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ABSTRACT

The Economic Effects of the 1918 Influenza Epidemic*

The 1918-19 influenza epidemic killed at least 40 million people worldwide and 675,000 people in the United States, far exceeding the combat deaths experienced by the US in the two World Wars, Korea, and Vietnam combined. Besides its extraordinary virulence, the 1918-19 epidemic was also unique in that a disproportionate number of its victims were men and women aged 15 to 44, giving the age profile of mortality a distinct 'W' shape rather than the customary 'U' shape, and leading to extremely high death rates in the prime working ages. We examine the impact of this exogenous shock on subsequent economic growth using data on US states for the 1919-30 period. Controlling for numerous factors including initial income, density, urbanization, human capital, climate, the sectoral composition of output, geography, and the legacy of slavery, the results indicate a large and robust positive effect of the influenza epidemic on *per capita* income growth across states during the 1920s.

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I. Introduction

In his Presidential Address to the Economic History Association, Neal (2000, p. 332) argued that his fellow economic historians would do the "economics profession, and the society at large, a big favor if we focused an increasing share of our research efforts on shocks, rather than on longer periods of 'normal' economic change." The 1918 influenza epidemic undoubtedly qualifies as a shock: in the last four months of 1918 and the first six months of 1919, at least 40 million people worldwide died from the influenza epidemic.[1] This death toll exceeds the cumulative twenty-year toll from the AIDS epidemic.

In the United States, Crosby (1989, pp. 206-207) estimated that 675,000 Americans died from influenza and pneumonic complications and that about 550,000 of these were "excess deaths" of Americans who would have otherwise lived during a normal year. These "excess deaths" surpass the number of combat deaths in the U.S. Armed Forces during World War I, World War II, the Korean War, and Vietnam combined.[2] However, the epidemic has been almost completely ignored by economists and economic historians. A comprehensive search of *EconLit* found only two articles relating to the 1918 influenza epidemic, and the epidemic is not even mentioned in any of the leading economic history textbooks or *The Cambridge Economic History of the United States*.[3]

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[1] The most recent estimate of the worldwide number of deaths due to the epidemic is 40 to 50 million (Potter 2001).

[2] Using U.S. Department of Defense and U.S. Coast Guard estimates, Ellis (2001, p. 209) reports 426,704 battle deaths during World War I, World War II, the Korean War, and the Vietnam War.

[3] A search on June 12, 2002 found only the articles by Noymer and Garenne (2000) and Bloom and Mahal (1997b) using the keywords "flu," "influenza," and "1918" separately. In addition, the textbooks by Atack and Passell (1994), Walton and Rockoff (2001), and Cain and Hughes (1997) fail to mention the epidemic.

The 1918 influenza epidemic is an important episode to study not only because of its sheer magnitude, but also because economists know little about how large population and labor force shocks affect economic growth: economic theory offers ambiguous predictions regarding the relationship between negative population shocks and economic growth, and the other major historical pandemics provide inconclusive evidence on the issue. The importance of understanding the relationship is further underscored by the massive loss of life due to AIDS in many developing countries; in part due to lack of evidence the effect of the AIDS epidemic on economic growth in these regions remains an unresolved issue. Although we emphasize below the differences between the influenza epidemic and the AIDS epidemic, the two are clearly linked by the almost incomprehensible scale of the deaths recorded in both crises.

The purpose of this paper is to examine the effects of the 1918-1919 influenza epidemic on subsequent economic growth using data from a sample of U.S. states. We find that the epidemic is positively correlated with subsequent economic growth in the United States, even after taking into account differences in population density, urbanization, levels of income per capita, climate, geography, the sectoral composition of output, human capital accumulation, and the legacy of slavery. Our results suggest that one more death per thousand resulted in an average annual increase in the rate of growth of real per capita income over the next ten years of at least 0.15 percent per year.

II. An Overview of the 1918-1919 Influenza Epidemic

Only three epidemics in world history resulted in mortality approaching or exceeding the mortality caused by the 1918 influenza epidemic: the Plague of Justinian

in the sixth century (100 million lives lost over 50 years), the Black Death of 1348-1351 (62 million), and the current AIDS epidemic (25 million).[4] Below we describe the main features of the epidemic, then turn to the theoretical and empirical evidence regarding the possible links between the epidemic and economic growth.

The influenza epidemic swept the world in three waves: the first in the spring of 1918, the second deadly wave in the fall of 1918, and a third wave that further afflicted some regions in early 1919. The precise origin of the epidemic is unknown, but the first recorded outbreak worldwide occurred in March 1918 among army recruits at Camp Funston, Kansas.[5] The virus spread quickly across the United States and reached Europe in a matter of weeks, apparently with the arrival of American troop ships. The influenza epidemic swept across Europe and had reached India, Australia and New Zealand by June 1918 (Patterson and Pyle 1991).

The virulence and mortality rate of the first wave of influenza only slightly exceeded normal levels and it therefore attracted little attention. The epidemic was, however, characterized by two traits that clearly linked it to the second deadly wave in the fall, and which distinguished the influenza epidemic from any epidemic before or since: it disproportionately killed young adults, and victims died with excessive accumulation of bloody fluid in their lungs, often with severe complications as a result of pneumonia (Crosby 1989).

[4] See Potter (2001) for estimates of the death toll during the Plague of Justinian and the Black Death. The estimate from the AIDS epidemic is UNAIDS (2001, 2000).

[5] Recent research indicates that the epidemic likely first originated in a large French mass transit camp in 1916, where influenza victims exhibited the distinctive symptoms that characterized the later epidemic (Oxford et al. 1999). The authors of this study hypothesize that the virus smoldered for a couple of years before exploding in 1918.

The second wave began in August 1918 with new, deadly outbreaks of influenza occurring nearly simultaneously in Brest, France; Freetown, Sierra Leone; and Boston, Massachusetts. Undoubtedly fueled by the troop movements and demobilization surrounding the end of World War I, the virus spread explosively around the world in the second wave, with worldwide mortality from the epidemic peaking in October and November 1918. A third wave affected some areas of the world in early 1919, principally England and Wales, Australia, and other countries in the southern hemisphere.

In the United States, the impact of the epidemic varied widely across regions and had a profound demographic impact on the country. Approximately 0.66 percent of the U.S. population died during the epidemic, which caused life expectancy at birth to plunge by nearly 12 years for both men and women in 1918 (see Figure 1).

