The Long-Term Impact of Military Service on Health: Evidence from World War II and Korean War Veterans

By KELLY BEDARD AND OLIVIER DESCHÊNES*

During the World War II and Korean War era, the U.S. military freely distributed cigarettes to overseas personnel and provided low-cost tobacco products on domestic military bases. In fact, even today the military continues to sell subsidized tobacco products on its bases. Using a variety of instrumental variables approaches to deal with nonrandom selection into the military and into smoking, we provide substantial evidence that cohorts with higher military participation rates subsequently suffered more premature mortality. More importantly, we show that a large fraction, 35 to 79 percent, of the excess veteran deaths due to heart disease and lung cancer are attributable to military-induced smoking. (JEL I10, I12, J14)

Recent U.S. military action in Iraq and elsewhere has once again focused attention on the health consequences of military service. Many veterans of the first Gulf War reported a collection of symptoms now known as "Gulf War Syndrome." While the scientific basis for Gulf War Syndrome has never been fully established, in 2002 the Department of Veterans Affairs (VA) established a new advisory committee composed of medical experts and veterans to help "focus on the research that we hope will improve the health of ill Gulf War veterans" (Secretary of Veterans Affairs Anthony J. Principi, January 23, 2002).[1] And later in 2002, the VA announced it had allocated $20 million for the 2004 fiscal year budget for research into Gulf War illnesses.[2]

The Gulf Wars are not the first time that health concerns associated with military service have surfaced. The VA pays compensation to veterans suffering from disabilities resulting from exposure to Agent Orange during military service.[3] And after World War II (WWII) and the Korean War there was substantial concern about the physical and mental health of former prisoners of war (POWs) as a result of extended, severe deprivation. As one might expect, many studies find that POWs have worse health outcomes and higher rates of premature death than non-POW veterans.[4]

Estimating the causal effect of military service on long-term health is made difficult by the positive selection into the military during WWII and the Korean War; military administrators selected individuals who satisfied minimum physical and mental aptitude criteria. As a result, differences in health outcomes between veterans and nonveterans may reflect the underlying differences in the characteristics of the two populations rather than the impact of military service on health. Carl C. Seltzer and Seymour Jablon (1974) provide a classic example of this phenomenon. They document the selection bias induced by prescreening based on mental and physical attributes by showing that

[1] http://www1.va.gov/rac-gwvi/docs/FOR_IMMEDIATE_RELEASE.doc.

[2] http://www1.va.gov/rac-gwvi/docs/pressrelease.doc.

[3] http://www1.va.gov/agentorange/docs/B3AOBRIEF72003A1.doc.

[4] Examples include M. D. Nefzger (1970), Robert J. Keehn (1980), William F. Page and Richard N. Miller (2000), Page and L. M. Brass (2001), and Catherine R. Gale et al. (1999).

WWII veterans have lower age-adjusted mortality rates than nonveterans.

In their seminal paper, Norman Hearst et al. (1986) resolve this selection bias problem by using the natural randomized experiment generated by the Vietnam draft lottery. They show that those draft-eligible men with low lottery numbers had higher mortality rates in the years immediately following Vietnam, and that the excess mortality was concentrated in suicides and motor-vehicle accidents. Nevertheless, there is still considerable uncertainty regarding longer-term impacts, additional causes of death, and the nature of the causal mechanism between health and military service.

This paper, therefore, seeks to provide credible estimates of the long-term consequences of military service by studying the morbidity and mortality of WWII and Korean War veterans 20 to 50 years after discharge from the military. We overcome the selection bias caused by military screening rules using a variety of data sources and instrumental variables strategies similar to those of Joshua D. Angrist and Alan B. Krueger (1994) and Guido Imbens and Wilbert van der Klaauw (1995). Our results are important for at least two reasons. First, previous studies focused on the short-run impacts—five to ten years after military discharge. Since many health conditions take many years to develop, short-run comparisons may greatly understate the impact of military service on post-service health. Second, we propose an important causal link in the military-health nexus that has so far received little attention: *smoking*. We show that military-induced smoking is an important cause of premature veteran mortality. The military encouraged smoking by freely distributing cigarettes to personnel stationed overseas and selling cigarettes at reduced prices on U.S. bases during WWII and the Korean War (U.S. Public Health Services, 1989 p. 425).[5] Moreover, in 1993 the VA acknowledged the link between their past tobacco policies and subsequent smoking-related illnesses when the VA general counsel issued the opinion that "injury or disease resulting from tobacco use initiated in active military service could serve as the basis for a service-connected claim for compensation" (see VAOPGCREC 2-93 in Jeffrey E. Harris, 1997). Despite these concerns, military personnel continue to have access to subsidized tobacco products on military bases even today.

The elevated risk of mortality due to military-induced smoking demonstrates that the health consequences of military service are not solely attributable to traumatic experiences or injuries suffered while in the military. Instead, they may partly be the result of the military facilitating the adoption of risky behaviors/habits early in the life of young soldiers.[6] This issue is important for current public policy since the VA is one of the largest providers of health insurance in the United States. Moreover, in recent years there has been a debate about the rising costs of the VA medical programs, which constitute nearly half of all VA outlays.[7]

We begin by analyzing the annual post-service age-specific mortality rates for men born in the cohorts at risk of serving during WWII and in Korea, and contrast them with the proportion of veterans in each cohort. To control for unobserved cohort differences in mortality, we use women from the same cohorts—who were essentially exempt from military service—as a comparison group, as well as simple inter-cohort trends.[8] The results reveal substantial excess post-service mortality among cohorts with higher military service rates: a 10-percentage-point increase in the fraction of veterans is associated with 0.38 more deaths per 1,000 men in every year between the ages of 40 and 75. This translates into a 2.5-percent increase in the average annual death rate for a 10-percentage-point rise in the veteran rate.

We then turn to the cause-specific mortality rates to uncover the specific margins on which excess veteran mortality is most pronounced. By examining the major causes of death, we expose the most important veteran-nonveteran

---

[5] While the distribution of free cigarettes in rations stopped in 1975, tobacco usage rates are still high among military personnel. During the 1995 fiscal year, sales of tobacco products by the Department of Defense totaled $747 million (U.S. Department of Defense, 1996).

[6] There is an extensive literature studying the relationship between early life conditions/behaviors and later health. See Jonathan Gruber (2001) and Robert W. Fogel (2003) for overviews.

[7] VA medical expenditures rose from $3 billion in 1980 to $19 billion in 2000 (in 2000 constant dollars).

[8] Variants of this difference-in-difference strategy have also been used in the context of the military by Imbens and van der Klaauw (1995) and David Card and Thomas Lemieux (1999).

mortality differences. This exercise reveals the somewhat surprising, and to our knowledge new, finding that excess veteran mortality after the age of 40 is most pronounced for heart disease and lung cancer—two causes that are strongly linked to smoking. In fact, according to the American Lung Association, smoking is responsible for 87 percent of lung cancer deaths.[9] While the link between smoking and lung cancer is widely recognized, the strong link between smoking and heart disease is less well known. According to the U.S. Department of Public Health Service, among people under the age of 65, cigarette smoking is responsible for 40 to 45 percent of coronary heart disease.[10]

The finding that the "veteran mortality effect" is strongest for heart disease and lung cancer is consistent with the notion that veteran status is an intervening variable for smoking, and that at least part of the health consequences of military service are caused by military-induced smoking. We substantiate the military-smoking connection using the Current Population Survey (CPS) tobacco usage supplements. The instrumental variables estimates indicate that military service increased the smoking rate of WWII and Korean War veterans by 30 percentage points. Combining the smoking and mortality rate estimates, we show that military-induced smoking explains 64 to 79 percent of excess veteran deaths due to heart disease and 35 to 58 percent of excess veteran deaths due to lung cancer, between the ages of 40 and 75. This may well be the ultimate smoking gun.

Finally, we corroborate the mortality results by looking at other health outcomes using data from the U.S. Census. Given the aforementioned veteran mortality effect, we should also observe higher disability rates among veterans. Using the same instrumental variables strategy and data from the 1980 and 1990 Censuses of Population, we find that by their fifties and sixties, veterans were 3.2 percentage points more likely to suffer from a work-preventing disability and 9.5 percentage points more likely to suffer from a work-limiting disability.

Overall, our results suggest a strong causal connection between military service and long-term morbidity/mortality, although not necessarily through the channels that have previously been identified as responsible for the link. In contrast to the Hearst et al. (1986) finding of elevated mortality rates in the years immediately following military discharge due to motor-vehicle accidents and suicide, the longer-run results reported in this study point to heart disease and lung cancer as major contributors. Based on our analysis of veteran mortality patterns, we estimate that military service caused approximately 2 million *additional* premature deaths for the 1920–1939 cohorts between the ages of 40 and 75 (out of a population of approximately 20 million men at age 40), relative to the cumulative death rate that would have been expected with a veteran rate of zero for these cohorts. As we will show in Section ID, on average 11.6 years of potential life were lost (YPLL) for each additional premature death due to military participation, relative to a life expectancy of 75 years. To put this in perspective, this implies that the total YPLL due to premature death between the ages of 40 and 75 is approximately the same as the total YPLL due to battle deaths during WWII and the Korean War combined.[11] The remainder of the paper is as follows. Sections I and II examine the mortality patterns for veterans, the connection between military smoking policies during WWII and the Korean War, and subsequent veteran mortality. Section III corroborates the mortality results by examining the causal link between higher disability rates and veteran status. Section IV concludes.

