PAA Extended Abstract

Testing the Influenza-Tuberculosis Selective Mortality Hypothesis in Australia*

Andrew Noymer†

23 September 2005

150-Word Abstract

I have postulated that there was selective mortality in the great 1918 influenza pandemic in the USA (Noymer and Garenne, 2000). Tuberculosis morbidity enhances influenza mortality; this selection hypothesis is potentially important for the way we think about mortality change. Influenza pandemics are also a timely public health topic, and since TB remains very prevalent in developing countries, this work is highly relevant.

Confirmation in another country is a next step. Australia is ideal, because (unlike many countries) there are good historical vital statistics and, like the USA, the 1914–18 war was far-removed, at least physically. Using mortality sex differentials and an external event as a natural experiment was the previous methodological innovation. Early analysis has already shown that the flu year (1919 in Australia) was a pivot point in TB mortality sex differentials; this is confirmatory. This paper will provide a valuable comparison to the American results.

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).

*This work is based on a dissertation chapter. Sociology Department, UC–Berkeley. Committee: Neil Fligstein (co-chair), Trond Petersen (co-chair), David A. Freedman (Statistics), George W. Rutherford (UCSF, Epidemiology and Biostatistics).

†andrew@demog.berkeley.edu

[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.

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. 3) 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 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 concen-

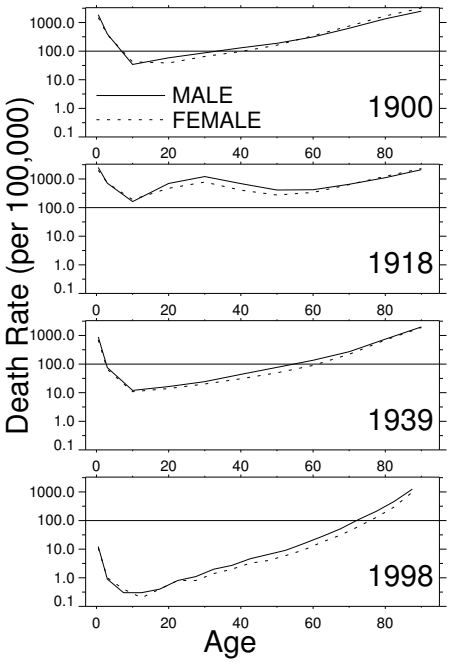

Figure 1: Age-mortality profiles, influenza and pneumonia, United States: 1900, 1918, 1939, 1998

trated 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. 3) 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 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

[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.

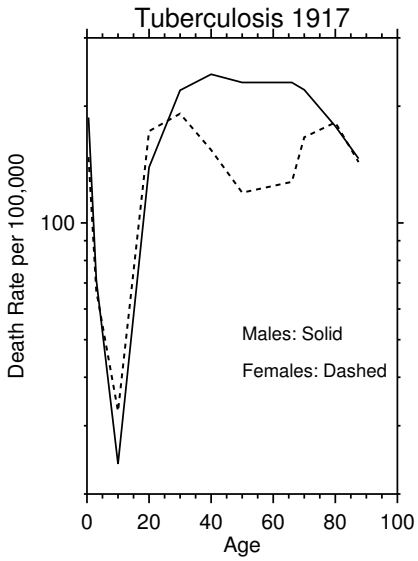

Figure 2: Age-mortality profile, tuberculosis (TB) (all forms), United States, 1917

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/robustness 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 disaggregated 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). The recent surge in interest in 1918 makes my this paper timely.

1.6 Why the Australian data will be important

Verifying the selection hypothesis in a different national population will provide a firmer empirical platform for the the selection hypothesis.

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 Australia in 1919 may be approximated in other countries today, bringing currency to this historical analysis.

2 Australia Paper

2.1 Verifying the selection hypothesis in another country

The selection hypothesis was built using data from the United States. With the exception of some anecdotal or quasi-anecdotal accounts from Europe (Dormandy 1999, p. 235, and Puranen 1991, p. 116, respectively), the selection hypothesis still rests exclusively on American data. Gage (1993) presents data from England and Wales that are congruent with the selection hypothesis, and he notes the importance of considering TB and influenza together, though stops short of articulating a selection concept as such (p. 63). It is important to investigate thoroughly whether the selection effect was idiosyncratically American. The clear way to do this is to investigate data from a different country.

