
A Stranger to Our Camps: Typhus in American History

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SUMMARY: Medical observers during the American Civil War were happily surprised to find that typhus fever rarely made an appearance, and was not a major killer in the prisoner-of-war camps where the crowded, filthy, and malnourished populations appeared to offer an ideal breeding ground for the disease. Through a review of apparent typhus outbreaks in America north of the Mexican border, this article argues that typhus fever rarely if ever extended to the established populations of the United States, even when imported on immigrant ships into densely populated and unsanitary slums. It suggests that something in the American environment was inhospitable to the extensive spread of the disease, most likely an unrecognized difference in the North American louse population compared to that of Europe.

KEYWORDS: typhus, Civil War, military medicine, relapsing fever, American Revolution, body louse, disease evolution

“We never had real typhus in the United States,” a historian of public health told me in the late 1970s; “something wasn’t right for it here.” This pronouncement recurred out of memory recently when I was considering disease patterns in the American Civil War. Even though all the conditions appeared ripe for typhus to flourish, it indeed did not erupt within the apparently fertile fields of Civil War encampments. The official medical history of the Civil War, written in the 1880s, noted that “the records do not furnish a single instance of undoubted typhus as having occurred among our troops in the field. . . . [T]yphus was fortunately a stranger

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to our camps.”¹ Typhus is spread by human body lice, among people weakened by malnutrition, huddled together for warmth, and deprived of clean clothes or bathing opportunities. The prison camps that housed lousy soldiers both north and south supplied this optimal environment in abundance, yet the organism that causes typhus failed to take advantage. Was the American environment somehow exempt from invasion by this major killer of nineteenth-century European soldiers, sailors, refugees, and prisoners?

Typhus cases have occurred in the United States, ranging in time from the American Revolution to 2001—in most instances, however, the disease remained contained within a community of very recent immigrants from Europe, and did not spread far beyond this nidus. The major exception to this North American pattern has been typhus in Mexico, where it appeared shortly after the Spanish occupation and persisted well into the twentieth century. Although one epidemic among southwestern Indian tribes in 1921 revealed cross-border transmission from Mexico, such transfer did not happen often. This pattern suggests that there is some factor that makes the American environment north of Mexico inhospitable for the disease. The match was repeatedly struck, but the straw did not catch. This paper will both demonstrate this curious American typhus pattern, and speculate about its explanation.

It is not easy to track the course of typhus in North America before the mid-twentieth century, and some historians would probably claim that the attempt is so fraught with confusion as to be not worth the effort. It is true that the modern category of typhus—a disease caused by the organism *Rickettsia prowazekii*—does not map neatly onto diagnoses common in the eighteenth and nineteenth centuries. Yet the fact that following an organism’s path is difficult does not make the story any the less fascinating. I follow Mirko Grmek in asking questions about the ecology of disease, and wondering what features of the environment, broadly conceived, promoted or inhibited the organism from flourishing.² It is somewhat peculiar to be talking about why a disease was *not* a major problem—it is far more typical to explain why major epidemics happened when and where they did. After all, there were no major outbreaks of plague, leprosy, legionnaire’s disease, or AIDS in the nineteenth-century United States either, and this has heretofore not troubled historians. But

1. Charles Smart, *The Medical and Surgical History of the Civil War*, 10 vols. (Washington, D.C., 1888; reprint Wilmington, N.C.: Broadfoot, 1991), 5: 324, 331.

2. Mirko Grmek, *History of AIDS: Emergence and Origin of a Modern Pandemic*, trans. Russell C. Maulitz and Jacalyn Duffin (Princeton: Princeton University Press, 1990).

in this case there is every reason to believe, based on events elsewhere, that typhus should have flourished in North American cities, or amidst the foul prison camps of the Civil War. Like counterfactual history, this story challenges our understanding of cause and effect, and may offer insight into disease etiology.

The Biology of Typhus

Typhus fever as defined in modern textbooks is caused by *Rickettsia prowazekii*, one of a family of true bacteria that are intermediate in size and character between viruses and more typical bacteria. Rickettsiae are obligate intracellular organisms, like viruses; unlike viruses, but like other bacteria, they are visible with proper staining through a microscope. All rickettsial diseases require an arthropod vector. The typhus organism is spread by the human body louse, *Pediculus humanus*. The louse sucks the infected host's blood, taking in the rickettsiae with the blood meal; the organism then multiplies in the louse gut, and is expelled in the feces. If the louse has moved to a new human host (as it is wont to do when a host becomes too hot with fever or too cold with death), microbes in the feces can penetrate the new victim through skin abraded by the itchy response to louse infestation. Dried feces in clothing or bedding can also land on the mucous membranes in the nose or mouth, allowing the typhus organism to enter a new host via that route. Ultimately, the louse dies due to digestive-organ damage. Typhus therefore appears to be highly contagious to caregivers, and can also be spread to people handling the soiled clothes or blankets of the typhus victim.³

Typhus spreads most rapidly in conditions of crowding, cold, and poverty: crowding enhances the inhalation of lice feces from the patient by the next victim; cold weather encourages the constant wearing of clothing and huddling together inside for warmth; poverty makes it likely that there is no change of clothes or opportunity for washing the body or belongings. Thus the association of typhus with camps, prisons, famines, and mass exodus has been common in the last five hundred years. Within such congeries of misery, the disease will spread very quickly, causing waves of prostration and death. Children acquire the infection, but rarely die of it.⁴

3. Alfred J. Saah, "*Rickettsia prowazekii* (Epidemic or Louse-Borne Typhus)," in *Principles and Practice of Infectious Diseases*, ed. Gerald L. Mandell, John E. Bennett, and Raphael Dolin (Philadelphia: Churchill Livingstone, 2000), pp. 2050–53.

4. *Ibid.*

Most cases of typhus begin with the sudden appearance of fever, headache, and searing pains in the bones and joints. As the disease progresses, the mental state can decline through apathy, delirium, and coma. On days five through seven the rash appears, consisting of small red spots that resemble petechiae; initially blanching, the spots gradually darken and persist as the inflamed blood vessels that originally showed red break down and small hemorrhages dot the body. Dependent areas rapidly form ulcers that can necrose, causing painful bedsores. Typhus tends to cause constipation, not diarrhea—a key point in the differential diagnosis. If the patient survives, the illness resolves by day fourteen.⁵

A case of typhus confers lifelong immunity, of sorts. Although persons lucky enough to have survived will not be at risk of acquiring the disease anew, they may suffer from a recrudescence years or even decades later. Now called Brill-Zinsser disease for the two physicians who identified the phenomenon, cases of recrudescence typhus are usually mild and nonfatal⁶—but the patient can communicate the disease, if lice are present. Since there is no major animal reservoir for typhus, the phenomenon of recrudescence offers the organism a place to hide while awaiting a new opportunity to spread in the population. Like shingles, which represents a recrudescence of chicken pox, Brill-Zinsser disease tends to occur when the host's immunity is depressed by age, malnutrition, fatigue, or concurrent illness.⁷

Confusing the diagnosis of epidemic typhus is a related disease called murine typhus, spread by another rickettsial organism, *R. typhi*. The two organisms, *R. prowazekii* and *R. typhi*, are extremely similar: serological tests for one cross-react with the other; immunity to one conveys immunity to the other;⁸ and their genomes are very close, with only minor differences.⁹ Yet *R. typhi* travels in different circles, living among rodent populations.