I. Excess Veteran Mortality

A. *Mortality Data*

The main objective of this paper is to provide a credible estimate of the long-term conse-

---

[9] http://www.alahv.org/bookfiles4/lung_cancer.html.
[10] See U.S. Public Health Service (1984) and Henry C. McGill et al. (2000).

[11] Our premature mortality estimates reported in Table 4 suggest that a total of 23.2 million years of potential life were lost (2 million × 11.6) as a result of military service during WWII and Korea, conditional on surviving up to age 40. Assuming that soldiers killed during WWII and the Korean War lost 50 years of potential life, relative to a 75-year life expectancy, the 450,000 battle deaths during the two wars (U.S. Bureau of the Census, 2000) caused a loss of 22.5 million years of potential life (450,000 × 50).

quences of military service on post-service mortality. More specifically, do veterans suffer higher rates of *premature* mortality than they otherwise would have? Because the sample sizes in the available microdata sets with individual mortality information (such as the National Longitudinal Mortality Survey) are relatively small, we investigate this question using grouped-data models for birth cohorts. If military service has a detrimental effect on health, then excess mortality should be observed in cohorts with higher veteran rates.

Data on individual mortality causes are taken from the Multiple Cause of Death (MCOD) Files for 1968–2000 (National Center for Health Statistics). The mortality records contain information about gender, race, age at death, place of residence at death, and cause of death for all deaths occurring in the United States in each year. Data on population counts by sex and single year of age are taken from the Postcensal Resident Population Estimates for 1968–2000. With the available 33 years of data we construct panels of all-cause and cause-specific mortality rates for different birth cohorts by sex and single year of age.[12] Throughout the analysis, we pool white and black men and women; however, the inclusion or exclusion of black individuals is inconsequential for the results. Unfortunately, the demographic information available from the MCOD files excludes veteran status. As a consequence, direct contrasts of the mortality rates by veteran status cannot be computed. We therefore analyze the relationship between cohort-specific mortality rates and veteran rates.

We restrict the analysis to individuals age 40 to 75 from the 1920–1939 birth cohorts. This choice is motivated by several factors. First, these represent all cohorts at risk of serving during the Korean War and the majority of the cohorts at risk of serving during WWII; men born before 1920 constitute only 23 percent of all veterans born between 1901 and 1939. Importantly, some of the cohorts born from 1920 to 1939 were at risk of serving in the years where the veteran rate rapidly declined, corresponding to the end of WWII and the Korean War. Second, we exclude the cohorts born before 1920 since the mortality records, which are available in computer-readable format starting in 1968, allow us to observe only the mortality rates of the pre-1920 cohorts at relatively old ages. For example, the average age of death for the pre-1920 cohorts we observe in the MCOD data is 74, as opposed to 56 for the post-1920 cohorts. Since we focus on premature mortality, it is essential to observe the members of a cohort for an extended period before they reach life expectancy. Moreover, since we have fewer observations for the older cohorts, it is more difficult to pin down empirically any permanent cohort effects in the statistical models discussed below. Finally, there is also evidence that the cohorts born during the 1918–1919 influenza pandemic have higher age-adjusted mortality rates for reasons unrelated to military service (Douglas Almond, 2005).[13]

The veteran rate for each cohort is calculated from the 1960–1980 Censuses of Population. All men from these cohorts who served in the military are classified as veterans, irrespective of the period of service. Most veterans from these cohorts are either WWII or Korean War veterans.[14] We pool the microdata from the 1960–1980 Censuses and use a model with unrestricted census year effects, birth cohort dummies, and quartic profile in age. The cohort-specific averages of the predicted values from this model are used as our measure of veteran rate.[15]

Table 1 reports the average all-cause annual deaths per 1,000 men (or women), as well as the rates for the major causes for men and women in the 1920–1939 birth cohorts who are between the ages of 40 and 75. There are 579

---

[12] Place of birth is not reported in the MCOD files before 1980. As a result, all analysis includes both natives and immigrants. Our tabulations of post-1980 data indicate that for the 1920–1939 birth cohorts, immigrant deaths constitute less than 5 percent of annual deaths, and consequently our analysis should not be greatly affected by their inclusion. The yearly population estimates also do not distinguish between natives and immigrants.

[13] While we believe that the 1920–1939 birth cohorts are the appropriate group to study, the results are generally similar if we use a more restrictive cutoff for the Korean War era (for example, restrict the sample to the 1920–1933 birth cohorts), or a wider window to include older WWII veterans (birth cohorts 1918–1939, for example).

[14] While 96 percent of veterans from the 1920–1932 cohorts served in WWII, Korea, or both, 40 percent of the 1933–1939 cohorts served in Korea, with the rest serving in the interwar period, between Korea and Vietnam.

[15] The results are the same if unadjusted sample averages are used.

	Men	Women
1. All mortality causes	15.41	8.85
	(10.90)	(6.71)
	100%	*100%*
Cause-specific mortality		
2. Ischemic heart disease	3.98	1.48
	(2.61)	(1.37)
	26%	*17%*
3. Lung cancer	1.85	0.86
	(1.44)	(0.70)
	12%	*10%*
4. Colon cancer	0.39	0.28
	(0.33)	(0.20)
	3%	*3%*
5. Cerebrovascular disease	0.64	0.51
	(0.57)	(0.44)
	4%	*6%*
6. Respiratory diseases	0.91	0.58
(chronic and pneumonia)	(1.04)	(0.66)
	6%	*7%*
7. Accidents and suicides	0.65	0.23
	(0.21)	(0.09)
	4%	*3%*
Fraction veterans	0.66	—
Fraction ever-smokers	0.75	0.50
Observations	579	579

Notes: Standard deviations in parentheses. Entries in italics represent the percentage of all-cause mortality attributable to each specific cause.

observations for the male and female samples, corresponding to the number of cohort-by-year cells for men and women aged 40 to 75 observed between 1968 and 2000. As one would expect, the premature mortality rates for men are nearly double those for women. Despite the difference in levels, the distribution across major causes of death is fairly similar, except for ischemic heart disease. On average, between the ages of 40 and 75, heart disease accounts for 26 percent of annual deaths for men compared to only 17 percent for women.

B. *Descriptive Analysis*

We begin the analysis by displaying the main patterns in the data. Figure 1 plots the average log mortality rates per 1,000 men born from 1920 to 1939, along with the fraction of male veterans in each cohort. Since we use an unbalanced panel, the log mortality rates are adjusted for differences in age using unrestricted age dummies, and all figures plot residual rates.

Figure 1 provides clear evidence of a strong positive association between military participation and mortality: cohorts with higher veteran rates also have average age-adjusted mortality rates (the correlation is 0.95). Most importantly, log mortality tracks, at least in part, the discontinuous changes in the veteran rate at the end of WWII (cohorts 1927–1929) and Korea (cohorts 1932–1935). In both cases, the decline in the veteran rate is mirrored by a corresponding decline in the age-adjusted log mortality rate.

Omitted cohort-specific factors may, however, confound this relationship. In Section IC, we describe two approaches to control for this possibility. First, we assume that the omitted cohort-specific determinants of male mortality evolve smoothly and can therefore be approximated by a smooth cohort trend. Second, we use women from the same birth cohorts as a comparison group in a "difference-in-difference" model. Since almost no women served in the military during WWII and the Korean War, the mortality rate of women may provide a reasonable counterfactual for the cohort mortality trend for men in absence of military participation.[16] The key assumption for this comparison to be valid is that the idiosyncratic cohort effects, representing the impact of the medical and economic conditions at birth, are the same for men and women.[17]

Figure 2 shows the trends underlying this second approach. It plots the average log relative mortality rates of men and women from the 1920–1939 cohorts, as well as the fraction of male veterans. Again, log relative mortality tracks the veteran rate closely: the correlation is 0.84. As in Figure 1, there is again a notable parallel between the sharp reduction in the veteran rates at the end of WWII and the Korean War and the relative mortality for men.

C. *Empirical Framework*

Although individual-level mortality data are not available, we begin with an individual-level model in order to illustrate the causal relation-

---

[16] Information on female veterans is not available prior to the 1990 Census of Population. Tabulations from these data indicate that 0 to 3 percent of females born between 1920 and 1939 are veterans.

