PAA Extended Abstract

Testing the Influenza-Tuberculosis Selective Mortality Hypothesis with Union Army Data[*,†]

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150-Word Abstract

In 2000, I postulated that there was selective mortality in the great 1918 influenza pandemic (Noymer and Garenne, 2000). This hypothesis — that tuberculosis (TB) enhances risk to death from influenza — is potentially important for mortality studies generally, and may add new information to the frailty modeling approaches that have been predominant in mortality studies since the 1979 watershed methodological papers by Keyfitz and Littman, and by Vaupel et al.

Elaboration with historical microdata is a next step. Fogel's Union Army data, which by now are well-known to historical demographers, fit the bill. I use UA data to examine, using Cox regressions (etc), if lifetime TB exposure influences death due to influenza. The hypothesis predicts: Yes. The data agree. The risk ratio is 2.0 and is statistically significant. [[see regression table, p. 14 of this Extended Abstract]]

This informs current debates in demographic methods, and tells an interesting story about life and death in the UA cohorts.

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[†]This paper uses data from the Early Indicators of Later Work Levels, Disease and Death study ("Union Army" data), Robert W. Fogel, principal investigator; supported by NIH/NIA grant P01 AG10120. I would like to thank Dora Costa for advice on the data.

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PAA Extended Abstract

1 Background

1.1 The 1918 influenza pandemic

The 1918 influenza pandemic[1], sometimes called the 'Spanish flu', was the most deadly outbreak of any disease in the twentieth century. Estimates of global mortality from the pandemic are 40–100 million (Johnson and Mueller, 2002). There were about 34 million combat deaths in all twentieth century wars combined (Brzezinski, 1993, p. 9), so it is difficult to understate the quantitative magnitude of the 1918 flu.

The 1918 flu was also qualitatively different. The age-mortality profile of influenza deaths (a plot with age on the horizontal axis and death rates on the vertical axis) is normally U-shaped. Children and the elderly have the weakest immunity, and the U-profile reflects that. Adults, who have the greatest resistance, form the base of the U. By contrast, in 1918 the age-mortality profile was W-shaped. Typical mortality among the youngest and oldest was accompanied by a third peak, among young adults, which is unprecedented for influenza as well as puzzling theoretically.

1.1.1 Influenza age-mortality profiles

Figure 1 (p. 4) shows the age profile of death rates for influenza and pneumonia (combined) for the United States. Since fatal cases of influenza involve pneumonia, it is customary for statistical bureaux to merge influenza and pneumonia in published vital statistics. Four panels are shown, representing, from top to bottom, the years 1900, 1918, 1939, and 1998; male rates are solid and female rates are dashed; rates are per 100,000 population. The patterns in the figure illustrate notable aspects of influenza demography and yield insight into mortality

[1] A pandemic is a global epidemic of the same strain of influenza virus (Kilbourne, 1987, p. 14); herein "1918 epidemic" and "1918 pandemic" are used interchangeably.

patterns more generally. To permit comparisons, all four panels are drawn with identical scale, with a horizontal rule across each panel at a mortality level of 100 per 100,000.

The influenza mortality rates exemplify three major mortality age patterns, named after letters of the alphabet: U, W, J. In 1900, the pattern is U-shaped (sometimes called V-shaped), with peak mortality at the upper and lower bounds of the age distribution. Though influenza occurs at all ages, mortality is concentrated among the youngest and oldest. A similar pattern is seen in 1939, except that the base of the U (but not the tops) has descended to a lower level and remains below the line (100 per 100,000) until a much later age. The 1939 panel represents the end of the pre-antibiotic era. Flu, being a viral disease, is not treatable with antibiotics, but secondary pneumonias often involve or are exacerbated by bacterial coinfection, which can be treated with antibiotics.

On the other hand, the pattern in 1918 is completely atypical, even for a pandemic. Due to that year's epidemic of hypervirulent influenza, the pattern is W-shaped, with the aforementioned mode at middle age in addition to modes at either extreme of the age distribution. Such a pattern is unusual among biological causes of death, with tuberculosis being the closest match among the major diseases. This is seen in figure 2 (p. 5), which illustrates death rates for tuberculosis[2] (TB) for the United States in the relevant time period (1917).

