TECHNOLOGICAL INNOVATION AND INEQUALITY IN HEALTH*

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The effect of education on health has been increasing over the past several decades. We hypothesize that this increasing disparity is related to health-related technical progress: more-educated people are the first to take advantage of technological advances that improve health. We test this hypothesis using data on disease-specific mortality rates for 1980 and 1990, and cancer registry data for 1973–1993. We estimate education gradients in mortality using compulsory schooling as a measure of education. We then relate these gradients to two measures of health-related innovation: the number of active drug ingredients available to treat a disease, and the rate of change in mortality from that disease. We find that more-educated individuals have a greater survival advantage in those diseases for which there has been more health-related technological progress.

A series of recent studies has shown that improvements in health have contributed significantly to improvements in the standard of living over the past 50 years. Nordhaus (2002) found that the value of mortality declines in the United States in the twentieth century was roughly equivalent to the contemporaneous change in gross domestic product (GDP) per capita. Murphy and Topel (2003), in analyses computing the social value of research and development between 1970 and 1990, found enormous gains from the resulting longevity—worth as much as $2.8 trillion annually. This period has seen many innovations that have contributed to improvements in health, from better public water systems, to increased awareness of risk factors for many diseases, and to improvements in the quality of medical care. These technological changes, whether in the form of new knowledge or new products and services, have enabled people to live longer and better lives (Cutler, Deaton, and Lleras-Muney 2006).

Socioeconomic disparities in health have also been increasing over the past century (Elo and Preston 1996; Kunst et al. 2002; Pappas et al. 1993). In the United States, between 1960 and 1986, the age-adjusted mortality rate for white men with high educational attainment, defined as one or more years of college in 1960 and four or more years of college in 1986, declined from 5.7 to 2.8 per 1,000; the rate declined only from 9 to 7.6 for those with low educational attainment, defined as those with fewer than 8 years of schooling in 1960 and fewer than 11 years of schooling in 1986 (Pappas et al. 1993). Similar divergences have also been documented in the United Kingdom and in other European countries, including those with comprehensive universal health insurance programs (Kunst et al. 2002; Mackenbach et al. 2003).

The growing disparities in health outcomes by socioeconomic status have attracted considerable research and policy attention. Many researchers have sought to explain the existence of socioeconomic differences in health status at a point in time (see, e.g., Cutler and Meara 2001). In their landmark study, Kitagwa and Hauser (1973) found significant disparities in mortality by education, which varied by cause of death.

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We hypothesize that the robust rate of technological change affecting health and the increased gradients described above are related. Sociologists have conjectured that socioeconomic status is a "fundamental social cause" of gradients in health (Link et al. 1998). A fundamental cause 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, which is one component of socioeconomic status, enables people to better exploit new information and resources. Phelan et al. (2004) tested this hypothesis by comparing gradients in socioeconomic status at a point in time for diseases that are more or less preventable and found that gradients are greatest for the most preventable diseases. Phelan and Link (2005) examined changes in the gradient over time for selected diseases that have and have not become more preventable or treatable over time (heart disease, colon cancer, and lung cancer compared with brain and ovarian cancer). They documented that gradients have increased in those diseases that have become more preventable or treatable.

This paper extends this literature by looking at whether education gradients in health are related to measures of technological change across a broad spectrum of diseases. The hypothesis we test is that improvements in health technologies tend to increase disparities in health across education groups because education enhances the ability to exploit technological advances. The most-educated make the best initial use of this new information and adopt newer technologies first. For this reason, the gradient increases where and when technological change occurs.[1]

Our hypothesis is also an extension to health of existing economic models of technological change, particularly the theory of Nelson and Phelps (1966:72) that in the labor market, "the return to education is greater the faster the theoretical level of technology has been advancing." A substantial literature examines this pattern empirically in the labor market (see, e.g., Allen 2001; Bartel and Sicherman 1999) and in the agricultural sector (Wozniak 1984). Our hypothesis is further 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 twentieth-century United States (Preston and Haines 1991).

In this paper, we test the hypothesis that education gradients increase when innovation increases by relating education gradients in mortality to two measures of health-related innovation: the change in mortality for a given disease (which accounts for all types of health-related progress) and the number of active drug ingredients recently approved to treat a disease. We use the Mortality Detail Files (MDF) for 1980 and 1990 (National Center for Health Statistics, multiple years; 1968–1988) to examine mortality rates by disease, and the Surveillance Epidemiology and End Result (SEER) individual data to look at mortality subsequent to cancer diagnosis for different types of cancer.

We estimate the effect of education on disease-specific mortality and cancer mortality conditional on diagnosis, respectively. We then look at whether the effect of education becomes larger for diseases for which there have been greater innovations. To obtain plausibly causal effects of education, we match individuals with the compulsory schooling laws that were in place in their state of birth when they were growing up. Because we can control for both state of birth and cohort of birth, our effects are estimated using changes in legislation within states over time.

We find that more-educated individuals have a larger survival advantage from precisely those diseases that have seen more health-related innovation. We find robust evidence that observed gradients in socioeconomic status are, in part, a consequence of the relationship between education and technological improvement.

This paper makes two contributions to the literature. First, it provides a test of the idea that innovation creates health inequalities, using measures of technology across all diseases.

1. In the absence of innovation, gradients may exist for other reasons, such as stress, and could even be reversed.

Second, by using compulsory schooling legislation as a measure of socioeconomic status, this paper makes a compelling case that education itself—rather than other characteristics that may be correlated with education—confers an increased health advantage in the presence of innovation.

THE RELATIONSHIPS BETWEEN EDUCATION, MORTALITY, AND TECHNOLOGICAL PROGRESS

Although many studies have documented that a strong correlation between education and health appears to be causal (see Grossman 2000 for a summary), skepticism remains because the mechanisms through which the relationship might arise are unclear. We suggest that more education increases the rate of adoption of innovations. This idea is accepted in many contexts, yet it is not as obvious that education accelerates diffusion in the context of health.

There are several mechanisms through which the relationship between education and health could be mediated by technological innovation. As in other contexts of diffusion, more-educated people are better informed about health-related innovation (National Science Board 2000). Greater access to information and more-positive valuations of the benefits of innovations could lead the more educated to adopt newer innovations before the less educated. As an example, de Walque (2004) reported that in Uganda, there was no relationship between educational level and the incidence of AIDS in 1990; however, by 2000 and after several information campaigns on prevention, the more educated were more likely to use condoms and less likely to have AIDS. When analyzing the effects of the 1964 Surgeon General's Report, Meara (2001:27) concluded that "the response to knowledge plays a more important role than knowledge itself in creating differential health behavior."

