PAA Extended Abstract

Selective mortality in Norway during the 1918 flu pandemic*

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150-Word Abstract

Using data from the USA, Noymer and Garenne (2000) postulated that there was selective mortality in the 1918-19 influenza pandemic. Specifically, declines of tuberculosis mortality after 1918 were accelerated by the influenza pandemic having consumed, all-at-once, a large umber of tuberculosis-infected people. This knocked-down tuberculosis transmission, and also simply killed in 1918 a large number of TB-infected people whose deaths would otherwise have been distributed throughout the early 1920s. This was not corroborated in Australian data (Noymer 2006). The explanation for this is that in Australia, TB was less prevalent than the USA, and moreover the influenza pandemic was less severe. On the other hand, this paper looks at Norway, where the influenza was severe and in which tuberculosis was famously a major cause of death. *A priori*, Norway ought to corroborate the American findings. It does. The present paper shows age- and time- specific declines consistent with selection.

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1 Background

This PAA paper will fit an important piece into the jigsaw puzzle of the demography of the 1918 influenza pandemic. It is relevant far beyond this niche, however. It touches on international mortality comparisons (Norway, Australia, United States), sex differences in mortality (part of the identification technique), historical mortality (due to the signature importance of tuberculosis in twentieth century mortality decline), and of course selection theories.

The final paper will review the prior results in appropriate detail, but for this extended abstract, I will give only a thumbnail sketch of the relevant prior work. The selection hypothesis is discussed in detail in Noymer and Garenne (2000, 2003) and Noymer (2006). The selection hypothesis holds that the 1918–19 influenza pandemic accelerated the decline of tuberculosis by killing all-at-once (viz., in 1918) a large number of tuberculosis-infected people. These deaths were "part of" the flu pandemic, so to speak, but they were also borrowed against the future ledger of tuberculosis deaths. Thus, tuberculosis deaths in the early 1920s were fewer than they would have been had the 1918–19 pandemic not occurred. What is more, transmission to new cases was also diminished.

1.1 Finding a replication group

The selection hypothesis was built using data from the United States. It is important to investigate thoroughly whether the selection effect was id-

iosyncratically American. The clear way to do this is to look at data from a different country.

Puranen (1991) notes that, in Sweden, "after 1919 the death rate from tuberculosis fell substantially more quickly than the proportion of all deaths from tuberculosis", but, he explains, "...after 1919 specific [medical intervention] measures against tuberculosis began to make their impact" (p. 116). Closer investigation of Swedish data is warranted, but if not for the pandemic it is unclear why 1918–19 should be a pivot point in decline of tuberculosis.

1.1.1 Sanatoria as canaries in the coal mine?

Dormandy (1999, p. 235) notes that tuberculosis sanatoria in Europe were "decimated" by the 1918 pandemic, an observation that is consistent with the selection hypothesis, but he does not provide further information. Hawes (1920) reviews the experiences of American tuberculosis sanatoria in 1918, albeit in more breadth than depth. One of his more detailed illustrations, from Massachusetts state sanatoria, is that in 1918 in the general (state) population, 9.5% of all influenza cases were fatal (presumably he means all clinical cases), whereas the same number was 16.5% inside the sanatoria, and 22% if one includes exacerbated tuberculosis fatalities (Hawes 1920, p. 588). On the other hand, most sanatoria were isolated, which was a countervailing force, making contradictory cases difficult to interpret. Indeed, Markel et al. (2006) report that the famous Trudeau Tuberculosis Sanatorium at Saranac Lake, New York, had no influenza deaths — because it had no influenza cases. And an earlier report by Hawes (1919) provides evidence

that the superintendents of these institutions did in fact take extra isolationary measures during the 1918 pandemic.

1.1.2 Choosing a comparison country

If sanatoria present problems as comparison groups, simply looking at different national data seems like a good way to simply confirm the selection hypothesis (or to point to the the US findings being *sui generis*). 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. At the very least, the selection hypothesis holds that the decline of tuberculosis was accelerated by the 1918 pandemic having killed-off a large number of the tuberculous, whether of not the tuberculous were individually more susceptible. What is needed, then, for a replication study, is a country with both tuberculosis and influenza.