[17] In the statistical models presented below, we also allow the age effects to differ across men and women.
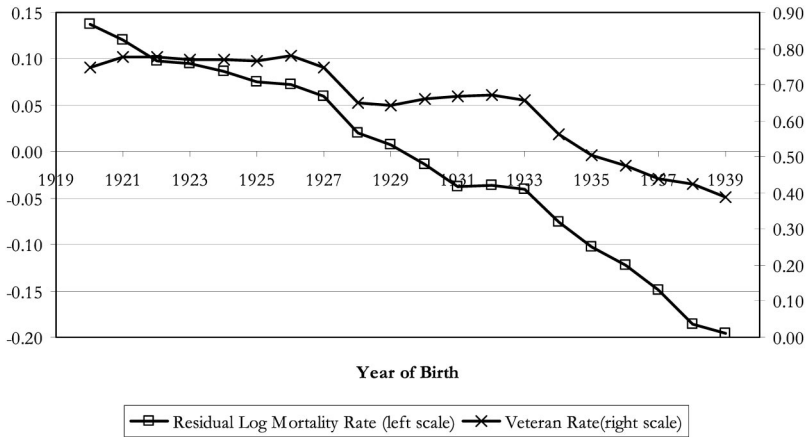
FIGURE 1. AGE-ADJUSTED LOG MALE MORTALITY RATE (RESIDUALS FROM REGRESSION ON AGE DUMMIES) AND MALE
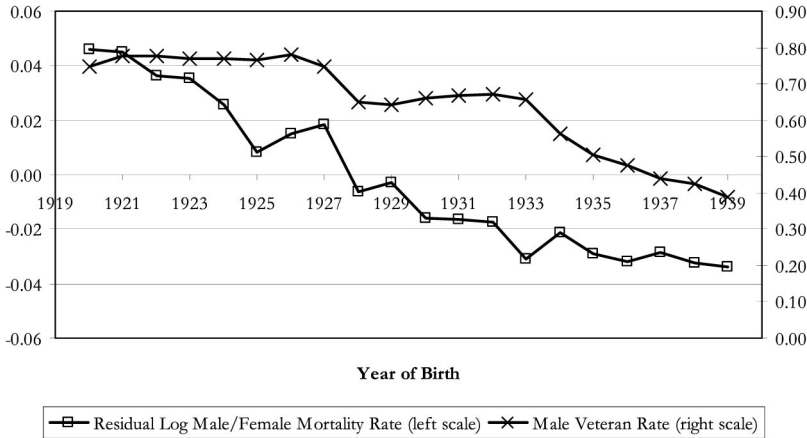VETERAN RATE, BY YEAR OF BIRTH

FIGURE 2. AGE-ADJUSTED LOG MALE/FEMALE MORTALITY RATE (RESIDUALS FROM REGRESSION ON AGE DUMMIES) AND
MALE VETERAN RATE, BY YEAR OF BIRTH

ship we seek to estimate. At the individual level, the impact of military participation on male post-service mortality could be estimated from the following equation:

$$(1) \quad M_{ict} = \alpha + \beta V_{ic} + \lambda_{t-c} + \varphi_t + u_{ict}$$

where M_{ict} equals 1 if person i from birth cohort c died in year t; V_{ic} is a dummy variable equal to 1 if person i from birth cohort c served in the military; λ_{t-c} represents unrestricted age effects (age $= t - c$); and φ_t represents unrestricted calendar year effects. Such other person-specific predictors of mortality as edu-

cation are excluded from the model for notational convenience. In this model, β represents the causal effect of military service on post-service mortality. Estimates of β based on cross-sectional comparisons, however, are unlikely to be informative about the causal effect of military service on mortality because selection into the military was partly based on mental and physical attributes. More precisely, V_{ic} and u_{ict} are likely negatively correlated, and hence simple cross-sectional estimates of the impact of military service on mortality will tend to be understated. We therefore use an instrumental variables (IV) approach to deal with this issue.

As noted above, the data underlying equation (1) are not available. The MCOD data provide information on the universe of deaths occurring during a given year, but no such data exist for the living population; only yearly population counts by single year of age are available. Fortunately, IV estimates of equation (1) can be derived without individual-level data. The OLS estimates of β in the grouped-data version of model (1) correspond to the IV estimates of β in the individual-level model when the percentage of veterans in each birth cohort, V_c, is used as an instrument for V_{ic} (see Angrist, 1990; Imbens and van der Klaauw, 1995).[18] While the absence of microdata means that we cannot estimate the reduced-form relationship between individual mortality and the cohort-average veteran rate, the first-stage relationship between veteran status and the cohort-average veteran rate can be estimated using data from the Census of Population. This is not technically required but is useful to assess the predictive power of the instruments for the endogenous variable. (We report the *F*-statistics on the excluded instruments from these regressions in Table 8 on p. 192.) Unfortunately, standard overidentification test statistics cannot be computed in this context since we do not have the microdata underlying the reduced-form for mortality.

The credibility of this particular IV approach depends on whether or not unobserved cohort effects are appropriately controlled for. Clearly, cohort-average veteran rates cannot be used as an instrument for veteran status if there are "direct" cohort effects in equation (1) in addition to the unrestricted age effects. We use two approaches to control for the possibility that there are cohort effects in equation (1), one using data on men only and the other pooling data for men and women. These two models control for cohort effects in specific ways. The first assumes that the cohort effects can be modeled as a smooth cohort trend, while the second assumes that the birth cohort effects are the same for men and women from the same cohorts, once we allow for sex specific unrestricted age effects.[19] The first approach will be invalid if the process by which year of birth affects future health evolves discontinuously across birth cohorts. The second approach allows for such discontinuities but assumes that the process is the same for men and women.

In all specifications, we use a linear model for the log mortality rate ($\log \overline{M}_{ct}$) of cohort c, in year t. Since the dependent variable is a proportion, linear models for rates suffer from the same drawbacks as the linear probability model for binary data. A popular approach to resolve this problem is to use a linear model for the log-odds ratio.[20] In our application, the cohort-by-year mortality rates are very small and, therefore, log-odds and log-rate models give similar results.[21] To proceed, consider the following log linear model for the male mortality rate:

$$(2) \quad \log \overline{M}_{ct} = \alpha + \beta \overline{V}_c$$
$$+ \delta(c) + \lambda_{t-c} + \varphi_t + u_{ct}$$

where $\overline{V}_c$ is the male veteran rate in cohort c, λ_{t-c} represents unrestricted age effects, $\delta(c)$ is a smooth function of year of birth (here restricted to be linear), and φ_t are unrestricted calendar-year effects.[22] The cohort trend will pick up the effect of all observable (i.e., average education and permanent income) and unobservable (i.e., medical conditions at birth) *time-invariant* cohort-specific predictors of mortality under the assumption that these factors evolve smoothly across cohorts. The year effects sim-

[18] Similarly, cohort dummies could be used as instruments for veteran status.

[19] Another possibility would be to exploit the important difference in the military service rates of white and black men in the difference-in-difference model. The important racial disparities in life expectancy at birth and in the trends across cohorts, however, make this approach questionable.

[20] Leslie E. Papke and Jeffrey M. Wooldridge (1996) discuss various regression models for proportions.

[21] Results from the log-odds ratio specification are available upon request.

[22] Since age, birth cohort, and calendar year are related by the identity age = $t - c$, unrestricted age and year effects cannot be included along with the linear trend in c. In practice, this requires an additional restriction on the age effects or the year effects. We remove an additional year dummy from the models to account for this. An alternative approach would be to impose more structure on the year effects (for example, defining the year effects as a set of dummies for each three calendar year, i.e., a dummy for 1968–1970, a dummy for 1971–1973, etc). Angus S. Deaton (1997) presents a useful discussion of the various alternative approaches to this problem.

ilarly control for the time-varying confounders that affect all cohorts equally, conditional on the age effects (i.e., medical technology). In practice, the inclusion or exclusion of the year effects has little impact on the results, once we control for the unrestricted age effects. Since the model includes age and year dummies, the parameter of interest, β, is identified from across cohort covariation in the mortality rate and the veteran rate, after accounting for the linear trend across cohorts.

