Pestis Minor : The History of a Contested Plague Pathology

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SUMMARY: *Pestis minor* is a pathological category that at the height of the third plague pandemic (1894–1959) fueled extensive debate and research among medical scientists. Referring to an attenuated or benign form of plague, evidence of *pestis minor* or *pestis ambulans* was produced in medical reports across the world so as to raise the question of whether the disease could survive measures against it by means of temporary transformation. Afflicting its victims only by the slightest lymphatic swellings, this theory went, the disease could thus lurk in the human body until conditions allowed it to break out again in its true, malignant form. This article draws for the first time a history of this contested pathology, the diagnostic and epidemiological questions raised by it, and the way in which it came to play a significant role in debates about the nature of plague at the turn of the nineteenth century.

KEYWORDS: plague, diagnosis, evidence, commission, India, Russia, Philippines, Hong Kong

Following the discovery of the plague bacillus by Alexandre Yersin in Hong Kong, in the summer of 1894, and in the context of the global pandemic of the disease that soon ensued, the study of plague witnessed phenomenal growth.[1] Across all inhabited continents a broad spectrum

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1. For a historical review of the third plague pandemic, see Myron J. Echenberg, *Plague Ports: The Global Urban Impact of Bubonic Plague, 1894–1901* (New York: New York University Press, 2007).

of medical and life scientists would engage in the bacteriological, clinical, epidemiological, and ecological study of the disease. Research was fueled by pressing imperial and governmental demands for information that could lead to stamping out the scourge and to the protection of humans and vital infrastructures. For over five decades this study produced a rich exchange on hypotheses about and ways of investigating plague's transmission pathways and its ability to persist in urban and rural environments. Key to these debates was the notion that plague possessed the ability to attenuate itself and thus remain hidden in the course of interepidemic periods. In the words of a member of the Quarantine Council of Istanbul in 1899, "the plague virus eludes the best efforts of struggle [against it], it annuls the effects of [our] best efforts and it awaits for the most favorable moment, unknown until now to science, for emerging out of its slumber so as to resume its morbid progress."[2]

In many cases, this notion was connected to concerns regarding plague as what Prashant Kidambi has called "an infection of locality," with the soil being considered the "breeding ground" of the disease in material terms: as a physical carrier of the plague bacteria.[3] Yet the idea of plague's natural attenuation was not limited to this inorganic medium and its epistemic or political framings. Instead, through the employment of a "seed and soil" metaphor, which, as Michael Worboys has demonstrated, played an important role in the development of germ theories, physicians also came to see human bodies as sites of bacterial natural attenuation.[4] This article examines the way in which this applied to the case of plague, a disease that was seen as able to assume a treacherous form in the human body itself, by transforming into a distinct and indeed elusive nosological entity. And it does this by exploring an important but historically neglected disease category, which, at the height of the third plague pandemic, emerged from and in turn fueled debates regarding plague's ability to render itself unseen and then "revive" so as to strike back at humanity.

The pathology in question relates to a condition considered at the turn of the century to be a less virulent and debilitating form of the disease than "true plague."[5] This was seen as a benign type of plague that,

2. D. Stekoulis, "Bulletin épidémiologique," *Gazette Médicale D'Orient* 43, no. 24 (February 15, 1899): 353–54, quotation on 353 (my translation).

3. Prashant Kidambi, "'An Infection of Locality': Plague, Pythogenesis and the Poor in Bombay, c. 1896–1905," *Urb. Hist.* 31, no. 2 (August 2004): 249–67. On plague and the soil, see Christos Lynteris, "A Suitable Soil: Plague's Urban Breeding Grounds at the Dawn of the Third Pandemic," *Med. Hist.* 61, no. 3 (June 2017): 343–57.

4. Michael Worboys, *Spreading Germs: Disease Theories and Medical Practice in Britain, 1865–1900* (Cambridge: Cambridge University Press, 2000).

5. On "true plague," see "The Cantlie-Lowson Debate" section, this article.

while inflicting illness upon its human hosts, failed to kill them, with the patient persisting in a relatively functional state. Proponents of the theory believed that, in this way, the patient was able to remain a long-term source of human infection. This diagnostic and epidemiological category, which first appeared in relation to the plague outbreaks in the Volga region in the late 1870s, initially took the name *pestis ambulans* or ambulatory plague, but remained little used by health professionals until two decades later. As of 1896, a new term was applied to describe this form of the disease, *pestis minor*, assuming global application in the context of the third plague pandemic. Over the following decades the two terms would sometimes be used alternatively in medical literature, although there were times when experts attempted to draw a sharp distinction between the two.

