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THE EFFECT OF COLLEGE EDUCATION ON HEALTH

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## ABSTRACT

We exploit exogenous variation in college completion induced by draft-avoidance behavior during the Vietnam War to examine the impact of college completion on adult mortality. Our preferred estimates imply that increasing college completion rates from the level of the state with the lowest induced rate to the highest would decrease cumulative mortality by 28 percent relative to the mean. Most of the reduction in mortality is from deaths due to cancer and heart disease. We also explore potential mechanisms, including differential earnings, health insurance, and health behaviors, using data from the Census, ACS, and NHIS.

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**I. Introduction**

Schooling is highly correlated with subsequent health outcomes. For example, in 2007, the age-adjusted mortality rate of high school graduates aged 25 to 64 was more than twice as large as the mortality rate of those with some college or a collegiate degree (Xu et al., 2010). If these associations between health and education reflect a causal relationship, they would represent a significant non-pecuniary return to education. They would also imply that policies meant to increase educational attainment could serve as an important means for improving health. However, there is substantial debate about whether these associations actually represent causal effects (see the reviews by Grossman, 2006, and Cutler and Lleras-Muney, 2010). This paper is the first to provide a causal estimate of the effect of college completion on mortality. In doing so, we contribute to knowledge about the impacts of education on health for a new part of the schooling distribution, where the observed health gradient in education is steeper.

We use variation in college attainment induced by draft-avoidance behavior during the Vietnam War in an instrumental variables strategy, as in Malamud and Wozniak (2012). This enables us to identify the effect of increased higher education on the later health status of men who were eligible to be drafted into the Vietnam War. This strategy builds on Card and Lemieux (2000, 2001) who document the excess educational attainment among cohorts induced to enter college in order to defer conscription. While Card and Lemieux focus on differences in induction risk across birth cohorts, we also exploit state level variation in induction risk within cohorts. The existence of state level variation allows us to decompose national induction risk into two constituent parts: induction risk faced by a young man's own state cohort and induction risk faced by young men of that cohort in the rest of the country. Our decomposition yields two instruments, which we use to identify the impact of the two endogenous variables—education and veteran status—in our empirical framework. This approach is an advance over studies that used the Card and Lemieux measures to

identify the effect of college going on health outcomes using only year to year variation in induction risk (e.g., De Walque, 2007; Grimard and Parent, 2007; MacInnis 2006). This strategy was developed by Malamud and Wozniak (2012); here we provide a more detailed explanation of how identification works in our setting in which there are two linked endogenous variables. We also develop a test for identification grounded in the assumptions of our structural model.

We merge our data on national and state-level induction risk with the Vital Statistics Mortality Files from 1981 to 2007 and the U.S. Censuses for 1980, 1990, and 2000 to construct birth state-by-cohort level mortality rates. We also include information about basic demographic characteristics, cohort size, and labor market conditions at the time of entry from a variety of additional sources. Our instruments predict both veteran status and educational attainment for men in the affected cohorts, with the increase in education coming primarily from increased post-secondary schooling attainment. We therefore have a viable instrument for educational attainment at higher levels that can be purged of its correlation with veteran status for the Vietnam cohorts.

We focus on the cumulative mortality rate between 1981 and 2007 and perform our main analysis using data aggregated to the birth state-birth year cohort level. We first establish that the well-known gradient between education and health status is present and statistically significant in our Vital Statistics data using OLS specifications. Specifically, OLS shows that a birth state-birth year cohort with 100 percent college completion is associated with 102 fewer deaths by 2007 per 1,000 persons compared to a cohort with no one completing college. Our instrumental variables estimates indicate an effect that ranges from 93 to 172 fewer deaths per 1,000 persons, with our preferred specification yielding a magnitude similar to the OLS estimate. For the birth state-birth year cohorts in our sample, this translates into a reduction in mortality of 42.8 per 1,000 for states with the highest induced completion rates versus the lowest. Furthermore, none of these 2SLS estimates are significantly different from the OLS estimates. Results using 10-year mortality rates for the 1980s,

1990s and 2000s show that the effects of college completion on health are not confined to any particular decade. The largest effects are found for the impact of college completion in lowering deaths due to cancer and heart disease, which represent the leading causes of mortality in our sample of older adults. College completion decreases the cancer mortality rate by 72 percent, with lung cancer accounting for over half of the reduction.

Before concluding, we use the American Community Survey (ACS) and the National Health Interview Survey (NHIS) to explore mechanisms that might explain the documented relationship between college education and mortality. We examine the causal effect of college completion on auxiliary outcomes such as health insurance and wages, as well as the cross-sectional relationship between college completion and various health behaviors such as smoking, exercise, and obesity.

This paper helps to fill an important gap in the literature on the relationship between education and health. Previous analyses of the causal impacts of education on health outcomes, such as mortality, have relied on variation at the lower part of the schooling distribution. For example, Lleras-Muney (2005), Clark and Royer (2010), and Meghir et al. (2012) all exploit changes in compulsory schooling requirements to examine whether increased schooling improved the health of students on the margin of dropping out before 12th grade. Lleras-Muney (2005) finds large and significant effects of increased education on declines in mortality in the United States, whereas Clark and Royer (2010) find no evidence for an impact of education on mortality in England.[1] Meghir et al. (2012) find temporary improvements in mortality and other health measures for affected cohorts following a reform in Sweden. However, regardless of the causal impact of schooling on health at the margin of dropping out of high school, the causal relationship may be different at the margin between high school and college. Moreover, estimating the effect of education on health at the

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[1] Arendt (2005) and Albouy and Lequien (2009) also find no statistically significant impact of compulsory school reforms on health outcomes in Denmark and France, respectively, but in both studies the estimated effects have large standard errors.

college margin may be of particular interest given that the largest increase in educational attainment in recent years has occurred among students entering college (Turner, 2004).

The findings in this paper have important implications for both health and education policy. People value health, and the health returns to education may represent a substantial fraction of the pecuniary returns. Indeed, Cutler and Lleras-Muney (2006) calculate that their estimates of the health benefits from education increase the total returns to education by 15 to 55 percent. Because higher education policy in recent years has been focused on increasing college completion, this represents a particularly important margin of analysis.[2] If individual investments in college education are suboptimal because of credit constraints, externalities, or lack of information, the presence of additional health returns to college provides an even stronger case for subsidizing education. This is particularly relevant given recent discussions about the rising cost of college and the decline in federal financial aid for college students. On the other hand, a positive causal impact of higher education on health may pose a dilemma for health policy. Health improvements, like smoking cessation, may reduce health care costs in the short run only to increase them in the long run as individuals live longer or as other unforeseen consequences arise (Bearman, et al., 2011). Our analysis will help inform policymakers interested in the link between education policy and national healthcare spending.

The effect of postsecondary education on certain health behaviors—smoking in particular—has been examined previously. De Walque (2007) and Grimard and Parent (2007) exploit year-to-year variation in induction risk faced by cohorts of young men during the Vietnam War to identify the impact of education on smoking. Using different datasets (NHIS and the CPS Tobacco Supplements, respectively) and different specifications, they find that additional education has a

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[2] Increasing college completion rates is a stated goal of the Obama-Biden administration. They have pushed for higher college completion rates on several fronts: by expanding Pell Grant access, proposing grants to state post-secondary systems, and devising action plans for states (Office of the Vice President, 2011).

negative and significant effect on the likelihood of smoking.[3] Our paper extends this identification strategy by incorporating within-cohort variation in induction risk to account for veteran status. Moreover, we examine a wide range of both health outcomes and behaviors across multiple decades, providing a much broader picture of higher education's potential health impacts.

Finally, although it is not our main focus, this paper contributes to research examining the impact of military service during the Vietnam War on health outcomes and behaviors (Angrist, Chen, and Frandsen, 2010; Dobkin and Shabani, 2007; Conley and Heerwig, 2009; Hearst, Newman, and Hulley, 1986).[4] We find that veteran status has a statistically significant protective effect on mortality conditional on survival to 1980. This is potentially explained by higher rates of health insurance access among veterans in our sample.

## II. Background on the Vietnam Draft

Our instrumental variables strategy exploits variation in the risk of induction (also referred to colloquially as the risk of "being drafted") to which young men in the US were exposed during the Vietnam conflict. This section provides a brief overview of the sources of this variation. A more detailed discussion can be found in Malamud and Wozniak (2012); hereafter MW.

Approximately 2 million American men were drafted during the Vietnam War. The Selective Service System, which comprised over 4,000 local draft boards across the nation at that time, was responsible for registering recruits and classifying them for either deferment or selection. Responsibility for devising and meeting the national target number of conscriptions rested with the federal Department of Defense (DoD). To achieve this target, the DoD issued monthly "draft calls"

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[3] MacInnis (2006) uses a similar identification strategy to document the effect of education in reducing obesity and its co-morbidities such as hypertension and adult-onset diabetes.

[4] These studies all exploit variation in veteran status induced by the Vietnam draft lottery, which is a different source of identification than our own. None reject the hypothesis that the impact of veteran status on health outcomes is zero.

that divided the national number into quotas assigned to state draft boards, which did the active work of ordering men to be inducted.

Faced with an excess of eligible draft age men, draft boards adopted generous deferral policies toward large categories of men. Enrollment in a four-year college was the second most common deferral category, after the exemption for dependents (Semiannual Reports of the Director of the Selective Service System, 1967-1973).[5] The Military Service Act of 1967 codified the existing de facto arrangement by stating that college students in good standing could defer induction until receipt of an undergraduate degree or age 24, whichever occurred first. Over 1.7 million college deferments were granted in 1967 alone.[6] Although men who received college deferments were technically eligible for induction until age 35, very few men between the ages of 26 and 35 were ever drafted. Card and Lemieux (2000) estimate that, among men born between 1945 and 1947, those with a college degree were only one-third as likely to serve in Vietnam as compared to those without a college degree. Thus, the incentive to enroll in college to avoid the draft during these years was large.

Our identification strategy relies on two sources of variation in induction risk: over time and across states. The existence of intertemporal variation is well-known (Card and Lemieux 2000) and has been used in previous research (e.g., De Walque, 2007; Grimard and Parent, 2007). Inductions varied considerably over the course of the Vietnam War. From 1960 to 1963, inductions were fairly low at approximately 8,000 per month. However, following the Gulf of Tonkin incident in August 2, 1964, Congress authorized an expanded role for the U.S. military in Vietnam. Inductions more than doubled from 1964 to 1965 and again from 1965 to 1966. By the spring of 1968, in the midst of raging student protests, the rate of inductions reached a peak of almost 42,000 a month.

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[5] See also Tatum and Tuchinsky, *Guide to the Draft*, Ch. 3. By contrast, enrollment in a two-year college was not considered grounds for automatic deferment. See Rothenberg (1968).
[6] The number of college deferments remained above 1.7 million in 1968 and 1969, and then fell to 1.5 million and 1.3 million in 1970 and 1971 respectively (Semi-annual Reports of the Director of the Selective Service System, 1967-1973).

The introduction of the draft lottery in 1969 led to a substantial change in the induction process. However, college deferments continued to be issued until September 1971, and men who were already enrolled were allowed to retain their deferment until the end of the school year. Risk of induction during this period was also much lower since men were at risk of induction for only a single year and the overall rate of inductions was substantially lower, falling from about 20,000 per month in late 1969 to 2,000 per month in late 1971. In February of 1973, the draft was suspended and no more inductions took place.

The existence of state level variation in induction risk was less well known at the time, and remains so today. This type of variation arose through two channels. The first was uneven and idiosyncratic application of formal procedures across the thousands of local draft boards.[7] In their influential study of the draft, Davis and Dolbeare write, "The conclusion seems inescapable: local board autonomy implies both within state and between state variability, even among socioeconomically similar board jurisdictions."[8] Similar idiosyncrasies were described in the report of the U.S. National Advisory Commission on Selective Service, in 1967. A second source of state and year variation in induction risk were communication delays between federal, state, and local officials. These delays meant that the DoD assigned quotas using registrant numbers that were several months old. Thus, draft risk for an eligible man at a point in time was not only a function of the number of men in his state currently eligible for the draft but also of the number available several months ago. The current pool could be much larger than the past pool if, for example, a large number of local men graduated high school thus becoming draft eligible or much smaller if a large number married or aged out of the draft pool in the intervening months.