5. Ibid.

6. Nathan Brill identified cases of mild typhus-like fevers among elderly Jewish immigrants living in louse-free surroundings, and published his results in 1910–11: Nathan E. Brill, "Pathological and Experimental Data Derived from a Further Study of an Acute Infectious Disease of Unknown Origin," *Amer. J. Med. Sci.*, 1911, 142: 196–218. Hans Zinsser and colleagues further clarified the identity of this disorder in the 1930s: Hans Zinsser, "The Rickettsia Diseases: Varieties, Epidemiology and Geographical Distribution," *Amer. J. Hygiene*, 1937, 25: 430–63.

7. Saah, "*Rickettsia prowazekii*" (n. 3). On the American flying squirrel as a reservoir for typhus, see the last section of this paper.

8. J. Stephen Dumler and David H. Walker, "*Rickettsia typhi* (Murine Typhus)," in Mandell et al., *Principles and Practice* (n. 3), pp. 2053–55.

9. Jan O. Andersson and Siv Andersson, "A Century of Typhus, Lice, and *Rickettsia*," *Res. Microbiol.*, 2000, 151: 143–50; M. P. McLeod et al., "Complete Genome Sequence of *Rickettsia typhi* and Comparison with Sequences of Other Rickettsiae," *J. Bacter.*, 2004, 186: 5842–55.

In North America it infests rats; it is spread from rat to rat by either fleas or lice, and to humans by the rat fleas, humans being incidental intruders into this zoonosis. Given the similarity of the organisms, some have speculated that epidemic typhus evolved from endemic typhus, and even that this may have happened when the murine typhus organism of Mexico met the European louse carried by Spanish invaders.¹⁰ Like syphilis, epidemic typhus appeared in Europe right around 1500, raising speculation that it had been imported from the New World.¹¹ In any event, it does not appear that a disease like epidemic typhus existed among the Inca, Maya, or Aztec, the civilizations of the Western Hemisphere with the most densely packed peoples, before Spanish contact.¹²

Murine and epidemic typhus are most easily distinguished by their epidemiology. Murine typhus kills at most 5 percent of its victims; epidemic typhus has a mortality ranging from 10 to 60 percent or more. This is true of the differential diagnosis of epidemic typhus in general. The specific case of fever and rash, laid side by side with a case of Rocky Mountain spotted fever, murine typhus, typhoid fever, meningococemia, relapsing fever, or multiple viral syndromes, may be difficult to diagnose except by sophisticated laboratory analysis. It is the mortality pattern of typhus, coupled with its rapid spread within a susceptible population, that makes it apparent in the historical record.

Typhus in North America

It is probable that the first typhus epidemics in North America occurred in Mexico in the three centuries after Spanish conquest. Although the colonial disease reports in the Spanish archives are fairly detailed, historians still argue over whether certain outbreaks were smallpox, typhus, or both.¹³ The disease that may have been typhus was called *tabardillo*; into the mid-twentieth century, researchers debated whether this was the same as European epidemic typhus, or was a virulent form of murine typhus.¹⁴

10. Patrick A. Buxton, *The Louse: An Account of the Lice Which Infest Man, Their Medical Importance, and Control* (Baltimore: Williams & Wilkins, 1946), p. 83.

11. Erwin Ackerknecht, *History and Geography of the Most Important Diseases* (New York: Hafner, 1965), p. 33.

12. Francisco Guerra, "Origen y efectos demográficos del tifo en el México colonial," *Colon. Lat. Amer. Hist. Rev.*, 1999, 8: 273–319.

13. Donald B. Cooper, *Epidemic Disease in Mexico City, 1761–1813: An Administrative, Social, and Medical Study* (Austin: University of Texas Press, 1965).

14. See, for example, Zinsser, "Rickettsia Diseases" (n. 6); H[erman] Mooser, "Tabardillo, an American Variety of Typhus," *J. Infect. Dis.*, 1929, 44: 186–93. On the recent distribution of typhus in Mexico, see Rudolfo Acuna-Soto et al., "Murine Typhus in Mexico City," *Trans.*

This confusion persists in modern dictionaries, which define the word variably as murine typhus, epidemic typhus of Mexico, a murine typhus that can occur in epidemic form, and so on.¹⁵

Modern studies have shown widespread antibodies to both *R. typhi* and *R. prowazekii* in the Mexican population, with the former common in the lowlands and the latter found in the highlands.¹⁶ Convincing evidence for both diseases can be found, and most probably both have prevailed in Mexico. Certainly some form of rickettsial fever occurred in epidemic outbreaks in Mexico well into the twentieth century, with high attack rates and high mortality rates. One such epidemic in 1910 killed Howard Ricketts, the scientist who first identified the causative agent of Rocky Mountain spotted fever (another rickettsia) and for whom the genus was named.¹⁷ And it almost killed Joseph Goldberger, of pellagra fame, who was likewise in Mexico in 1910 to study this interesting disease.¹⁸ The diagnosis of typhus before the twentieth century is difficult; its diagnosis for the past one hundred years is confused by the presence of these two organisms and their controversial relationship.

The relevance of the Mexican experience to that of the United States and Canada is even murkier. It is not entirely clear that the Mexican *R. prowazekii* and the European were the same; whether *R. typhi* in Mexico could sometimes be spread by lice, from human to human; and whether these infecting lice of Mexico were the same in all particulars as their American cousins. Kenneth Maxcy identified murine typhus only in 1926, and he found it in southern cities of the United States; since then, it has been detected in much of the world. Murine typhus cases continued to appear in the American south well into the 1980s, involving isolated inci-

Roy. Soc. Trop. Med. & Hygiene, 2000, 94: 45; V. E. Alcantara et al., "Typhus Group Rickettsiae Antibodies in Rural Mexico," *Emerg. Infect. Dis.*, 2004, 10: 549–51. To complicate matters further, a third strain of rickettsiae, *R. felis*, found primarily in cats, opossums, and their fleas, can also infect humans: see Abdu F. Azad et al., "Flea-Borne Rickettsioses: Ecological Considerations," *Emerg. Infect. Dis.*, 1997, 3: 1–12.

15. See, e.g., the tabardillo entries in *Dorland's Illustrated Medical Dictionary* (Philadelphia: Saunders, 2000); *Taber's Cyclopedic Medical Dictionary* (Philadelphia: Davis, 2001); *Stedman's Medical Dictionary* (Philadelphia: Lippincott Williams & Wilkins, 2000); *Butterworth's Medical Dictionary* (London: Butterworth, 1978); *Blakiston's Gould Medical Dictionary* (New York: McGraw-Hill, 1979); *International Dictionary of Medicine and Biology* (New York: Wiley, 1986).