The effects seen in the United States are too large and internally consistent to have been caused by random fluctuations in the strict sense (i.e. sampling error). However, each national population has its own history, and it is important to establish that the selection effects are not driven by circumstances unique to the United States. For example, the US began a bovine tuberculosis eradication program in 1917 (Olmstead and Rhode, 2004). Significant positive externalities for human health have been attributed to this program. Olmstead and Rhode's estimates of the human health improvements from the veterinary TB program are perhaps overly generous, but nonetheless this illustrates the sort of specific historical factors that make it desirable to look at a second country. Another factor could be systematic errors introduced by incomplete death registration in the United States at the time (discussed below).

2.2 The appropriateness of Australia as a comparison

The selection hypothesis is based on the simple idea that those who died in the influenza pandemic were disproportionately likely to have had underlying tuberculosis. In 1918, tuberculosis was worldwide in its distribution, even more so than today. The 1918 influenza pandemic was likewise worldwide in its distribution. Therefore, there is no reason the selection effect should be seen only in the United States. Thus, not only is it desirable methodologically to look at another country for confirmation, but as a theoretical matter there is every reason to expect to see the selection effect in other countries.

Because the selection hypothesis involves the combination of tuberculosis prevalence and the influenza pandemic, there are a variety of countries that could serve as a comparison. As noted, the influenza pandemic was global (Johnson and Mueller, 2002), as was tuberculosis (Bloom and Murray, 1992), so *any* country with well-collected vital statistics can serve as a comparison. The goal of looking at another country is not so much to conduct a factorial pseudo-experiment (for instance where both countries experienced the pandemic but one was poor and the other rich, thus testing the effect of poverty), but do demonstrate that the selection effect was not uniquely American. Nonetheless, one cannot choose any country out of a hat.

During the period of interest, Europe was embroiled in the 1914–18 world war, making European data difficult to interpret. Winter (1976, p. 539) noted "statistical confusion which plagues studies of the 1914–18 conflict" and calls this period "the 'dark ages' of British historical demography". This is especially true of the belligerent countries in Europe, but it applies also to the other European countries, whose societies were affected as well (cf. e.g., Vigness 1932; Romero Salvadó 1999; Beckett 2001, pp. 92–98). Ironically, in Britain, a belliger-

ent power, redistributive wartime programs had a positive effect on population health (Winter, 1977, 1986); for a different perspective see Harris (1993).

Before 1920, not every country kept scrupulous vital statistics records. Not even all the then-industrialized countries had complete registration of vital events, to say nothing of the vast areas under colonial rule. Indeed, even the US did not have complete death registration in 1918 (Dublin, 1915, 1926; Tobey, 1922; Davis, 1926; Linder and Grove, 1943). What makes pre-1930s American data analyzable at all is that the registration area, though less than the entire country, has known denominators. The task of completing the American vital statistics system was was the subject of much contemporary discussion among demographers in the first three decades of the twentieth century (see, for example, Willcox 1906; Cummings 1907; Wilbur 1907, 1911; Dunn 1936; Shapiro 1950).

Although Australia was involved in the war, like the US the actual battlefields were far-removed from the general population, so the aforementioned difficulties of using European data are skirted. Another similarity with the United States is that the Australian population was comprised mostly of European immigrant stock. The institutional exclusion of Aborigines from Australian society extended as far as vital statistics records until the 1960s (Smith, 2005), so none of the analyses I will perform will include any data from what were termed "full blood" Aborigines. As a member of the British Commonwealth, Australia was a party to the 1914–18 World War for longer than the United States, however.

The University Library here has Australian vital statistics volumes that provide death counts, by age and sex and cause. Stanford has volumes to fill in several gaps in Berkeley's collection. Volumes I have examined so far are: Knibbs (1908, 1909, 1910, 1911, 1912, 1913, 1914, 1915, 1916, 1917, 1918, 1919), anonymous (1920, 1921), and Wickens (1922, 1923, 1924, 1925, 1926, 1927). It is hard to grade the quality of data without being able to do an independent cross-check

against the population. In the case of the Australian historical statistics, the populations are long gone, making a definitive statement on data quality impossible. However, the Australian statistics are richly detailed, with cause-of-death data cross-classified not only by age and sex and cause, but also by occupation, state/territory of residence, marital status, month, and so on (though not all of these simultaneously). The meticulousness that this presentation required in the pre-computer era bodes well for the data being of excellent quality. Having good-quality data will be especially important for Australia, where the denominators are smaller than in the United States, making the results more sensitive to potential sampling (or systematic) error in the numerators.