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[7] Davis and Dolbeare, *Little Groups of Neighbors*, Page 18.
[8] Davis and Dolbeare, *Little Groups of Neighbors*, Page 84.

### III. Empirical Strategy

We use variation in college attainment caused by draft-avoidance behavior during the Vietnam conflict to isolate the causal effect of education on health. An artifact of this identification strategy is that the likelihood an individual is a veteran also varies systematically across cohorts in our sample. Given that veteran status is a plausible determinant of health, it is important to control for this variable in our estimation strategy.[9] However, selection into military service during the Vietnam War was likely based on characteristics that are unobserved in our data, which would confound our estimates of veteran status and potentially other covariates as well. To deal with this, we exploit changes in both national and state-level induction risk to generate exogenous variation in both college-going and veteran status.[10]

This strategy is identical to the one described in MW. However, we extend the MW analysis in two ways. First, we provide formal econometric evidence of the relevance of our instruments by developing and implementing a test for first-stage power under the identifying assumptions appropriate to our model. Second, we provide a detailed discussion of the interpretation of our estimates when both direct and indirect effects are possible.

### A. Identification in a Structural Model with Direct and Indirect Effects

We begin by presenting a simple structural model that relates veteran status and college-going to health through both direct and indirect channels. The parameters of interest for our analysis of a health outcome $Y$ are the coefficients on the college-going indicator $C$ and the veteran status indicator $V$ in the first equation of the structural equation system[11]

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[9] As noted above, the literature on this question is mixed but tends to find no causal relationship between veteran status and later health among Vietnam veterans. Nevertheless, because veteran status will be affected by our instrumental variables for educational attainment, we think it is important to control for it.

[10] A number of related papers face the same problem. De Walque (2007) includes veteran status but treats it as an exogenous variable. MacInnis (2006) instruments for veteran status using a quartic polynomial in age, while Grimard and Parent (2007) consider a specification that instruments for veteran status using information about early health problems.

[11] Our model assumes that education only has a direct effect on health at the state-cohort level (that is, there is no indirect effect through veteran status). We believe this reflects the historical record. Certainly, college students could

(1) $$Y = \alpha_1 C + \alpha_2 V + X' \alpha_4 + \varepsilon$$

(2) $$C = \gamma_2 V + Z' \gamma_3 + X' \gamma_4 + \nu$$

(3) $$V = Z' \beta_3 + X' \beta_4 + \eta.$$

Suppose for simpliclity that the vector of instruments $Z$ has two entries $Z_1$ and $Z_2$. The $p$-dimensional vector $X$ contains additional covariates, including a constant. Both $Z$ and $X$ are exogenous in the sense that the unobserved error components $\varepsilon$, $\nu$, and $\eta$ satisfy $\mathrm{E}(\varepsilon \,|\, Z,X) = \mathrm{E}(\varepsilon)$, $\mathrm{E}(\nu \,|\, Z,X) = \mathrm{E}(\nu)$, and $\mathrm{E}(\eta \,|\, Z,X) = \mathrm{E}(\eta)$. Since we have constants in the system, we can without loss of generality take $\mathrm{E}(\varepsilon) = \mathrm{E}(\nu) = \mathrm{E}(\eta) = 0$. The variables $C$ and $V$ are assumed to be endogenous in (1) so that $\mathrm{Cov}(\varepsilon, C) \neq 0$ and $\mathrm{Cov}(\varepsilon, V) \neq 0$.

For our purposes, identification of $\alpha_0 := (\alpha_1, \alpha_2)'$ is best thought of in terms of reduced-form restrictions. We can write the reduced form of the structural system (1)-(3) as

(4) $$(Y \quad C \quad V) = (Z' \quad X') \begin{pmatrix} \Pi_{11} & \Pi_{12} \\ \Pi_{21} & \Pi_{22} \end{pmatrix} + \Psi.$$

Here $\Psi$ is a row vector of reduced-form errors and $(\Pi_{ij})_{ij=1,2}$ is the matrix of reduced-form parameters. The dimensions of $\Pi_{11}$ and $\Pi_{12}$ are $2 \times 1$ and $2 \times 2$, respectively; $\Pi_{21}$ and $\Pi_{22}$ are $p \times 1$ and $p \times 2$. The reduced form of (2) and (3) is therefore

$$(C \quad V) = (Z' \quad X') \begin{pmatrix} \Pi_{12} \\ \Pi_{22} \end{pmatrix} + \Psi_2,$$

where $\Psi_2$ consists of the second and third entry of $\Psi$. Denote the first entry by $\Psi_1$ and plug the preceding display into (1) to see

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delay or avoid conscription by staying in school, but induction rates for a birth state-birth cohort were unlikely to be affected by individual draft-avoidance behavior. This is because local draft boards needed to fulfill specific manpower requirements set by the Department of Defense. This assumption is also supported by evidence (available upon request) showing that veteran status is not predicted by graduation rates at the birth-state birth-cohort level in our data. Ultimately this assumption simplifies our econometric analysis, but it is not required for identification.

$$Y = (Z' \quad X') \begin{pmatrix} \Pi_{12}\alpha_0 \\ \Pi_{22}\alpha_0 + \alpha_4 \end{pmatrix} + \Psi_1.$$

We conclude from (4) that $\Pi_{12}\alpha_0 = \Pi_{11}$, and therefore $\alpha_0 = \Pi_{12}^{-1}\Pi_{11}$. The sample equivalent of this is just the IV estimator of $\alpha_0$. Since the reduced-form parameters are identified as long as $E[(Z',X')'(Z',X')]$ is invertible, it follows that $\alpha_0$ is identified if and only if

(5) $\qquad \text{rank}(\Pi_{12}) = 2,$

which requires the correlation structure of $Z$ and $(C,V)$ to be rich enough to disentangle the effects coming from $C$ and $V$.[12]

We now briefly outline a test of this rank condition based on minimum-distance considerations. Partition $\Pi_{12}$ into 2-dimensional vectors $\pi_1$ and $\pi_2$ so that $\Pi_{12} = (\pi_1, \pi_2)$. In particular, equations (2) and (3) imply $(\pi_1, \pi_2) = (\gamma_2\beta_3 + \gamma_3, \beta_3)$. Under the null hypothesis

(6) $\qquad H_0: \text{rank}(\Pi_{12}) = 1$

there exists some $\lambda_0 \in \mathbb{R}$ so that $\pi_2 = \lambda_0\pi_1$. Under the alternative (5), there is no such $\lambda_0$. Denote the OLS estimate of $\pi_0 = \text{vec}\,\Pi_{12} = (\pi_1', \pi_2')'$ by $\hat{\pi}$ and let $\widehat{W}$ be an estimate of the covariance matrix of $\hat{\pi}$. For $\pi \in \mathbb{R}^2$, define the function $g(\pi, \lambda) = (\pi', \lambda\pi')'$. Because $\pi_0 - g(\pi_1, \lambda_0) = 0$ under the null hypothesis, minimum-distance estimates of $(\pi_1, \lambda_0)$ can be found by minimizing

$$D(\pi, \lambda) = (\hat{\pi} - g(\pi, \lambda))'\widehat{W}^{-1}(\hat{\pi} - g(\pi, \lambda))$$

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[12] In the standard case with a single endogenous variable, $\Pi_{12}$ would be a vector and the usual first-stage $F$ statistic could be used to test the null hypothesis $\Pi_{12} = 0$ against the alternative of identification. In the present case, large $F$ statistics in first-stage regressions for $C$ and $V$ only provide evidence that there is correlation between the instruments and the endogenous variables, but do not rule out that the effects of $Z_1$ and $Z_2$ on $C$ and $V$ are too similar to prevent identification of $\alpha_0$. The more relevant pair of hypotheses is therefore the null of an insufficiently rich correlation structure, $\text{rank}(\Pi_{12}) = 1$, with the alternative of identification, $\text{rank}(\Pi_{12}) = 2$.

with respect to $(\pi, \lambda)$. Denote these minimizers by $(\tilde{\pi}_1, \tilde{\lambda})$. Provided that standard conditions are satisfied (Wooldridge, 2002), $D(\tilde{\pi}_1, \tilde{\lambda})$ has an asymptotic $\chi_2^2$ distribution under the null hypothesis (6) and otherwise diverges in probability as the sample size grows to infinity.

In the more general case with two endogenous variables and $k \geq 2$ instruments, the null and alternative hypotheses of this test remain unchanged, but $\Pi_{12}$ has $k$ rows and $D(\tilde{\pi}_1, \tilde{\lambda})$ converges to a $\chi^2_{2(k-1)}$ distribution under the null. Hence, if we divide $D(\tilde{\pi}_1, \tilde{\lambda})$ by $2(k-1)$, we can interpret

$$F(\tilde{\pi}_1, \tilde{\lambda}) = \frac{D(\tilde{\pi}_1, \tilde{\lambda})}{2(k-1)}$$

as a generalized first-stage $F$ statistic for a pair of endogenous variables. This is the Cragg and Donald (1993) test for identifiability.[13] In the standard case with a single endogenous variable, it reduces to the usual first-stage $F$ test. In our two-variable setting, the standard first-stage $F$ test and the Cragg-Donald test complement one another: we can use standard $F$ statistics to separately test, in each first stage model, the null hypothesis of no correlation between the instruments and the endogenous variables against the alternative of correlation. Rejecting the null in each case provides evidence that there is correlation, but the correlation structure is not necessarily rich enough to identify our structural parameter $\alpha_0$. The presence of correlation with insufficient structure is precisely the null hypothesis of the Cragg-Donald $F$ test. The alternative is identification of $\alpha_0$. Hence, if we reject the null in both the first-stage $F$ tests and the Cragg-Donald test, we have evidence that the structural parameters are identified.

Another test that is sometimes proposed in settings with multiple endogenous variables is the adjusted $F$-statistic developed in Angrist and Pischke (2009). It tests, separately for each first

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[13]Note that, as opposed to the Cragg-Donald minimum eigenvalue statistic routinely reported in statistical software, this test remains valid when cluster-robust covariance matrices are used.

stage model, whether the correlation between the instruments and a given endogenous variable is weak while accounting for the fact that some of the variation in the instruments is used in the remaining first stages. Although they do not represent a direct test of our structural model and identifying assumptions, the Angrist-Pischke $F$ statistics are potentially of interest as a diagnostic test of weak instruments so we report them as well.

## B. Interpretation of the Structural Parameters

We now show that $\alpha_0 = (\alpha_1, \alpha_2)'$ has a causal interpretation by using the instruments $Z_1$ and $Z_2$ to mimic otherwise impossible interventions that change $C$ while holding $V$ fixed (or vice versa) without affecting the error term $\varepsilon$. To avoid obscuring the problem with unnecessary notation, we do not explicitly include the covariates in the discussion, although all of the results below remain valid conditional on $X$. To interpret $\alpha_1$, it follows from (5) that we can find values $(z_{11}, z_{21})$ and $(z_{12}, z_{22})$ such that

(7) $\qquad \mathrm{E}(C \mid Z_1 = z_{11}, Z_2 = z_{21}) - \mathrm{E}(C \mid Z_1 = z_{12}, Z_2 = z_{22}) \neq 0 \qquad$ and

(8) $\qquad \mathrm{E}(V \mid Z_1 = z_{11}, Z_2 = z_{21}) - \mathrm{E}(V \mid Z_1 = z_{12}, Z_2 = z_{22}) = 0,$

i.e., switching from $(z_{11}, z_{21})$ to $(z_{12}, z_{22})$ changes the probability of college education without changing the probability of veteran status. From these two relations, equation (1), and the exogeneity assumption, we can conclude

$$\alpha_1 = \frac{\mathrm{E}(Y \mid Z_1=z_{11}, Z_2=z_{21}) - \mathrm{E}(Y \mid Z_1=z_{12}, Z_2=z_{22})}{\mathrm{E}(C \mid Z_1=z_{11}, Z_2=z_{21}) - \mathrm{E}(C \mid Z_1=z_{12}, Z_2=z_{22})}.$$

The parameter $\alpha_1$ therefore measures the average change in the outcome variable for individuals where an intervention changed the probability of college education without changing the probability of veteran status, adjusted for the fact that a change in the probability does not

necessarily induce an effect in the outcome. A similar argument can be made for $\alpha_2$ with different

values for $(Z_1, Z_2)$ and reversed equality signs in (7) and (8).

