

ABSTRACT

Title of dissertation: MODERATE SHOCKS TO WEALTH AND
 HEALTH: ESTIMATES OF THEIR IMPACT ON
 THE MORTALITY OF ELDERLY

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In Chapter One, we examine evidence for a causal connection between income and mortality. There is widespread and longstanding agreement that life expectancy and income are positively correlated. However, it has proven much more difficult to establish a causal relationship since income and health are jointly determined. We use a major change in the Social Security law as exogenous variation in income to examine the impact of income on mortality in an elderly population. We compare mortality rates after age 65 for males born in the second half of 1916 and the first half of 1917. Data from restricted-use versions of the National Mortality Detail File combined with Census data allows us to count all deaths among elderly Americans between 1979 and 1993. We find that the higher income group has a higher mortality rate, contradicting the previous literature. We also found that the younger cohort responded to lower incomes by increasing post-retirement work effort. These results suggest that moderate employment has beneficial health effects for the elderly.

In Chapter Two, we examine another potential determinant of mortality among the elderly, pre-natal shocks to the mother's health. D.J.P. Barker has presented epidemiologic evidence that maternal health status affects the later-life mortality of children. We use the 1918 influenza epidemic as a health shock which is orthogonal to chronic health status. Our findings are that the influenza-exposed cohorts do not experience significantly higher mortality. This allows us to bound any Barker effect of the epidemic as raising mortality less than ten percent.

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by

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DEDICATION

To the memory of Henry Nogi and David Snyder, my first instructors

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CHAPTER ONE
THE IMPACT OF INCOME UPON MORTALITY: EVIDENCE
FROM THE SOCIAL SECURITY NOTCH

1. Introduction

In 1996, the Advisory Commission to Study the Consumer Price Index, informally known as the Boskin Commission, released a report concluding that the Consumer Price Index (CPI) overstated the growth in prices by about 1.1 percentage points per year. Because payments in many Federal programs are indexed by the CPI, the Commission concluded that this over-indexation has contributed to an excessive growth in these programs. The Commission calculated that over the period 1997–2008, the over-indexation of Federal programs would add an additional \$1.07 trillion to the national debt. In their recommendations, the Commission suggested that if “...the purpose of indexing is accurately and fully to insulate the groups receiving transfer payments ...” then “[t]his could be done in the context of subtracting an amount partly or wholly reflecting the over-indexing from the current CPI-based indexing.” (p. 9)

The recommendation to adjust the CPI downward was criticized by a number of groups including those representing unions and senior citizens. The incomes of these constituencies are in many cases tied to the CPI and any adjustment downward in how inflation is calculated would reduce future incomes for these groups. Those testifying before Congress painted a grim picture of the elderly on fixed incomes

forced to choose between purchasing food or prescription drugs, a situation that would obviously be made worse if cost of living adjustments to Social Security payments were more modest after the CPI was adjusted downward. Testimony from a number of witnesses even suggested that adopting the Boskin Commission's recommendations would raise mortality rates among the elderly.

While the identity of the groups objecting to the Boskin Commission's recommendations is predictable, some of their concerns are not without empirical backing. A large body of literature that spans many disciplines has established that those with lower incomes have poorer health outcomes and higher mortality rates (Kitigawa and Hauser, 1973; Duleep, 1986; Wolfson et al., 1993; Fuchs 1993; Chapman and Hariharan, 1994; McDonough et al., 1997; Ettner, 1996; Lantz et al., 1998; and Deaton and Paxson, 1998 and 1999). A relationship between health and socioeconomic status (SES) has been documented for virtually all measures of health (infant mortality, mortality, disease incidence, health habits, and violence) and SES (income, wealth, occupation, and education¹), within many countries (including Canada, the United Kingdom, The Netherlands, Sweden, France, the United States²) and over time,³ and recent research suggests that the statistical correlation between SES status and mortality may have actually increased over the past 40 years (Feldman

¹ See Menchik, 1993; Marmot and various co-authors, 1984, 1987, and 1991; and Townsend et al., 1988.

² See Wolfson et al., 1993; Marmot et al., 1991; Townsend et al., 1988, Kunst et al., 1990; and Feinstein, 1993.

³As one example, Adler et al. (1993) cite evidence that wealthy Rhode Island taxpayers in the 1860s had mortality rates less than half the population average.

et al., 1989; Pappas et al., 1993; Preston and Elo, 1995; Deaton and Paxson, 1998). A large literature also exists about the correlation between socioeconomic status and health in an elderly population, the particular interest of this paper (Mare, 1990; Menchik, 1993; Smith and Kington, 1997).

It has, however, been difficult isolating income as the causal element in this relationship. The inferential problems are described in detail by Smith (1999). There is for example the simple problem that an equally large literature demonstrates that poor health reduces earnings (Haveman et al., 1995; Bound, 1989) and therefore, low current income may be caused by poor health and not the other way around. Smith (1999) also demonstrates that the onset of a poor health shock greatly increases a families' out of pocket health expenses, possibly decreasing resources available in the future. Likewise, low income and high mortality may reflect outcomes of the same process, thereby subjecting the income/mortality relationship to an omitted variables bias. For example, Fuchs (1982) suggests that poor health and low income may both be generated by high discount rates. A high discount rate will discourage both investment in human and health capital, thereby lowering income and raising mortality (Farrell and Fuchs, 1982). This hypothesis is bolstered by evidence which suggests those with lower education have higher mortality and much poorer health habits (Kenkel, 1991; Pincus et al., 1987; Adler et al., 1993; Evans and Montgomery, 1994; and Evans, Ringel and Stech, 1999).

One could isolate the impact of income on mortality by assigning different groups higher or lower income independent of observed characteristics. Absent this ideal research design, we must find field variation in income that mimics random

assignment. Unfortunately, finding such variation has proven to be difficult. The heart of this paper is the use of what is frequently termed the “benefits notch” in Social Security as an exogenous source of variation in the income of Social Security beneficiaries. Concerned with rapidly increasing benefit payments, in 1977, the Federal government changed the way benefits were calculated for new beneficiaries, substantially decreasing the size of payments for recipients born after January 1, 1917. As a result of these changes, two people with identical earnings histories but different birth dates would receive substantially different retirement incomes. Those born after the Notch had little time to adjust since the changes happened late in their work lives. Most, for example, did not realize the impact of the law changes on payments until after they retired.

We examine the link between income and health in an elderly population by estimating a reduced-form relationship between the benefits Notch and mortality. Our econometric model is a simple difference-in-difference estimator where we compare five-year mortality rates for those born in the fourth quarter of 1916 with those born in the first quarter of 1917. Because there may be consistent differences in mortality based upon quarter of birth, we use as a comparison group mortality of men born in the fourth quarter of 1915 and the first quarter of 1916. Because there may be cohort-specific differences in mortality rate, we use as a second comparison group women born in 1916:4 and 1917:1. As we demonstrate empirically below, going into retirement, there is little to distinguish those born just before and after January 1, 1917. In the 1970 Census for example, there is no difference in the observed characteristics between those born in the fourth quarter of 1916 and the first quarter

of 1917. If income does have a causal impact on mortality, we should find those born just before 1917 to have lower mortality after retirement.

The Notch is an excellent opportunity to examine the income/mortality link for four reasons. First, the changes in monthly payments generated by the notch were substantial. Analysis from a variety of sources suggests that those born in the fourth quarter of 1916 had about 7–10 percent higher monthly Social Security payments and about 4 percent higher incomes than those born just one quarter later. Second, mortality rates in the impacted groups are relatively high, making it easier to detect an effect of income on mortality if one exists. Third, the manner in which the Notch came to be minimized any pre-retirement reaction among the affected populations. Fourth, and maybe most importantly, the incomes of the elderly are routinely changed by the Federal government through such factors as cost of living adjustments and Medicare premiums, and therefore, this research answers a question of direct policy relevance.⁴

⁴Here are three examples of proposed Federal programs that would have changed the incomes of the elderly in amounts comparable to the income shift produced by the Notch. As we mentioned above, The Boskin Commission's report suggested that the Federal Government might consider indexing Social Security at a rate lower than the CPI. Had the Commission's recommendations been adopted, monthly Social Security payments would have dropped by about 5 percent over a 5-year period. In 1988, Congress passed legislation providing catastrophic health for seniors. This proposal would have provided, among other benefits, unlimited coverage for hospital and nursing home stays. Currently, Medicare only pays for the first 180 days of a hospital stay and does not normally pay for nursing home care. This insurance would have been financed by a premium based upon a share of income up to \$800/year for a single person and \$1,600/year for married couples. Finally, in the early 1980s, Congress instituted the QMB (Qualified Medicare Beneficiary) and SLMB (Specified, Low-income Medicare Beneficiary) programs. QMB paid Medicare Part A (hospitalization) co-payments and deductibles for low income seniors while the SLMB program paid Part B premiums for low-income seniors with incomes too high

We are not the first to use the Notch as an exogenous source of variation in income. Krueger and Pischke (1992) use the large increases in retirement benefits enjoyed by the pre-Notch cohorts to examine the impact of Social Security payments on labor supply among the elderly. Recently, Engelhardt, Gruber and Perry (2002) use the income changes generated by the Notch to examine the role that Social Security benefits play in determining living arrangements. The quasi-experiment we examine is conceptually similar to the one outlined in Case (2001). In that paper, the author uses large unanticipated changes in the South African pension system to examine the impact of income on health. In South Africa, pensions for elderly Blacks and Coloured men and women were increased to be on par with those received by whites. Case found that in households that pooled income, individual health was positively related to the number of pensioners in the household while in households that did not pool resources, health is correlated with only the pension status of the recipient.

Our results are however quite different from the current literature. Examining mortality rates after age 65, we find those born in the last half of 1916 have higher mortality than those born in the first half of 1917, even though the older group receives higher Social Security benefits. Investigation of post-retirement labor supply

to qualify for the QMB program. The income limit for QMB eligibility is \$716 for an individual and \$958 for a couple while the SLMB limits are \$855 for an individual⁴(*continued*) \$1,145 for a couple (<http://www.aarp.org/confacts/money/qmb.html>). The current one-day deductible for a Medicare hospital stay is \$792 or roughly one-twelfth of the income of the highest-earning QMB-eligible person. Likewise, the Part B premium is \$50/month, or about 5.8 percent of the income of the highest-earning eligible SLMB beneficiary. (<http://www.hhs.gov/news/press/2000pres/20001018.html>)

for these two groups suggests that the younger cohort has more part-time work than those born in 1916. These results are consistent with research that suggests social isolation may increase mortality among the elderly. If part-time work keeps the elderly engaged in the community, then there may be some mortality benefits from staying employed past retirement.

This chapter is structured as follows. In the next section, we present an empirical baseline that provides an estimate of the impact of income on mortality for those just entering retirement. The results in this section are in the spirit of previous work in the literature and provide a useful backdrop from which we can compare the reduced-form models presented later on in the paper. In Section 3, we provide a short description of the Social Security notch and some estimates of how much the notch changed Social Security payments and family income. In Section 4, we propose a simple difference estimator to examine this issue and describe how we use data from restricted-use versions of the Mortality Detail data files to implement this model. In Section 5, we present our basic results. Contrary to conventional wisdom, we find that those who received higher Social Security benefits actually had higher mortality than those from younger cohorts. In Section 6 we explore possible explanations for this result and suggest that increased part-time work of the elderly after age 65 is the likely cause. Using data from the March Current Population Survey, we show that the Notch cohorts have substantially higher probabilities of work than older, more highly compensated cohorts. Greater labor force participation can explain the lower mortality among the Notch cohorts if work reduces social isolation among an elderly. A number of researchers have demonstrated a strong positive correlation between

social isolation and mortality, especially among the elderly. We close with some concluding remarks in Section 7.

2. An Empirical Baseline

In later sections, we examine whether reduced Social Security payments generated by the Notch produced higher mortality rates for younger cohorts. This section sets a baseline for discussing the likely magnitude of the effects by estimating a single-equation model for our population of interest. The backdrop for this work is the large social science literature that has examined the income/mortality relationship. The genesis for much of the work in social sciences is the research of Kitagawa and Hauser (1973) who matched survey data from the 1960 Census long form, conducted in April of 1960, to death records from the May–October 1960 period. The stylized facts from their work are that mortality rates decline with income but at a decreasing rate. This relationship is present for all age groups but Kitagawa and Hauser find less variation in mortality across socioeconomic groups for the elderly. The more democratic nature of mortality among the elderly has also been recently documented by Hurd, McFadden and Merrill (1999) and Deaton and Paxson (1998). As we illustrate below, these stylized facts are present in data sets 30 years later than the one analyzed by Kitigawa and Hauser.

The data for the analysis in this section is a sample of individuals from the National Health Interview Survey's (NHIS) Multiple Cause of Death (MCOB) file. The NHIS is an annual survey of 100,000 people from 40,000 households designed to track illness and disability among the non-institutionalized population. Each NHIS

had two components. The first is a household file that contains basic demographic information, self-reported health status, height and weight, lists of chronic and acute conditions, and counts of doctor visits and hospitalizations as well as a measure of family income for all household members. The second component of the NHIS are special-interest modules that survey samples of core respondents about current health topics. Modules vary in size and scope and in many years there are numerous special topics.

In an important extension of the NHIS data, the MCODE data file was constructed by merging individual-level records from the 1986-1994 data files with the National Death Index. The MCODE/NHIS identifies whether individuals in the NHIS have died by the end of 1995, when they died, and the multiple causes of death.

As we explain below, our test for whether the Notch altered mortality is to compare mortality rates over the first five to eight years of retirement for those born just before and after the notch. The relevant population is therefore people roughly 65 years of age. We want our baseline to reflect the long follow-up periods we use later on so we must eliminate the latest years of analysis from NHIS/MCODE data. We also delete the first year of data, 1986, because the NHIS/MCODE was only one-half the size of other years. Because our population of interest is such a small birth cohort, we would like to pool as many NHIS surveys together as possible to enhance the sample sizes. Unfortunately, one shortcoming of the NHIS is that family income

is a categorical variable where categories have not changed for many years.⁵ Inflation makes it impossible to group more than a few years worth of data. To provide as large a data set as possible, we pool data from the first three full-size surveys, 1987-1989.

For each year of the NHIS/MCOD, we have detailed demographic information from the core NHIS data file as well as month, year and cause of death for those who died. NHIS respondents are surveyed throughout the year and the quarter and week within the quarter when the interviews are conducted are coded on the data file. From this information, as well as the data on the month and year of death, we can construct an indicator that measures whether a person died within 5 years to the month of their initial NHIS interview.⁶

To illustrate that this data set can reproduce the stylized facts regarding the correlation between income and mortality, we estimate linear probability equations for three populations: males aged 21–44, 45–59, and 60 and up. The dependent variable is whether a person dies within 5 years of the initial survey. The controls include a complete set of single-year age effects, indicators for white and black respondents (with other race being the reference group), an indicator for Hispanics, seven education dummy variables, plus measures of family income.

⁵For the years of NHIS/MCOD that we consider, the family income variable has 27 groups: 20 categories in \$1000 increments through \$20,000, 6 groups in \$5,000 increments through \$50,000, and those making in excess of \$50,000 per year.

⁶We should note that the NHIS/MCOD is limited in that it only contains data for the non-institutionalized. This is not a problem for those aged 21-44, but for higher age groups, the fraction of those institutionalized increases and deaths are high for these groups. In contrast, our reduced-form results in section V contain data for those institutionalized as well.

In the top portion of Table 1, we report the sample means and sample sizes for each age group. The stark difference in five-year mortality rates across age groups illustrates the need to estimate models for separate age groups. In the next block of results, we report linear probability estimates where the key covariate is income coded as a categorical variable in \$10,000 increments. The reference groups in these models are those with annual family incomes less than \$10,000. The results from these models illustrate that for all age groups, higher income groups have lower mortality but as incomes rise, the coefficients increase (in absolute value) at a decreasing rate. Consider the case of those aged 45–59. Moving from the lowest to the second lowest income group decreases the 5-year mortality probability by 2.5 percentage points. However, moving from \$30,000–\$39,000 to incomes in excess of \$40,000 only changes this probability by .61 percentage points. In the final column of Table 1, we report results for those aged 65 and 66 which is representative of the population we examine below. In this case, we see the protective effects of income but a much more linear relationship between income and five-year mortality. As we move through the income groups, adding \$10,000 in income (1st to 2nd income group, 2nd to 3rd, 3rd to 4th, 4th to 5th) reduces the probability of death by 5.67, 0.48, 2.20 and 4.62 percentage points, respectively. In general, moving from the 1st to 2nd group does produce a larger decline in mortality than movements between other income groups, but the estimated impact is not monotonic.

Table 1
 Linear Probability Estimates, 5-Year Mortality Equations
 1987-1989 NHIS/MCOD Data

Variable	Age Group			
	21-44	45-59	60+	65-66
Observations	53,606	20,748	19,967	2,253
Sample mean of Dep. Variable	0.010	0.042	0.197	0.130
Model 1				
Income \$10,000 - \$19,999	-0.0049 (0.0018)	-0.0250 (0.0065)	-0.0395 (0.0084)	-0.0567 (0.0235)
Income \$20,000 - \$29,999	-0.0073 (0.0018)	-0.0456 (0.0065)	-0.0743 (0.0096)	-0.0615 (0.0259)
Income \$30,000 - \$39,000	-0.0092 (0.0018)	-0.0538 (0.0066)	-0.0842 (0.0114)	-0.0835 (0.0304)
Income \$40,000 +	-0.0116 (0.0018)	-0.0599 (0.0064)	-0.1016 (0.0108)	-0.1297 (0.0290)
Model 2				
In (Income)	-0.0037 (0.0005)	-0.0178 (0.0019)	-0.0402 (0.0039)	-0.0504 (0.0103)
Elasticity at sample mean	-0.370	-0.424	-0.204	-0.388

Other covariates include a complete set of single-year age effects, indicators for white and black respondents, an indicator for Hispanics, and seven education dummy variables.

We can capture the nonlinear relationship between income and mortality using log-income as the single covariate of interest. Unfortunately, income is top-coded at \$50,000 in the NHIS and 19 percent of all men aged 21 and higher report this top-coded value. We use the following procedure to compensate for top-coding. First, we assume income is log normally distributed with a mean of μ and a standard deviation of σ and use the responses to the 26 income categories to estimate an ordered probit model. This model produces maximum likelihood estimates of $\hat{\mu}$ and

☞, and with these values, we calculate the expected value of log income given that it is top coded. Finally, we use this amount for top-coded responses. For all other persons we use the log of their income category's midpoint.

The results from models with log income as the covariate of interest are reported in the bottom of Table 1. Comparing the first three broad age groups, a fixed percentage change in income actually has a larger impact on mortality for older respondents, but the implied income/mortality elasticity is much lower for the oldest respondents. Both of these results are driven by the higher probabilities of death for those aged 60 and up. The marginal impact of a change in income for our population of interest, those 65 and 66, is 20 percent larger than for those aged 60 and up, but the elasticity is roughly similar to the one for those aged 45–50.

Our results, similar to those of other researchers suggest that if single-equation estimates are consistent, for those aged 65, we would expect a 10 percent increase in income to reduce five-year mortality rates by a half a percentage point, from 13.5 percent to 13 percent, a change in the rate of 3.88 percent. This is the standard of comparison for the estimates that follows.

3. The Social Security “Benefits Notch”

The codes establishing Social Security were contained in three sections of Public Law 271, enacted in 1935 by the 74th Congress. Initial Old Age and Survivors Insurance (OASI) payments were a function of the beneficiary's “average nominal wage” and the retiree's age at the time of retirement. These payments remained fixed until Congress passed legislation altering either the method for calculating the

average wage or the schedule of benefits. Although beneficiaries lost ground to inflation until Congress acted, it did so frequently, amending the benefit formula 16 times between 1935 and 1972. The real value of Social Security benefits increased substantially over this time. Part of this increase was due to the higher real wages of younger cohorts, but much was due to Congress' generosity. By the early 1970s, Social Security was the largest and least controversial government social program (Munnell, 1977; Tynes, 1996).

Social Security has, from the start, been a pay-as-you-go system. Taxes on current workers finance current retirees' benefits. Until the 1970s benefits were figured by computing an average wage and reading the appropriate benefit from a table set by statute. During this pre-70s period, nominal wages rose faster than inflation and the pool of workers paying Social Security taxes expanded. This led to a large current surplus in Social Security and a large projected surplus when current benefit levels were compared to inflated future wages. Based on these projected surpluses, between 1972 and 1974 Congress substantially increased benefit levels. At the same time, Congress instituted a system for indexing the benefit table to the Consumer Price Index (CPI). The early 1970s were a period of relatively high inflation; waiting for statutory adjustment was increasingly costly, and consequently unpopular with seniors.

In figuring benefits based upon an unindexed average wage and an indexed benefit table Congress set the stage for Social Security's first financial crisis. Indexation shields current retirees from inflation, but it also leads to higher initial benefits for new retirees with a given (nominal) wage. However, wages do not stay

fixed during a period of inflation. A worker with rising nominal but flat real wages still has a rising “average wage,” thus even an unindexed benefit schedule will lead to higher (nominal) initial Social Security payments. When the benefit schedule is fully indexed to inflation, initial benefits will rise faster than inflation.

Looking back, the double indexation of benefits seems clearly an error. If, however, wages and prices had behaved in the 1970s as they had in the previous decades, the increase in real benefits would have been consistent with long term trends and the wage base would have been sufficient to support such benefits. However, during the 1970s, wages’ growth lagged inflation and projections showed a possibility of insolvency as soon as the mid-1980s (Commission on the Social Security “Notch” Issue, 1996). This led Congress to address the problem with uncharacteristic speed. The result was the first widespread reduction in the generosity of the Social Security system and as a side effect, the creation of the “Benefit Notch.”