Alternatively, more-educated people may be more adept at implementing new technologies (such as computers) in their early stages. Over time, technologies (which may vary in their complexity at introduction) tend to become simpler to use, and the best techniques for using them are developed. This process of adaptation may make the technology 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, the drugs diffused to other populations (Cunningham et al. 2000).

A third explanation, focusing on innovations in medical care, draws on the enormous variations in the practice patterns of medical professionals (e.g., see Chandra and Skinner 2003). More-educated people could have an advantage in the presence of innovation if they are more effective at searching for high-quality providers with access to newer technologies.[2] Researchers have indeed 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). Several studies have documented that educational attainment substantially increases the propensity to seek care from medical specialists, even after controlling for health status, and even in countries with national health insurance systems (Bongers et al. 1997; Van Doorslaer, Koolman, and Jones 2004).

Fourth, the correlation between education and technology adoption could operate directly or indirectly through income, which is another component of socioeconomic status. A higher level of education raises income, and income might provide better financial access to quality care. Those with higher incomes are also able to support better-endowed hospitals and more medical specialists. Unfortunately, our data do not allow us to examine any of these mechanisms directly and systematically.

2. For example, Bradley (1991) reviewed the literature on prescription behavior by physicians. He found wide variation in the prescription behavior of doctors and in the rate at which doctors start prescribing new drugs.

DATA

We use two sources of mortality data for this project: the Mortality Detail Files for 1980 and 1990 (MDF 80–90) and the SEER data for 1973–1993. Both these data sources include information on state of birth, which we need to assign individuals to the years of compulsory schooling they were subject to.

MDF 80–90

We calculate four-year mortality rates by disease by matching cause-specific mortality counts in the MDF files with population data from the census in 1980 and 1990. We construct death rates by cause of death (25 diseases, using the 34 causes-of-death classification and excluding deaths from maternal causes, congenital debilities, and external causes), gender, cohort, and state of birth for two periods: 1980–1983 and 1990–1993.[3] We match each cell in these data to the compulsory attendance and child labor laws in place in that state of birth in the year when the cohort was 14 years old.[4] We restrict our analyses to whites born between 1901 and 1925 in the 48 U.S. states (thus excluding foreign-born) because compulsory schooling laws were most effective in the first half of the twentieth century and because they affected only whites (see Lleras-Muney 2002).

Table 1 provides summary statistics for these data. There are 119,975 observations, each representing the mortality rate for a given cause (25), gender (2), cohort (25), state of birth (48), and year group (2). Note that the sample size is not exactly equal to 120,000 because of a few missing cells. The main causes of death in these data are cardiovascular disease, cancer, and respiratory diseases. The number of compulsory years of schooling ranged from 0 to 10 for the cohorts we study.[5]

SEER

We use the Surveillance Epidemiology and End Result (SEER) Cancer Incidence Public Use Database collected by the National Cancer Institute to examine the effect of progress on cancer mortality. Unlike the MDF, these data allow us to examine the effects of death conditional on incidence, at the individual level, using more covariates.

The SEER data contain information on *every* person diagnosed with cancer from 1973 to 1998 in nine SEER registries. (Registries are composed of specific counties located in San Francisco, CA; Connecticut; Detroit, MI; Hawaii; Iowa; New Mexico; Seattle, WA; Utah; and Atlanta, GA.) The National Cancer Institute suggests that these registries constitute a nationally representative sample of patients. Individuals who leave these counties are followed by the registry to establish their status as of the last year of the data—in our case, 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 the probability of dying within five years (the standard in epidemiology), conditional on cancer diagnosis, for individuals diagnosed with cancer between 1973 and 1993.[6] We exclude individuals who died from external causes and assume that individuals died from the cancer with which they were initially diagnosed.[7] The SEER data include information on state of birth and year of birth, so we can match SEER

3. We also conducted the analyses in the paper using a finer disease categorization (60 diseases based on the 72 cause-of-death recodes) and omitting any residual category. The results are qualitatively very similar to those presented in the current paper (results are available from the authors upon request).

4. There may be some misclassification error in the assignment of compulsory schooling attributable to family mobility, but prior research suggests that the effect of this error is likely to be small (Lleras-Muney 2002).

5. The data on compulsory attendance and child labor laws were collected from multiple sources (for details, see Lleras-Muney 2002). We use only two age-limit laws: the age at which a child had to enter school and the age at which a child 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. To avoid censoring, we drop individuals diagnosed after 1993.

7. Our results are not sensitive to using the actual cause of death instead of the diagnosed cancer.

Table 1. Mortality Cause-of-Death Files, 1980 and 1990 (MDF 80–90): Summary Statistics

Variable	Disease Recode	Mean	*SD*
Four-Year Cause-Specific Mortality Rate		0.0067	0.0148
Years of Compulsory Schooling		6.6185	1.3879
Year		1984.9	5
Age		71.9965	8.7725
25 Causes-of-Death Distribution (ICD-9 codes in parentheses)[a]			
Tuberculosis (010–018)	10	0.0001	0.0002
Syphilis (090–097)	20	0.0000	0.0001
Other infectious disease (001–009, 020–041, 042–044, 045–088, 098–139)	30	0.0020	0.0024
Digestive organs cancer (150–159)	50	0.0102	0.0057
Respiratory organs cancer (160–190)	60	0.0113	0.0074
Breast cancer (174–175)	70	0.0023	0.0026
Genital organs cancer (179–187)	80	0.0060	0.0071
Urinary organs cancer (188–189)	90	0.0020	0.0020
Leukemia (204–208)	100	0.0016	0.0014
Other cancers (140–149, 170–173, 190–203)	110	0.0075	0.0040
Diabetes (250)	120	0.0037	0.0030
Rheumatic fever and rheumatic heart disease (390–398)	150	0.0006	0.0007
Hypertensive heart disease (402, 404)	160	0.0019	0.0020
Ischemic heart disease (410–414)	170	0.0511	0.0403
Other heart disease (415–429)	180	0.0198	0.0206
Hypertension with or without heart disease (401, 403)	190	0.0008	0.0012
Cerebrovascular diseases (430–438)	200	0.0159	0.0163
Atherosclerosis	210	0.0022	0.0030
Other diseases of arteries, arterioles, and capillaries (440)	220	0.0027	0.0026
Pneumonia and influenza (480–487)	230	0.0082	0.0117
Chronic obstructive pulmonary disease, COPD (490–496)	240	0.0103	0.0089
Ulcer of stomach and duodenum (531–533)	250	0.0007	0.0008
Chronic liver disease or cirrhosis (571)	260	0.0014	0.0009
Nephritis, nephritic syndrome, and nephrosis (580–589)	270	0.0020	0.0025
All others (excluding pregnancy, maternity, congenital, and external causes)	280	0.0026	0.0030

Notes: The sample is white cohorts born between 1901 and 1925 in the 48 U.S. states, matched to compulsory schooling by state of birth and year of birth. Death rates are calculated at the gender, year, state-of-birth, and year-of-birth level for each disease. The statistics are not weighted by cell size. $N = 119{,}975$.