These cause-of-death data, combined with population counts (i.e. denominators) from Smith (2005), will allow me to construct death rates comparable to those that demonstrated the selection effect in the United States. This will allow confirmation of the selection hypothesis in an unrelated population, or it may show differences. Either way, it will be an extremely valuable comparison.

2.3 Analysis

The analysis in this paper will be a straight-forward reproduction of that in Noymer and Garenne (2000), but with Australian data. Specifically, changes in age-specific death rates from tuberculosis will be tracked using explanatory data analysis (using this term in the statistical sense, after Tukey, 1977). Declines in age-specific tuberculosis death rates (particularly pulmonary TB), relative to pre-pandemic trend, will be tracked. Under the selection hypothesis, cohort-specific declines in tuberculosis prevalence (for which TB mortality is an excellent proxy, in the pre-chemotherapeutic era) are expected in proportion to the impact of the influenza pandemic. Graphs are well-suited to this purpose. The importance of

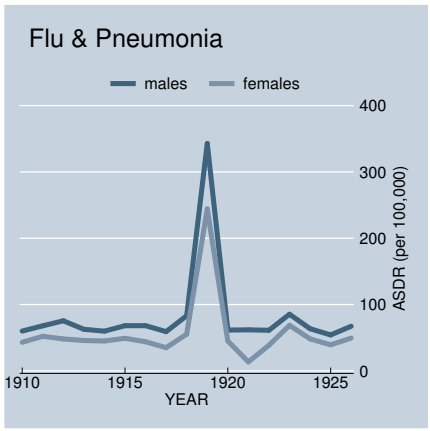

Figure 3: Influenza & pneumonia, Australia, 1910-1926

this paper lies not in new methodological territory, but in testing the selection hypothesis using new data from a different national population.

2.3.1 preliminary results

Data for 1910–26 have been keyed-in and some preliminary comments are possible. More data remains to be keyed-in, which will provide a longer time series, with a richer breakdown.

Figure 3 (p. 14) shows that Australia experienced a dramatic influenza epidemic in 1919. As with the US it was more deadly for males than for females (figure 4, p. 15). The age-standardized death rates (ASDRs) in all the Australian graphs were calculated using the US 1940 standard million (Grove and Hetzel, 1968, p. 37). Using a population from one country as a standard for data from another country is consistent with the logic of demographic standardization (see Wolfenden, 1923, for the canonical reference on standardization), and doing so permits comparison with the graphs in § 1.

Figure 5 (p. 15) illustrates that, like the US, the sex differential in Australian ASDR dropped after the flu pandemic, and remained below the pre-epidemic

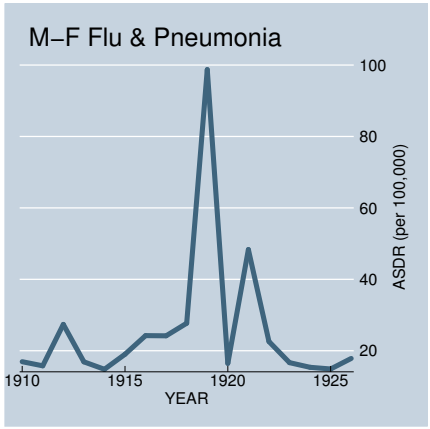

Figure 4: Sex differential, influenza & pneumonia, Australia, 1910-1926

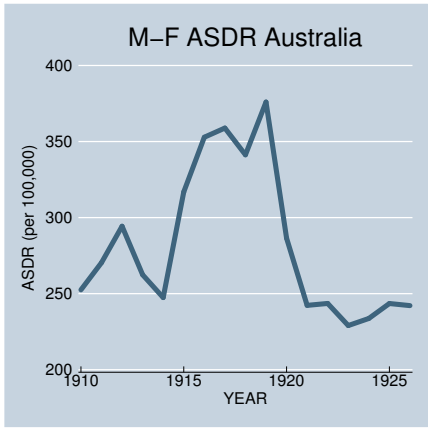

Figure 5: Sex differential, age-standardized death rate (ASDR), Australia, 1910-1926

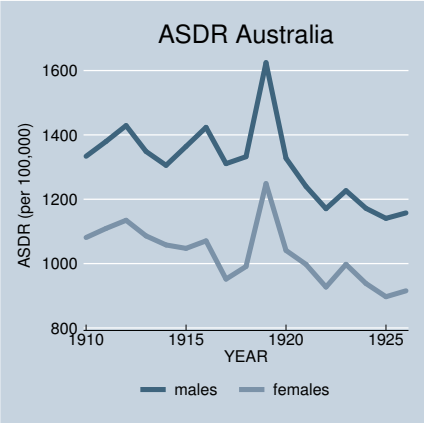

Figure 6: Age-standardized death rate (ASDR [US standard]), Australia, 1910-1926

level through at least 1926; this may be regarded as congruent with the selection effect. However, figure 6 (p. 16) shows that the same effect when plotted for each sex separately is less distinct than in the US.