We can also show that $\alpha_1$ and $\alpha_2$ measure the direct causal effect of a change in $C$

(respectively $V$) status holding $V$ (respectively $C$) status constant in a potential outcomes

framework. Write the observed outcome $Y$ as a function of the potential outcomes $Y_{11}$, $Y_{01}$, $Y_{10}$, or

$Y_{00}$ depending on realizations $(C,V) = (1,1)$, $(0,1)$, $(1,0)$, or $(0,0)$,

(9)
$$Y = Y_{11}CV + Y_{01}(1-C)V + Y_{10}C(1-V) + Y_{00}(1-C)(1-V)$$

$$= \big(Y_{11} - Y_{01} - (Y_{10} - Y_{00})\big)CV + (Y_{10} - Y_{00})C + (Y_{01} - Y_{00})V + Y_{00}.$$

Under linear regression (constant effects) assumptions, we have $\alpha_1 = Y_{11} - Y_{01} = Y_{10} - Y_{00}$ and

$\alpha_2 = Y_{11} - Y_{10} = Y_{01} - Y_{00}$. In particular, the marginal effect of a college education is the same for

veterans and non-veterans and the marginal effect of veteran status is the same for college graduates

and non-college graduates. Hence, the first term in the second line of the preceding display drops

out and, if we take $E(Y_{00})$ as the intercept and $\varepsilon = Y_{00} - E(Y_{00})$, we are back in the framework of

the structural system in equation (1). As such, $\alpha_1$ is the "average treatment effect" because $\alpha_1 = $

$E(Y_{11} - Y_{01}) = Y_{11} - Y_{01} = Y_{10} - Y_{00} = E(Y_{10} - Y_{00})$. The same argument, with the roles of $C$ and

$V$ reversed, yields the equivalent result for $\alpha_2$.

Finally, it can also be seen that the parameters $\alpha_1$ and $\alpha_2$ are "local average treatment

effects" from two different interventions. Within the constant effects framework, $\alpha_1$ measures the

causal effect of an intervention described by (7) and (8) on individuals that comply with the

intervention. Intuitively, as long as condition (5) holds, our instruments vary enough to mimic a

situation in which variation in college access and variation in veteran status come from two

independent randomized experiments.[14] This breaks the dependency between outcomes in equations (1)-(3). It may be that some individuals would be "randomly selected" to receive both interventions, but their receipt of both interventions is random and unrelated to the relationships in the structural model. However, we emphasize that this interpretation crucially depends on our parametric assumptions. For a general nonparametric potential outcomes model, we cannot simplify (9). We therefore we do not claim to *non-parametrically* identify local average treatment or any other (direct, indirect, or total) causal effect.

## C. Instruments for College Education and Veteran Status

Finding instrumental variables that meet the conditions on $Z$ discussed in the previous section is obviously critical to our analysis. Fortunately, we are able to employ the same strategy as MW, who in turn extend an instrumental variables strategy inspired by Card and Lemieux (2000, 2001); henceforth CL. Like CL, we assume that draft avoidance was proportional to the risk of induction. To account for the mechanical relationship between inductions and veteran status, we exploit state level variation within the cohort level variation identified by CL. The existence of state-cohort level variation allows us to break national induction risk into its constituent parts and obtain two separate instruments that can be used to identify both college attainment and veteran status. Thus, young men faced state cohort risk that is analogous to the CL measure, where $s$ indexes state of residence and $c$ indexes one-year birth cohorts:

$$(10) \qquad staterisk_{sc} = \frac{\left(\sum_{t \mid c=19\,to\,22} I_{sct}\right)/4}{N_{sc}}$$

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[14] In other words, this is analogous to the thought experiment in which names are randomly selected from an urn to be treated with college access without affecting veteran status. For $\alpha_2$, all names are then placed back in the urn, and a second round of names is randomly drawn to be treated with veteran access without changing college status.

$I_{sct}$ is the number of inductions from birth state–birth year cohort *sc* in year *t*. Like CL, we construct

an average draft risk for the years a man was 19 to 22 since draft risk was non-trivial for men ages 20

to 22. $N_{sc}$ is birth state-cohort size. This measure of state cohort risk is our first instrument. We

then use our state level data on *I* and *N* to construct a second instrument in the following manner:

(11)
$$nationalrisk_{sc} = \frac{\left(\sum_{t\,|\,c=19\,to\,22}\sum_{-s} I_{-sct}\right)/4}{\sum_{-s} N_{-sc}}$$

This measure defines national cohort risk for a man born in state *s* and in birth cohort *c* as the

number of inductees from the set of all other states, denoted *–s*, and birth cohort *c*, divided by the

total number of such men at age 17.[15] In other words, the numerator and denominator in (11) are

national level inductions and cohort size for a cohort *c* minus the birth state inductions and cohort

size for the same cohort, respectively.

To construct the measures in equations (10) and (11), we obtained data on the number of

inductees from 1961 to 1972 in each state from reports of the Selective Service. We estimate state

cohort size using enrollment numbers spanning 1959 to 1970, the academic years in which our

cohorts of interest were in 11$^{th}$ grade. Thus state-cohort level risk (henceforth state risk) for a young

man born in Alabama in 1950 equals the number of inductees from Alabama in 1969 (the year he

turned 19) divided by the number of students enrolled in 11$^{th}$ grade in Alabama in 1967. National

level risk for the same young man roughly equals the number of men inducted nationally in 1969

divided by the size of his birth cohort; more precisely, we subtract own state inductions from the

numerator and own state cohort size from the denominator. We then construct an average national

draft risk for the years a man was 19 to 22. Figure 1 graphs the variation in state-level induction risk

for a selection of states over our period of interest. Panel A shows raw induction risk as defined in

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[15] Note that MW use variation at the six month birth cohort level. Here we use yearly variation because month of birth
is not available in the public use mortality data.

(10), Panel B shows residual state-level induction risk after controlling for a cohort trend, state-of-birth fixed effects, and national risk, and Panel C shows residual national-level induction risk after controlling for a cohort trend, state-of-birth fixed effects, and state-level risk. Given that we include these controls in our main empirical specifications, the patterns in Panels B and C most closely approximate our identifying variation.[16] We present our first stage analysis in the later results section.

**D. Validity of Our Instruments**

Given the novelty of our identification strategy, some of the IV assumptions bear more discussion. Our instruments could fail if young men attempted to exploit local variation in induction risk by moving between localities. In this case, risk would not be truly randomly assigned. Our risk measures would only bind for men who were unwilling or unable to move to low risk jurisdictions, which might in turn be correlated with other unobservable characteristics related to health. MW document that this type of "local board shopping" was prohibited by draft board regulations.

Identification further requires the assumption that induction risk only affected health through either education or veteran status. There are two ways in which this might fail. First, the health screenings required to determine draft eligibility might uncover an important health condition earlier than it might otherwise have been detected, thereby encouraging individuals to treat the condition and improving future health. Our reading of the historical literature suggests that this was unlikely. The required exams were very standard and did not involve any invasive testing for health conditions not easily observed in a doctor's office. It is therefore unlikely these exams provided young men with information they did not already have. [17]

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[16] For a more detailed discussion of the variation in residual induction risk seen in Figure 1, see MW.

[17] In fact, Tatum and Tuchinsky describe the exams this way, "Since the [Army] examines large numbers of men each day, and since the doctors, orderlies, and clerks assigned there must process them in assembly-line fashion as quickly as possible, the examinations are often careless. If you have a medical or other condition which should disqualify you, bring letters and other evidence from your own doctors…" (Ch. 6). A 1964 report to the president noted that "The current published medical standards are roughly the same as those which were in effect at the close of World War II," (U.S. Department of Health, Education, and Welfare, 1964, Ch. 3).

Second, young men may have viewed marriage or fertility choices as a means to reduce their chances of being drafted, and these choices may have connections to health. Marriage was not in itself grounds for deferment (Shapiro and Striker, 1970). Fatherhood, on the other hand, was grounds for deferment and was the most common draft exemption. However, there is little evidence that male fertility among affected cohorts responded to this. Bitler and Schmidt (2012) examine fertility changes among women who were likely to have been affected by the absence of men in affected cohorts. Their findings indicate that men in our affected cohorts were modestly but statistically significantly less likely to become fathers during the Vietnam years. Whether there is an effect on completed fertility is unknown. Nevertheless, we know of no evidence linking fertility timing to later adult health for men. Since the known effects (for women) are small, and since there is no known channel to link any potential effects for men to health, we believe that the exclusion restriction is valid in our context.[18]

## IV. Data Sources and Estimating Equations

We focus on mortality as our measure of health. We perform our main analysis on data aggregated to the birth state-birth year cohort level, and we refer to these cells as birth state-cohorts. Our main measure of mortality at the birth state-cohort level is the cumulative mortality rate per 1000 persons between 1981 and 2007, constructed as follows:

(12) $$MR_{sc} = \frac{deaths_{sc} \ between \ 1981 \ and \ 2007}{cohort \ size_{sc}^{1980}} * 1{,}000$$

where $s$ is state of birth and $c$ is year of birth. Thus the mortality rate gives the fraction of the cohort that died by 2007, conditional on having been alive in 1980. We also construct cumulative mortality rates by cause. These are defined as above, but where $MR_{sc}^{j}$ is mortality due to cause $j$, and the

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[18] Also, fertility effects were largest among blacks and low skilled whites (Bitler and Schmidt, 2012). Since we omit blacks from our sample, this further reduces concerns about fertility changes in young adulthood driving later health for men.

numerator is all deaths due to cause *j* over the period.

The data for our analysis come from two sources. First, we use data from the IPUMS microdata 5% samples of the 1980 Census (Ruggles et al., 2004) to construct the denominators in the mortality rate measures as well as the birth state-cohort rates of college completion and veteran status. We restrict our sample to men born between 1942 and 1953. These are the years for which both inductions and enrollments are available at the state level, which are the two components of our induction risk measures. Finally, we omit non-white men from our sample because they may have been less able to avoid the draft by enrolling in college.[19]

Our second source is the Vital Statistics mortality data for the period 1981 to 2007, which contains observations on all deaths in the United States, at the annual level. We use these data to construct the numerators in our mortality rate measures, after applying the same sample restrictions applied to the Census data. We match numbers of deaths to the appropriate state-cohort information using year and state of birth. We also have information on the primary (or underlying) cause of death, and we use this to construct cause-specific mortality rates.

Our primary measure of educational attainment is a dummy variable for college completion. To inform this choice, we explored the impact of our instruments on educational attainment and confirmed that higher national and state-level induction risk increased male educational attainment at all post-secondary levels, including completion. We also experimented with alternative specifications using years of schooling and years of college as our measure of educational attainment. The substance of our conclusions appears robust to the choice of education measure.[20]

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[19] MW discusses this possibility and its potential consequences in more detail.

[20] College completion has been shown to be the most accurately measured higher education outcome and to contain little measurement error overall (Black, et al., 2003). On the other hand, years of post-secondary schooling is unlikely to be plagued by error that is negatively correlated with the recorded value, as is the case with the dummy variable schooling measures (Kane, Rouse, and Staiger, 1999, and Black, et al., 2000).