In a typical influenza epidemic, the majority of the victims are young children and the elderly, giving the age profile of mortality a distinct 'U' shape. A distinguishing characteristic of the 1918 epidemic was that it disproportionately killed men and women ages 15 to 44, so that the age profile of mortality instead followed a 'W' pattern. This is illustrated in Figure 2, which plots the age-specific death rates for men and women in the 1918 epidemic and the average rates from 1914 to 1916, along with the same data for whites and nonwhites separately. It is evident from Figure 2 that over one percent of males ages 25 to 34 died as a result of the epidemic. For both whites and nonwhites, the male mortality rate for those ages 15 to 44 exceeded the female mortality rate by 50-75 percent in 1918, in contrast to the non-epidemic years in which the death rates by gender

are virtually identical. The death rate for nonwhites also exceeds that of whites, although the 'W' pattern characterizes the age-specific death rates of both races.[6]

Other than the age, sex, and racial distribution of deaths, little else is known about the pattern of deaths across different subgroups of the population. Some observers argue that there were few differences in mortality rates across income groups (Crosby 1989; Rice 1988), while other evidence suggests that households with higher income levels had lower mortality rates.[7] It does seem clear that the influenza epidemic did not simply kill the weakest members of each cohort. Numerous eyewitness accounts by doctors and other medical personnel attest that influenza killed the most robust individuals in the population. For example, the Acting Surgeon General of the Army remarked that the influenza epidemic "kills the young vigorous, robust adults;" public health specialists agreed as well that most influenza victims were those who "had been in the best of physical condition and freest from previous disease" (quoted in Crosby 1989, pp. 215-16).

The geographic spread of the influenza epidemic in the United States appears to have been arbitrary, with few regional tendencies in the pattern of influenza mortality across states (Figure 3). The states with the highest death rates from the epidemic –

[6] All countries for which age-specific death rates are available also recorded a 'W'-shaped age distribution of mortality; this is true, for example, in India, Australia, New Zealand, and South Africa (Mills 1986; Rice 1990; Union of South Africa 1921).

[7] Door-to-door Public Health Service surveys of more than 100,000 individuals conducted in nine cities during the summer of 1919 suggest that the mortality rate of whites "was nearly twice as great among the 'very poor' as among the 'well-to-do' and those classified as in 'moderate' circumstances" (Sydenstricker 1931, p. 160). These survey results should be weighed with extreme caution. Since the surveys were conducted after the flu epidemic, it is unclear whether the poverty was a cause or a result of flu mortality within each household. In addition, the categorization of economic status was unclear and certainly not comparable across surveyors or cities. Sydenstricker (1931, p. 156) reported that "each enumerator was instructed to record at the time of her visit to the household her impression of its economic condition in one of four categories – 'well-to-do,' 'moderate,' 'poor,' or 'very poor' . . . They were purposely given no standards for comparison . . . [with] the intention being to have them record their own impressions naturally."

Pennsylvania, Maryland, and Colorado – shared few common features climatically or economically, and in some cases neighboring states, cities or even counties experienced highly dissimilar mortality rates during the epidemic. For example, describing the geographic incidence of influenza mortality in Indiana, Ensley (1983, p. 7) writes, "there is no discernible regional pattern in the severity of the epidemic….Unlike previous epidemics which traveled on a slow east-west axis, the Spanish Lady struck in a sudden, random fashion." Cities in the same region experienced markedly different influenza mortality rates; for example the death rate from influenza and pneumonia in Chicago was 5.2 per 1,000 population in 1918, compared with a rate of 2.8 per 1,000 in Grand Rapids, Michigan which is less than 200 miles away.

Some have conjectured that exposure to the first wave of the influenza virus in the spring of 1918 conferred immunity to the second wave, which may explain some of the regional patterns in influenza mortality, but evidence on this issue is unavailable. It is unlikely that differing effectiveness of the public health services across states can explain the variation in state mortality rates, because the public health measures taken by local authorities proved completely ineffective at halting the spread of the virus.[8]

The statistical evidence also supports the notion of influenza mortality as an exogenous shock to the population. There is virtually no relationship between state-level mortality rates from influenza and pneumonia in 1918-19 and levels of real personal per capita income in either 1910 or 1919-21: the simple linear correlation between influenza and pneumonia deaths in 1918-19 and the level of real personal per capita income in 1910 is 0.028, while the correlation between flu deaths and the level of real personal per

capita income in 1919-21 is 0.084. To summarize, state-level mortality rates appear to be randomly distributed and do not seem to be related to the level of economic development, climate or geography. The empirical evidence presented in Section V further supports this conclusion.

III. Theoretical Predictions

Theoretical models of economic growth offer conflicting predictions of whether an influenza epidemic, and the accompanying negative shock to population and the labor force, should increase or decrease the rate of growth of output per capita over the medium and long run. For illustrative purposes, consider two simple models of growth: the neoclassical Solow (1956) model which assumes diminishing marginal returns to capital, and the AK model, based on the work of Romer (1987) and Rebelo (1991), which assumes constant returns to capital accumulation.

The Solow diagram is depicted in Figure 4a, where k is the amount of capital per worker, s is the saving rate, δ is the rate of depreciation, n is the population growth rate, and f(k) is a Cobb-Douglas production function with diminishing marginal returns to capital. If an economy begins at the steady-state level k_0, the initial effect of an influenza epidemic will be to increase the amount of capital per worker and output per worker, moving the economy immediately to k_1. After the initial shock, the economy will gradually converge back to the steady-state level k_0, and the growth rate of output per

[8] For example, in some cities and towns residents were required to wear surgical masks to protect themselves from the virus. Because the influenza virus can penetrate even tightly woven cloth, this measure failed to prevent transmission (Ensley 1983).

worker will be negative during this transition.[9] Moreover, the larger the epidemic, the larger the initial increase in capital per worker and output per worker, and the more negative is the subsequent growth in output per worker as the economy converges back to the steady-state.

In the simple AK model shown in Figure 4b, an influenza epidemic will also initially increase the amount of capital per worker and output per worker. However, in contrast to the Solow model, growth of output per worker will be positive in the years after the initial shock. Immediately after the negative shock to population, the amount of capital per worker increases from k_0 to k_1. In contrast to the Solow model, the subsequent growth rate is positive since the amount of capital per worker continues to increase. In addition, the larger the initial shock the larger the subsequent growth rate of output per worker.

Thus far, we have assumed that the only impact of an influenza epidemic is an initial shock to the population. While there was certainly a large negative shock to the population and the labor force in 1918 and 1919, there is also strong reason to believe that this shock affected population and labor force growth rates substantially beyond these two years. Since a large proportion of the deaths occurred among those ages 15 to 44, the epidemic undoubtedly adversely affected family formation and fertility for years after the epidemic. Data limitations, however, make it difficult to precisely estimate the effect.[10]

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[9] With exogenously given labor-augmenting technological progress, g, the growth rate of output per worker will be slower than g as the economy moves back from k_1 to k_0.

[10] Fertility rates decreased substantially across the 1920s. Keyfitz and Flieger (1968) estimate that the total fertility rate of women ages 15 to 49 decreased from 3.378 children in 1919-1921 to 2.547 in 1929-1931. However, there are undoubtedly many reasons for this decline besides the flu epidemic. These factors include urbanization, increasing incomes, restrictions on immigration, increasing opportunities for young women, etc.

