Tuberculosis and World War I

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Consumption (or "phthisis"), later renamed tuberculosis, ravaged Europe in the sixteenth, seventeenth, and eighteenth centuries. Some say more than 1 billion people died of the disease during that 300-year epoch of extraordinary mortality. To compound the problem, deaths from consumption climbed even higher during the first half (1760 to 1810) of the Industrial Revolution. But by 1800, or somewhat before, a remarkable about-face took place: the scourge peaked and then began a nearly steady decline that lasted for more than a century (1). Note that the reduction in mortality was underway well before Robert Koch announced his discovery of *Mycobacterium tuberculosis* in 1882, and without significant change mortality kept falling for another 32 years. But then the downward trend abruptly ceased—on July 28, 1914—the start of World War I (WWI): the greatest cause of death and destruction to that time in history. And the carnage didn't stop until November 11, 1918, nearly four and one-half years later.

The purpose of this essay is to examine what actually happened to tuberculosis during WWI: (*1*) why did mortality rates suddenly escalate so strikingly at the outset of hostilities, (*2*) increase considerably higher during wartime, (*3*) decrease precipitously shortly after peace was declared, and (*4*) end up at virtually the same level of mortality that would have been expected if the war never happened?

World War I

Background

WWI loomed ever closer on June 28, 1914 when Serbian terrorists murdered Archduke Franz Ferdinand, heir apparent to the throne of Austria-Hungary, and his wife Sophie. Cognoscenti knew, though, that the Great Powers had long been planning for war, and exactly one month later it happened: Austria-Hungary declared war on Serbia; Russia mobilized its troops; Germany invaded neutral Belgium on its way to battle in France; and Britain declared war on Germany (2). By its end, 32 different nations were fighting.

Major wars have typically led to important advances in medicine, including one by renowned French scientist Marie Curie; after receiving both her Nobel Prizes before the conflict, in 1914 Curie invented and showed how to use small, portable X-ray machines, "Little Curies," for clinical diagnosis near the front lines (3). But such advantages were greatly overwhelmed by the depredations of warfare, including the remarkable buildup and resolution of death-dealing tuberculosis.

Recruitment and Tuberculosis

Wilhelm Conrad Röntgen discovered X-rays in 1895, and within a few years, technical advances and scientific headway established the astonishing clinical benefits of radiography, albeit in primitive form, for examining the human body, including the lungs. Although radiology had progressed considerably, it was unprepared for large-scale, routine high-tech screening of tens of thousands of candidates for conscription into the French Army, both in preparation for WWI and then during it. Within the first 5 months after the beginning of the war (1914), owing to the relatively crude means of evaluation, 86,000 French recruits were discharged to civilian life because of presumed (but not verified) tuberculosis; three years later (1917), the number sent home from the army had climbed to 150,000 (4).

Given the nearly 3-year interval to prepare for impending battle in WWI, the U.S. Army sought to upgrade its evaluation process and refine its criteria for rejection or acceptance of recruits. The army hired 600 tuberculosis-naive general practitioners who took a 6-week course on thoracic physical examination aimed at differentiating among active, quiescent, and absent disease (5). Although advocates for radiological screening were gaining strength, in 1918 the majority of experts favored physical examination over radiology, in part because the few available comparative studies had proved unconvincing. (It is of interest to note that only a few years after the development of X-ray techniques and recognizing their usefulness in warfare, the U.S. Army began to use "roentgenograms" to locate bullets and identify fractures in injured soldiers [6].)

Owing to the perceived logistic shortcomings of mass radiology, U.S. Army estimates indicated that 10,000 recruits with tuberculosis served on active duty, another 5,000 were later diagnosed during wartime with the disease, and tuberculosis became the most common reason for discharge from military service (5). This policy, of course, generated an enormously costly bill to the federal government for medical care and disability pensions for the large number of former military personnel afflicted with tuberculosis during WWI.

Upsurge in Tuberculosis

Findings

Figure 1 shows that from 1885 to 1914, tuberculosis mortality rates declined progressively in England–Wales, Scotland,