Congress chose to correct the system by replacing the nominal wage with an indexed wage. The average wage in the year a claimant turned 60 would be used as a basis, and the claimant’s earnings in year x would be multiplied by the ratio between average earnings in year x and average earnings in the year he or she turned 60. The legislation, enacted in 1977, allowed those who were eligible for retirement before the new amendments became effective to stay in the old system. Those who were not yet eligible would be forced to use the new system. The effective date of the new amendments was January 1, 1979. Therefore, those born on or before January 1, 1917 could stay in the old system. Those born after January 1, 1917 would be in the new system.

To minimize the abruptness of this change, Congress created a special five-year transitional method for people born between 1917 and 1921. Retirees born between 1917 and 1921 are the group commonly known as the “Notch Babies.” The transitional method was identical to the old method except earnings after age 61 could not be used in figuring benefits, and after 1978, no inflation adjustments would be made until age 62. Retirees in the 1917–21 cohorts could take the higher of the two benefits, the new or the transitional. Since earnings after the age of 61 were generally years of high nominal earnings, these rules were of limited assistance. They typically helped only those who retired in or near 1979, but were of little help to the majority who worked into the 1980s. The transitional method was only designed to lessen the impact of the law change. It did not alter the fact that people born after January 1, 1917 would receive, with few exceptions, lower benefits than those born prior to that year. This of course was the intent of the 1977 law. This was the first and, to date, only time the generally rising trend in Social Security benefits was reversed.

The principal novelty of what has become known as the “benefit notch” is that younger cohorts of Social Security beneficiaries generally receive less in old age benefits than older cohorts with similar work histories. The Social Security Act had been amended many times, but before 1977, changes in the act generally allowed a beneficiary to choose whether to claim under the new provisions or under the prior law. This meant that new provisions could only increase a retiree’s benefits. The 1977 amendments offered no such choice; unlike earlier amendments their purpose was to reduce payments.

In the next section, we document the differences in monthly benefits between those born before and after January 1, 1916. The Notch became as large as it did for two reasons. First, those born before 1917 could continue to benefit from the effects of over-indexation no matter when they retired. Second, the years when the pre-1917 cohorts were likely to remain in the labor force were years of high inflation. In the years 1979 through 1982, the annual increases in the CPI were 9.9, 14.3, 11.2 and 7.4 percent, respectively. Had inflation remained at 5 percent between 1979 and 1982, the difference in monthly payments between those born before and after January 1, 1917 would have been much smaller.

It is customary to refer to the cohorts born between 1917 and 1921 as the “Notch babies.” These workers were subject to the transition rules and they received less money than those born slightly earlier. Their benefits however, were in line with long term trends. It is probably more helpful to think of those born just before the Notch cohorts as the “treatment” group. They received more income than the long run trend of OASI payments and more income than Congress intended to give. This is an important distinction in understanding the continuity of retirement behavior across the cohorts. In effect, the Notch was a windfall for the older cohorts, not a calamity for younger cohorts.

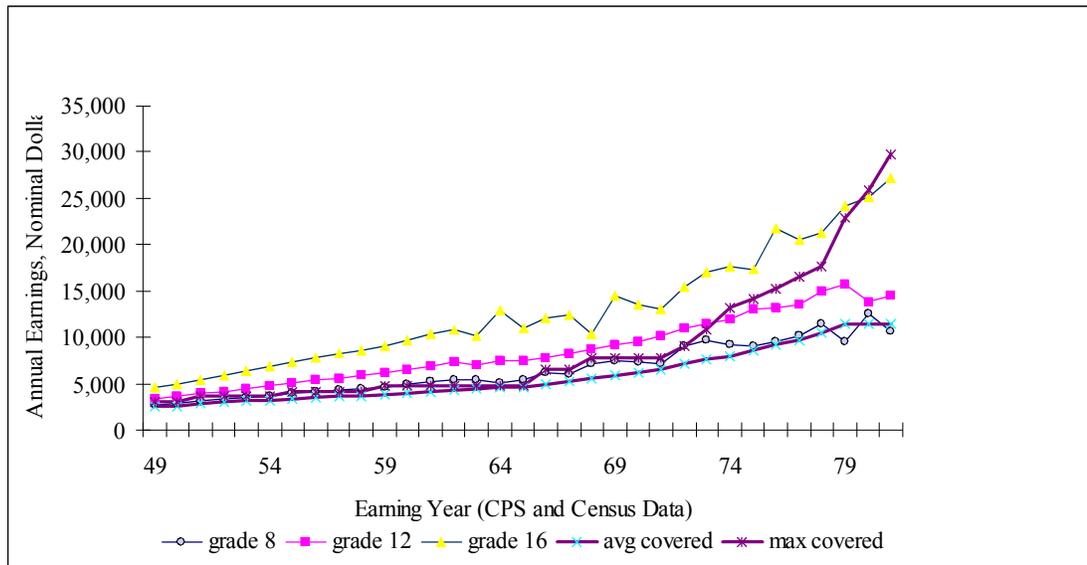
There is scattered evidence about the impact of the Notch on Social Security payments. The bipartisan Commission on the Notch used estimates calculated by the Social Security Administration’s (SSA) Office of the Actuary using the same computer program that Social Security field offices use to calculate actual benefits. A necessary input into the program were estimates of the work history of the Notch

cohorts. Because the impact of the Notch on payments is a function of retirement age, simulations were conducted for two groups: those that retire at 62 and 65. The numbers from these simulations suggest that the Notch generates a loss of \$7 for those born after January 1, 1917 and retiring at 62. The difference however rises to \$110 if a worker retires at age 65. Krueger and Pischke (1992) take a similar approach, but they calculate benefits for each cohort at many different retirement ages. In both cases, the authors use as wage histories the average covered earnings for the years these cohorts were working.

We use a similar methodology as the two previous efforts, but we use a different time series of earnings histories for the Notch cohorts. Since the wage profile varies with age, and since there are significant cohort effects on wages, using average earnings for all workers potentially introduces errors into the calculation of benefits. We use instead cohort-specific earnings profiles constructed from Census data and various March Current Population Surveys (CPS). In particular, we calculate time series for three groups of male workers: those that report 8, 12, or 16 or more years of education. Data for the years 1964 through 1982 are taken from the March CPS. We calculate the earnings for only those who are from the 1916 and 1917 birth cohorts (e.g., approximated by those who report ages of 59 and 60 in the March 1976 CPS). Data from the 1950, 1960, and 1970 Census PUMS generates estimates for 1949, 1959, and 1969 respectively. Data for the period 1950 through 1963 are interpolated using the Census estimates. Revisions to the Social Security law eliminate the need to consider earnings before 1950 in calculating benefits;

benefits are figured only on post-1950 wages for those with income after 1950. The time series of earnings for the three education groups is displayed in Figure 1.

Figure 1
Wage Profiles of Men Born 1916-1917 by Education



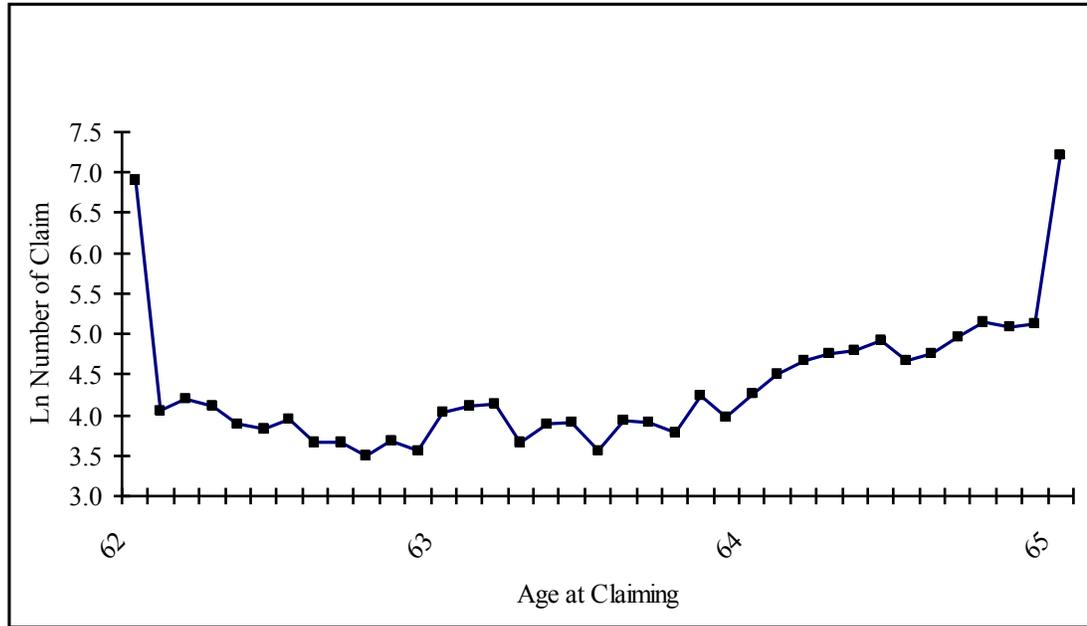
Our calculation of OASI benefits is done by first breaking the Notch cohorts into a series of cells based on age at retirement and years of education. We then calculate a benefit for each side of the Notch in each cell, take the differences, and figure an average difference by weighting each cell according to its share of the cohort. To obtain estimates of the fraction of the population that retires at particular ages, we use data from the Social Security Administration's New Beneficiary Survey (NBS). The NBS is a one-percent sample of all those making an initial claim for benefits between June 1980 and May 1981. We use the entire population of male retirees to estimate the age distribution of new claimants. Since the NBS contains month of birth and month of initial claim we can figure age at claiming to the month. Figure 2 shows that the distribution of claims is extremely lumpy. Virtually half of all

claims by men are filed the month of the pensioner's 62 birthday (22 percent) or 65th birthday (25 percent).⁷ After the first month, claims are steady at one percent per month for ages 62 and 63, rising to 2 percent per month at age 64. Over 95 percent of males filing initial claims were between the ages of 62 and 65. In the early 1980s there was only a very small enhancement to benefits for working beyond age 65. From the data in Figure 1 we create five (year/month) cells, 62.0, 62.6, 63.6, 64.6 and 65.0. We have placed those working past 65 into the 65.0 cell.

The number of sample points in each year's data is quite small, but this makes little difference in the final estimates for two reasons. Social Security is figured from a simple average of the highest N years of covered earnings (N=21 or 22 for these two cohorts), so the averaging across years reduces the impact of any year-to-year estimate. Second, the "covered" earnings are limited to a certain maximum, so variations in wages above the maximum will have no effect on benefits. Figure 1 shows the three profiles and the maximum covered wage for each year. For most of the period under consideration the Social Security system was quite egalitarian, average wages of high school graduates (and certainly college graduates) exceeded the maximum. A large fraction of the working population earned identical Social Security "credits," even when their wages differed substantially.

⁷A change in the law governing minimum age for claiming actually occurred during the year from which the NBS drew its data. We use 62.0, even though the minimum age changed to 62.1 in January 1981 to simplify exposition.)

Figure 2
 Distribution of Age at Initial Social Security Claim
 New Beneficiary Survey Men Age 62–65 - Log Scale



In figuring the weights for each education/retirement age cell, we used the proportions from the 1980 Census data for each of the three levels of educational attainment. We did not make any effort to assign other educational levels to one group or another. Given the mild gradient in the Notch effect across the educational levels, inclusion of other education levels could have only minor effects on the results.

Table 2 presents our findings from these simulations. They are in line with previous estimates, showing that the Notch is very small for those retiring at 62 and much larger for those who work an additional 3 years. The resulting 7 percent difference in benefits is thus an amalgam of a large group with a reduction around 10 percent, and a smaller group with a reduction of 1 or 2 percent. Also, the 10 percent reduction is conservative, since there is a small group working past 65, who will

continue to add additional years of high earnings to their benefits. In the end, we estimate that the 1916 cohort was receiving on average \$41 more per month than those born in 1917. For these groups, average family income was about \$12,000 per year when they reached age 65, so this represents about a 4 percent increase in income.

Table 2
OASI Monthly Payments By Age of Retirement and Education Level

Payment for Men Born 1st Quarter 1917 and
(Difference between 1916:4 and 1917:1)

Age at retirement (year/months)	% that retires at age	Grammar school graduate (28%)	High school graduate (62%)	College graduate (11%)	All
62.0	19%	\$471.60 (\$5.20)	\$525.50 (\$7.10)	\$543.50 (\$5.70)	\$517.64 (\$6.48)
62.6	11%	\$491.30 (\$7.70)	\$546.40 (\$12.60)	\$566.10 (\$14.10)	\$539.22 (\$11.52)
63.6	12%	\$530.60 (\$27.50)	\$591.20 (\$28.60)	\$611.40 (\$44.70)	\$582.37 (\$30.35)
64.6	28%	\$569.90 (\$53.00)	\$635.00 (\$47.20)	\$656.70 (\$79.20)	\$625.51 (\$52.82)
65.0	30%	\$589.50 (\$70.20)	\$656.80 (\$62.30)	\$679.30 (\$102.20)	\$647.00 (\$69.52)
Total					\$596.79 (\$41.49)

We would like to examine the impact of the Notch on Social Security incomes in a regression context with micro data but most data sets do not identify month and year of birth. Had the 1990 census asked for quarter of birth we could have used that data. The 1980 Census PUMS does identify quarter of birth but the earnings data

from the Census related to 1979 when the 1916:4 and 1917:1 cohorts turned 63 and 62 respectively. We can however obtain an estimate of the Notch effect on Social Security payments using data from the Annual Demographic File of the March CPS. The March CPS contains information about income received in the previous year and one category is Social Security payments. The main drawback to the CPS is the poor identification of birth year. The questionnaire asks for age at the time of the survey (mid-March) and for earnings and employment in the previous calendar year. Year of birth can therefore only be defined as (survey year) - age - 1. This will place about 80 percent of respondents with the appropriate calendar year of birth, but the other 20 percent (those born January through mid March) will be grouped with those born in the previous year. Since it is calendar year that changes the applicable benefit formula, this will bias any estimate of a Notch effect on Social Security payments downward.

To conduct the test, we first extract a sample of males who report their age as 68 in 1985, 69 in 1986, or 70 in 1987. These males are roughly from the 1916 birth cohort and in these surveys, this cohort reports Social Security payments received when they were 67–69 years of age. We then draw a companion sample of those aged 67 in 1985 through 69 in 1987. Most of these men were born in 1917 and these respondents report Social Security earnings at ages 66–68.⁸ Next, we regress real annual Social Security earnings in 1987 dollars on a complete set of fixed effects for

⁸It is important to only include earnings starting in 1984 for the 1917 cohort because including data for 1983 would add Social Security payment data for this group in a year when these men turn 65 and as a result, many will not have a full year's worth of earnings data.

race, education, marital status, and reporting year, plus a dummy variable representing the older (1916) birth cohort. The results from this exercise are reported in Table 3. This sample has a large number of observations (3059) and real mean annual Social Security earnings over these four years is slightly under \$5,900. During this period, the 1916 birth cohort received \$496 more per year than their nearest younger cohort, about 8.4 percent of than the sample mean. Since only 80 percent of the people we put into the 1917 birth cohort are actually from that group, with 20 percent coming from the 1916 group, we would expect this number is low by about 20 percent. Inflating the parameter estimate by 20 percent and dividing by 12, we find that the Notch increased payments to the 1916 birth cohort by about \$50 a month, similar to the estimate we produced through our simulation exercise in Table 2.⁹

⁹Respondents in the CPS are in the sample for the same four months over a two year period (for example, January through April in both 1985 and 1986). As a result, our sample will sometimes include two observations from the same household but in different years. Restricting our attention to people who were in the sample for the first year, we cut the sample in half, but the coefficient on the “Notch” dummy variable is essentially unchanged dropping to \$487.51 with a standard error of \$155.76.

Table 3
Impact of Notch on Annual OASI Payments, in Real 1987 Dollars,
March CPS

	1916 cohort: earnings during ages 67-69		1915 cohort: earnings during ages 67-69
	1917 cohort: earnings during ages 66-68		1916 cohort: earnings during ages 66-68
	Males	Females	Males
Mean of dependent variable	\$5,879	\$4,171	\$5,950
“Notch” effect—dummy variable that equals 1 for the oldest cohort (standard error)	495.74 (109.74)	8.47 (69.54)	8.00 (109.78)
Number of Obs.	3,059	3,786	3,189

All models include fixed effects for education, marital status, race and year of the survey. The Notch effect is for the “oldest” cohort.

To establish that this difference is not found in the other populations we will compare to the “Notch” cohort, we report comparable results for two other groups, women born in 1916/1917 and men born 1915/1916. During this time period, most women who receive OASI payments are qualifying for payments based on their husband’s earnings (Reno and Ycas, 1982; Iams and Ycas, 1988). Consequently, for women there should be no Notch effect when we compare the 1916 and 1917 cohorts. In the next column of the table, we redo the same exercise with women from the same years and age ranges, we see that women from the 1916 birth cohort earn only \$9 more per year than their nearest younger cohort. These results are presented in column (2) of Table 3. If we take men who were 67 in 1984, 68 in 1985, and 69 in 1986, we generate a sample of Social Security earnings from the 1916 cohort when these men were 67 through 69. If we match this group to men who were 68 in 1984, 69 in 1985, and 70 in 1986, we produce a sample of earnings for men aged 67–69 from the 1915 cohort. Defining the “Notch” dummy variable for the “older” cohort

(those born in 1915), we see in the final column of the table that men from the 1915 cohort only earn \$8 more per year from Social Security than their next younger cohort. These last two results are important because later on, we will use mortality differences for women from the 1917 and 1916 cohorts and men from the 1916 and 1915 cohorts as comparison groups for our primary comparison – the mortality difference between the 1917 and 1916 cohorts. Therefore, after retirement, there are large differences in Social Security earnings in our primary groups of interest (1916 versus 1917) but no such differences in our comparison groups.

Although the benefits Notch was a windfall for some cohorts, there is little evidence that the differences in OASI payments for cohorts born in the 1911–1921 time period altered retirement behavior. For those born prior to 1917, we might expect the increase in retirement income to induce an earlier exit from the labor force. However, because Congress continued to allow over-indexation for older cohorts and these workers reached age 62 at a time when inflation was high, the longer an individual stayed in the labor force the greater the impact of over-indexation. In effect, Congress created a substitution effect to offset any wealth effect. The cost of retiring young was high, which would tend to delay retirement. Krueger and Pischke (1992) find no difference in the retirement profiles for those born before and after January 1, 1917.

Another explanation for the apparent non-impact of the 1977 amendments was that the changes in the benefits generated by the new law were not widely understood. The true impact of the benefit changes was produced through the interaction of legislation and the inflation experienced after the legislation passed. The perceived

unfairness of the Notch only became a political issue after the 1917–1921 cohorts began to retire. The Commission’s account of public awareness of the Notch is instructive:

“After comparing their benefit checks against the larger checks of their pre-“Notch” colleagues, neighbors, and friends with similar employment records, they began expressing their dissatisfaction to public officials—and the “Notch” issue was born.” (Commission on the Social Security “Notch” Issue 1996) (Our italics)

Some may find it puzzling that there was not more foresight among the general public regarding the change in benefits. In 2001, new retirees are accustomed to thinking of a given real benefit level as an entitlement. Until the late 1970s, however, Social Security benefit levels had always been subject to arbitrary adjustments. No one could predict their benefit, even if they could predict their wages. Survey data suggests that today still, workers have poor understanding of their future OASI benefits (Gustman and Steinmeier, 2001). The Reader’s Guide to Periodical Literature, an index of popular magazines, contains fewer than 20 references to the over-indexation of Social Security benefits in the crucial 1974-1976 time period.

4. Econometric Model

As mentioned above, the 1977 amendments to Social Security changed the way initial OASI benefits are calculated, which in turn produced sharply lower payments for recipients born after January 1, 1917. We examine the income/mortality hypothesis in an elderly population by examining whether the higher payments received by older cohorts lead to lower mortality rates. The

econometric model we propose is a “difference-in-difference” estimator where the structure of the test is driven by the available data and the need for large samples.

Within a group of pregnant women, it is essentially a random process that generates who gives birth today and who gives birth tomorrow. We would therefore expect to find very little difference in the social, economic and behavioral characteristics of people born just before and after January 1, 1917. However, these two groups face different streams of OASI payments once they retire. Therefore, any difference in post-retirement mortality between these groups can reasonably be attributed to the difference in OASI payments.

This simple analysis is complicated by two facts. First, the difference in income generated by the Notch is not huge and as we demonstrate below, we will need a large data set to have any hope of detecting a meaningful difference in mortality across the two groups. This makes it impossible to compare cohorts born in the days right before and after January 1, 1917. Expanding the size of the cohorts does come at a price. There are a number of secular trends in the economic, health and social characteristics of cohorts. On average, younger cohorts have more education and live longer than older groups. If we compare outcomes for full year cohorts born in 1916 and 1917, these groups contain people that vary in age anywhere from 1 day to almost 2 years. If mortality itself or the determinants of mortality (such as education or income) are changing rapidly, comparing two groups that span so many months may introduce a difference in mortality that is produced by secular trends rather than OASI payments. Consequently we cannot rely on a simple difference estimator as the basis of our analysis.

The difference-in-difference estimator is generated by finding a control group that can accurately measure the difference in mortality after age 65 between the 1917:1 and 1916:4 groups that would have existed in the absence of the treatment (the Notch). In this case, we have two potential control groups. Each helps control for a different type of secular variation in mortality. The first includes men born one year earlier. The second control group is women born during the same months as our male cohort. Each provides a control for a different alternative cause of differences in mortality.