16. Acuna-Soto et al., "Murine Typhus" (n. 14); Alcantara et al., "Typhus Group" (n. 14).

17. Victoria A. Harden, *Rocky Mountain Spotted Fever: History of a Twentieth-Century Disease* (Baltimore: Johns Hopkins University Press, 1990).

18. Alan M. Kraut, *Goldberger's War: The Life and Work of a Public Health Crusader* (New York: Hill and Wang, 2003).

dents of humans in the company of rats.¹⁹ Whether murine typhus existed in the United States before the twentieth century is unknown—likely, but impossible to prove.

The best-documented appearance of typhus north of the Rio Grande occurred during the American Revolution, when British physicians familiar with the jail typhus of England diagnosed similar cases among prisoners and soldiers kept aboard British ships anchored along the American coast.²⁰ There is no clear evidence to suggest that the infection spread significantly on the mainland, however. The disease that laid American General Nathanael Greene low in August 1776, perhaps changing the course of history by allowing the British to take New York, has been attributed to typhus by some historians. His illness occurred amid an outbreak of fever that affected a quarter of his troops. This epidemic may have stemmed from poor camp sanitation, for Greene noted in orders issued 18 July 1776 that “Tis with Pain the General [has] of late Discovered to[o] much Inattention to the filling and Digging the Necessary vaults for the Regiment,” and that the troops had been “easing themselves in the Ditches of the Fortifications, a practice that is Disgraceful to the last Degree.”²¹ This report, coupled with the time of year, makes a disease spread by the fecal-oral route, such as typhoid, a more likely diagnosis than typhus. Greene and his physician, John Morgan, called it a putrid and bilious fever—labels that would have included typhoid fever, along with many other disorders.²² The distinction between typhoid and typhus fevers would not be made for another half-century.

19. Kenneth R. Maxcy, “Endemic Typhus of the Southeastern United States: The Reaction of the White Rat,” *Pub. Health Rep.*, 1929, 44: 1935–43; R. E. Dyer, “A Virus of the Typhus Type Derived from Fleas Collected from Wild Rats,” *ibid.*, 1931, 46: 334–38; Norman H. Topping and R. E. Dyer, “Apparent Recent Extension of Typhus in the United States,” *Amer. J. Trop. Med.*, 1943, 23: 37–42; J. Stephen Dumler, Jeffrey P. Taylor, and David H. Walker, “Clinical and Laboratory Features of Murine Typhus in South Texas, 1980 through 1987,” *JAMA*, 1991, 266: 1365–70.

20. Robert Robertson, *Observations on the Jail, Hospital, or Ship Fever* (London: Murray, 1783), describes his experience with typhus as a surgeon aboard British ships employed in America during the revolution.

21. General Greene’s Orders, 28 July 1776, in *The Papers of General Nathanael Greene*, ed. Richard K. Showman, 2 vols. (Chapel Hill: University of North Carolina Press, 1976), 1: 267–68.

22. Nathanael Greene to George Washington, 15 August 1776, *ibid.*, 1: 287–88; John Morgan, *A Vindication of His Public Character in the Station of Director-General of the Military Hospitals, and Physician in Chief of the American Army* (Boston: Powers and Willis, 1777), pp. 107–8. Theodore E. Woodward, “Typhus Verdict in American History,” *Trans. Amer. Clin. Climat. Assoc.*, 1970, 82: 1–8, attributes the illness to typhus.

Benjamin Rush, famous as a signer of the Declaration of Independence and professor at the medical college of the University of Pennsylvania, described typhus fever among Washington's soldiers at Bethlehem, Pennsylvania, in the summer of 1776.²³ In an 1815 publication, he remembered that typhus had not spread until the armies from north, middle, and south came together at New York and Ticonderoga, and then "typhus became universal, and spread with such peculiar mortality in the armies of the United States."²⁴ Yet this statement is not as definitive as it sounds. Rush did not use the word "typhus" as modern textbooks do today. First of all, he claimed that "there is but one fever. . . . I do not admit of its artificial division into genera and species."²⁵ By "[t]he *typhus* state of fever" he meant a fever that was "generally preceded by all those circumstances which debilitate the system. . . . It is known by a weak and frequent pulse, a disposition to sleep, a torpor of the alimentary canal, tremors of the hands, a dry tongue, and in some instances, by a diarrhoea."²⁶ While he claimed that these symptoms were common to "what is called the jail, the ship, and the hospital fever," the state also had other names and appearances; he preferred the name "*low chronic* state of fever" for these symptoms.²⁷ Rush might have seen an outbreak of typhus, or, more likely, one of typhoid.

The attempt to track typhus outbreaks in the next decades continues to be hampered by contemporary diagnostic uncertainty. Until the mid-nineteenth century most European and American physicians thought typhus and typhoid were varieties of the same fever. The work of P. C. A. Louis brought this into question, however, when he found on autopsy that a consistent feature of typhoid victims was the inflammation of Peyer's glands in the small intestine. The American William Gerhard came home from studies in France and Britain with an awareness that there seemed to be two different diseases, called by the same name, in the two countries; he would later argue that there was little typhus in France during the 1820s when Louis was pursuing his researches, and that, on the other

23. Oscar Reiss, *Medicine and the American Revolution: How Diseases and Their Treatments Affected the Colonial Army* (Jefferson, N.C.: McFarland, 1998), p. 188.

24. Benjamin Rush, "The Result of Observations Made upon the Diseases which Occurred in the Military Hospitals of the United States, during the Revolutionary War between Great Britain and the United States," in *Medical Inquiries and Observations*, ed. Benjamin Rush, 4 vols. (Philadelphia, 1815; reprint New York: Arno Press, 1972), 1: 145–50, on p. 149.

25. Benjamin Rush, "Outlines of the Phenomena of Fever," in Rush, *Medical Inquiries* (n. 24), 3:1–36, on p. 19.

26. *Ibid.*, p. 27.

27. *Ibid.* (italics in original).

hand, typhoid was relatively uncommon and typhus common in England at the time. He had an opportunity to further explore the disease common in England and Ireland when an epidemic of continued fever broke out among Irish immigrants in Philadelphia during the winter of 1835–36. Gerhard's Philadelphia outbreak was typical of the typhus experience in the United States during the nineteenth century: the disease was clustered in a Philadelphia slum that was home to very recent Irish immigrants, less than a month off the boat. Gerhard was able to distinguish the British/Irish fever from the French one on the basis of pathological changes in the gastrointestinal tract.²⁸ It took a couple of decades, but by 1860 most well-educated American physicians would agree that the two diseases could be differentiated by autopsy: typhoid fever resulted in Peyer's-gland inflammation, and typhus did not.²⁹

Still, the historian of typhus in nineteenth-century America has to tread carefully. Autopsies were rarely done—so even after doctors agreed upon a distinction based on pathology, the key information was rarely available. It is likely that in any epidemic, cases of other diseases are mixed in with the supposed typhus statistics. The very concept of fever remained fluid throughout the century. Gerhard himself noted that typhus acquired a diarrheal component as the summer came on; but since typhus is usually a disease of the winter months, it is likely that his typhus cases with diarrhea (but without Peyer's-patch inflammation) were caused by some other microorganism. As late as 1881, a physician could describe an outbreak of typhus as acquiring some typhoid characteristics. Further confusing the medical writing of the time was typhomalarial fever, an entity first labeled during the American Civil War and abandoned only after research during the Spanish-American war.³⁰

28. W. W. Gerhard, "On the Typhus Fever, which Occurred at Philadelphia in the Spring and Summer of 1836; Illustrated by Clinical Observation at the Philadelphia Hospital; Showing the Distinction between this Form of Disease and Dothinenteritis, or the Typhoid Fever, with Alteration in the Follicles of the Small Intestine," *Amer. J. Med. Sci.*, 1837, 19: 289–322.