Though the category is still, albeit sporadically, in use today, since no recent scientific studies have been conducted to prove or disprove the veracity of this pathological condition, it is beyond the scope of this article to argue on the true or imaginary character of *pestis minor*.[6] What is more important is to excavate the historical emergence and discursive transformations of this capacious category so that we may understand its role in the problematization of plague during the third pandemic. Asking what allowed this fluid category, which first arose within nonbacteriological understandings of plague, to persist and remain useful in spite of evidence contrary to its existence into the early twentieth century, the article stresses the productivity of *pestis minor*. It will be argued that, rather than being a handicap for epidemiological knowledge, the categorical fluidity of *pestis minor* permitted a fertile exchange of evidential frameworks and perspectives regarding plague's natural attenuation. And, at the same time, it will be shown that by contributing to the deferral of plague's configuration into what Charles Rosenberg has called a stable disease entity, *pestis minor* did not simply delay plague's ontological solidification.[7] Instead it fostered ideas about the fundamentally transformative character of plague, which

6. See, for example, M. Ratsitorahina, L. Rabarijaona, S. Chanteau, and P. Boisier, "Sero-epidemiology of Human Plague in the Madagascar Highlands," *Trop. Med. Internat. Health* 5, no. 2 (February 2000): 94–98. It has been maintained that this condition may have influenced Black Death infection patterns, where "the symptoms are so mild that the patient is able to walk around"; Robert Sallares, "Ecology, Evolution, and Epidemiology of Plague," in *Plague and the End of Antiquity: The Pandemic of 541–750*, ed. Lester K. Little (Cambridge: Cambridge University Press, 2002), 231–89, quotation on 244n56. The Latin etymology of "ambulatory," as in *pestis ambulans*, is derived from the verb *ambulare*, "to walk."

7. Charles E. Rosenberg, "The Tyranny of Diagnosis: Specific Entities and Individual Experience," *Milbank Quart.* 80, no. 2 (2002): 237–60.

would fuel and maintain epidemiological interest in the role of the human body in the maintenance of the disease well after rats and wild rodents were admitted as its urban and sylvatic reservoirs.

Plague in Calcutta?

Historians of plague in India, and the third plague pandemic more generally, make passing note of what is commonly referred to as the 1896 Calcutta plague scare. The incident involved William J. Simpson, a leading British expert on tropical hygiene and editor of the *Indian Medical Gazette*, who would later become known for his authoritative reports on plague in Hong Kong and the Gold Coast (Ghana) and his *Treatise on Plague*.[8] Simpson was not a member of the Indian Medical Service (IMS) and was not accountable to the colonial government of Calcutta. He rather acted as the city's health officer employed by the Corporation of Calcutta. Under this capacity, the standard history of the so-called plague scare goes, he diagnosed plague among the Shropshire Regiment, which was at the time stationed in Calcutta.[9] This diagnosis was, however, severely challenged by the regiment's command, and upon further investigation a Medical Board composed of Indian Civil Service sanitary officers and IMS doctors concluded there was no case of plague. Ivan Catanach has argued that the incident led to a questioning of the city's sanitary con-

8. William John Simpson, *Report on the Causes and Consequences of Plague in Hongkong and Suggestions as to Remedial Measures* (London: Waterlow and Sons, 1903); William John Simpson, *A Treatise on Plague; Dealing with the Historical, Epidemiological, Clinical, Therapeutic and Preventive Aspects of the Disease* (Cambridge: Cambridge University Press, 1905); William John Simpson, *Report on Plague in the Gold Coast in 1908* (London: J. & A. Churchill, 1909). William John Richie Simpson (1855–1931) was a health officer in Calcutta (1886–97). After his return to London (1898) he was appointed professor of hygiene in King's College, joining in 1899 the newly founded London School of Tropical Medicine, where he lectured until his retirement (1923). Serving at numerous government commissions on epidemics and public health, Simpson was a key figure in the development of tropical medicine; G. Carmichael Low, "Obituary. Sir William John Ritchie Simpson," *Brit. Med. J.* 2, no. 3691 (October 3, 1931): 633; R. A. Baker and R. A. Bayliss, "William John Ritchie Simpson (1855–1931): Public Health and Tropical Medicine," *Med. Hist.* 31, no. 4 (1987): 450–65.

9. Ivan J. Catanach, "Plague and the Tensions of Empire: India 1896–1918," in *Imperial Medicine and Indigenous Societies: Disease, Medicine and Empire in the Nineteenth and Twentieth Centuries*, ed. David Arnold (Manchester: Manchester University Press, 1988), 149–71, quotation on 155. See also Mary P. Sutphen, "Not What but Where: Bubonic Plague and the Reception of Germ Theories in Hong Kong and Calcutta, 1894–1897," *J. Hist. Med. & Allied Sci.* 52, no. 1 (January 1997): 81–113. For a general history of plague in Calcutta, see Srilata Chatterjee, "Plague and Politics in Bengal 1896 to 1898," *Proc. Indian Hist. Cong.* 66 (2005–6): 1194–1201.

