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### **Cholera and the Environment in Nineteenth-Century Japan**

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#### **Abstract**

Among acute infectious diseases, cholera gained more attention in Japanese popular culture and among policymakers than any other. Although many facts about cholera's historical existence seem settled, when one looks closely at the medical, biological, and environmental science, several of these facts—especially about the definitions of pandemics, epidemics, and endemicity—become fuzzy. In an attempt to reach a modicum of clarity, this article asks: What biological and environmental conditions allowed cholera to become endemic in nineteenth-century Japan? In answering this question, it makes the case that understanding this disease in history and, more specifically, why it took the course that it did in nineteenth-century Japan requires knowledge of the scientific literature. This claim is part of a broader argument about how understanding the physical world in its concrete manifestations through biological and environmental science is important to understanding human history. It is applicable not only to the history of disease, epidemics, and pandemics but to other fields, including environmental history, the history of technology, and the history of war, as well. This article also questions a number of boundaries: between disciplines, across time and space, and between evidence and assumption in what we consider facts.

**Keywords:** Japan, disease, cholera, pandemic, epidemic, endemic, environment, history, science, biology, historiography of cholera, GIS analysis

#### **Introduction: Cholera, Science, and History**

More than any other acute infectious disease, cholera was a defining element of the nineteenth century. This was as true for those living on the Japanese archipelago as for other people across the globe. Cholera's pandemics are established as historical facts, but how its worldwide spread depended on environmental forces is less known. This article examines the role those forces played on the course that the disease took in Japan during the nineteenth century. It begins with the epidemic waves that then established cholera's endemicity, punctuated by later epidemics that arose autochthonously. After that, cholera remained endemic but insignificant in terms of mortality rates throughout the twentieth century. This article asks: What biological and environmental conditions allowed cholera to become endemic? The answer to this question can help us understand why the disease unfolded in time

and space as it did across the Japanese archipelago. The answer also can suggest explanations why cholera in Japan quickly declined during the early twentieth century, although space limitations here prevent a full exploration of this question.

The medical and biological sciences related to cholera open other useful epistemological windows. They help us empathize with the experiences that individuals and populations had with this disease. Its signs and symptoms all carried stigma and other cultural baggage that were particular to a time and place. Yet the effects of the disease on the human body—in particular, when left untreated with modern medicine—were and remain more similar than different across time and space. Clearly, the Japanese experience with cholera resembled other individual and national experiences with the disease. Those commonalities become apparent when looking at the physical realities of the disease and of the organism that causes it, *Vibrio cholerae*, in both the body and the environment. In short, a clear understanding of cholera's historical trajectory in a specific geographic location requires more than archival sleuthing and a close reading of contemporary observations. The sciences can help.

More broadly, this is yet another call to bridge the “two cultures” that C. P. Snow discussed in the Rede Lecture at the University of Cambridge in 1959 and later published in *The Two Cultures and the Scientific Revolution* (Snow 2008). Although *The Two Cultures* has received its share of criticism, the main ideas still resonate today. Snow, who was both a physicist and novelist, urged that the gap between the culture of scientists and that of humanists be bridged by educating people in each camp about the other. Two years later, Snow presented his ideas on how a “third culture” can exist through genuine interdisciplinarity (Fins 2010). Hence, although this article focuses on issues regarding both epidemic—indeed pandemic—and endemic disease, it is part of a broader argument about how understanding the physical world in its concrete manifestations through biology and environmental science is important to understanding human history. It is applicable to not only the history of disease, epidemics, and pandemics but also other fields including environmental history, the history of technology, and the history of war.

The literature on the history of cholera is extensive, yet little has been written in which the biological and environmental sciences of the disease have any role. This is as true in Japan as for the rest of the world. In order to properly examine the relevant science about cholera, it is useful to take a look at how previous historians have understood this disease. With the goal of positioning this article in the context of the literature that has preceded it, without disrupting the article, the Appendix: The Historiography of Cholera at the end of the article offers a brief discussion of some of the major works on the history of cholera, especially in Japan.

### **The Emergence of Pandemic Cholera**

A disease with symptoms much like those of cholera as we define it today seems to have existed in South Asia at least since the sixteenth century and possibly since ancient times (MacPherson 1884; Pollitzer 1954; Colwell 1996, 2025; Barua 1992, 1–7). Yet it did not, at least in its virulent form, spread beyond the Bay of Bengal until 1817. Genetic analysis suggests that this virulent form of *Vibrio cholerae* might

not have existed long before then (Devault et al. 2014). Previously, “cholera,” often referred to as “cholera morbus,” signified a set of bilious symptoms that usually included diarrhea; this was true in most European languages (Sydenham and Rush, 1809). After this new affliction appeared, “cholera morbus” was usually associated with the peculiar signs and symptoms of the pre-1817 affliction, and “Asiatic cholera,” leaving aside the question of whether it actually originated in Asia, designated the new disease (Speck 1993, 642–643). Nevertheless, throughout much of the nineteenth century “cholera” and “cholera morbus” were sometimes used interchangeably with “Asiatic cholera” both in Japan and across the globe (Nagayo 1877, ii–iii). This was not unusual. Until the twentieth century when nosologies and nosographies became fairly standardized, disease names were frequently used inconsistently. Yet many, if not all, observers at the time saw this as a new disease or at least one that they believed was previously limited to the Indian subcontinent (Whitney 1833, 307). “Asiatic cholera” was the pre-1817 cholera morbus on steroids. Whereas cholera morbus was tedious, sometimes terrible, and infrequently deadly, “Asiatic cholera” tended to be lethal. Left untreated, it could kill more than 70 percent of those who developed symptoms (Harris et al. 2012, 2469). Most nineteenth-century treatments were ineffective at best. At worst, they were, as one historian of medicine remarked, “a form of benevolent homicide” (Howard-Jones 1972, 373).

This new form of cholera appeared just as a new political entity, the modern nation-state, was emerging globally. Modern political organizations, along with the technologies of biopower and forms of environmental and social control that emerged often in response to cholera, made the disease a driver of modernity. The resulting policies largely subdued the disease in the long run but with certain costs. As recent works such as Anna Lowenhaupt Tsing’s *The Mushroom at the End of the World: On the Possibility of Life in Capitalist Ruins* (2017) and Donna J. Haraway’s *Staying with the Trouble: Making Kin in the Chthulucene* (2016) have shown, the idea that modernity inevitably goes in the direction of enlightened benevolence is illusory. Although modernity has improved the lives of many, if not all, people worldwide, it also has provided new tools of oppression—often in the name of protecting lives. State policies regarding cholera were similarly double-edged. In various countries, the disease caused innumerable deaths, but it also led to the creation of the clean water supplies and waste treatment processes on which capitalistic civilization depends. It also led to quarantine policies in which households whose members suffered from the disease were sealed off from their communities and hospitalization policies in which patients were sent to quarantine hospitals where the patients died more frequently than they recovered. This was very much the case in nineteenth-century Japan—whose biopolitics were often neck and neck with those of contemporary European nation-states.