Our second empirical strategy is to pool data for men and women. This enables us to estimate β while controlling for unrestricted year-of-birth effects (assumed to be the same for men and women), sex-specific age effects, and year effects. Equation (3) illustrates this model:

$$(3) \quad \log \overline{M}_{sct} = \alpha + \beta \overline{V}_{sc} + \omega_s$$
$$+ \delta_c + \lambda_{st-c} + \varphi_t + u_{sct}$$

where s denotes sex and δ_c are unrestricted year-of-birth effects. Note that this model also includes a dummy for men ($\omega_s = 1$ if sex = male) and sex-specific unrestricted age effects (λ_{st-c}). Since the veteran rate $\overline{V}_{sc}$ is defined to be zero for women, this is essentially a difference-in-difference model. The assumption underlying this approach is that, after controlling for year and sex-specific age effects, the female mortality rate provides a valid counterfactual for male mortality, absent military service.[23]

We estimate equations (2) and (3) by OLS using a variety of specifications. All models are weighted by the square root of the inverse sampling variance of the log mortality rate.[24]

---

[23] Female attrition due to mortality during childbearing years could potentially invalidate this assumption. While this might be a concern for cohorts of women giving birth before 1930 (i.e., female cohorts born before 1910), when the per-birth maternal death rate was 0.006 deaths per live birth, by the late 1940s (the post–WWII baby boom) maternal deaths during childbirth had fallen to fewer than 0.002 deaths per live birth (see Bernard Guyer et al., 2000). One might also be concerned that changes in total fertility per woman could have an impact on long-run female health. However, for the 1920–1939 female birth cohorts, total fertility was relatively constant, ranging from 2.7 children per woman to 3.1 children per woman (see Donald J. Hernandez, 1993).

[24] The sampling variance for the log mortality rate is given by $(1 - \overline{M}_{sct})/(n_{sct} \times \overline{M}_{sct})$, where n_{sct} is the number of observations in each cell (see Gary S. Maddala, 1983).

Throughout, we report standard errors that allow for clustering at the birth cohort level.[25] Finally, for interpretive ease, the parameter estimates reported in all tables are converted back into mortality rates per 1,000 (as opposed to log mortality rates). This is done by calculating the sample average of the marginal effects as follows: let $\hat{\beta}$ be the estimated coefficient on the veteran rate in the log mortality rate regression, and let $\hat{\mu}_{ct}$ denote the fitted values from our model of the conditional mean of log mortality. The effect in levels is then the sample average of $1000 \times \hat{\beta} \exp(\hat{\mu}_{ct})$. Standard errors of the marginal effects are computed using the delta method.

D. Veterans and Mortality

Table 2 reports the results obtained from estimating equation (2) by OLS. The key point about this specification is that it uses a linear birth cohort trend to control for cohort effects.[26] Columns 1 and 2 exclude and include year indicators, respectively. Column 3 reports the average annual mortality rate per 1,000 men, and columns 4 and 5 transform the estimates in column 2 into the predicted nonveteran and veteran average annual mortality rates per 1,000 men assuming that 66 percent of men are veterans.

The first row in Table 2 reports the average annual excess death rate for veterans. A 10-percentage-point rise in the veteran rate is associated with an average of 0.34 more annual deaths per 1,000 men between the ages of 40 and 75, and is precisely estimated. This point estimate implies a nonveteran average annual mortality rate of 13.1 per 1,000 men and a veteran annual mortality rate of 16.6 per 1,000 men.

The remaining rows in Table 2 report the average annual excess veteran mortality rates for the major causes of death. In particular, row 2 reports the estimates for ischemic heart disease and row 3 reports the results for lung cancer. For

---

[25] Standard errors that ignore clustering are typically 10 to 30 percent smaller than those that account for clustering (i.e., those reported in the tables).

[26] Similar results are obtained if the linear trend is replaced by a quadratic trend, with the exception of the lung cancer effect, which is somewhat attenuated.

TABLE 2—IMPACT OF VETERAN STATUS ON MORTALITY, MALE-ONLY SAMPLE

	Veteran effect		Mean rate	Implied mortality rates[a]	
				Nonvets	Vets
	(1)	(2)	(3)	(4)	(5)
1. All mortality causes	3.562	3.424	15.406	13.1	16.6
	(0.97)	(0.97)			
Cause-specific mortality					
2. Ischemic heart disease	0.965	0.740	3.979	3.5	4.2
	(0.27)	(0.21)			
3. Lung cancer	0.986	1.027	1.851	1.2	2.2
	(0.18)	(0.20)			
4. Colon cancer	0.024	0.016	0.388	0.4	0.4
	(0.04)	(0.04)			
5. Cerebrovascular disease	0.043	−0.015	0.642	0.7	0.6
	(0.05)	(0.05)			
6. Respiratory diseases	0.255	0.309	0.910	0.7	1.0
(chronic and pneumonia)	(0.09)	(0.11)			
7. Accidents and suicides	0.233	0.097	0.646	0.6	0.7
	(0.08)	(0.05)			
Observations	579	579			
Unrestricted age dummies	Yes	Yes			
Linear cohort trend	Yes	Yes			
Unrestricted year dummies	No	Yes			

Notes: The estimated standard errors in parentheses allow for cohort-level clustering. Each regression is weighted by the inverse of the sampling variance of the dependent variable.

  [a] Implied mortality rates based on the estimates in column 2.

both of these causes, there is substantial excess veteran mortality. A 10-percentage-point rise in the veteran rate implies a rise in deaths due to ischemic heart disease of 0.07 per 1,000 men. To put this in context, this estimate implies an average annual veteran mortality rate per 1,000 men due to heart disease of 4.2 compared to only 3.5 for nonveterans. A similar pattern is found for lung cancer. The excess annual lung cancer mortality rate among veterans is 1.03 per 1,000 men; a 10-percentage-point rise in the veteran rate causes 0.10 more deaths per 1,000 men. Mortality caused by respiratory diseases (including pneumonia) is also more prevalent among veterans, as indicated in row 6. However, the magnitude of the effect is smaller.

In contrast to the significant excess veteran mortality for all causes, heart disease, lung cancer, and respiratory diseases, there is less evidence of important veteran effects for the remaining three major causes of death. The veteran effect is both extremely small and imprecise for colon cancer and cerebrovascular disease. The veteran effect for suicides/accidents is precisely estimated, but small in magnitude: a 10-percentage-point rise in the vet-

eran rate leads to a 0.01 per 1,000 men rise in average annual deaths due to suicides/accidents.[27]

The evidence in Table 2 is consistent with the possibility that veteran status is an intervening variable for smoking, and supports the hypothesis that some of the health consequences of military service are caused by smoking. Cohorts with more veterans have higher rates of smoking-related mortality, as indicated in rows 2 and 3, but similar mortality rates for nonsmoking-related illnesses. We will return to this important finding in Section II.

Table 3 replicates Table 2 using equation (3). These models use women to control for cohort-effects rather than the linear trend used in equation (2). The estimates in Table 3 are similar to

[27] It is worth pointing out that the impact on suicide/accidents is much lower than the one reported for Vietnam-era veterans by Hearst et al. (1986). Based on the data in Hearst et al. (1986), Angrist et al. (1996) report IV estimates of suicides/accidents for Vietnam veterans that are 10 to 20 times larger than the ones reported in Table 2. Part of this difference likely reflects the Hearst et al. (1986) focus on the years immediately following military discharge and our focus on middle- to old-age men.

| | Veteran effect | | Mean rate (men) | Implied mortality rates[a] | |
| | | | | Nonvets | Vets |
	(1)	(2)	(3)	(4)	(5)
1. All mortality causes	3.847	3.754	15.406	12.9	16.7
	(0.44)	(0.43)			
Cause-specific mortality					
2. Ischemic heart disease	1.147	0.919	3.979	3.4	4.3
	(0.10)	(0.09)			
3. Lung cancer	1.643	1.688	1.851	0.7	2.4
	(0.22)	(0.23)			
4. Colon cancer	−0.094	−0.086	0.388	0.4	0.4
	(0.02)	(0.02)			
5. Cerebrovascular disease	−0.054	−0.041	0.642	0.7	0.6
	(0.03)	(0.03)			
6. Respiratory diseases (chronic and pneumonia)	0.809	0.836	0.910	0.4	1.2
	(0.12)	(0.13)			
7. Accidents and suicides	−0.092	−0.114	0.646	0.7	0.6
	(0.02)	(0.02)			
Observations	1158	1158			
Unrestricted sex ∗ age dummies	Yes	Yes			
Unrestricted cohort dummies	Yes	Yes			
Unrestricted year dummies	No	Yes			

Notes: The estimated standard errors in parentheses allow for cohort-level clustering. Each regression is weighted by the inverse of the sampling variance of the dependent variable.

[a] Implied mortality rates based on the estimates in column 2.

those in Table 2. This is important since the credibility of our results hinges on the validity of the controls for "direct" cohort effects in the mortality equations. The fact that two different approaches yield similar estimates adds to our confidence. Nevertheless, some differences are worth noting. Lung cancer and respiratory disease estimates using women to control for cohort effects are larger than those using a linear cohort trend. To be specific, the estimated veteran effect rises from 1.03 (standard error = 0.20) to 1.69 (standard error = 0.23) for lung cancer and from 0.31 (standard error = 0.11) to 0.84 (standard error = 0.13) for respiratory disease. In light of the standard errors, however, the difference between the lung cancer and respiratory disease estimates in Tables 2 and 3 do not appear too important. Despite this, it is possible that men and women become less comparable at older ages (even after controlling for sex-specific age effects). Thus, the counterfactual experiment provided by women may become less credible at older ages. We evaluate this possibility in Table 4. One anomaly is that the coefficient on accidents/suicides changes sign, from 0.10 (Table 2) to −0.11 (Table 3).