Our measure of veteran status is based on veteran information in the Census. Specifically, we define a veteran in our cohorts as someone who answered affirmatively that he was a Vietnam veteran.[21] We exclude anyone from our sample that is on active duty in the military. We also exclude observations with imputed values for a number of key variables.[22]

We estimate our main regressions at the birth state by birth cohort level. We use the aggregated data for several reasons. First, our source of exogenous variation occurs at this level. Second, since we cannot observe veteran status, education, and mortality for a representative set of individuals in a single data set, it allows for construction of a birth state-cohort panel from which we can estimate our main econometric model. Finally, an individual's risk of death in a given year is low, so the fit of our model is likely better at the aggregate level than in a model estimating rare outcomes in individual level data. Our main econometric model is as follows:

(13) $$MR_{sc} = a_1 C_{sc} + a_2 V_{sc} + \alpha_4 X_{sc} + \tau trend_{sc} + \delta_s + \varepsilon_{sc}$$

where $s$ indexes state of birth and $c$ indexes birth year, and the dependent variable $MR_{sc}$ is the mortality rate per 1,000 persons as defined in (12)—either in total or by cause. The variable $V_{sc}$ is the fraction of veterans, and $C_{sc}$ is the fraction with a college degree. In addition, *trend* is a region-specific linear trend in birth cohort, $X_{sc}$ is a set of state-cohort level controls, and $\delta_s$ represents a full set of state of birth dummies. Allowing for state of birth fixed effects removes variation arising from states with persistently higher or lower than average induction rates, which may be associated with other state characteristics (e.g., industrial composition) that are correlated with mortality rates. $X_{sc}$ includes two variables to capture labor market conditions facing a cohort at the time of the college enrollment decision: (a) the employment to population ratio in the individual's state of birth

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[21] Census respondents are asked whether they are veterans, and if they answer yes, they are asked to identify a specific period of conflict. A small number of men in our cohorts report that they are veterans but not Vietnam veterans. For 1990, we define Reservists or National Guard members as non-veterans to match the veteran definition in the 1980 and 2000 Censuses. We also omit those living in group quarters.

[22] Specifically, we drop observations with imputed values for age, education, birth place, and veteran status. Our results are not sensitive to including the imputed values and those living in group quarters or on active duty in the military.

the year his cohort turned 19, and (b) the log of the number of respondents from a birth state and year cohort in the 1960 Census. Together, these approximate the changes in labor demand and labor supply which may have occurred alongside changes in state-level induction risk.[23]

Our endogenous variables, $C$ and $V$, are predicted from first stage equations that include the remaining right hand side covariates in equation (13) plus functions of *staterisk* and *nationalrisk* as defined in (10) and (11), respectively. Our main results show estimates from three different first stage specifications. In the first, *staterisk* and *nationalrisk* are entered linearly. The second and third specifications include quadratic and cubic functions of both risk measures, respectively. In results not shown, our inspection of the relationship between our identifying state risk variation and our covariates of interest (veteran status and college completion) suggests that the relationship may be non-linear, in which case including higher order risk terms is appropriate. This is an advance over MW that is especially relevant here because there has been discussion in the literature about alternative specifications in identifying the impact of education on health (Lleras-Muney, 2005; Mazumder, 2008). Estimation is done via standard linear 2SLS as well as LIML for the higher order specifications, weighted by the number of observations in each state-cohort cell.[24] We follow Bertrand et al. (2004) and cluster standard errors at the birth state level after collapsing our data to birth state-cohort cells.[25]

Descriptive statistics are shown in Table 1, which summarizes the variables used in our analysis for the sample of white men born between 1942 and 1953. The average cumulative

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[23] The literature tends to find no consistent, significant relationship between local labor market conditions and college attendance (Wozniak, 2010; Card and Lemieux, 2001). However, early labor market conditions affect longer-run labor market outcomes (Wozniak, 2010; Kahn, 2010; Oreopoulos, et al., 2012) and labor market outcomes have been linked to later adult health (Sullivan and von Wachter, 2009). We do not include fertility and marriage rates as controls since these may be endogenous to education and may therefore interfere with identification of education's total effect on mortality.

[24] See Wooldridge (2002) pp. 622-624 concerning 2SLS versus an approach with a probit first stage when the endogenous variable is a dummy variable. In some cases, the latter is more efficient but may tend to produce larger point estimates. Given our concerns about possible upward bias, we implement 2SLS estimation.

[25] Because our mortality measures combine Census data with data on the universe of mortality outcomes, we do not further correct our standard errors to account for multiple data sources, as for example, in two-sample IV.

mortality rate per 1,000 persons as defined in (12) is 138.58. Table 1 also gives mortality rates by cause-of-death for ten important causes for these cohorts, in order of prevalence. The most common causes of death were cancers (36.5 per 1,000 white men) and heart disease (35.7/1,000). External causes (accidental injury, suicide, and homicide) accounted for 23.0 deaths per 1,000 men. Figure 2 shows the fraction of deaths attributable to common causes by age for our cohorts. For younger men, external causes like accidental injury and suicide are the leading causes of death, but by their forties cancer and heart disease are the leading causes.

## V. Results

### A. First Stage Results

Table 2 presents results from estimating the first stage, with linear, quadratic, and cubic specifications of the risk measures. For transparency, we estimate two first stage equations for each specification—predicting college graduation and veteran status separately—although 2SLS estimates these equations jointly. Consistent with the manner in which 2SLS identifies endogenous variables, both equations include national and state cohort risk as identifying variables. We also report the Cragg and Donald (1993) $F$-statistic for identifiability developed in 3.A, the Angrist-Pischke $F$-statistics, and the traditional single equation first stage $F$-statistics.

The first two columns show results from the linear specification. A 10 percentage point increase in national cohort risk (roughly the entire range of this variable) increased the percent with a college degree by 9.4 percentage points. In other words, cohorts with higher national risk were more likely to graduate college.[26] Coefficients from the quadratic and cubic specifications also indicate an overall positive relationship between higher national risk and college graduation over the

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[26] The comparable estimate from Table 1 in Card and Lemieux (2001) shows a 4.6 percentage point increase. Our estimate is larger because we restrict the sample to whites. When we do not make this restriction, our estimates are very close to those in Card and Lemieux.

range of the risk variable. On the other hand, college graduation is negatively related to state cohort risk when national induction risk is included, particularly in the linear and quadratic specifications. As discussed in MW, this may be an artifact of the high correlation (collinearity) between national and state level risk. Across specifications, the *F*-statistics suggest both that we meet the identification assumptions in our model (implied by the Cragg-Donald statistics) and that this first stage has substantial power (implied by the Angrist-Pischke *F* statistics).[27]

The first stage estimates with veteran status as the dependent variable show that veteran status positively varies with both national and state cohort risk. This is reassuring since higher rates of induction risk at both the state and national level should lead more young men to go to war. We interpret the fact that the coefficient on national risk exceeds the one on state risk in the veteran equation to mean that the time series variation in draft risk generated by the massive fluctuation in military manpower demands is responsible for more of the variation in veteran status than are the differences in induction risk across states.

## B. OLS and 2SLS Effects of College Education on Total Mortality

Table 3 presents our OLS and 2SLS estimates of the effect of college graduation on cumulative mortality (equation 13). For the IV results, we show specifications in which the induction risk variables are linear, quadratic, and cubic. For the quadratic and cubic specifications the model is over-identified, so we can estimate the model using both 2SLS and limited-information maximum likelihood (LIML). We do this to further investigate the validity of our instruments—LIML is less precise than 2SLS but is also less biased in the presence of confounding variables. If the coefficients from the two approaches are similar, this is evidence that the bias in 2SLS is small (Angrist and Pischke 2009).

The OLS coefficient for college graduation is -101.97. This indicates that increasing college

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[27] Reduced form results available upon request.

completion rates from the level of the state with the lowest rates to that with the highest (a 42 percentage point change) is associated with 42.8 fewer deaths per 1,000 men. This is consistent with the well-documented educational gradient in most health outcomes. Veteran status is also negatively associated with mortality for these cohorts, but it is less clear what we should expect for the sign on this coefficient, even in our OLS estimates. There are two reasons for this. First, we observe mortality conditional on surviving to 1980. Veteran status may increase mortality at young ages, both via combat injuries and death but also through risky behavior (Hearst et al., 1986). Less is known about the relationship between veteran status and health in the longer term, and the existing studies find mixed evidence, as noted above.

Table 3, Column [2] shows the point estimates of the causal effect of college graduation and veteran status on mortality using a linear specification for the instruments. The results are remarkably similar to the OLS estimates, though they are less precise. With the quadratic specification, the estimated effect of college graduation is substantially larger and is statistically significant; with the cubic specification the point estimates are again very close to OLS and are statistically significant. In columns [3] through [6], the 2SLS and LIML results are nearly identical, further supporting our identification strategy. Taken together, the 2SLS results imply a large causal role for both college graduation and veteran status in reducing cumulative mortality for these cohorts. The magnitude of our estimated impacts is also economically significant. For example, the estimate from Table 3, Column [5], implies that increasing college completion rates from the level of the state with the lowest rates to that with the highest (a 42 percentage point change) leads to 39.5 fewer deaths per 1,000 men.

Table 3 shows the impact of high college completion rates on later mortality is robust to three specifications of the first stage equation. We have examined robustness to other alternative specifications. In results not shown but available upon request, we substitute a common quadratic

time trend for the linear birth region trends. Results are very similar. We have also verified that our estimates are robust to excluding the non-linear state-cohort varying controls (employment to population ratio and log cohort size).

A final check is shown in Appendix Table 1. Here we estimate our baseline OLS specification on subgroups of the sample defined by birth year. The "late" cohorts, those born 1951-1953, are somewhat different from the "early" cohorts, those born 1942-1944, and middle cohorts in that they were partially exposed to the draft lottery and had access to more generous GI Bill benefits upon release. One might be concerned that the late cohorts drive our results. If so, this might raise questions about our identification strategy. We do not have the first stage power to estimate our 2SLS specifications for a subset of only three cohorts.[28] However, the late cohorts are unlikely to contribute much to our estimates of the negative impact of college on mortality because the OLS results show no college completion health gradient for them. This is likely because these cohorts have not yet reached the ages where mortality is due to causes for which college completion has a greater effect (something we show in more detail later). We therefore view our main results as robust to concerns about differential policy treatment across cohorts.

Returning to our main analysis, one might wonder whether the effect of education on mortality changes over time, since the causes of death vary considerably over the period, as Figure 2 shows. In Table 4, we show OLS and IV estimates for the effect of college completion rates on mortality by decade. In these and most remaining results, we show results for the cubic 2SLS specification only.[29] For each decade, this measure is the total number of deaths over the decade (1981-1990, 1991-2000, 2001-2007) divided by the cohort size in the beginning of the decade (taken

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[28] We generally lose first stage power when we drop more than two cohorts from the analysis.
[29] We choose to show the cubic specification because the estimates are generally more precise, but results using lower order polynomial functional forms for the first stage equations are qualitatively similar. De Walque (2007) uses a non-linear specification of induction risk to instrument for both education and veteran status. The 2SLS and LIML results for decadal mortality are similar using the cubic functional form (though more so for 1980 and 2000 than for 1990), which again suggests that the bias from using 2SLS is small.

from the 1980, 1990, and 2000 Censuses, respectively). These shorter-term mortality rates allow us to determine whether the role of education changes as the likely reasons for mortality evolve.[30]

For each decade, college graduation and veteran status are negatively associated with mortality. For the 1980s (where the first stage is strongest), IV results are again very similar to OLS. The estimated effect of increased college attainment on mortality during the 1980s is -24.38; increasing college education from the level of the least educated birth state-cohort to the highest would decrease mortality by 10.2 per 1,000 men (or 40% relative to the mean). For the 1990s and 2000s, the IV estimates are larger than the OLS estimates and indicate that moving from the lowest-education birth state-cohort to the highest would decrease mortality by 61% and 57% relative to the mean, respectively. Results for these decades are less precise, however, which may be driven by the weaker first stage for college graduation (first-stage $F$-statistics are reported in the table). So while we interpret these results with caution, there is little evidence that the negative effect of college education on total mortality is confined to a particular decade. Veteran status has a negative and statistically significant negative effect on mortality in all periods.

