.6e-05 (1.4e-05)	-1.3e-05 (2.1e-05)	4.1e-05*** (1.6e-05)
Compulsory school*number of drugs in past 5 years	-1.9e-05*** (2.8e-06)	-1.5e-05*** (4.2e-06)	-2.2e-05*** (3.3e-06)
Compulsory school*number of drugs between 5 and 10 yeas ago	-6.8e-06** (3.1e-06)	-9.8e-06** (4.7e-06)	-3.8e-06 (3.6e-06)

Notes: Standard errors in parentheses. Sample consists of whites born in the 48 states between 1901 and 1925.

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

**Table VI: Individual education versus geographic disparities
SEER cancer data**

	Males	Females (excluding cancers of the reproductive system)
Baseline: Compulsory school*number of drugs	-1.4e-04** (6.1e-05)	-2.28e-04** (11.33e-05)
<u>Panel A: Add county education and income^(*)</u>		
Compulsory school*number of drugs	-1.5e-04*** (6.0e-05)	-0.0002** (0.0001)
Mean education in registry, gender, cohort	-9.4e-04 (0.002)	-0.0008 (0.0019)
Log of per capita personal income	-0.042** (0.021)	-0.092*** (0.032)
<u>Panel B: cardiovascular mortality^(*)</u>		
Compulsory school*number of drugs	-0.00065** (0.00026)	0.00033 (0.0007)
Cardiovascular white mortality rate in county and year (CMR)	27.11* (15.87)	-36.695 (21.017)
CMR*compulsory school	-2.25 (2.12)	5.828* (3.050)
CMR*number of drugs	-0.661 (0.514)	1.752 (1.509)
Compulsory school*CMR*number of drugs	0.164** (0.075)	-0.2653 (0.2164)
<u>Panel C: number of specialists^(*)</u>		
Compulsory school*number of drugs	-3.9e-04*** (1.2e-04)	-0.00002 (0.0003)
# of MD specialists in county, year	0.023*** (0.004)	-0.002 (0.007)
# of specialists*compulsory school	-0.001 (6.9e-04)	0.00029 (0.00086)
# of specialists *number of drugs	-3.3e-04** (1.6e-04)	0.00029 (0.00040)
Compulsory school*# of specialists *number of drugs	4.7e-05* (2.4e-05)	-0.00004 (0.00005)

Notes: Standard errors (in parentheses) are clustered at the cancer site 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.
(*) See data Appendix for the sources of these data . * Significant at 10%; ** significant at 5%; *** significant at 1%