ditions, and consequently to "the severe abridgement of the Calcutta Corporation's powers in the Calcutta Municipal Act of 1899."[10] Thus the Calcutta plague scare has been approached as an incident of intracolonial antagonism in the midst of the Indian plague epidemic, similar to the one between the IMS and the discoverer of the first effective antiplague vaccine, Waldemar Haffkine.[11] Projit Mukjarji has provided an alternative perspective of how the incident fueled conflict in Calcutta in his study of daktari medicine, focused on how Simpson's diagnosis antagonized native doctors and their medical practices.[12] Important as these readings of the incident are, in terms of illuminating colonial dynamics, at the same time they do not illuminate an intriguing aspect of the debacle: the fact that what Simpson claimed to have diagnosed was not at all supposed to be plague as commonly understood by us today or by colonial medicine at the time. In other words, Simpson's contention was not that he had come across a plague outbreak in Calcutta, but rather that he had discovered a series of cases of what he called *pestis ambulans* and would soon be known as *pestis minor*. Hence, if Mary Sutphen is right to note in her analysis of the incident that what was at stake "was not whether plague bacilli were associated with plague . . . but how to interpret the bacteriological evidence," in turn the actual object of medical debate was not plague in general but its supposed ability to naturally assume an insidious, attenuated form that allowed it to persist silently within a given population and thus potentially lead to future outbreaks by means of confounding its medical recognition.[13]

The Indian story of *pestis minor* begins in a telegram dated October 10, 1896, a month after bubonic plague first appeared in Bombay, causing increasing anxiety about the possibility of a pan-Indian epidemic.[14] The

10. Catanach, "Plague and the Tensions of Empire" (n. 9), 155; on the impact of plague on urban planning in Calcutta, see Partho Datta, "How Modern Planning Came to Calcutta," *Planning Perspect.* 28, no. 1 (2013): 139–47.

11. On the Haffkine controversy, see David Arnold, *Colonizing the Body: State Medicine and Epidemic Disease in Nineteenth-Century India* (Berkeley: University of California Press, 1993); Barbara J. Hawgood, "Waldemar Mordecai Haffkine, CIE (1860–1930): Prophylactic Vaccination Against Cholera and Bubonic Plague in British India," *J. Med. Biog.* 15 (2007): 9–19. On provincial-imperial antagonism in India's "scientocracy," see Deepak Kumar, "Emergence of 'Scientocracy': Snippets from Colonial India," *Econ. Polit. Weekly* 39, no. 35 (August 28–September 3, 2004): 3893–98.

12. Projit Mukharji, *Nationalizing the Body: The Medical Market, Print and Daktari Medicine* (London: Anthem Press, 2009).

13. Sutphen, "Not What but Where" (n. 9), 190.

14. On concerns that plague would soon spread to Calcutta, see Anon., "The Plague. Important Letter from the Chamber of Commerce," *Indian Lancet* (October 16, 1896): 395–96.

message, written by Drs. Ross and Dyson, described the case of a seventeen-year-old "Eurasian" Goanese boy, James Cotta, who was reported to have arrived from Byculla, a plague-stricken area in Bombay, to Howrah, in the outskirts of Calcutta, on September 26, 1896.[15] The boy had been experiencing a painful swelling in the left inguinal gland fifteen days before his departure, followed by an enlargement of the right inguinal gland, symptoms that after arriving in Calcutta were accompanied by remittent fever and urticaria. As reported by a local newspaper, "the Howrah police reported that they thought the case was one of plague."[16]

Cotta was thus subjected to a series of tests, with bacteriological examination of his blood, performed by Simpson, revealing what was believed to be plague bacilli: "Dr. Simpson in conjunction with the medical gentleman named gave a certificate to the officials of Howrah to the effect that the case was one of bubonic plague of a mild type, but still highly infectious."[17] Drs. Ross and Dyson were, however, reluctant to conclude that the boy suffered from plague, as the symptoms appeared too mild and Cotta was able to walk and function in a way unusual for plague cases. This reluctance took a more acute form when it came to Surgeon-Lieutenant-Colonel Sanders, who stated this to be a venereal or syphilitic condition.[18] Yet other colonial doctors involved in the case, like Surgeon-Lieutenant-Colonel A. Tomes, were convinced by Simpson's bacteriological tests and were moved to argue that this was in fact a case of "*pestis ambulans*, the mild form of plague."[19] Tomes in particular excluded the implication of venereal disease, noting that it was quite common to mistake plague buboes for syphilitic ones on the onset of the ailment.[20] Sanders, he claimed, "appeared to have fallen into that error."[21] Tomes

15. House of Commons Parliamentary Papers, House of Commons Parliamentary Papers, Cd.139, Indian Plague Commission, 1898–99, Minutes of Evidence Taken by the Indian Plague Commission with Appendices, vol. 1, appendix 21, Note of the Special Medical Board, Calcutta, on the Cases Reported as Plague in Calcutta, 1896, with Reports on Suspected Cases. "Eurasian" was a term applied to the "mixed race" offspring of British and Indian couples, a perilous racial category in the colonial imagination.

16. "Plague," *Amrita Bazar Patrika*, October 12, 1896, 5.