Not only would one expect the population growth rate to be affected by the epidemic, other parameters may also change as a result of an influenza epidemic. For example, the aggregate savings rate may change. Households that experienced the death of the family's primary breadwinner would likely see a decrease in their subsequent savings rates. However, the ninety-nine percent of households that did not have a death in the immediate family, but undoubtedly witnessed the premature deaths of friends and acquaintances, may have increased their precautionary savings as a result. If either the population growth rate or savings rate is affected, then this leads to greater uncertainty regarding the theoretical effect of the epidemic on growth. For example, a sufficient increase in s and decrease in n can generate a positive increase in the growth of output per worker even in the neoclassical Solow model.

The point of this exercise is only to show that even simple models of growth lead to different predictions. More complicated models, such as those that include human capital or models in which the rate of innovation depends positively on the size of the population, lead to further ambiguities.[11]

Finally, it may be the case that a market clearing growth model is not the appropriate way to analyze the effects of a flu epidemic. With price stickiness, the influenza epidemic may have caused shocks to aggregate demand that affected the path of output per capita in the short run. One data limitation discussed in Section V is that we can only examine the growth of real personal per capita income across U.S. states between 1919-21 and 1930. That is, we examine the growth effects immediately after the epidemic. It may be the case that the epidemic caused only a temporary negative shock

to aggregate demand, perhaps as a result of reduced consumer confidence, increased precautionary savings, or business failures and bankruptcies caused by the deaths of hundreds of thousands of breadwinners and business owners. States with the highest mortality rates may have experienced the biggest decreases in per capita income by 1919-1921. What we may be witnessing is not a change in trend, but only a return to trend across the 1920s after a temporary aggregate demand shock.

IV. Related Empirical Evidence

Like the ambiguous theoretical predictions, empirical studies of demographic catastrophes of the past also provide conflicting evidence on the relationship between population health shocks and growth. The most relevant empirical evidence examines the effect of the Black Death in Western Europe and the impact of the 1918 influenza epidemic in India.

The plague killed roughly one-third of the West European population between 1348 and 1351, and recurring epidemics continued to inflict high death tolls on the continent over the next quarter-century. The conventional view of the effect of the plague is that it sharply reduced the size of the working population, leading to a rapid increase in real wages for the laboring classes that persisted into the fifteenth century (Hirshleifer 1987). Bloom and Mahal (1997a) re-examine the effect of the plague on the wages of unskilled agricultural laborers in England and France during the epidemics, and find a positive but statistically insignificant relationship between real wages and population growth in both countries. While the authors conclude that the evidence fails

[11] Mankiw, Romer, and Weil (1992) add human capital to the neoclassical model, while Romer (1990) and Helpman (1991) are emblematic of endogenous growth models where innovation depends positively on the

to support the hypothesis that the Black Death resulted in higher wages for the laboring classes, given the limited data available to study the issue (n=13) the effect of the Black Death on wages remains uncertain.

The same study also examines the effect of the 1918-19 influenza epidemic on acreage sown per capita in India across 13 Indian provinces. As in the case of the plague, the authors find no relationship between the magnitude of the population decline and changes in acreage sown per capita across provinces. However, Schultz (1964, pp. 66-67) argues that the agricultural labor force was reduced by 8 percent as result of the 1918-19 influenza epidemic, but that agricultural production fell by only 3.3 percent, thus implying an increase in per capita output.

A contemporary demographic catastrophe merits discussion as well, although the parallels with the influenza epidemic are less clear than in the case of the Black Death: the current AIDS epidemic in developing countries. While the magnitude of the population shock caused by AIDS will ultimately be at least as severe as that of the influenza epidemic in many countries, and AIDS – like the 1918 influenza epidemic – is primarily affecting prime-age adults, the AIDS epidemic differs from the influenza epidemic in important ways that likely have significant implications for its effect on economic growth. First, in contrast to the influenza epidemic which claimed victims within a matter of days of infection, AIDS is a slowly evolving disease which can be associated with long periods of reduced productivity, high medical expenditures, and extended periods of care by family members. Second, as discussed previously, the influenza epidemic was an exogenous shock in that its mutation into a deadly form in the summer of 1918 and its geographic distribution across regions appear to have been

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size of the market.

randomly determined. The spread and severity of the AIDS epidemic, on the other hand, is likely related to income levels which complicates econometric analyses of its impact with endogeneity problems. Using cross-country data to empirically test the correlation between AIDS incidence and per capita GDP growth, Bloom and Mahal (1997b) find a statistically insignificant coefficient on the AIDS variable and conclude that AIDS has had little impact on growth. However, it is possible that the 1980–1992 period examined in this study is too early in the epidemic to fully assess the effect of AIDS on growth.

To summarize, the predictions of neoclassical and endogenous growth models are ambiguous regarding the impact of an exogenous shock to population and labor force on economic growth. The empirical evidence is inconclusive as well, and is problematic in that other historical episodes of mortality shocks are either affected by endogeneity between mortality and income – such as the AIDS epidemic – or lack sufficient statistical evidence to draw clear conclusions. While data on the 1918 influenza epidemic in the United States are far from perfect, a study of this episode appears to provide a unique opportunity to analyze the effects of a large exogenous shock to population and labor force size on economic growth.

V. Empirical Evidence from U.S. States

A. Data

The dependent variable in the specifications is the growth of real personal per capita income from 1919-1921 to 1930. Easterlin (1957) constructed nominal estimates of state income per capita at twenty-year intervals, reporting the average level of personal per capita income over the 1919 to 1921 period. Barro and Sala-i-Martin (1992) used

these estimates, after deflating the nominal estimates by an aggregate consumer price index, in their study of long run convergence across U.S. states. Their procedure, however, assumes that prices were the same across all U.S. states, which may not have been the case. Lindert (1978), however, has constructed real estimates of personal income per capita after taxes from the nominal Easterlin estimates.[12] Instead of using a national consumer price index, Lindert created regional price indexes from a variety of sources to deflate the nominal estimates for each state. More importantly, he provided estimates for both the period immediately after the epidemic (1919-21) and 1930 as well, thus allowing us to examine growth over the 1920s. In order to avoid the difficulties associated with the Great Depression, we omit the 1930s from the empirical analysis.[13]

One limitation of the real per capita income data is that we only have observations for two points in time: 1919-1921 and 1930. As a result, we first observe real personal per capita income immediately after the epidemic. It would be preferable to have an annual measure for the years both before and after the epidemic, but this is simply not available at the state level. Another limitation is that the personal income measure attributes capital income to the state of the asset holder instead of the state in which the business activity actually occurred. However, Barro and Sala-i-Martin (1992, p. 239) report that the post-World War II results using gross state product (where capital income

[12] More recently, Mitchener and McLean (1999) have published state personal per capita estimates at six census years adjusted for differences in prices and labor input per capita across states. These data are not used for two reasons. Mitchener and McLean (1999, p. 1025) constructed "relative price indexes that measure, for a particular year, how the price level for any given state deviates from the U.S. average. . ." However, this method does not allow for comparisons over time. Second, they only report estimates for 1920 and 1940, making it impossible to examine only the 1920s before the Great Depression. For our purposes, the choice of the price index, whether based on regional prices like Lindert (1978) or aggregate prices like Barro and Sala-i-Martin (1992), is not likely to matter. Mitchener and McLean (1999, p. 1026) show that "part of the regional dispersion in incomes in the Easterlin data for 1880 and 1900 is due to the effect of differences in regional price levels, but this effect is trivial by 1920 and thereafter."

is attributed to the state where the business activity occurs) and personal income "are nearly equal."