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

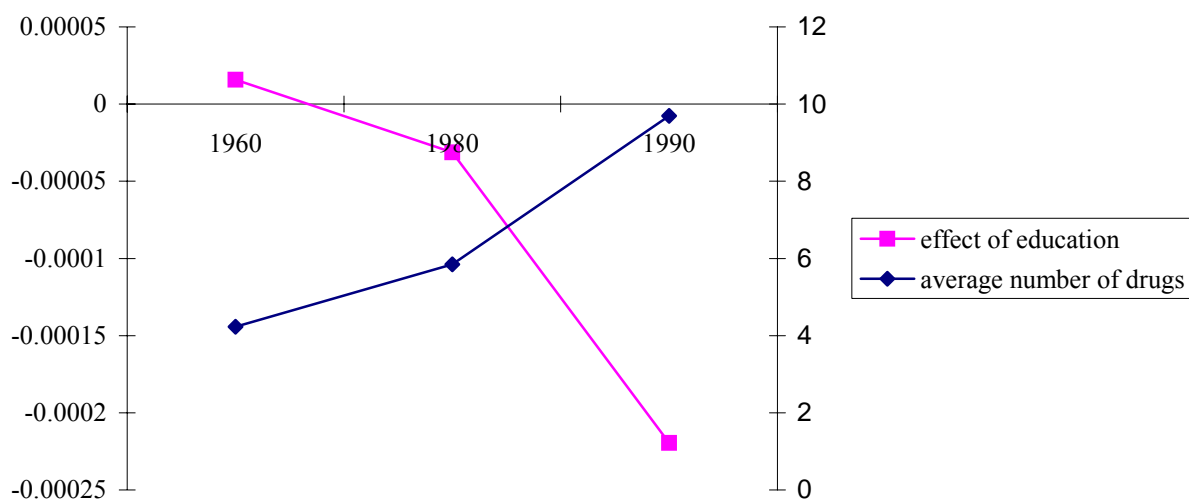
SEER cancer data

	Males	Females (excluding cancers of the reproductive system)
Baseline	-1.4e-04** (6.1e-05)	-2.3e-04** (1.1e-06)
Panel A: Medicare versus non Medicare population		
Medicare (ages 65 and above) Compulsory school* drugs	-1.9e-04*** (5.6e-05)	-1.7e-04 (1.1e-04)
Non-Medicare (age below 65) Compulsory school* drugs	1.8e-04 (2.2e-04)	-3.0e-04 (2.1e-04)
Panel B: prices of medical care		
Compulsory school*number of drugs	-1.7e-04** (7.8e-05)	-3.9e-04* (6.1e-05)
Medicare Expenditure (ME)	-1.2e-05 (2.6e-05)	9.5e-06 (1.9e-05)
ME*compulsory school	1.9e-06 (1.4e-06)	1.1e-06 (1.5e-06)
ME *number of drugs	-2.4e-07 (3.3e-07)	-4.8e-07 (5.5e-07)

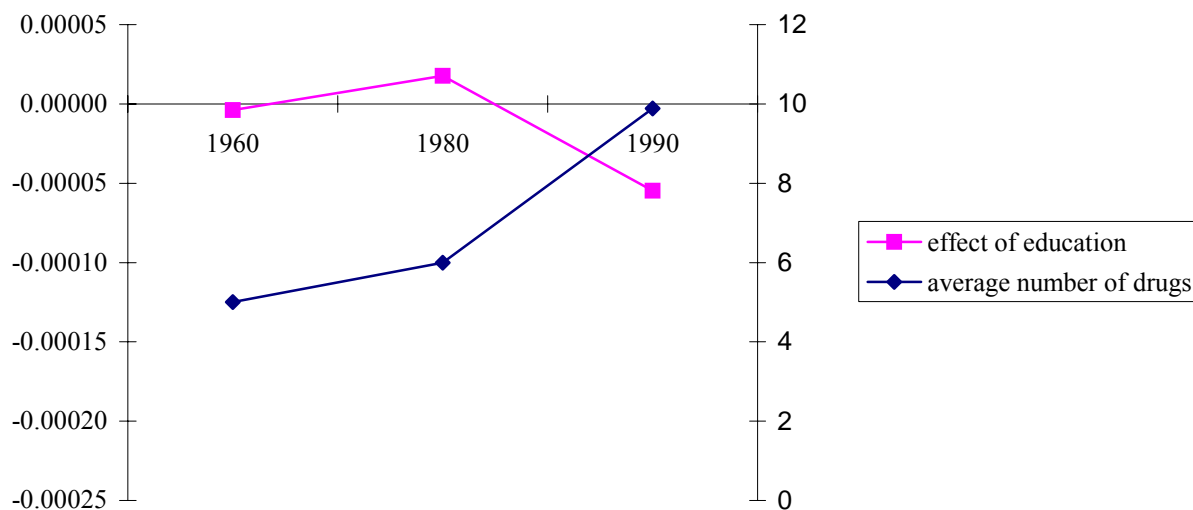
Notes: Standard errors (in parentheses) are clustered at the cancer site level. Regressions include average education and per capita personal income ion county of residence, age at diagnosis, age at diagnosis squared, 47 state of birth dummies, 24 cohort dummies, 80 cancer site dummies, county of residence 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.