### **Cholera in Japan and the Question of Endemicity**

The Japanese experience with cholera paralleled that of people in many European and other Western countries. A rich documentary record reveals the course of the disease through the country and the various medical and state-based responses to it. The statistical records for the epidemics that occurred in 1822, 1858–1860, and 1861–1862 are vague. However, by the last quarter of the century, when the country experienced multiple epidemics, national and prefectural records provide separate snapshots of disease patterns.

Before cholera first reached Japan in 1822, Dutch physicians warned their Japanese colleagues that a new and dangerous epidemic disease had spread through its colony in Batavia (Jakarta), so its first appearance in Kyushu was not entirely a surprise (*Bunsei jingo aki tenkō byō koreri morubusu keiken* 1822). Once it did reach Japan, some thought it had come from Korea; others believed the Dutch had brought it from Java (Hirahara 1842; Nagayo 1877, 2–3; Yamamoto 1982, 6–8). Possibly both explanations were correct. For whatever reasons, this epidemic, which started in Kyushu during October of that year, did not go much farther east than Kyoto and for the most part was over by December (Fujikawa 1944, 157–163; Kikuchi 1978, 63; Yamamoto 1982, 8–13). Despite its geographic limitation, this epidemic left an indelible mark on Japanese medical culture and society, especially in the name *mikka korori* (literally, “three-day drop-dead”), and the fact that it was a disease that at least some Japanese physicians described as previously unknown (Hirahara 1842).

Cholera spread widely across the globe during the 1830s, reaching Western Europe and North America, but it did not emerge again in Japan until 1858. Some who had lived through the epidemic of 1822 recognized the disease, and it was once again called *mikka korori* (Shingū, Ōmura, and Shingū 1858). This sobriquet spread widely and remained in national circulation until the end of the century, though names differed depending on locale (Fujikawa 1944, 155–157; Yamamoto 1982, 15). Cholera reached Japan in 1858 when American sailors on board the *U.S.S. Mississippi* brought the disease to Nagasaki. Some scholars have doubted this explanation of this epidemic’s origins in Japan, but there is evidence for it. William F. Gragg, the steward to the *Mississippi*’s surgeon, described an “epidemic form” of diarrhea that occurred two days after the ship left Hong Kong for Nagasaki. Once there, he also noted that the Japanese allowed “some [American] officers” who were sick to come ashore to recover (Gragg 1860, 31–32). The *Mississippi* arrived on June 23, 1858; by mid-July, J. L. C. Pompe van Meerdevort, the Dutch physician in Nagasaki, reported that cholera had spread throughout the city (Yamamoto 1982, 16). From there, the disease moved eastward across the Japanese archipelago. Whereas the previous epidemic had stopped before reaching Edo, this one decimated much of the city and reached into northeastern Honshu. Record-keeping for causes of mortality at this time was loose at best. Nevertheless, deaths from the disease clearly reached into the tens of thousands in Edo alone (Kanagaki 1858; Fujikawa 1944, 165–167; Yamamoto 1982, 19–23). Moreover, although the epidemic stalled late in 1858, it reemerged in the following year (Kikuchi 1978, 453–455; Yamamoto 1982, 24–26). The next epidemics occurred in 1861 and 1862, as contemporary observers widely reported (“Interesting from Japan” 1862; “Affairs in Japan” 1863; Simmons 1880, 4; Fujikawa 1944, 169; Kikuchi 1978, 455–460).

Cholera apparently disappeared from the Japanese archipelago for the next fifteen years. In 1877, it spread across much of China and likely from there to Japan (Simmons 1880, 6–8; Yamamoto 1982, 29). The previous year, the Japanese government had begun to collect national statistics for acute infectious diseases, including cholera; there were cases of typhoid, dysentery, and smallpox reported in 1876, but none for cholera. The following year, more than eight thousand deaths from cholera were reported. Figures in the *Statistical Yearbook of Imperial Japan* report either patients or deaths from the disease every year from 1877 to 1924—oddly, in 1904, 1905, 1915, and 1918, no patients were reported in the national statistics, but several dozen deaths appeared in the official record. Major epidemics, causing more than ten thousand recorded deaths, occurred in 1879, 1882, 1886, 1890, and 1895, after which cholera caused deaths reaching into four figures only during six more years in the twentieth century. All the major epidemics in Japan occurred before 1900.

With the exception of the epidemic that occurred in 1822, cholera in Japan was a disease of the last half of the nineteenth century. Nevertheless, it left a profound mark on popular culture and, in particular, on expressions of fear. Of importance here are the ways those expressions of cholera-induced fear changed between the 1850s and 1860s and the later years of the century. In the epidemics of the 1850s and 1860s, many Japanese associated cholera with traditional demons and evil spirits, as did many people across the globe, despite the fact that widespread reports described how American sailors had brought the 1858 epidemic on board the *U.S.S. Mississippi* to Nagasaki. Many contemporary Japanese knew this, as indicated by the popular name for the disease at the time, *amerika kitsune* (American fox), which provided an interpretation of the disease in Japanese terms—as a fox spirit—while also suggesting a foreign origin (Takahashi 2005, 62–68).

Nevertheless, many others understood the cholera epidemic that swept 1858 Japan in the established terms of epidemic disease without any reference to foreign origins. It was widely believed that the disease was the result of *kami* (unhappy deities), and could be appeased or warded off through prayer, amulets, the action of benevolent spirits, or the use of other time-trusted offerings and methods. What is remarkable is that during its first epidemic years in Japan, people thought of the disease both as a foreign invader and an expression of domestic deities gone rogue—in much the same way they thought of epidemic smallpox and measles. When the next cholera epidemic occurred in 1877, this was no longer the case. Popular imagery depicted this epidemic as originating in China or Korea; many Japanese similarly responded to later epidemics (Padilla 2009, 19, 115; Johnston 2017). This view was based on the idea that cholera epidemics invariably came from abroad and could not have been repeated outbreaks of endemic disease. Nagayo Sensai concurred. In his report on the epidemic of 1877, he wrote that endemic cholera could be found only in India and that epidemic cholera invariably came to Japan either from Java or China (Nagayo 1877, i, iv).