While there is no clear explanation for this result, the implied difference in mortality between veterans and nonveterans is negligible in both cases.

Table 4 breaks the sample into age groups: 40–54, 55–64, and 65–75. This stratification allows us to explore the age-gradient of excess veteran mortality. Columns 1 to 3 report the average annual excess veteran mortality rate per 1,000 men using the linear cohort control and the male sample, and columns 4 to 6 report the same estimates using the male and female sample and women to control for cohort effects. All models include age and year dummies. As can be seen in the first row ("all mortality causes"), there is an age-increasing veteran effect. The veteran effect rises from 2.0 to 3.0 to 8.4 using equation (2) and from 0.6 to 4.9 to 15.2 using equation (3). The large increase in the veteran effect for the 65–75 group using women as the cohort control is likely the result of women being a poor control for male cohort effects near to male life expectancy, which is not surprising as male and female life expectancy were changing differentially across men and women for some of the 1920–1939 birth cohorts. As such,

TABLE 4—IMPACT OF VETERAN STATUS ON MORTALITY, BY AGE GROUP

	Veteran effect men only			Veteran effect men and women			Male mortality rate (per 1000)		
	40–54 (1)	55–64 (2)	65–75 (3)	40–54 (4)	55–64 (5)	65–75 (6)	40–54 (7)	55–64 (8)	65–75 (9)
1. All mortality causes	1.998	2.971	8.414	0.589	4.831	15.079	6.627	15.948	33.040
	(0.42)	(0.86)	(3.73)	(0.06)	(0.37)	(2.43)			
Cause-specific mortality									
2. Ischemic heart disease	0.535	0.864	1.316	0.262	1.143	1.760	2.003	4.190	7.953
	(0.12)	(0.23)	(1.06)	(0.03)	(0.12)	(0.55)			
3. Lung cancer	0.355	0.800	2.216	0.568	1.811	4.497	0.603	2.121	4.141
	(0.08)	(0.24)	(0.54)	(0.07)	(0.22)	(0.75)			
4. Colon cancer	0.001	−0.019	0.197	−0.036	−0.094	−0.079	0.125	0.415	0.919
	(0.02)	(0.05)	(0.17)	(0.01)	(0.02)	(0.10)			
5. Cerebrovascular disease	0.011	−0.055	0.434	−0.036	−0.049	0.380	0.263	0.590	1.551
	(0.03)	(0.06)	(0.16)	(0.01)	(0.01)	(0.12)			
6. Respiratory diseases (chronic and pneumonia)	0.132	0.244	0.184	0.127	0.789	2.872	0.203	0.798	2.633
	(0.03)	(0.05)	(0.45)	(0.01)	(0.08)	(0.68)			
7. Accidents and suicides	0.087	0.129	0.048	−0.200	−0.062	0.409	0.711	0.529	0.689
	(0.08)	(0.06)	(0.13)	(0.02)	(0.03)	(0.13)			
Observations	264	194	121	528	388	242			
Unrestricted age dummies	Yes	Yes	Yes	Yes	Yes	Yes			
Unrestricted cohort dummies	No	No	No	Yes	Yes	Yes			
Linear cohort trend	Yes	Yes	Yes	No	No	No			
Unrestricted year dummies	Yes	Yes	Yes	Yes	Yes	Yes			

Notes: The estimated standard errors in parentheses allow for cohort-level clustering. Each regression is weighted by the inverse of the sampling variance of the dependent variable.

we view the equation (2) estimates as the preferred estimates for the age subgroups.

Focusing on columns 1 to 3, while the veteran mortality effect for all-cause mortality, heart disease, lung cancer, and respiratory disease are all increasing with age, only all-cause mortality and lung cancer are sufficiently precisely estimated to make this claim with any confidence. Further, as shown in columns 7 to 9, average annual death rates are also rising with age. As a result, even though the effect of veteran status on mortality rises with age, the effect is roughly constant in percentage terms across age groups.

To put the age-increasing veteran effect for the overall death rate in context, it is helpful to approximate the years of potential life lost (YPLL), relative to a life expectancy of 75, implied by the estimates in Table 4 for men born from 1920 to 1939. Based on the estimates in columns 1 to 3, the annual per 1,000 mortality rates from age 40 to 54, 55 to 64, and 65 to 75 are 2.0, 3.0, and 8.4 higher than what would have been expected if the veteran rate for these cohorts were zero. Given a population of approximately one million men per birth cohort,

at age 40, and an average veteran rate of 66 percent, approximately 336,402, 359,304, and 1,138,223 additional men died between the ages of 40 and 54, 55 and 65, and 65 and 75[28] with average YPLLs of 28, 15, and 5.5, respectively. Taking the weighted average of these estimates implies an overall average YPLL for each additional death due to military participation of approximately 11.6 years, relative to a life expectancy of 75. This shows that military service both increases post-service premature mortality and accelerates the process substantially.

E. *Potential Identification Strategy Issues*

Throughout the analysis, the causal effect of military service on mortality is identified from variation in the veteran rate across birth cohorts under the assumption that our cohort specifications, a smooth cohort trend or the male/female

---

[28] By comparison, approximately 450,000 men died from battle-related and other deaths during WWII and the Korean War (U.S. Bureau of the Census, 2000).

difference, adequately control for the unobserved cohort effects. We now discuss the two most likely confounders that would violate these exclusion restrictions: education and income.

Veterans from WWII and the Korean War are better educated than their nonveteran counterparts (see Table 7). This reflects positive selection into the military as well as increased veteran educational attainment due to the education subsidy (GI Bills) available to returning veterans (see John Bound and Sarah Turner, 2002; Marcus Stanley, 2003). However, as education has been found to reduce mortality (Adriana Lleras-Muney, 2005), the confounding effects of GI Bill–induced education will, if anything, lead to an understatement of the negative effect of veteran status on long-term health and mortality.

In the case of income there is more uncertainty about the sign and magnitude of the causal link. While cross-sectional evidence suggests that income and measures of health are positively related (see Angus S. Deaton and Christina H. Paxson, 1998; James P. Smith, 1999), recent evidence from an analysis of the "Social Security Notch" suggests that income and mortality are positively related (Stephen E. Snyder and William N. Evans, 2002). But again, for our purposes, the important point is that permanent income differences across cohorts will be absorbed by the cohort controls included in equations (2) and (3). Further, to the extent that cohorts share the same age-income profile, the age and year effects included in the models should capture any effect of transitory income on mortality.[29]

II. Smoking in the Military

The excess average annual mortality rates attributable to lung cancer and heart conditions for cohorts with higher military participation rates suggest that veteran status may be an intervening variable for smoking in the health equations. At the center of this contention is the military tobacco policy in place during WWII and the Korean War. As stated earlier, overseas soldiers received free cigarettes as part of their rations, and tobacco products were sold at low prices on U.S. military bases. At one extreme, one could therefore view military-induced smoking as the result of tobacco companies "donating" cigarettes to the military under the pretense of supporting the troops in order to gain a long-term increase in cigarette sales by getting a large number of young men addicted to tobacco. At the other extreme, it is possible that smoking is simply an unavoidable consequence of wartime military service. While military-induced smoking is likely a combination of these two factors, as well as such other factors as peer group effects, it seems unlikely that the combat-induced smoking avenue is the driving force behind our results, since many veteran cohorts had low combat participation rates but still had higher smoking rates than nonveterans. For example, men born in 1927 (who turned 18 in 1945) are more likely to have served in WWII and more likely to smoke than men born in 1928, despite the fact that neither cohort is likely to have experienced combat.

A. Data and Results

Our analysis of smoking and veteran status is based on data from the 1967 and 1968 August CPS, which contain information about smoking habits, veteran status, and other demographic variables.[30] At the time of these surveys, the cohorts of interest (birth years 1920–1939) were between the ages of 28 and 48. To the best of our knowledge, this is the earliest microdata source containing information on smoking practices and veteran status. Contrasting smoking behavior and veteran status in the years immediately following military separation for veterans minimizes the confounding effect of attrition due to smoking-related mortality. Recent descriptive studies by R. Monina Klevens et al. (1995) and W. Paul McKinney et al. (1997) likely suffer from this sort of bias as they compare smoking behavior of veterans and nonveterans over a large age range. Moreover, neither study addresses the endogeneity of veteran status. As discussed below, we avoid the attrition problem by focusing on relatively young

---

[29] Moreover, Angrist and Krueger (1994) find that the earnings gap between WWII veterans and nonveterans is essentially zero once positive selection in the military is accounted for.

[30] Responding to the interest generated by the 1964 Report of the Surgeon General, the 1967 and 1968 August CPS contained a section with 22 items on the smoking habits of the population.