There can also be variation across cohorts in mortality rates based on a number of factors that may confound a simple difference analysis. For example, we know that age-adjusted mortality rates are lower for younger cohorts. Although the 1917:1 cohort is only born one quarter later than the 1916:4 cohort, one might be suspicious that a higher mortality rate after age 65 for the 1916:4 group is a secular difference rather than a shock produced by the impact of the Notch. Therefore, we will use mortality differences for males between the 1916:1 and 1915:4 cohorts as one control group.

There is growing evidence that conditions present before and right after birth have a lasting impact on health. Barker (1998) for example argues that when the fetus is faced with a poor environment, changes in the supply of nutrients received by vital organs can hard wire these organs for later susceptibility to disease. Using a variety of models, Almond (2003) finds that the 1918 birth cohort, which was born during a major influenza epidemic, has substantially worse labor market outcomes than adjacent cohorts. We discuss this subject in detail in the next chapter. If there is

something that adversely impacted either the 1917:1 or 1916:4 birth cohorts, we might find differences in mortality across these groups that is unrelated to the Notch. To control for this possibility, we use women born in the 1917:1 and 1916:4 periods as a control. Women born in 1916/1917 face the same perinatal infant environment as males. As we note above, most women born in this generation receive Social Security benefits based on their husband's earnings, and, since most women marry men older than themselves, there should be little impact of the Notch on Social Security income across these narrow birth cohorts. This is in fact verified in Table 3. Women at age 65 do however have substantially lower mortality rates than men the same age, so in this model we use difference in log mortality rates as the key outcome.

4.1 A Note on Sample Sizes

We showed using two different procedures that the Social Security Notch generated about 4 percent higher incomes among those born in the 1916 compared to those born in the next year. In Table 1, we showed that for people aged 65 and 66, the coefficient on log income is about -0.05 in a linear probability model where the outcome is 5-year mortality. Therefore, if this relationship represents the "true" impact of income on total mortality, we would expect those born before 1917 to have two-tenths of one percentage point lower five-year mortality rate than those born in 1917 ($0.04 * (-0.05) = -0.002$). In this section, we calculate an estimate for the sample sizes necessary to produce a statistically significant estimate of -0.002. This calculation is necessary because we have to expand our samples around January 1,

1917 and we want to expand them enough to have a fair test of detecting a statistically precise relationship.

Suppose we have two equally size cohorts—one born after January 1, 1917 and one born just before. The reduced-form equation of interest can be written as a simple bivariate regression:

$$(1) \quad y_i = \alpha + \beta_{RF} z_i + \varepsilon_i.$$

Where y_i is an indicator that equals 1 if the respondent died within 5 years of their 65th birthday and z_i is an indicator for whether the respondent is a pre-Notch baby, i.e., someone born in 1916. Let $\hat{p}_1 = (\bar{y}|z_i=1)$ and $\hat{p}_0 = (\bar{y}|z_i=0)$. Because both z and y are discrete, the estimate for β_{RF} in equation (1) can be shown to equal:

$$(2) \quad \hat{\beta}_{RF} = (\bar{y}|z_i=1) - (\bar{y}|z_i=0) = \hat{p}_1 - \hat{p}_0$$

This reduced-form estimate will only be statistically significant if:

$$(3) \quad \hat{\beta}_{RF} / \hat{\sigma}_{RF} \geq 1.96$$

where $\hat{\sigma}_{RF}$ is the standard error of $\hat{\beta}_{RF}$. Under the assumptions we have made, we can solve this expression for the minimum number of observations necessary to generate a statistically significant coefficient of $\hat{\beta}_{RF} = -0.002$. Because y is discrete and both samples are assumed to have the same number of observations, σ_{RF}^2 approximately equals $[\tilde{p}_1(1 - \tilde{p}_1) + \tilde{p}_0(1 - \tilde{p}_0)]/n$. For equation (3) to be true, it must be the case that $n > [1.96 / \hat{\beta}_{RF}]^2 [\hat{p}_1(1 - \hat{p}_1) + \hat{p}_0(1 - \hat{p}_0)]$. Using the means from Table 1, we can set $\hat{p}_0 = 0.13$ and notice that $\hat{p}_1 = \hat{p}_0 + \hat{\beta}_{RF}$. It is then easy to show that n , the size of the treatment and control groups, must be approximately 214,000 observations. This is

roughly the size of the number of people born in the 1st quarter of 1917 who are alive at the time of the 1980 Census. At the start, we then want to compare the post-retirement mortality for people born in the 4th quarter of 1916 and the 1st quarter of 1917.

4.2 Pre-retirement Characteristics of the 1916:4 and 1917:1 Male Cohorts

Going into retirement, there is little difference along any demographic characteristic between these two cohorts. This is easily demonstrated with data using the 1970 Census Public Use Micro Samples (PUMS). The PUMS samples are drawn from the one-sixth of the households who received the Census long form. In 1970, Public use data is available from six different samples. The samples differ in geographic information available and in the questionnaires they received, but the core demographic information we need is common to all samples. By aggregating data from all of these samples, we obtain a 6-percent nationally representative sample. For our purposes, one key piece of information in the PUMS is the respondents' quarter of birth. Using the fact that the Census day is April 1, 1980, the first day of the second quarter, we can then back out the quarter and year of birth. Respondents from the 1916:4 and 1917:1 cohorts would report 53 years of age in the 1970 Census. In Table 4, we compare the means across the 1916:4 and 1917:1 cohorts along a number of different demographic characteristics. In the table, we report the sample average for each variable when these two cohorts are pooled together, the difference in means across these cohorts, and the t-statistic on this difference. We see there is no statistically significant difference in earnings, years of education, the fraction with a

high school degree, weeks worked, hours worked per week, the fraction that worked full time, the fraction self employed, marital status, or the fraction disabled. The small differences which exist in observed characteristics between the two cohorts are not unique. We report the same type of results for different 4th quarter/1st quarter contrasts and the only persistent difference is that those born in the 1st quarter have lower education levels. In this case, the result is statistically significant in three of five cases. This result has been documented, notably by Angrist and Krueger (1991), who use data from the 1970 and 1980 Census PUMS data sets to show that in cohorts born between 1920 and 1949, men born in the 1st quarter tend to have about one tenth of a year fewer years of education than do those born at other times of the year. Angrist and Krueger interpret this as being generated by interactions between laws governing minimum school start age and compulsory education laws—those born in the 1st quarter start school at an older age and they are more likely to age out of compulsory education laws, making them more likely to drop out. The differences in education in our data are very close to those reported in Angrist and Krueger.

Table 4
Comparison of Means, 1st and 4th Quarter Births
1970 PUMS 6% Sample

Mean for First Quarter [Difference in Mean for Fourth Quarter] (t Value of Difference)

Outcome of Interest	1914:4 vs 1915:1	1915:4 vs. 1916:1	1916:4 vs 1917:1	1917:4 vs. 1918:1	1918:4 vs 1919:1
Annual Labor Earnings	7386 [-21] (-.25)	7640 [-15] (-.18)	7700 [46] (.56)	7923 [185] (2.23)	7986 [57] (.69)
Years of Education	10.41 [.037] (.86)	10.55 [.088] (2.09)	10.65 [.068] (1.63)	10.76 [.115] (2.81)	10.77 [.147] (3.54)
< High school diploma	53.7% [-.15%] (-.25)	51.8% [-.91%] (-1.49)	50.8% [-.43%] (-.71)	49.2% [-1.69%] (-2.85)	48.9% [-1.11%] (-1.88)
Weeks worked, 1969	45.4 [.11] (.58)	45.8 [.04] (.21)	46.2 [-.11] (-.63)	46.1 [.53] (3.21)	46.3 [.41] (2.59)
Hours worked/week, 1969	35.23 [.04] (.16)	35.61 [.36] (1.68)	36.22 [-.03] (-.13)	36.27 [.40] (1.94)	36.28 [.428] (2.09)
Worked full time, 1969	73.3% [-.0012] (-.21)	74.6% [.0046] (.87)	75.5% [-.0022] (-.42)	75.7% [.0023] (.44)	75.6% [.0084] (1.65)
Self-employed, 1969	16.4% [.4%] (.83)	16.0% [.5%] (1.16)	15.6% [.4%] (.89)	15.4% [-.2%] (-.38)	15.3% [.1%] (.33)
Disabled (1=yes)	9.7% [.08%] (.21)	9.6% [-.16%] (-.44)	8.7% [.49%] (1.42)	8.3% [-.21%] (-.65)	8.5% [-.20%] (-.62)
Married (1=yes)	86.0% [.69%] (1.65)	86.8% [-.24] (-.58)	86.8% [.13%] (-.33)	87.0% [.04%] (.09)	86.6% [-.08%] (-.20)
Number of observations, both quarters	27,024	27,215	27,737	28,618	28,306

4.3 Restricted-Use Mortality Detail Data

Although there are some retrospective data sets that identify mortality for a cross-section of people (such as the National Longitudinal Mortality Survey and the NHIS/MCOD file introduced above), the sample size calculations show these data sets are inappropriate for the task at hand. The only data sets of the appropriate size are the Mortality Detail data sets. Detailed micro data on births and deaths are available starting in 1968 from the NCHS Natality and Mortality Detail data files. Mortality Detail files contain a census of births in all years except in a few when a 50 percent sample is available. The Mortality Detail data provides information on age, sex, race, ethnicity, plus detailed geographic data about the place of residence and death. Some states also provide education and the industry and occupation of the deceased. The mortality data also identifies the month and cause of death. Public use tapes do not provide year or month of birth. This data is, however, available on research files available to NCHS staff. We reached a special agreement with the NCHS and they provided us with restricted-use Mortality Detail files that contain all public-use information plus the month and year of birth. We received data for deaths over the 1979–1990 period.

With this data, we can count the total deaths from a birth cohort after a particular date. We must, however, control for the possible differences in cohort size that exist. For example, the cohort born in the 1st quarter of 1917 is slightly larger than the cohort born in the previous quarter, so we would expect to find some difference in the number deaths across these two groups. We therefore need to construct death rates that condition on the number of people alive in a cohort at the

start of the period. To calculate the denominator in the death rate, we use population counts of cohort size from the 1980 Census Public Use Micro Samples (PUMS). The 1980 PUMS data is available from three samples: the 5-Percent, a 1-Percent Urban/Rural sample and a 1-Percent Metro Sample. All three are nationally representative samples of the population and merged together represent a 7-percent random sample of the population. Dividing the number of people in a particular birth quarter by .07 in these combined samples, we then have an estimate of the number of people from the cohort who were alive on April 1, 1980. Counting deaths in previous or subsequent quarters for cohorts and subtracting or adding these numbers from population total from the Census, we have an estimate of the number of people alive at the start of any particular quarter. For example, those born in 1916:4 turn 65 in the 4th quarter of 1981. Subtracting deaths for this cohort between 1980:2 and 1981:3 from the population numbers from the Census gives us the number of people alive as of the start of the quarter this group turns 65.

For each cohort, we want to construct death rates over a fixed period of time. The beginning periods for the death rates will be defined by birth dates rather than calendar time. Although cohorts born before and after January 1, 1917 are roughly the same age, calculating death rates for say the January 1, 1982–December 31, 1986 period will, by construction, produce differences in death rates simply because the 1917 cohort is younger than the 1916 cohort. We therefore must define death rates over the same age range. Because the vast majority of people on Social Security have retired by their 65th birthday and because there is little Notch effect for those who

retire, we construct five-year mortality rates for people who died within 5 years of the quarter in which their 65th birthday occurred.

Death rates are constructed as:

$$(1) \quad DR(J)_i = D(J)_i/n_i$$

where $D(J)$ is the number of deaths over the next J years after the quarter cohort i turns 65, and n_i is the number of people from a cohort who lived until the quarter they turned 65. Standard errors on rates such as $DR(J)_i$ are typically defined by sampling variance. In this case, there is no sampling variance in the numerator—we have a complete census of deaths for the cohort for the J years after their 65th birthday.

Variance is introduced into the variable from two sources. First, the numerator is the outcome from a random process. Of n_i people from the cohort, only a fraction will die. Second, the denominator is based on a 7-percent sample. If $d(j)_i$ is the actual true j -year death rate for cohort i after age 65, the variance of $D(j)_i$ is $n_i d(j)_i (1-d(j)_i)$. Similarly, if there are N people in the U.S., the chance a person from cohort i is sampled in the Census is given by p_i , and because the PUMS is a 7-percent random sample of the population, the number of people we estimate in the cohort is therefore $n_i = N_i * 0.07$. Since whether a sampled person is from cohort n_i or not is the result of a Bernoulli process, $\text{Var}[n_i] = N p_i (1-p_i) / (0.07)^2$. Using a 1st-order Taylor's Series expansion, the variance on $DR(J)_i$ is then defined as:

$$(1) \quad \text{Var}[DR(J)_i] = d(j)_i (1-d(j)_i) / n_i + d(j)_i^2 N p_i (1-p_i) / (0.07)^2 / n_i^2$$

A consistent estimate of this variance is obtained by using estimates of p_i , n_i and $d(j)_i$.

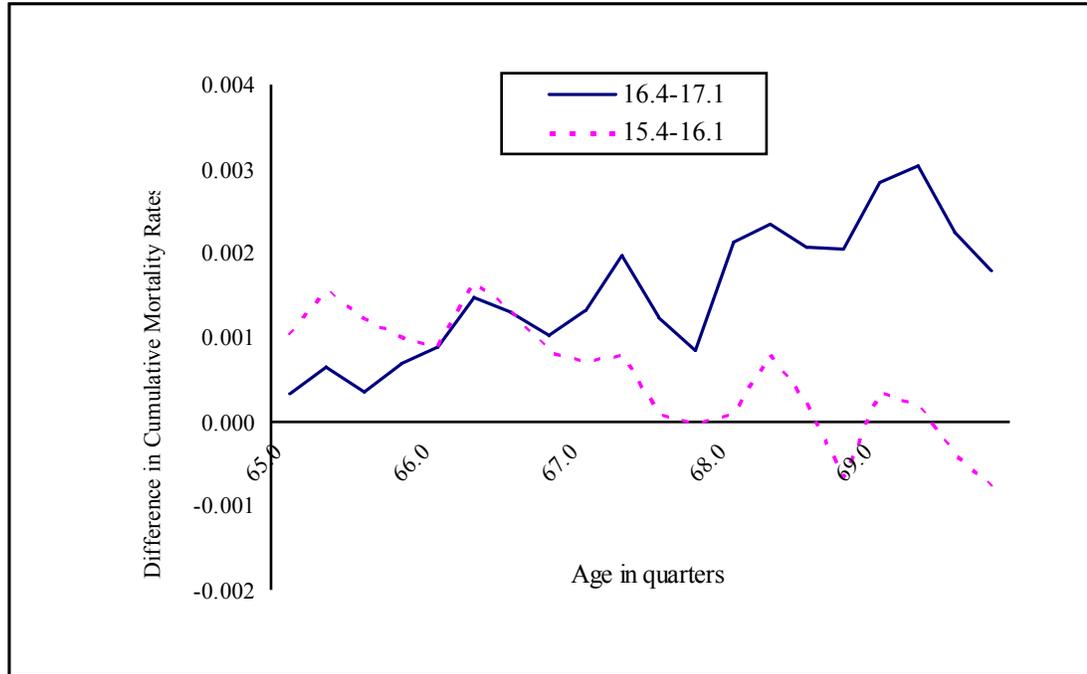
5. Results

In this section, we present basic difference-in-difference estimates, comparing the five-year mortality rates after age 65 for those born in the 1916:4 and 1917:1 quarters. To control for any difference in mortality for men generated by a quarter of birth effect, we use our first control group (men born one year preceding the year of the Notch) as comparison groups. In the first test, we examine the difference in cumulative mortality rates after age 65 for males from the 1916:4 and 1917:1 cohorts. There may be persistent differences in mortality between these two cohorts generated by their quarter of birth. For example, we know that among the elderly, there are more deaths in the fourth quarter than in 1st quarter. We also know that death rates increase with age. Among the 1916:4 and 1917:1 cohorts, using mortality from age 65:0, the older group is turning older at a worse time of year. As the cohort ages, we may see above-average mortality for this group, solely due to an interaction between the quarter of birth and the age profile of mortality. To eliminate this type of bias, we can use the contrast in mortality between younger cohorts with 4th and 1st quarter births as a difference control group for this analysis. Specifically, we start by using the difference in the five-year mortality rate between the 1915:4 and 1916:1 cohorts as a measure of the expected difference in mortality due to these seasonal factors. Because the five-year mortality rate is on roughly the same scale for the 1915:4/1916:1 and 1916:4/1917:1 contrasts, we focus on differences in mortality.

A graphical presentation of this estimate is presented in Figure 3. In this figure, we graph the difference in cumulative mortality for the 1916:4 and 1917:1 cohorts as well as the 1915:4 and 1916:1 groups. Notice that this latter difference

tends to hover around zero for the 20 quarters after age 65. In contrast, the difference in cumulative mortality for the Notch cohorts (1916:4–1917:1) grows steadily over time.

Figure 3
Difference-in-Difference Estimates—Males



The numeric results that correspond to the graphical presentation are reported in the top half of Table 5. There is almost no difference in five-year mortality rates for the two pre-Notch cohorts and the difference-in-difference estimate again suggests that if anything, the 1916:4 cohort has higher mortality than the 1917:1 group. The results in Table 5, although statistically imprecise, indicate that the older cohorts who received *higher* Social Security payments actually have a 0.0028 percentage point higher mortality rate than their younger, less wealthy cohorts. If the results in Table 1 represent a causal relationship, the expected change in mortality should be -0.002 percentage points – a 4 percent increase in income should produce about a -0.002

percentage point drop in mortality. To increase the precision of the estimates in Table 5 we expand the study population so that we compare 1916:3 and 4 to 1917:1 and 2 using the difference in mortality between 1915:3 and 4 and 1916:1 and 2 as the control group. We present these results in Table 6, but the difference-in-difference estimates in Table 6 are still not statistically significant. If we take the results in Table 1 as our null hypothesis, we would expect the higher incomes generated by the Social Security notch to reduce mortality by .002. Although they have opposite signs, a 95 percent confidence interval on the Table 1 estimate and the difference in difference estimate in Table 6 overlap.

Table 5
Difference in Difference Estimates,
Impact of Notch on Five-Year Mortality Rates for Males

A: Males			
	4 th Quarter (1)	1 st Quarter (2)	Difference (1) - (2)
(a) Notch (1916:4 and 1917:1)	0.1519 (0.0021)	0.1494 (0.0021)	0.0025 (0.0029)
(b) Pre-Notch (1915:4 and 1916:1)	0.1524 (0.0021)	0.1527 (0.0021)	-0.0003 (0.0029)
		Difference (a) - (b)	0.0028 (0.0042)
B: Females			
	4 th Quarter (1)	1 st Quarter (2)	Difference (1) - (2)
(c) Notch (1916:4 and 1917:1)	.0820 (.0012)	.0823 (.0012)	-.0003 (.0017)
(d) Pre-Notch (1915:4 and 1916:1)	.0827 (.0012)	.0839 (.0012)	-0.0012 (.0017)
		Difference (c) - (d)	-0.0015 (0.0024)

Standard errors in parenthesis.

As a check on our estimates, we can repeat the analysis from Figure 3 and the top halves of Tables 5 and 6 using female rather than male mortality rates. As we noted previously, most women from these cohorts qualify for Social Security based on their husbands' earnings. Consequently, there should be little Notch-effect on Social Security income for women in the 1916:4 and 1917:1 cohorts—a result we verified above in Table 3. If our difference in difference model is controlling for the secular differences in mortality that should be impacting the Notch cohorts, then we should see a small notch effect on mortality when we re-run the basic models using data for women.

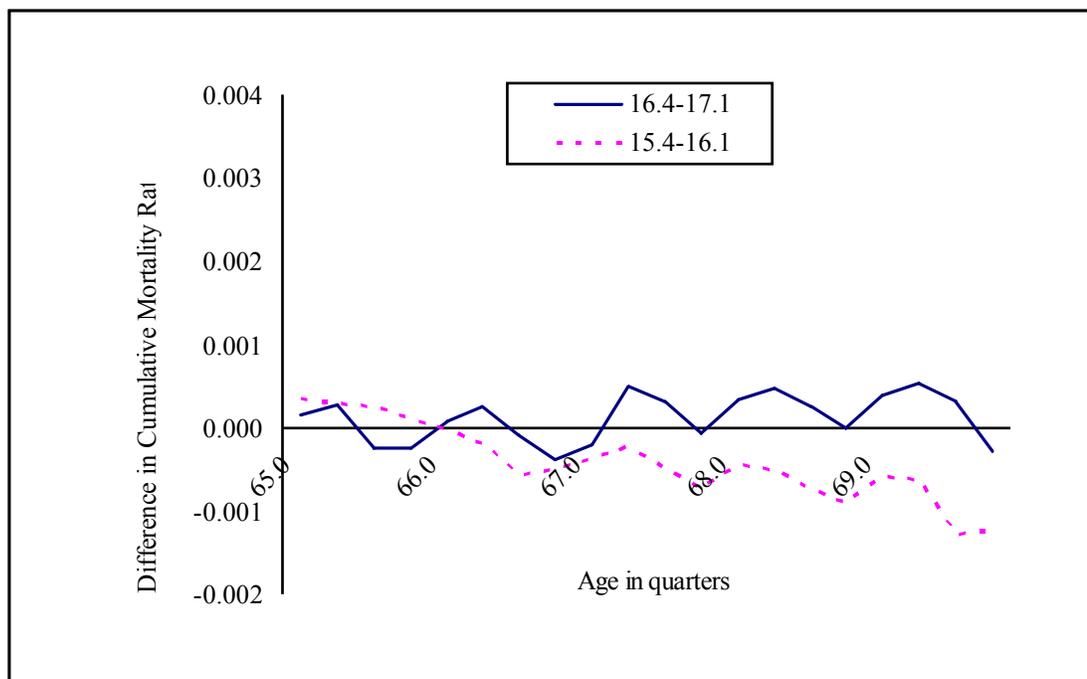
Table 6
Difference in Difference Estimates,
Impact of Notch on Five-Year Mortality Rates for Males

A: Males			
	3 rd and 4 th Quarter (1)	1 st and 2 nd Quarter (2)	Difference (1) - (2)
(a) Notch (1916:3,4 and 1917:1,2)	0.1517 (0.0015)	0.1482 (0.0015)	0.0036 (0.0021)
(b) Pre-Notch (1915:3,4 and 1916:1,2)	0.1520 (0.0015)	0.1521 (0.0015)	-0.0001 (0.0021)
		Difference (a) - (b)	0.0037 (0.0029)
B: Females			
	3 rd and 4 th Quarter (1)	1 st and 2 nd Quarter (2)	Difference (1) - (2)
(c) Notch (1916:3,4 and 1917:1,2)	0.0820 (0.0009)	0.0819 (0.0009)	0.0001 (0.0012)
(d) Pre-Notch (1915:3,4 and 1916:1,2)	0.0820 (0.0009)	0.0835 (0.0009)	-0.0014 (0.0012)
		Difference (c) - (d)	0.0015 (0.0017)

Standard errors in parenthesis.