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

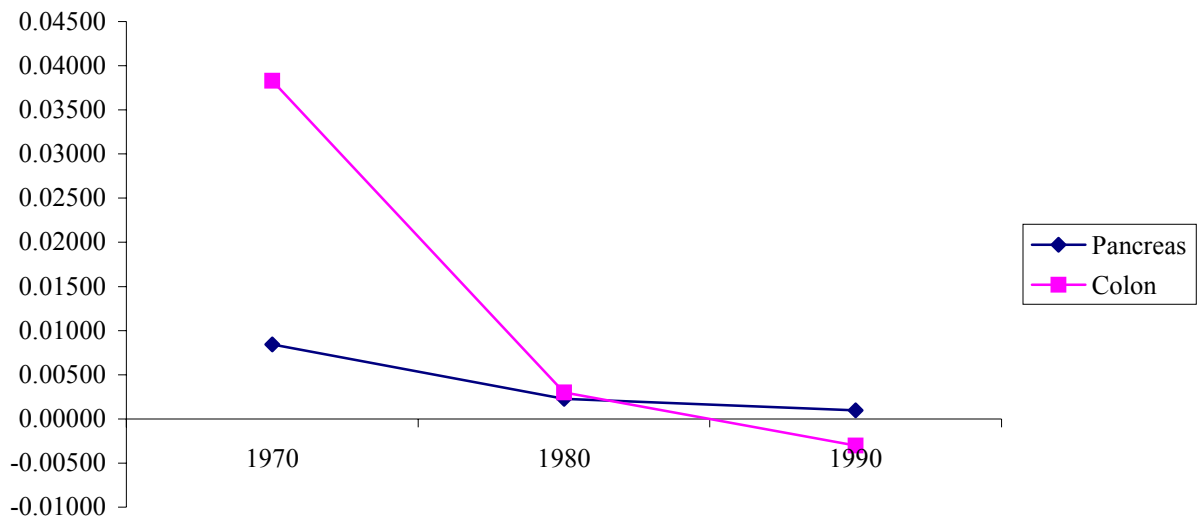
Graph 1a: Education gradients and number of drugs for Cardiovascular mortality, 1960-1990



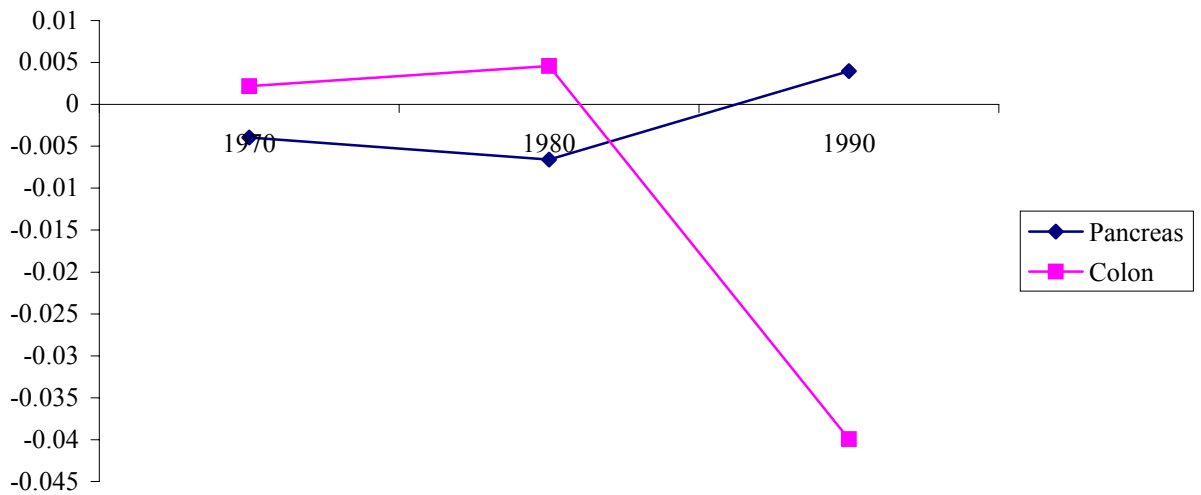
Graph 1b: Education gradients and number of drugs for Cancer mortality, 1960-1990