17. Ibid., 5.

18. House of Commons Parliamentary Papers, Cd.139 (n. 15), 489.

19. Ibid., 489.

20. Tomes stressed that in Cotta's case the enlarged glands appeared "both above and below Poupart's ligament, and it is clear there had been no true chancre"; A. Tomes, "The First Case of Plague in Howrah," *Indian Med. Gazette* 31 (December 1896): 447, quotation on 447. Venereal or syphilitic buboes were the subject of several medical treatises in the nineteenth century; see, for example, Alexandre B. E. A. Lasnet, *Étude bactériologique du chancre mou et du bubon* (Bordeux: G. Goinouilhou, 1893).

21. House of Commons Parliamentary Papers, Cd.139 (n. 15), 489.

further noted that this "typical case of 'Pestis Ambulans'" is known to be present at the beginning of plague outbreaks, and to be "less infective."[22] Arbitrating between conflicting medical opinions, Surgeon-Major Walsh, the officiating civil surgeon at Howarth, reported that, having examined Cotta's blood specimen against plague specimens provided by Haffkine from Bombay, he was convinced that this was not plague in its true form, but perhaps, once again, "a mild form of '*pestis ambulans.*'"[23]

Cotta was removed from isolation on October 26. Still, in the meantime, the case seemed to have fueled the interest of the medical community, something understandable within the growing climate of colonial anxiety regarding the spread of plague in British India at the time. Within days of reading about the Cotta case, Surgeon-Major Skinner of Calcutta's Station Hospital wrote to Cobb, confiding to him that soldiers stationed at Fort William had been suffering from nonsyphilitic buboes. Cobb visited the hospital and concluded that the soldiers in question were victims of an infection similar to the Cotta case, a verdict he claimed was confirmed by microbiological tests performed by himself and Simpson. What struck Cobb as pertinent was that the men belonged to no other than the Shropshire Regiment, famous for its antiplague work in Hong Kong two years earlier (1894). Having arrived in Calcutta in January 1895, they were reported to have had ever since suffered from "this peculiar illness and glandular swellings, new drafts of men being affected who had never been in Hong-Kong."[24] Could it be that the heroes of the Whitewash Brigade, decorated for stamping out the disease in the Crown Colony, were so insidiously infected by it, and, worse, spreading it unbeknownst to themselves across India?[25]

Faced with such questions, investigations were taken over by the Calcutta-based Scottish physician David Douglas Cunningham, known for having established the first British Indian research laboratory, aimed at the study of cholera, in Bombay in 1884.[26] In October 10, 1896, Cunningham

22. Tomes, "First Case of Plague in Howrah" (n. 20), 447.

23. House of Commons Parliamentary Papers, Cd.139 (n. 15), 490.

24. Ibid., 495.

25. On the work of the Shropshire Regiment in Hong Kong, see Robert Peckham, "Hong Kong Junk: Plague and the Economy of Chinese Things," *Bull. Hist. Med.* 90, no. 1 (2016): 32–60; Jerome J. Platt, Maurice E. Jones, and Arleen Kay Platt, *The Whitewash Brigade: The Hong Kong Plague of 1894* (London: Dix Noonan Webb, 1998).

26. Jeremy D. Isaacs, "D. D. Cunningham and the Aetiology of Cholera in British India, 1869–1897," *Med. Hist.* 42, no. 3 (1998): 279–305; Pratik Chakrabarti, *Bacteriology in British India: Laboratory Medicine and the Tropics* (Rochester, N.Y.: University of Rochester Press, 2012); Mark Harrison, "A Question of Locality: The Identity of Cholera in British India, 1860–1890," in *Warm Climates and Western Medicine: Emergence of Tropical Medicine, 1500–1900,*

was appointed to the newly founded Medical Board with his duties being "restricted to advising on bacteriological questions."[27] Headed by H. H. Risley (the government secretary for the Financial and Municipal Department), the Medical Board was composed "for the purpose of determining the action to be taken by all executive authorities, whether official or municipal, with the object of preventing the spread to Bengal of bubonic plague" from Bombay where the disease had been raging since September 1896.[28] Cunningham received cultures and bacterial preparations sent to him by Cobb and Simpson, none of which were found to "accurately correspond in character with those of type-specimens obtained from Bombay from M. Haffkine," with the presence of other microbes found therein attributed to contamination from external sources.[29] Following a special meeting of the Commissioners of Calcutta on the subject of plague, which saw Simpson subjected to pressing questions on his experience with the disease and the ability to distinguish plague clinically and bacteriologically from other disease, like mumps, the Medical Board sat on October 20, 1896, to consider the evidence.[30] Reflecting on Cunningham's verdict, it declared that "the bacteriological data available did not point to any certain conclusion as to the character of the prevailing disease."[31] In order to allay fear among the general public, fueled by a popular attribution of "any swelling of the glands to the effect of plague in cases which would not have attracted any attention in the ordinary times," the board issued a note declaring the cases not to be plague; copies of this were sent to officials and to the press.[32]

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ed. David Arnold (Amsterdam: Rodopi, 1996), 133–59. Cunningham (1843–1914) was educated in Munich, under Max Joseph von Pettenkofer. Entering the Indian Medical Service in 1868, he was appointed special assistant to the sanitary commissioner of India (1874) and professor of physiology in the Medical College in Calcutta (1879). He was noted for his contribution to heated debates about Robert Koch's theory of the etiology of cholera; Mark Harrison, *Public Health in British India: Anglo-Indian Preventive Medicine 1859–1914* (Cambridge: Cambridge University Press, 1994).