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. 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 examining the effect of poverty), but to 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". Even the great Karl Pearson noted in 1919, "the Great War has rendered it almost impossible for us to feel our way in mortality statistics" (p. 376). Pulmonary tuberculosis death rates increased in the civilian population of England and Wales during the 1914–18 period (Hill 1936, Mercer 1986). There is disagreement about whether this was due to nutritional stress (Mercer 1986) or wartime concentration of people (Winter 2003), but there is no dispute that tuberculosis death rates increased during this period. Apart from an increase in tuberculosis deaths in the pandemic year itself (discussed in Noymer 2006), which is expected other countries, great deviations from trend in pulmonary tuberculosis mortality in 1914–18 muddy the waters a lot. Another factor arguing against using British statistics for a replication study is that in Britain, a belligerent power, redistributive wartime programs had a positive effect on population health apart from tuberculosis (Winter 1977, 2003); for a different perspective see Harris (1993).

The data difficulties are especially severe in the belligerent European countries, but they apply to some extent to neutral European countries, whose societies were affected as well (cf. Vigness 1932, Beckett 2001, pp. 92–98). The name "Spanish flu", as the 1918 pandemic is sometimes called (cf. e.g., Collier 1996), suggests Spain, a neutral country, as a place to look. The war did not affect Spanish industrial output *per se* (Harrison 1978). Nonethe-

less, Romero Salvadó (1999) cites social privation and wartime food shortages.

In the relevant time period (i.e. the 1910s and 1920s), many countries did not record vital statistics scrupulously. In fact, the flu pandemic was the event that spurred some countries to modernize their vital statistics systems (e.g. Canada [cite McGinnis]). 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 United States 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 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). Nothing about the specific pattern of the expansion of the death registration area in the United States suggests it could, in and of itself, account for the selection effect. Only three states (Delaware, Florida, and Mississippi) were in the death registration area of the United States in 1919 but not in 1918 (Linder and Grove 1943). These three states together accounted for about three percent of the size of the death registration area in 1919. In 1920, Nebraska joined the registration area, adding about another 1.5 percent population. No states joined the registration area in 1921. Even making the extreme hypothetical assumption that the additions had no tuberculosis and therefore were purely diluting to the tuberculosis death rates, they can-

not account for the steep drops in age-specific tuberculosis death rates seen after the pandemic. And the age- and sex-specific patterns of tuberculosis decline documented in Noymer and Garenne (2000) are not consistent with a dilution effect.

1.2 Norway

This § will discuss the rationale for using Norway as a comparison country. Norway is a good choice for comparison. It was a neutral country in the world war. It had famously high tuberculosis prevalence (as reflected by TB death rates about twice the contemporary rates in the United States). And, like most other countries, it was struck severely by the flu (as is illustrated in figure 1, p. 9).

2 Evidence

The evidence at this stage is still graphical in nature, though appropriate numerical analyses will be developed. The data come from Backer (1961), though I am exploring other, more detailed, data from the archives. Nonetheless, the evidence in Noymer and Garenne (2000) was likewise primarily graphical in nature.

Figure 1 demonstrates that the 1918 flu pandemic struck Norway with force, and with the characteristic W-shape. The middle mode of the W is highly peaked, as highlighted by the shading in figure 1.

Figures 2–13 plot time series of age-specific tuberculosis death rates for Norway, for the years 1871 to 1940. I am working on getting more time-

resolute data, but these data are in mostly 4- and 5-year groupings by time. Grouping by age is in 10-year groups above age 20.

Because figures 2–13 are grouped in time, each data series is plotted as a step function not as connected points (cf. the graphs). Due to the time-grouping, one should not expect to see any changes immediately after the pandemic (viz., in 1919), as the data are grouped from 1916 up to and including 1920.

This will obviously be elaborated greatly, but we see in these graphs a pattern that is congruent with the original (Noymer and Garenne 2000) findings. Tuberculosis was in decline before 1918 — this is not in doubt. But in the crucial 20–29 age group (figure 7, p. 12), 1918 was a pivot point in the time series. In all the graphs, 1918 is noted by a vertical rule. This age group, had the highest TB death rates among all the ages, and coincided with the peak of the selector (i.e. influenza — cf. figure 1). The key 20–29 year age group did not experience any decline until after 1918. Ages which were comparatively unaffected by the flu (cf. figure 1) do not exhibit any change in trend (as would be expected).