The entire influenza mortality curve in 1918 (figure 1, p. 4) lies above the 100 per 100,000 line, reflecting the severity of the epidemic. The male excess death rate, in absolute terms, was also exaggerated in 1918, particularly at the middle-age mode of the W. The leading explanation for the decline in death rates above age 35, which gives rise to the W shape, is that at least one flu strain circulating in the late nineteenth century was similar to the 1918 strain, imparting partial

[2]Figure 2 shows all forms of tuberculosis. By far the most deadly form is pulmonary TB, and a graph of death rates from pulmonary TB would not look markedly different except that the mode among infants would be smaller, since other forms of TB are more important at the youngest ages.

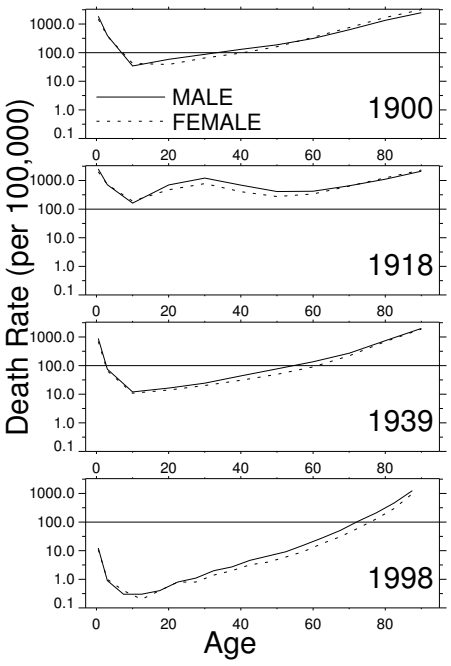

Figure 1: Age-mortality profiles, influenza and pneumonia, United States: 1900, 1918, 1939, 1998

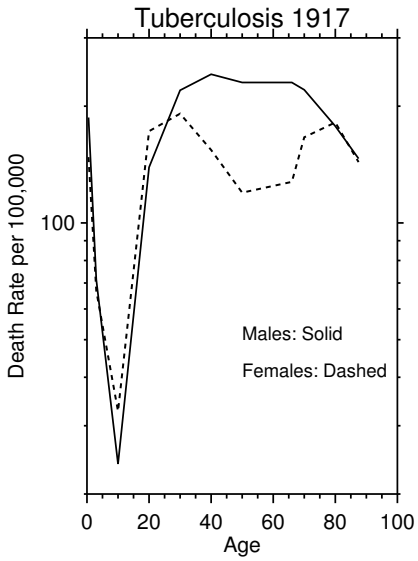

Figure 2: Age-mortality profile, tuberculosis (TB) (all forms), United States, 1917

immunity to those who, by 1918, were above age 35; in other words, a cohort explanation. The W-shape is considered more thoroughly in another paper of my dissertation.

In the last half century, influenza death rates in developed countries have declined more at young ages than among the elderly, transforming the U shape into a J shape, as seen in the 1998 data.

1.2 The selection hypothesis in plain English

Selection theories in demography are often highly mathematical, but selection in the 1918 flu can be summarized as: who died, who survived, and did this change the ante- *vs.* post-epidemic population composition?

The selection hypothesis centers on the W-shaped age-mortality profile: it posits that young adults who died of the 1918 influenza — the middle of the W — were the unhealthiest members of society. The surviving population, in

1919 and afterward, was therefore that much healthier on average. Tuberculosis is the nexus with "unhealthy" because the lungs are attacked by both diseases. Since many influenza deaths were among tubercular people, the post-epidemic population was healthier. The hypothesis is corroborated by a variety of data, including plummeting TB death rates in 1919 and thereafter. It is no coincidence that TB was, in that era, typically a disease of adults rather than of children or the elderly (cf. figure 2), and it was the most important cause of death among adults.

1.3 Relevance to frailty and heterogeneity

The concept of selection, though not always referred to by name, is of cardinal importance to virtually all current lines of research in mortality, longevity and long-term health. Frailty models postulate a distribution of frailty such that mortality selection of the frail causes cohorts to become more robust as they get older. This has an important bearing on longevity, the flip-side of mortality. In theory, older, less frail, cohorts fare better in the face of a baseline mortality risk than would be expected were the frailty not taken into account; this is sometimes called "cohort inversion" (Hobcraft et al., 1982). This line of inquiry began twenty-five years ago (Keyfitz and Littman, 1979; Vaupel et al., 1979) and has continued in a large technical literature (e.g., Hougaard, 1984). An analogous and similarly technical literature on labor markets began around the same time (Lancaster, 1979).