27. *History and Proceedings of the Plague Commission, Bengal. 1896 to 1898* (Calcutta: Bengal Secretariat Press, 1899), 1.

28. Ibid. In November 10, 1897, the board's designation would be changed to "Plague Commission."

29. Note by Brigade-Surgeon-Lieutenant-Colonel Cunningham, in R. Nathan, *The Plague in India 1896, 1897* (Simla: Government Central Printing Office, 1898), vol. 2, app. 1, 6–8, quotation on 7.

30. "The Plague Scare," *Amrita Bazar Patrika*, October 19, 1896, 6. As Sutphen, "Not What but Where" (n. 9), has noted, the attitude toward Simpson was part of broader suspicion on his person, especially within the Indian community.

31. The Medical Board, "Plague in Calcutta," *Indian Lancet*, November 1, 1896, 453.

32. *History and Proceedings of the Plague Commission* (n. 27), 2. For an account of popular perceptions of the "plague scare" and the role of Simpson from the viewpoint of an Ayurvedic

This sequence of events, which divided the medical community of Calcutta, would perhaps have remained but a footnote in the history of the long and devastating march of plague in South Asia had they not attracted the interest of a leading plague expert at the time, who took it upon himself to systematize information on what he coined *pestis minor*.

The Cantlie-Lowson Debate

In a lecture on the spread of plague, delivered before the Epidemiological Society of London on December 18, 1896, the Scottish physician James Cantlie explored the pressing issue of the spread of bubonic plague from South China to British India.[33] The lecture comprised a meditation upon his direct experience with the disease in Hong Kong two years earlier and a study of available data on plague from medical and colonial reports and research at the time. One of Cantlie's concerns was the exploration of the notion that rats may be related to the spread of the dreaded disease. Native ideas regarding a connection between the particular animal and what was believed by medical officers to be plague had been noted in the Indian Himalayas as well as in Yunnan over the previous decades.[34] Still the prevalent idea at the time was that the rat was merely a copatient, not a transmitter or source of plague.[35] The fact that rats had been observed

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practitioner, see "The Bubonic Plague," *Amrita Bazar Patrika*, October 26, 1896, 26. On "plague panic" in relation to this case, see "Panic Plague," *Amrita Bazar Patrika*, October 29, 1896, 5. For historical discussion of the phenomenon in India, see David Arnold, "Disease, Rumor, and Panic in India's Plague and Influenza Epidemics, 1896–1919," in *Empires of Panic: Epidemics and Colonial Anxieties*, ed. Robert Peckham (Hong Kong: Hong Kong University Press, 2015), 111–29; Rajnarayan Chandavarkar, "Plague Panic and Epidemic Politics in India, 1896–1914," in *Epidemics and Ideas: Essays on the Historical Perception of Pestilence*, ed. Terence Ranger and Paul Slack (Cambridge: Cambridge University Press, 1992), 203–40.

33. Published in the *Lancet* in two parts: James Cantlie, "A Lecture on the Spread of Plague, Delivered before the Epidemiological Society on Dec 18th, 1896," *Lancet* 149, no. 3827 (January 2, 1897): 4–7; James Cantlie, "A Lecture on the Spread of Plague, Delivered before the Epidemiological Society on Dec 18th, 1896," *Lancet* 149, no. 3828 (January 9, 1897): 85–91. James Cantlie (1851–1926) resigned from the position of surgeon at Charing Cross Hospital (1887) to accept Patrick Manson's invitation and become dean of Hong Kong's School of Medicine. Cantlie was cofounder of what would become Hong Kong University and taught Dr. Sun Yat-sen. Returning to London (1896) he took the chair of applied anatomy at Charing Cross Hospital and founded the Royal Society of Tropical Medicine and Hygiene (1907); Mark Harrison, "Cantlie, Sir James (1851–1926), Physician and Medical Administrator," in *Oxford Dictionary of National Biography* (September 2004), http://www.oxforddnb.com/view/article/50530.

34. For discussion, see Christos Lynteris, *Ethnographic Plague: Configuring Disease on the Chinese-Russian Frontier* (London: Palgrave Macmillan, 2016).