Later government documents offer mixed accounts regarding cholera's endemicity. The anonymously written *Brief Review of the Operations of the Home Department in Connection with the Cholera Epidemic of the 18th Year of Meiji (1885)*

(1886 [?]) presents a historical account of cholera in Japan up to that year. It notes that the disease was epidemic in 1822, 1858–1860, and 1877–1879; strikingly, it elides the well-documented epidemic that occurred in 1862 (*A Brief Review* 1886 [?], 2–3). It then notes that after the disease was absent in 1880 and 1881, it reappeared in 1882 but was absent the subsequent two years. Its next documented outbreak is in 1885, with a note that cholera spread from Nagasaki that year (*A Brief Review* 1886 [?], 15–17). The following year, however, an outbreak that started in Osaka spread throughout the country with a severity that surpassed the epidemic of 1879 (“Causes of the Cholera Epidemic in Japan in 1886” 1888). This later narrative account implies that the disease was reintroduced from abroad multiple times until 1886. As noted earlier, there were cholera patients or deaths every year between 1877 and 1924. Nevertheless, at least until the 1880s, there existed the view not only in the popular media but also among at least some government officials that cholera came from the outside and was not endemic (Padilla 2009, 119–120).

The perceived ambiguity of cholera’s reintroduction raises several questions. Was cholera a disease that traveled around the globe in epidemic waves, transmitted from person to person like smallpox or polio? Or, did other organisms act as vectors for the cholera bacterium in the same way that rodents can harbor the plague bacterium? Was it possible that the disease-causing organism could remain in the environment but invisible to humans? These are key questions not only in historical perspective but also in considering how best to control this disease. It is helpful to examine the recent biological, environmental, and epidemiological science that answers these questions and apply it to the case of nineteenth-century Japan. First, a brief review of cholera’s global spread and its multiple pandemics is useful for putting the Japanese experience into a wider perspective.

### **A Difficulty with Pandemics: Fuzzy Borders**

Cholera pandemics and modern medical science and technology arose at the same time. As previously noted, the disease apparently first spread beyond the Bay of Bengal in 1817. That was the year after French physician René-Théophile-Hyacinthe Laennec invented the stethoscope. During the next few decades, cholera became a global scourge, and by the end of the century it was as widespread as the stethoscope. The disease’s epidemics coincided with the rise of cell theory, anesthetics, asepsis, bacteriology, and other cornerstones of modern medicine. According to many histories of epidemiology, British physician John Snow’s demonstration in 1854 that cholera was definitely a water-borne disease was foundational to the field. That same year, Filippo Pacini, an Italian anatomist, discovered the cholera bacterium, which he called *Vibrio* because of its vibrating motion (Bentivoglio and Pacini 1995). Pacini’s discovery, however, was neglected until after German physician and microbiologist Heinrich Hermann Robert Koch rediscovered the bacterium nearly thirty years later, something contemporaries considered a milestone in the history of bacteriology (*British Medical Journal* 1884). Nevertheless, much about the epidemiology and transmission of cholera remained obscure until well into the twentieth century.

Epidemiologists have long considered cholera a quintessentially pandemic disease. Although cholera first spread from India to other parts of the globe in 1817, reaching Japan in 1822, it took another decade to reach Western Europe and North America. Most historians and epidemiologists concur that 1817 marked the beginning of seven cholera pandemics, the last of which most consider as ongoing. Many have disagreed over exactly *when* those pandemics occurred, although most agree that six of those seven either occurred or started during the nineteenth century. There has been considerable disagreement regarding the beginning and end points of the pandemics. J. N. Hays, a historian who has written on the global impact of epidemics and pandemics, dates the seven pandemics as 1817–1824, 1827–1835, 1839–1856, 1863–1875, 1881–1896, 1899–1923, and 1961 to the present (Hays 2005). Notably, Hays does not include 1858, the year of Japan’s second cholera epidemic.

The history of cholera written in 1992 by Dhiman Barua, a physician from Bangladesh who specialized in cholera control, lists six different sets of dates for cholera pandemics. Barua’s history uses the dates established by Robert Pollitzer, an Austrian physician who worked for the World Health Organization during the 1950s (Barua 1992, 8). These are 1817–1823, 1829–1851, 1852–1859, 1863–1875, 1881–1896, 1899–1923, and 1961–present. Anybody who looks up the dates of the seven cholera pandemics on Wikipedia will see yet a different set of dates that are similar to, but not identical with, those used by Pollitzer for the first six pandemics. Proximity in time did not make the matter any more obvious to observers who lived through much of the nineteenth century. In 1910, A. J. McLaughlin, who served in the U.S. Public Health Service, described six pandemics between 1817 and 1910, but assigned different beginning and end dates from any of those listed above (McLaughlin 1910, 129).<sup>1</sup> In 1919, E. D. W. Greig, a British physician and specialist in tropical medicine, described seven pandemics in the nineteenth century alone, all with different dates from any listed above, the first beginning only in 1826; it seems that for him, pandemics counted when they affected Western populations (Greig 1919, 4–5).<sup>2</sup> All of this points to the difficulty of establishing the temporal and geographical boundaries of epidemic and pandemic cholera and an inevitable arbitrariness in establishing those boundaries, although recent genetic studies have enabled a more finely tuned dating of the seventh pandemic (Chun et al. 2009). Indeed, based on bacteriological evidence, one observer has ventured to say that the first six pandemics were nothing more than a single, continuing pandemic (Blake 1994, 294).

The symptoms, course, and outcomes of cholera as most texts today describe them are so distinct that the disease would seem easy to spot: cholera begins with mild but increasing nausea and malaise followed sometimes by vomiting and almost inevitably by severe diarrhea that starts soon after the onset of symptoms. In reality, infection with *Vibrio cholerae* can result in a clinical spectrum

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<sup>1</sup> McLaughlin’s dates for the pandemics were 1817–1823, 1826–1837, 1846–1862, 1864–1875, 1883–1896, and 1902–1910.

<sup>2</sup> Greig’s dates for the pandemics were 1826–1837, 1840–1849, 1853–1856, 1863–1868, 1868–1873, 1881–1885, and 1892–94.



ranging from asymptomatic infection to cholera gravis, the current term for the most severe form of the disease (Nelson et al. 2009, 694). If left untreated, the latter can cause rapid dehydration with a loss of up to one liter (about a quart) of fluids per hour, resulting in lethargy, cold skin, sunken eyes, cyanosis, cardiac arrhythmia, and death in up to 70 percent of cases (Harris et al. 2012, 2469–2470). Yet not all cases present so clearly. Depending on a person's immune system, the amount of acid in the stomach, and a number of other physiological factors, infection can be asymptomatic or cause nothing more than mild symptoms mimicking more ordinary gastroenteritis or a minor case of food poisoning. Even more importantly, the reporting of cases in the past was frequently spotty and inconsistent, and the European and North American men who created the category of pandemic and established the dates and places to be included had a selection bias.