The primary explanatory variable is the number of influenza and pneumonia deaths per 1,000 persons in each state in 1918 and 1919 reported in the U.S. Bureau of the Census *Mortality Statistics*. Both influenza and pneumonia are combined in the Census figures because they usually occurred together during the epidemic, and because "it is not believed to be best to study separately influenza and the various forms of pneumonia . . . for doubtless many cases were returned as influenza when the deaths were caused by pneumonia, and vice versa."[14] The primary limitation of the mortality data is that some states are excluded from the sample. In 1918, the Registration Area, the area from which the Census Bureau received transcriptions of all death certificates, contained only 78.6 percent of the total estimated U.S. population.[15] With the exception of data from a few cities outside of this area, only 30 states are included in the Registration Area at the outset of the epidemic.[16] While nearly 80 percent of the U.S. population is included in the sample, most of the least populous states are excluded. As a result, the states in the South and West are underrepresented in the sample, although states from all Census regions are included.

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[13] See Lindert (1978, Appendix G, pp. 381-390) for a complete description. While data for 1929 would have been preferred, the first year of the Great Depression, 1930, was relatively mild.
[14] U.S. Bureau of the Census, *Mortality Statistics 1919*, p. 28.
[15] Ibid., p. 30.
[16] In 1918, the following states were omitted from the Registration Area: Alabama, Arizona, Arkansas, Delaware, Florida, Georgia, Idaho, Iowa, Mississippi, Nebraska, Nevada, New Mexico, North Dakota, Oklahoma, South Dakota, Texas, West Virginia, and Wyoming.

Economic theory and previous empirical work guided the selection of the other explanatory variables.[17] Density, the number of persons per square mile in each state in 1910, is included to ensure that influenza and pneumonia deaths are not simply acting as a proxy for density.[18] Education levels (the share of the population age 6 to 20 in school in 1910) and the share of the population foreign born in 1910 are included to control for differences in labor force skills across states. We control for convergence by including initial income – real income per capita in 1919-1921 – in the regressions, and also include controls for geographic characteristics of the states such as climate. In addition, a dummy variable for Southern states is included to account for the legacy of slavery, which may have prevented convergence of the South before the imposition of federal minimum wage laws during the New Deal (Wright 1986).[19] Finally, Barro and Sala-i-Martin (1992) find that the beginning of the decade agricultural share of personal income is an important control variable in their study of convergence across U.S. states during the 1920s. They find that states with large agricultural shares grew more slowing during the 1920s as a result of decreasing farm prices and land values. Moreover, this is an important control variable in our study because one might expect agricultural states to be more rural and to suffer lower influenza mortality rates as a result. The precise sources and definitions of all of the variables are listed in Appendix 1.

[17] One advantage of using data from U.S. states rather than cross-country data is that U.S. states are relatively homogenous. As a result, we do not need to include the array of variables to control for institutional differences that have been so difficult to control for in cross-country growth studies.
[18] Various measures of urbanization were also tried as well. These results are discussed in Section V.C.
[19] In addition to using a Southern dummy variable, the percent of the state population that were slaves in 1860 was also examined as an explanatory variable. These robustness checks are discussed in Section V.C.

B. Results

As is evident from Table 1, the size and significance of the flu coefficient is remarkably robust and stable across various empirical specifications. The flu coefficient ranges between 0.219 and 0.235 in the specifications in Table 1, and is always statistically significant at the 5 percent level or lower. Column (1) reports the general unrestricted model including all of the explanatory variables described above. In addition, density, the proportion of the population foreign born in 1910, and initial agricultural share are also statistically significant. It is clear from Table 1 that the flu variable is not just serving as a proxy for density since it remains positive and significant even with density included.

The specification in column (2) was obtained by using PcGets (ver. 1.0), an econometric modeling program designed to implement the general-to-specific approach to econometric modeling often associated with the London School of Economics.[20] PcGets automatically selects an undominated, congruent model even though the precise formulation of the econometric relationship is not known *a priori*. While the size of the flu coefficient is quite similar to the general specification in column (1), the standard error is smaller so that it is now significantly different from zero at the 1 percent significance level. Initial income, density, foreign-born, and the initial agricultural share are the other robust variables in the restricted model.

The specifications in columns (3) through (5) illustrate that the size and significance of the flu coefficient is not greatly affected by the inclusion or omission of the other explanatory variables. The regression in column (3) includes both the initial

level of real income per capita and flu deaths. The insignificant and economically small coefficient on initial income suggests that there was no convergence taking place across states in the 1920s without controlling for the initial agricultural share.[21] Column (4) is consistent with Goldin's (1998) work on the importance of the high school movement and human capital accumulation in the early twentieth century, while column (5) reports the simple ordinary least-squares relationship between growth and flu deaths.

The results reported in Table 1 strongly suggest that influenza and pneumonia deaths are nearly orthogonal to all of the other explanatory variables in the sample. This result formalizes what Crosby (1989, p. 66) suspected when he concluded that "the states with the highest excess mortality rates – Pennsylvania, Montana, Maryland, and Colorado – had little indeed in common economically, climatically or geographically." Regardless of the specification, the size and significance of the flu coefficient remains quite constant.

The results shown in Table 2 replace the total state-level mortality rate from flu and pneumonia with the state-level mortality rates of those in the prime-working ages, defined as those ages 10-44 in 1918-1919.[22] As is evident from Table 2, the size of the flu coefficients are similar to those reported in Table 1, and the coefficient is still statistically significant at the 5 percent or 1 percent level in all specifications. The coefficient on flu-prime age ranges from 0.157 in the general unrestricted model [column (1)] to 0.281 in the simple ordinary least-squares equation [column (5)]. The initial

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[20] Phillips (1988) and Hendry (1995) explain the general-to-specific methodology in detail. Hoover and Perez (1999, 2001) show that the general-to-specific modeling strategy often reaches a specification near the true data generating process and outperforms many other specification search procedures.