35. In 1898 Paul-Louis Simond established the rat and its flea as the respective host and vector of plague; it would take another decade for his hypothesis to be widely accepted;

to die in great numbers before the onset of human plague epidemics was largely attributed to the proximity of the animal's snout to the earth, believed to be the natural abode of the disease.[36]

Cantlie, who also believed plague to be a "soil disease," supported the idea that the disease among rats was a forerunner of it among humans. And although he avoided tackling the question regarding rats being "a means of infection," he did seem to implicate the animal's susceptibility to plague as a necessary component of true human plague outbreaks.[37] Cantlie established this by turning his attention to the controversial case of plague in Calcutta a few months earlier, noting that no rats died of the disease. The explanation for this phenomenon, he claimed, was simple: "The rats did not die, as true plague did not exist in Calcutta, the cases of fever with bubonic swellings being found to be minus a toxic bacillus."[38] "The Calcutta form of disease," he concluded, "is benign and no record of rats dying during its prevalence has ever been made."[39] In other words, what was in place was a nonvirulent, attenuated form of the bacillus, such as the one found by Alexandre Yersin in Hong Kong's soil two years earlier.[40]

Cantlie thus concluded that the presence of plague in the absence of rat epizootics constituted evidence of a benign form of the disease. Reflecting a broader mistrust of bacteriology (in the case of India, exemplified in D. D. Cunningham) and its use in knowing plague, as especially pronounced in India, Cantlie stressed that the *pestis minor* cases in Calcutta called for a cautious approach of diagnosis, and not one solely based on bacteriological evidence.[41] These cases provided evidence that "glandular swellings may attack a dweller in a district where true plague rages, and that at a subsequent period . . . may become virulent and kill the patient

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Marc Simond, Margaret L. Godley, and Pierre D. E. Mouriquand, "Paul-Louis Simond and His Discovery of Plague Transmission by Rat Fleas: A Centenary," *J. Roy. Soc. Med.* 91, no. 2 (February 1998): 101–4.

36. Hong Kong Government Gazette GA 1895 no. 146; Medical Report on the Epidemic of Bubonic Plague in 1894 (incorporating J. A. Lowson, "The Epidemic of Bubonic Plague in Hong Kong, 1894") (April 13, 1895): 369–422.

37. Cantlie, "Lecture on the Spread of Plague" (n. 33), 85.

38. Ibid., 86.

39. Ibid., 86.

40. Lynteris, "A Suitable Soil" (n. 3).

41. Cantlie's assumption of an absence of diseased rats was later contradicted by Simpson, who in a note as Calcutta's health officer stressed he had found several cases of rats suspected of dying from plague in the Burra Bazaar, where he had examined a *pestis ambulans* case; W. J. Simpson, "Calcutta and Plague, Note by the Health Officer—To the Chairman of the Calcutta Corporation," *Indian Lancet*, February 16, 1897, 181–82.

by true plague."[42] This transformation of *pestis minor* into "true plague" was judged by Cantlie to be "not at all a comfortable doctrine" insofar as the former, "larval" or "ambulatory" form of the disease was "so indefinite in its duration, so unstable in its relation to malignant plague, and so uncertain in its onset and departure" that its scientific study remained an elusive task.[43] For Cantlie, this posed epidemiologically significant questions regarding *pestis minor*'s disease ontology: "1. Does pestis minor occur as a precursor, as a collateral ailment, or a sequela of true plague? 2. Is it confined to the plague belt delineated in the map? 3. Has it anything to do with plague? 4. Is it a disease per se?"[44]

To answer these questions Cantlie compiled and assessed evidence of *pestis minor* from descriptions given during epidemics, mid-nineteenth-century outbreaks in Astrakhan and Mesopotamia, and mid-1890s cases in Hong Kong and Calcutta. He concluded that the transformation of *pestis minor* into malignant or "true" plague could require a long intervening period, with glandular swellings persisting as long as nine years before the appearance of the latter, and for as many or more after its disappearance. However, he also opined that independent *pestis minor* outbreaks often did not lead to true plague epidemics.[45] What remained a mystery for Cantlie was exactly how one type or form of plague transformed into another. His inclination was to reason that "the bacillus of the benign variety attains malignancy by passing through some intermediate host, possibly, but not probably, the rat."[46]

A little more than a month after the publication of Cantlie's influential lecture in the *Lancet*, James Lowson, superintendent of the Government Civil Hospital in Hong Kong during the 1894 outbreak, published a scathing response. Continuing a personal war that had started while the two doctors were battling the Hong Kong plague, Lowson accused Cantlie of "inaccuracies regarding the Hong-Kong epidemic," as well as of having opinions that people more experienced with plague than himself did not share.[47] Among the points attacked was Cantlie's alleged confusion of *pestis minor* with *pestis ambulans*. Following Lowson, while the former

42. Cantlie, "Lecture on the Spread of Plague" (n. 33), 87.

43. Ibid., 87, 88.

44. Ibid., 5. The "belt" stretched from Yunnan through the Himalayas into Northwest India, Persia, Mesopotamia, and the mouth of the Volga.