At the beginning of an epidemic, symptoms tend to be more severe. The main reason is hyperinfectivity, a characteristic of the bacterium that makes it easier to transmit between individuals immediately after it has been first introduced from the environment into the human body (Nelson et al. 2008, 697–698; Harris et al. 2012, 2468–2469). Another reason is that as populations are exposed to cholera, they build up immunity to the disease (Harris et al. 2010, 1963). Over time, the symptoms become less severe, sometimes masking the distinction between cholera and other gastrointestinal infections—yet another reason that historical and sometimes even contemporary data regarding both the numbers of cases and deaths from the disease are less than completely reliable. Nevertheless, the historical data is useful as an index of cholera's presence, even if it is not an exact measure of its epidemiological and demographic consequences. This index of cholera's presence is important in the following analysis, which makes use of Japan's historical cholera morbidity and mortality data not to reveal the exact numbers of cases but to demonstrate the relative presence of the disease.

A look at other relevant dimensions of *Vibrio cholerae* will help set the stage for this article's argument that after 1877, cholera epidemics in Japan were more likely to have occurred as a result of indigenized bacteria than from repeated invasions by bacteria from abroad. The reasons are based not only on the historical data but also the biological and environmental circumstances that make the bacterium dangerous to humans and allow it to exist for extended periods of time without human contact. The science on which this reasoning is based is relatively recent. Some of it dates from the last half of the twentieth century, and other studies come from the first two decades of the twenty-first century.

### **The Cholera Bacterium, the Environment, and Disease**

It took what might seem a surprisingly long time to discover how the cholera bacterium causes disease. A total of sixty-six years passed between the beginning of the first cholera pandemic in 1817 and Koch's rediscovery of *Vibrio cholerae* in 1883, despite Pacini's initial discovery in 1854. It took even longer, seventy-six years, between Koch's confirmation that *Vibrio cholerae* caused the disease and the discovery of the cholera toxin. This protein causes the human body to lose enormous volumes of water and electrolytes, especially sodium, whose depletion

can lead to a rapid death. In 1959, an Indian scientist, Sambu Nath De, identified the cholera toxin by taking it outside the bacterium and subsequently showing that it could cause fluid depletion in a living animal (De 1959). Another thirty-seven years passed between De's discovery and the realization in the late 1990s that the presence of the cholera toxin in *Vibrio cholerae* was the result of horizontal gene transfer between a filamentous bacteriophage, an independent virus (which will be discussed later), and the cholera bacterium (Waldor and Mekalanos 1996). Since the 1990s, researchers have significantly expanded our knowledge of the environment's role in the life cycles of *Vibrio cholerae*. For years, there was a popular and scientific opinion that cholera could be transmitted only between humans via contaminated food and water, a view that has been slow to change even among highly informed writers (Quammen 2012, 381). The progress in the scientific knowledge about cholera and its transmission has been helpful for health policymakers—and is just as promising for helping us understand the history of the disease and, more specifically, how it became endemic in Japan.

Beginning with the heyday of bacteriology in the late nineteenth century, medical specialists and laypeople alike equated the presence of a bacterium with the disease itself. This was most obvious in the case of tuberculosis, a disease also known by the initials TB, which stand for tubercle bacillus. Much the same occurred with cholera. The disease and the bacterium *Vibrio cholerae* tended to be equated until it became clear by the early twentieth century that there existed numerous species of the *Vibrio* genus as well as sub-types of *Vibrio cholerae*. By the 1930s, researchers had discovered multiple strains of *Vibrio cholerae*, realizing that not all of them caused the symptoms of cholera (Gardner and Venkatraman 1935). Later in the twentieth century, researchers discovered that the cholera bacterium existed globally in aquatic environments. More than thirty species of *Vibrio* and more two hundred serogroups of *Vibrio cholerae*—of which only two, the O1 and O139, are associated with epidemic cholera—have been discovered (Kaper, Morris, and Levine 1995, 54–56; Pruzzo et al. 2005, 217). In short, by no means does it make sense to equate the disease cholera with the bacterium *Vibrio cholerae*. Science has shown that conditions for the outbreak of epidemic cholera depend on much more than the presence of the bacterium. Indeed, this multifactored model of cholera's causality is closer to the model forwarded by one of the founders of modern public health, Bavarian chemist and hygienist Max von Pettenkofer, who believed that a confluence of water, soil, and bacterial conditions needed to occur before the disease could be transmitted from person to person (Morabia 2007, 1234–1235). Although Pettenkofer's theory about soil and the importance of the bacterium was incorrect, its approach was closer to contemporary ideas about multifactorial causation of disease than Koch's ideas about bacteria being the sole cause (Morabia 2007, 1236–1237). Although the bacterium is a necessary cause, it is not in itself sufficient.

One of the most important discoveries leading to a better understanding of the relationship between cholera and the environment was that bacteriophages needed to insert genetic material into the bacterium to make it toxic to humans. Bacteriophages—literally, “eaters of bacteria”—are viruses that constitute one of the most pervasive biological entities on earth (Weinbauer 2004; Srinivasiah et al.

2008). There are two main cycles, or ways in which phages, as they are commonly called, reproduce in bacteria: lytic and lysogenic. In a lytic cycle, the phages insert genetic material into the bacteria; that material uses the bacteria's systems to reproduce and form new phages, which then burst out of the bacteria, killing them. These phages are called virulent phages. In a lysogenic cycle, the phage is reproduced through the reproductive cycle of the bacterium, which then remains viable; these are called temperate phages. Only under special circumstances do temperate phages initiate a lytic cycle, killing the bacterial host and sending phages into the environment (Touchon, Moura de Sousa, and Rocha 2017).

In natural environments, both aquatic and terrestrial, there is an ongoing contest between phages and bacteria. The relative presence of phages in the environment depends on conditions such as temperature, pH, and salinity; their presence is a powerful controlling force on bacteria populations, including *Vibrio cholerae* (Faruque et al. 2005a, 2005b; Nelson et al. 2009, 699–700; Faruque and Mekalanos 2012; Faruque 2014). Yet in the case of *Vibrio cholerae*, phages not only have a limiting impact on populations, but also are the origin of toxicity in the bacterium. For *Vibrio cholerae* to cause epidemic disease, it requires the sequential insertion of genetic material from two lysogenic bacteriophages. The first is a phage whose genes create pili, filamentous structures on the surface of the cholera bacterium, which takes a form reminiscent of a wiener sausage with a long, thin tail; keep in mind that this is an organism whose size allows thousands to fit on the head of a pin. The pili add a kind of fuzz to the outside of the bacterium and have three functions: (1) to anchor the bacterium to the cell wall of the human intestine; (2) to act as a receptor for the phage, which, after attaching itself to a pilus, inserts into the bacterium the genetic code for the production of the cholera toxin; (3) to facilitate the creation of biofilm, which allows the bacterium to attach itself to chitinous surfaces, such as the outside of zooplankton (Vezzulli et al. 2008). To become toxic to humans, the *Vibrio cholerae* must undergo two separate lateral gene transfers (Waldor and Mekalanos 1996; Donnenberg 2000; Nelson et al. 2009). The toxic cholera bacterium constitutes a chimera in the classical Greek sense: it requires elements from multiple organisms to make it dangerous to humans. *Vibrio cholerae* without infection with these phages can cause gastroenteritis, septicemia, and wound infections. Its gastrointestinal infections tend to be mild and rarely contagious (Pruzzo et al. 2005, 217). Depending on the environmental conditions, horizontal gene transfer through phage infections can turn autochthonously existing yet benign *Vibrio cholerae* in aquatic environments into new epidemic strains (Faruque and Mekalanos 2012). In other words, this process makes it entirely plausible for *Vibrio cholerae* to exist in aquatic settings for extended periods without causing disease, only to appear in epidemic form as a result of changes in environmental conditions (Huq et al. 1983; Colwell and Huq 2001; de Magny et al. 2008; Vezzulli et al. 2008; Nelson et al. 2009).