29. Dale C. Smith, "Gerhard's Distinction between Typhoid and Typhus and Its Reception in America, 1833–1860," *Bull. Hist. Med.*, 1980, 54: 368–85.

30. On the mutability of typhus in 1881, see Henry E. Branin, "The History, Origin, etc. of the Epidemic of Typhus Fever at the Camden Co. Alms-house," *Trans. Med. Soc. New Jersey*, 1881, 115: 216–34. Joseph J. Woodward promoted the new designation "typho-malarial" in his *Outline of the Chief Camp Diseases of the United States Armies as Observed During the Present War* (Philadelphia, 1863; reprint New York: Hafner, 1964). Dale C. Smith chronicles the history of this hybrid diagnosis, and further exposes the nineteenth-century medical confusion over spotted fevers, in "The Rise and Fall of Typhomalarial Fever," *J. Hist. Med. & Allied Sci.*, 1982, 37: 182–220, 287–321. Vincent J. Cirillo describes the investigation of camp diseases,

The first widespread importation of typhus to the United States and Canada began in 1847, via the immigrant ships fleeing the Irish famine. A Boston physician treating the outbreak reported that it was “clearly different from any fever which has hitherto been known in this country,” and identified it with the jail, hospital, and camp fever of Britain.³¹ His comments reflect the likelihood that typhus had almost disappeared in the intervening years since Gerhard’s Philadelphia experience. Around 100,000 Irish entered Canada in 1847; at least 15,000 of them died from starvation and disease, with epidemic typhus a major killer both on ship and once landed in the New World.³² Although statistics are less complete for arrivals into the United States, thousands of Irish immigrants either arrived ill or became sick soon after landing, often stricken with typhus fever. The disease was kept alive as more and more immigrants came, but epidemic typhus did not spread significantly beyond these groups. In one New York hospital there were 138 cases of typhus in May and June 1847; only 7 were American, and 5 of those had had direct contact with new immigrants in their jobs as ferry boatmen or dock peddlers.³³ In 1852 the Marine Hospital on Staten Island admitted 3,040 cases of ship fever, and 17 percent of these patients died; they were all recent immigrants.³⁴ Irish emigrants also shipped to Louisiana in droves, and typhus traveled there as well. Charity Hospital in New Orleans admitted 1,045 cases in 1847, 9 percent of its patients; again, the disease was limited to the immigrants themselves and the people in their immediate vicinity, such as doctors and nurses.³⁵

especially typho-malaria, in his book *Bullets and Bacilli: The Spanish-American War and Military Medicine* (New Brunswick, N.J.: Rutgers University Press, 2004). A commission headed by Walter Reed concluded that in almost all cases, the correct diagnosis was typhoid fever.

31. Henry Grafton Clark, *Ship Fever, So Called; Its History, Nature, and Best Treatment* (Boston: Ticknor, Reed and Fields, 1850), p. 1.

32. A. B. Hawke to Sir, 16 October 1847, *British Parliamentary Papers, Correspondence and Other Papers Relating to Canada and to Immigration in the Provinces, 1847–48*, orig. vol. 47, new vol. 17 (rpt. Shannon, Ire.: Irish University Press, 1969), pp. 19–20. Daniel Drake described the Canadian epidemic in “The Irish Immigrants’ Fever,” *Boston Med. & Surg. J.*, 1847, 37: 149–57.

33. Arthur L. Gelston and Thomas C. Jones, “Typhus Fever: Report of an Epidemic in New York City in 1847,” *J. Infect. Dis.*, 1977, 136: 813–21. See also “Typhus Fever in New York,” *Boston Med. & Surg. J.*, 1848, 38: 66; John H. Griscom, “Summary of, and Observations upon, the Medical Practice of the New York Hospital, in the Months of July, August, and September, 1847,” *New York J. Med.*, 1847, 9: 347–54.

34. *Report of the Select Committee of the Senate of the United States on the Sickness and Mortality on Board Emigrant Ships* (Washington, D.C.: Beverley Tucker, 1854), p. 55.

35. E. Harold Hinman, “History of Typhus Fever in Louisiana,” *Amer. J. Pub. Health*, 1936, 26: 1117–24.

A similar pattern occurred in New York City in the 1860s, where a typhus outbreak sent Stephen Smith to the slums to investigate. He found abundant typhus among the miserable immigrant Irish.³⁶ Smith described a filthy hovel, with “every available place, from cellar to garret, . . . crowded with immigrants—men, women, and children. The whole establishment was reeking with filth, and the atmosphere was heavy with the sickening odor of the deadly typhus, which reigned supreme in every room.”³⁷ New York City inspectors found 2,000 cases during this outbreak in the New York slums—an impressive number, to be sure, but not next to the estimate that 500,000 potential victims lived in those crowded, filthy tenements. Other accounts detail the fact that most of the cases were new arrivals, or had had close contact with new arrivals.³⁸ A similar pattern prevailed in the slums of Philadelphia during the decade. Typhus does not seem to have “caught fire” even here, where the spark was repeatedly lit under ready tinder.³⁹

Nor did it appear in the most likely sites during the Civil War, in spite of the concurrent cases in New York and Philadelphia, and in spite of the abundant possibility of Brill-Zinsser recrudescence among the Irish immigrants who had been infected earlier. Even while recognizing the difficulties of retrospective diagnosis in the case of typhus, the historian can still be impressed at its absence during the war. As Dr. Charles Smart noted in *The Medical and Surgical History of the Civil War*, “the experience of other armies shows definitely that if the contagion of typhus had gained access to our camps, no search of the records of individual cases would have been required to substantiate the fact. The death-roll of our medical officers and hospital nurses would have been a sufficient demonstration.”⁴⁰

During the war Joseph Jones, a Confederate physician who wrote prodigiously on the subject of gunshot wounds and infectious diseases, deliberately searched for typhus. He reported in 1867:

During the recent civil war I sought for typhus fever amongst the Confederate troops serving in the field, and amongst the general hospitals in various parts of the Confederate States; thousands of sick and wounded were examined with

36. George Rosen, “Tenements and Typhus in New York City, 1840–1875,” *Amer. J. Pub. Health*, 1972, 62: 590–93.

37. Stephen Smith, *The City That Was* (New York, 1911; rpt. Metuchen, N.J.: Scarecrow Reprint, 1973), p. 36.

38. *Ibid.*, pp. 88–113.