In Figure 4, we present a graphical presentation of the basic difference in difference results using women from the 1917:1, 1916:4, 1916:1 and 1915:4 cohorts. In this graph, although women have lower mortality rates than men, we have kept the scale of the graph the same to allow a comparison with the results from Figure 3. The solid line in the figure is the difference in quarterly cumulative mortality rates between the 1916:4 and 1917:1 cohorts. In contrast to the results for males, for these cohorts, there is no systematic difference in mortality rates for these Notch cohorts – the difference in mortality rates hovers around zero for the 20 quarter period.

Figure 4
Difference-in-Difference Estimates—Females



In the bottom half of Table 5, we report the basic difference in difference estimates for 20-quarter mortality for females that corresponds to the graph in Figure 4. Notice that the difference in the 20-quarter mortality rate between the 1916:4 and

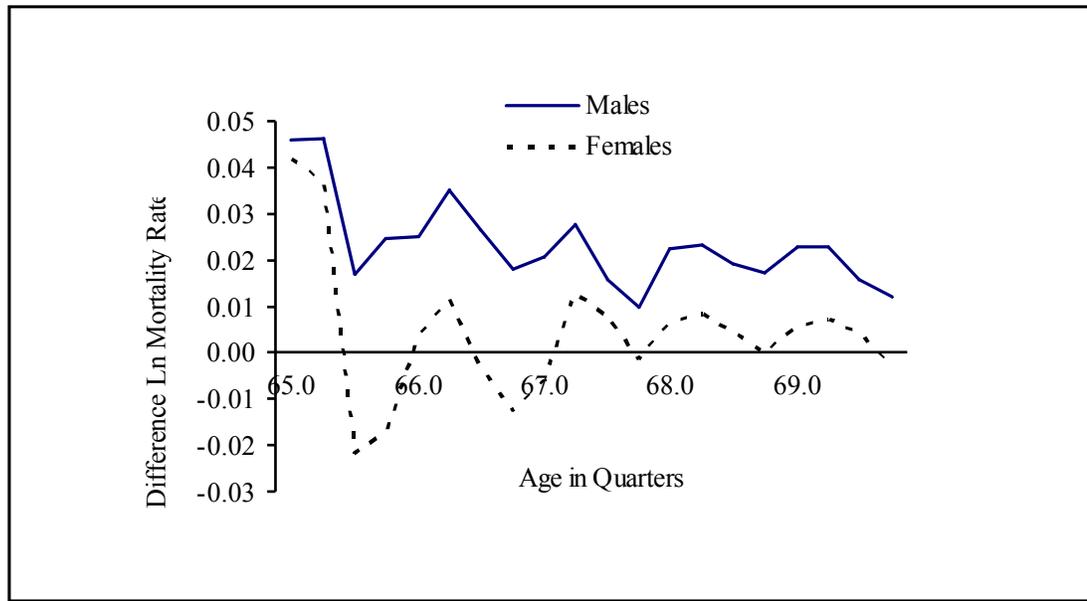
1917:1 cohorts is only four-tenths of a percent of the mortality rate for the 1916:4 group. The difference in difference estimate generated by using the pre-Notch difference of 1915:4 minus 1916:1 produces a small, negative and statistically insignificant Notch effect. In Table 6, when we expand the cohorts to include half-year samples, we find a statistically insignificant difference in difference estimate for women only 40 percent of the size of the result for males.

As we mentioned above in Section 4, comparing the difference in mortality in the 1916:4 and 1917:1 cohorts with the difference in the 1915:4 and 1916:1 groups controls differences in mortality that are associated with quarter of birth but are persistent across birth cohorts. There could however be differences in mortality between the 1916:4 and 1917:1 birth cohorts that are cohort specific. To control for permanent differences in mortality associated with these quarters, we can use women from the 1916:4 and 1917:1 as a second comparison group. In this case, because the mortality rates are larger for males than females, we compare differences in log mortality rates.

In Figure 5, we graphically illustrate this contrast. The dashed line is the difference in cumulative log mortality rates, 1916:4 minus 1917:4, for females. After the first quarter, there is a very small difference in cumulative log mortality rates for these women, with the difference bouncing around zero for the first 20 quarters past age 65. The solid line represents the difference in cumulative log mortality for males over the same period and for the same cohorts. We see a pronounced positive difference in log mortality with the 1916:4 cohort having higher mortality throughout

the entire period, suggesting once again that in contrast to the conventional wisdom, the higher income 1916:4 group, has higher mortality rates.

Figure 5
Difference in Ln of Cumulative Mortality Rates, 1916:4 minus 1917:1



A numeric version of this analysis is contained in Table 7. The top row of the table reports the 20 month (five-year) mortality rates starting from the quarter when the cohort turns 65. If the higher incomes received by the 1916:4 cohorts lead to lower mortality, we should see a lower mortality rate for this group. This is not the case. The difference in log mortality rates is actually a positive 0.0164. The second row of the table reports results for women over the same time period. If our results were driven by some time-specific variation in mortality rates we would expect the data for women to show a similar pattern. It does not. There is a slightly lower mortality rate in the fourth quarter and a difference-in-difference estimate suggests that the 1916:4 male cohort has a 2-percent *higher* mortality rate after age 65 than the 1917:1 cohort. However, recall that the elasticity of five-year with respect to income

in a single-equation model is about -0.388 and we estimate that the Notch elevated incomes of the 1916:1 cohort by about 4 percent over the 1917:1 group, so if the single-equation estimates are true, we would expect a coefficient of about -0.01552 and the 95-percent confidence interval on this estimate would be (-0.0093,-0.0217). Given the standard error of 0.0096 on the difference-in-difference estimate of 0.0200, we can reject the hypothesis that the results in Tables 1 and 7 are statistically identical.

Table 7
Difference in Difference Estimates,
Impact of Notch on Mortality Rates
5 Year Mortality from Age 65, Women as Control Group

	Before Notch Born in 4 th Q. (1)	After Notch Born in 1 st Q. (2)	Log Difference log(1) - log(2)
(a) Men born 1916:4 or 1917:1	0.1519 (0.0021)	0.1494 (0.0021)	0.0164 (0.0076)
(b) Women born 1916:4 or 1917:1	0.0820 (0.0021)	0.0823 (0.0012)	-0.0036 (0.0060)
		Difference (a) - (b)	0.0200 (0.0096)

Standard errors in parenthesis.

Mortality Rates are computed from the quarter each quarter-of-birth cohort turns 65 and proceeding forward for 20 quarters. The cohort born in 1916:4 turns 65 in 1981:4. Their mortality rate is based upon the population as of January 1, 1982 and the count of deaths between January 1, 1982 and December 31, 1986. The 1917:1 cohort's mortality rate is based upon the population on April 1, 1982 and mortality between April 1, 1982 and March 31, 1987.

To increase the power of the test, we expand the cohorts by one quarter and compare mortality rates for 1916:3 and 1916:4 with 1917:1 and 1917:2. This doubles the sample size and will decrease the standard errors by square root of 2. The results

for these expanded samples, reported in Table 8, are nearly identical but more precise than the estimates in Table 7.

Table 8
Difference in Difference Estimates,
Impact of Notch on Mortality Rates
5 Year Mortality from Age 65, Women as Control Group

	Before Notch	After Notch	Log Difference log(1) - log(2)
	Born in Q. 3,4 (1)	Born in Q. 1,2 (2)	
(a) Men born 1916:3,4 or 1917:1,2	0.1517 (0.0012)	0.1482 (0.0012)	0.0239 (0.0053)
(b) Women born 1916:3,4 or 1917:1,2	0.0820 (0.0009)	0.0819 (0.0009)	0.0012 (0.0042)
		Difference (a) - (b)	0.0227 (0.0067)

Standard errors in parenthesis. Rates calculated as in Table 6, referenced from older quarter-of-birth (16.3 and 17.1) for each column

6. Why Are Mortality Rates Higher for Pre-Notch Group?

The results in the previous section are rather striking—although the 1916 birth cohort receives larger Social Security payments than the 1917 group, the point estimate of five-year mortality for the older group, adjusted for the slight difference in age, is larger. This result is in stark contrast to the conventional wisdom. In particular, our results differ from those in Case (2001) who finds improved health from higher pension in South Africa, although that experiment provided a much larger income change to a much poorer population than the one we consider here.

Our results are, however, not alone in the literature. Eibner and Evans (2001) find that much of the impact of high income on mortality is actually driven by a

person's relative position in their reference group's income distribution. Ruhm (2000) finds that state-specific total mortality and eight of the ten cause-specific mortality rates are pro-cyclic. The lone death category that has a statistically significant counter-cyclic relationship is suicides. In this work, the level of economic activity is measured as the unemployment rate and even though a small fraction of elderly work, Ruhm also claims a pro-cyclic relationship for those aged 65 and over. Ruhm finds some evidence that poor health habits such as smoking rise but health investments such as routine exams decline when the economy improves.

Our first thought was that changes in some health habit such as smoking rates could explain our counter-intuitive result. Smoking rates are negatively related to incomes (Evans, Ringel and Stech, 1999), but in this population, as we demonstrate below, smoking quit rates are negatively related to income. Smoking cessation, even at advanced ages, has tremendous health benefits. According to the 1990 Surgeon General's Report on smoking, male smokers aged 65–69 have mortality rates that are three times higher than those who have never smoked. In this age group, former smokers who have been off cigarettes six to nine years have a mortality rate that is 40 percent lower than current smokers (Table 7, page 95). In the end however, we found that the impact of higher incomes on smoking rates was not large enough to explain the difference in mortality across the birth cohorts. To demonstrate this point, we examined data on complete smoking histories that is available as part of the Tobacco Use Supplements (CPS/TUS) to the regular September 1992, January 1993 and May 1993 monthly CPS surveys. These three samples are designed to be pooled together to form one large data set on smoking histories. Limiting the sample to 948 males

aged 65 who smoked five-year prior to the CPS/TUS, we find that 25 percent quit over the next five years. In a linear probability model, we regressed this outcome on controls for marital status, race, ethnicity, education, the month of the survey and log income. The coefficient on log income was -0.055 with a t-statistic of -2.5, suggesting that an eight percent increase in income will reduce five-year smoking quit rates by .44 percentage points. Given this modest change in the smoking rate, our back of the envelope calculation suggests that a lower smoking quit rate induced by higher Social Security payment can explain about 5 percent of the higher mortality rate among the 1916 birth cohort.

One area that we thought could possibly explain the results noted above is changing social networks. In recent years a growing literature has demonstrated a link between social networks and mortality with those less connected to their community, friends, relatives or coworkers experiencing a higher mortality rate. This literature is reviewed in Putnam (2000). The first empirical evidence for this relationship was generated from mortality follow-up surveys from small geographic areas like Alameda County, California and Tecumseh, Michigan. These surveys tracked a random sample of people over time, collecting important demographic data at baseline, plus measures of the social network such as church and group membership and contact with friends and relatives (Berkman and Syme, 1979; Blazer, 1982; House et al., 1982; Berkman, 1995 and 2000; Cohen et al., 1997; Colantonio et al., 1993; Zuckerman et al., 1984). Berkman and Syme (1979) for example found that the age-adjusted death rates for those most isolated were 2.3 to 2.8 times that of others. This relationship was found to be independent of self-

reported health status, socioeconomic status and health practices. The mortality impacts for the elderly are particularly important. Seeman et al. (1987) found that social ties remain a significant predictor of mortality risk even for those aged 70 and older. Their results suggest that for the elderly, contacts with friends and/or relatives are the most important ties. The study by Blazer (1982) of 30-month mortality in a sample of persons aged 65 years or older at baseline found that both a general lack of social ties with children and siblings as well as low perceived support from one's social network were each independently associated with increased mortality risk.

Given this literature, we looked for ways in which more income may reduce social networks. For example, many seniors move from their lifelong homes after the age of 65. If this is positively correlated with income, the 1916 cohorts may be more likely to move than younger groups. Migration may have many benefits, but the movement away from a known social network may negatively impact health. We could, however, find no evidence that the Notch impacted mobility. In the 1990 Census, because the census is conducted on the first day of the second quarter, three-quarters of the people 72 were born in 1917 and three-quarters of those aged 73 were born in 1916. In these age groups, nearly all who will receive Social Security have begun to claim benefits, so we can safely assume that the roughly 10 percent of the population without Social Security income in the Census are people not eligible. Looking at the difference in 5-year migration rates between 73- and 72-year-olds with Social Security and comparing this to the difference for those outside of the system, we find no "Notch" impact on migration rates.

In a related paper, Englehardt, Gruber and Perry (2002) use the Notch as an instrument for OASI payments in models that related whether seniors live independently. The authors find that living independently is a normal good and the elasticity of living independently given a change in OASI payments is -0.40. In this case, if living independently detracts from health, then the higher rates of living independently we would expect to find among the older yet higher-income cohorts could explain part of our results.

Another area where we might find some explanation for our results is in the post-retirement careers of the Notch generation. Research by others suggests that the Notch did not alter the *pre-retirement* age behavior of affected populations, but a change in retirement income might be expected to alter *post-retirement* behavior. In particular, because of lower Social Security benefits, we might expect the Notch cohorts (those born after 1917) to work more than older groups. As Lumsdaine and Mitchell (1999) suggest in their survey of the literature on retirement, the behavior of older Americans is increasingly complex. Older workers may leave the full-time labor force, but return as part-time workers, often working in different industries, and generally at a reduced wage from their primary career employment. This less intense form of labor force participation is aptly characterized as post-career employment. This work could have positive health benefits if the work keeps the seniors connected to the community and reduces social isolation.

We would expect to only find an impact of the Notch on part-time work. During the period for which we present mortality data individuals at or above age 65 could claim Social Security benefits and earn up to a specified amount without

incurring any reduction in the OASI benefits. Once the earnings threshold was crossed, however, the reduction in benefits was severe. Benefits were reduced by \$1 for every \$2 earned. These penalties were in effect until age 70 (72 before 1982, the penalty was reduced to \$1 for every \$3 earned in 1990). The penalty threshold increased in both nominal and real terms during the relevant time period, from \$5,000 in 1980 to \$9,720 in 1991, but even this higher level is less than half average full time earnings. The Social Security system discouraged full-time employment for OASI recipients, but not part-time employment, if the compensation was low enough. One possible response to the Notch is to increase part-time employment while receiving retirement benefits.

To investigate the possibility of a post-retirement labor force response to the Notch, we began by constructing a balanced data set of labor force information for those born between 1909 and 1920. Our source was the March CPS for years when the year-to-year cohorts were between the ages of 56 and 70. Only the CPS offers a large sample of the appropriate ages with earning and labor force status reported in a consistent manner. As discussed above, the main drawback to the use of the CPS is the poor identification of birth year. Since it is calendar year that changes the applicable benefit formula, this is a serious problem. We therefore define the Notch to equal 1 for people born in 1917 after they reach age 65. Since retirement prior to age 65 has little impact on Social Security payments, there should be little labor supply effects of the Notch. To control for the noisy identification of birth cohorts, we let the value equal 0.2 for the 1916 birth cohort.

With this data, we can construct an indicator that equals 1 if a person worked in a particular age/year cell. However, hours worked last week is only available starting in 1976 so to preserve the balance in the panel, we are forced to restrict our attention to people from the 1913–1920 birth cohorts when they were 62–70 years of age. We then construct two outcomes: an indicator for “worked” and hours worked per year. We regress this on year of birth and age effects, controls for education, race, and marital status, plus an indicator for cohorts impacted by the Notch. This model is similar in spirit and specification to that of Krueger and Pischke, with the primary difference being our use of balanced cohort samples and the definition of the treatment effect.

The results of this exercise are reported in Table 9. In the first column, workers have a 3 percentage point higher labor force participation rate than those from earlier birth after age 65. Breaking the treatment effect up into different age groups, we find in the next column that all of the difference is produced by a large increase in work after age 67. In the final two columns of the table, we continue the analysis using hours worked as the dependent variable. The same pattern is apparent. The Notch has increased work after age 65 for those born 1917 and later and all of this increase is concentrated in the post 67 age range. Can this higher level of work at ages 68–70 explain the relative fall in mortality for the 1917 cohort relative to 1916? The timing is consistent with Figure 3. Notice that the cumulative difference in mortality between the 1916 and 1917 cohorts does not start to appear until after age 68. Table 9 provides empirical support for the hypothesis that the size of Social

Security benefits do alter labor supply among the elderly. They also offer an explanation for the counter-intuitive results in Tables 5–7.

Table 9
 OLS Estimates, the Impact of the Notch on Post-65 Labor Supply
 Men Born 1913-1920, Aged 62-70,
 March CPS

Independent Variables	Worked last year	Worked last year	Hours last year	Hours last year
Notch effects				
1917-20 Cohorts, aged 65+	0.0286 (0.0099)		69.50 (19.66)	
1917-20 Cohorts, aged 65-67		0.0072 (0.0109)		35.42 (21.62)
1917-20 Cohorts, aged 68-70		0.0518 (0.0113)		109.13 (22.53)
R ²	0.120	0.120	0.163	0.163
Mean of dependent variable	0.456	0.456	738	738
Mean of dependent variable, age 68-70 (N=12,135)	0.287	0.287	355	355

Both models control for education, race, marital status, year of birth and age.

7. Conclusion

Heading into retirement, men born in the last part of 1916 looked similar in many respects to those born in the first half of 1917. They had similar incomes, labor force participation rates and intensity of work. Once they reached retirement age however, one group was rewarded with substantially higher Social Security payments than the other. We use this variation to examine the potential impact of income on mortality in an elderly population. To be clear, our research design answers a very narrow question: will mortality be impacted by transferring more income to an elderly population? Our results are somewhat counterintuitive: the 1916 birth cohort which

received more income in Social Security benefits ends up to have higher mortality than the lower earning 1917 birth cohort. We suggest that these results could be driven by changes in labor supply by the older cohort. The lower incomes received by the Notch babies encouraged them to have more post-retirement part-time work. In fact, our results suggest that this cohort is five percentage points more likely to work during ages 68–70 than older cohorts. A number of researchers have suggested that among the elderly, social isolation is an important cofactor in mortality. If part-time work keeps the elderly engaged and helps prevent social isolation, then the increased part-time work may have reduced mortality.

Do higher incomes reduce mortality? These results suggest that in this particular case, the source of the income is probably as important as the amount of income. Here, the lower incomes for the 1917 cohort seem to have encouraged an activity that is in the end healthy. We might then expect very different results from an “income effect” generated by a wage change for example. This type of income change is particular to a specific population so the results cannot be readily applied to other groups. But as we note in the introduction, this is not an uninteresting group or policy consideration. The Federal government routinely proposes changes to social insurance programs that resemble the Notch experiment in magnitude of dollars.

CHAPTER TWO

THE 1918 INFLUENZA EPIDEMIC AND LATER-LIFE MORTALITY

1. Introduction

Inter-generational transfers of socioeconomic status are a topic of continuing interest in the social sciences. Researchers from a variety of disciplines have demonstrated that a variety of economic outcomes such as earnings, income, wealth, education, and occupations are correlated across generations. As health economists developed the idea of health capital it became apparent that inter-generational transfers of health are also plausible through genetic endowment or through expenditure on children's health through immunization, nutrition, good health habits, and public health expenditure (Grossman, 1972; Smith, 2000). In the last 20 years, medical researchers have begun to consider another possible channel for the intergenerational transfer of health. (Barker and Osmond 1986; Ben-Shlomo et al., 1991; Barker et al., 2001). This is neither genetic, nor a parent purchasing improved health for their children; it is the mother's health status directly affecting the child's health status. The theory suggests that the mother's health changes the child's *in utero* development in ways that may be difficult or impossible to alter later in life. This theory is most closely associated with the work of D.J.P. Barker, and is referred to as (by other researchers) the Barker hypothesis.

Evidence in support of this theory falls into four broad groups. First, there are epidemiologic studies showing that birth in geographic areas with poor health indicators predict high incidences of cardiovascular and other chronic diseases late in

life. These same studies do not show similar elevated susceptibility to other major disease groups, such as cancer. Second, perinatal and neonatal studies that show maternal deprivation is associated with underdevelopment of the vascular, pulmonary and endocrine systems of infants. Third, studies of older adults which document higher rates of abnormal vascular, pulmonary and endocrine conditions in individuals born in regions with low maternal health indicators. Fourth, animal studies which follow offspring from fetus through youth to adulthood, directly relating *in utero* conditions to the development of chronic disease in old age.