Figure 7 (p. 17) shows that, like the US, pulmonary tuberculosis in Australia exhibited considerably higher mortality for males. It also demonstrates that TB death rates dropped after 1919, again confirmatory (these same data will be examined also with logarithmic y-axis to test proportionality hypotheses[3]). As before, the effect is less distinct than in the US. Looking at the mortality sex differential of pulmonary TB in Australia (figure 8, p. 17), there are again some similarities, with the 1919 epidemic being a pivot point in the trend line; again confirmatory.

[3] I thank Nick Jewell for reminding me of this.

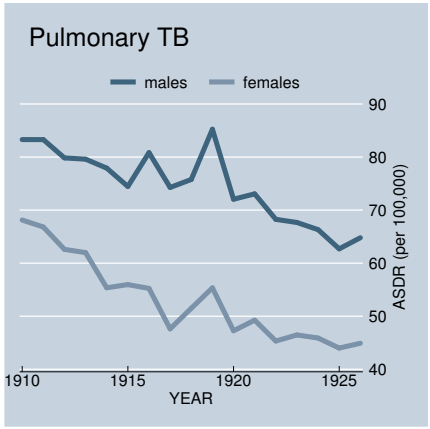

Figure 7: Pulmonary Tuberculosis, Australia, 1910-1926

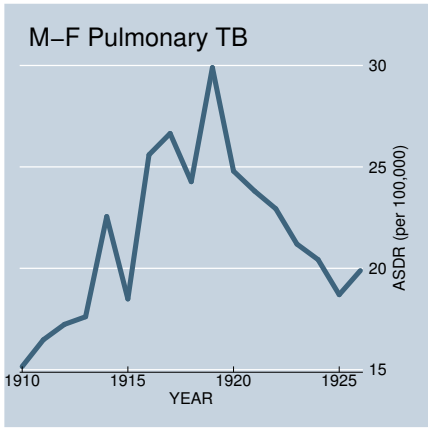

Figure 8: Sex differential, pulmonary Tuberculosis, Australia, 1910-1926

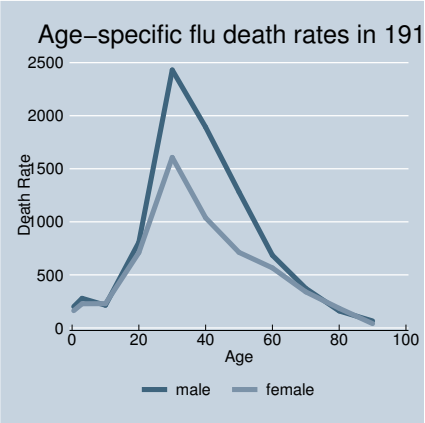

Figure 9: Influenza & pneumonia, pandemic year (1919), Australia

2.3.2 upside-down V *vs.* W

I have already found one attribute of the Australian data deserving further analysis. The flu pandemic in Australia did not follow the W-shape age-mortality profile that characterized the 1918 flu in the United States and elsewhere. In Australia, the age-mortality pattern is an upside-down V, illustrated in figure 9. This aspect of the Australian epidemic has been shown before, in data for Sydney, (McCracken and Curson, 2003), but is noteworthy nonetheless.

In the United States, the W-shape may be thought of as young adults having the *same* flu (actually influenza and pneumonia, combined) death rates as infants and the elderly. It was not unusual in that time period for flu to have high death rates at the extremes of the age distribution; what was different was that the middle mode of the W matched the extremal modes, as discussed in § 1.1 of this prospectus. The typical U-, V-, or J-shaped age-mortality profile for flu is seen in figure 10 (p. 19), with the downturn at the oldest ages being due to small sample size at these extreme ages.