45. Cantlie, "Lecture on the Spread of Plague" (n. 33), 87, 88.

46. Ibid., 91.

47. James A. Lowson, "Some Remarks on Plague," *Lancet* 149, no. 3833 (February 13, 1897): 439–42, quotation on 439. On Lowson's role in the Hong Kong outbreak, see T. Solomon, "Hong Kong, 1894: The Role of James A. Lowson in the Controversial Discovery of the Plague Bacillus," *Lancet* 350, no. 9070 (July 5, 1997): 59–62.

is "bubonic plague which occurs (?) before an outbreak of plague," the latter consists in "glandular enlargements occurring during or after an epidemic which may probably be due to a less potent infecting power."[48] Lowson claimed that, according to his clinical experience, benign cases of fever and inguinal buboes appearing in Hong Kong before 1894 (classified by Cantlie as *pestis minor*) were in fact venereal in origin, and, in the case of children, mumps-related parotitis.[49] Rejecting the very notion of *pestis minor*, Lowson noted that, nonetheless, a form of ambulatory plague, called *pestis ambulans*, did in fact occur. This he attributed tacitly to a possible absorption into the human body of "a small amount of toxin . . . without the necessary entrance of bacilli, as the products of the bacilli may hang about a neighbourhood long enough after all bacilli are dead."[50]

Lowson's wrath against Cantlie's notion of *pestis minor* can be understood in the context of the former's long-standing antagonism with the idea that plague could be retained in nonvirulent form and reemerge, having regained its virulence, after some time so as to cause new outbreaks. This theory, which originally framed the soil as the receptacle of this attenuated form of plague, had been first formulated in 1894 by Alexandre Yersin in Hong Kong and was enjoying considerable popularity in British India at the time.[51] In the context of a most acute animosity against Yersin, whose research he had sought to obstruct in favor of the work of the Japanese rival discoverer of the bacillus, Kitasato, Lowson had violently attacked the notion before a special committee in August 1894.[52] Could *pestis minor* have represented for him a return to the notion he had battled against in Hong Kong, led no less by one of Yersin's few supporters in the course of the 1894 outbreak, James Cantlie? After all, it had been only five years since Yersin and Roux had argued that the pseudo-diphtheria bacillus, which was commonly found in human bodies, was not a separate species, as Robert Koch maintained, but an attenuated form of the diphtheria bacillus.[53] Lowson's retort underlined a common

48. Lowson, "Some Remarks on Plague" (n. 47), 439.

49. James Cantlie later adopted a differentiation between the two terms, claiming that *pestis ambulans* is a mild form of the disease observed in the course of an outbreak, whereas *pestis minor* is "possible a distinct disease" involving nonvenereal buboes and preceding true plague outbreaks. Cantlie claimed *pestis minor* could be treated by removing the infected glands; James Cantlie, "Plague: Its Symptoms and Spread," *Pub. Health* 13 (October–September 1900–1901): 165–75, quotation on 166.

50. Lowson, "Some Remarks on Plague" (n. 47), 440.

51. Lynteris, "A 'Suitable Soil'" (n. 3).

52. Ibid.

53. J. Andrew Mendelsohn, "'Like All That Lives': Biology, Medicine and Bacteria in the Age of Pasteur and Koch," *Hist. Philos. Life Sci.* 24, no. 1 (2002): 3–36.

epistemic thread between the possibility that the soil retained and even spread plague and the ability of plague to hide in an attenuated form in the human body, from which it could reemerge and strike again.

What was so alarming about this epistemic transference of plague's ability to become naturally attenuated between a geological and a physiological register (the soil and the human body) becomes clear in the writings of another leading plague expert at the time. In his seminal work on the Venice Sanitary Conference of 1897, the French doctor Adrien Proust complained about the implications of differentiating between what he called sporadic and epidemic plague. He denounced the prevalent idea that the two bore different symptoms and that the former is not contagious whereas the latter is. If that were true, Proust warned, it would mean that in the former case all quarantine and isolation measures could be defied with impunity. The very idea offered itself to a collapse of painstakingly achieved and maintained interstate agreements and public order in the course of epidemics. Proust hence insisted that the only difference between the two forms of plague is one of *degree*, regarding how many people are affected, and was not truly typological.[54] Far from being academic, the question of this "larval" form of plague was thus related to pressing issues of epidemic control.[55]

When then, in 1898, plague finally struck Calcutta, questions about Simpson's 1896 diagnosis and Cantlie's theory of a dangerous, silent form of the disease were immediately raised.[56] Were the disputed cases from 1896 and the 1898 outbreak connected? Were Simpson's and Cantlie's warnings ineptly and dogmatically ignored?[57] Or were the cases in fact unconnected with Simpson's microbes, these being nothing more that "common atmospheric bacilli," as the head of the Calcutta Medical Board, Risley, maintained before the Bengal Legislative Council?[58] The agency ultimately responsible for adjudicating on such matters was a body of medical practitioners and colonial officers under the direction of Thomas R. Fraser, known as the Indian Plague Commission (est. November 26, 1898, also known as the Fraser Commission).[59]

54. Adrien Proust, *La défense de l'Europe contre la peste et la conférence de Venise de 1897* (Paris: Masson, 1897).

55. Cantlie, "Lecture on the Spread of Plague" (n. 33), 88. The notion of "larval" plague was also used in the daily press to refer to *pestis minor*. See, for example, Anon., "Plague and Its History," *Queenslander* (June 21, 1902): 1370.

56. *Calcutta Gazette*, "Extraordinary," April 30, 1898, Medical Department.