There are at present no longitudinal human medical studies that replicate the animal studies; that is, no researcher has conclusively demonstrated that degenerative diseases in later life occur in individuals with underdeveloped systems as infants. This is not surprising given the recent vintage of the line of research; the babies in the first infant studies are barely twenty, but it has meant that the Barker hypothesis is still controversial within the medical profession. Critics contend the relationship between infant development and chronic disease is an association, but not a causal relationship. The confounding variable is poverty. Poor regions have poor health indicators and parents pass on poor health habits and poverty to their children, so epidemiology cannot prove the hypothesis. Fetal and neonatal underdevelopment may not be persistent, so perinatology cannot furnish definitive studies. Animal models are not persuasive since human behavior is adaptive in ways animal behavior is not. The Barker hypothesis remains controversial; there is substantial evidence for it, but no definitive proof. (*The Lancet*, 2001)

It is hard to imagine an ethical means of testing the hypothesis using the tools of medical research. We cannot randomly assign women to deprived and enriched environments during pregnancy and track their children's mortality without intervention. This has led to interest in other possible tests of the hypothesis, a field experiment which would constitute a definitive test, as the British Medical Journal has put it, "an ordeal"¹⁰ (Susser, 1999). Similarly, health economists have begun to consider how health shocks might enable us to devise econometric tests of the Barker hypothesis. The endogeneity of health status is a problem similar to many in empirical microeconomics, trying to isolate the impact of one variable in a complex social and economic environment.

Establishing Barker's theory has important implications for economists interested in development and intergenerational transfers. Under the Barker hypothesis, there can be no clear distinction between one generation's welfare and that of the next. An investment in the current adult generation becomes an investment in their children. A Barker effect of sufficient magnitude could mean that the cheapest means of investing in children becomes investing in their parents.

Recently, an economist (Almond, 2003) has used the 1918 influenza epidemic as a quasi-experiment to identify the relationship between maternal health and various social and health outcomes among birth cohorts from 1915–1920. Health indicators in previous research have been chronic, so it is not possible to separate the effects of the health environment from other persistent regional differences. (Barker, 1994) The flu

¹⁰ The phrase refers to a statement of Galileo who was arguing against scientists use of accumulated suggestive results as proof.

epidemic was a very specific event that raised mortality rates over a 6 to 9 month period (Frost, 1920). The rate of influenza mortality also differed substantially from place to place (Mortality Statistics, 1918). Do cohorts born in areas that suffered more from the flu show worse outcomes than do those from states that suffered less? Almond concludes that both time series and cross-sectional evidence is consistent with the Barker hypothesis. This appears to lend strong support to the theory, and to extend it by estimating the impact of a particular acute health shock on the next generation's health outcomes. If his conclusions are correct, Almond has made an important contribution, not only to health economics, but also to establishing the value of health economics for medical research.

The influenza epidemic of 1918 was unlike any other before or since. For both the world and the United States influenza was a minor infectious disease problem before 1918. Cases of influenza were not tabulated separately by the Public Health Service (Beveridge, 1977). In 1918, it suddenly, and for reasons which are still not completely clear, became a severe, life-threatening illness for all ages and classes (Reid and Taubenberger, 2003; Beveridge, 1977; Kolata, 1999; Niall, 2002). Because it came as a shock, the epidemic had no effect on the decision to begin a pregnancy for couples who conceived before the fourth quarter of 1918, and because most pregnant women survived and delivered viable offspring, a comparison with preceding cohorts approximates random assignment between treatment (influenza exposed *in utero*) and control (born before the epidemic). We discuss the influenza epidemic in more detail in Section 3.

The other health shock that has been proposed as an ‘ordeal’ or a quasi-experiment to identify a Barker effect is the Dutch famine winter of 1944–1945 (Stein et al., 1975). This was a much more severe and long lasting shock to health, but the population sizes are much smaller, and the exposed cohort has not yet begun to experience substantial mortality from chronic diseases. (Stein et al., 1975; Roseboom, 2000, 2001).

The current chapter extends Almond’s analysis using a census of deaths from a restricted-use version of the National Mortality Detail Files (NMDF) of the United States Center for Vital Statistics. Almond’s conclusions about a Barker effect on mortality are limited by his reliance on census data. By combining census data with the NMDF, we are able to calculate mortality rates with considerable precision, and test the extent to which the ‘flu-baby’ cohort experienced elevated mortality rates due to maternal exposure to influenza.

Through the generous co-operation of the National Center for Health Statistics we have had access to the NMDF between 1979 and 1990. Observations in the NMDF include state of birth, month and year of birth, and month and year of death. To calculate mortality rates we must relate the death counts to population size at some reference date. This is accomplished by using the 1980 Integrated Public Use Micro-Samples, a 7% sample of the United States’ 1980 Census. The 1980 Census includes age, quarter of birth, and state of birth, allowing us to merge these two data sets to construct mortality rates for state of birth by quarterly cohort. The precision this gives in observing the ‘flu babies’ and their adjacent cohorts’ mortality is the principal contribution of this paper.

The 1980s' mortality rates are combined with mortality rates from the 1919 Mortality Statistics of the United States (Census Bureau, 1920) which summarized the data published in the first 5 years of regular publication, 1915–1919. In this way we can potentially investigate time series and cross sectional variation in the likelihood of exposure to influenza.

2. The Barker Hypothesis

Until the 1980s there had been little consideration of the role *in utero* experience might play in the rates of heart disease and other chronic degenerative diseases in the developed world. That this is no longer the case is principally due to the work of David J. P. Barker and his collaborators. Barker's own account of his work (Barker, 1998) is that insults to health *in utero* result in poor development of important physiological systems, notably the vascular. These systems are far less plastic in the child than they are in the fetus, and still less plastic in the adult. Thus, early life "programming" (his term) can have consequences late in life when these systems become less robust. No person's vascular system works as well at age 65 as it did at age 25; those who have poor vascular (or lung, or endocrine) development fall below the threshold necessary for proper physiologic function and become ill or die. The theory fits nicely into the health capital framework developed by Grossman (1972); some individuals start with a lower endowment of health capital due to their mother's health status. However, while Grossman stresses that individuals can invest in their health, Barker believes that poor programming is difficult to offset after the developmental window has closed.

The biological basis for a Barker effect is centered in the importance of programming. The programming effect that Barker outlines suggest that when, due to under-nutrition or placental abnormality the fetus cannot fully develop at the appropriate time, then the organ will be mal-formed and will remain so throughout life. Under-nutrition of the fetus may be the result of restricted maternal nutrient intake (Godfrey 2001), or it may result from illness interfering with nutrients available to the fetus (Gewold 1983). Barker cites evidence that undernourished babies show differences in liver metabolism, endocrine development, and changes in the structure of the heart, blood vessels and kidneys. Irving et al. (2000) find serological evidence of influenza exposure in pregnant women's blood samples is associated with an increased rate of complications in pregnancy, thus connecting the Barker hypothesis with exposure to influenza. Barker (1998) shows that in the early 20th century, young women in London had unusually low rates of infectious diseases, and that their children have unusually low rates of cardiovascular disease in the 1980's (pp. 172-175). The effects are summarized by Barker (1998 p. 145):

“A mother's ability to nourish her baby ... has little effect on the baby's size at birth, but nevertheless programs the baby. The fetus adapts to undernutrition by changing its metabolism, altering its production of hormones and the sensitivity of tissues to them, redistributing its blood flow and slowing its growth rate....Adaptations to undernutrition that occur during development can permanently alter the structure and function of the body.”

Evolutionary biologists find Barker's ideas plausible. Since it is the first 40–50 years of life when humans reproduce and raise offspring to adulthood, survival of individuals beyond that point is not of great significance for survival of a line of

descent (Diamond, 1997). It would confer an evolutionary advantage if, during periods of scarcity, mothers can protect their welfare by stinting on development of the fetus in a way that does not reduce the offspring's fitness during its own period of reproductive importance. What happens late in life is less important for transmitting genetic material to subsequent generations. There are now numerous animal studies which confirm that deprivation during pregnancy affects the offspring's health in later life. Robinson et al. (2001) summarize these results. Langley-Evans et al. (1994A, 1994B, 1995, 1997) and Gardner et al. (1997, 1998) have published a number of studies showing higher blood pressure and increased dyslipidemia in rats whose mothers faced restricted protein intake. Similar findings exist for sheep (Hoet and Hanson, 1999). The debate continues over whether this effect is quantitatively important in humans.

Barker believes that the effect is very large. He asserts (Barker, 1998, 2001) that early life programming accounts for the rising and then falling rates of heart disease in the developed world. Studies such as the Framingham study that attribute the falling incidence of heart disease to lifestyle changes¹¹ overestimate lifestyle effects, because they omit the influence of rising levels of maternal health on the next generation (Barker, 1998, 2001; Azambuja, 2002). As living standards rose and more people lived to old age, the rate of chronic disease rose because maternal health was poor, and then began to fall because the next generation was in a better position to nourish their babies.(Barker, 2001; Azambuja, 2002)

¹¹ Generally, age-specific rates of heart disease are estimated to have declined approximately 40% over the last 30 years. Barker believes this may be largely due to the effects of fetal programming. (See Barker, 1998, Chapter 4.)

Because statistical data on maternal well being in the early 20th century is in short supply, Barker's original research associated late in life death rates with infant mortality rates (Barker, 1986). Barker asserts that infant mortality is a proxy for maternal health, not simply a proxy for social conditions. This is a controversial point in itself and critics of Barker's work feel this is an unproven assertion (The Lancet, 2001; Susser, 1999). Even if infant mortality rates measure maternal health to some extent, that is clearly not all they measure, and disease vectors such as crowding, unsanitary water supplies, environmental toxins, restricted or low quality food supplies, rather than maternal health, might explain the association between infant and adult mortality. Barker considers this possibility, but rejects it on the grounds that later life mortality effects are concentrated in diseases which represent the breakdown of physiologic systems. Other diseases of mature adulthood, such as cancer, are linked to lifestyle and environmental factors, but are not associated with early life conditions (Barker, 1998). The possibility remains that it is neonatal rather than perinatal environment that provides the link, but at that point we are more than half-way to accepting Barker's larger point about the connection between health in later life and early life programming.

Almond's claim of a strong association between the influenza epidemic and chronic disease in later life would, if confirmed, be a major contribution. Because the influenza epidemic was an acute shock distributed across the socioeconomic spectrum, establishing such a connection would imply that not only was maternal health an important variable for predicting later life mortality; short term alterations in maternal health have quantitatively important effects.

As noted above, the Barker hypothesis is largely consistent with the models developed by health economists. In the health capital model originally proposed by Grossman (1972) and recently restated in Smith (2000), an individual survives until their stock of health falls below a critical level. Some individuals are born with a greater stock of health than others, some individuals receive greater health investment by their parents during childhood, and all individuals try as adults to achieve an optimal combination of health, wealth and consumption. *Ceteris paribus* those with higher levels of health endowment will tend to live longer. Barker adds another path by which parents can influence their children's health. Previously, the endowment was either fixed (genetic) or immediately observable (smoking while pregnant, taking a child for immunization). Barker adds a pathway that is latent for fifty or sixty years, but that, unlike the genetic component, is alterable both by giving individuals better information and by social policies that affect the health status of pregnant women.

3. The Great Influenza Epidemic

There is a consensus on the epidemiology of the great influenza epidemic in the United States (Kolata, 1999). It begins in Boston at the very end of August 1918 with a handful of cases among some recently arrived sailors. It spread through Boston and to a nearby army camp by early September. After that, there is little variation in the time at which the illness strikes different parts of the country (Kolata, 1999). October 1918 is the peak month for deaths in 21 of the 30 states reporting data from 1918.¹² November is the peak month for the remaining states. In reporting states the

¹² Six states' peak month was November, three states' peak month was December. Of the states with peaks after October, two had high mortality rates, three had low

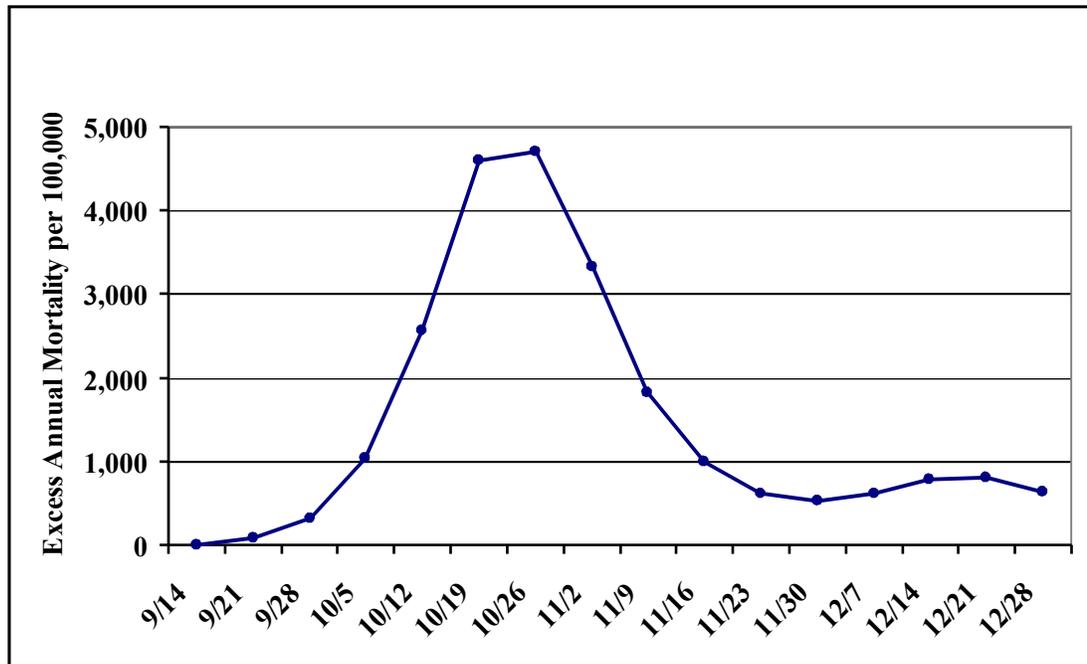
epidemic was responsible for over 500,000 deaths in an eight-month period, 21 percent of all deaths recorded during these months. Perhaps the most unusual feature of the epidemic is the large number of healthy young and middle-aged adults who died. Mortality rates were as high for those aged 20–29 as those under 1 or over 70 (Jordan, 1927). The disease seemed to particularly single out the healthiest, most robust young people, this according to four of the most prominent physicians in America, who had been dispatched by the surgeon general to investigate the outbreak in Boston. This included W.H. Welch, a founding faculty member of Johns Hopkins Medical School and the pre-eminent pathologist of his time. When Welch began his first autopsy he was stunned by the appearance of the cadaver’s lungs and remarked, “This must be some new kind of infection or plague.” (quoted in Kolata, 1999). A disease that shocked Welch must be considered an unforeseen shock to the general public.

There is no other documented case of an influenza epidemic with a similar mortality pattern (Kolata, 1999). While the onset was sudden, and the peak realized in October (see Figure 6), deaths from influenza did not fall back to their pre-1918 levels for several years, and another smaller epidemic occurred in 1920 (PHR, 3-12-1920). This is one reason we compare the ‘flu-babies’ to preceding, not succeeding cohorts. Also, in most epidemics the poor and those in urban areas suffer higher death rates than the general population during an epidemic caused by a contagious virus, but this was not true of the 1918 influenza epidemic. Mortality rates were higher in

mortality rates, and four were on neither the high nor low list. Eight are west of the Mississippi River. (Mortality Statistics, 1918, 1919)

rural areas than in cities, and African Americans suffered less mortality from the disease than Caucasians (MS 1919; Frost 1920).

Figure 6
'Excess Mortality' from Influenza—Fall 1918 by Week



A puzzling aspect of the pandemic is that it followed closely upon a mild influenza epidemic which occurred in the spring of 1918, but which did not involve substantial mortality (Niall 2002; Patterson 1991). Recent research suggests that the two epidemics may be related, but that between the spring and fall of 1918 the virus underwent a genetic shift, possibly by exchanging DNA with an avian influenza (Reid and Taubenberger, 2003). However, exposure to the spring virus does not appear to have conferred immunity, and the cause of the virulence, especially among young adults, remains an enigma.

Unfortunately, there is little direct evidence on the neonatal health of *survivors* in the birth cohort exposed to influenza *in utero*. There is also some

controversy over whether birth weight and Barker effects are closely related (Barker 1998) and, the circumstances under which birth weight is properly used as an index of fetal health (Wilcox 2001). Nevertheless, it is the only measure of the fetal health of survivors we are likely to develop for this cohort. Mortality rates are high for this group (BS 1918), and Englund (2003) documents maternal and infant morbidity from modern influenza, but the epidemic predated any systematic data collection on newborn health indicators. Steckel (1999) cites series on birth weights for 6 European and American cities, but none of the American cities' data include the 1915-1920 period. Data for the European cities (Dublin, Edinburgh, Vienna) shows a local minimum at about the right time period, but since World War I was also affecting European food supplies, it is hard to draw conclusions about the impact of influenza. Birth weight data gathered from hospital records at this period also suffers from changing selection bias. Wealthier families tended to prefer home deliveries, but the fraction doing so was shrinking with the spread of scientific medicine. There is no good source of evidence on the direct effects of the epidemic upon birth weights.

As a general rule episodic illnesses are not believed to have much effect on newborn health in the developed world (Hartert 2003). They have, however, been shown to have an influence in the contemporary developing world, which may be a more relevant comparison for the 1918-1919 time period in the United States.(Spencer 2003) Also, the 1918 influenza epidemic was far more virulent than other episodic illnesses, and it is well known that children suffer from low birth weight when exposed to chronic disease. Malaria has been shown to reduce birth weight in a number of studies (Spencer 2003), and children born in the wet season in

Gambia have been shown to be stunted compared to those born during the dry season. The wet season is known as the high-disease portion of the year (Moore 1997). The best documented developmental disorder associated with influenza is neurological impairment, especially Schizophrenia (Wright 1995). This is associated not only with the 1918 pandemic, but later, less severe outbreaks as well (Wright 1995). It is also associated with low birth weight (Kunugi 2001). While the evidence we have is fragmentary and only indirectly relevant it points toward a relatively unhealthy group of newborns in the 1918.4-191.3 time period.

While it is clear that the epidemic did not affect the whole country equally, how to measure the severity of the epidemic for the purpose of testing the Barker hypothesis is much less clear. The only reliable data for most of the country is found in "Mortality Statistics of the United States." Thirty states report mortality statistics for 1918, twenty-five report for the all five years 1915–1919. We want to measure the effect of the influenza epidemic upon fetal health, or at least, infant health, but it was not deceased women who bore children, nor is it deceased infants who die of heart disease seventy years later. When we use mortality rates to draw conclusions about the impact of a particular health event upon the living we must exercise caution to avoid making serious errors of interpretation. For acute conditions such as influenza, epidemiologists decompose mortality rates into two components; an incidence rate and a case fatality rate which when multiplied together produce the mortality rate (Frost, 1920). Whether infant, adult or general mortality is used as a measure of influenza's intensity, it is only variation in the incidence rate which measures the likelihood of a survivor's exposure. In the extreme case where incidence rates are

constant but case fatality rates are high and variable, survivors from high mortality states may have a lower rate of exposure than those from low mortality states.

Consider the following example for an arbitrary disease: incidence rates across two areas for all age groups are constant at 40%, case fatality rates for the target population are either 25% (in area X) or 50% (in area Y). Thus, the mortality rate in X is 10%, in Y, 20%. How many of the survivors were exposed? In area X, it is $(.4-.1)/(1-.1) = .33$. In area Y, it is $(.4-.2)/(1-.2) = .25$. If area Y experiences higher later-life mortality, it is not because its inhabitants were more likely to be exposed. The relevance of this example to the case of influenza is this: while incidence may not be correlated with chronic health conditions or socioeconomic status, case fatality is more likely to exhibit such correlation. Those in poor health are more vulnerable to health shocks. (Grossman, 1972) Empirical evidence for this includes the relatively low incidence, but higher case fatality of African-Americans (Frost, 1920).

4. Methods

Using the influenza epidemic to test for a Barker effect will require a dataset with health variables from the time of the epidemic, mortality experience at a point where chronic disease mortality is substantial, and information on sufficient covariates to control for potential confounders. No single data source contains all three types of information. Accordingly, we combine data from several sources to create our analytic data set. As the design of the data set determined the econometric specification, we begin by describing the data sources. Next we develop our estimation strategy, and then discuss interpretation of the chosen variables.

4.1 Data Sources

As outlined in the previous chapter, merging restricted use data from the National Mortality Detail Files (NMDF) with the 1980 Census Public Use Micro-Samples allows us to construct mortality rates by state for quarterly birth cohorts. An advantage of this approach is that it is based upon a census of deaths occurring in the United States between 1979 and 1989, not the experience of a sample population. One disadvantage is the absence of individual-level variation; we cannot match a death back to a particular individual in the census. When compared to available micro-level data sets, however, the cohort-based analysis is superior for two reasons beyond the size of the population covered and the completeness of the coverage. Most importantly, death and birth are precisely observed; we have the birth month and death month for each individual. We also have state of birth provided from physician records, or by next of kin. Second, because the NMDF is drawn from the information on death certificates we avoid issues of attrition. There is a high degree of confidence that the information is complete and accurately recorded. The NDMF is restricted to those who die in the United States. Since our interest is those born in the United States and emigration has been minimal, the NDMF is nearly ideal as a source of death information.