[21] These results are consistent with those reported by Barro and Sala-i-Martin (1992, Table 1, line 14, p. 231) who also find evidence of convergence conditional on the initial agricultural share. It evident that the 1920s were a very difficult time for farmers and that states with larger agricultural sectors grew more slowly during the 1920s.

agricultural share remains negatively and significantly related to subsequent growth, and there is also support for convergence across the 1920s. The specific model suggested by PcGets [column (2)] includes prime age flu mortality, initial income, percent foreign, and the initial agricultural share. Along with the flu variables, these other three variables appear in both of the specific models in Tables 1 and 2. Once again, there is evidence that the lagged schooling share is positively and significantly related to subsequent economic growth [column (4)].

The key finding in Tables 1 and 2 is that both the total mortality rate from influenza and pneumonia in 1918 and 1919, and the mortality rate of those of prime working age, is significantly and positively related to the subsequent growth in real income per capita from 1919-1921 to 1930 across U.S. states.

C. Tests of Robustness

It is possible that the regressions reported in Tables 1 and 2 contain specification errors, such as omitted variable bias, that could cause the positive and statistically significant impact of the flu on growth to be spurious. However, the diagnostic tests reported in the lower panels of Tables 1 and 2 suggest that none of the equations suffer from non-spherical errors, as there is no evidence of non-normality or autoregressive conditional heteroskedasticity. In addition to these tests based on the residuals themselves, we also include a general test for misspecification; the RESET (regression error specification tests) based on the work of Ramsey (1969). While a significant RESET test could indicate the evidence of omitted variable error or functional form misspecification, there is no evidence for this as reported in Tables 1 and 2.

[22] While Figure 2 shows that mortality rates peaked in the 15 to 44 year age group, different age groupings between the *Census of Mortality* statistics and the *Bureau of the Census* population estimates meant that the

The results are also robust to the inclusion of several alternative explanatory variables. Density (persons per square mile) may not adequately capture the importance of population clusters for growth and these population clusters may be highly collinear with flu mortality, thereby implying that the flu results may be spurious. To account for this possibility, we also examine several measures of urbanization in place of the density variable included in Tables 1 and 2. We first include the Census definition of urbanization at the time (the percent of the population living in towns and cities of 2,500 or more). This definition may not, however, adequately capture the importance of larger centers of population. In addition to the Census definition, we also include the percent of persons living in cities with populations of greater than 50,000 and greater than 100,000 in each state in 1920.[23] McLean and Mitchener (2001) also show that the percent of a state's population that were slaves in 1860 is perhaps a better predictor of subsequent growth than the South dummy variable included in Tables 1 and 2.

None of these changes substantially impact our results. For example, with percent slave and percent living in cities of greater than 50,000 included, instead of density and South, the results are quite similar. The flu coefficient increases from 0.219 in column (1) of Table 1 to 0.236 and is significant at the 1 percent level instead of the 5 percent level as reported in Table 1. However, the adjusted R^2 is higher in the original specification (0.471 vs. 0.431).

The results using prime age flu with percent slave and urbanization are robust as well. The coefficient on prime age flu increases from 0.157 to 0.221, and is now significant at the 1 percent level instead of the 5 percent level in Table 2. The adjusted

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10 to 44 age group had to be examined instead.

R^2 increases from 0.428 in Table 2 to 0.448 with urbanization (percent in cities with population of 50,000 or greater) and percent slave replacing density and South. The results do not change markedly when other alternative definitions of urbanization are used as well.[24]

There is also the concern that flu mortality could just be serving as a proxy for the general health status of the population. If influenza death rates are negatively correlated with general health status and health status is important for growth, then our results could be spurious as well. In many ways, the results in Tables 1 and 2 already account for this since the regressions include the level of real income per capita, climate, density, and South, and all of these variables are likely correlated with general health status. Noymer and Garenne (2000) have argued that the reason for the high rates of flu mortality, and the disproportionately higher flu deaths for men and those in the prime working ages, was the high incidence of tuberculosis, particularly for young males. However, there is no statistically significant correlation between tuberculosis death rates in 1915 and influenza death rates in 1918 and 1919. Moreover, the coefficients on flu and prime age flu both remain near 0.2 and statistically significant at the 5 percent levels with the inclusion of the 1915 TB death rates added to the base regressions in Tables 1 and 2. In both cases, the TB coefficient is near zero and far from statistically significant.

It is also possible that the epidemic resulted in a negative shock to aggregate demand that caused real per capita incomes to fall by 1919-1921, so that the observed increase we observe to 1930 is partly or largely a return to an unchanged long run trend. Unfortunately, there is still great uncertainty regarding the timing and severity of

[23] The authors wish to thank Kris Mitchener for this suggestion and for providing us with the urbanization and percent slavery variables from McLean and Mitchener (2001).

business cycles in the years during and after the epidemic, even at a national level. According to the NBER business cycle chronology, there was a cyclical peak in August 1918 and a trough in March 1919. These dates are almost exactly coincident with the epidemic that began in August 1918 and had nearly run its course by March 1919. Another peak occurs in January 1920, followed by a trough in July 1921. Although there is debate regarding how far below trend the economy was in 1919 to 1921, there is no doubt that the economy was generally below trend during these years.[25] Moreover, what matters to us are not the national aggregates, but the differential impact across U.S. states. To measure this differential impact, we use one of the few comprehensive data series on business conditions across U.S. states during this time: Dun's business failure rate data.[26]

While both measures of the flu are positively correlated with subsequent business failure rates in 1919, 1920, and 1921, these correlations are not statistically significant. However, there is evidence that consumption may have fallen and savings increased as a result of the epidemic. The simple correlation between flu and pneumonia mortality and the change in the average deposits per depositor from 1918 to 1919 is 0.445 (p-value = 0.057). Although this evidence is consistent with the view that the influenza epidemic caused the economy to fall below trend by 1919, the available evidence is not conclusive. Moreover, including business failure rates in the general specifications reported in Tables 1 and 2 do not significantly alter the results.

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[24] All of these results are available from the authors.
[25] See Romer (1988) and Balke and Gordon (1989) for a discussion of this period.
[26] The total number of firms listed is well over one million and included most manufacturing, retailing, wholesale, transportation and contracting firms in the U.S. However, the professions, farms, railroads, amusements, one-person services and firms in the "FIRE" sector (finance, insurance, and real estate) are excluded. A failure is defined as a closure leading to or likely to lead to a loss to creditors. Mergers and acquisitions are excluded.

To further examine the robustness of our results, we replicate the general unrestricted regressions in Table 1 for each decade in the post-World War II period using both contemporaneous influenza and pneumonia death rates and 1918-1919 rates.[27] If influenza and pneumonia deaths are correlated with an omitted variable that generally causes growth, then we should find that the flu variable is important in other time periods as well. The results reported in Table 3 include influenza and pneumonia deaths for the two years immediately proceeding each decade. For example, the 1950s regression includes 1948 and 1949 death rates from influenza and pneumonia, while the 1960s regression includes 1958 and 1959 death rates and so on. Although the flu coefficient is significant at the 10 percent level for the 1950s, it is far from significant for any of the other decades in the postwar period.