There is a black-box aspect to this state of affairs, because while death rates are observed, there are two free parameters in the theory — the baseline mortality rate and the frailty distribution. These two free parameters combine to produce one observed phenomenon, death rates. The observed death rates identify a unique frailty distribution assuming a baseline mortality rate, or the observed

death rates identify a unique baseline mortality rate assuming a frailty distribution, but one cannot simultaneously identify both the frailty distribution and the baseline mortality from observational data. Put another way, the observed death rates determine the baseline mortality against a counterfactual frailty distribution, or vice versa (Noymer, 2001). Recognizing this, research has moved in the direction of trying to open the black-box, through genetics (as in Weiss, 1990, or Yashin and Iachine, 1997), kinship analysis (e.g., Kerber et al. 2001; Smith et al. 2002; Mineau et al. 2002), analysis of biological (viz., laboratory) populations (cf. for example, Carey, 2003), and the study of early life influences (such as: Bengtsson and Lindström 2000; Costa 2000; Almond and Mazumder 2005). By bringing in more information *a priori*, the challenge of understanding two phenomena (baseline mortality and frailty) from one (observed death rates) becomes easier.

The selection hypothesis paper (Noymer and Garenne, 2000) used the 1918 influenza pandemic as a natural experiment to show how exposure to a disease at a certain point in time can affect mortality from another cause at a later point in time. This is another way to open the black-box, and is, in effect, a way of looking at early life conditions, albeit loosening the restriction that the early conditions take place *in utero* or during development.

To reiterate, a thumbnail sketch of the previous results is that although excess mortality in 1918 was attributed to influenza, tuberculosis death rates plummeted in the following years, indicating that the tuberculous population was diminished by the epidemic. One condition, having tuberculosis (including latent cases), affected the chances of dying from an unrelated condition (infection with the 1918 strain of influenza), which in turn diminished death rates from tuberculosis for the affected cohorts, relative to what would have been expected had the 1918 epidemic not occurred. The results hold up when dis-

aggregated by age and sex. This is an example of cohort inversion because the influenza epidemic had the perverse effect of reducing cohort mortality in the post-epidemic period.

1.4 Why the selection hypothesis is important

Firstly, the 1918 epidemic killed more people than any other epidemic of the twentieth century, and as such it is worth understanding as much as possible about it, as a matter of demographic, epidemiologic and social history. The W-shaped mortality profile in particular continues to be a medical mystery, and this work can help to address it.

Secondly, this work will help adjudicate a current debate in demography about selective mortality. This debate may be summarized as a school of thought that mortality is selective in general *vs.* a school of thought that mortality is fairly random. One group believes that longevity tends to increase because deaths at younger ages leave behind a more robust population. The other group cautions against taking ever-increasing life expectancy for granted. Both arguments are underpinned by counterfactuals that cannot be tested directly. Examining cause-specific mortality selection helps shed some empirical light on these questions.

Thirdly, influenza pandemic mortality is of current public health interest, especially because of bird-to-human transmission of H5N1 influenza virus (a new strain) in southeast Asia (Specter, 2005). Pandemics are caused by new strains, so there is concern about the recent events. Tuberculosis is still highly prevalent in the developing world, indicating that the impact of a new pandemic may vary greatly internationally.

1.5 The 1918 epidemic is under-studied

Despite the severity of the 1918 flu and the peculiar age-mortality profile, demographers have paid relatively little attention to it. Part of the reason is that the 1918 epidemic was short-lived. Although it shortened US life expectancy by 12 years in 1918, mortality decline continued apace in 1919 as if nothing had happened. Until recently the 1918 influenza has not fit well into the story of long-term expansion of life expectancy. Ironically, the selection hypothesis postulates that the 1918 flu actually hastened the decline in mortality in the years following 1918.

As the title suggests, a major theme of Crosby's landmark *America's forgotten pandemic: The influenza of 1918* (1989) is that the 1918 pandemic has been ignored not only in technical fields such as demography, but also among historians. The same holds in other countries (e.g., Rice and Palmer, 1993). Duffy (1977) does not find this unusual, however, noting "historians have generally paid little attention to epidemics other than the Black Death and the Great Plague of London", referring to events in the fourteenth century and 1665, respectively.

Except for brief mentions, the 1918 epidemic does not figure in the almost 800-page account of twentieth century European population produced by Bardet and Dupâquier (1999), and the same is true for a recent volume on the demographic history of North America (Haines and Steckel, 2000).