Use of the NDMF forces us to take a cohort-based approach. The NDMF is a census of deaths. It does not provide information on the population in which the deaths occurred. To develop mortality rates we need a census of the U.S. population at a point in time. The 1980 Census is preferred because it is the census when the

relevant birth cohorts attained the age of sixty and the last to identify respondents' quarter of birth. Because we do not need geographic identification beyond the state of birth, we can use the 5 percent PUMS sample and two additional 1 percent PUMS samples that differ only in their level of geographic identification. Thus, our cohort sizes are based upon a 7 percent sample of the U.S. population. By merging the death counts from the NMDF with the population estimates from the 1980 PUMS we construct mortality rates for each state and quarter of birth cohort in every quarter between January 1, 1980 and December 31, 1989.

The third type of information we desire is data on exposure to the influenza epidemic. The ideal data source would provide the number of live births in each state whose mothers had contracted influenza. However, at this time the vital statistics of the United States consisted of birth and death statistics for only a portion of the states. There was no ongoing collection of incidence data of any kind, and certainly none focused on perinatal health experience. In studies of the Barker hypothesis, mortality data is typically used as a proxy for incidence, which we have already noted introduces a possible source of bias, a subject to which we return in more detail below. The alternative is to simply use birth during or shortly after the influenza epidemic as the proxy variable. In this case, the lack of mortality data is not a limitation. We will present results based upon both approaches. Were we to develop negative results using only the time series approach, the natural question would be whether a combination of cross-sectional and time series variation would produce a different conclusion. Our data on mortality is drawn from the Mortality Statistics for the Death Registration Area of the United States for 1919. In that year the Census

Bureau summarized and restated death rates by cause of death and state for the period 1915–1919. In so doing, they added additional years to the results for some states, because results that met the Bureau’s standards had been generated too late for the original report.¹³

Combining the three sources of data produces an analytic file that combines mortality rates in the 1915–1919 period, mortality rates in the 1980s, and demographic characteristics for each quarterly birth cohort in each of the 25 states.

4.2 Mortality as a Proxy for Incidence, with a Note on Available Incidence Data

How good a proxy for exposure to influenza *in utero* are mortality rates? For an acute condition such as influenza the mortality rate is the product of the incidence rate and the case fatality rate. Barker and other researchers of the Barker hypothesis typically use mortality rates as health measures, but our interest is not in how many died from influenza in 1918, it is how many pregnant women contracted influenza. When we examine mortality rates there is certainly the possibility that the variation observed comes not from variations in incidence, but variations in case fatality. Variations in incidence might be uncorrelated with existing health status. Even very healthy people get influenza, and accounts of the 1918 epidemic describe the young and healthy as particularly susceptible. Case fatality—the risk of death given that an individual has contracted influenza—is another matter. It is likely that variations in

¹³ We know there was substantial concern over the quality of the data, because in a few instances in the published report data is omitted for a state with a note that the data did not meet the department’s quality standards.

case fatality are correlated with health status. If this is so and substantial variation comes from case fatality, the mortality rates for influenza will be correlated with pre-existing health status, and we will only be demonstrating the sort of association between community health status at the time of birth and later-life mortality that has already been demonstrated many times before.

Incidence data for any epidemic occurring before WWII is very unusual. However, because of the dramatic impact of the influenza epidemic, the United States Public Health Service undertook a survey of incidence in ten cities and one rural area¹⁴ where USPHS field offices were located. This is not a nationally representative sample, and only the report, not the field data, is available, but W.H. Frost, a very eminent epidemiologist, issued the report. His main subject is the relative impact of incidence and case fatality upon mortality rates. Given the rarity of incidence data, and the importance of the source of mortality variation for establishing Barker's hypothesis, his opinion is of some interest.

Frost's report was published in the 12 March 1920 issue of Public Health Reports. In each community canvassers went door-to-door in 10 or more enumeration districts (i.e., neighborhoods) until the greater of 5% or 5,000 persons could be surveyed. Inspectors conducted the canvasses between December 1918 and March 1919. They recorded the race, sex and age of each individual and whether the individual had been sick since September 1, 1918, with "influenza, pneumonia or an

¹⁴ Localities were: San Francisco, CA; San Antonio, TX; Little Rock, AR; Des Moines, IA; Louisville, KY; Macon, GA; Augusta, GA; Spartanburg, SC; Baltimore, MD; minor Maryland communities; and New London, CT.

indefinite illness which might have been influenza” (Frost, 1920). It is not clear how individuals were selected from households, but Frost asserts that the inspectors recorded “date of death if death resulted” (Frost, 1920, p.586). This implies some procedure to detect deceased residents and thus avoid survivor bias.

Frost presents incidence rates for each area, but groups fatality rates into three groups as he considers the number of deaths in some areas too small to be statistically useful. Frost then graphs incidence, case fatality and mortality rates. While incidence shows a consistent age/sex pattern across localities, case fatalities show different pattern between regions, and it is this pattern that is reproduced in the mortality rates. His analysis of variation in mortality rates by age and sex concludes:

“It is evident, however, that the relative mortality in these various groups is determined more largely by case fatality, which varies within wider limits than case incidence, and so without a full and exact knowledge of the variations in case fatality, statistics of mortality are by no means translatable to terms of relative morbidity.” (Frost, 1920)

The use of mortality as a proxy for incidence is problematic, but no other measure is available. Use of age-and sex-specific mortality rates appears to make mortality worse as a proxy for incidence, not better, since influenza is transmitted across ages and genders, but case fatality may vary locally due to health status.

Frost’s data presents a substantial problem, not only for the present study, but for the use of mortality rates generally as a proxy for incidence. His examination of race shows that while African-Americans were less likely to contract influenza than whites, their case fatality was generally higher. We cannot draw firm conclusions from such limited data, but it appears that case fatality is likely to be correlated with unobserved health status, and thus mortality rates from the flu may be biased by pre-

existing health status. Our strategy to control for this problem is to include a state-level measure of persistent mortality as a potential explanatory variable. We prefer this strategy to state fixed effects because the parameter estimates generated can be compared to those for influenza mortality. State effects do not lend themselves to testing an alternate hypothesis; however, we use state effects as an alternate specification to test our results.

4.3 Selection of Time Period and Variables

The decision to make the oldest cohort in the analysis those born in the first quarter of 1915 is a simple one. Mortality statistics are only available for 1915 and after. The reasons for choosing the fourth quarter of 1919 as the younger cohort are subtler. All the cohorts chosen have a family structure defined before the onset of the epidemic, those born in 1920 or later have a family structure defined after the epidemic, that is, by survivors of the epidemic. Because we are testing a hypothesis about physiologic development *in utero*, it is desirable to use a cohort that did not include those whose parents were widows or widowers as a result of the epidemic. Given the ‘W’-shaped mortality that characterized the 1918 epidemic, it is possible that four or five percent of households with young children were in the widow or widower category in some high-mortality states. Children conceived after the epidemic differ from those born before or during the epidemic in a non-random way. Their parents survived, and (in the case of widows and widowers) attracted new spouses. This could potentially cause different mortality experience due to selection rather than fetal development.

The next task is to define the treatment population, or more precisely, since we do not observe incidence, the intention-to-treat population. The basic facts are not in dispute, the influenza epidemic in the United States began in September 1918 in a few localities, became a national epidemic very quickly, and after the initial peak continued at a lower level for some months. Over 80% of all influenza mortality occurred in four months, from October 1918 through January 1919. Figure 6 demonstrates how sharp the peak was. From a level near zero in mid-September, the mortality rate rose to an annualized rate of almost 5,000 per 100,000 during late October¹⁵ (Mortality Statistics 1918). The national mortality rate then falls below 1,000 by December, but stays above the background level for some months. The timing of the epidemic varied comparatively little across states. Of the 30 registration states in 1918, 21 had their peak mortality rate during October, six during November, and three during December. Table 10 shows that the influenza mortality rates before 1918 were less than 10% of 1918's level, and this understates the difference, as the 1918 mortality occurs almost entirely in the fourth quarter. The natural candidate for the treatment population is those babies born or conceived during the fourth quarter of 1918, quarterly cohorts 1918:4 through 1919:3. The evidence of relative cohort size in the 1980 Census supports this. Figure 7 shows that those four cohorts are unusually small given their age, and form a distinct group. The usual pattern of large first and third quarter cohorts, and small second and fourth quarter cohorts is suppressed. The 1918:4–1919:3 cohort is the natural choice for the treatment

¹⁵ Technically this is excess mortality, but the background mortality was very low.

population. Figure 8 displays the same data as a separate series for each quarter of the year and emphasizes the singularity of the 1918:4-1919:3 time period. One explanatory variable will be a dummy denoting membership in this cohort.

Table 10
Influenza Mortality Rates by Year: 1915-1919
Deaths per 1,000 Population—Death Registration States

Year	Influenza Mortality	All Cause Mortality
1915	0.2	13.3
1916	0.3	13.9
1917	0.2	14.1
1918	3.0	18.0
1919	1.0	12.8

There are several possible approaches to defining the relevant influenza mortality rate. One would be to use the mortality rate in the state and quarter of birth. However, it is the mortality shock that is relevant, not mortality in the quarter of birth. Figure 6 shows that the shock is highly concentrated in late 1918, so the fourth quarter 1918 mortality rate is the preferred variable. Here the limitations of the Mortality Statistics become a binding constraint. National figures for mortality rates by cause are available by quarter, even by month, but state-specific estimates are only available annually. Given the low background level of mortality, the 1918 state-specific influenza mortality rate does not introduce a confounding variation. This mortality rate applied to the treatment cohort will be a second explanatory variable. It is the best available proxy for capturing the cross-sectional variation in the incidence of the influenza epidemic.

Figure 7
QOB Cohort Size - 1980 IPUMS

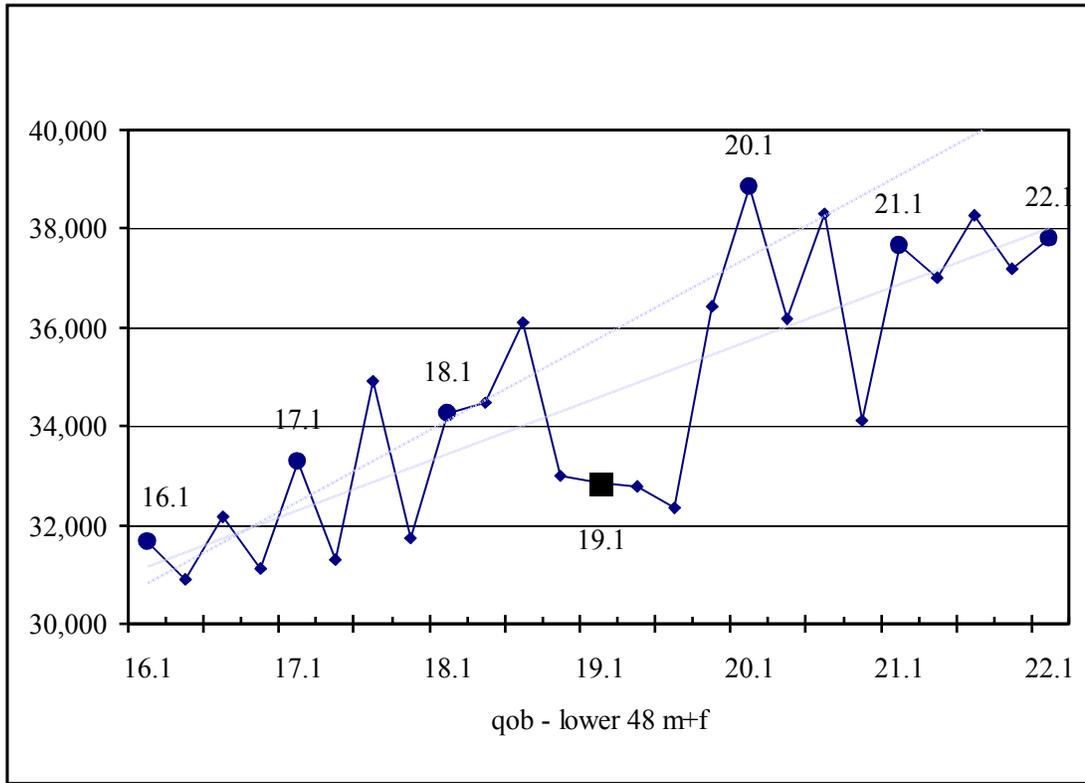
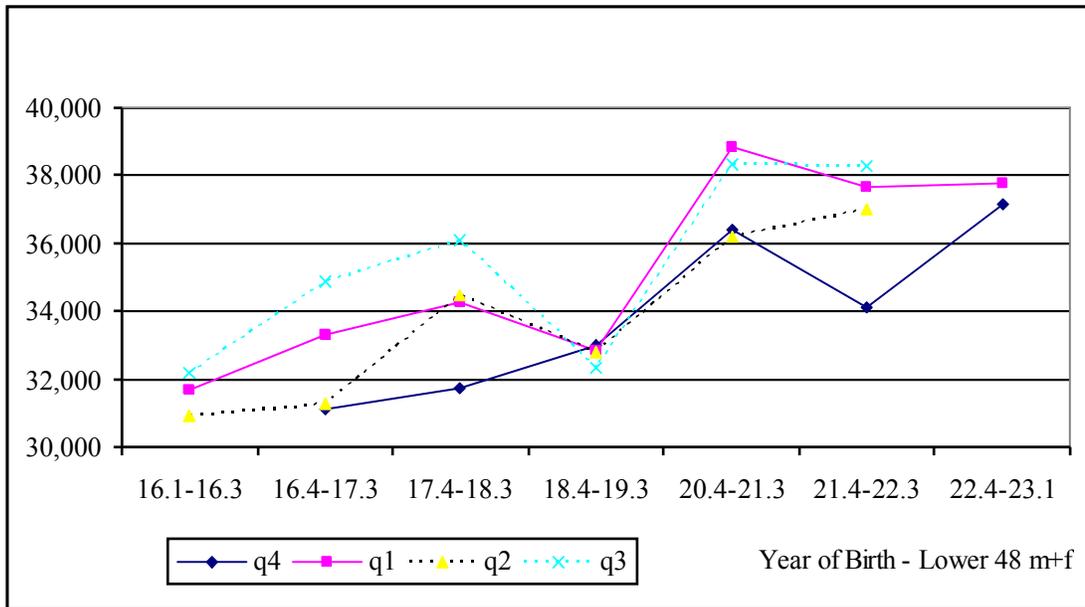


Figure 8
Cohort Size by Quarter and Birth Year - 1980 PUMS



There are several possible approaches to defining the relevant influenza mortality rate. One would be to use the mortality rate in the state and quarter of birth. However, it is the mortality shock that is relevant, not mortality in the quarter of birth. Figure 6 shows that the shock is highly concentrated in late 1918, so the fourth quarter 1918 mortality rate is the preferred variable. Here the limitations of the Mortality Statistics become a binding constraint. National figures for mortality rates by cause are available by quarter, even by month, but state-specific estimates are only available annually. Given the low background level of mortality, the 1918 state-specific influenza mortality rate does not introduce a confounding variation. This mortality rate applied to the treatment cohort will be a second explanatory variable. It is the best available proxy for capturing the cross-sectional variation in the incidence of the influenza epidemic.

A potential problem with the influenza mortality rate is that there may be state-to-state variations in assigning cause of death. Many patients develop pneumonia as a result of contracting influenza, and pneumonia was itself a significant cause of death in the 1915–1919 period. It is possible that some states were more likely to assign co-morbid cases to one disease or the other; in that case, high mortality could potentially represent high reporting rather than high fatality. To test for this possibility we computed the correlation coefficient for 1918 influenza and 1918 pneumonia deaths. If there is variation in reporting we might expect to find a negative correlation between the two causes of death, since a single death is assigned a single cause. Instead, we find a positive correlation coefficient, 0.435. High

influenza mortality rates are associated with high pneumonia mortality; there is no evidence of a state-specific selective reporting problem.

The alternative hypothesis we propose to test is that any observed relationship between influenza mortality and later-life mortality is not due to the shock of the influenza epidemic, but to correlation between influenza mortality and the background level of mortality in a community. Mortality rates here stand in their usual role in this literature, as proxies for health levels in a community. Given the variety of epidemiologic evidence for the Barker hypothesis, it would be surprising if there were no relation between mortality at time of birth and later-life mortality. In this case, the alternative hypothesis is a negative one, that the evidence from the influenza epidemic is really no different from that in the existing literature, and does not provide a stringent test of Barker's theory.

To this end, we take as our measure of persistent mortality the mean all cause mortality rate within each state for the years 1915–1917. We do not attempt to limit this measure to the mortality of young women or any other target population. As was pointed out in the discussion of incidence and mortality above, we desire a proxy for exposure to influenza, not maternal mortality. Taken together, the three variables, birth in the influenza cohort, cohort birth interacted with influenza mortality, and persistent mortality, should allow us to test the Barker hypothesis using the 1918 influenza epidemic as a shock to maternal health.

4.4 Estimation

The three data sources are combined into two analytic files, one measuring mortality as 5-year mortality from age 65, the other measuring mortality from the reference date of January 1, 1980, through the last quarter of 1989. We use the natural log of the mortality rate as our dependent variable, and can use generalized least squared to regress the explanatory variables and covariates upon the logged mortality rate. For either dependent variable, our basic estimating equation is one of the following:

$$(1) Y_{ij} = \alpha + \beta_1 FLU_i + \beta_2 FLU_i * FluMort_j + \beta_3 P-Mort_j + Z_{ij} \gamma + YEAR_i \mu + \varepsilon_{ij}$$

$$(2) Y_{ij} = \alpha + \beta_1 FLU_i + \beta_2 FLU_i * FluMort_j + Z_{ij} \gamma + YEAR_i \mu + v_j + \varepsilon_{ij}$$

where ‘i’ indexes the quarterly birth cohort and ‘j’ indexes the state of birth. ‘FLU’ is a dummy variable for membership in the 1918:4–1919:3 cohorts. ‘FluMort_j’ and ‘P-Mort_j’ represent the 1918 influenza mortality and 1915–1917 mean all cause mortality respectively in the jth state. ‘Z’ represents an array of covariates—age, percent male, percent African-American and their interaction terms. ‘YEAR’ is an array of dummy variables for the calendar year of birth, so ‘μ’ is a vector of the 4-year effects (1915 is the reference year). ‘v_j’ represents a vector of state effects. Since we have only state-level variation in persistent mortality, we cannot include both persistent mortality and state fixed effects in the regression. Equation (1) omits state effects, the second equation omits the P-Mort variable.

The resulting parameter estimates for both FLU and FLU*FluMort represent the effect of being in a population exposed to the influenza epidemic, not the effect of actually having a mother contract influenza. If we take the consensus estimate of 25

percent for the national incidence of the influenza epidemic, and put the estimation in an instrumental variable context, the effect of actually receiving the ‘influenza treatment’ is:

$$\beta_{iv} = \beta_{rf}/\beta_{1st} = \beta_{rf}/.25 = 4*\beta_{rf}$$

If micro-level incidence data were available, we would regress membership in the cohort upon ‘contracted influenza,’ in the first stage. In the reduced form, we regress membership in the cohort upon later-life mortality. Unfortunately, the first stage is not feasible; we do not know who actually received *in utero* exposure. Since the dependent variable is logged, the parameter estimate represents the percentage change in the outcome for a one-unit change in the explanatory variable. A $\hat{\beta}_1 = 0.05$, for example, would imply that actually being exposed to influenza as a fetus raises later life mortality rates by 20 percent.

The two analytic files provide complementary ways of controlling for the age differences across cohorts. The fixed-age file measures mortality over the same ages—65 to 70—so, *ceteris paribus*, we expect mortality rates to be the same. However, there is a secular trend toward declining mortality and the age variable is a means to parameterize this change. This approach requires that we use a subset of the data we have; the 1919 cohorts do not turn 65 until 1984, the 1915 cohorts turn 70 in 1985. It also introduces a potential confounding variable, changes in health environment during the 1980s. Changes in health technology, for example the diffusion of bypass surgery, might decrease the later cohorts’ mortality, and since these include the influenza-exposed cohorts, it could potentially attenuate the relationship between exposure *in utero* and later mortality.

The second analytic file avoids these problems by using the full 10 years mortality observations. This means that each cohort is traversing a different segment of its mortality curve, so the expected mortality in each cohort should be different even before considering secular changes in mortality. The limitation of this approach is that mortality is a function of age and that to misspecify the functional form could bias our results. Fortunately, demographers have determined that a log-linear specification closely approximates end-of-life mortality when chronic diseases are the predominant cause of death. This principle dates back to the work of Benjamin Gompertz in the early 19th century, and is known as Gompertz' Law¹⁶ (Tropp 1972; Olshansky 1997). As both the necessary conditions apply to mature populations in late 20th century America, we have a natural choice for our specification. In the second set of regressions age controls for both the secular changes in mortality and the differences in cohorts' mean ages.

In all regression results we use only data from those states with mortality rates available for the 1915–1919 period. In figures we additionally present, when feasible, data on the full 48 contiguous states plus the District of Columbia.

5. Results

Figure 9 displays cohort mortality rates for the ten years from 1980 to 1989 for those born in the United States from 1915 to 1919. Both annual and quarterly

¹⁶ Gompertz published his work in 1825 and in slightly modified form it is still widely used in demography and gerontology. It states, "... the average exhaustion of a man's power to avoid death to be such that at the end of equal infinitely small intervals of time he lost equal portions of his remaining power to oppose destruction which he had at the commencement of these intervals." (Gompertz 1825; Tropp 1972)

cohorts are displayed. As expected, younger cohorts exhibit generally lower mortality rates, though there are exceptions in the quarter of birth cohorts. Among those *in utero* during the influenza epidemic, the 1919:1 cohort has higher mortality than 1918:4, but the rise is not large, and other cohorts not exposed to the epidemic *in utero* exhibit similar patterns, notably 1918:2 and 1918:1, and 1916:1 and 1915:4. In this simple time series analysis there is certainly no dramatic difference associated with the influenza-exposed cohorts as defined in the methods section (1918:4–1919:3), neither do we observe any other obvious breaks in the mortality pattern from cohort to cohort.