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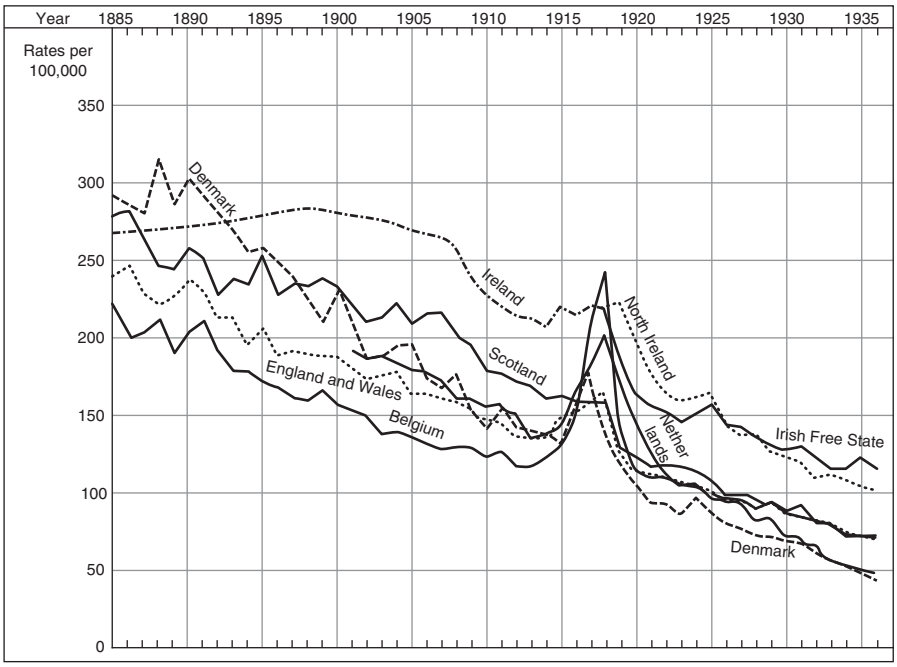

Figure 1. Mortality rates from tuberculosis per 100,000 population during the 50-year period 1885–1935. Countries included are Denmark (*top dashed line* at 1890, continuing to *bottom line* at 1930), Ireland (at 1910), Scotland (at 1910), England and Wales (starting 1895), Belgium (*bottom* at 1905), and the Netherlands (*descending* at 1920). Reprinted by permission from Reference 7.

Belgium, Denmark, and the Netherlands, but less impressively in Ireland (7). Then in 1914, mortality increased sharply and dramatically during wartime until after its end in 1918. Figure 1 further demonstrates that—in general and in all six countries illustrated—without exception, death rates from tuberculosis fell steeply to values in most instances well below those recorded a year or two after the end of the war, and that the prewar, century-plus-long reduction of tuberculosis had resumed its previous downward track.

A way of documenting the effects on tuberculosis mortality in various countries during WWI is to compare rates per 100,000 inhabitants before, during, and after the war: in 1913 (prewar), in 1917 (midwar), and in 1920 (postwar) (7). (Because the first wave of the global pandemic of influenza erupted in March 1918, death rates that year were spuriously elevated; by 1920, the influenza effect had largely dissipated.)

Tuberculosis mortality in both the United States and France remained constant between 1913 and 1917 and did not show the wartime increases seen in several other countries; postwar values, however, did show the expected steep drop in mortality (7). Germany, Austria, and Hungary had typical increases in mortality during warfare, but in 1920 the levels had not yet fallen below the 1913 (prewar) values. Although neutral, Norway, Sweden, Denmark, and Switzerland showed similar war-related increases in tuberculosis death rates as belligerent countries during WWI; all revealed sizable peacetime reductions except Switzerland, whose immediate postwar tuberculosis mortality failed to decrease.

The apparent apogee of increasing death rates from tuberculosis during WWI was established in Warsaw, Poland: in 1913, mortality was an impressive 306 per 100,000 population but more than tripled to 974 in 1917, and then dropped sharply to 337 in 1920 (7).

Mechanisms

Tuberculosis mortality up to the start of WWI was high but continuously decreasing. The striking spike in death rates from tuberculosis during the war years and equally impressive declines shortly afterward appear to be linked phenomena, which strongly suggests that the causative mechanisms suddenly burst forth, and then quickly subsided. In rough order of their relative importance, the possible reasons underlying the increases and decreases, alone or in concert, include the following (8).

Poison gas. A staunch patriot and gifted German chemist, Fritz Haber, invented and first used poison gas as a potentially lethal offensive weapon (9). Haber oversaw the release of chlorine fumes by Germans in the front-line trenches of Ypres, Belgium, in 1915; the British and French quickly produced protective gas masks and retaliated. Not long afterward, mustard gas, which both blinded and blistered, became the favorite. Chemical weapons created countless casualties, but were not decisive in the ultimate allied victory. (In 1919, Nobel Prizes were bestowed during the war years, and the declared winner in chemistry in 1918 was Fritz Haber—for converting nitrogen gas into ammonia for use as fertilizer. Nothing was said about his use of chemical weapons, which unleashed a torrent of protests [10].)