39. V[ictor] C. Vaughan, “History and Epidemiology of Typhus Fever,” *JAMA*, 1915, 64: 1805–10.

40. Smart, *Medical and Surgical History* (n. 1), 5: 324.

a view to the determination of the existence or non-existence of this disease amongst the Confederate armies; and even the prisoners confined upon Belle Isle, in the Libby Prison, and in Castle Thunder, in Richmond, Va., were not neglected in these examinations, and numerous medical officers of the Confederate army were interrogated upon this subject personally and by letter. No case of true typhus fever came under my observation during the war in any army, in any field hospital, general hospital, or military prison.⁴¹

He went on to say that cases reported as typhus turned out to be typhoid on closer inspection. Jones reviewed the medical situation at Andersonville Prison for the Confederate government, and continued his search for typhus there.⁴² “I supposed that if typhus fever existed anywhere in the Confederate States it would be found at Andersonville,” he wrote, “and especially amongst the foreign element of the Federal armies, which had been but recently imported from the bogs of Ireland, and from the hovels of the densely populated European countries.”⁴³ But it was not there.

Jones and others drew an important conclusion from this absence:

This great experiment of Andersonville, perhaps the greatest and most remarkable of modern times, strongly sustains the view that typhus and typhoid fevers are dependent upon the action of special poisons, the conditions for the origin and action of which are as definite and as limited as in the case of the poisons of smallpox and measles. It would appear from the results of the experiment at Andersonville, as well as from the large number of well established facts presented during the course of the present inquiry, that neither typhoid or typhus fevers can be generated by animal exhalations from putrefying excrements or bodies; but that these diseases are propagated by a special poison emitted by the living body, either directly or through the excretions and secretions.⁴⁴

The absence of major typhus outbreaks drove the evolving theories of infectious diseases toward belief in the specificity of causation, paving the way for the specificity of bacteriology.⁴⁵

41. Joseph Jones, “Investigations upon the Diseases of the Federal Prisoners Confined in Camp Sumpter, Andersonville, Ga.,” in *Contributions Relating to the Causation and Prevention of Disease and to Camp Diseases*, ed. Austin Flint (New York: Published for the U.S. Sanitary Commission by Hurd and Houghton, 1867), pp. 469–655, quotation on p. 600.

42. On Andersonville, see William Marvel, *Andersonville: The Last Depot* (Chapel Hill: University of North Carolina Press, 1994).

43. Jones, “Investigations” (n. 41), pp. 600–601.

44. *Ibid.*, p. 643.

45. Roberts Bartholow, “Camp Fevers—Remittent, Typhoid, Typho-malarial or Malarial Typhoid, Common Continued, and Mountain Fever,” in Flint, *Contributions* (n. 41), pp. 195–217; K. Codell Carter, *The Rise of Causal Concepts of Disease* (Aldershot: Ashgate, 2003).

There were some typhus cases listed in the official records, it is true: the *Medical and Surgical History* recorded 2,624 cases and 958 deaths among Union troops. Commentators at the time questioned these statistics, however, and noted that the diagnosis became more rare as the war went on and physicians acquired more experience. It is likely that there were some isolated cases of Brill-Zinsser disease, and perhaps a few limited outbreaks stemming from them, so some of the reported cases may have been true typhus. But contemporary commentators believed that most of these cases were typhoid or other infections, including meningococcal meningitis.⁴⁶

The Wilmington Epidemic

Some historians have argued that an epidemic that erupted in Wilmington, North Carolina, during the last months of the war, was typhus. If it was, this fact significantly challenges my argument.⁴⁷ The epidemic occurred shortly after Wilmington had fallen to (or been liberated by) Federal troops on 22 February 1865. Seeing defeat near, with General Sherman's army pushing into central North Carolina, the Confederates decided to close the Salisbury, North Carolina, prison camp, and send the Union prisoners to (now Federal) Wilmington. The prisoners began to arrive in late February, and over the next month some 8,600 moved through the city. The men were starving, near-naked, and covered in lice. "Language would utterly fail to describe their condition," the commanding general who received them told a representative of the U.S. Sanitary Commission; in the men "filth, rags, nakedness, starvation, were personified," and many were described as idiots, unable to tell their names.⁴⁸ Others had suffered so much gangrene in their feet, secondary to frostbite, that they crawled into camp on their hands and knees. "Their condition [was] that of men who have for months suffered from chronic starvation. Their arms and legs look like coarse reeds with bulbous joints. Their faces look as though a skilful taxidermist had drawn tanned skin over the bare skull, and then placed false eyes in the orbital cavities."⁴⁹ One man who

46. Smart, *Medical and Surgical History* (n. 1), 5: 268–69, 324–25.

47. Paul E. Steiner, *Disease in the Civil War: Natural Biological Warfare in 1861–1865* (Springfield, Ill.: Thomas, 1968), p. 15.

48. C. R. Agnew, "Preliminary Report of the Operations of the U.S. Sanitary Commission in North Carolina, March, 1865. Sanitary Commission Report # 87," in *Documents of the U.S. Sanitary Commission*, nos. 61–95, 2 vols. (New York: U.S. Sanitary Commission, 1866), 2: 7.

49. *Ibid.* *The Herald of the Union*, a federally run Wilmington newspaper that first appeared on 28 February 1865, likewise reported on the pitiful condition of the released prisoners.

had weighed about 180 pounds upon capture in October 1864 weighed in at 87 pounds when he arrived in Wilmington on 2 March 1865.⁵⁰ Although as many ex-prisoners as could travel were packed off directly to the north on steamers, some three to four thousand remained in Wilmington, too sick to go further. In this setting, with hospitals sprouting up all over town in houses, churches, and other public buildings, a deadly fever broke out.

This highly contagious fever killed at least three hundred soldiers, and caused a high mortality among refugee blacks living in a large camp nearby. Many medical officers contracted the fever, along with chaplains and other attendants, and many of these men died as well. Even people with slight contact, such as attendants during travel, came down with the disease. The fever had a multitude of symptoms, including myalgias, diarrhea, suppressed urine, slow pulse, brain and lung congestion, and a red rash; severe jaundice was frequent.⁵¹ A Minnesota surgeon, Daniel Hand, who was in charge of the medical department of North Carolina in the last years of the war, was sure the disease was typhus: "The condition of affairs was worse than had been reported," he revealed in his memoirs two decades later; "The number of deaths daily was frightful, and every house in town had crape on the windows. The fever was violently contagious and was typhus. It had been engendered in the filthy prison-pen at Salisbury, was brought down by the released prisoners, and by them communicated."⁵² Hand also sent dispatches to the Army medical authorities while the epidemic was going on, and his reports found their way into Charles Smart's *Medical and Surgical History*.⁵³ Although Hand was from Minnesota, where he would have seen little typhus, he had trained in Philadelphia, where he may have had opportunities to study the disease.⁵⁴

The fever thus had many of the characteristics of typhus. Certainly there were lice in abundance; although some men came into Union lines

The paper says little about the subsequent epidemic, although there are references to military orders regarding street-cleaning crews, and demands that homeowners clean up their grounds, which suggests that the tool of sanitation was being brought to bear against an epidemic enemy. See editions of 2 March, 13 March, 17 March, 21 March, and 1 April 1865.