3 Conclusion

These preliminary results support the findings of Noymer and Garenne (2000) that the 1918–19 was a pivot point in tuberculosis mortality.

In the United States, I previously argued, the 1918 flu pandemic accelerated the decline of TB.

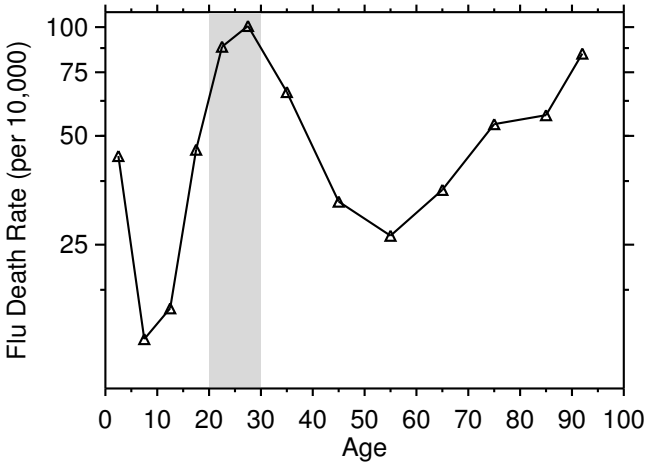

Figure 1: Age-mortality profile, influenza and pneumonia (combined) death rates, Norway, 1918, both sexes combined. The modal ages (20–30) are highlighted with a grey bar. (Confer the post-pandemic changes in figure 7 [p. 12], which corresponds to the age range of the grey bar.) I will obtain sex-specific curves to the extent to which they are available.

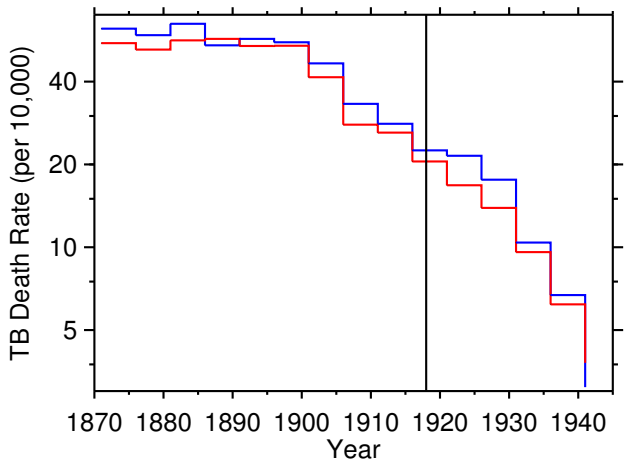

Figure 2: TB death rates, Norway, 1871–1940, males (blue), females (red). The pandemic year (1918) is accented by a vertical rule. Age: 0–1.

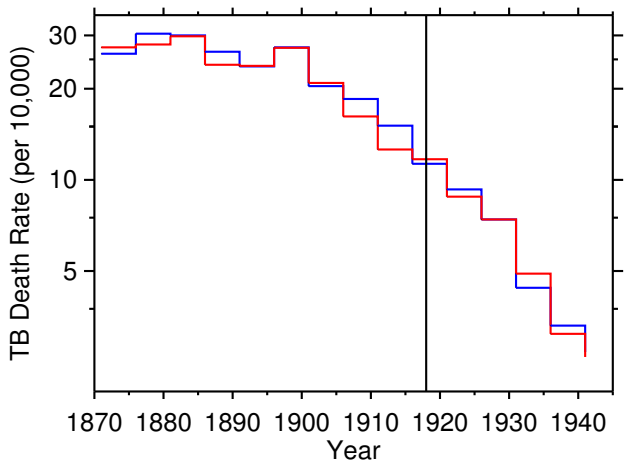

Figure 3: TB death rates, Norway, 1871–1940, males (blue), females (red). The pandemic year (1918) is accented by a vertical rule. Age: 1–4.