<small>TABLE 5—IMPACT OF VETERAN STATUS ON SMOKING BEHAVIOR</small>

	OLS: men only		TSLS: men and women		TSLS: men only	
	(1)	(2)	(3)	(4)	(5)	(6)
1. Probability ever-smoker: veteran (1 = yes)	0.080 (0.005)	0.123 (0.004)	0.276 (0.03)	0.346 (0.02)	0.237 (0.14)	0.301 (0.03)
2. First stage F-statistic: year of birth * male (p-values)	—	—	86.55 (0.0001)	184.51 (0.0001)	2.49 (0.0003)	53.32 (0.0001)
Dataset	CPS 67/68	CPS 90s	CPS 67/68	CPS 90s	CPS 67/68	CPS 90s
Cohorts	1920–1939	1920–1939	1920–1939	1920–1939	1920–1939	1920–1939
Age range	28–48	53–75	28–48	53–75	28–48	53–75
Unrestricted age dummies	Yes	Yes	Yes	Yes	Yes	Yes
Sample size	31,969	64,104	68,879	141,557	31,969	64,104

Note: Eicker-White standard errors are reported in parentheses.

men and the endogeneity problem by using an instrumental variables approach.

Despite our concerns about nonrandom premature mortality, we supplement our analysis using CPS data from the 1990s, specifically: 1992, 1993, 1995, 1996, 1998, and 1999. At the time of these surveys, the 1920–1939 birth cohorts ranged in age from 53 to 75. In all cases, the models are estimated separately for the 1967/68 data and the data from the 1990s. A drawback of the CPS data from the 1990s is the possibility of sample selection due to increased premature death among smokers. As such, all results using 1990s CPS data should be interpreted with caution.

We begin by estimating the cross-sectional differences in the smoking rate of veterans and nonveterans using a simple linear probability model:[31]

$$(4) \quad Smoke_{ict} = \alpha + \beta V_{ic} + X_{ict}\delta + \lambda_{t-c} + u_{ict}$$

where $Smoke_{ict}$ is a dummy variable taking a value of 1 if person i in cohort c reports ever being a smoker in survey year t,[32] V_{ic} is a veteran status dummy, X_{ict} is a vector of personal characteristics (including education and indicators for marital status, race, and census division), and λ_{t-c} are unrestricted age effects. The OLS estimates of β are reported in the first row in columns 1 and 2 in Table 5. In the 1967/68 sample, veterans were 8 percentage points more

likely to have ever smoked, and in the 1990s they were 12.3 percentage points more likely to report having smoked. The higher smoking rate among veterans in the 1990s may reflect the slower death rate of veterans due to positive selection into the military (Seltzer and Jablon, 1974), despite a higher smoking rate.

Since veteran status is not randomly assigned, it is possible that V_{ic} and u_{ic} are correlated and hence simple cross-sectional estimates of the impact of veteran status on smoking are biased, although the direction of the bias is unclear. If smoking and socioeconomic status (SES) were negatively related during the WWII and the Korean War era, the positive selection of men in the military during WWII and the Korean War would lead cross-sectional comparisons to understate the inducement effect of military service. We therefore use instrumental variables to deal with this issue, and estimate equation (4) using two-stage least squares (TSLS).

In the two CPS samples (1967/68 and 1990s), we begin by pooling the male and female samples and using year-of-birth dummies interacted with a male dummy as instruments for veteran status in the smoking equation.[33] This allows us to identify the effect of veteran status on smoking, while controlling for unrestricted year-of-birth and age effects. The models also include controls for race, education, marital status, and division of residence. The identifying assumption is that cohort and age effects in the smoking equation are the same for men and women.

---

[31] Estimates of marginal effects from probit models are similar.

[32] Ever-smokers are defined as individuals who have smoked at least 100 cigarettes during their life.

[33] Remember that we assume throughout that the female veteran rate is zero.

TABLE 6—PERCENTAGE OF VETERAN MORTALITY EFFECT EXPLAINED BY MILITARY-INDUCED SMOKING

	Deaths rate per 1000 (Table 1) (1)	% Deaths caused by smoking (2)	Death rate per 1000 smokers due to smoking (3)	Additional deaths per 1000 due to veteran smoking (4)	% Veteran effect due to smoking (Table 2) (5)	% Veteran effect due to smoking (Table 3) (6)
Ischemic heart disease	3.98	40	2.12	0.586	79	64
Lung cancer	1.85	87	2.14	0.591	58	35

The IV estimates for veteran status are reported in the first row in columns 3 and 4 in Table 5. For the 1967/68 sample, military service caused a 27.6-percentage-point increase in smoking, with a standard error of 3. Consistent with the hypothesis that the military selected men with lower smoking propensities, the TSLS estimate is substantially larger than the OLS estimate reported in column 1.[34] While we are concerned about smoking-related attrition in the 1990s sample, the results are remarkably similar to those obtained for the 1967/68 sample. Column 4 reports a 27.6-percentage-point increase in smoking due to military service, with a standard error of 3. Row 2 reports the *F*-statistics and *p*-values associated with the excluded instruments in the first-stage equations. For both samples, the interaction of year of birth with a male indicator is a powerful predictor of veteran status. Importantly, the *F*-statistics reported are significantly larger than the rule-of-thumb values for weak instruments, as reported in Douglas Staiger and James H. Stock (1997).

An alternative to pooling males and females is to reestimate the instrumental variables version of equation (4) for the male-only sample. In this case, the models are identified by the exclusion of year-of-birth dummies in the smoking equation, while controlling for unrestricted age effects. We have concerns about this identification strategy because it precludes us from controlling for underlying cohort differences in smoking behavior. With this caveat in mind, we nevertheless report the TSLS estimates based on the male-only sample in columns 5 and 6 in Table 5. Regardless of the

choice sample and identification strategy, the estimates in columns 3 to 6 are almost identical.

B. *The Long-Run Impact of Military-Induced Smoking on Mortality*

The results reported in Sections ID and IIA reveal two important findings: veterans from this era suffered increased premature mortality; and military service during WWII and the Korean War caused smoking. In this section, we ask how much of premature veteran mortality was caused by military-induced smoking?

Using the causal estimates for veteran status on mortality reported in Tables 2 and 3 and the IV estimates for veteran status on smoking reported in Table 5, we can approximate the fraction of excess veteran mortality explained by military-induced smoking. For this exercise, we assume that the veteran rate for men born between 1920 and 1939 is 66 percent (as reported in Table 1) and that the overall male ever-smoking rate is 75 percent (as reported in Table 1). We will focus attention on deaths due to heart disease and lung cancer for two reasons. First, these are the causes for which the veteran effect is largest. Second, these are the conditions for which we were able to obtain outside estimates of the fraction of deaths "caused" by smoking. As reported in the introduction, smoking is responsible for approximately 87 percent of lung cancer deaths and approximately 40 percent of deaths due to heart disease.

Column 1 in Table 6 reports the male death rate (per 1,000) due to ischemic heart disease (3.98) and lung cancer (1.85), and column 2 reports the fraction of these deaths attributable to smoking. Multiplying the entries in column 1 by the entries in column 2 and dividing by the 750 smokers per 1,000 men gives the death rate per 1,000 smokers due to smoking, for each

---

[34] In both cases, the Jerry R. Hausman (1978) test, testing the null hypothesis that the difference between TSLS and OLS coefficients is due only to sampling error, is rejected at the 5-percent level.

cause. These death rates are reported in column 3. We then multiply the figures in column 3 by the IV estimate of the effect of veteran status on smoking to obtain the number of additional deaths (per 1,000) caused by military-induced smoking. Given the similarity of the IV estimates reported in Table 5, the choice of a particular point estimate is inconsequential. We therefore use the estimate in column 3 in Table 5 (0.276). The entries in column 4 indicate that smoking induced by military service caused approximately 0.6 additional deaths due to heart disease and lung cancer per 1,000. Finally, we divide the entries in column 4 by the excess veteran mortality estimates for lung cancer and heart disease, from column 2 in Tables 2 and 3, to calculate the fraction of excess veteran mortality that is attributable to military-induced smoking (reported in columns 5 and 6 in Table 6).

The results reported in the last two columns in Table 6 show the relatively large fraction of excess veteran deaths due to heart disease and lung cancer that are attributable to military-induced smoking. For heart disease, military-induced smoking accounts for approximately 64 to 79 percent of excess premature veteran mortality and for lung cancer it accounts for 35 to 58 percent.[35] While smoking does not explain the entire difference in mortality, it is clearly an extremely important component of the mechanism linking military service and subsequent health.

III. Veterans and Disability

We complete our analysis of the long-term impact of military service on health by examining the connection between veteran status and measures of health other than mortality. In particular, we exploit the large samples of microdata from the 1980–1990 Censuses of Population, which contains information on veteran status and disability.[36] Our objective is to

corroborate the mortality evidence with results for other indicators of health.[37]

A. *Census Data*

The samples are constructed in a similar manner to those used in Section II. All samples include men and women born in the continental United States between 1920 and 1939. The 1980–1990 Censuses allow us to track these cohorts between the ages of 40 and 69. Following the definition of veteran in Section I, all men from these cohorts who served in the military are classified as veterans, irrespective of the period of service.