In Australia (in 1919, when the pandemic hit), as in the United States (in 1918), the extreme age groups had a typical flu year. What was unusual in both

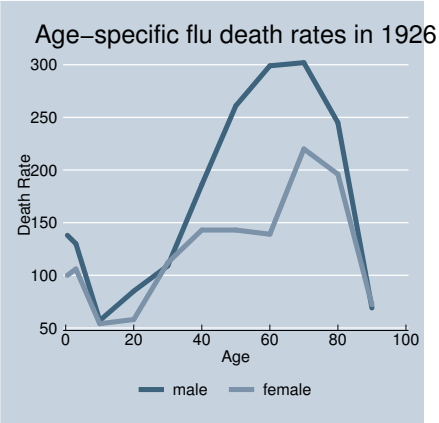

Figure 10: Influenza & pneumonia, "typical year" (1926), Australia

instances were the middle age groups. In Australia, however, the peculiar age-mortality profile was still more exaggerated, so the middle mode of the would-be W is so dominant that it becomes an upside-down V (this could also be described as an A-frame shape). In other words, young adults did not have the same influenza and pneumonia death rates as infants and the elderly — they had rates *far exceeding* the extremes of the age distribution. This is potentially of enormous significance, though it would be premature to draw firm conclusions at present. It would seem to predict, *inter alia*, an even bigger selection effect, due to an even bigger age-mortality anomaly.

2.4 Summary

The point of using a different data set is really two-fold. First, as noted, there is nothing uniquely American about the selection hypothesis, so we expect to see it in other countries. Thus, to establish the selection hypothesis more firmly, it makes sense to demonstrate that selection operated in Australia as well as in the United States. Second, the selection hypothesis was *formed* using the American data set. Once the hypothesis was formed, it was further tested using the same

data set in different ways. This inductive approach is a natural way to go about things, but leaves open an epistemological concern of the circularity of using the same data both to formulate and to test a hypothesis. By examining the Australian data in exactly the same way as the American data, I can test the selection hypothesis on a data set other than the one that was generative of the hypothesis.

This a chapter in my PhD dissertation that I am finishing this year, so delivering the goods by March will not be a problem.

Works Cited

Almond, Douglas. 2005. "Is the 1918 influenza pandemic over? Long-term effects of *in utero* influenza exposure in the post-1940 U.S. population." working paper. `http://www.nber.org/~almond/jmp3.pdf`.

Almond, Douglas V. and Bhash Mazumder. 2005. "The long-term health effects of fetal exposure to the 1918 influenza pandemic: An analysis of SIPP data." *American Economic Review: Papers and Proceedings* forthcoming.

anonymous. 1920. "Official Statstics, Commonwealth of Australia. Population and Vital Statistics." Bulletin No. 38, Commonwealth Bureau of Census and Statistics, Melbourne.

———. 1921. "Official Statstics, Commonwealth of Australia. Population and Vital Statistics." Bulletin No. 39, Commonwealth Bureau of Census and Statistics, Melbourne.

Azambuja, Maria Inês Reinert. 2004. "Spanish flu and early 20th-century expansion of a coronary heart disease-prone subpopulation." *Texas Heart Institute Journal* 31(1):14–21.

Azambuja, Maria Inês Reinert and Bruce B. Duncan. 2002. "Similarities in mortality patterns from influenza in the first half of the 20th century and the rise and fall of ischemic heart disease in the United States: A new hypothesis concerning the coronary heart disease epidemic." *Cadernos de Saúde Pública* 18(3):557–566.

Bardet, Jean-Pierre and Jacques Dupâquier (eds.). 1999. *Histoire des populations de l'Europe: III, Les temps incertains 1914–1998*. Fayard, Paris.

Beckett, Ian F. W. 2001. *The great war 1914–1918*. Longman, London.

Bengtsson, Tommy and Martin Lindström. 2000. "Childhood misery and disease in later life: The effects on mortality in old age of hazards experienced in early life, southern Sweden, 1760–1894." *Population Studies* 54(3):263–277.

Bloom, Barry R. and Christopher J. L. Murray. 1992. "Tuberculosis: Commentary on a reemergent killer." *Science* 257(5073):1055–1064.

Brainerd, Elizabeth and Mark V. Siegler. 2003. "The economic effects of the 1918 influenza epidemic." Centre for Economic Policy Research Discussion Paper Series, No. 3791. `http://www.cepr.org/pubs/dps/DP3791.asp`.

Brzezinski, Zbigniew. 1993. *Out of control: Global turmoil on the eve of the twenty-first century*. Scribner, New York.

Carey, James R. 2003. *Longevity: The biology and demography of life span*. Princeton University Press, Princeton.

Costa, Dora L. 2000. "Understanding the twentieth-century decline in chronic conditions among older men." *Demography* 37(1):53–72.