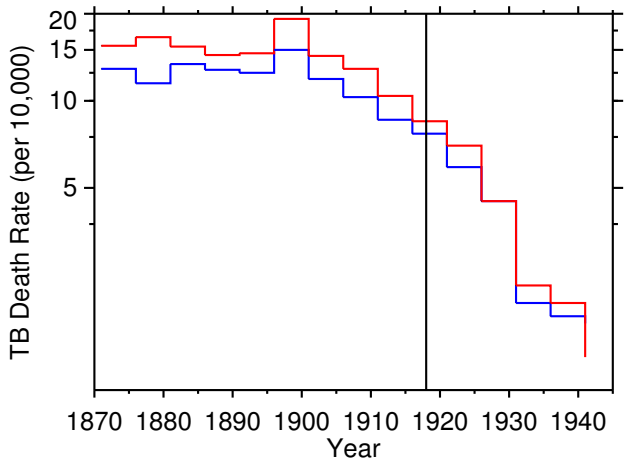

Figure 4: TB death rates, Norway, 1871–1940, males (blue), females (red). The pandemic year (1918) is accented by a vertical rule. Age: 5–9.

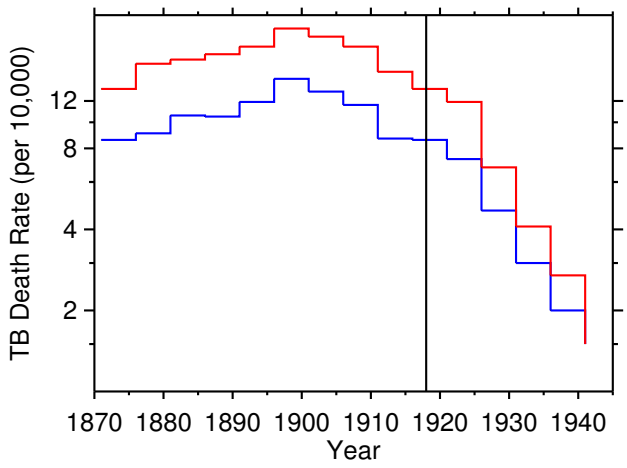

Figure 5: TB death rates, Norway, 1871–1940, males (blue), females (red). The pandemic year (1918) is accented by a vertical rule. Age: 10–14.

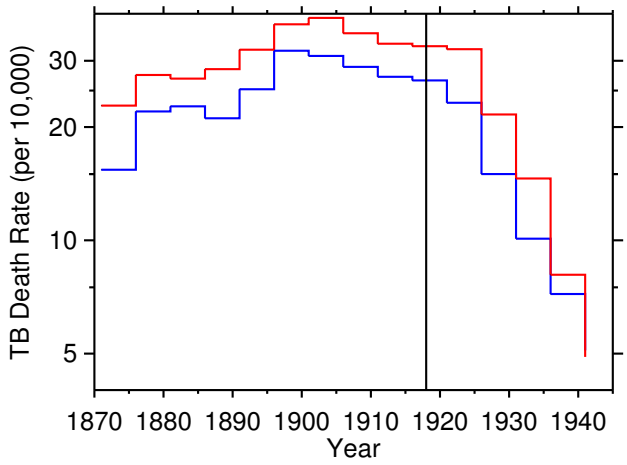

Figure 6: TB death rates, Norway, 1871–1940, males (blue), females (red). The pandemic year (1918) is accented by a vertical rule. Age: 15–19.