[a]ICD-9 causes of death were classified into 25 categories using the 34-cause categorization that is used in the National Center for Health Statistics publications.

registrants to compulsory attendance and child labor laws. We limit the sample to whites born in the 48 states between 1901 and 1925 (as in the MDF 80–90).

Summary statistics for the final SEER sample are shown in Table 2. The sample consists of 625,958 individuals. Our sample is relatively old because we exclude people born after 1925; therefore, average age at diagnosis for this sample is around 70, which is older

Table 2. SEER Summary Statistics

	Full Sample: All Individuals Diagnosed Between 1973 and 1993, With No Missing Values (N = 625,958)		Restricted Sample: Individuals Diagnosed in 1983 and 1993 Only (N = 67,749)	
Variable	Mean	*SD*	Mean	*SD*
Years of Compulsory School	6.939	1.065	6.966	1.022
Female = 1	0.468	0.499	0.455	0.498
Age at Diagnosis	69.397	7.991	72.530	7.391
Hispanic = 1	0.020	0.142	0.022	0.145
Married = 1	0.640	0.480	0.603	0.489
Died Within Five Years of Diagnosis = 1	0.634	0.482	0.682	0.466
Year of Diagnosis (1973 = 1)	11.747	5.726	15.576	4.982
Cancer Site (broad categories)				
Bones and joints	0.00	0.03	0.00	0.03
Brain and other nervous system	0.01	0.11	0.01	0.11
Breast	0.12	0.33	0.11	0.32
Digestive system	0.23	0.42	0.24	0.43
Endocrine system	0.00	0.07	0.00	0.06
Eye and orbit	0.00	0.04	0.00	0.04
Genital system	0.20	0.40	0.20	0.40
Leukemia	0.03	0.16	0.03	0.16
Lymphomas	0.03	0.18	0.03	0.18
Buccal cavity and pharynx	0.03	0.17	0.03	0.16
Multiple myeloma	0.01	0.11	0.01	0.11
Ill-defined and unspecified sites	0.03	0.17	0.03	0.18
Respiratory system	0.20	0.40	0.20	0.40
Skin	0.02	0.13	0.02	0.13
Soft tissue	0.00	0.06	0.00	0.06
Urinary system	0.07	0.26	0.07	0.26

than the average age in the full SEER data (62). About two-thirds of the population died within five years of diagnosis,[8] most frequently from cancers of the digestive system, the respiratory system, and the genital system. We also report summary statistics for individuals diagnosed only in 1973 and 1983 because we use this subsample in many specifications in this paper. As the table shows, this sample, although much smaller (N = 67,749), has the same average characteristics as the larger sample.

Measures of Progress

Drug approvals. One measure of innovation is the number of active ingredients (new molecular entities) approved by the Food and Drug Administration (FDA) to treat a particular

8. Not surprisingly, because we are calculating mortality conditional on a cancer diagnosis, the average five-year mortality rate is much higher than in the cause-specific data.

disease. This measure captures the role of one component of innovations in medical care in generating gradients. Estimates suggest that about half of the recent improvement in mortality is attributable to improvements in medical care (Cutler, Rosen, and Vijan 2006).

We use the number of active ingredients rather than drug approvals because the former constitute greater pharmaceutical innovations: only about 2,000 active ingredients have appeared in the United States since the FDA's inception in 1938, yet more than 80,000 drugs have been approved by the FDA.[9] Because new ingredients are considered to be major innovations, our measure excludes drugs that are close substitutes for existing treatments. For simplicity, we refer to active ingredients as *drugs* in the rest of the paper. The data on active ingredients, the condition(s) they were approved to treat,[11] and their FDA approval dates were given to us by Frank Lichtenberg and originally were obtained through a Freedom of Information Act request to the FDA.[10]

We match the active ingredient data to our sample using ICD-9 condition codes.[11] For the disease specific mortality data (MDF 80–90), we match mortality rates with the number of drugs approved in the previous five years (e.g., 1980–1983 mortality is matched to the number of drugs approved between 1975 and 1979) or in the previous 10 years (1970–1979). We also repeat these analyses using the stock of drugs—that is, the total number of drugs in existence to treat a condition.

In the SEER cancer data, we use both the stock of drugs at a point in time and the number of drugs newly introduced (the flow of drugs). When using the flow of new drugs, we match individuals with the number of drugs approved in the prior 10 years and restrict our attention only to those diagnosed in 1983 and 1993. Alternatively, we match individuals with the number of drugs in existence as of 2000 and look at all individuals diagnosed between 1973 and 1993. We prefer the stock measure because very few new cancer drugs are approved in any given year, and many of these are used for several cancer types, so there is little variation in our data. The stock measure overstates the number of drugs available to individuals at the beginning of the period. The stock of drug measure also 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, and the extent of such secondary innovations depends on the stock of existing drugs available.

The match between drugs and diseases is better in the SEER than in the mortality data because drugs typically treat specific diseases (rather than causes of death), and in the SEER, we know each individual's particular condition. In the mortality data, the match between drugs and causes of death can be misleading: for example, drugs used to control diabetes can reduce death rates attributable not only to diabetes but also to heart disease, stroke, kidney failure, and other conditions.

Changes in mortality rates. New drug approvals are a tangible but very limited measure of health-related innovation. Many drugs are subsequently used to treat diseases that they were not originally approved for (i.e., off-label), and these innovations would not be captured in our drug measure. The drug measure does not capture nondrug medical innovations 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 surgical or diagnostic innovations (Fuchs and Sox 2001). These latter innovations may

9. See Lleras-Muney and Lichtenberg (2002) for a discussion of how the data were constructed and how FDA approval dates were coded.

10. Unfortunately, not all drugs in our data can be dated: some may have been invented prior to the creation of the FDA in 1938, and there are others for which we could not impute a date of approval. The dates of approval in our data range from 1938 to 2001, with a mean of 1973.