Other environmental conditions facilitate not only the existence but the flourishing of *Vibrio cholerae* in various aquatic settings. The bacterium has been discovered worldwide, in areas ranging from Chesapeake Bay in the United States to locations in Australia, England, Italy, and Sweden—all places where outbreaks of cholera have not occurred for very long periods (Colwell and Huq 2001; Lutz et al.

2013). Most importantly, *Vibrio cholerae* has been shown to survive in aquatic environments ranging from those with little or no salinity, such as freshwater streams and rivers, to more saline water found in estuaries and even open seawater (Colwell 1996, 2027; Vezzulli et al. 2008; Nelson et al. 2009). These conditions in conjunction with the activity of bacteriophages help explain cholera's endemicity in inland sea regions, a pattern made more apparent in a later section of this article.

As researchers looked for ways that *Vibrio cholerae* could exist for extended periods of time in such a range of environmental conditions without contact with the human body, they discovered new qualities about both the bacterium itself and previously unidentified places it inhabited. One key discovery was that *Vibrio cholerae* could enter a state that scientists call viable but nonculturable (VBNC). Reports of this state appeared as early as 1982, but some researchers questioned whether bacteria could assume such a form (Xu et al. 1982; Nyström 2001). This debate continued until the early twenty-first century but has been settled with the identification of viable but nonculturable states for not only the cholera bacterium but numerous bacteria species as well (Barcina and Arana 2009; Oliver 2010). It is clear that *Vibrio cholerae* enters this state depending primarily on environmental conditions, especially changes in salinity, temperature, sunlight, and the presence of nutrients. Once conditions become favorable again, the bacteria in this state can revert to their active form in which they again become infectious. Just how long bacteria can survive in this state is unclear, but it has been shown to range between one and at least six years (Oliver 2010, 418). The length of survival implies that *Vibrio cholerae* in the environment can cause epidemic disease long after having disappeared from human populations. This finding has great significance when considering the endemicity of the disease in nineteenth-century Japan.

Another survival method for *Vibrio cholerae* is its ability to attach itself to the surface of numerous other species. These include copepods and other zooplankton, phytoplankton, various other aquatic plants, and numerous macroscopic sea creatures ranging from oysters, shrimp, and crabs to fish and even birds (Huq et al. 1983; Sedas 2007, 232; Vezzulli et al. 2008, 255; Halpern, Senderovich, and Izhaki 2008; Senderovich, Izhaki, and Halpern, 2010). It does this generally through the formation of biofilm, which establishes a surface adhesion between the bacterium and the various host organisms to which it becomes attached (Lekshmi et al. 2018). These interactions with other organisms allow *Vibrio cholerae* to not only exist for extended periods outside the human body but move considerable distances, especially when ships carry infected zooplankton or mollusks. Outbreaks of epidemic cholera can occur when seasonal conditions stimulate the increase of phytoplankton, providing increased nourishment for copepods and other zooplankton and in turn for *Vibrio cholerae* itself, resulting in significantly higher concentrations of the bacterium in ways that can facilitate its transmission to humans. For example, oysters filter copepods and other zooplankton containing *Vibrio cholerae*, leading to concentrations over one hundred times those found in ambient waters (Morris 2003, 272).

As shown here, the environmental biology of *Vibrio cholerae* indicates that the bacterium has the ability to remain viable in aquatic environments for extended periods. In short, epidemic cholera can arise from the consumption of infected

oysters and other mollusks, fish, and some forms of seaweed—all of which were common foods in nineteenth-century Japan. More importantly, the environmental biology of cholera strongly suggests that the Japanese coastline could easily have provided a highly suitable environment for the long-term sustenance of *Vibrio cholerae*, facilitating swings between endemicity and epidemic disease. There is compelling evidence that this indeed occurred. It appears in the prefectural data from the *Statistical Yearbook of Imperial Japan*. The use of geographic information system (GIS) analysis in conjunction with this data makes patterns visible that otherwise would be more difficult to discern.

### **The Epidemiological Record of Cholera in Japan**

In advance of examining the data using a GIS analysis, it is helpful to look briefly at the epidemiological record regarding cholera in Japan as a country and in the context of several specific prefectures. Despite the attention given to cholera, its national mortality rates throughout the entire period between 1877 and 1920 were relatively low. The disease was the focus of numerous health policies and much public attention primarily because its epidemics were socially and economically disruptive, especially in prefectures with high mortality rates. Between 1877 and 1920, crude cholera mortality rates per 100,000 of the population ranged from highs of 290 in 1879 and 283 in 1886—the only two years in which they reached three figures—to lows of zero in thirteen of the forty years studied. After 1920, national cholera mortality rates never again rose past 1 per 100,000 (calculated based on the *Statistical Yearbook of Imperial Japan*). The average national mortality rate from cholera for the entire period between 1877 and 1920 stood at 22 per 100,000. By comparison, mortality from pulmonary consumption and tuberculosis between 1886 (the first year for which figures are available) and 1920 ranged from a low of 86 per 100,000 in 1886 and a high of 257 per 100,000 in 1918; the average for that period was 180 per 100,000—over eight times the mortality caused by cholera. As another basis for comparison, the mortality rate for heart disease, the leading cause of mortality in the United States in 2016, stood at 197 per 100,000; malignant neoplasms stood at 185 per 100,000 (CDC).<sup>3</sup> National mortality rates for cholera, however, mask significant prefectural differences. In the epidemic of 1886, for example, Tokyo Prefecture experienced a mortality rate from cholera of 926 per 100,000; for Osaka, the rate that year was even higher, at 983. The rate for Kagoshima that year, however, did not even reach 3 per 100,000 (Naikaku Tōkei Kyoku, ed. 1888). National averages, in other words, hardly begin to tell the story of cholera's regional impact; rather, they hide important differences.