**C. OLS and 2SLS Effects of College Education on Mortality by Cause of Death**

Table 5 shows the effect of state-cohort college graduation rates on leading causes of death for men in these cohorts. Causes are shown in descending order of prevalence between 1980 and 2007. OLS results are in Panel A and 2SLS results are in Panel B. Again, all specifications include birth region trends and a control for veteran status. For cancer and heart disease—the two leading causes of death—the point estimates on college graduate are negative and statistically significant for both OLS and IV and are larger for IV. Moving from the lowest-education birth state-cohort to the

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[30] Ideally, we would construct mortality rates by age, rather than by decade (for example, rates for men in their forties). However, to construct the denominators for the mortality rates, we would need to know the number alive at certain ages, by birth state. We would also need the average education level and veteran status for the cell. These cannot be constructed from the Census since it is only conducted every 10 years, and the SEER population counts are not available by state of birth and do not have information on education and veteran status.

highest would reduce cancer deaths by 25.9 per 1,000 (71% relative to the mean) and heart disease deaths by 18.9 (42% relative to the mean). The effect of veteran status on mortality due to these causes is also negative, though not statistically significant for heart disease.

In results not shown here, we estimated the model for deaths by specific type of cancer. Lung cancer accounts for nearly one-third of the cancer deaths in our sample; the next leading specific cause is colon cancer, which accounts for about 10% of cancer deaths. The IV estimate of the effect of a college degree on lung cancer is -35.59 (s.e. = 5.09), suggesting that 57% of the decline in total cancer deaths is due to lung cancer. This is greater than the decline that would be observed if college education had an equal effect on deaths from all cancers. That college education has a particularly big effect on death from lung cancer is not surprising, as De Walque (2007) and Grimard and Parent (2007) both find that education has a large and statistically significant negative effect on smoking. Cigarette smoking is known to be a leading cause of both lung cancer and cardiovascular (heart) disease (Chaloupka and Warner, 2000).

College education has a negative and statistically significant effect on mortality from three other causes: diabetes, stroke, and chronic low respiratory conditions (CLRCs). In the IV specifications, a college degree decreases deaths per 1,000 white men from both diabetes and stroke by 7 and from CLRCs by 9. The latter effect is especially large and is also likely related to the effect of college on the likelihood of smoking, since smoking is a known cause of CLRCs like chronic bronchitis and emphysema (Chaloupka and Warner, 2000). Veteran status decreases deaths due to chronic low respiratory conditions and stroke, while its effect on diabetes is small and statistically insignificant at the 5 percent level.

The OLS results show a negative association between a college degree and death by two external causes—accidental injury and homicide. But for both of these, the IV coefficient is smaller and statistically insignificant. There is also no evidence that college decreases deaths by suicide.

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Veteran status, however, does decrease deaths due to accidents and suicide, conditional on surviving to 1980.

Finally, for infectious and parasitic diseases and liver diseases, the point estimate for college graduate is negative in the OLS specification but positive and statistically significant in the 2SLS specifications. For infectious and parasitic diseases, 78% of deaths for these cohorts were due to HIV alone, and HIV deaths are driving the positive coefficient. There are several ways in which college-going might increase one's risk of contracting HIV during this period. For example, college-going is associated with living in an urban area and engaging in same-sex relationships, which are significant risk factors for contracting HIV (Shilts 1987).[31] Liver diseases, on the other hand, include cirrhosis which can be caused by excessive alcohol consumption—48% of deaths to cirrhosis in 2007 were alcohol-related (Yoon and Yi, 2010). If a college degree increases this behavior, graduates may be more at risk for liver diseases.[32] We investigate the relationship between college education and alcohol consumption in the next section. The effect of veteran status on deaths by infectious diseases and liver disease is negative.

The results in Tables 3, 4, and 5 indicate that college education has a negative effect on total mortality; that the effect was not confined to any particular decade; and that deaths due to heart disease and cancer are particularly affected. A college education has a particularly strong negative effect on deaths from causes related to smoking.

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[31] About half of all HIV cases in the U.S. are for men who have sex with men (Center for Disease Control 1997). Lambert et al. (2006) find that upper-class college students had more positive attitudes toward gay and lesbians than underclassmen, suggesting that college increases acceptance of homosexuality. Smith (1991) found that among respondents to the General Social Survey in 1989, college graduates were more likely to have engaged in risky sexual behaviors than less educated groups, although shares in the highest risk group were similar across education levels. Smith also finds that risky behavior is significantly higher among younger and more urban individuals, who are more likely to be college educated.

[32] Competing risks may also explain these positive coefficients, (as well as the coefficients on other specific causes of death). Since the first two columns in the table indicate that college prevented some men from dying of cancer and heart disease, these men may now be more susceptible to death by other causes (Honoré and Lleras-Muney, 2006).

**VI. Potential Mechanisms**

A number of potential mechanisms might explain the negative effect of college completion on cumulative mortality and the heterogeneity in the effects across specific causes of death. For example, if the college educated are less likely to smoke and more likely to drink, this may explain the negative coefficient on cancer and the positive coefficient on liver disease, respectively. Mokdad et al. (2004) find that behavioral factors such as smoking, obesity, and excessive alcohol consumption represent almost half of the "actual" causes of death in the United States in 2000. Cutler and Lleras-Muney (2010) describe the role of cognition, resources, prices, and health behaviors in mediating the relationship between education and health for nationally-representative populations in the US and UK. In this section, we focus on our cohort in particular and explore whether we can find evidence supporting or refuting potential mechanisms that would explain the cause-of-death mortality results.

First, using data from the Census, we examine the causal relationship between education and earnings or wages for our cohort. Second, we assess the impact on access to health insurance in the 2008-2010 American Community Survey (ACS) and 1980 National Health Interview Survey (NHIS) Health Insurance Supplement. Finally, using data from the 1985 National Health Interview Survey (NHIS), Health Promotion and Disease Prevention (HPDP) Supplement, we consider acute health outcomes, including diabetes, hypertension, and BMI, and health behaviors, including smoking, exercise, and alcohol consumption.

**A. Earnings and Wages**

An extensive literature demonstrates that increased schooling is causally related to higher wages and earnings (Card, 1999). To the extent that higher income enables individuals to purchase better health care or acquire a healthier lifestyle, it represents a potentially important mechanism in explaining the relationship between education and mortality. Cutler and Lleras-Muney (2010)

demonstrate that family income is an important mediating factor between education and health. However, health may directly affect earnings potential, so these results must be interpreted only as suggestive evidence of a possible mechanism. We examine this potential mechanism by estimating the effect of college completion on wages and earnings for cohorts who came of age during the Vietnam War. Using data from the 1980 Census, Table 6 presents OLS and IV estimates based on our preferred cubic first-stage specification.[33]

The OLS estimates for log earnings indicate that a cohort moving from a 0 to 100 percent college completion rate is associated with an increase of 43 percent in earnings for that cohort. Thus, increasing college completion rates from the level of the state with the lowest rates to that with the highest (a 42 percentage point change) is associated with 18 percent higher wages for the more educated cohort. The IV estimate suggests that college completion causes a significant increase in earnings of approximately 78 percent. The OLS estimate for hourly wages indicates that moving from a 0 to 100 percent college completion rate is associated with an increase in hourly wages of 23 percent. The IV specifications yield large and significant impacts indicating that universal college completion would raise wages by about 40 percent. As with the earnings results, we cannot reject that the IV estimates are significantly different from the OLS estimates.

These findings suggest that wages and earnings may be an important mediating factor in the effect of education on health. If health is a normal good, we expect that higher incomes would lead to lower mortality rates. However, quantifying the causal effect of income on mortality is difficult due to the endogeneity of income (Smith, 1999). International studies find no difference in mortality rates by average income differences between countries (e.g., Wilkenson, 1996). So while it is plausible that the effect of college graduate rates on mortality rates operates through earnings and wages, we explore other potential mechanisms as well.

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[33] All of the regressions are restricted to full-time workers. Hourly wages are further truncated to remove the bottom and top 0.5 percent in order to reduce measurement error.

**B. Health Insurance Measures in the American Community Survey**

We consider the role of health insurance as an additional potential mechanism through which education might reduce mortality. Earning a college degree qualifies individuals for jobs that are more likely to include benefits such as employer-provided health insurance (Hipple and Stewart, 1996). Health insurance has been linked to better access to preventative care, which should lead to better health and lower mortality, all else equal (Miller, 2012). However, Finkelstein and McKnight (2008) find that the introduction of Medicare, i.e., nearly universal access to health insurance after age 65, had very little impact on mortality rates. When considering health insurance status among those under age 65, those with poorer health or who are at higher risk of needing expensive medical services might be more inclined to purchase health insurance, complicating the measurement of a causal effect of health insurance on mortality. Here we explore whether college attainment is associated with a higher probability of being covered by health insurance in order to assess whether access to health insurance is a channel through which education might improve health.

Our primary data on insurance comes from the 2008-2010 ACS. In Section VI.C, below, we present parallel results using the 1980 NHIS Health Insurance Supplement which confirm these main findings. We first explore whether the individual has any health insurance; we then consider private and public health insurance coverage separately. Private health insurance includes both employer-provided health insurance and plans that individuals purchase in the private market. We anticipate that having a college degree is associated with a higher probability of having private health insurance. Our public health insurance measure includes Medicaid, Medicare and disability, and the veteran's health insurance program CHAMPUS. We therefore anticipate that veterans are much more likely to have public health insurance coverage. We expect that both having a college degree and being a veteran are associated with a higher probability of having any health insurance. Note that in the ACS individuals can be classified as having both private and public health insurance.

The main results are reported in Table 7. The IV estimates suggest that increasing college

completion rates from the level of the state with the lowest rates to that with the highest (a 42

percentage point change) leads to a 5.7 percentage point higher rate of having any health insurance

coverage (which is about 6.1 percent of the mean of 92.4). Next, we consider private coverage.

While the coefficient on college graduate is only significant in the OLS specification, the magnitude

is similar in both OLS and IV and suggests that a cohort having a 100 percent college graduation

rate versus 0 percent is associated with a 10-16 percentage point higher probability of individuals in

that cohort having private health insurance coverage. Finally, we see that rates of college

completion are unrelated to public coverage rates, although the OLS relationship is negative.

Veterans have higher rates of public coverage in both the OLS and IV specifications, which appears

to drive their higher rates of coverage overall.

## C. Health Insurance Measures in the National Health Interview Survey

We next present results for access to health insurance from the more detailed 1980 National

Health Interview Survey (NHIS) Health Insurance Supplement. The 1980 NHIS is chosen because

this is the baseline year from our mortality analysis. Estimation using the NHIS data necessitates

some changes in our specifications since we cannot observe state of birth or state of residence (but

we do observe the Census region of residence) in the public use versions of the NHIS. We

therefore estimate our insurance models using individual-level data, rather than data aggregated to

the birth state-birth year cohort level. Our econometric model for the NHIS data is the following:

(14) $$insurance_{irc} = a_1 C_{irc} + a_2 V_{irc} + \tau trend_{rc} + \delta_r + \varepsilon_{irc}$$

where $i$ indexes individuals, $r$ indexes region of residence, and $c$ indexes birth year. Here the

dependent variable is an indicator for either any insurance coverage, private coverage, or public

coverage. The variable $V$ is an indicator for whether the individual is a veteran, and $C$ is an

indicator for whether the individual has a college degree. In addition, *trend* is a region-specific linear

trend in birth cohort, and $\delta_r$ represents region of residence dummies. Note that because of the lack of state identifiers in the NHIS, we cannot use the set of state-cohort level controls described above (the employment to population ratio and the cohort size).