Crosby, Alfred W. 1989. *America's forgotten pamndemic: The influenza of 1918*. Cambridge University Press, Cambridge. Orig. publ. 1976 as: *Epidemic and peace, 1918*.

Cummings, John. 1907. "Mortality statistics: 1905." *The Journal of Political Economy* 15(6):364–367.

Davis, William H. 1926. "The past and future development of vital statistics in the United States: IV, 1926 progress report in the campaign to bring every state into the registration." *Journal of the American Statistical Association* 21(155):274–279.

Dormandy, Thomas. 1999. *The white death: A history of tuberculosis*. New York University Press, New York.

Dublin, Louis I. 1915. "The improvement and extension of the registration area." *Publications of the American Statistical Association* 14(110):578–582.

———. 1926. "The past and future development of vital statistics in the United States: V, The present registration system." *Journal of the American Statistical Association* 21(155):279–281.

Duffy, John. 1977. "Review of: Alfred W. Crosby, *Epidemic and Peace, 1918*." *Journal of the History of Medicine and Allied Sciences* 32(1):92.

Dunn, Halbert L. 1936. "Vital statistics collected by the government." *Annals of the American Academy of Political and Social Science* 188:340–350.

Gage, Timothy B. 1993. "The decline of mortality in England and Wales 1861 to 1964: Decomposition by cause of death and component of mortality." *Population Studies* 47(1):47–66.

Grove, Robert D. and Alice M. Hetzel. 1968. *Vital statistics rates in the United States, 1940–1960*. United States Department of Health, Education, and Welfare: National Center for Health Statistics, Washington, DC.

Haines, Michael R. and Richard H. Steckel (eds.). 2000. *A population history of North America*. Cambridge University Press, New York.

Harris, Bernard. 1993. "The demographic impact of the first world war: An anthropometric perspective." *Social History of Medicine* 6(3):343–366.

Hobcraft, John, Jane Menken, and Samuel Preston. 1982. "Age, period, and cohort effects in demography: A review." *Population Index* 48(1):4–43.

Hougaard, Philip. 1984. "Life table methods for heterogeneous populations: Distributions describing the heterogeneity." *Biometrika* 71(1):75–83.

Johnson, Niall P. A. S. and Juergen Mueller. 2002. "Updating the accounts: Global mortality of the 1918–1920 "Spanish" influenza pandemic." *Bulletin of the History of Medicine* 76(1):105–115.

Kerber, R.A., E. O'Brien, K.R. Smith, and R.M. Cawthon. 2001. "Familial excess longevity in Utah genealogies." *Journals of Gerontology Series A — Biological Sciences and Medical Sciences* 56(3):B130–B139.

Keyfitz, N and G Littman. 1979. "Mortality in a heterogeneous population." *Population Studies* 33(2):333–342.

Kilbourne, Edwin D. 1987. *Influenza*. Plenum, New York.

Knibbs, G. H. 1908. "Official Statstics, Commonwealth of Australia. Population and Vital Statistics." Bulletin No. 14, Commonwealth Bureau of Census and Statistics, Melbourne.

———. 1909. "Official Statstics, Commonwealth of Australia. Population and Vital Statistics." Bulletin No. 20, Commonwealth Bureau of Census and Statistics, Melbourne.

———. 1910. "Official Statstics, Commonwealth of Australia. Population and Vital Statistics." Bulletin No. 25, Commonwealth Bureau of Census and Statistics, Melbourne.

———. 1911. "Official Statstics, Commonwealth of Australia. Population and Vital Statistics." Bulletin No. 29, Commonwealth Bureau of Census and Statistics, Melbourne.

———. 1912. "Official Statstics, Commonwealth of Australia. Population and Vital Statistics." Bulletin No. 30, Commonwealth Bureau of Census and Statistics, Melbourne.

———. 1913. "Official Statstics, Commonwealth of Australia. Population and Vital Statistics." Bulletin No. 31, Commonwealth Bureau of Census and Statistics, Melbourne.

———. 1914. "Official Statstics, Commonwealth of Australia. Population and Vital Statistics." Bulletin No. 32, Commonwealth Bureau of Census and Statistics, Melbourne.

———. 1915. "Official Statstics, Commonwealth of Australia. Population and Vital Statistics." Bulletin No. 33, Commonwealth Bureau of Census and Statistics, Melbourne.