### **The Prefectural Record through GIS Analysis**

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<sup>3</sup> National and state mortality data for 2016 can be found at [https://www.cdc.gov/nchs/data/dvs/lcwk/lcwk5\\_hr\\_2016.pdf](https://www.cdc.gov/nchs/data/dvs/lcwk/lcwk5_hr_2016.pdf).

Prefectural data obviously does reveal regional patterns, yet displaying the data for all forty-seven prefectures over the course of forty years is unwieldy at best. This is where the use of GIS analysis, illustrating annual mortality rates for each prefecture, is extremely helpful.<sup>4</sup> The prefectural mortality data, when animated, allows us to see the geographical distribution of cholera mortality by year and to consider that distribution in light of the environmental biology of *Vibrio cholerae*.

The prefectural data does have its limitations. It does not begin until 1881, though national data regarding infectious diseases is available from 1876. The data for some regions is not completely sequential; for Hokkaido, for example, it begins with separate data for Kaitakushi (The colonial office) during 1882, and for Sapporo, Nemuro, and Hakodate during the years 1883 and 1884. No prefectural data is available in the *Statistical Yearbook of Imperial Japan* between 1901 and 1909. Even more problematically, the population data for each prefecture, which of course is necessary for the calculation of crude mortality rates, is not consistent across the entire period from 1880 to 1920. From 1880 to 1897, the population data depends on the *honseki jinkō* (registered population); only from 1898 does the *genjū jinkō* (actual population) become available. It is assumed that when a person died of cholera it was reported to the police in the location where the death occurred and that those are the numbers available for annual deaths from the disease in any one prefecture. The *honseki jinkō* for places such as Tokyo was smaller than the *genjū jinkō* as shown in the figures for 1898, when the former was 1,507,642 and the latter 1,877,412; this difference would have skewed mortality rates per 100,000 of population upward before 1897 and downward after that year. This use of different population data is unavoidable after 1897 because *honseki jinkō* is available annually before that but only once every five years thereafter. Rural prefectures out of which residents moved to work in urban areas have larger registered than actual populations, meaning that the crude mortality rates from cholera would have been skewed in the opposite direction. Another problem is simply that of reporting. It must be assumed that reporting of cholera cases was not consistent throughout the country in that many people wished to avoid association with such a malignant affliction. In short, the prefectural mortality rates for cholera as well as those for all of Japan must be interpreted as an index rather than an exact measure.

Yet even as an index, these figures and the visual information they impart through this animated map reveal important patterns. In particular, in non-epidemic years, cholera remains endemic in most of the prefectures of Japan that faced the Inland Sea. Throughout the 1880s and 1890s, these prefectures rarely went a year without reported deaths from cholera. Based on the current environmental biology of *Vibrio cholerae*, this finding should be unsurprising. This area has large stretches of complex coastlines and contains hundreds of small islands; a large number of rivers drain into the Inland Sea from both Honshu and Shikoku, providing nutrients

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<sup>4</sup> An animated map of Japan illustrating changes in crude mortality rates from cholera by prefecture between 1881 (the year in which prefectural data is first available) and 1920 (after which, as previously noted, cholera mortality in Japan becomes relatively insignificant) is available online at <https://cross-currents.berkeley.edu/e-journal/issue-30/johnston>.

for both *Vibrio cholerae* itself and the various organisms that it can colonize. The result is a favorable environment for *Vibrio cholerae*, one that is rich in plankton blooms and large numbers of mollusks. Hiroshima, for example, was famous for its oysters, but experience limited their consumption to the cold months. Because of the ability of oysters to concentrate the bacterium in their tissue, as noted in the discussion about the environmental biology of *Vibrio cholerae* above, it is easy to imagine them becoming infected with *Vibrio cholerae* and providing a vector for cholera when warmer waters provided a favorable climate for the bacterium. It is conclusive that cholera was indeed endemic to Japan at least during the last two decades of the nineteenth century. Moreover, it is probable that *Vibrio cholerae* remained viable in the Inland Sea between 1901 and 1909, when deaths from cholera were not reported on the prefectural level but were reported nationally; in 1902, 8,104 deaths from cholera were reported, and in 1907, there were 1,702. When prefectural data again becomes available in 1910 through 1920, the consistent hot spots continue to be the prefectures surrounding the Inland Sea and coastal areas in western Honshu.

Another pattern that the animated map available online makes visible is the impact of cholera on populations that had minimal previous exposure to the disease. Areas such as large parts of northeastern Japan (Tōhoku) and Hokkaido tended to have low mortality rates from cholera, but in epidemic years such as 1886 and 1895 they experienced high mortality rates. This occurrence matches patterns observed in other parts of the world in which populations that previously had low exposure to the disease then suffered relatively high mortality once *Vibrio cholerae* began to spread (Nelson et al. 2009, 694). By no means does this knowledge amount to an epidemiological history of cholera in Japan, but that is not the goal of this article, which is, rather, to show that the biological and environmental science of the disease can help us understand some of the patterns of its historical epidemiology.

## Conclusion

Based on current science regarding the *Vibrio cholerae* bacterium and the disease it causes in conjunction with a GIS-enabled illustration of regional mortality patterns, it is clear that cholera was indeed endemic in Japan during the last two decades of the nineteenth century, a time when popular media if not public health officials depicted the disease as coming from abroad (Johnston 2017). This endemicity was in no small part the result of cholera's being primarily a zoonosis, or disease of animals, that infects copepods (Huq et al. 1983; Colwell and Huq 2001). As in other places, cholera in Japan during these decades infected humans who lived in coastal regions, especially in non-epidemic years. In this way, the overall epidemiological pattern of cholera fits patterns seen in many other regions across the globe.

By the 1910s, the disease had become localized primarily in areas where it had existed endemically before that, and by the 1920s cholera's national mortality and morbidity rates had become insignificant. This decline remained true even during the hardship years of World War II, with a minor uptick in cholera mortality in 1946, when 560 individuals died of the disease nationwide—not enough to register significantly among other causes of disease, much less other causes of mortality. The

decline certainly was not from the expansion of sewage systems, the widespread construction of which had to wait until after the 1970s. Until then, most human waste was collected in septic tanks that dealers in night soil regularly emptied. Although sewage systems remained rudimentary through the first half of the twentieth century, it appears that the current methods of treating individuals infected with cholera did, for the most part, help prevent the spread of the bacterium. For example, a popular manual for home nursing and first aid published in 1910 emphasized the need both to take care with effluents that cholera patients produced and to wash hands to prevent the spread of the cholera bacterium (Kodama 1910, 34–35). Arguably, the key change was to stop the reintroduction of *Vibrio cholerae* from the environment into human populations. This decline in cholera mortality likely was tied to the rapid expansion of pure water supplies throughout Japan during the late nineteenth and early twentieth centuries (Kasahara 2003; Kojima 2018).