The results reported in Table 4 include 1918-1919 influenza and pneumonia death rates in each of the postwar regressions. The 1918-1919 flu coefficients are close to zero and statistically insignificant in all of the postwar decades. If influenza mortality rates are correlated with any omitted variable that generally causes growth, then the flu coefficients in Tables 3 and 4 should be significantly different from zero. In addition, if the impact of the 1918-1919 epidemic substantially affected growth during the postwar period, we would also expect to find a significant 1918-1919 flu coefficient. We find no evidence that this is the case, thus lending support to the conclusion that the results for the 1920s are not spurious. The results reported in Table 4 also show that the impact of the 1918 epidemic was no longer evident by the 1950s, suggesting that the large effects initially observed during the 1920s were only transitory and did not affect the long run growth trend.

[27] The postwar data are available from the authors.

VI. Conclusion

The death toll exacted by the 1918-1919 influenza epidemic was one of the highest ever recorded during a health crisis in world history. The epidemic disproportionately claimed young adults, and, although fueled by the troop movements that accompanied the end of World War I, the geographic distribution of influenza mortality across the United States appears to have been largely random. This exogenous shock to population size provides a unique opportunity to examine the impact of a large population decline on subsequent economic growth in an episode that is largely free of the endogeneity between economic growth and mortality that affects other historical episodes of population health shocks.

This study finds a large, positive effect of mortality from the 1918-1919 influenza epidemic on growth over the next decade across U.S. states in the Registration Area, and this result is robust across a variety of specifications.[28] Identical tests of the relationship between influenza mortality and economic growth in subsequent decades across U.S. states indicate that the positive coefficient on influenza mortality is not simply acting as a proxy for the interconnectedness of a region's population that may positively contribute to growth. Along with conditional convergence, the rise of education, and agricultural difficulties, the lingering impact of the influenza epidemic also appears to be an important part of the economic history of the 1920s.

[28] Obviously, this result does not imply that the epidemic improved social welfare. Growth in income per capita is only one component of well-being, and does not include the pain and suffering of the friends and families of the 675,000 victims.

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Appendix 1: Data Sources

U.S. States: Interwar Period

Data Name	Description	Source
Growth	Average annual growth rate of real personal income per capita after taxes between 1919-21 and 1930, computed as the log differences and expressed as a percent.	Lindert (1978), Table G-6, p. 390.
Flu	Influenza and pneumonia deaths per 1,000 population in 1918 and 1919.	U.S. Bureau of the Census, *Mortality Statistics 1920*, p. 30.
Flu - prime age	Influenza and pneumonia deaths per 1,000 population aged 10-44 in 1918 and 1919.	U.S. Bureau of the Census, *Mortality Statistics 1918*, and *Mortality Statistics 1919*, Table 8 for both years for the numerator. The denominator [state populations at various ages from Miller and Brainerd (1957), Table L-2].
Initial income	Real personal income per capita after taxes in 1919-21 (1960 consumer dollars).	Lindert (1978), Table G-6, p. 390.
Climate	Number of average annual cooling degree-days defined as the number of degrees the air temperature is above 65 degrees Fahrenheit multiplied by the number of days. The average of the entire length (years) of recorded temperatures was used.	*Statistical Abstract of the United States* (2001), Table No. 377. If a state has more than one reporting station, then an arithmetic average of all the stations in that state was computed.
Density	Persons per square mile in 1910.	Total population in 1910 from the *United States Historical Census Data Browser* (http://fisher.lib.virginia.edu/census/).

Foreign	Percent of persons foreign born in 1910.	*United States Historical Census Data Browser* (http://fisher.lib.virginia.edu/census/).
School	Percent of persons age 6-20 in school in 1910	*United States Historical Census Data Browser* (http://fisher.lib.virginia.edu/census/).
South	Dummy variable equal to one if the state was a member of the Confederacy, and zero otherwise.	
Initial agricultural share	Farm income as a percent of personal income in 1920.	Barro and Sala-i-Martin (1992). Data (AGRY20) from http://www.columbia.edu/~xs23/data.htm.
Deposits	Average deposits per depositor in mutual and savings banks in 1918 and 1919.	*Statistical Abstract of the United States* (1918,1919).
Business failure rate	Number of business failures divided by number of business concerns, expressed as a percent.	*Statistical Abstract of the United States* (1921), pp. 292-293 (original source: *Dun's Review*, NY: NY).