11. This measure does not capture off-label use of drugs. If off-label use is common, there will be more measurement error in our innovation measure. Off-label use would be captured in analyses that use changes in mortality by disease, discussed in the next section.

affect education gradients differently than do drugs, potentially generating either greater or smaller gradients. Finally, the drug measure fails to capture innovations in knowledge about modifying disease risk through, for example, diet and exercise, which explain the remaining half of mortality gains.

As an alternative measure of progress, we compute the change in age-adjusted mortality by disease (from published age-adjusted mortality rates by cause and year) and the change in age-adjusted five-year survival conditional on diagnosis (for the SEER data) in the previous decade. Thus, for the disease-specific data, we can calculate the absolute change in age-adjusted mortality from 1970 to 1979 and 1980 to 1989.[12] We match mortality rates in 1980 and 1990 to the absolute change in age-adjusted mortality from 1970 to 1979 and 1980 to 1989, respectively.

Likewise, for the SEER cancer data, we calculate the change in five-year survival conditional on diagnosis from 1973 to 1982 (matched to individuals in 1983) and from 1983 to 1992 (matched to individuals in 1993). We also calculate changes in survival conditional on the stage of cancer at diagnosis (for cancers for which stage is assessed).

The mortality measure, unlike the drug measure, captures all aspects of progress in disease prevention and treatment. This measure of progress does not precisely capture the timing of innovations that led to mortality declines. That is, innovations in prevention and screening that took place several decades earlier may be associated with mortality declines today. Our analysis would infer that such innovations (which we observe only as declines in overall mortality today) generate disparities today. We cannot identify the source or timing of the original innovation. These indices are also based on past observations of the data that also compose our dependent variables, so that the specifications using the mortality indices are not as econometrically robust as those using the drug measures.[13]

Summary statistics on our progress measures are provided in Table 3. These measures suggest that for all causes of mortality, the rate of health-related innovation measured using drugs accelerated in the 1980s, whereas the rate measured using age-adjusted mortality shows more progress in the 1970s. This suggests that nondrug innovation was a more important factor in the earlier period. For cancer, the rate of innovation using drugs was more rapid in the 1970s, whereas changes in survival saw larger improvements between 1983 and 1993 than in the prior decade. Moreover, a greater share of the progress against cancer in the earlier period appears to be attributable to improvements in diagnosis because conditioning on stage reduces the magnitude of progress in the early period, but not in the later period.

In sum, we measure education gradients in all-cause mortality (using the MDF data) and education gradients in cancer mortality conditional on diagnosis (using the SEER data). We use two measures of progress in each case. The first measure—new drug approvals—captures pharmaceutical innovations in medical care. The second measure—changes in age-adjusted mortality—captures all innovations, wherever they may occur, that affect health.

EMPIRICAL STRATEGY

The hypothesis that gradients are related to progress suggests an empirical strategy in which we would estimate a model of the probability of dying, where education is interacted with progress:

$$P(died = 1) = \beta_0 + \beta_1 education + \beta_2 education \times progress + \beta_3 progress + \mathbf{X}\gamma + e, \quad (1)$$

12. For small causes of death, percent changes are very misleading.

13. These indices include lagged dependent variables, which can cause inconsistency in panel models with fixed effects (Nickell 1981). If we use changes in mortality as a measure of progress and do not include fixed effects, we find very similar estimates (results available upon request).

Table 3. Summary Statistics for Various Progress Measures

	Number of Observations	Mean	*SD*	Minimum	Maximum
Measures for All Causes of Death					
Number of drugs					
Total (ever approved)	25	62.6	74.69	0	338
By decade					
Approved between 1970 and 1979	25	7.6	9.6	0	41
Approved between 1980 and 1989	25	9.72	12.81	0	52
By five-year period					
Approved between 1975 and 1979	25	3.64	4.62	0	15
Approved between 1985 and 1989	25	4.6	5.96	0	26
Improvements in mortality					
Change in age-adjusted mortality, 70–79	25	6.72	32.89	–56.21	144.1
Change in age-adjusted mortality, 80–89	25	3.25	17.56	–13.82	79.3
Measures for Cancer Progress					
Number of drugs					
Number of drugs by 1993	81	9.654	10.015	0.00	48.00
Number of drugs approved between 1973 and 1982	81	1.72	1.68	0	5
Number of drugs approved between 1983 and 1992[a]	80	1.24	1.6	0	6
Improvements in mortality					
Change in five-year survival conditional on diagnosis from 1973 to 1982	81	0.025	0.108	–0.26	0.55
Change in five-year survival conditional on diagnosis from 1983 to 1992[a]	80	0.037	0.087	–0.29	0.32
Change in five-year survival conditional on diagnosis and local stage from 1973 to 1982[b]	141	0.018	0.121	–0.38	0.55
Change in five-year survival conditional on diagnosis and local stage from 1983 to 1992[b]	145	0.037	0.121	–0.33	1.00

Notes: Age-adjusted mortality is calculated as the number of deaths per 100,000. Changes in age-adjusted mortality are calculated as the level difference between the early period and the later period; therefore, positive numbers are decreases in age-adjusted mortality or progress. Because small diseases have very small mortality rates, we did not calculate percentage changes.

Sources: Data on drug approvals were provided by Frank Lichtenberg; see the text. Age-adjusted mortality by cause since 1950 by year and disease were obtained from unpublished tables provided by the National Center for Health Statistics "290 Trend Tables from CDC/NCHS, National Vital Statistics System, Mortality Data" (tables HIST290_5059, HIST290_6067, HIST290_6878, and HIST290_7998), which can be found online at http://www.cdc.gov/nchs/datawh/statab/unpubd/mortabs/hist290.htm. Changes in five-year survival rates by diagnosis or by diagnosis and stage were calculated by the authors using SEER data.

[a]There was no one diagnosed with one of the cancers in the list of 81 possible cancers in 1992.

[b]The number of observations is not 81 × 2 because not all cancers have stage defined. Also there are some cancers × stage cells that are empty.

where **X** might include disease fixed effects, year fixed effects, disease × year fixed effects, and other individual characteristics. Prior research suggests that β_1 should be negative, reflecting the steady-state advantage of the more educated (the more educated have lower mortality rates), and β_3 should be negative because progress improves survival. Although,

a priori, the interaction term β_2 may be negative, positive, or zero, if the more educated do indeed benefit first from innovation, β_2 (the interaction between education and progress) should be negative, meaning that education should lower mortality more for those diseases with larger innovations in the recent past.