In 1927, A. R. Koontz (11) dispelled the long-held, postwar prevailing belief that exposure to poison gases both predisposed to the development of pulmonary tuberculosis and reactivated old disease: neither proved true. Seventy-five years later, another retrospective look confirmed that victims of gassing had evidence of chronic, nontuberculosis respiratory disease, and also (probably) of cancers of the pharynx, larynx, and lung (12). *Poison gas both killed and maimed extensively during WWI, but had little impact on death rates of tuberculosis.*

Inadequate ventilation. Restricted ventilation favors the spread of tubercle bacilli. Mandatory blackouts and closing of windows reduces ventilation in dwellings and factories in wartime; time spent in sequestered cellars, basements, and bomb shelters worsens exposure. *Abundant fresh air was a therapeutic feature of the sanatorium movement in the late nineteenth and early twentieth centuries* (13)*, and restriction of ventilation during the war years may have made a minor contribution to worsening tuberculosis.*

Overcrowding. Two different phenomena closely linked to WWI and subsequent global conflicts consistently create congestion. First, displacement of people, especially women and children, from war-besieged cities to safer, less vulnerable areas leads to overcrowding.

More importantly, marked overcrowding of refugees seeking safety regularly follows military activity, invasion, or occupation. *Congestion must have played a role in war-torn countries, but similar amplifications of tuberculosis occurred in neutral nations as well.*

Malnutrition. Weakening of immunologic defenses from malnutrition is one of the fundamental mechanisms that enhances the development and progress of tuberculosis. Britain introduced food rationing in February 1918, but serious food shortages were uncommon; the United States, whose army was always well fed, sent food to its Allies before and after the war. The Allied blockade of Germany and its partners was an "express weapon of war" and led to widespread malnutrition and accompanying starvation, which undoubtedly contributed to the Central Power's defeat and, moreover, which lasted until 1919 (14). *The important but marked variations in nutritional intake between Allied countries on the one hand and those under German control on the other, fail to account for the coincident sharp rise and later brisk fall of tuberculosis mortality in many European countries during the war.*

Prolonged physical and mental strain. Like malnutrition, prolonged physical and mental strain is believed to lead to breakdown of resistance to infection and favor development of tuberculosis. In 1915, a new name was fashioned for WWI-caused psychological collapse: "shell shock" (15). It has since been relabeled "posttraumatic stress disorder." No one had a clue how to assess psychiatric meltdown during WWI, let alone how to treat it, and the disorder was considered a sign of weakness and femininity (16). *Shell shock was an important cause of new-onset and lengthy disability but was unrelated to tuberculosis.*

Shortage of medical care. Tuberculosis mortality rates during peacetime before the outset of WWI in the six countries depicted in Figure 1 were roughly 150–200 deaths per 100,000 population (7); thus, routine, prewar medical care in Western European countries involved a gigantic public health undertaking, which included identifying newly diagnosed patients with tuberculosis and managing huge numbers of both chronically sick and moribund patients with the disease. The shifting of tuberculosis-related activities from peacetime to wholesale warfare forced limitation or abandonment of crucial control programs. In 1916, the newly enfranchised Rockefeller Foundation was asked to help address the huge problem of tuberculosis in the French Army, which later blossomed into the Commission for the Prevention of Tuberculosis in France, a United States–French partnership, with robust educational and public health components (17); by 1919, the Commission had mushroomed from 22 to 600 dispensaries and from 8,000 to 30,000 beds. *A shortage of medical care and a breakdown of accompanying socioeconomic progress undoubtedly contributed to the rise in tuberculosis mortality during WWI* [*see also* the next explanation].

Conversion of latent infection to active disease. Figure 1 showing the tall, 6-year-wide triangles of increased tuberculosis mortality indicates that—compared with the declining numbers of deaths that were predicted to occur between 1914 and 1918 in the absence of hostilities—countless "extra" cases and deaths *must* have occurred during wartime. Remember, nearly all adults at that time were infected with *Mycobacterium tuberculosis* (8); it follows that the overwhelming majority of unforeseen deaths certainly included numerous patients already afflicted with latent infection or with smoldering, quiescent, or inactive disease whose tuberculosis ripened into full-blown, rapidly fatal disease actuated by the aggravations of warfare. *The potential role/impact of reporting bias has been proposed as accounting for the observed heightening of tuberculosis mortality during WWI; another point of view indicates that the remarkable congruence of both timing and manifestations originating from multiple sources appears to signify real events.*

Conclusions and Implications

Happily, the more recent results of genetic analysis and whole genome sequencing offer plausible clues to the uncertainties enveloping the transient rise and fall of tuberculosis in WWI (18). Successive waves of extensive global spread of *M. tuberculosis* Beijing lineage have been documented during the last 200 years; the first during the Industrial Revolution, the second during WWI, and later during the HIV epidemic. Strains of the Beijing lineage are supposedly endowed with "selective advantages," such as increased transmissibility, supervirulence, and enhanced progression from infection to disease. So far, so good. But the burst of worsening mortality was short-lived, didn't persist as it should have, and rapidly returned to its previous well-established rate of decline. The conspicuous escalation in mortality of tuberculosis during WWI is telling us something important, but we still don't know the full explanation. ■

Author disclosures are available with the text of this article at www.atsjournals.org.

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