Figure 1. U.S. Male and Female Life Expectancy at Birth, 1900-1945

Figure 2a. Age-Specific Death Rates from Influenza and Pneumonia, United States

Figure 2b. Age-Specific Death Rates, Nonwhite and White Men

Figure 2c. Age-Specific Death Rates, Nonwhite and White Women

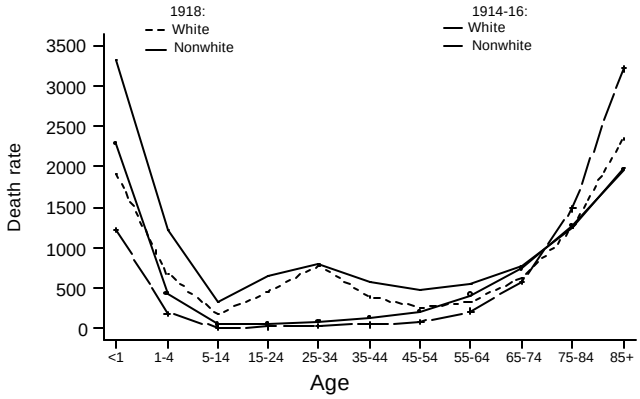

Figure 3. Influenza and Pneumonia Death Rates, 1918-1919

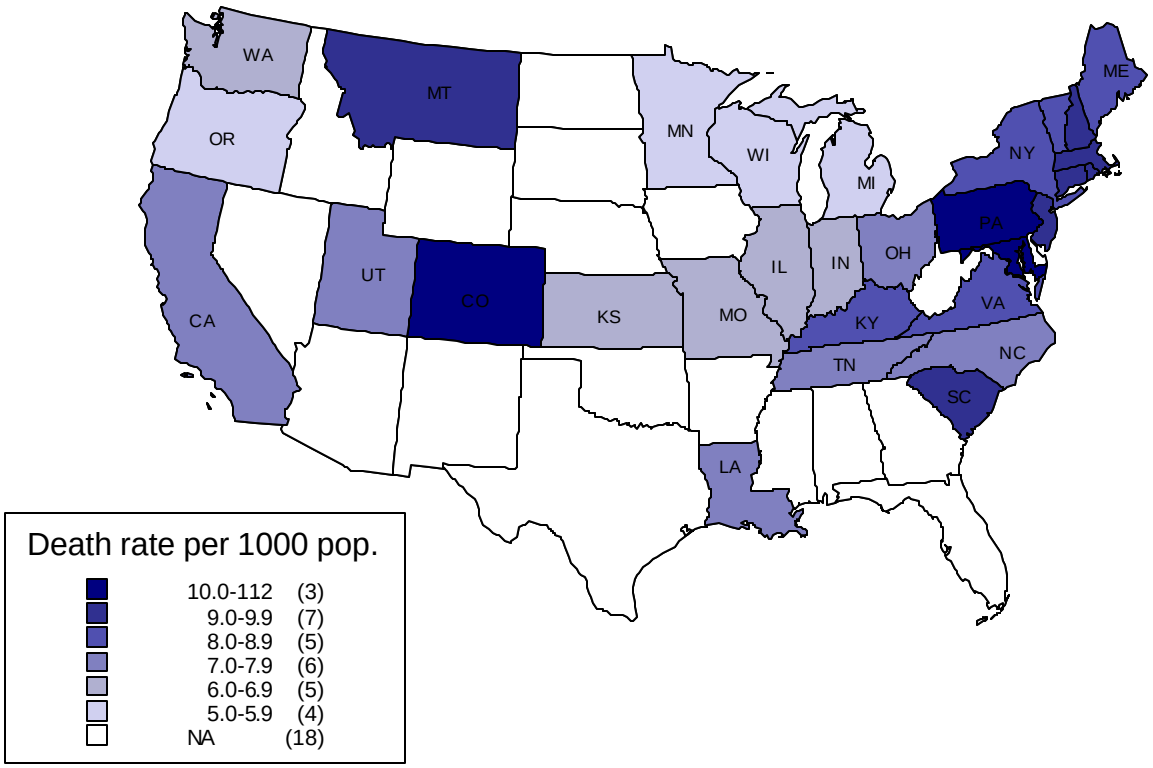

Figure 4a.

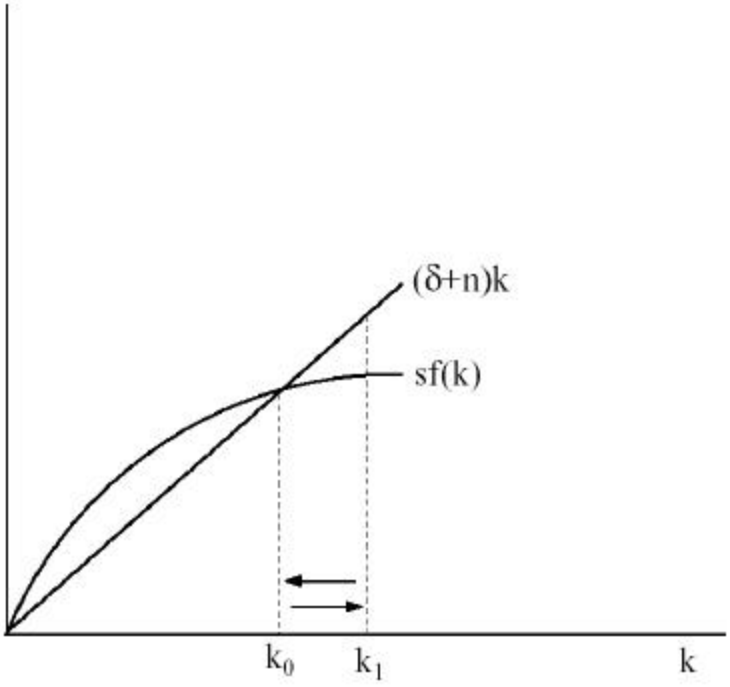

Figure 4b.

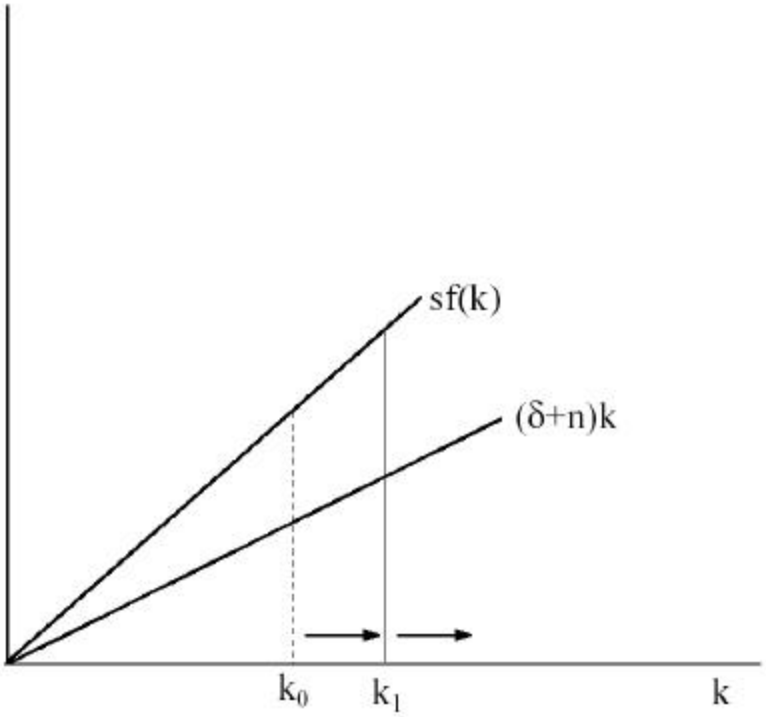

Table 1
Dependent Variable: Average Annual Growth in
Real Personal Per Capita State Income from 1919-21 to 1930

	(1)	(2)	(3)	(4)	(5)
Constant	-2.448		-2.818***	-4.992**	-2.031***
	(3.411)		(0.921)	(1.955)	(0.629)
Flu	0.219**	0.221***	0.222**	0.226***	0.235***
	(0.078)	(0.065)	(0.083)	(0.081)	(0.083)
Initial income	-0.002**	-0.002***	0.001	-0.001	
	(0.001)	(0.0005)	(0.001)	(0.001)	
Climate	0.0004				
	(0.0007)				
Density	-0.003**	-0.0036**			
	(0.001)	(0.0014)			
Foreign	0.080***	0.081***			
	(0.027)	(0.020)			
School	0.045			0.076**	
	(0.044)			(0.032)	
South	-0.235				
	(0.646)				
Initial agricultural share	-0.064**	-0.053***		-0.048*	
	(0.029)	(0.016)		(0.027)	
R^2	0.617	0.576	0.220	0.417	0.155
Adj. R^2	0.471	0.508	0.163	0.324	0.125
Schwarz criterion	2.829	2.476	2.858	2.794	2.826
Jarque-Bera normality (p-value)	0.541	0.715	0.168	0.973	0.159
ARCH (p-value)	0.824	0.874	0.931	0.641	0.918
Ramsey RESET (p-value)	0.996	0.487	0.325	0.911	0.801
N	30	30	30	30	30

Notes: White (1980) heteroskedasticity-consistent standard errors are in parentheses. * denotes significance at the 10 percent level, ** denotes significance at the 5 percent level, and *** denotes significance at the 1 percent level. See Appendix 1 for the precise definitions and sources of the variables above.