Figure 9
Mortality Rate by YOB (and qob)
Deaths 1/1980–12/1989

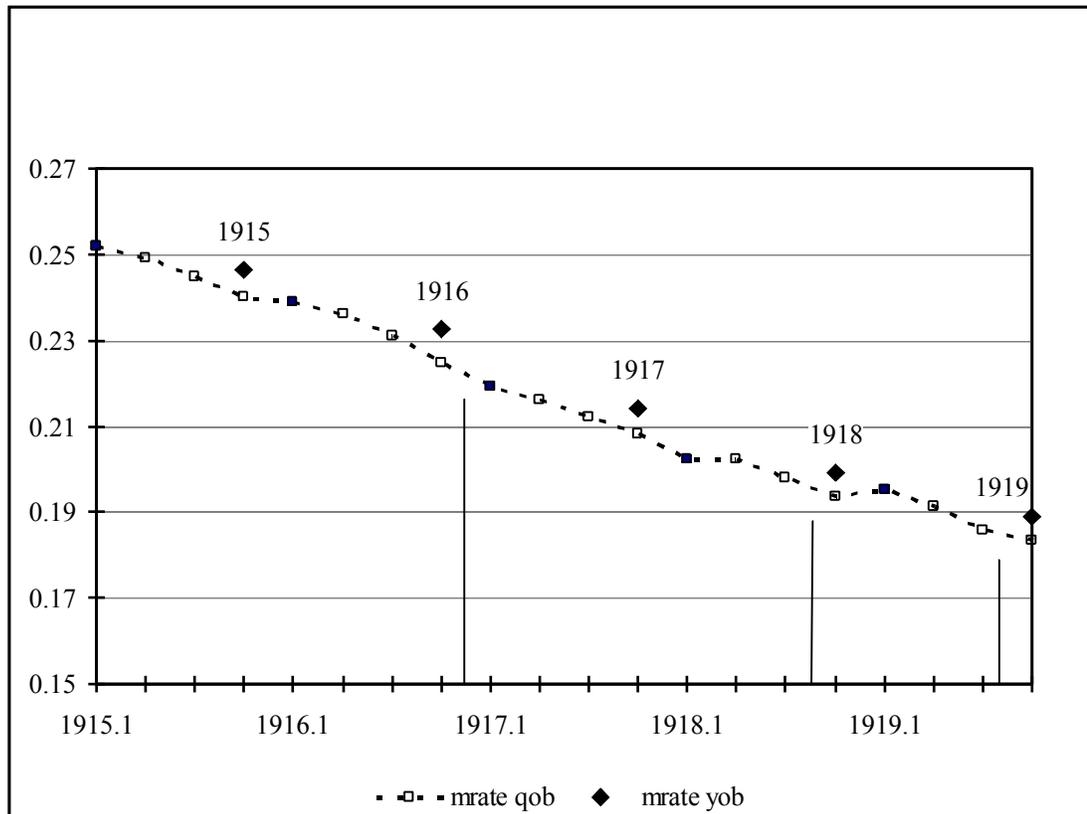
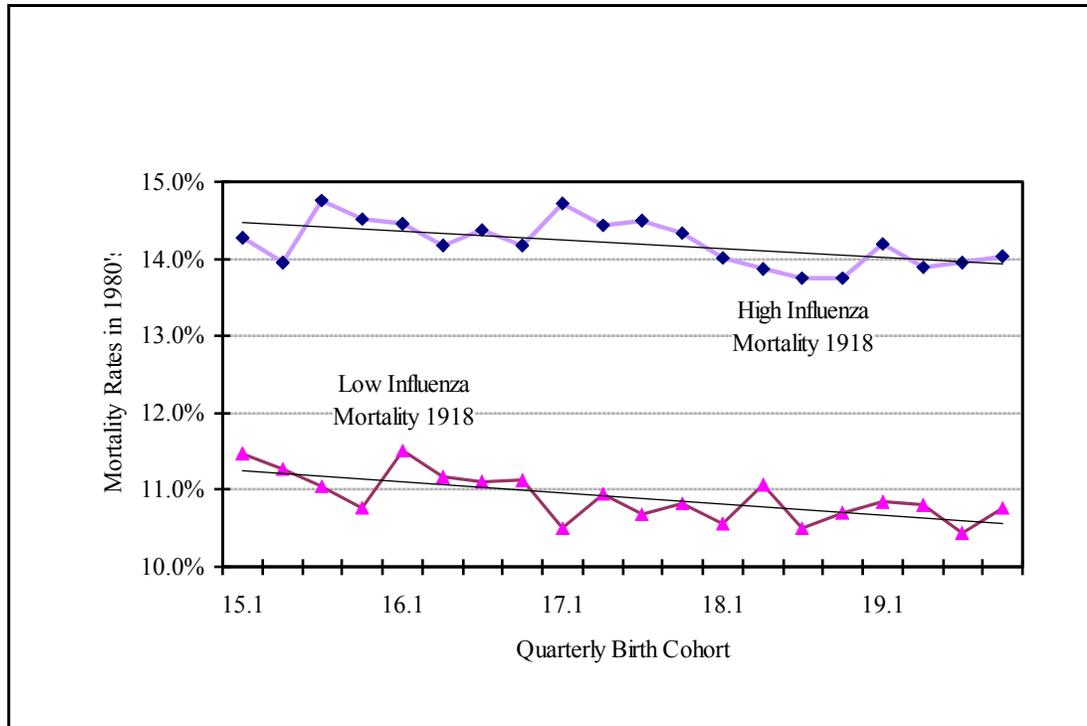


Figure 10 presents a subset of the data used to produce Figure 9 arranged so as to illustrate the problem in associating cross-sectional variation in influenza mortality rates with later-life mortality. In this instance, we eliminate the effect of age differentials between the cohorts by measuring 5-year mortality from a fixed age, in this case the quarter of the 65th birthday of cohort members. The six registration states with the highest 1918 influenza mortality and the six registration states with the lowest 1918 influenza mortality are displayed as two units of analysis. The jagged lines represent actual mortality experience; the associated straight line represents a least squares fit. While the influenza-exposed cohorts have unremarkable mortality experience within each group, the states with high influenza mortality have consistently higher later-life mortality than the low influenza mortality states. Thus, any cross-sectional regression is liable to confound the effects of the influenza epidemic with persistent differences in mortality rates.

Figure 10
 Five Year Mortality from Age 65—Six States with the Highest 1918 Influenza
 Mortality Rates vs. Six States with the Lowest Influenza Mortality Rates

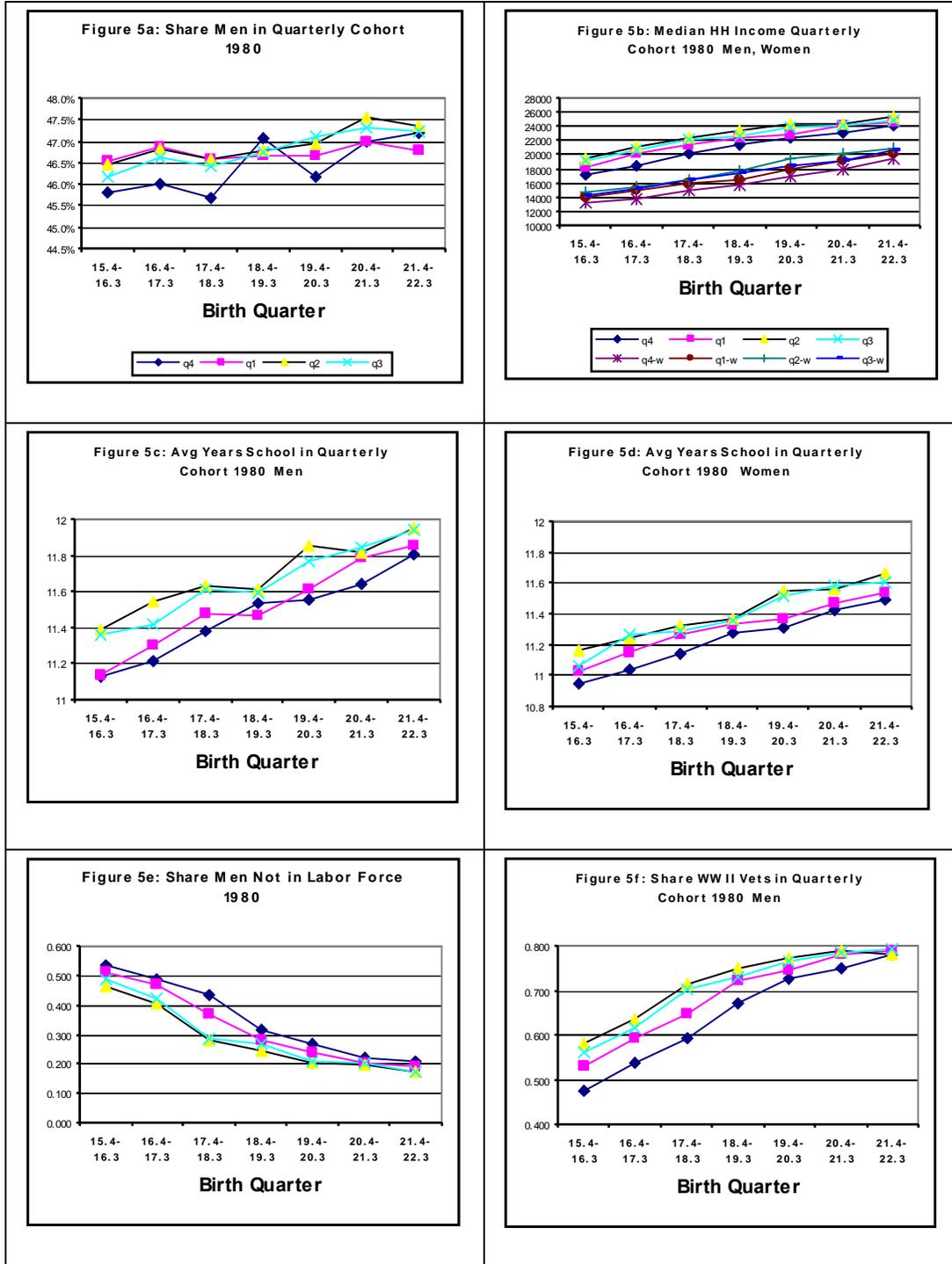


While we do not possess microdata associating influenza exposure and later-life mortality, we use the 1980 Census Public Use Micro-Sample to verify the extent to which the population receiving the influenza ‘treatment’ (1918:4–1919:3) appears similar to surrounding cohorts in demographic and economic characteristics. The six panels of Figure 11 compare the ‘treatment’ group to surrounding cohorts. Since there are clearly seasonal effects, to facilitate comparison each quarterly cohort (e.g., q1, q2, q3, q4) is graphed as a separate line. Each cohort is a different age on the census reference date, of course, thus any comparisons between them are necessarily approximate, especially as the 1915–1919 age group was approaching retirement age in 1980. While the influenza birth year is clearly a locus of some differences, these differences are not the sort likely to generate large differences in mortality of

themselves. Any differences may reflect underlying health differences, but in that case the argument that a treatment effect would be apparent in regression results is strengthened. The share of men in the 1918:4 cohort is a percentage point above trend, for example. Years of schooling is depressed for the 1919:1–1919:3 cohorts, with a larger effect for men than women. This agrees with Almond’s findings (Almond, 2003) but we do not see an associated reduction in male labor force participation, nor is there any notable pattern in the share of World War II-era veterans among the men. The reduced labor force participation and declining incomes as we go from youngest to oldest is compatible with standard retirement behavior (Leonesio, 1996). Years of schooling also declines from youngest to oldest, but this is a fairly small effect, .3 years mean level of schooling over five birth years, and comparable to standard accounts of the spread of high school education as a standard (Goldin, 2001). The share of World War II veterans declines fairly steadily from cohort to cohort; the likely explanation is that the younger cohorts were of prime draft age when the United States went to war in 1941; the older cohorts were more likely to have married, established families, and found work in war-related industries. While the ‘flu-baby’ cohorts seem to show some differences as compared to surrounding cohorts, the magnitude of the differences does not suggest a large mortality effect. A Barker effect of the hypothesized magnitude should be detectable.

Figure 11 Panels A - F

Cross Cohort Comparison of Socioeconomic Variables in 1980 IPUMS



We now turn to a regression analysis of the effects of the influenza epidemic on later-life mortality. We begin with Table 11, which presents multivariate regressions in the fixed-age format discussed above. The unit of analysis is a quarterly birth cohort for a specific state in the 1915–1919 registration area. The dependent variable is 5-year mortality from age 65, attainment of age 65 as defined above. Our explanatory variables are:

- a) Flu cohort: membership in a quarter-of-birth cohort potentially exposed to the 1918 influenza epidemic *in utero* (i.e. 1918:4 to 1919:3),
- b) Flu mortality: the interaction between flu cohort and state-specific 1918 influenza mortality rates, and
- c) Persistent mortality: the state-specific mean all cause annual mortality rate for 1915–1917.

Table 11
Effects of Influenza Epidemic on Later-Life Mortality: Fixed Age Regressions
Five Year Mortality from Age 65—Quarterly Birth Cohort by State of Birth

Dependent Variable: Ln (Mortality Rate), Mean = 4.6907 (10.96% mortality)

Independent Variables	Model 1	Model 2	Model 3	Model 4	Model 5
a) Flu Cohort (1918:4-1919:3)	0.0143 (0.0143)	-----	0.0579 (0.0350)	0.0545 (0.0304)	-----
b) a*1918 flu mortality	-----	0.0046 (0.0130)	-0.0434 (0.0318)	-0.0419 (0.0281)	0.0117 (0.0141)
c) Persistent Mortality	0.2645 (0.0275)	0.2638 (0.0276)	0.2702 (0.0278)	-----	-----
d) Age in 1980 (years)	0.0212 (0.0526)	0.0260 (0.0525)	0.0155 (0.0527)	0.0047 (0.0433)	0.0580 (0.0570)
e) % Male	1.7287 (6.8583)	2.3819 (6.8331)	0.9299 (6.8772)	-1.9391 (5.6423)	.6894 (7.4255)
f) % Afram	-12.6784 (16.6551)	-12.5421 (16.6700)	-12.7413 (16.6405)	-19.6063 (13.5062)	-3.8726 (18.1275)
g) d*e	-0.0355 (0.1095)	-0.0457 (0.1091)	-0.0229 (0.1098)	0.0288 (0.0902)	-0.1152 (0.1186)
h) d*f	0.2087 (0.2658)	0.2064 (0.2660)	0.2095 (0.2656)	0.3128 (0.2161)	0.0714 (0.2893)
i) e*f	37.9419 (37.9606)	37.6533 (37.9934)	38.4232 (37.9287)	52.7341 (30.8419)	18.8173 (41.3217)
j) d*e*f	-0.5947 (0.6065)	-0.5902 (0.6070)	-0.6022 (0.6059)	-0.8410 (0.4927)	-0.2930 (0.6602)
k) Intercept	-3.3061 (3.3008)	3.0043 (3.2900)	3.6640 (3.3083)	-----	1.2801 (3.5776)
Year effects (ref 1915)	Y	Y	Y	Y	Y
State effects	N	N	N	Y	N
N	500	500	500	500	500
R ²	0.4356	0.4346	0.4377	0.6566	0.3280
Adj R ²	0.4205	0.4195	0.4215	0.6291	0.3114

Since levels of influenza mortality are lower than all cause mortality the two series have been normalized by dividing by the mean across states. This facilitates comparison of parameter estimates associated with each variable. Covariates include age, percent male, percent African-American, their interactions and an array of year effects. State effects replace explanatory variable c) in one specification as discussed above.

Table 11 displays the results of five separate regressions using different combinations of the explanatory variables. Across all regressions neither a) (the cohort effect), nor b) (the state-specific influenza mortality rate) is ever significant, while c) is significant (p value < 0.0001) in the three specifications where it is included. In short, we do not find evidence of a Barker effect, whether we identify risk of 1918 influenza exposure by a cohort dummy (Model 1), by the influenza mortality rate (Model 2), or use both variables simultaneously (Model 3). In Model 3 the parameter estimates approach significance with a p -value of 0.0989 for flu cohort and 0.173 for flu mortality, however the signs of the estimates are different, and the flu mortality effect is negative. That is, conditional on being born between 1918:4 and 1919:3, being in a state with high influenza mortality *reduces* later-life mortality. In Model 4 we use state-level fixed effects, and consequently must exclude persistent mortality, as it would be co-linear with the state effects. The R-squared value is higher in this specification, but the estimates for the explanatory variables a) and b) change very little from the nearest equivalent, model 3. Persistent mortality is excluded from Model 5 to test whether the correlation between persistent mortality and influenza mortality is sufficient to produce a Barker effect through omitted

variable bias, the omitted variable being the general health status of the community. While the point estimate for flu mortality doubles in this misspecification compared to model 2, it still does not approach significance. An analogous misspecification using variable a) produced a similar absence of statistically significant results.

Table 12 repeats the analysis of Table 11, but in a setting where we use the log-linear relationship between mortality and age. This enables the use of the full ten years of mortality data, and means that cohorts are compared over the same time interval, but have different mean ages. In this specification the age variable controls for both this difference and the small secular change in the age/mortality effect between cohorts, while in the fixed-age specification age controls only for the latter. Because there is an additional source of variation in the dependent variable that is largely accounted for in the regression by age and its interactions, Table 12 has higher R-squared values than Table 11 for each regression model. While point estimates and standard errors change slightly, the results support the same conclusion: there is no evidence of a significant Barker effect, but there is strong evidence of a relationship between persistent mortality in an individual's state of birth and later-life mortality.

In Model 4 we do see statistically significant results for the both the flu cohort and the cohort* influenza mortality rate, but as in Table 11, Model 4, the coefficients have opposite signs and comparable magnitudes, evidence for the difficulty in measuring incidence, but not evidence for a Barker effect.

Table 12
Effects of Influenza Epidemic on Later-Life Mortality: Gompertz Regressions
Mortality 1980–1989 - Quarterly Birth Cohort by State of Birth

Dependent Variable: Ln (Mortality Rate), Mean = 5.3301 (20.88% Mortality)

Independent Variables	Model 1	Model 2	Model 3	Model 4	Model 5
a) Flu Cohort (1918:4-1919:3)	0.0103 (0.0128)	-----	0.0585 (0.0314)	0.0584* (0.0263)	-----
b) a*1918 flu mortality	-----	0.0006 (0.0116)	-0.0480 (0.0285)	-0.0497* (0.0243)	0.0075 (0.0129)
c) Persistent Mortality	0.2593 (.0247)	0.2591 (.0247)	0.2656 (.0249)	-----	-----
d) Age (years)	0.0868 (0.0472)	0.0911 (0.0471)	0.0805 (0.0473)	0.0618 (0.0373)	0.1225 (0.0520)
e) % Male	1.9579 (6.6476)	2.5878 (6.6217)	1.0050 (6.6593)	-1.8766 (5.259)	.1602 (7.3079)
f) % Afram	-9.6714 (16.1426)	-9.5222 (16.1529)	-9.7390 (16.1124)	-16.6658 (12.5904)	-.3428 (17.8395)
g) d*e	-0.0363 (0.0983)	-0.0455 (0.0979)	-0.0224 (0.0985)	0.0253 (0.0778)	-0.1137 (0.1081)
h) d*f	0.1451 (0.2386)	0.1429 (0.2387)	0.1460 (0.2381)	0.2433 (0.1865)	0.0103 (0.2637)
i) e*f	36.7201 (36.7954)	36.4549 (36.8184)	37.2936 (36.7279)	51.9085 (28.7487)	16.4923 (40.6683)
j) d*e*f	-0.5262 (0.5444)	-0.5224 (0.5447)	-0.5345 (0.5434)	-0.7597 (0.4253)	-0.2304 (0.6017)
k) Intercept	-0.5809 (3.1877)	-0.8730 (3.1989)	-0.1534 (3.2030)	-----	2.7238 (3.5204)
Year effects (ref 1915)	Y	Y	Y	Y	Y
State effects	N	N	N	Y	N
N	500	500	500	500	500
R ²	0.7404	0.7400	0.7419	0.8539	0.6813
Adj R ²	0.7334	0.7330	0.7343	0.8422	0.6734

1) * denotes p<.05.

2) Substituting variable (a) for (b) in Model 5 does not produce p<.05, changes R-squared by .0001, and no covariate changes more than 10%.

3) Year effects and state effects refer to year of birth and state of birth.

6. Discussion

No variant of either fixed-age or log-linear specifications provide support for the Barker hypothesis. Our measure of the general health of the community shows a strong positive association between the community's health indicators at the time of birth and later-life mortality. This is equivalent to what has been demonstrated previously, and suffers from the same limitations; poverty is a confounding variable. We cannot demonstrate a causal relationship because poverty is persistent. Areas with poor health indicators, in this case high mortality rates, tend to be socio-economically poor; poor individuals have higher mortality rates. Is fetal programming the important variable, or is it poverty? Our strategy to separate the two by using the shock of the 1918 influenza epidemic on maternal health has produced negative results. The simplest explanation is that poverty is the important variable. In the discussion which follows we explore alternative explanations for our results, and whether they are compatible with our data.