———. 1916. "Official Statstics, Commonwealth of Australia. Population and Vital Statistics." Bulletin No. 34, Commonwealth Bureau of Census and Statistics, Melbourne.

———. 1917. "Official Statstics, Commonwealth of Australia. Population and Vital Statistics." Bulletin No. 35, Commonwealth Bureau of Census and Statistics, Melbourne.

———. 1918. "Official Statstics, Commonwealth of Australia. Population and Vital Statistics." Bulletin No. 36, Commonwealth Bureau of Census and Statistics, Melbourne.

———. 1919. "Official Statstics, Commonwealth of Australia. Population and Vital Statistics." Bulletin No. 37, Commonwealth Bureau of Census and Statistics, Melbourne.

Lancaster, Tony. 1979. "Econometric methods for the duration of unemployment." *Econometrica* 47(4):939–956.

Langford, Christopher. 2002. "The age pattern of mortality in the 1918–19 influenza pandemic: An attempted explanation based on data for England and Wales." *Medical History* 46(1):1–20.

———. 2005. "Influenza in China 1918–19 and the possible significance of the Chinese labor corps in the development of the 1918–19 influenza pandemic." *Population and Development Review* forthcoming.

Lee, Ronald. 2000. "The Lee-Carter method for forecasting mortality, with various extensions and applications." *North American Actuarial Journal* 4(1):80–91.

Lee, Ronald D. 1992. "Stochastic demographic forecasting." *International Journal of Forecasting* 8(3):315–327.

Lee, Ronald D. and Lawrence R. Carter. 1992. "Modeling and forecasting U.S. mortality." *Journal of the American Statistical Association* 87(419):659–671.

Linder, Forrest E. and Robert D. Grove. 1943. *Vital statistics rates in the United States, 1900–1940*. United States Department of Commerce: Bureau of the Census, Washington, DC.

Mamelund, Svenn-Erik. 2003. "Spanish influenza mortality of ethnic minorities in Norway 1918-1919." *European Journal of Population* 19(1):83–102.

———. 2004. "Can the Spanish influenza pandemic of 1918 explain the baby boom of 1920 in neutral Norway?" *Population* 59(2):269–301.

McCracken, Kevin and Peter Curson. 2003. "Flu downunder: A demographic and geographic analysis of the 1919 epidemic in Sydney, Australia." In Howard Phillips and David Killingray (eds.), *The Spanish influenza pandemic of 1918–19: New perspectives*, chap. 8, pp. 110–131. No. 12 in Routledge Studies in the Social History of Medicine, Routledge, London.

Mills, Christina E., James M. Robins, and Marc Lipsitch. 2004. "Transmissibility of the 1918 pandemic influenza." *Nature* 432:904–906.

Mineau, G.P., K.R. Smith, and L.L. Bean. 2002. "Historical trends of survival among widows and widowers." *Social Science & Medicine* 54(2):245–254.

Noymer, Andrew. 2001. "Mortality selection and sample selection: A comment on Beckett." *Journal of Health and Social Behavior* 42(3):326–327.

Noymer, Andrew and Michel Garenne. 2000. "The 1918 influenza epidemic's effects on sex differentials in mortality in the United States." *Population and Development Review* 26(3):565–581.

Olmstead, Alan L. and Paul W. Rhode. 2004. "An impossible undertaking: The eradication of bovine tuberculosis in the United States." *Journal of Economic History* 64(3):734–772.

Olson, Donald R., Lone Simonsen, Paul J. Edelson, and Stephen S. Morse. 2005. "Epidemiological evidence of an early wave of the 1918 influenza pandemic in New York City." *Proceedings of the National Academy of Sciences of the United States of America* 102(31):11,059–11,063.

Puranen, Bi. 1991. "Tuberculosis and the decline of mortality in Sweden." In Roger Schofield, David Reher, and Alain Bideau (eds.), *The decline of mortality in Europe*, chap. 5, pp. 97–117. Clarendon Press/Oxford University Press, Oxford.

Reid, Alice. 2005. "The effects of the 1918–1919 influenza pandemic on infant and child health in Derbyshire." *Medical History* 49(1):29–54.

Rice, Geoffrey W. and Edwina Palmer. 1993. "Pandemic influenza in Japan, 1918–19: Mortality patterns and official responses." *Journal of Japanese Studies* 19(2):389–420.

Romero Salvadó, Francisco J. 1999. *Spain, 1914–1918: Between war and revolution*. Routledge, London.

Shapiro, S. 1950. "Development of birth registration and birth statistics in the United States." *Population Studies* 4(1):86–111.