We do not estimate this model for two reasons. First, there are no major data sets that contain both educational attainment and detailed cause of death, and span a long period of time. Thus, we could not estimate this model directly even if we wished to do so.

Second, a major concern in estimating the model and using actual education is that for many reasons, those individuals who choose to obtain more education are also more likely to be healthier than those who do not; for example, they may be more patient (Fuchs 1982), or their parents may have been wealthier. In response to this concern, recent papers have used compulsory schooling laws to obtain consistent causal estimates of the effects of education, exploiting the fact that compulsory education laws (which specified the number of years that a child had to attend school) meant that individuals within a state no longer could choose the amount of schooling they obtained.[14] This implies that these laws can be used as instruments for education. Studies (most recently, Moretti and Lochner 2004) have shown that these laws had a substantial impact on educational attainment,[15] and that it is plausible that compulsory schooling laws affected mortality only though their effect on education (Lleras-Muney 2005; Oreopolous 2003). We follow this strategy here, using compulsory schooling as a measure of educational attainment. Because the specification includes state-of-birth and cohort-of-birth fixed effects, the effect of compulsory schooling is identified by variation within states over time. If some states are consistently more progressive in social policy than others, this state characteristic will be captured by the state fixed effect and will not bias the estimated effects of education. Similarly, if some birth cohorts were more likely to be exposed to some common health shock (such as the 1918 influenza pandemic) than others, this effect will be captured by the cohort fixed effect. Changes in other state policies or cohort characteristics would bias the effects of increased education only if they were exactly contemporaneous with changes in compulsory schooling within each state.

Empirical Model for the MDF Data

Using the grouped MDF data, we estimate the following linear probability model:

$$MR_{dsct} = \beta_0 + \beta_1 CS_{sc} + \beta_2 CS_{sc} \times progress_{dt} + \mathbf{X}\gamma + \boldsymbol{\eta}_{\mathbf{c}} + \boldsymbol{\mu}_{\mathbf{s}} + \boldsymbol{\lambda}_{\mathbf{dt}} + e_{dsct}, \tag{2}$$

where MR is the four-year mortality rate for cause d in year t, for individuals born in state s of cohort c. CS is the number of years of compulsory schooling for individuals of cohort c born in state s, and progress is a measure of innovation for disease d that occurred in the years up to year t. We control for 47 state-of-birth dummy variables ($\boldsymbol{\mu}_{\mathbf{s}}$), 24 cohort dummy variables ($\boldsymbol{\eta}_{\mathbf{c}}$), 24 disease dummy variables, a decade dummy variable, and disease × year dummy variables ($\boldsymbol{\lambda}_{\mathbf{dt}}$); thus, progress main effects are not estimated. We also control for age and age squared ($\mathbf{X}$) and estimate this equation separately by gender.[16] The coefficient of interest is β_2, which measures whether the effect of education is larger for diseases with greater innovation. Because we are including disease dummy variables, our estimates are

14. However, these laws may have had differential effects on populations within a state (e.g., urban versus rural residents). Average differences across states are captured by the state-of-birth dummy variables, but we cannot identify these differential effects within state when using these data.

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

16. We repeat our analyses separately by gender. Our hypothesis does not specifically predict variation in the effect of education by gender, but this specification is consistent with prior research, which suggests that the effect of most covariates (e.g., age and education) varies by gender as well as the fact that men and women are susceptible to different diseases (particularly in the case of cancers).

identified using variation within diseases over time: thus, we are testing whether education gradients increase for a given disease when innovation increases rather than comparing gradients among diseases at a point in time.

Empirical Model for the SEER Cancer Data

Using the SEER cancer data at the individual level, we estimate the following linear probability model:

$$P_{idsct}(died = 1) = \beta_0 + \beta_1 CS_{sc} + \beta_2 CS_{sc} \times progress_{dt} + \mathbf{X_{idsct}}\gamma + \boldsymbol{\eta}_c + \boldsymbol{\mu}_s + \boldsymbol{\lambda}_{dt} + e_{idsct}, \quad (3)$$

where P is the probability of dying within five years for individual i diagnosed with disease d in year t, of cohort c and born in state s. CS is the number of years of compulsory schooling for individuals of cohort c born in state s, and progress is a measure of innovation for disease d that occurred in the years up to year t. $\mathbf{X_{idsct}}$ is a vector of individual characteristics including age, age squared, 138 county-of-current-residence dummy variables, five stage-of-diagnosis dummy variables, year-of-diagnosis dummy variables, 80 disease dummy variables, stage × disease dummy variables (comparing cancers at the same stage over time), and disease × year dummy variables $\boldsymbol{\lambda}_{dt}$. Thus, progress main effects are not estimated. This specification also controls for 47 state-of-birth dummy variables ($\boldsymbol{\mu}_s$) and 24 cohort dummy variables ($\boldsymbol{\eta}_c$). We estimate this equation separately by gender. Unlike the MDF, the SEER data include information on county of current residence. By including dummy variables for each of these counties, we can control for differences that arise because of geographic disparities in access and quality of care.

Choice of Statistical Model

Although for both the SEER data (individual level) and the MDF data (group rates), outcome variables fall between 0 and 1, we report estimates of Eqs. (2) and (3) using linear probability models. We use this functional form because our primary interest is in the coefficients on the interaction terms between compulsory schooling and technological progress. The interpretation of interaction terms in nonlinear models is complex and problematic. As Ai and Norton (2003:123) showed, "The magnitude of the interaction effect in nonlinear models does not equal the marginal effect of the interaction term, can be of opposite sign, and its statistical significance is not calculated by standard software." Additionally, calculating the marginal effects is particularly challenging given that our model includes disease fixed effects. We repeated our analyses by using a logistic model. The signs and significance levels of our point estimates for the principal coefficients of interest (compulsory school and its interaction with the two progress measures) are fully consistent with those of the linear probability model (results available from the authors on request). The results reported here use a linear probability model, which is more straightforward to interpret.

DOCUMENTING THE GRADIENT

We begin by documenting the education gradient in the MDF mortality and SEER cancer data using the number of years of compulsory schooling as a measure of education. If compulsory schooling laws affected mortality only through their effect on education, estimates from this (reduced-form) equation can be interpreted as causal.