Definitive proof that the effective separation of water supplies and human waste combined with increasingly effective methods of dealing with effluents from cholera patients calls for yet another study. Yet this article does illustrate how current science can inform answers to historical questions, especially regarding the environment and disease patterns. In doing so it offers a degree of agency to actors such as bacteriophages that do not appear in the archival record but clearly played an important role in the history of this disease. This work might encourage others who specialize in the history of disease to make themselves as familiar as possible with the current science on their subjects, as long as that knowledge can contribute to understanding how people in the past experienced disease or how diseases waxed and waned in time and space. In addition, it would be beneficial to apply similar methods to the study of environmental pollution, climate change, and habitat destruction in historical perspective, providing useful analyses for not only historians but policymakers as well. In this way C. P. Snow's call for a bridge between the humanities and the natural sciences is as urgent today as it was he first made it over sixty years ago.

## Appendix

For many decades, historians left the histories of diseases and epidemics to physicians based on the assumption that they required medical rather than historical training. Rather than being considered as world-shaping forces, the history of disease and epidemics became a niche field. Early histories of cholera tended to examine questions of interest to physicians and other medical specialists. Aside from the earliest histories of cholera by nineteenth-century observers such as John MacPherson and Duane B. Simmons, the modern history of the disease was largely initiated by Robert Pollitzer. He tracked its history from earliest times to the twentieth century based on the assumption that today's "cholera" was the same as what premodern writers meant by that term—or translations of it. Pollitzer was thoroughly grounded in the contemporary science relevant to the history of cholera, but he wrote before genetic, environmental, and other discoveries changed the way people thought about the disease (Pollitzer 1954; 1959; Hamlin 2009, 268). Dhiman



Barua wrote a more up-to-date version of cholera's history in this vein, extending his study to include the seventh pandemic (Barua 1992). More recently, two cardiologists, S. L. Kotar and J. E. Gessler, have written a global history of cholera that tracks its pandemics from the earliest one in 1817 to the present era (Kotar and Gessler 2014). These works focused on the course of pandemic cholera across the globe or on changing medical responses to the disease. Although their authors were medically trained, they did not generally use current science to explain how pandemic cholera spread and integrated itself in local environments.

An early foray into the history of cholera by a trained historian was Charles E. Rosenberg's *The Cholera Years: The United States in 1832, 1849, and 1866*. Published when he was twenty-six years old, its relevance remains perennial. It focuses on the religious, cultural, and public health responses to the disease in those years (Rosenberg 1962). Another monumental work is Richard J. Evans's *Death in Hamburg: Society and Politics in the Cholera Years, 1830–1910*. It provides an in-depth look at the scientific, social, and political events relevant to that city's experience with the disease, especially Max Joseph von Pettenkofer's tragic objections to Robert Koch's theory that a specific germ, *Vibrio cholerae*, caused the disease (Evans 1987). Frank M. Snowden's *Naples in the Time of Cholera, 1884–1911* is another model study not only of this disease in a single city but of its broader cultural implications and why, unlike tuberculosis or syphilis, its symptoms prevent it from being the stuff of romantic novels, narratives of rebellion, or operatic arias (Snowden 1995, 112–113). Going geographically and temporally to the other extreme is Myron Echenberg's *Africa in the Time of Cholera: A History of Pandemics from 1817 to the Present*, which examines the history of the disease on a continental scale from the first pandemic to the time of its publication. To a certain extent, Echenberg does examine relevant scientific sources, especially with regard to transmission and endemicity (Echenberg 2011). Notably, at least one scholar has engaged fully with the literature regarding the medical, biological, and ecological sciences relevant to cholera's transmission and endemicity. Christopher Hamlin, in *Cholera: The Biography*, argues that the current medical and scientific knowledge about *Vibrio cholerae* and its global environmental distribution decenters the disease from Asia, with which it had been associated from the time of its first pandemic in 1817. He points out how current science has made the boundaries around our conceptions of cholera much fuzzier than previously thought (Hamlin 2009, 268). Although the term "Asiatic cholera" has long been in circulation, it is no more accurate than calling the 1918 influenza pandemic by the moniker "Spanish flu." In many respects, Hamlin's work is a model for interdisciplinary work inclusive of the sciences as well as social science theory in the history of disease and public health (Hamlin 2012).

The historiography of cholera in Japan is rich, especially when compared to that of most other non-Western countries. Fujikawa Yū, the doyen of medical history in Japan, who trained as a physician but practiced as a historian, first published his *Nihon shippei shi* (A history of disease in Japan) in 1912. It included an extensive discussion of cholera but focused on understandings of the disease in the medical literatures in Japan (both Chinese- and Western-based) and, to a certain degree, in popular culture (Fujikawa [1912] 1944). Another physician, Yamamoto Shunsuke,

who taught medicine at Tokyo University, published his magisterial *Nihon korera shi* (A history of cholera in Japan) in 1982. It makes an enormous contribution, especially as a compendium of primary sources on cholera in Japan, but offers minimal analysis. This work's contradiction of many other studies by claiming that no cholera epidemic occurred in 1862 suggests that its strengths are in its examination of the disease during and after the Meiji-era reforms (Yamamoto 1982).

Fujikawa Yū also initiated formal studies of the medical history of cholera in Japan—focusing on theories of causation, prevention, and cure—with the initial publication of his *Nihon igaku shi* (A history of Japanese medicine) in 1904 (Fujikawa [1904] 1979). Tatsukawa Shōji's *Kinsei yamai zōshi* (Stories of maladies in early modern Japan) includes a chapter on cholera before the Meiji Restoration in 1868, with sections on the 1858 epidemic, the widespread confusion that epidemic caused, and popular beliefs about the disease (Tatsukawa 1979). Similarly, Nakajima Yōichirō's *Byōki Nihon shi* (Disease in Japanese history) includes a section on cholera, focusing on premodern conceptions of the disease's cause, names, and famous people who suffered from it (Nakajima 1982). An examination of how the changing names of cholera-like diseases in nineteenth-century Japan reflected changing medical epistemologies can be found in my essay (Johnston 2014). Roberto Padilla has written a more tightly focused essay that examines problems associated with disease categories that the Japanese military used during the First Sino-Japanese War (1894–1895) (Padilla 2016).