Smallman-Raynor, M., N. Johnson, and A.D. Cliff. 2002. "The spatial anatomy of an epidemic: Influenza in London and the county boroughs of England and Wales, 1918-1919." *Transactions of the Institute of British Geographers* 27(4):452–470.

Smith, K.R., G.P. Mineau, and L.L. Bean. 2002. "Fertility and post-reproductive longevity." *Social Biology* 49(3-4):185–205.

Smith, Len. 2005. "Australian Demographic Databank." (digital data file) Version 3.2b.

Specter, Michael. 2005. "Nature's bioterrorist: Is there any way to prevent a deadly avian-flu pandemic?" *The New Yorker* 28 February:50–56.

Tobey, James A. 1922. "The Division of Vital Statistics of the United States Bureau of the Census." *Journal of the American Statistical Association* 18(138):268–271.

Tognotti, E. 2003. "Scientific triumphalism and learning from facts: Bacteriology and the 'Spanish flu' challenge of 1918." *Social History of Medicine* 16(1):97–110.

Trostle, James. 1986. "Anthropology and epidemiology in the twentieth century: A selective history of collaborative projects and theoretical affinities, 1920 to 1970." In Craig R. Janes, Ron Stall, and Sandra M. Gifford (eds.), *Anthropology and epidemiology: Interdisciplinary approaches to the study of health and diseases*, pp. 59–94. Reidel, Dordrecht.

Tukey, John W. 1977. *Exploratory data analysis*. Addison–Wesley, Reading, Massachusetts.

Vaupel, James W., Kenneth G. Manton, and Eric Stallard. 1979. "The impact of heterogeneity in individual frailty on the dynamics of mortality." *Demography* 16(3):439–454.

Vigness, Paul G. 1932. "The neutrality of Norway in the world war." *Stanford University Publications, University Series: History, Economics, and Political Science* IV(1):188 pp.

Weiss, Kenneth M. 1990. "The biodemography of variation in human frailty." *Demography* 27(2):185–206.

Wickens, Chas. H. 1922. "Official Statstics, Commonwealth of Australia. Australian Demography: Summary of Australian Population and Vital Statistics." Bulletin No. 40, Commonwealth Bureau of Census and Statistics, Melbourne.

———. 1923. "Official Statstics, Commonwealth of Australia. Australian Demography: Summary of Australian Population and Vital Statistics." Bulletin No. 41, Commonwealth Bureau of Census and Statistics, Melbourne.

———. 1924. "Official Statstics, Commonwealth of Australia. Australian Demography: Summary of Australian Population and Vital Statistics." Bulletin No. 42, Commonwealth Bureau of Census and Statistics, Melbourne.

———. 1925. "Official Statstics, Commonwealth of Australia. Australian Demography: Summary of Australian Population and Vital Statistics." Bulletin No. 43, Commonwealth Bureau of Census and Statistics, Melbourne.

———. 1926. "Official Statstics, Commonwealth of Australia. Australian Demography: Summary of Australian Population and Vital Statistics." Bulletin No. 44, Commonwealth Bureau of Census and Statistics, Melbourne.

———. 1927. "Official Statstics, Commonwealth of Australia. Australian Demography: Summary of Australian Population and Vital Statistics." Bulletin No. 45, Commonwealth Bureau of Census and Statistics, Canberra.

Wilbur, Cressy L. 1907. "The outlook for a general system of registration of vital statistics in the United States." *Publications of the American Statistical Association* 10(80):395–420.

———. 1911. "The census and the public health movement." *Annals of the American Academy of Political and Social Science* 37(2):42–60.

Willcox, Walter F. 1906. "Death-Rate of the United States in 1900." *Publications of the American Statistical Association* 10(75):137–155.

Winter, J. M. 1976. "Some aspects of the demographic consequences of the first world war in Britain." *Population Studies* 30(3):539–552.

———. 1977. "The impact of the first world war on civilian health in Britain." *Economic History Review* 30(3):487–507.

———. 1986. *The great war and the British people*. Harvard University Press, Cambridge.

Wolfenden, Hugh H. 1923. "On the methods of comparing the moralities of two or more communities, and the standardization of death-rates." *Journal of the Royal Statistical Society* 86(3):399–411.

Yashin, Anatoli I. and Ivan A. Iachine. 1997. "How frailty models can be used for evaluating longevity limits: Taking advantage of an interdisciplinary approach." *Demography* 34(1):31–48.