The Census defines a disability as the presence of a "physical, mental, or other health condition which has lasted 6 or more months, and which limits or prevents a certain type of activity." The work disability question therefore provides important information on the long-term health of individuals. Work disability is classified as either being work-preventing or work-limiting (which includes work-preventing).

B. *A Simple Comparison of Veterans and Nonveterans*

We begin our analysis of disability rates and veteran status by comparing disability rates and socioeconomic characteristics across veteran status. Table 7 reports the unadjusted difference in means for male veterans and nonveterans from the 1920–1939 birth cohorts in 1980 and 1990. The entries indicate that veterans are less likely to suffer from work-limiting or work-preventing disabilities by 1 to 2 percentage points. These unadjusted differences are significant and remain roughly constant as the average member of these cohorts age from 50 to 60.

While veterans have lower disability rates than nonveterans, it is important to remember that veterans are positively selected. The bottom panel of Table 7 provides suggestive evidence.

---

[35] Since these estimates are directly proportional to the assumed fraction of deaths caused by smoking, it is straightforward to compute alternative shares of excess veteran mortality attributable to military-induced smoking that correspond to different assumptions on fraction of deaths caused by smoking.

[36] In the 1970 census, the disability and veteran status items were part of two separate questionnaires. As a consequence, the information is not available for the same individuals. We elected not to use these data for this reason.

[37] The National Health Interview Surveys (NHIS) are an alternative source of data to explore veteran morbidity. We have chosen not to use them for two reasons. First, the sample sizes for the 1920–1939 cohorts are too small for examining relatively infrequently observed health problems, like lung cancer. Second, interpreting morbidity incidence is somewhat difficult, since mortality is associated with the key morbidities—lung cancer and heart disease.

TABLE 7—VETERAN-NONVETERAN COMPARISONS, 1980–1990 U.S. CENSUS OF POPULATION

	1980			1990		
	Vets	Nonvets	Difference	Vets	Nonvets	Difference
Disability outcomes						
Work-limiting	0.14	0.15	−0.01*	0.21	0.22	−0.01*
Work-preventing	0.06	0.08	−0.02*	0.13	0.15	−0.02*
Socioeconomic characteristics						
Age	50.56	47.62	2.94*	60.25	57.05	3.20*
Fraction black	0.07	0.15	−0.08*	0.07	0.14	−0.79*
Born in the south	0.33	0.43	−0.10*	0.33	0.41	−0.11*
Fraction high-school graduates	0.36	0.30	0.06*	0.31	0.26	0.05*
Fraction college graduates	0.21	0.17	0.04*	0.22	0.18	0.04*
Currently married	0.85	0.80	0.05*	0.82	0.77	0.05*
Labor force participation	0.91	0.88	0.03*	0.68	0.74	−0.06*
Labor force participation, work-limited	0.54	0.46	0.08*	0.36	0.35	0.01*
Annual wages	27,839	23,308	4,531*	20,384	21,970	−1,585*
Annual income	34,320	28,579	5,741*	33,323	30,399	2,923*
Observations	640,820	361,776	—	611,235	316,774	—

* Indicates that the veteran-nonveteran differences are statistically significant at the 5-percent level.

The differences in average socioeconomic characteristics between veterans and nonveterans are statistically significant at the 5-percent level for all relevant variables. Veterans are slightly older, more educated, wealthier, and more likely to be white, married, and employed. Interestingly, even the labor force participation rate among the work-limited group is higher for veterans. The sign and magnitude of these differences are consistent with the positive selection of veterans from these cohorts. However, some of these socioeconomic characteristics, like educational attainment, may have been altered after military service (see Bound and Turner, 2002; Stanley, 2003).

C. *The Impact of Military Service on Post-Service Health*

To illustrate the conceptual issues, we use a simple linear model. Let y_{ict} be an indicator for the presence of a disability for person i, born in cohort c, observed in year t. Suppose that disability and veteran status are related in the following way:

$$(5) \qquad y_{ict} = \alpha + \beta V_{ic} + X_{ict}\delta + u_{ict}$$

where V_{ic} is a veteran status indicator ($= 1$ if veteran), β is the causal effect of veteran status on disability, and X_{ict} is a vector of observable predictors of health, including a quartic profile in age. We pool the 1980 and 1990 Censuses

because it is impossible to control for age when using year-of-birth dummies as instruments in a single cross section of data. In addition to the quartic age profile, all models include controls for race, education, marital status, and marital status and residence in a metropolitan area.

Table 8 reports a series of OLS and TSLS estimates of effect for military service on disability. The left column reports the results for work-limiting disabilities and the right column reports the results for work-preventing disabilities. Row 1 reports the average male disability rate for the 1980–1990 sample: 17.8 percent of men report being work-limited and 10.2 percent of men report being work-prevented. Row 2 displays the OLS estimates of β.[38] For both disability measures, the estimates indicate that veterans are less likely to suffer from work disability by 1.0 to 1.2 percentage points. Both estimates are statistically significant at the 5-percent level. However, it is unlikely that this simple covariate adjustment adequately controls for the positive selection in the military (i.e., even after conditioning on X_{ict}, u_{ict} and V_{ic} may still be negatively correlated).

To control for the positive selection in the military, which confounds the relationship between veteran status and health, we use the male/female instrumental variables approach used in Sections I and II. This estimation strat-

---

[38] Marginal effects from probit equations are similar.

TABLE 8—IMPACT OF VETERAN STATUS ON DISABILITY

	Work-limiting	Work-preventing
Men only		
1. Mean outcome	0.178	0.102
2. OLS veteran (1 = yes)	−0.010	−0.012
	(0.001)	(0.000)
Observations	1,930,605	1,930,605
Men and Women		
3. TSLS: veteran (1 = yes)	0.095	0.032
	(0.003)	(0.002)
4. F-Statistics on excluded IV [p-value]	8,832.0	8,832.0
	[0.001]	[0.001]
Quartic in age	Yes	Yes
Observations	4,048,052	4,048,052

Note: Eicker-White standard errors are reported in parentheses.

egy is based on the exclusion of year-of-birth dummies interacted with a male dummy in the disability equations, while including unrestricted year-of-birth dummies, a male dummy, and a quartic in age in the first-stage and both disability equations. This permits the identification of the effect of veteran status on disability, while controlling for unrestricted year-of-birth effects, under the assumption that cohort effects are the same for men and women after conditioning on the other covariates.

The TSLS estimates are reported in row 3 in Table 8. The estimates show that veterans are more likely than nonveterans to suffer from work-limiting and work-preventing disabilities. The TSLS point estimates are positive and are larger in magnitude than the OLS estimates, consistent with the fact that veterans are positively selected.[39] As one might expect, given the more severe nature of work-preventing disability, the estimated excess veteran work-preventing disability rates are smaller than those for work-preventing disability: the TSLS point estimate is 3.2 percentage points compared to 9.5 percentage points.

Overall, the evidence in Table 8 corroborates the lower long-run levels of health for veterans reported in Section I. Moreover, the estimated veteran impacts, which range from 3.2 to 9.5 percentage points, are not trivial relative to the disability rates reported in row 1. Perhaps more

importantly, consistent with the mortality estimates, the disability results are also contrary to the notion that the negative health effects of military service disappear relatively quickly after discharge. Finally, the excess disability among veterans reported in Table 8 is even more striking in light of recent evidence documenting the improved health among the older population (see Kenneth G. Manton et al., 1997; David Cutler, 2001).

IV. Conclusion

This paper presents substantial evidence of excess age-adjusted mortality among veterans from the 1920–1939 cohorts, compared to nonveterans from the same cohorts. Moreover, excess veteran mortality is concentrated among two causes, ischemic heart disease and lung cancer. More importantly, we provide smoking-gun evidence of the link between military-induced smoking and long-run increases in premature mortality due to heart disease and lung cancer for veterans. While the near ubiquity of smoking during this period is well known, the role of military service in promoting smoking is less well known. The causal estimates of the impact of military-induced smoking on premature mortality reported in this paper are the first from a large-scale study for a representative sample. Our estimates suggest that 36 to 79 percent of the excess veteran deaths due to heart disease and lung cancer are attributable to military-induced smoking for veterans from the WWII and Korean War era.

Although the United States stopped provid-

[39] A Hausman (1978) test of the null hypothesis that the difference between TSLS and OLS coefficients is due only to sampling error is easily rejected at the conventional level (all *p*-values < 0.001).

ing cigarettes to overseas personnel as part of the K and C rations in 1975, tobacco products are sold at subsidized prices on military bases even today. A direct implication of our results is that the military should reconsider their tobacco subsidy policies and design better programs to reduce smoking incidence among veterans. But even if these changes are made, our results indicate that the VA can expect high medical costs at least until the Korean War veteran cohorts are gone, and potentially longer if other later wartime, or even peacetime, veteran cohorts were similarly induced to begin smoking.