50. B. F. Booth, *Dark Days of the Rebellion, or Life in Southern Military Prisons: Libby and Salisbury, 1864–1865* (Indianola, Iowa: Booth, 1897), pp. 327–28.

51. Smart, *Medical and Surgical History* (n. 1), 5: 331–32.

52. Daniel Hand, "Reminiscences of an Army Surgeon," *Glimpses of the Nation's Struggle*, 1887, 1: 276–307, quotation on p. 305.

53. Smart, *Medical and Surgical History* (n. 1), 5: 331–32.

54. "Daniel W. Hand, M.D.," *Trans. Minn. State Med. Soc.*, 1889, 21: 234–39. My thanks to Leonard Wilson for drawing my attention to this obituary and providing other information about Hand and continued fever diagnosis in this era.

naked, what clothing they had was “ragged, filthy and full of vermin,” and “[w]ith the great majority of the feebler ones, personal cleanliness was a thing which they appeared to have entirely forgotten.”⁵⁵ This is just the sort of population within which typhus traditionally prevails. Yet other physicians had their doubts. Smart himself began his discussion of the Wilmington epidemic by saying that “there appears strong ground for believing” that the infection was typhus, not that it was.⁵⁶ And he cited another opinion, a physician who noted that “a form of low fever with eruption prevails among the recent arrivals from General Sherman’s troops. It is unusually fatal, and though differing somewhat from true typhus, bears more resemblance to it in its essential features than any other fever I have met with.”⁵⁷ It is possible that this was typhus, perhaps with enough other diseases mixed in to confuse the diagnostic picture. These men were no doubt concomitantly suffering from scurvy, chronic dysentery, and other forms of malnutrition, which may have altered their presentation. But it is odd that jaundice was a prominent symptom of this outbreak: jaundice is not associated with typhus. Other fevers that feature jaundice—such as yellow fever, hepatitis, and malaria—are less likely here: it was the wrong time of year for mosquito-borne diseases, and physicians at that time would have been quite familiar with those symptoms. Hepatitis A is the only form of hepatitis that spreads via common sources such as food or drink, but it is not likely to be so contagious or so fatal.

One possibility is that this was an epidemic of relapsing fever, an infection also spread by lice and producing symptoms similar to typhus. Relapsing fever often traveled with typhus, and indeed was a major source of comorbidity both among the Irish and among Irish immigrants. So common was relapsing fever with its accompanying jaundice in Ireland that it was called *fiabhras buidhe*, “yellow fever,” and was contrasted with its common companion, *fiabhras dubh*, “black fever” or typhus.⁵⁸ The symptoms match fairly well, with the exception that relapses were not noted as a prominent feature of the Wilmington outbreak—this is not a fatal flaw, however, for relapses are not an inevitable part of the disease, and many of the victims were in such a weakened condition that they may have died before reaching the relapse point. A more troubling critique lies in the question of where the disease came from, if it was relapsing fever.

55. Agnew, “Preliminary Report” (n. 48), p. 11.

56. Smart, *Medical and Surgical History* (n. 1), 5: 331–32.

57. George Lyman, quoted in *ibid.*, 5: 333–34.

58. William P. Macarthur, “Medical History of the Famine,” in *The Great Famine: Studies in Irish History, 1845–52*, ed. I. R. Dudley and T. Desmond Williams (New York: New York University Press, 1957), pp. 263–315.

It is unknown where relapsing fever hides out between outbreaks; two mechanisms for latency—an animal/arthropod cycle, or a shingles-like recrudescence—have been postulated but not proven.⁵⁹ If relapsing fever hides in prior victims as typhus does, then among the many Irish soldiers of the Union army there might have been one recrudescence to spark this epidemic. If so, then the Wilmington epidemic would mark the only major indigenous outbreak of this disease in North America. In any event, the widespread jaundice makes it unlikely that this was typhus.

Typhus after the Civil War

After the war, typhus continued to appear in the sort of immigrant-related outbreaks that had typified its antebellum courses. Northern port cities saw limited typhus epidemics in the early 1880s, but it was only in the 1890s that it became a major cause of panic.⁶⁰ Russian Jewish immigrants brought the disease to New York City in 1892, and the Metropolitan Board of Health mounted a rapid response, isolating the sick and potentially sick on North Brother Island. Howard Markel said of this episode: “The 1892 epidemic in New York City was especially remarkable because almost every case of typhus fever, with the exception of some medical attendants, police guards, and close neighbors, occurred among newly arrived Russian Jewish immigrants who had traveled on the same steamship . . . [and were housed] in eight boarding houses on the Lower East Side.”⁶¹ This had become the typical pattern for typhus in the United States, although public health officials of course took credit for containing the disease: “Typhus fever . . . developed in the early part of February to an extent which would, without doubt, have resulted in an epidemic, had not the Medical Inspectors of this Department . . . succeeded in stamping it out by a system of well-directed efforts,” crowed the annual report of the Board of Health of New York City.⁶²

59. Willy Burgdorfer, “The Epidemiology of Relapsing Fevers,” in *The Biology of Parasitic Spirochetes*, ed. Russell C. Johnson (New York: Academic Press, 1976), pp. 191–200; A. D. M. Bryceson et al., “Louse Borne Relapsing Fever: A Clinical and Laboratory Study of 62 Cases in Ethiopia and a Reconsideration of the Literature,” *Quart. J. Med.*, 1970, 39: 129–70. I thank Powel Kazanjian, University of Michigan, for his thoughts on this question.

60. Thomas McCrae, “Typhus Fever,” in *Modern Medicine, Its Theory and Practice*, ed. William Osler (Philadelphia: Lea, 1907), pp. 231–44.

61. Howard Markel, *Quarantine! East European Jewish Immigrants and the New York City Epidemics of 1892* (Baltimore: Johns Hopkins University Press, 1997), p. 16.

62. *Annual Report of the Board of Health of the Health Department of the City of New York for the Year Ending December 31, 1892* (New York: Brown, 1894), p. 30.