A number of well-researched works have been written on the Edo-period experience with cholera, mostly focusing on the epidemics that occurred in 1858 and 1862, although the epidemic of 1822 does not go completely unmentioned in the secondary literature. An examination of death records from Buddhist temples in different regions of the country during the three major Edo-period epidemics appears in an article by Kikuchi Kazuo. Kikuchi (1978) demonstrates that in 1859 cholera was likely more deadly than in 1858, but the earlier year gains greater attention because of its impact on the city of Edo. In addition, he persuasively argues that the 1862 epidemic was the worst of the three that occurred during the Edo period. Bettina Gramlich-Oka's "The Body Economic: Japan's Cholera Epidemic of 1858 in Popular Discourse" provides, at this point in time, the most comprehensive English-language overview of the popular cultural responses to that epidemic (Gramlich-Oka 2009). In Japanese, Takahashi Satoshi's *Bakumatsu orugii: Korera ga yatte kita!* (A Bakumatsu orgy: Cholera has arrived!), despite its lurid title, provides a rich analysis of local reactions, primarily based on religious and folk beliefs among villagers and townspeople, to the epidemics of 1858 and 1862 (Takahashi 2005). A close examination of the interaction between J. L. C. Pompe van Meerdervoort, a Dutch physician in Nagasaki, and Ogata Kōan, the founder of the school of Western Learning in Osaka, Tekijuku, and how they treated cholera in the 1858 epidemic can be found in Härmen Beukers's 1992 "Pompe to Ogata Kōan no korera chiryōhō" (Remedies for cholera of Pompe and Ogata Kōan). My "Cholera and Popular Culture in Nineteenth Century Japan" (2017) contextualizes the popular responses to cholera during the three epidemics of the Edo period but also discusses continuities and discontinuities in those responses before and after the Meiji Restoration in 1868.

In one of the most important English-language studies of epidemics and public health in modern Japan, Susan L. Burns argued in “Constructing the National Body: Public Health and the Body in Nineteenth-Century Japan” (2000) that cholera was Japan’s first modern epidemic, and showed how it was central to the formation of modern Japan’s public health system. Another study of how the body became the subject of hygienic modernization and state control is the subject of Miri Nakamura’s *Monstrous Bodies: The Rise of the Uncanny in Modern Japan*, in which she shows how cholera, an invisible yet potent health threat, was visualized using contemporary metaphors and tropes (Nakamura 2015, 16–23). In another seminal essay that examines the obstacles that contemporary public health officials faced among the general public, Akihito Suzuki and Mika Suzuki explore the modernization of medicine in nineteenth-century Japan through the lens of consumerism and citizenship (Suzuki and Suzuki 2009). Oginō Natsuki’s study of public health in the context of “civilization and enlightenment” shows how officials attempted to change the way laypeople thought about cholera and replace their thinking with “civilized” ideas during the epidemic that occurred in 1879 (Oginō 2012). Another important public health response was the establishment of quarantines on ships entering Japanese harbors, which raised important issues of international law. “Informal Imperialism and the 1879 *Hesperia* Incident: Containing Cholera and Challenging Extraterritoriality in Japan” by Harald Fuess (2014) examines many of these issues. An essay on the Japanese quarantine placed at approximately the same time on the Korean port of Busan and the meeting of empire and international law is Jeong-Ram Kim’s “The Borderline of ‘Empire’: Japanese Maritime Quarantine in Busan c.1876–1910” (2013). Chester J. Proshan’s study of epidemic cholera in Yokohama takes up many of the same issues of quarantine while looking more broadly at the expatriate communities there from the late Edo period to the end of the century (Proshan 2018).

As one might expect because of cholera’s impact on public health policy formation and institutional reforms, the body of literature dealing with these themes is relatively large and well developed, with Burns’s (2000) groundbreaking essay examining these themes in comparison with other infectious diseases widespread at the time. Similarly, Hozuki Rie’s study of the development and social integration of *eisei* (hygiene) emphasizes the immense role cholera had in the rise of hygienic thought and public health practices in modern Japan (Hozuki 2010). Numerous other studies detail cholera’s importance in this respect. Utsumi Takeshi described hygienic measures aimed at eliminating unsanitary conditions throughout the country during the cholera epidemic of 1877 (Utsumi 1992). Following a parallel argument, my article examines how the Meiji state collaborated with the established Buddhist churches after the epidemic of 1879 in an attempt to persuade people throughout Japan that, although during epidemics they certainly should pray to the *kami* and buddhas, they also should complement those responses with “scientific” approaches to prevention and treatment (Johnston 2016). Two government bureaucrats, Nagayo Sensai, Director of the Central Sanitary Bureau of the Home Ministry, and Gotō Shinpei, who also worked in the Central Sanitary Bureau and later became governor of Taiwan, and their responses to cholera stand out in the history of public health in Japan (Nagayo 1877; Gotō 1890). Many works, including Burns,

discuss Nagayo, but more focused studies can be found in essays by Kasahara Hidehiko (2007) and Kojima Kazutaka (2009). These works build on Kasahara's earlier essay that examined the impact of Nagayo and Gotō on the establishment of modern Japan's public health administration; cholera control was a main objective (Kasahara 1996). Not surprisingly, Nagayo also played an important role in *eisei kōji* (public health engineering) projects, one of whose main goals was cholera prevention through the establishment of reliable clean water sources (Kasahara 2003; Kojima 2018).

Several works have raised issues that link geography with cholera during the nineteenth and early twentieth centuries in Japan. In his doctoral dissertation, Padilla (2009) discusses at length Japanese perceptions of the rest of Asia as being diseased and the idea that China and Korea in particular harbored infestations of cholera that threatened Japan, at least until the 1880s. Yet Padilla emphasizes that by 1886, Japanese health officials considered cholera to have become endemic to the country (2009, 120). Taking a different perspective on modernization and the spread of cholera, economist John P. Tang (2017) claims that the growth of railroads during the 1880s and later facilitated the spread of acute infectious diseases, especially cholera. Two groundbreaking essays that use GIS-based analysis appeared in 2007. One focuses on the cholera outbreak in 1890, using GIS to examine the spread of the disease primarily in northern Kyushu but also throughout Japan. This study reveals the general course of the disease across the country during this epidemic, with hot spots located long transportation routes (Kuo and Fukui 2007a). The other, a study of cholera in Fukushima Prefecture during the epidemics of 1882 and 1895, uses similar methods and also reveals that cholera outbreaks were concentrated in areas surrounding transportation hubs, especially where they had high degrees of traffic from outside the prefecture (Kuo and Fukui 2007b).

This is by no means an exhaustive survey of the secondary literature on the history of cholera in Japan. Nevertheless, it seems safe to conclude that, so far, works by historians of cholera in Japan have taken a broad variety of approaches, but virtually none have attempted to show how the physical reality of *Vibrio cholerae* in its environment was a major determinant in its epidemics and establishment as an endemic disease.

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