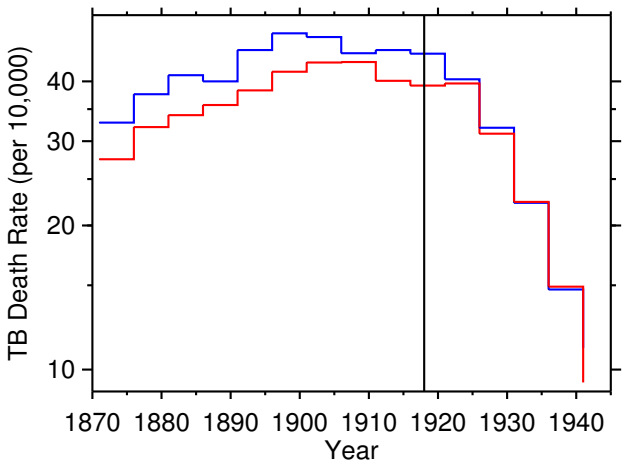

Figure 7: TB death rates, Norway, 1871–1940, males (blue), females (red). The pandemic year (1918) is accented by a vertical rule. Age: 20–29. *This is the crucial part of the quasi-natural-experiment of this paper.*

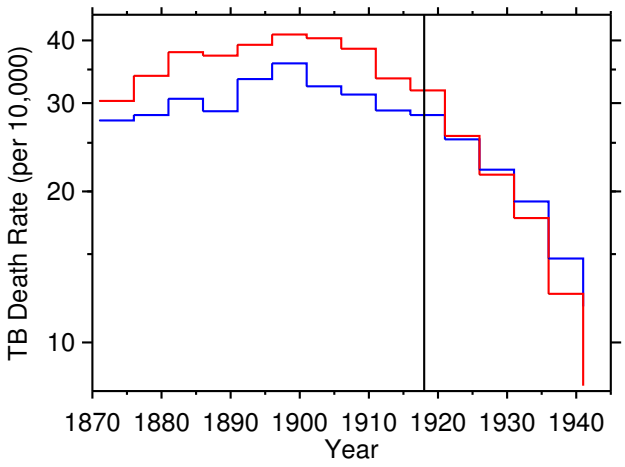

Figure 8: TB death rates, Norway, 1871–1940, males (blue), females (red). The pandemic year (1918) is accented by a vertical rule. Age: 30–39.

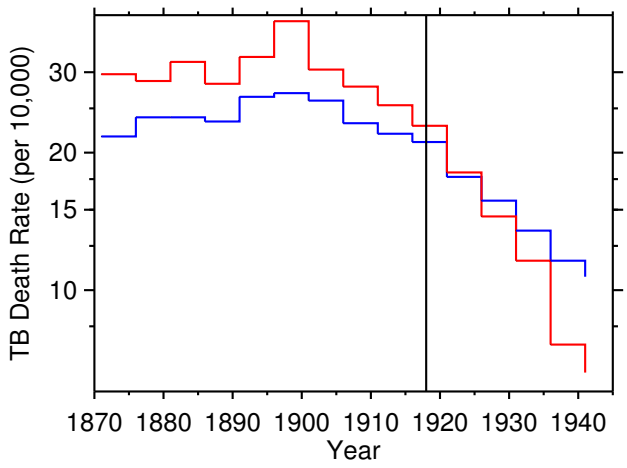

Figure 9: TB death rates, Norway, 1871–1940, males (blue), females (red). The pandemic year (1918) is accented by a vertical rule. Age: 40–49.

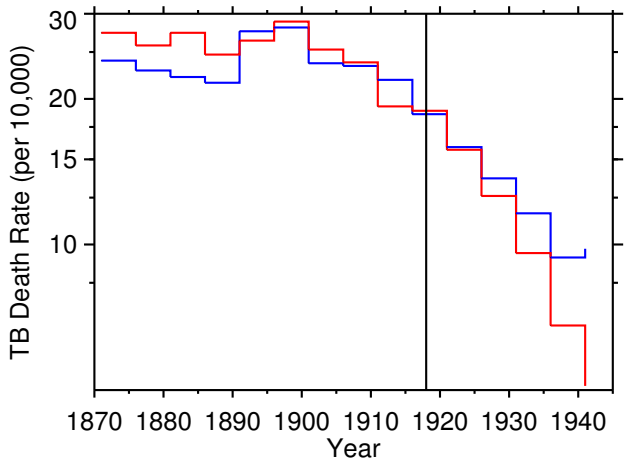

Figure 10: TB death rates, Norway, 1871–1940, males (blue), females (red). The pandemic year (1918) is accented by a vertical rule. Age: 50–59.