Another typhus panic took hold in 1916, when health authorities feared that Mexican laborers would import the typhus then raging in Mexico. Many immigrants were infested with lice, and the rate of immigration from Mexico was increasing. A popular border-crossing point was El Paso, where not only immigrants came through, but also hundreds of laborers who worked in El Paso during the day and returned to Mexico at night. The U.S. Public Health Service set up a cumbersome quarantine and disinfection system, including dousing immigrants with gasoline to kill lice. After one crowd of twenty-six detainees burned to death when a lit cigarette came too near, riots broke out at the border crossing. A less onerous system of cleansing and inspection was put in place, and life returned to normal.⁶³

Mexican typhus did cross the border in November 1920, causing an epidemic that continued until June of the next year. The location was a Navaho reservation that included parts of New Mexico, Arizona, and Utah. USPHS investigator Charles Armstrong found sixty-three cases resulting in twenty-seven deaths in this nomadic population. No overt disease contact with Mexico was identified, although Armstrong noted that a pathway for itinerant Mexican laborers crossed the reservation. He observed that the Navaho bathed rarely, because water was scarce: "Left to themselves, the Indians of the San Juan Reservation are 90 to 100 per cent infested with vermin, usually both head and body lice being found."⁶⁴ Because the Navaho believed that lice were a natural body product that came from inside the body, Armstrong had trouble convincing them of the value of delousing techniques; only when he told them that someone evil from Mexico had brought bad Mexican lice to make them sick did the community cooperate.

Explaining the American Typhus Pattern

There is no obvious answer to the peculiar pattern of typhus in America north of the Rio Grande. Perhaps it was simple luck, and no other explanation is necessary. Hans Zinsser noted that typhus declined markedly in Europe during the last half of the nineteenth century, for no apparent reason, before emerging again in World War I. Even then, the disease was limited to the eastern front, and was not a problem in France: "Among

63. Howard Markel, *When Germs Travel: Six Major Epidemics That Have Invaded America since 1900 and the Fears They Have Unleashed* (New York: Pantheon Books, 2004), pp. 111–40.

64. Charles Armstrong, "Typhus Fever on the San Juan Indian Reservation, 1920 and 1921," *Pub. Health Rep.*, 1922, 37: 685–93, quotation on p. 685.

the most remarkable phenomena of the war is the total absence of typhus from the Western front. No completely satisfactory explanation for this can be offered. Soldiers in the trenches on this front were as universally lousy as soldiers have always been.”⁶⁵

Whatever is concluded about the Wilmington epidemic, the absence of major typhus epidemics elsewhere in the Civil War particularly calls for explanation. There is every indication that the typhus organisms were present and ready to spark an epidemic. Perhaps there were not enough lice? This seems unlikely. Lice were common companions of Civil War soldiers, both north and south. One commentator described the Confederates at the Union prison camp at Alton, Illinois, in words that leave no doubt as to their fitness for a typhus outbreak: “Prisoners are permitted to lounge about in their filth, with no other duty to perform seemingly than to amuse themselves by slaughtering the vermin crawling about their filthy persons. This seems to be their general avocation and amusement.”⁶⁶ Opportunities for laundry and personal bathing were few. The population density inside prisons was tight, making it easy for lice to move from body to body. The Civil War soldier was often malnourished, as evidenced by the frequent reports of scurvy among the troops, and the prisoners in camps were even worse off, with foul living conditions and frank starvation as their lot.⁶⁷ It is hard to make an argument that the environment prevented disease transmission.

Another hypothesis to be considered is that clothing during the Civil War, and in America more generally, differed from that in Europe, and that this difference was significant. What if, for example, lice preferred wool over cotton, and more Americans than Europeans were wearing cotton undergarments or shirts? Body lice tend to live in the folds and seams of clothing, traveling to the skin to dine on blood but otherwise staying on the fabric, and indeed it turns out that they do not like all fabric types equally: one researcher actually studied this, and found that lice preferred wool, cotton, and linen, but disliked silk.⁶⁸ In fact, visitors to Mexico during the 1910s typhus outbreak were advised to wear silk underclothes to avoid infection.⁶⁹ But few Civil War soldiers or other louse-ridden Ameri-

65. Hans Zinsser, *Rats, Lice and History: A Bacteriologist's Classic History of Mankind's Epic Struggle to Conquer the Scourge of Typhus* (Boston: Little, Brown, 1934), pp. 290–92, 298, quotation on p. 298.

66. Dr. Keeney quoted in Smart, *Medical and Surgical History* (n. 1), 5: 51.

67. Lonnie R. Speer, *Portals to Hell: Military Prisons of the Civil War* (Mechanicsburg, Pa.: Stackpole Books, 1997).

68. Buxton, *Louse* (n. 10), p. 43.

69. Markel, *When Germs Travel* (n. 63), p. 117.

cans would have had the resources for silk underwear, and the louse is equally at home in wool, cotton, or linen. Nothing about the conditions during the Civil War appears to explain the scarcity of typhus.

Could it be that Americans were already immune to typhus when European immigrants brought it to their doors? The cross-immunity generated by murine typhus makes this at least a reasonable question. But investigations of murine typhus indicate that it is not a disorder likely to achieve high levels of penetrance in a population. In Mexico City some 17 percent of the population tested positive for *R. typhi* in 2000, and one could argue that the exposure to rats and their fleas in this population is analogous to the slum experiences of nineteenth-century American cities.⁷⁰ Remember, too, that many slum dwellers lived near those of similar ethnicity. If the incoming Irish of the 1860s settled in Irish neighborhoods, some proportion of their contacts would have lived through typhus in Ireland or on the way over. Twenty-five percent of New York's population in 1860 was born in Ireland.⁷¹ These immunities may well have hindered the spread of typhus outside the immediate immigrant circle—but it is hard to see this as having a major effect on situations such as the Civil War prison camp.

One possible explanation of the American typhus pattern is that the American louse was a less efficient disease vector than its European counterpart. If typhus remained largely a problem of recent immigrants and their immediate contacts, it might be because the disease depended on European lice imported upon the immigrant's body and clothing. As those lice died of typhus (and typhus is deadly to the louse), the disease chain was quickly broken. Now for this theory to hold water, it would be helpful if entomologists distinguished American lice from European or Asian or African lice—but they draw no such distinction. The human body louse is the human body louse the world over, according to accepted classification schemes.⁷²

The possibility that entomologists may have overlooked such a differentiation is not particularly far-fetched. In the 1930s Lewis Hackett wondered why certain areas of Europe suffered from malaria while certain other areas, with abundant anopheles mosquito populations, appeared

70. Acuna-Soto et al., "Murine Typhus" (n. 14).

71. Alan M. Kraut, *Silent Travelers: Germs, Genes, and the "Immigrant Menace"* (New York: Basic Books, 1994), p. 34.

72. Ke Cheung Kim, Harry D. Pratt, and Chester J. Stojanovich, *The Sucking Lice of North America: An Illustrated Manual for Identification* (University Park, Pa.: Pennsylvania State University Press, 1986); Joanna Ibarra, "Lice (Anoplura)," in *Medical Insects and Arachnids*, ed. Richard P. Lane and Roger W. Crosskey (London: Chapman and Hall, 1993), pp. 517–28.

immune. No one had asked the question in quite that way before, so no one had discovered the heretofore hidden differences in behavior and anatomy that led Hackett to identify clear subspecies within the anopheles population. It turned out that one subspecies liked human blood and the other preferred animals: no human bites, no human malaria.⁷³ A similar situation may exist here, of subtle subspeciation among lice that has rendered the American louse an inefficient vector for typhus, and has thus spared the North American continent from the worst ravages of this dread disease.