Trostle (1986, p. 60) notes the role of the pandemic in the development of modern notions of host-environment interactions in disease processes. But for the most part, even when the population literature includes an awareness of the 1918 epidemic, it treats the event as a one-off curio. For example the Lee-Carter mortality model uses a dummy variable to cleanse the time series of the distorting effects of the pandemic (Lee and Carter 1992; Lee 1992, 2000).

More recently, having realized the 1918 flu is under-studied, scholars have begun to devote more attention to this topic. Lead by techniques unavailable until recently, paleovirologists have practically made the 1918 pandemic a cottage industry within their field. There has been a similar if smaller change in the social science literature, for example: Azambuja and Duncan (2002); Azambuja (2004); Langford (2002, 2005); Smallman-Raynor et al. (2002); Brainerd and Siegler (2003); Tognotti (2003); Mamelund (2003, 2004); Reid (2005); Almond (2005). Epidemiologists also show a renewed interest in the pandemic (for example Mills et al., 2004; Olson et al., 2005).

1.6 Conclusion

This work touches on several important areas.

The tuberculosis/influenza selection hypothesis merits reproduction and elaboration, and historical demography is the right approach for this. By using microdata, a more specific putative causal story can be told. By looking at a period prior to 1918, as well as another country, the generality of the selection hypothesis can be assessed.

This work is one way to open the black-box in the frailty/longevity literature, and as such it can potentially lead to a better understanding of how causes of death interact.

Future influenza pandemics are of much interest in public health, and demographers should not be left out of the debate. Much of the world still suffers from tuberculosis, so the conditions of the USA in 1918 may be approximated in other countries today, bringing currency to this historical analysis.

2 Union Army Paper

2.1 Elaborating the selection hypothesis with Union Army data

The data used to substantiate the selection hypothesis were mostly aggregate data, death rates for age- and sex-groups taken as a whole. Because the selection hypothesis concerns individuals sick with tuberculosis dying of influenza, it is desirable also to analyze individual-level data.

Individual-level historical data sets rich in detail on mortality as well as pre-death illnesses are difficult to come by. However, one example is the Union Army data set, collected by the University of Chicago and made available to the scholarly community (discussed further in § 2.2). Although most of the veterans died before 1918, these data provide a unique opportunity to investigate connections between TB and influenza in historical context. If the selection hypothesis in its strongest interpretation is correct, it should apply even in years other than 1918. These data will be analyzed using statistical tools for event history analysis, to determine if underlying TB presents a risk factor for death due to influenza.

This paper will explore and test the selection hypothesis in two ways: First, microdata (the Union Army Dataset) will be analyzed whereas the original work predominantly dealt with data at various levels of aggregation. Second, the period analyzed will encompass all of the late nineteenth and early twentieth century morbidity and mortality experiences of the Union Army cohorts, illuminating whether or not the selection hypothesis holds in non-pandemic years.

Although the 1918 flu pandemic, due to its unusual severity, has certain advantages for use as a natural experiment, there is nothing about the tuberculosis/influenza selection hypothesis *per se* that requires a hypervirulent influenza

pandemic. If the selection hypothesis in its strongest form is correct, having tuberculosis should predispose one to death from influenza in years other than the pandemic.

2.2 Data

The data set is the "Union Army" data, collected at the University of Chicago's Center for Population Economics under the direction of Robert W. Fogel. The data are described on the Internet (Chicago Center for Population Economics, n.d.) and by Fogel (1993). The sample "consists of 35,747 white males mustered into the Union Army during the Civil War, for whom military, socio-economic, and medical information from several sources throughout their lifetimes has been collected" (Chicago Center for Population Economics, n.d.). The data are representative of the military age white male population of the Union states in the early 1860s. Of the 35,747 records, 35,570 are made available to the public, with certain modest restrictions, via a web-based data extraction system. Of these, 3,609 (10.1%) are specifically classified as "wartime" deaths in a data subset, though these are not necessarily combat fatalities. Overall, 4,984 records (14.0%) list a death on or before 26 May 1865 — the date of surrender of the last significant Confederate army — which forms the most encompassing definition of wartime deaths. Wartime survivors are followed through the nineteenth and early twentieth century, until death in most cases. Pension information, visits to the doctor's office, and eventual cause of death are recorded for most records.