We do not present results using instrumental variables with the NHIS data because of power issues. The 1980 Census sample is over 200 times larger than the 1980 wave of the NHIS. Although theoretically one could merge together data from three decades of the NHIS using restricted-access data that includes state of birth, even then the sample size is not sufficient to have a strong first stage.[34] Thus, using the NHIS data, we present results from linear probability model regressions, with standard errors clustered by region of residence. We also report the sample size for each specification and the mean of the dependent variable.

The results are reported in the top of Table 8. We see that the OLS estimates for health insurance coverage using the 1980 NHIS are very similar to those from the more recent ACS data, with the exception of the probability of having public health insurance coverage. We find that being a college graduate is associated with a 9.4 percentage point higher probability of having any health insurance, which is approximately 10 percent of the mean of 89 percent. Similarly, college graduates are 11.1 percentage points more likely to have private health insurance coverage than non-college graduates.

When we consider public health insurance coverage, we see some differences. In the NHIS, individuals can be classified as having public coverage only if they do not have any private health insurance coverage. Those that do not have private insurance or Medicare are asked to state the reason, to which they may respond, among other things, that they receive services through Medicaid, welfare, or CHAMPUS. Only 2 percent of the sample report having any public insurance coverage

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[34] Results using pooled data from the 1997-2009 NHIS are available upon request, but show a weak first-stage and no statistically significant coefficients in 2SLS. The power issues arise because we are limited to looking at men from only 11 birth year cohorts. Other studies using the NHIS (e.g. DeWalque 2007) use larger samples.

in the NHIS. The restrictiveness of the definition contributes to the differences between this figure and the one based on the ACS, where 17.9 percent have public coverage. Our cohort is also younger in 1980, so these men are much less likely to be disabled and qualify for Medicare than they will be at ages 55-64. Indeed, when we look at individuals who are ages 55-64 in 1980 (born between 1916-1925), we see that 11.9 percent have public health insurance coverage, which is much closer to the ACS mean. Although the estimated coefficient on college graduate is much larger in the ACS data, the effect size relative to the sample mean is quite similar between the two datasets. In the NHIS data, we do not see any association between veteran's status and public health insurance coverage, while the effect is positive and significant in the ACS. Taken together, these results confirm that access to health insurance is another potential mechanism through which college attainment could improve health and reduce mortality.

**D. Acute Health Outcomes and Health Behaviors in the National Health Interview Survey**

Our final set of results considers how acute health conditions and health behaviors are associated with college attainment and veteran status for our cohort of men. Here we use the 1985 NHIS Health Promotion and Disease Prevention (HPDP) Supplement, which allows us to measure outcomes for our particular cohort as close to the 1980 baseline year as possible. We again estimate equation (14) at the individual-level, but where the dependent variable is a measure of either a health outcome or a health behavior. We report estimated coefficients on college graduate and veteran status in the bottom of Table 8.

We first consider several acute health measures that could help to explain the association between education and mortality. First, we find that college graduates have significantly lower body mass index (BMI) and are significantly less likely to be obese. We find that college graduates are 3.9 percentage points more likely to report having been under a lot of stress in the past two weeks relative to non-college graduates. This result is in contrast to the finding in Table 5 that college

graduates are significantly less likely to die from heart disease. However, it may be that higher stress levels among college graduates are due to reporting differences across education groups, rather than true underlying stress levels. We do not find any relationship between being a college graduate and the probability of missing work or having restricted activity or bed days in the past two weeks.

The bottom of Table 8 explores a series of health behaviors that Cutler and Lleras-Muney (2010) identify as important mediating factors between education and health. Consistent with prior literature, we find that college graduates in our cohorts are less likely to smoke while veterans are more likely to smoke. Similarly, we see that college graduates are more likely to exercise. Therefore, we observe that college graduates are exhibiting healthier behaviors than non-college graduates, while veterans are more likely to smoke. The evidence on heavy drinking is mixed. According to Table 8, college graduates report similar levels of heavy drinking to less educated respondents in two of the three categories, but lower levels of heavy drinking in the third category. This behavior alone is therefore unlikely to explain the increase in mortality from liver disease seen in Table 5, although competing risks (and reporting differences across education groups) may explain the higher mortality rates from liver disease among college graduates. Health behaviors also cannot explain the protective effect of veteran status on mortality.

## VII. Conclusion

This paper examines the causal impact of a college education on early adult mortality. We exploit changes in national and state-level risk of induction into military service during the Vietnam conflict as a source of exogenous variation in college completion. Using Census data from 1980 to 2000 and Vital Statistics data from 1981 to 2007, we show that the magnitude of causal impacts of higher cohort college completion rates estimated by 2SLS is quite similar to the OLS gradient. In our cohorts, these estimates indicate that increasing college completion rates from the level of the birth-

state with the lowest rate to that with the highest would decrease deaths by about 39 per 1,000 men. This is equivalent to 28 percent of the mean of total mortality over our period, or a full standard deviation in birth state-cohort mortality. Alternatively, given the fraction of our sample with a college degree, our IV coefficient indicates that mortality rates over the period are about 2.21 times greater for non-college graduates than for college graduates.[35] We find large negative effects of college completion on deaths from cancer and heart disease, the leading causes of mortality among older adults. Increasing college completion rates in the lowest-education birth state-cohort to that of the highest would reduce early cancer deaths by 25.9 per 1,000 (71% relative to the mean) and early heart disease deaths by 18.9 (42% relative to the mean).

For comparison, 10-year mortality rates for 50-year old men in the U.S. are 2.6 times higher for smokers than for non-smokers (Woloshin et al. 2008).[36] We find that non-college graduates are over twice as likely to report being a smoker in 1985 relative to college graduates. Thus, smoking alone could explain a large portion of the mortality differential we find. We also shed some light on other potential mechanisms for the effect of college education on mortality by examining the causal effect of college completion on auxiliary outcomes such as health insurance and earnings, as well as the cross-sectional relationship between college completion and various health behaviors such as smoking, exercise, and obesity. Using data from the Census and the American Community Survey and employing the same IV strategy used to establish the mortality results, we show that increased college completion leads to both higher earnings and higher rates of health insurance. Using data from the National Health Interview Study, we show that college completion is also associated with less smoking and more exercise for our cohorts of interest.[37] These results suggest that the impact of

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[35] To see this, note that with 35% of the sample having a college degree and a mean mortality rate of 138.58, the coefficient implies that mortality rates would be 171.5 for non-college graduates and 77.5 for college graduates.
[36] A study of Norwegian men found that mortality by age 70 (conditional on living to age 40) was 2.9 times higher for smokers than non-smokers (Vollset et al. 2006).
[37] See Cutler and Lleras-Muney (2010) for similar results in the NHIS for a broader set of cohorts.

college completion on overall mortality may partially operate through greater financial and health resources, and that the impacts on cancer (especially lung cancer) and heart disease may be partially explained by the differences in behavior between college graduates and non-graduates.

The findings in this paper indicate that there is a large return to schooling on health at the top part of the education distribution. Given that higher education policy in recent years has been focused on increasing college completion, this represents a particularly important margin of analysis. Of course, whether these findings would point towards education subsidies depends on the presence of market failures, either in the form of externalities, credit constraints, or lack of knowledge about the health benefits of college graduation. To the extent that this paper provides useful information about the benefits of college completion in reducing adult mortality, we hope that it may address one possible source of inefficient investments in human capital.

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**Figure 1:  Birth State-Birth Year Cohort Variation in Induction Risk**

## Panel A: Induction Risk
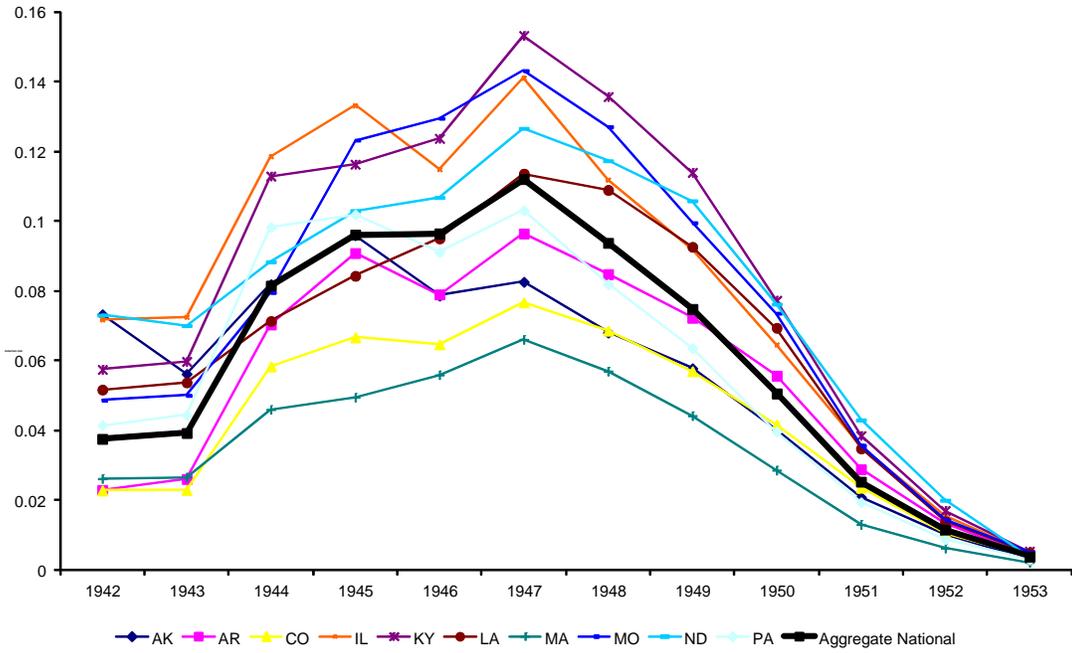


## Panel B: Residual state induction risk
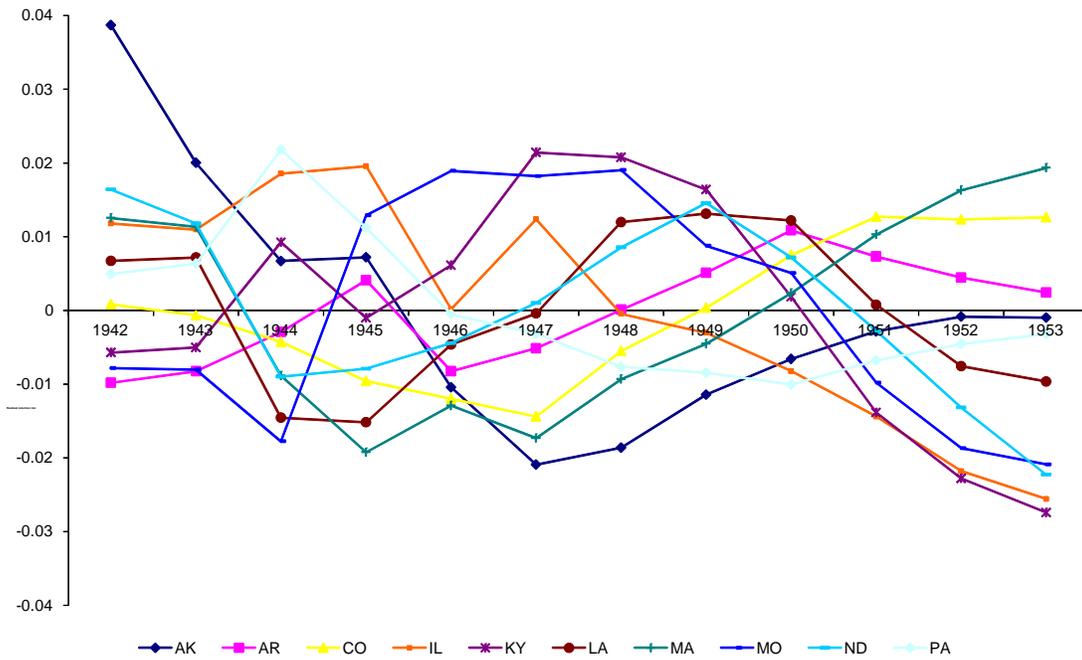
## Panel C: Residual national induction risk
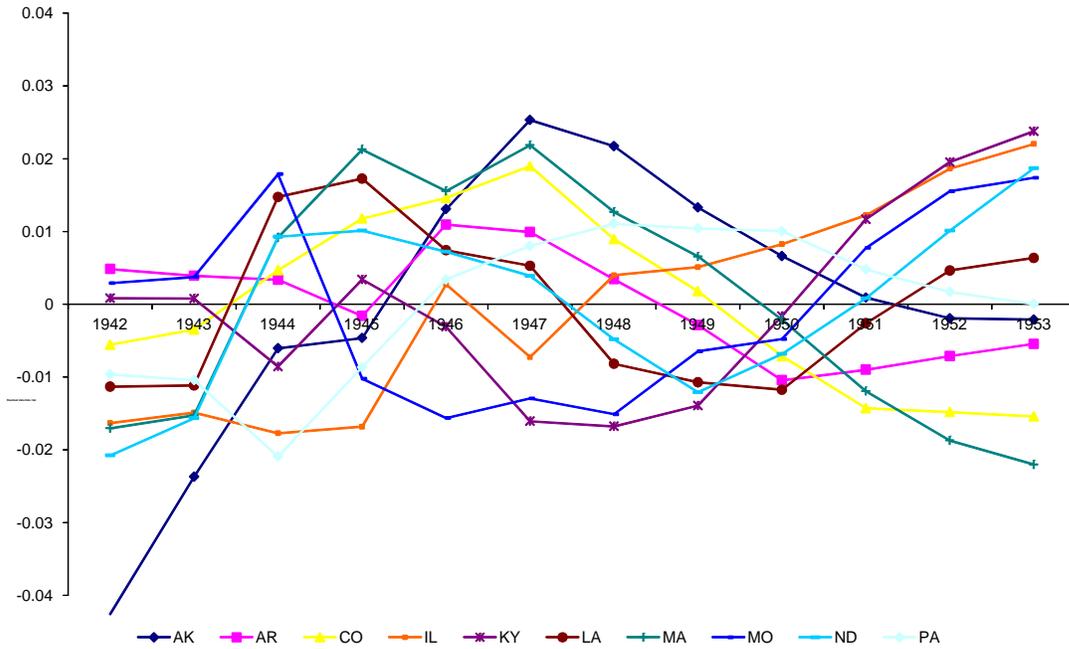