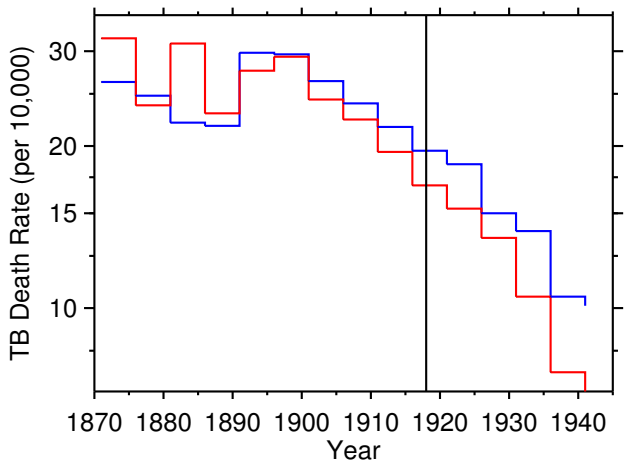

Figure 11: TB death rates, Norway, 1871–1940, males (blue), females (red). The pandemic year (1918) is accented by a vertical rule. Age: 60–69.

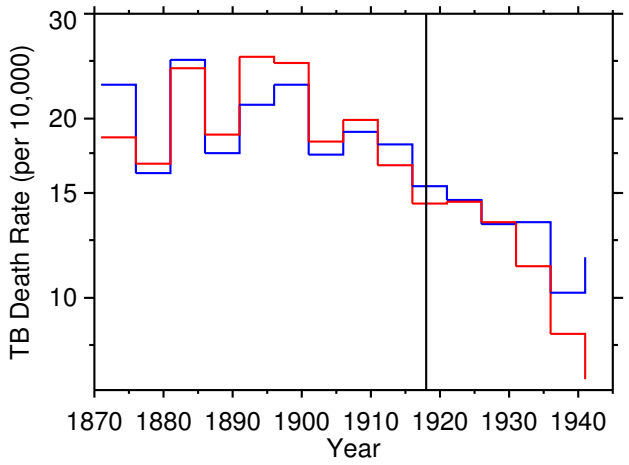

Figure 12: TB death rates, Norway, 1871–1940, males (blue), females (red). The pandemic year (1918) is accented by a vertical rule. Age: 70–79.

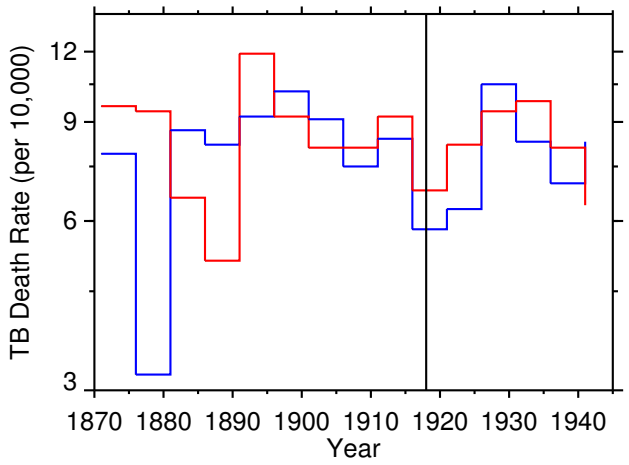

Figure 13: TB death rates, Norway, 1871–1940, males (blue), females (red). The pandemic year (1918) is accented by a vertical rule. Age: 80 and over.

In Norway, in the key age group of 20–29, there was no decline of TB prior to 1918. So we cannot speak of accelerating the decline of TB, but rather, for this age group, we must speak of the flu as catalyzing the decline of TB. Catalyzing is an intentional word choice — a catalyst does *not* cause a chemical reaction, but it helps it along. This is an interesting result as Norway was famously a high-TB country, with perhaps double the overall TB prevalence of the United States.

The results are consistent with the previously-identified selection occurring in Norway as well, especially since the selector (the flu) was highly concentrated in the 20–20 age group (see figure 1).

These results fit in with the US findings, but also the Australia findings. Influenza–TB selection in 1918 was not a universal phenomenon. It occurred

where the flu was severe (most countries, but not all) and where tuberculosis was reasonably severe.

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