Results Using the MDF Data

Table 4 describes these gradients in the disease-specific, four-year mortality data (MDF 80–90). Recall that these data are aggregated into cells defined by cause of death, gender, cohort, state of birth, and year; and that the regression includes gender, age, age squared, year dummy variables, and dummy variables for cohort and state of birth, as well as cause-of-death dummy variables interacted with year. Because we include state-of-birth and cohort

Table 4. Two-Sample Instrumental Variables (TSIV) Estimates of the Effect of Education on Four-Year Mortality Rates by Year, Gender, State of Birth, Year of Birth, and Cause of Death Estimated using Mortality Cause-of-Death (MDF 80–90) Data

	(1) Effect of Compulsory School on the Four-Year Mortality Rate ($\times 10^4$)	(2) Effect of Compulsory School on Education, From Census	(3) TSIV Effect of Education on the Four-Year Mortality Rate ($\times 10^4$)	(4) Effect of One More Year of Education on Mortality at the Mean
Mean Mortality				0.0067
(%)				–14.1
All	–0.41†	0.044*	–9.41	
	(0.24)	(0.0010)	(5.80)	
Males	–0.73*	0.054*	–13.43†	
	(0.35)	(0.012)	(7.10)	
Females	–0.10	0.041*	–2.46	
	(0.29)	(0.012)	(7.08)	

Notes: Standard errors are in parentheses. Controls are age, age squared, female dummy variables, state-of-birth dummy variables, cohort dummy variables, decade dummy variables, cause-of-death dummy variables, and cause-of-death × decade dummy variables. The sample consists of whites born in the 48 U.S. states between 1901 and 1925. Standard errors for the TSIV estimates were calculated by using the Delta method. Coefficients in columns 1 and 3 have been multiplied by 10^4 for ease of reporting.

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

dummy variables, the effect of compulsory schooling laws is identified from variations in the laws within states over time. We find negative effects of compulsory schooling on mortality for both genders, although they are statistically significant only for men.

Results Using the SEER Cancer Data

In Table 5, we document education gradients in the SEER data. These data contain observations on individuals, and the model we estimate includes state-of-birth dummy variables, cohort dummy variables, county dummy variables, year-of-diagnosis dummy variables, site dummy variables, stage-of-diagnosis dummy variables, stage × disease dummy variables, and disease × year dummy variables. Again, we identify the effect of compulsory schooling from variation in the laws within states overtime. We find a negative and significant effect of compulsory schooling on mortality for both males and females. We repeat the analyses for those diagnosed only in 1983 or 1993 because this sample is the one that we use to examine changes in survival rates or flows of new drugs. The coefficients are of similar magnitude but are statistically insignificant in this smaller sample.

Magnitude of the Effects

To better interpret these effects, we compute the effect of education that is implied by our estimates of the effect of compulsory schooling on mortality.

First, we use the 1970, 1980, and 1990 censuses to estimate the effect of compulsory schooling on educational attainment, measured in years. 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 (see column 2 of Table 4).

The estimate of the effect of education on mortality can be calculated as the ratio of the effect of compulsory schooling on mortality reported in column 1, to the effect of compulsory schooling on education reported in column 2. (This is known in economics as the *two-sample instrumental variables estimate.*) Using this method, we find that the implied effect of education on unconditional mortality is –0.0009. At the mean, this coefficient implies

Table 5. Two-Sample Instrumental Variables (TSIV) Estimates of the Effect of Education on the Probability of Dying Within 5 Years (dependent variable = 1 if died within five years of diagnosis): SEER Data

	(1) Effect of Compulsory School on the Probability of Dying in Five Years	(2) Effect of Compulsory School on Education, From Census[a]	(3) TSIV Effect of Education on the Probability of Dying in Five Years	(4) Effect of One More Year of Education on the Probability of Dying at the Mean
Results for All Years, 1973–1993				
Mean mortality				0.634
(%)				–9.5
All	0.0025*	0.0430*	–0.0604*	
	(0.0008)	(0.0084)	(0.0220)	
Males	0.0018*	0.0430*	–0.0436†	
	(0.0009)	(0.0104)	(0.0231)	
Females	–0.0032*	0.0451*	–0.0731*	
	(0.0012)	(0.0101)	(0.0313)	
Results for 1983 and 1993 Only				
Mean mortality				0.681
(%)				–10.3
All	–0.0031	0.0440*	–0.0704	
	(0.0022)	(0.0098)	(0.0524)	
Males	–0.0040	0.0541*	–0.0739	
	(0.0030)	(0.0124)	(0.0580)	
Females	–0.0027	0.0405*	–0.0666	
	(0.0033)	(0.0121)	(0.0838)	

Notes: Standard errors, shown in parentheses, are clustered at the cancer-site level. Standard errors for the TSIV estimates were calculated by using the Delta method. Regressions include age at diagnosis, age at diagnosis squared, 47 state-of-birth dummy variables, 24 cohort dummy variables, 80 cancer-site dummy variables, county-of-residence dummy variables, stage-of-cancer-at-diagnosis dummy variables, site × stage dummy variables, and site × year dummy variables. The sample is whites born in the 48 U.S. states between 1901 and 1925.

[a]Data for all years (1973–1993) are from the 1970, 1980, and 1990 census. Data for 1983 and 1993 only are from the 1980 and 1990 census.

†Significant at 10%. *Significant at 5%.

that one more year of schooling reduces cause-specific, four-year mortality by about 14% (which is obtained by computing the ratio of the effect of one more year of school divided by mean mortality: 0.00094 / 0.0067).

In the SEER data (Table 5), the effect of education on cancer mortality conditional on diagnosis computed in the same manner is roughly –0.06. At the mean, this coefficient suggests that one more year of education reduces the probability of dying of cancer within five years of diagnosis by about 10% (0.06 / 0.634). The effect of education we estimate is somewhat larger than ordinary least squares (OLS) estimates from other studies, as is the generally the case in studies that use compulsory education.[17]

17. Estimates of the effect of education in other studies suggest that one more year of education reduces five-year mortality rates by 2% to 5% (Elo and Preston 1996). Our estimates are larger. This is probably because compulsory education affected individuals whose returns to education were larger than those of the average person. This could be the case if, for example, compulsory schooling affected individuals at the low end of the education

We find that for the cause-specific mortality data (MDF 80–90), the effect of education on mortality is greater for men than for women. This is a commonly found result in the literature (e.g., see Elo and Preston 1996). In the cancer data, the pattern is less clear, with larger gradients for women in the full sample but larger gradients for men in the restricted sample (1983 and 1993 only). We also find that the effect of education on mortality is somewhat larger in the disease-specific data than in the cancer data, which suggests that education reduces the incidence of disease as well as improving survival conditional on disease.