Is there any evidence that the New World louse is indeed a less efficient typhus carrier? This is not the sort of question that attracts much modern attention, especially among American medical scientists. But there is one intriguing account from the 1920s that supports my hypothesis that American lice were different. In 1920 S. Burt Wolbach headed a Red Cross commission charged with studying a typhus outbreak in Poland.⁷⁴ Wolbach's group wanted to expand on work done by other scientists that suggested that typhus was indeed caused by *Rickettsia prowazekii* and was transmitted by the human body louse. In order to be sure that the lice used in the investigation were uninfected, Wolbach brought North American lice with him; he acquired these in Montreal, and kept them alive on his body during the transatlantic voyage in boxes strapped to his legs.⁷⁵

Wolbach was familiar with research done by Henrique da Rocha-Lima four years earlier in Germany, and he and da Rocha-Lima followed a similar research protocol. They filled small flat boxes, about the size of a pack of cigarettes, with lice, and covered the side with very fine gauze. The lice could feed through the gauze, but could not escape. After keeping the lice alive on healthy humans, including themselves, they let them go hungry for a day or two and then applied the lice box to a patient with typhus. After the lice had fed on the typhus patient, they were later used to infect guinea pigs. The lice were then dissected and examined for organisms via serial slices, or by smearing the contents of the gastrointestinal tract on a slide. Da Rocha-Lima, using lice acquired in Germany, succeeded in infect-

73. L. W. Hackett, *Malaria in Europe: An Ecological Study* (London: Oxford University Press, 1937).

74. On the typhus outbreak in Poland and the American response, see Alfred E. Cornobise, *Typhus and Doughboys: The American Polish Typhus Relief Expedition, 1919–1921* (Newark: University of Delaware Press, 1982); Gaines M. Foster, "Typhus Disaster in the Wake of War: The American-Polish Relief Expedition, 1919–1920," *Bull. Hist. Med.*, 1981, 55: 221–32.

75. S. Burt Wolbach, John L. Todd, and Francis W. Palfrey, *The Etiology and Pathology of Typhus: Being the Main Report of the Typhus Research Commission of the League of Red Cross Societies to Poland* (Cambridge: League of Red Cross Societies at the Harvard University Press, 1922).

ing almost all of them after applying them to typhus patients.⁷⁶ Wolbach, on the contrary, achieved much lower infection rates in his lice:

We have found it to be more difficult to infect lice with *Rickettsia prowazeki* [sic] than is apparently indicated in the accounts of da Rocha-Lima (1916, p. 29). Even in heavily infected boxes [of lice] in the later half of our work there was always a varying percentage of lice in which rickettsia could not be demonstrated, and in Box LII not one of the twenty-one lice recovered and examined, twelve by serial sections, nine by smears, showed rickettsia.⁷⁷

Wolbach confessed his bewilderment at this phenomenon.

It is thus possible that there exists a subspecies of human body louse common to the United States and Canada that is subtly different from that of Europe (and Mexico). One suspects that the difference was not absolute, but that it may have been significant enough that the chain of human-to-human typhus transmission was much more easily broken in the United States and Canada than in Europe, Latin America, and Africa. It is otherwise hard to explain the lack of typhus epidemics in locales so favorable for its propagation.

Epilogue

That might be the end of the story were it not for a curious discovery reported in 1975: *R. prowazekii* had been found in a flying-squirrel species that lives east of the Mississippi River, in a range from Maine to Florida.⁷⁸ In the United States from 1976 to 2001, “a total of 39 human *R. prowazekii* infections were documented in persons with no reported contact with body lice,” and nearly all of these were in the eastern part of the country. In approximately one-third of the cases, contact with flying squirrels or their habitats was reported.⁷⁹ In a typical case, such as one recorded in February 2002, a forty-four-year-old man in West Virginia who entered the emergency room with headache, fever, and chills had spent several nights in a hunting cabin recently infested by flying squirrels; he had not

76. H. da Rocha-Lima, “Beobachtungen bei Flecktyphusläusen,” *Archiv für Schiffs- und Tropen-Hygiene*, 1916, 20: 17–31; da Rocha-Lima, “On the Etiology of Typhus Fever,” in *Selected Papers on the Pathogenic Rickettsiae*, ed. Nicholas Hahon (Cambridge: Harvard University Press, 1968), pp. 74–78.

77. Wolbach, Todd, and Palfrey, *Etiology* (n. 75), p. 49.

78. F. M. Bozeman et al., “Epidemic Typhus *Rickettsiae* Isolated from Flying Squirrels,” *Nature*, 1975, 255: 545–47.

79. Mary G. Reynolds et al., “Flying Squirrel-Associated Typhus, United States,” *Emerg. Infect. Dis.*, 2003, 9: 1341–43, quotation on p. 1341.

seen any squirrels, but had cleaned up nests, feces, and other debris.⁸⁰ It is not clear how the infection moves from squirrels to humans, although flea bites and the aspiration of flea feces have been suspected. How and when this organism moved into the flying-squirrel population is at present unknown. If it was seeded by the urban epidemics of the nineteenth century, it is odd that such rural creatures should provide the reservoir.⁸¹

In early June 1999, a man walking along the beach on Padre Island off the coast of Texas splashed through an eddy pool filled with brackish water; sand flies assaulted his ankles, leaving itchy red bumps. Ten days later, now back home in Zuni, New Mexico, he developed a fever, stiff neck, photophobia, and abdominal pain. His physicians quickly suspected meningitis, performed a lumbar puncture, and delivered empiric antibiotics while awaiting results. The laboratory identified no conventional bacteria, but finally serum titers came back suggestive of murine typhus, and proper therapy was begun. He recovered completely. Murine typhus is found occasionally in Texas, so this case was not entirely surprising—but samples of cerebral spinal fluid went off to the CDC, where PCR amplification revealed a surprising result: this southwestern American male, with no exposure to flying squirrels, rodents, lice, or any other of the usual suspects, had just survived epidemic typhus meningitis. The genetic determination left no doubt that his organism was *R. prowazekii*.⁸² Where did it come from? Is there another indigenous reservoir of epidemic typhus fever, as yet unrecognized? If so, why does the organism emerge so rarely? Typhus remains a puzzling disease, and its geographic distribution continues to stymie easy explanation.



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80. Ibid.

81. See also Cecilia G. Moron et al., "Phylogenetic Analysis of the *ROMPB* Genes of *Rickettsia Felis* and *Rickettsia Prowazekii* European-Human and North American Flying-Squirrel Strains," *Amer. J. Trop. Med. Hygiene*, 2000, 62: 598–603.

82. Robert F. Massung et al., "Epidemic Typhus Meningitis in the Southwestern United States," *Clin. Infect. Dis.*, 2001, 32: 979–82.