First, however, we want to interpret the coefficient on membership in the influenza cohort. While not statistically significant, is the point estimate demographically important? We take Table 11 Model 1 as our reference value. Since the dependent variable is the logged mortality rate, the point estimate of 0.0143 implies a rise in predicted mortality of 1.43%. Using a standard life table and assuming rates rise with age at a constant rate as Gompertz' law predicts, when we calculate the equivalent change in life expectancy we find that it is 0.35 years. To put this number in perspective, if we look at variation in life expectancy associated with the statistically significant coefficient on persistent mortality, a change in 0.35 years

is equivalent to going from a state at the 44th percentile for mortality to a state at the 56th percentile, considerably smaller than the difference between being born in New York compared to birth in Pennsylvania. If we construct a life table model

As we discussed in the Methods section, mortality rates may not be a proxy for exposure as much of the literature on the Barker hypothesis assumes, at least in the case of a health shock such as the influenza epidemic. The strongest negative result, therefore, is the absence of any significant cohort effect (explanatory variable a) in Model 1, Tables 11 and 12. While mortality rates may or may not be a cross-sectional measure of incidence, the timing of the epidemic is not in doubt. If *in utero* programming has an effect, it should be evident between October 1918 and September 1919, but it is not. We have considered the possibility that a year is too long a time period, that the effect may be more localized, and that the inclusion of inappropriate birth dates in the treatment population leads to attenuation of the effect for which we are testing. Table 13 shows the results of rerunning Model 1, Table 12 using single quarters of birth as our definition of the treatment effect. This is the smallest cohort which can be run using census-derived cohort sizes as no more specific birth information was recorded in the census. None of the estimated coefficients are statistically significant; the largest estimate is for those born in quarter 1919:1. Still, the p-value on this coefficient is .18, not close to conventional levels of statistical significance. Because we do not use influenza mortality rates in this specification, it is possible to use a larger data set, the full 50 states. By increasing the sample size we can reduce our standard errors, however, the results are less supportive of the Barker hypothesis, not more (not shown in table). The

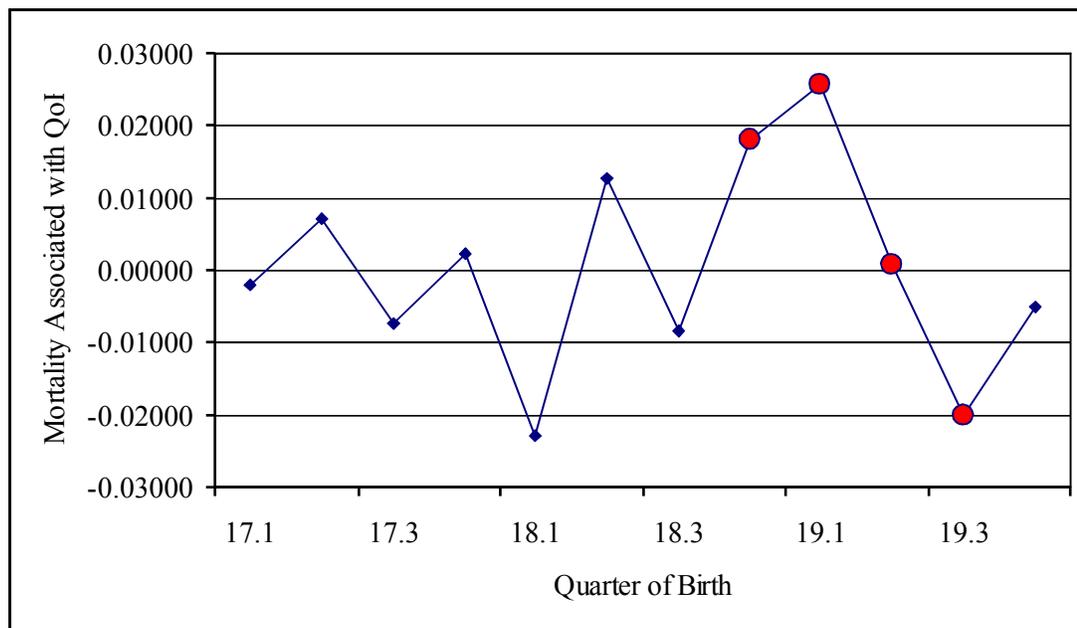
coefficient on the 1919:1 dummy variable is -0.0112 (se 0.0160), not significant and with a different sign from the 25 state sample. None of our alternate specifications helps to substantiate the Barker hypothesis.

Table 13
 Alternate *in Utero* Influenza Exposure Cohort Definition—Gompertz Regressions

Quarter of Birth	Model 1			Model 2		
	Variable a) Regression Coefficient	Standard Error	R ²	Variable b) Regression Coefficient	Standard Error	R ²
1918:4	0.0180	(0.0192)	0.7405	0.0108	(0.0182)	0.7402
1919:1	0.0258	(0.0192)	0.7410	0.0206	(0.0184)	0.7407
1919:2	0.0008	(0.0181)	0.7400	-0.0059	(0.0172)	0.7401
1919:3	0.0200	(0.0183)	0.7406	-0.0212	(0.0174)	0.7408
	From Table 12:					
1918:4– 1919:3	0.0103	(0.0128)	0.7404	0.0006	(0.0116)	0.7400

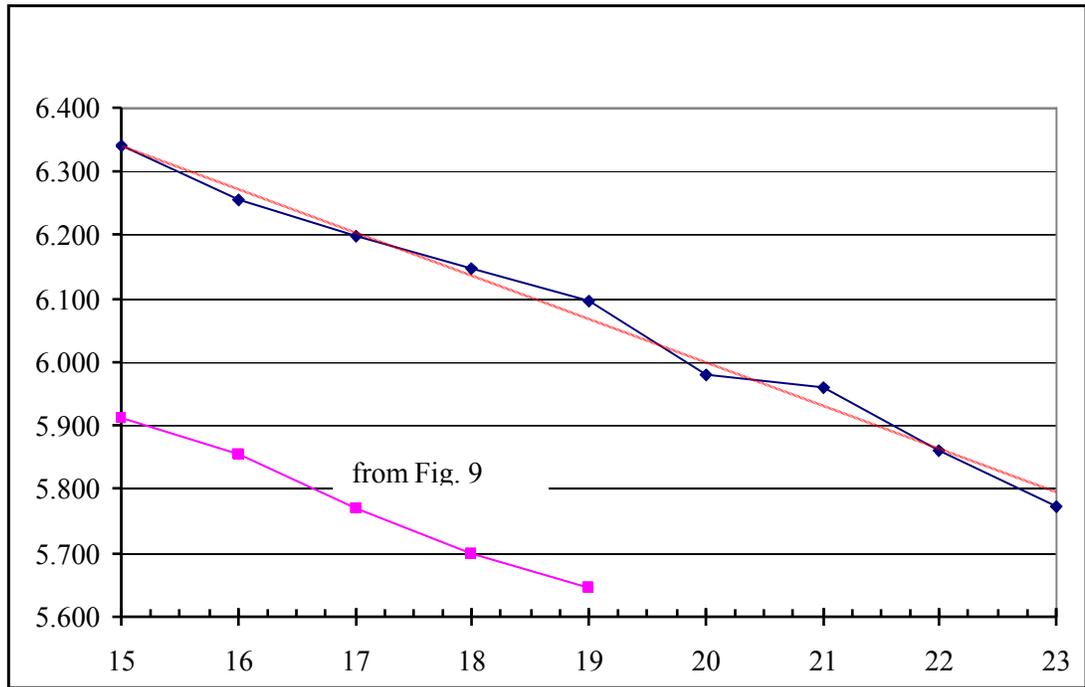
In a cohort analysis, one question is whether results are large compared to background variation due to unobserved causes. Figure 12 shows the coefficients associated with each particular quarter of birth, again using the Model 1, Table 12 specification, for each of the 12 quarters between 1917:1 and 1919:4. While the 1919:1 coefficient is still the highest value, we see that the coefficient on 1918:1 has almost the same magnitude, but the opposite sign. The conclusion of this analysis is that the background variation in mortality rates is large enough to account for the results for quarter 1919:1. There is no strong evidence for the singularity of any of the cohorts exposed to influenza *in utero* when mortality is the outcome of interest.

Figure 12
Regression Coefficient on Quarter of Birth



There is also no evidence of increased mortality when we examine mortality rates using one year cohorts from the 1970 and 2000 censuses (figure 13). The visible departure from linearity appears in year of birth 1920 (to be precise, 1920.2-1921.1). Because the mortality over the 30 year period is so high (35% – 55%) measurement problems which limit the usefulness of census survival figures over shorter periods become less important. Over this period in which half of those alive in 1970 died, there is no apparent spike due to the 1918 influenza epidemic.

Figure 13
Ln Mortality Rate by Year of Birth IPUMS 1970-2000



None of the models in Tables 11 and 12 includes any economic variables such as income, assets, occupation or migration. Because our unit of analysis is the quarter-of-birth cohort, income would necessarily be a state-wide mean, which would limit variation. The alternative would be to construct smaller state-of-birth, state-of-residence-1980 cells which would increase the number of observations, but at a substantial cost in clarity. Many cells would be very small or empty, for example, not many people were born in Vermont in 1917:4 and living in Wyoming in 1980. Instead we use an array of state dummy variables in Model 4. This absorbs all effects consistently associated with differences in state of birth. Note that results for Model 4 do not differ substantially from Models 1-3.

Alternative specifications confirm the basic results. The 1918 influenza epidemic does not generate a statistically significant effect on mortality using quarterly birth cohorts as the unit of analysis.

6.1 Influenza Mortality Rates

We have discussed in Methods why the use of mortality rates as a proxy for incidence is problematic. However, because this is the only national measure of the epidemic's intensity that exists, we have used the 1918 influenza mortality rates as our measure of cross-sectional variation in intensity of the epidemic. Other possible statistics were considered for the regression analysis and rejected. The mortality rates are drawn from published tables; the data which produced the tables are no longer available. There is no tabular breakdown of death rates by age, state and quarter of birth in the published tables. If we wish to use the published death rates, annual mortality by state and cause of death is the most specific measure available.

An alternative strategy would be to use death counts published in Mortality Statistics, which are available by month and age group, and compute death rates using the 1920 census as the denominator. Unfortunately, this census (reference date January 1) lacks reliable measures of cohort size finer than single year of age, so the only feasible alternative measure is calendar year age-specific influenza mortality. Figures 7 and 8 show that this is not a good definition of the influenza cohorts. Also, the procedure necessarily involves error, as the numerator and denominator are counted at different points in time. It is also likely that age specific mortality is a better measure of the mortality experienced by women of child-bearing age, but a

worse measure of incidence, as Frost (1920) observes. This alternative strategy also involves reducing the number of states included in the regression analysis from 25 to 20 as the specific death counts are found in the annual Mortality Statistics, rather than the 1919 summary.

There are at least two reasons to prefer the interaction of the 1918 influenza rate and the treatment dummy rather than each year's influenza mortality. First, the 1918 epidemic was not the same virus that caused deaths in other years. It was much more lethal, and appears to have had a more pronounced effect on healthy individuals than other strains of influenza¹⁷ (Reid and Taubenberger, 2003). Because mortality rates were so much higher in 1918, there is little reason to use influenza mortality from other years. The second reason is that the effect of the influenza spanned calendar years. Exposure and birth may take place in the same calendar year, or adjacent years. Use of the interacted 1918 rate allows greater flexibility in specifying the treatment group and does not diminish measurement accuracy.

The limitation of our approach is that it does not account for differences in the age distribution of the population across states. If differences in population age distribution contribute to the differences in mortality rates, it could be argued that our measure of influenza incidence is confounded, and this is the reason for our negative result. To test for this possibility we computed the share of the population under 2 years of age or over 65 years of age using the 1920 IPUMS sample. Given the 'W'-shaped mortality pattern of the epidemic, these are the other two population groups

¹⁷ That is, it was an influenza virus which had become more virulent due to genetic shift, genetic drift, or some combination.

aside from young adults that contribute disproportionately to mortality rates. We wish to develop a proxy for the influenza incidence among young adults. The population share of the other two groups might plausibly confound this measure. Table 14 shows that while population share in the infant/old age group is positively correlated at 0.48, the correlation between infant/old share and persistent mortality is substantially higher at 0.66. If the negative result were caused by confounding differences in age distribution, it would affect persistent mortality more than influenza mortality, but persistent mortality shows a strong positive effect.

Table 14
States Reporting Mortality Data 1915 – 1919
Influenza Mortality Rates per 100,000 and
Population Share Under 2 or over 65 Years

State of Birth	1915–1917	1918	Population Share <2 years or >65 years
	Persistent Mortality	Influenza Mortality	
California	1321	310	0.082
Colorado	1173	456	0.084
Connecticut	1538	424	0.113
Indiana	1327	198	0.096
Kansas	1130	252	0.047
Kentucky	1292	328	0.095
Maine	1574	335	0.144
Maryland	1610	362	0.099
Massachusetts	1506	342	0.103
Michigan	1324	191	0.083
Minnesota	1040	250	0.061
Missouri	1293	212	0.070
Montana	1195	522	0.120
New Hampshire	1649	457	0.135
New Jersey	1452	303	0.096
New York	1520	225	0.104
North Carolina	1461	325	0.095
Ohio	1341	259	0.119
Pennsylvania	1471	465	0.102
Rhode Island	1602	368	0.107
Utah	1053	327	0.068
Vermont	1554	396	0.143
Virginia	1420	420	0.099
Washington	935	241	0.099
Wisconsin	1127	245	0.067
Correlation with Share of Young and Old	0.6651	0.487	

While plausible, the confounding problem is by no means certain. Again, it is incidence that matters, not mortality. The old and the young may pass their influenza to caregivers, frequently women of child-bearing age. Excluding their experience

may lead to a better measurement for case fatality but a worse measure for incidence. There is no theoretical or empirical basis for preferring age-specific mortality

Table 15 further examines the issue of correlations in mortality rates relevant to the 1918 experience. All mortality rates are positively correlated, but the correlation between 1918 influenza mortality and the 1915–1917 average mortality is the lowest shown, 0.344. If we wish to compare the effects of different sources of mortality, co-linearity should not prevent our doing so. Table 15 also offers further confirmation that there is no evidence of differential measurement of the influenza epidemic across states. The potential alternative cause of death would be pneumonia (Jordan, 1927; Niall, 2002). The pneumonia vs. persistent mortality correlation is quite high, 0.597, while the pneumonia vs. flu correlation is 0.435, positive, but somewhat lower. States with poor health status report high pneumonia and influenza mortality, but the relation between influenza and pneumonia does not appear particularly close. Finally, note that persistent mortality is closely related to all cause mortality, non-influenza mortality in 1918, and infant mortality in 1918. Persistent mortality is a good measure of the 1918 experience, but avoids the measurement problems inherent in using two coincident mortality measures when there can be substantial co-morbidities between them. As proxies for influenza incidence and health status, the two measures chosen are the best available.

Table 15
Correlations of Different Measures of Mortality:
All States Reporting Mortality 1915-1919

	Infant	Pre-Flu	All 1918	All NFlu	Flu 1918	Pneu. 1918	Int. Coeff.
States Reporting	20	25	30	30	30	30	25
Infant							
Pre-Flu	.850						
All 1918	.916	.857					
All non-Flu 1918	.942	.895	.964				
Flu 1918	.649	.344	.655	.431			
Pneumonia 1918	.839	.597	.820	.826	.435		
Interaction Coefficients	.733	.541	.802	.682	.726	.714	

Notes:

- 1) Infant Mortality Rates are taken from Census Report "Birth Statistics 1918."
- 2) All other Mortality Statistics are taken from, or based upon, Census Report "Mortality Statistics 1919." In that year statistics are restated for the years 1915-1918, with additional states added to the statistics. All rates are crude mortality rates for all ages.
- 3) Pre-flu is the average mortality rate from all causes for the years 1915-1917.
- 4) Interaction Coefficients are the result of regressions run on the 25 states reporting data for all years 1915-1919 with year effects, state effects and the interaction of the year 1918 and the state effects. The coefficients for these interaction terms are used in the correlations.
- 5) Non-flu deaths + Flu deaths = All deaths. So correlations are algebraically related.
- 6) All correlations use the smaller of the relevant "States Reporting" counts. Each larger group is a superset of the smaller group. The selection problems between the different groups of states do not appear large. Correlation for 1918 All and 1918 Flu for the 20 states reporting infant mortality is .731.
- 7) Correlation for infant mortality rates across all years is very high. For example, 1918 and 1917 infant mortality have correlation of .972.

6.2 Comparison with Other Results

The present paper builds on the cohort analysis used by Douglas Almond (2003), but Almond reports only graphical results on mortality, and they, unlike his regression analysis, are based upon data from the National Longitudinal Mortality Study, where year of birth is intentionally blanked. Almond carefully analyses the education, income and disability data from the Public Use Micro-Samples of the 1960, 1970 and 1980 and finds generally worse outcomes for the influenza ‘treatment’ cohort. The differences he finds, though statistically significant, are not nearly large enough to cause measurable mortality effects. For example, Almond finds total personal income in 1980 reduced by 2.5 percent for the cohort, and a commensurate decline in educational attainment. While a striking finding, the effect of the lower income would lead to overestimates of any Barker effect in our regressions. As we find no effect, Almond’s results combined with our own tend to confirm the absence of a Barker effect.

As regards interpretation, Almond follows Barker in tending to ascribe fetal origins to observed differences. He carefully considers whether selective attrition is producing his results, but gives less attention to possible changes in family structure resulting from the creation of widows and widowers by the influenza epidemic. To reconcile Almond’s interpretation with our own, we would need to believe that the influenza epidemic had long lasting health effects sufficient to reduce high school graduation rates, for example, but not later-life mortality effects. Moreover, the consistency of Almond’s findings over the twenty years between 1960 and 1980 suggest that differential mortality before 1980 cannot have been large. In short,

Almond's results suggest that whatever produced the poor outcomes of his 1919 cohort, it was not something that produces large mortality effects.

Neither this paper nor Almond's has good measures of the health of neonates and infants who survived the influenza epidemic. One possible explanation for our failure to find a measurable Barker effect is the presence of an offsetting beneficial shock. For example, if the influenza epidemic led to deaths among weaker infants and fetuses, then the average health of the surviving cohort might have improved thereby offsetting the negative shock of poor *in utero* nutrition. There is no evidence for this type of censoring, and Almond's labor market findings (Almond 2003) point towards poorer health from childhood on, not better health. Nevertheless, in the absence of good data on the health of the cohort in infancy, we cannot definitively rule out some compositional change masking the Barker effect.

6.3 An Upper Bound for the Barker Effect

If we imagine Barker's hypothesis as a two-dimensional graph associating the magnitude of a fetal shock with an increase in later-life mortality, we have shown that the shock associated with the influenza epidemic cannot produce a large later-life effect. More intense shocks, such as the Dutch famine winter, might produce a measurable effect (Roseboom, 2000, 2001).

Even the influenza shock, if we had a sufficiently large longitudinal data set, might produce a measurable effect. How large a non-zero effect could the preceding cohort analysis have missed? The six standard errors associated with a regression in which there is a single explanatory variable are Models 1, 2 and 5 in Tables 11 and

12. The largest of these, the standard error on the cohort specification of Model 1 in Table 11, is 0.0143. The smallest is the standard error on Model 2 in Table 12, 0.0116. If we again take the incidence of influenza as 25% and the critical t-value as 2, a Barker effect of 9–11% could go undetected, depending on which specification best captures the pattern of *in utero* exposure.

The calculation on the cohort variable is straightforward; it is either zero or one. In the calculation which uses mortality rates (Model 4, Table 13) we have evaluated the effect at the mean treatment effect, 0.998. (The variable was parameterized to a mean which approximated one to simplify such calculations.) This assumes that the entire cohort is subject to the Barker effect if the mother contracts influenza at any point in her pregnancy. If we define a short enough critical period for exposure, we can, of course, increase the size of a potential effect which goes undetected; this is a limitation of our methods. If, for example, the critical period for development of the circulatory system is a gestational age of exactly week X, then the effective level of incidence may be less than 1%, so our treatment group has a very low percentage actually receiving the treatment. This line of argument defends the Barker hypothesis by limiting its importance. That is, a Barker effect associated with a severe famine which persisted for several months shows there is an effect at some point in pregnancy if the shock is severe enough, but does not tell us when the critical period occurs, or what the threshold is. For the developed world prolonged famine is a rare event, smaller shocks are more common. The results in this paper show that even a relatively dramatic shock has few later-life physiological consequences.

To test definitively for a Barker effect using the influenza epidemic as a shock, a large longitudinal individual-level data set with precisely observed date of conception and detailed information on maternal health history is the ideal. Such a data set is unlikely to appear for U.S. data at least. Failing that, our results strongly suggest that the use of mortality measures is unnecessary. The influenza epidemic is sufficiently localized in time that a cohort definition is sufficient if date of birth is well-observed. The NMDF do have a precisely observed month of birth, the problem is the absence of a denominator. But it is possible to make some assumptions about monthly cohort sizes and test to see if *any* reasonable assumptions for monthly cohort sizes can produce evidence for a Barker Effect. This is a possible direction for future work.

7. Conclusion

This chapter has subjected the Barker hypothesis to an ordeal; it has not emerged victorious. We find no evidence in support of a large Barker effect. Because we use an exogenous shock to health to identify the effect, our results cast doubt on those who claim a large effect from observations where the conditions of birth are a function of persistent health patterns. The possibility remains that a larger or more long-lived shock might produce a measurable effect. Our findings do not refute the Barker hypothesis, but do limit its scope. Given the severity of the 1918 epidemic, it appears unlikely that small insults to health produce irreversible changes in life expectancies. This chapter began by considering the implications of a strong Barker effect for economists. This appears to be one problem the discipline can safely ignore.

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