Graph 2a: Gradients in education overtime for men
in two types of cancer



Graph 2b: Gradients in education overtime for women
in two types of cancer



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 approved 1973-1993
			Number of drugs (active ingredients)	(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 & orbit	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 leukemia	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 leukemia	30	1	0
35033	Other monocytic leukemia	40	1	0
35041	Other acute leukemia	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 amebiasis	0.00016	7
20	Other Intestinal infections	0.00020	16
40	Tuberculosis of respiratory system	0.00026	23
50	Other tuberculosis	0.00011	6
60	Whooping Cough	0.00003	2
70	Streptococca, sore throat, scarlatina and erysipelas	0.00011	16
80	Meningococcal infection	0.00008	6
90	Septicemia	0.00162	50
100	Acute poliomyelitis	0.00005	0
110	Measles	0.00005	0
120	Viral Hepatitis	0.00018	5
130	Syphillis	0.00014	11
140	All other infections	0.00038	230
160	Neoplasms-lip, oral cavity and pharynx	0.00061	0
170	Neoplasms-digestive system	0.00752	21
180	Neoplasm-respiratory system	0.00829	27
190	Neoplasms-breast	0.00284	30
200	Neoplasms-genital organs	0.00455	51
210	Neoplasms-urinary organs	0.00165	22
220	Neoplasms-unspecified site	0.00339	58
230	Leukemia	0.00125	39
240	Other malignant neoplasms of lymphatic tissues	0.00178	51
250	Benign neoplasms	0.00055	65
260	Diabetes	0.00441	10
270	Nutritional deficiencies	0.00048	24
280	Anemias	0.00044	27
290	Meningitis	0.00012	28
320	Rheumatic fever and rheumatic heart disease	0.00068	20
330	Hypertensive heart disease	0.00161	27
340	Hypertensive heart and renal disease	0.00032	0
360	Acute myocardial infarction	0.02587	0
370	Other forms of ischemic heart disease	0.00037	2
380	Angina pectoris	0.00504	36
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	129
420	Hypertension with or without renal disease	0.00076	116
440	Intracerebral and other intracranial hemorrhage	0.00158	0
450	Cerebral Thrombosis	0.00172	6
460	Cerebral embolism	0.00020	1
470	All other late effects of cerebrovascular disease	0.00857	15
480	Atherosclerosis	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	42
500	Acute bronchitis and bronchiolitis	0.00023	1
520	Pneumonia	0.00576	71
530	Influenza	0.00028	5
550	Bronchitis, chronic and unspecified	0.00053	61
560	Emphysema	0.00172	21
570	Asthma	0.00035	47
580	other chronic obstructive pulmonary diseases	0.00561	24
590	Ulcer of stomach and duodenum	0.00065	39
600	Apendicitis	0.00013	1
610	Hernia	0.00062	2
620	Chronic liver disease and cirrhosis	0.00134	10
630	Cholelithiasis and other disorders of the gallbladder	0.00038	4
650	Acute glomerulonephritis and nephrotic syndrome	0.00012	16
660	Chronic glomerulonephritis and nephrotic syndrome	0.00028	4
670	Renal failure	0.00194	7
680	Infections of the kidney	0.00028	2
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.

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Appendix D: The Effect of Drug Innovation on Mortality

Dependent variable: Death

Coefficient on Number of Active Ingredients

Panel A: SEER⁽¹⁾

Use number of drugs in existence in 1990

Sample mean 21, s.d. 14

All	-0.00369*** (0.0014)
Males	-0.0045 (0.0029)
Females	-0.00310** (0.00138)

Panel B: Mortality⁽²⁾

Use number of drugs approved in past 5 years

Sample mean 2.7, s.d 4

All	-0.00037*** (7.72e-06)
Males	-0.0005378*** (0.0000117)
Females	-0.00021*** (010e-06)

Standard errors in parenthesis. Standard errors are clustered by site for cancer regressions.

(*) Basic controls include married, Hispanic, diagnosis year, age, and age squared, cohort dummies, state-of-birth dummies, and stage dummies.

Appendix E: Robustness checks in the SEER

Compulsory school*number of drugs	Male	Female
Basic controls ^(*) (*10 ⁵)	-65*** (4.2)	1.91 (47.45)
Add site dummies and site*stage dummies (*10 ⁵)	-14** (6.1)	-21.98* (11.2)
Add education and log income (*10 ⁵)	-14** (6.1)	-22.26* (11.3)
Add County of residence dummies (*10 ⁵)	-14** (6.1)	-23.1** (11.35)

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.

**Appendix F: Robustness checks—
Estimates for randomly matched drugs and disease**

	Mean	Standard Error	95% confidence interval	
<u>Cancer Data</u>				
Men				
True match: -0.00014				
Compulsory school*number of drugs	-5.73e-06	8.18e-06	-0.0000219	0.0000104
Women				
(excluding cancers of the reproductive system)				
True match: -0.00023				
Compulsory school*number of drugs	-0.0000146	0.000207	-0.0029281	1.75e-07
<u>All-cause mortality (full sample)</u>				
Compulsory school*number of drugs	1.48e-07	0.0000197	-0.0001219	0.0000405

Notes: 200 simulations were performed with each data set. Specifications are identical to those presented in table IV.