THE EFFECT OF PROGRESS ON THE EDUCATION GRADIENT IN MORTALITY

We now relate the education gradients by disease to progress (measured by changes in mortality/survival and by number of drugs) for that disease. We report results by using years of compulsory schooling as our measure of education.

MDF

In Table 6, Panel A, we report estimates of Eq. (2) by using the mortality files (MDF 80–90) and by using the number of drugs approved in the previous five years for treatment of the disease as our measure of progress. We find that the interaction between compulsory schooling and this progress measure is negative and significant.

To calculate the magnitude of the effect of technological innovation on the gradient, we first calculate the share of mortality explained by compulsory schooling at the mean level of innovation in the sample (about four drugs approved in the prior five years; see Table 3). The gradient is computed by multiplying this level by the coefficient on compulsory schooling interacted with drugs approved in the prior five years in Table 6 (–4.84 × 10–5) and adding the coefficient on compulsory schooling itself (15.81 × 10–5). This provides an estimate of the effect on mortality of one year of compulsory schooling at the mean level of drug innovation (–0.00003555). To assess the magnitude of this value, we divide this estimate by the average mortality in the sample (from Table 1, 0.0067). We find that the effect of one additional year of compulsory schooling in the presence of the average level of drug innovation is 0.5% of average mortality. We then repeat this analysis, increasing the number of drugs approved by 1 standard deviation (about five additional drugs, see Table 3). The effect of one year of additional compulsory schooling increases to 4% of average mortality for diseases with 1 standard deviation above the average (about 9) number of drugs approved in the preceding five years.

We repeat the analysis using alternative measures of innovation. In Panel B, we use the number of drugs approved in past 10 years (rather than in the past five years) and again find negative and significant interaction effects. Finally, we repeat this analysis using the total number of drugs approved by disease as of 1990 (not the flow) as the measure of progress (Panel C). We do this because date of approval is missing for several drugs and was sometimes imputed. Moreover, we want to test whether using the stock (rather than the flow of drugs) results in any bias because we use stocks in our analyses of cancer survival. We find that the interaction with total number of drugs is also negative and significant. The interaction coefficient is smaller, as one might expect given that this is a noisier measure of innovation.[18] Our results are therefore not very sensitive to how we define the period over

distribution and these individuals have larger returns to education. It could also happen if individuals affected by the laws were credit constrained and would not otherwise be able to afford further education. Research on the effects of compulsory schooling in Norway has indeed found that the wage returns to those affected by the reforms are much larger than the average returns (Salvanes, Vaage, and Aakvik 2003). Similar arguments have been made, for example, in Moretti and Lochner (2004) and Lleras-Muney (2005).

18. We repeated these analysis using drugs approved in the concurrent five years and obtained almost identical results.

Table 6. Is the Effect of Education on Mortality Larger for Diseases With More Progress: Mortality Cause-of-Death Data (MDF 80–90)

	Compulsory School × Progress	Compulsory School
Panel A. Progress measure: Number of drugs approved in the past five years		
All (× 10^5)	–4.84**	15.81**
	(0.35)	(2.80)
Males (× 10^5)	–5.92**	17.12**
	(0.50)	(4.08)
Females (× 10^5)	–3.77**	14.52**
	(0.41)	(3.31)
Panel B. Progress measure: Number of drugs approved in the past 10 years		
All (× 10^5)	–3.15**	23.12**
	(0.16)	(2.79)
Males (× 10^5)	–3.76**	25.28**
	(0.28)	(4.07)
Females (× 10^5)	–2.54**	20.98**
	(0.19)	(3.30)
Panel C. Progress measure: Number of drugs approved as of 1990		
All (× 10^5)	–0.33**	16.49**
	(0.03)	(2.87)
Males (× 10^5)	–0.41**	18.25**
	(0.04)	(4.18)
Females (× 10^5)	–0.25**	14.74**
	(0.03)	(3.39)
Panel D. Progress measure: Change in mortality in prior decade		
All (× 10^5)	–4.45**	18.05**
	(0.07)	(2.40)
Males (× 10^5)	–4.87**	17.01**
	(0.10)	(3.49)
Females (× 10^5)	–4.03**	–19.09**
	(0.08)	(2.82)

Notes: Standard errors in parentheses. Controls are age, age squared, female dummy variable, state-of-birth dummy variables, cohort dummy variables, decade dummy variables, cause-of-death dummy variables, and cause-of-death × decade dummy variables. The sample is whites born in the 48 U.S. states between 1901 and 1925. All coefficients and standard errors have been multiplied by 10^5 to facilitate reporting.

**Significant at 1%.

which innovation in drugs is measured. These results are also robust to using the number of observations in a cell as weights, which suggests that the results are not driven by a few common or a few uncommon diseases (results available from the authors upon request).

In Panel D, we repeat the analyses using changes in mortality in the previous decade as the measure of progress. Again, the coefficients on the interaction are negative and significant. The estimates of the interaction are quite similar for men and women. The gradient increases from 0.6% of average mortality (at the mean level of mortality change, about 5 per 100,000) to 17% of average mortality for diseases with 1 standard deviation greater

than average progress between 1980 and 1990 (a reduction in cause-specific mortality of about 25 per 100,000).

SEER

Table 7 reports the results of estimating Eq. (3), using the individual SEER data. In Panel A, we use the number of drugs approved between 1973 and 1993 and look at individuals diagnosed only in 1983 and 1993. Here, all the coefficients are negative but statistically insignificant, most likely because the sample size is small and there is little variation in number of drugs approved. In Panel B, we report the results using the stock of drugs available to treat this type of cancer as our progress measure, and using the full sample of all individuals diagnosed between 1973 and 1993. We find that the coefficients for men (and for the overall sample) are negative, as predicted, but that this pattern does not hold for women.

In both Panels A and B, we limit the types of progress considered to the approval of new drugs, thus excluding progress that takes the form of new drug combinations or off-label uses, as well as progress that is not drug-related. The small and generally insignificant coefficients here suggest that other forms of progress, such as new combinations of existing drugs or new surgical or radiation therapies, may be more important in generating education gradients in cancer survival.

In Panel C, we examine the interaction between changes in five-year survival and compulsory schooling. The coefficient is negative and significant for the overall sample and for men but, again, not for women.

The stage at which cancer is diagnosed may have changed over time differentially for more-educated and less-educated people. To address this concern, we repeat our analyses using the change in survival for each cancer at each stage of diagnosis in Panel D of this table. We find that the gradient in cancer survival (conditional on diagnosis) is significantly related to broader measures of progress overall and for males, but not for females.