Notes:  Panel A plots state risk as defined in equation (10) for a selected set of states, as well as the aggregate national risk calculated by summing enrollments and inductions over all states.  Panel B plots residual state risk adjusted for birth state fixed effects, birth year trend, and national risk as defined by equation (11).  Panel C plots residual national risk adjusted for birth state fixed effects, birth year trend, and state risk.

**Figure 2: Fraction of Deaths Attributable to Common Causes, by Age**
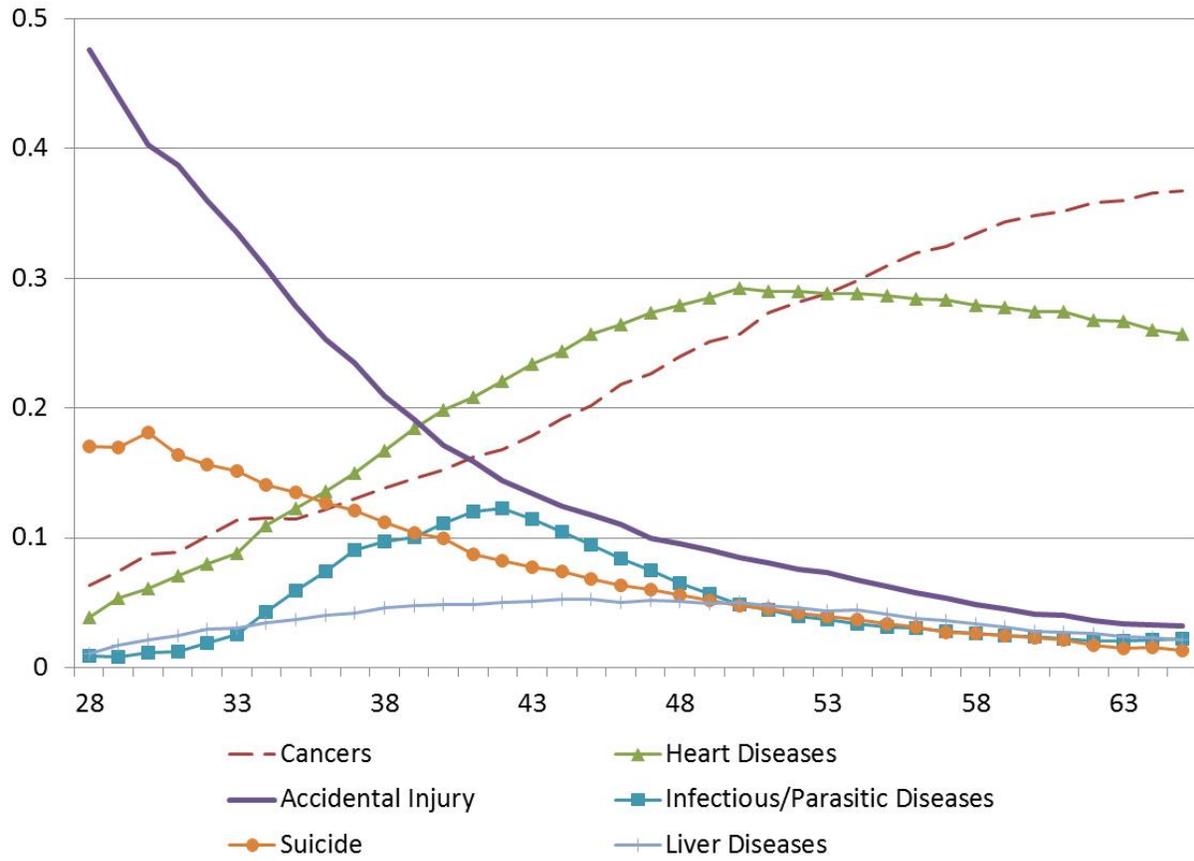


Source: Mortality Detail Files, 1981-2007.

**Table 1: Summary Statistics**

| VARIABLES | Mean | SD |
|---|---|---|
| **Census Data** | | |
| Years of Higher Education | 1.99 | 0.38 |
| College Graduate | 0.3508 | 0.0666 |
| Veteran | 0.3092 | 0.1231 |
| Individual Observations | 14,392,122 | |
| | | |
| **Mortality Data** | | |
| 1981-2007 Mortality Rates by Cause of Death: | | |
| Total | 138.58 | 38.03 |
| Cancers | 36.52 | 15.18 |
| Heart Disease | 35.73 | 13.99 |
| Accidental Injury | 13.67 | 3.49 |
| Suicide | 7.31 | 1.37 |
| Infectious and Parasitic Diseases | 7.20 | 2.41 |
| Liver Disease | 5.75 | 1.93 |
| Diabetes | 3.82 | 1.44 |
| Cerebrovascular Disease (Stroke) | 3.61 | 1.47 |
| Chronic Low Respiratory Disease | 3.21 | 2.16 |
| Homicide | 1.98 | 0.97 |
| Total Deaths | 1,994,481 | |
| State/Birth Year Cells | 600 | |

Notes: Census data are from the 5% sample of the 1980, 1990, and 2000 U. S. Census, available from IPUMS. Mortality data are from the Vital Statistics Multiple Cause of Death files from 1980 to 2007. The sample is restricted to white men born between 1942 and 1953. Veterans include any respondent that served in active duty in the Vietnam War. Respondents currently in active duty are excluded. Means are weighted by cell size. Mortality rates are deaths over the period per 1,000 population, where population is the cohort size in 1980.

**Table 2: First Stage Estimates of Effect of Induction Risk on College Completion and Veteran Rates**

| | Dependent Variable: | | | | | |
|---|---|---|---|---|---|---|
| | College Graduate | Veteran | College Graduate | Veteran | College Graduate | Veteran |
| National Induction Risk | 0.935** | 2.349** | 1.899** | 2.865** | 2.368** | 1.237* |
| | (0.091) | (0.185) | (0.263) | (0.354) | (0.437) | (0.728) |
| National Risk ^2 | | | -7.484** | -3.891* | -22.748** | 25.194** |
| | | | (1.644) | (2.068) | (5.946) | (9.238) |
| National Risk ^3 | | | | | 101.276** | -154.080** |
| | | | | | (29.076) | (42.970) |
| State Induction Risk | -0.240** | 0.768** | -0.380 | 0.653** | 0.588 | 1.171* |
| | (0.089) | (0.162) | (0.233) | (0.311) | (0.375) | (0.702) |
| State Risk ^ 2 | | | 0.770 | 0.627 | -12.254** | -5.907 |
| | | | (1.260) | (1.731) | (4.646) | (8.736) |
| State Risk ^ 3 | | | | | 54.507** | 25.502 |
| | | | | | (19.888) | (36.988) |
| Cragg-Donald F-stat | 14.23 | | 11.64 | | 13.14 | |
| [p-value] | [0.00] | | [0.00] | | [0.00] | |
| Angrist-Pischke F-Stat | 19.18 | 123.00 | 16.07 | 269.77 | 21.89 | 277.53 |
| F-Stat | 255.23 | 2154.74 | 169.47 | 1137.29 | 129.27 | 851.75 |

Notes: Standard errors are in parenthesis and are clustered by birth state. ***, **, and * indicate statistical significance at the 1, 5, and 10 percent level respectively. Specification refers to the functional form of the induction risk variables in the first stage regression. See the text for a discussion of the null hypotheses of the three F-statistics. See Table 1 notes for data sources, sample restrictions and variable definitions. Number of observations is 600 in each regression and regressions are weighted by cell size. Additional controls include birth state fixed effects, the employment to population ratio, the cohort size (derived from the 1960 Census and defined at the birth-year level), and birth-region trends.

**Table 3:  OLS and IV Estimates for the Impact of College Completion Rates on Cumulative Mortality, 1980-2007**

| | OLS [1] | Linear-2SLS [2] | Quadratic-2SLS [3] | Quadratic-LIML [4] | Cubic-2SLS [5] | Cubic-LIML [6] |
|---|---|---|---|---|---|---|
| | | | IV Specification: | | | |
| College Graduate | -101.97*** | -99.26 | -170.76*** | -172.46*** | -93.93*** | -93.53*** |
| | (19.59) | (77.55) | (38.34) | (39.22) | (25.56) | (26.69) |
| Veteran Status | -46.36*** | -42.93*** | -27.21*** | -26.82*** | -44.45*** | -44.50*** |
| | (5.14) | (16.43) | (9.67) | (9.86) | (6.81) | (7.03) |
| Observations | 600 | 600 | 600 | 600 | 600 | 600 |
| R-squared | 0.9516 | 0.9515 | 0.9505 | 0.9504 | 0.9515 | 0.9515 |

Notes:  Standard errors are in parenthesis and are clustered by birth state.  ***, **, and * indicate statistical significance at the 1, 5, and 10 percent level respectively.  See Table 1 notes for data sources, sample restrictions and variable definitions.  The IV specifications use the national and state-level induction risk to instrument for college graduate and veteran status.  The column headings indicate the functional form of the instruments in the first stage equation (linear, quadratic, or cubic) and the choice of model (two-staged least squares or limited-information maximum likelihood).  Number of observations is 600 in each regression and regressions are weighted by cell size.  Additional controls include birth state fixed effects, the employment to population ratio, the cohort size (derived from the 1960 Census), and birth region trends.