REFERENCES

Almond, Douglas. "Is the 1918 Influenza Pandemic Over? Long-Term Effects of *In Utero* Influenza Exposure in the Post-1940 U.S. Population." Unpublished Paper, 2005.

Angrist, Joshua D. "Lifetime Earnings and the Vietnam Era Draft Lottery: Evidence from Social Security Administrative Records: Errata." *American Economic Review*, 1990, *80*(5), pp. 1284–86.

Angrist, Joshua D.; Imbens, Guido W. and Rubin, Donald B. "Identification of Causal Effects Using Instrumental Variables." *Journal of the American Statistical Association*, 1996, *91*(434), pp. 444–55.

Angrist, Joshua D. and Krueger, Alan B. "Why Do World War II Veterans Earn More Than Nonveterans?" *Journal of Labor Economics*, 1994, *12*(1), pp. 74–97.

Bound, John and Turner, Sarah. "Going to War and Going to College: Did World War II and the G.I. Bill Increase Educational Attainment for Returning Veterans?" *Journal of Labor Economics*, 2002, *20*(4), pp. 784–815.

Card, David and Lemieux, Thomas. "Dropout and Enrollment Trends in the Post-War Period: What Went Wrong in the 1970s?" in Jonathan Gruber, ed., *Risky behavior among youth: An economic analysis*. Chicago: University of Chicago Press, 2001a, pp. 439–82.

Card, David and Lemieux, Thomas. "Going to College to Avoid the Draft: The Unintended Legacy of the Vietnam War." *American Economic Review*, 2001b (*Papers and Proceedings*), *91*(2), pp. 97–102.

Cutler, David. "Declining Disability among the Elderly." *Health Affairs*, 2001, *20*(6), pp. 11–27.

Deaton, Angus S. *The analysis of household surveys: A microeconometric approach to development policy*. Baltimore: Johns Hopkins University Press for the World Bank, 1997.

Deaton, Angus S. and Paxson, Christina H. "Aging and Inequality in Income and Health." *American Economic Review*, 1998 (*Papers and Proceedings*), *88*(2), pp. 248–53.

Fogel, Robert W. "Changes in the Process of Aging during the Twentieth Century: Findings and Procedures of the Early Indicators Project." National Bureau of Economic Research, Inc., NBER Working Papers: No. 9941, 2003.

Gale, Catherine R.; Braidwood, Elizabeth A.; Winter, Paul D. and Martyn, Christopher N. "Mortality from Parkinson's Disease and Other Causes in Men Who Were Prisoners of War in the Far East." *Lancet*, 1999, *354*(9196), pp. 2116–18.

Gruber, Jonathan. "Introduction," in Jonathan Gruber, ed., *Risky behavior among youth: An economic analysis*. Chicago: University of Chicago Press, 2001, pp. 1–27.

Guyer, Bernard; Freedman, Mary A.; Strobino, Donna M. and Sondik, Edward J. "Annual Summary of Vital Statistics: Trends in the Health of Americans during the 20th Century." *Pediatrics*, 2000, *106*(6), pp. 1307–17.

Harris, Jeffrey E. "Cigarette Smoking Practices, Smoking-Related Diseases, and the Costs of Tobacco-Rated Disability among Currently Living U.S. Veterans," Report Commissioned by the Department of Veterans Affairs Assistant Secretary for Policy and Planning, September 15, 1997.

Hausman, Jerry A. "Specification Tests in Econometrics." *Econometrica*, 1978, *46*(6), pp. 1251–71.

Hearst, Norman; Newman, Thomas B. and Hulley, Stephen B. "Delayed Effects of the Military on Mortality." *New England Journal of Medicine*, 1986, *314*(10), pp. 620–24.

Hernandez, Donald J. *America's children: Resources from family, government, and the economy*. New York: Russell Sage Foundation, 1993.

Imbens, Guido and van der Klaauw, Wilbert. "Evaluating the Cost of Conscription in the Netherlands." *Journal of Business and Economic Statistics*, 1995, *13*(2), pp. 207–15.

Keehn, Robert J. "Follow-up Studies of World War II and Korean Conflict Prisoners."

American Journal of Epidemiology, 1980, *111*(2), pp. 194–211.

Klevens, R. Monina; Giovino, Gary A.; Peddicord, John P.; Nelson, David E.; Mowery, Patricia and Grummer-Strawn, Laurence M. "The Association between Veteran Status and Cigarette-Smoking Behaviors." *American Journal of Preventive Medicine*, 1995, *11*(4), pp. 245–50.

Lleras-Muney, Adriana. "The Relationship between Education and Adult Mortality in the United States." *Review of Economic Studies*, 2005, *72*(1), pp. 189–221.

Maddala, Gary S. *Limited-dependent and qualitative variables in econometrics*. Cambridge: Cambridge University Press, 1983.

Manton, Kenneth G.; Corder, Larry and Stallard, Eric. "Chronic Disability Trends in the Elderly United States Populations: 1982–1994." *Proceedings of the National Academy of Sciences*, 1997, *94*(6), pp. 2593–98.

McGill, Henry C., Jr.; McMahan, C. Alex; Herderick, Edward E.; Tracy, Richard E.; Malcom, Gary T.; Zieske, Arthur W. and Strong, Jack P. "Effects of Coronary Heart Disease Risk Factors on Arteriosclerosis of Selected Regions of the Aorta and Right Coronary Artery." *Arteriosclerosis, Thrombosis and Vascular Biology*, 2000, *20*(3), pp. 836–45.

McKinney, W. Paul; McIntire, Donald D.; Carmody, Thomas J. and Joseph, Anne. "Comparing the Smoking Behavior of Veterans and Nonveterans." *Public Health Reports*, 1997, *112*(3), pp. 211–17.

Nefzger, M. D. "Follow-up Studies of World War II and Korean Conflict Prisoners." *American Journal of Epidemiology*, 1970, *91*(2), pp. 123–38.

Page, William F. and Brass, Lawrence M. "Long-Term Heart Disease and Stroke Mortality among Former American Prisoners of War of World War II and the Korean Conflict: Results of a 50-Year Follow-Up." *Military Medicine*, 2001, *166*(9), pp. 803–08.

Page, William F. and Miller, Richard N. "Cirrhosis Mortality among Former American Prisoners of War of World War II and the Korean Conflict: Results of a 50-Year Follow-Up." *Military Medicine*, 2000, *165*(10), pp. 781–85.

Papke, Leslie E. and Wooldridge, Jeffrey M. "Econometric Methods for Fractional Response Variables with an Application to 401(k) Plan Participation Rates." *Journal of Applied Econometrics*, 1996, *11*(6), pp. 619–32.

Pfefferman, D. and Smith, T. M. F. "Regression Models for Grouped Populations in Cross-Section Surveys." *International Statistical Review*, 1985, *53*(1), pp. 37–59.

Seltzer, Carl C. and Jablon, Seymour. "Effects of Selection on Mortality." *American Journal of Epidemiology*, 1974, *100*(5), pp. 367–72.

Smith, James P. "Healthy Bodies and Thick Wallets: The Dual Relation between Health and Economic Status." *Journal of Economic Perspectives*, 1999, *13*(2), pp. 145–66.

Snyder, Stephen E. and Evans, William N. "The Impact of Income on Mortality: Evidence from the Social Security Notch." National Bureau of Economic Research, NBER Working Papers: No. 9197, 2002.

Staiger, Douglas and Stock, James H. "Instrumental Variables Regression with Weak Instruments." *Econometrica*, 1997, *65*(3), pp. 557–86.

Stanley, Marcus. "College Education and the Midcentury GI Bills." *Quarterly Journal of Economics*, 2003, *118*(2), pp. 671–708.

U.S. Bureau of the Census, Department of Commerce. *Statistical abstract of the United States*. Washington, DC: U.S. Government Printing Office, 2000.

U.S. Department of Defense, Office of Inspector General. *Economic impact of the use of tobacco in DOD. Report 97-060*. Washington, DC: Department of Defense, 1996.

U.S. Department of Health and Human Services, National Center for Health Statistics. *Multiple causes of death in the United States*. Hayattsville, MD: U.S. Department of Health and Human Services, National Center for Health Statistics, Multiple years.

U.S. Public Health Service, Office of the Surgeon General. *The health consequences of smoking: Cardiovascular disease: A report of the Surgeon General*. No. 84-50204. Rockville, MD, 1984.

U.S. Public Health Service, Office of the Surgeon General. *Reducing the health consequences of smoking: 25 years of progress. A report from the Surgeon General: 1989*. No. 89-8411. Rockville, MD: Center for Chronic Disease Prevention and Health Promotion, Office of the Surgeon General, 1989.