One possible reason for this lack of robustness to gender in the SEER data may be that there is an unusual relationship between education and cancers of the reproductive organs for women, including breast cancer (Kaufmann et al. 2003). The incidence and severity of cancers of the reproductive system are 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 higher among those with more education (Martin 2000). When we repeat the analyses for women excluding cancers of the female reproductive system, we find results more similar to those for men. The coefficients are negative and significant in the analysis using drugs (Panel B) but insignificant when using other progress measures (Panels A, B, and D).

When the stock of drugs available for treatment is used as the measure of progress, the gradient increases from 0.35% of baseline mortality on average at the mean number of drugs (Table 3) to 0.39% of baseline mortality when we increase the number of drugs by 1 standard deviation above the mean (about 19 drugs). When the survival change is used as the measure of progress, we find that the survival advantage of one year of additional compulsory schooling increases from 0.5% to 1.3% of baseline mortality when survival progress increases by 1 standard deviation above average.

Overall, we find support for the hypothesis that the education gradient is steepest for those diseases that have seen the most health-related innovation. These results are quite robust to the inclusion of covariates, including average income in the county of residence.[19]

19. As a final test of the robustness of these results, we examined the effect on the interaction between the gradient and progress if we randomly matched drugs to diseases (rather than matching drugs to the disease for which they are indicated). We did this to check that the results we obtained are not an artifact of the empirical model that we estimate. We randomly matched drugs to diseases 200 times in the mortality cause-of-death data and the SEER data, reestimated Eq. (3) for each random match, and compared our correctly matched estimate with these random matches. For the disease-specific mortality, we randomized the match only within years. The results suggest that in

Table 7. Is the Effect of Education on Mortality Larger for Diseases With More Progress: SEER Cancer Data

	Compulsory School × Progress	Compulsory School
Panel A. Progress measure: Number of drugs in the past 10 years Sample: People diagnosed in 1983 or 1993 only		
All	–0.0010 (0.0008)	0.00007 (0.00262)
Males	–0.0012 (0.0012)	–0.0003 (0.0046)
Females	–0.0009 (0.0010)	0.00006 (0.00416)
Females, excluding cancers of the reproductive system	0.0007 (0.0006)	–0.0066 (0.0039)
Panel B. Progress measure: Number of drugs available to treat the condition Sample: All people diagnosed between 1973 and 1993		
All ($\times 10^5$)	–2 (9)	–205 (235)
Males ($\times 10^5$)	–16* (7)	144.5 (203)
Females ($\times 10^5$)	2 (6)	–371 (226)
Females, excluding cancers of the reproductive system ($\times 10^5$)	–31* (14)	189 (246)
Panel C. Progress: Change in five-year survival (conditional on diagnosis) in the previous decade Sample: People diagnosed in 1983 or 1993 only		
All	–0.057* (0.020)	–0.0008 (0.0023)
Males	–0.082** (0.020)	0.0005 (0.0027)
Females	0.006 (0.046)	–0.0029 (0.0032)
Females, excluding cancers of the reproductive system	–0.029 (0.067)	–0.0014 (0.0042)
Panel D. Progress: Change in five-year survival by stage (conditional on diagnosis) in the previous decade Sample: People diagnosed in 1983 or 1993 only		
All	–0.069** (0.015)	–0.0010 (0.0020)
Males	–0.073** (0.012)	–0.0008 (0.0028)
Females	–0.067 (0.041)	–0.0016 (0.0029)
Females, excluding cancers of the reproductive system	–0.038 (0.101)	–0.0031 (0.0048)

Notes: Standard errors are shown in parentheses. Clustered at the cancer-site level for cancer regressions. Regressions include age at diagnosis, age at diagnosis squared, 47 state-of-birth dummy variables, 24 cohort dummy variables, 80 cancer-site dummy variables, county-of-residence dummy variables, stage-of-cancer-at-diagnosis dummy variables, and site × stage dummy variables. The sample is whites born in the 48 U.S. states between 1901 and 1925.

*Significant at 5%. **Significant at 1%.

In the cancer data, after cancer-site dummy variables are included, adding other covariates (including stage-of-cancer dummy variables) has little effect on the magnitude and significance of the interaction estimates (results available upon request).

CONCLUSION

Health-related innovation has been an important source of improvements in the length and quality of life. Studies of technological diffusion in other contexts consistently point to education as a factor that increases the diffusion rate (Hall and Khan 2003). In this paper, we look at whether this pattern holds true in the context of health-related innovations. Specifically, we test the hypothesis that education gradients increase when health-related innovation increases. We relate education gradients in mortality to two measures of health-related innovation and show that education gradients become larger for diseases with more innovation. We find that the pattern holds for mortality rates from all causes and for cancer mortality conditional on diagnosis.

To obtain plausibly causal effects of education, we match individuals to the compulsory schooling laws that were in place in their state of birth when they were growing up. Because we can control for both state of birth and cohort of birth, our effects are estimated using changes in legislation within states over time.

Our approach has several other advantages. We use two very different measures of innovation: the change in mortality for a given disease, which accounts for all types of health-related progress; and the number of active drug ingredients recently approved to treat a disease, which is a very specific measure of medical innovation that can be dated. Each measure has its advantages and disadvantages, but our results do not depend on the measure we use.

Our findings have some limitations. The use of compulsory schooling allows us to make causal statements with more confidence, but it also limits the extent to which the findings can be generalized to other settings. In particular, this study exploits changes in secondary schooling; we cannot make any inferences about whether increases in schooling at higher levels (e.g., obtaining a college degree) would result in patterns similar to those that we report here. The limited variation in compulsory schooling laws means that our approach requires using very large data sets to find statistically significant results. Our results for women are smaller and less consistent than the results for males, particularly for mortality conditional on cancer diagnosis. Finally, our data do not allow us to systematically examine the mechanisms through which education interacts with technology. These limitations suggest that further research in this area would be very valuable, particularly approaches that examine more recent changes in education and where schooling levels are more variable than in this study.

Our results are consistent with Link and Phelan's (1995) fundamental cause hypothesis and with recent findings that gradients are larger in diseases that can be treated (Phelan et al. 2004). The substantial recent increases in socioeconomic gradients in health suggest that further research in this area that explores how gradients arise is warranted.

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both the SEER and the mortality data, we cannot reject the hypothesis that the estimate from the random match is significantly different from 0. Additionally, the point estimate of the correctly matched interaction term is outside the 95th percent confidence interval of the simulated interactions. These results are available upon request.

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