**Table 4:  OLS and IV Estimates for the Impact of College Completion Rates on Mortality by Decade**

| VARIABLES | 1981-1990 | | 1991-2000 | | 2001-2007 | |
|---|---|---|---|---|---|---|
| | OLS | IV | OLS | IV | OLS | IV |
| College Graduate | -24.51*** | -24.38*** | -20.00** | -79.46*** | -38.58*** | -100.37*** |
| | (4.56) | (6.43) | (9.10) | (30.46) | (11.26) | (37.05) |
| Veteran Status | -9.93*** | -9.33*** | -35.54*** | -27.32*** | -36.99*** | -28.80*** |
| | (1.10) | (1.36) | (2.20) | (4.49) | (2.53) | (6.06) |
| Cragg-Donald F-Stat | 13.14 | | 5.46 | | 3.60 | |
| [p-value] | [0.00] | | [0.00] | | [0.00] | |
| A-P F-Stat for: | | | | | | |
|    College Grad | 21.89 | | 8.91 | | 5.81 | |
|    Veteran | 277.53 | | 100.11 | | 101.06 | |
| F-Stat for: | | | | | | |
|    College Grad | 129.27 | | 38.51 | | 50.81 | |
|    Veteran | 851.75 | | 1002.92 | | 977.61 | |
| Mean Mortality Rate | 25.46 | | 54.88 | | 73.33 | |
| Total Deaths | 366,365 | | 727,108 | | 901,008 | |

Notes:  Standard errors are in parenthesis and are clustered by birth state.  ***, **, and * indicate statistical significance at the 1, 5, and 10 percent level respectively.  See the text for a discussion of the null hypotheses of the three F-statistics.  See Table 1 notes for data sources, sample restrictions and variable definitions.  The IV specifications use a third-order polynomial in the national and state-level induction risks to instrument for college graduate and veteran status using a 2SLS model.  Number of observations is 600 in each regression and regressions are weighted by cell size.  Additional controls include birth state fixed effects, the employment to population ratio, the cohort size (derived from the 1960 Census), and birth region trends.

**Table 5:  OLS and IV Estimates for the Impact of College Completion Rates on Mortality, by Leading Causes of Death**

| | Cancers | Heart Disease | Accidental Injury | Suicide | Infectious/ Parasitic | Liver Disease | Diabetes | Stroke | Chronic Low Resp. | Homicide |
|---|---|---|---|---|---|---|---|---|---|---|
| **Panel A:  OLS** | | | | | | | | | | |
| College Graduate | -38.34*** | -32.29*** | -5.21** | -0.68 | -2.62 | -1.19 | -2.41 | -2.98*** | -5.33*** | -1.62** |
| | (6.57) | (5.95) | (2.11) | (1.47) | (2.46) | (1.54) | (1.52) | (0.86) | (1.56) | (0.63) |
| Veteran Status | -15.88*** | -10.16*** | -3.20*** | -1.71*** | -0.980** | -1.250*** | -0.730** | -1.79*** | -4.00*** | 0.04 |
| | (1.69) | (1.81) | (0.60) | (0.46) | (0.45) | (0.41) | (0.36) | (0.28) | (0.36) | (0.16) |
| **Panel B:  IV** | | | | | | | | | | |
| College Graduate | -61.90*** | -45.07*** | -0.71 | 4.24 | 14.75*** | 12.20*** | -7.04*** | -6.61*** | -9.11*** | -0.10 |
| | (9.18) | (11.32) | (4.92) | (3.02) | (5.19) | (4.16) | (2.21) | (1.94) | (2.65) | (1.49) |
| Veteran Status | -9.72*** | -6.64** | -3.81*** | -2.74*** | -4.56*** | -4.04*** | 0.35 | -0.88* | -3.05*** | -0.22 |
| | (2.45) | (3.20) | (1.15) | (0.80) | (1.18) | (0.94) | (0.48) | (0.45) | (0.56) | (0.35) |

Notes:  Standard errors are in parenthesis and are clustered by birth state.  ***, **, and * indicate statistical significance at the 1, 5, and 10 percent level respectively.  See Table 1 notes for data sources, sample restrictions and variable definitions.  The IV specifications use a third-order polynomial in the national and state-level induction risks to instrument for college graduate and veteran status using a 2SLS model.  Number of observations is 600 in each regression and regressions are weighted by cell size.  Additional controls include birth state fixed effects, the employment to population ratio, the cohort size (derived from the 1960 Census), and birth region trends.

**Table 6:  The Causal Impact of College Completion Rates on Earnings and Wages**

| VARIABLES | Log Earnings | | Log Wages | |
|---|---|---|---|---|
| | OLS | IV | OLS | IV |
| College Graduate | 0.427*** | 0.780*** | 0.230*** | 0.393*** |
| | (0.071) | (0.157) | (0.058) | (0.100) |
| Veteran Status | 0.170*** | 0.0701* | 0.125*** | 0.0753*** |
| | (0.020) | (0.041) | (0.014) | (0.029) |
| Mean Earnings/Wages | 9.542 | | 1.964 | |

Notes: Standard errors are in parenthesis and are clustered by birth state.  ***, **, and * indicate statistical significance at the 1, 5, and 10 percent level respectively. Data are birth state-cohort cell averages from white men in the 1980 Census IPUMS extracts, born between 1942 and 1953. First stage specifications and estimating sample are identical to those in Table 2. Dependent variable is log real earnings or log real wages. Underlying microdata sample is therefore restricted to those with valid earnings in 1980. We truncate the top and bottom 1 percent of the hourly wage distribution to deal with measurement error in hours and weeks worked. Specifications are otherwise identical to those in Table 3. Number of observations is 600 in each regression and regressions are weighted by cell size.

### Table 7: College Completion Rates and Health Insurance Access

|  | Any Insurance | Private Coverage | Public Coverage |
|---|---|---|---|
| **Panel A: OLS** | | | |
| **College Graduate** | 0.093*** | 0.163*** | -0.083** |
|  | (0.025) | (0.028) | (0.035) |
| **Veteran Status** | 0.049*** | -0.038* | 0.241*** |
|  | (0.011) | (0.021) | (0.028) |
| **Panel B: IV** | | | |
| **College Graduate** | 0.135* | 0.107 | 0.082 |
|  | (0.072) | (0.108) | (0.135) |
| **Veteran Status** | 0.074*** | -0.027 | 0.306*** |
|  | (0.017) | (0.020) | (0.023) |
| **Mean of dep. variable** | 92.4% | 83.7% | 17.9% |

Notes: Standard errors are in parentheses and are clustered by birth state. ***, **, and * indicate statistical significance at the 1, 5, and 10 percent level, respectively. Data are from ACS survey years 2008-2010. Samples restricted to white, male respondents born between 1942 and 1953, and aged 64 or younger at the time of the survey. Each "College Graduate" and "Veteran" pair are coefficients from a single estimation using the cubic OLS and IV specifications from Table 3. C-D F statistic is 0.72 (p-value of 0.49). First stage standard F statistics are 13.6 and 11.2 for college graduate and veteran status, respectively; A-P F statistics are 13.3 and 146.1 for college graduate and veteran status, respectively.

## Table 8: Acute Health Outcomes and Health Behaviors

| *Dependent Variable* | N | Mean / Percent | College Graduate | | Veteran | |
|---|---|---|---|---|---|---|
| **1980 Health Insurance Supplement:** | | | | | | |
| Any Health Insurance | 6,292 | 89.0% | 0.094*** | (0.010) | 0.036** | (0.009) |
| Private Coverage | 6,292 | 86.8% | 0.111*** | (0.010) | 0.035*** | (0.005) |
| Public Coverage | 6,292 | 2.0% | -0.019*** | (0.002) | -0.001 | (0.004) |
| **1985 NHIS HPDP Supplement:** | | | | | | |
| Body Mass Index (BMI) | 3,069 | 25.82 | -1.081** | (0.294) | 0.084 | (0.093) |
| Obese (BMI ≥30) | 3,069 | 10.9% | -0.066* | (0.022) | -0.011 | (0.008) |
| Ever told had: | | | | | | |
|    Hypertension | 3,031 | 18.7% | -0.017 | (0.012) | 0.006 | (0.007) |
|    High cholesterol | 3,035 | 4.5% | 0.013 | (0.014) | -0.015** | (0.004) |
|    Heart Condition | 3,033 | 3.2% | -0.003 | (0.006) | 0.002 | (0.011) |
|    Stroke | 3,035 | 0.5% | -0.004* | (0.001) | 0.003 | (0.002) |
| Has Diabetes | 3,032 | 1.3% | 0.002 | (0.002) | -0.006 | (0.003) |
| Any Restricted Days in Past 2 Wks | 3,069 | 8.0% | -0.008 | (0.009) | 0.013 | (0.013) |
| Any Work Loss Days in Past 2 Wks | 3,069 | 4.7% | 0.000 | (0.006) | 0.016 | (0.015) |
| Any Bed Days in Past 2 Wks | 3,069 | 4.2% | -0.001 | (0.006) | 0.010 | (0.009) |
| A Lot of Stress in Past 2 Weeks | 3,018 | 22.7% | 0.039* | (0.012) | -0.010 | (0.017) |
| Ever Smoked 100 Cigarettes | 3,030 | 67.2% | -0.200*** | (0.008) | 0.074*** | (0.012) |
| Smoke Now | 3,006 | 37.6% | -0.213*** | (0.016) | 0.062** | (0.013) |
| Any days in Past Year 5+ Drinks | 2,977 | 44.1% | -0.030 | (0.011) | 0.071** | (0.015) |
| 25+ days in Past Year 5+ Drinks | 2,977 | 14.0% | -0.082** | (0.023) | 0.005 | (0.009) |
| Any Drunk Driving in Past Year | 2,996 | 20.9% | -0.008 | (0.014) | 0.014 | (0.023) |
| Exercise or play sports regularly | 3,036 | 45.0% | 0.249*** | (0.019) | 0.011 | (0.014) |

Notes: The sample is restricted to white, male respondents born between 1942 and 1953. Each "College graduate" and "Veteran" pair are coefficients from a single regression. Specifications are estimated using microdata at the individual-level and include region-specific linear trend in birth cohort and region of residence fixed effects. Standard errors are clustered by the four Census regions of residence. ***, **, and * indicate statistical significance at the 1, 5, and 10 percent level respectively. Health insurance information is from the 1980 NHIS Health Insurance Supplement. For this sample 30.1% are college graduates and 41.7% are veterans. For public coverage, individuals are reported as having Medicare, or could report receiving services through Medicaid/Welfare or CHAMPUS. The remaining rows report data from the 1985 NHIS HPDP Supplement. With the exception of BMI, all dependent variables are dichotomous and estimates are from a linear probability model. For this sample, 32.6% are college graduates and 38.1% are veterans.

**Appendix Table 1: OLS Estimates for the Impact of College Completion Rates on Cumulative Mortality, 1980-2007, for Early and Late Cohorts**

| | Baseline OLS | | OLS - Early Cohorts | | OLS - Late Cohorts | |
|---|---|---|---|---|---|---|
| | [1] | [2] | [3] | [4] | [5] | [6] |
| College Graduate | -101.97*** | -115.40*** | -101.88 | -146.15** | 1.42 | -3.76 |
| | (19.59) | (19.50) | (65.98) | (66.38) | (23.46) | (23.62) |
| Veteran Status | -46.36*** | -49.62*** | -73.29 | -97.98 | -44.85 | -70.11** |
| | (5.14) | (5.25) | (63.50) | (66.05) | (32.35) | (31.93) |
| Controls? | Y | N | Y | N | Y | N |
| Birth Cohorts | 42-53 | 42-53 | 42-44 | 42-44 | 51-53 | 51-53 |
| Observations | 600 | 600 | 150 | 150 | 150 | 150 |
| R-squared | 0.9516 | 0.9491 | 0.9212 | 0.9112 | 0.9568 | 0.9559 |

Notes: Standard errors are in parenthesis and are clustered by birth state. ***, **, and * indicate statistical significance at the 1, 5, and 10 percent level respectively. See Table 1 notes for data sources, sample restrictions and variable definitions. "Early cohorts" include men born 1942-1944, while "late" cohorts include men born 1951-1953. Regressions are weighted by cell size. Additional controls include birth state fixed effects, the employment to population ratio, the cohort size (derived from the 1960 Census), and birth region trends.