The 35,570 records are not equally complete. When date of birth information is known, the sample size is 12,977. Since all men in the data are of military age when they were mustered into the army, it is possible to impute an approximate age when birthdate is missing, but this is clearly not ideal and has not been attempted here, so far. When date of death information is known, the sample size

is 24,912. When both date of death and cause of death are known, the sample size is 15,833. When date of birth and death and cause of death are all known, the sample size is 6,900. It is unfortunate, although not at all unexpected, that the usable sample size for this study is smaller than the total of 35,570 records. Knowing cause of death is a difficult standard to meet in many cases, and such record loss is par for the course in historical work. Having thousands of nineteenth century records with cause of death information — and therefore well over 100,000 person-years of exposure — is a unique resource in American demographic data.

The Union Army dataset provides an unequaled opportunity for historical demographic research on the USA in the late nineteenth century. Using data from this time period is ideal. After the 1918 epidemic, as the prior work showed, tuberculosis death rates fell. Though tuberculosis death rates in the 1920s and 1930s were large by today's standards, the wake of the 1918 epidemic seems an inauspicious place to look for the selection phenomenon repeating itself. In the 1940s, there is the Second World War followed by the introduction of antibiotics against tuberculosis, both of which complicate matters. In the nineteenth century, tuberculosis prevalence and mortality were both high, and influenza ever-present. In addition, as noted above, using pre-1918 data will allow us to test whether the selection hypothesis only works in the *sui generis* context of the 1918 flu, which was unusually severe by orders of magnitude.

An alternate possibility as a way to test the tuberculosis/influenza selection hypothesis would be to collect contemporary data from developing countries, where tuberculosis prevalence remains high. Even so, the longitudinal nature and large sample size of the Union Army data set make it more appealing than any data that could be collected easily in developing countries. Existing surveys, including the Demographic and Health Surveys (DHS), do not contain as much

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Cox regression -- Breslow method for ties

No. of subjects =         6892                     Number of obs   =      6892
No. of failures =          620
Time at risk    =  317107.2361
                                                   LR chi2(1)      =     44.16
Log likelihood  =   -4674.4219                     Prob > chi2     =    0.0000

------------------------------------------------------------------------------
          _t | Haz. Ratio   Std. Err.      z    P>|z|     [95% Conf. Interval]
-------------+----------------------------------------------------------------
  alltime_TB |   2.066719   .2095822     7.16   0.000     1.694192    2.521159
------------------------------------------------------------------------------
```

Table 1: Preliminary event history analysis results

cause-specific mortality microdata as the Union Army dataset, and certainly lack the longitudinal aspect of the Union Army dataset. It is also worth bearing in mind that HIV complicates matters considerably today because, in addition to being a major cause of death, HIV is a risk-enhancer for any disease that the body fights with cell-mediated immunity. This changes both the physiology and epidemiology of the disease in ways that complicate the tuberculosis/influenza selection hypothesis.

2.3 Analysis

The analysis will be a continuous time event history analysis where, conditioning on ("controlling for") a host of observable factors, tuberculosis morbidity (recorded in the veterans' medical/pension file) will be assessed as a risk factor for death due to influenza (and vice versa, since there is some evidence from the original work that death codes get mixed-up: tuberculosis deaths spiked in 1918). I have the necessary data in-hand and I have begun the analysis.

Table 1 is a first step, and is meant to be a proof-of-concept example, to show that I have been able to download the raw data, get the data dictionary

(etc.), and, especially the left- and right-censoring variables, sorted-out, and read the data into an analysis package (Stata). The final analysis will include more covariates and will be more refined in terms of sample specification and statistical modeling. Specifically, in addition to useful controls such as height (a proxy for childhood nutrition, and therefore for childhood social status, cf. Floud et al., 1990) the proportional hazard (PH) assumption of the Cox model will be tested. In addition, a number of 2×2 and possibly higher dimension $2 \times \cdots \times 2$ contingency tables will be produced, which have a tangible aspect that the models lack.

The model shows that this is a promising line of inquiry. The regression is a Cox proportional hazard model, one of the workhorses of event history analysis (survival analysis) (see Kalbfleisch and Prentice, 2002), and it shows that a history of tuberculosis has a statistically-significant effect on the hazard of death by influenza. In other words, it agrees in sign and in significance with the central theoretical prediction of interest. Other potential controls include region of residence information, socioeconomic information, marital status, and so on. As noted, the analysis will be refined further in time for the conference. This a chapter in my PhD dissertation that I am finishing this year, so delivering the goods by March will not be a problem.

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