Table 2
Dependent Variable: Average Annual Growth in
Real Personal Per Capita State Income from 1919-21 to 1930

	(1)	(2)	(3)	(4)	(5)
Constant	-1.292		-2.714***	-4.020**	-2.125***
	(3.582)		(0.777)	(1.472)	(0.869)
Flu-prime age	0.157**	0.165**	0.263***	0.233***	0.281***
	(0.071)	(0.065)	(0.112)	(0.065)	(0.061)
Initial income	-0.002**	-0.002***	0.0007	-0.001	
	(0.001)	(0.0005)	(0.0007)	(0.001)	
Climate	-0.0003				
	(0.0007)				
Density	-0.002				
	(0.002)				
Foreign	0.066**	0.060**			
	(0.031)	(0.022)			
School	0.039			0.064**	
	(0.046)			(0.027)	
South	-0.153				
	(0.710)				
Initial agricultural	-0.062**	-0.032**		-0.047*	
share	(0.029)	(0.013)		(0.025)	
R^2	0.586	0.498	0.327	0.476	0.286
Adj. R^2	0.428	0.441	0.277	0.392	0.260
Schwarz criterion	2.906	2.531	2.712	2.688	2.658
Jarque-Bera normality	0.433	0.967	0.617	0.849	0.257
(p-value)					
ARCH (p-value)	0.961	0.406	0.739	0.897	0.861
Ramsey RESET	0.661	0.420	0.567	0.713	0.634
(p-value)					
N	30	30	30	30	30

Notes: White (1980) heteroskedasticity-consistent standard errors are in parentheses. * denotes significance at the 10 percent level, ** denotes significance at the 5 percent level, and *** denotes significance at the 1 percent level. See Appendix 1 for the precise definitions and sources of the variables above.

Table 3
Postwar Regressions
Beginning of the Decade Influenza and Pneumonia Death Rates
Dependent Variable: Average Annual Growth in
Real Personal Per Capita State Income

	1950s	1960s	1970s	1980s	1990s
Constant	3.093**	3.860*	1.030	-2.172	1.794
	(1.525)	(2.023)	(1.979)	(4.467)	(2.302)
Flu	0.649*	-0.010	-0.012	1.250	0.568
	(0.328)	(0.449)	(0.507)	(1.143)	(0.499)
Initial income	-0.0004***	-0.0003***	-0.00010	-0.00017*	-0.00002
	(0.00007)	(0.00007)	(0.0000)	(0.00008)	(0.00003)
Climate	0.00008	0.00008	0.00002	-0.0002*	-0.0002***
	(0.00009)	(0.00006)	(0.00006)	(0.0001)	(0.00006)
Density	-0.0003	0.0007*	-0.0003	0.0018***	0.00005
	(0.0006)	(0.0004)	(0.0003)	(0.0005)	(0.0003)
Foreign	0.031*	0.0146	-0.055	0.059	-0.045***
	(0.017)	(0.019)	(0.034)	(0.043)	(0.016)
School	0.008	0.015	0.032	0.069	0.002
	(0.019)	(0.026)	(0.214)	(0.059)	(0.027)
South	-0.019	0.504**	0.111	0.790***	0.320**
	(0.251)	(0.209)	(0.162)	(0.282)	(0.135)
Initial agricultural share	-0.038***	-0.002	-0.039***	-0.023	0.008
	(0.009)	(0.013)	(0.013)	(0.051)	(0.028)
R^2	0.686	0.715	0.458	0.530	0.523
Adj. R^2	0.622	0.657	0.347	0.434	0.428
Schwarz criterion	1.509	1.259	1.440	2.177	1.116
Jarque-Bera normality (p-value)	0.673	0.928	0.000	0.415	0.099
ARCH (p-value)	0.311	0.161	0.457	0.951	0.371
Ramsey RESET (p-value)	0.221	0.161	0.615	0.021	0.176
N	48	48	48	48	49

Notes: White (1980) heteroskedasticity-consistent standard errors are in parentheses. * denotes significance at the 10 percent level, ** denotes significance at the 5 percent level, and *** denotes significance at the 1 percent level. See Appendix 1 for the precise definitions and sources of the variables above.

Table 4
Postwar Regressions
1918-1919 Influenza and Pneumonia Death Rates
Dependent Variable: Average Annual Growth in
Real Personal Per Capita State Income

	1950s	1960s	1970s	1980s	1990s
Constant	4.982***	9.492***	-1.856	7.436	-2.143
	(1.727)	(3.131)	(2.862)	(7.138)	(2.093)
Flu (1918-1919)	0.029	0.001	0.064	0.076	-0.031
	(0.057)	(0.043)	(0.038)	(0.083)	(0.049)
Initial income	-0.0003***	-0.00015	0.00001	-0.00003	0.000006
	(0.00009)	(0.00010)	(0.00007)	(0.00010)	(0.00004)
Climate	0.0002	0.00002	0.00004	-0.0005	0.00002
	(0.0003)	(0.0002)	(0.0002)	(0.0003)	(0.0001)
Density	-0.001	0.0004	-0.0005	0.001*	-0.0003
	(0.0008)	(0.0003)	(0.0003)	(0.0006)	(0.0004)
Foreign	0.050**	0.034	-0.072*	0.040	-0.450**
	(0.024)	(0.023)	(0.038)	(0.053)	(0.0176)
School	-0.017	-0.070	0.046	-0.055	0.049**
	(0.019)	(0.042)	(0.032)	(0.085)	(0.236)
South	-0.207	0.292	0.330	0.544	-0.052
	(0.397)	(0.316)	(0.403)	(0.451)	(0.191)
Initial agricultural share	-0.050*	0.024	-0.032	-0.142	-0.058
	(0.027)	(0.033)	(0.023)	(0.156)	(0.081)
R^2	0.619	0.757	0.650	0.555	0.352
Adj. R^2	0.474	0.665	0.517	0.385	0.106
Schwarz criterion	1.608	1.186	1.000	2.303	1.189
Jarque-Bera normality (p-value)	0.588	0.175	0.692	0.596	0.091
ARCH (p-value)	0.785	0.353	0.307	0.764	0.982
Ramsey RESET (p-value)	0.001	0.363	0.076	0.013	0.804
N	30	30	30	30	30

Notes: White (1980) heteroskedasticity-consistent standard errors are in parentheses. * denotes significance at the 10 percent level, ** denotes significance at the 5 percent level, and *** denotes significance at the 1 percent level. See Appendix 1 for the precise definitions and sources of the variables above.