57. See, for example, Anon., "Calcutta and the Plague," *Times of India*, April 27, 1898, 4.

58. Anon., "Plague in Calcutta," *Indian Lancet*, May 16, 1898, 512–14, quotation on 514.

59. Not to be confused with the early twentieth-century Indian Plague Commission, which published its research results in the *Journal of Hygiene*. For a discussion on the differences

Simpson's Defense

Gathering evidence on the Indian plague epidemic by means of interviews with a range of witnesses, Fraser's Commission set out to clarify the clinical, bacteriological, and epidemiological profile of the outbreak.[60] When it came to the issue of the 1896 Calcutta cases, the commission collected past reports and telegrams, while summoning key players in the Cotta and Shropshire Regiment cases. In the course of the proceedings, Cobb and Simpson's identification of the glandular swellings of the Shropshire Regiment men as resulting from a mild form of plague was openly contested before the commission in January 1899 by Major B. M. Skinner, the man who had initially invited them to examine the cases in Fort William. Skinner claimed the bacilli cultivated from his regiment's men, including from buboes that he himself suffered, were not the same as the Bombay bacilli sent over by Haffkine.[61] Skinner appeared particularly agitated before the commission by the notion that he, a senior officer, stood suspect of having carried (or even imported) the disease, and declared similarly that the Cotta bacilli bore no resemblance to the true bacillus but were in fact streptococcal. Following Skinner's scathing testimony, Simpson would be subjected to an evidential grilling by the commission. On May 6, 1899, the doctor was called as a witness, though the tone of the "minutes of evidence" suggests that his role was more that of an accused.[62] In spite of the deriding questioning, Simpson stood his ground, providing as evidence in support of his diagnosis images of bacterial cultures developed from the Shropshire Regiment incident. Still, arguing that the bacteriological evidence on which he based his report were "fallacious," the commission claimed that the buboes observed by Simpson were "climatic" and "stood in no connexion at all either with the fact that the Shropshire Regiment had previously been engaged on plague duties in Hong Kong, or with the fact that plague afterwards broke out in Calcutta."[63] This was a humiliat-

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between the two commissions, see Nicholas H. A. Evans, "Blaming the Rat? Accounting for Plague in Colonial Indian Medicine," *Med. Anthrop. Theory* 5, no. 3 (June 2018): 15–42; http://doi.org/10.17157/mat.5.3.371. On the international plague commissions operating in India at the time, see Harrison, *Public Health in British India* (n. 26).

60. The commission concluded its interviews in March 1899, having conducted seventy sessions with 260 witnesses (or a total of 27,415 questions); Evans, "Blaming the Rat?" (n. 59).

61. House of Commons Parliamentary Papers, Cd.140, Indian Plague Commission, 1898–99, Minutes of Evidence Taken by the Indian Plague Commission with Appendices, vol. 2, Evidence Taken from 11th January 1899 to 8th February 1899, 129.

62. For a daily press coverage of Simpson's testimony, see Anon., "The Plague Commission," *Times of India*, May 29, 1899, 7.

63. House of Commons Parliamentary Papers, Cd.810, Indian Plague Commission, 1898–99, Report of the Indian Plague Commission with Appendices and Summary, vol. 5,

ing moment for Simpson, who sought to defend his position through an article published in September 1899 in the *British Medical Journal*.[64] This "indiscreet act" so vexed the Calcutta Medical Board that it "took steps to prevent the recurrence of any such undesirable publication by bringing the matter to the notice of Government."[65]

What is striking in Simpson's article is that, rather than defending his diagnosis of the Calcutta cases, he opted to support the category of *pestis minor* itself. This he did by reference to historical rather than firsthand bacteriological or clinical evidence. As we have already seen, such evidence had been first put forward by James Cantlie in his December 1896 lecture. Simpson was certainly aware of these, as during his Indian Plague Commission session he made passing reference to J. D. Tholozan's studies of plague in Mesopotamia, indicating "that from epidemic to epidemic the bridge is filled up by these mild cases of plague, which produce very little constitutional disturbance."[66] In his *BMJ* article such historical evidence lost its peripheral character and instead assumed central stage, with emphasis placed on the Astrakhan outbreak of 1877. Without providing bibliographical references, Simpson asserted that the outbreak on the mouth of the Volga affected more than two hundred individuals without leading to a single death.[67] As was also true in the cases he had examined in Calcutta, "beyond the inconvenience and discomfort caused by the buboes . . . the general symptoms were not such as to prevent the patient from moving about."[68] Simpson's narrative proceeded by relating that a year after the "glandular sickness of Astrakhan" erupted the devastating outbreak of plague in Vetlyanka (north of Astrakhan, on the banks of

149. For a later review of the notion of climatic buboes, see Botho Scheube, *The Diseases of Warm Countries: A Handbook for Medical Men*, 2nd rev. ed., trans. Pauline Falcke (London: John Bale, Sons & Danielsson, 1903). For a discussion of climatic buboes and endemic forms of plague in Reunion, see André Thiroux, "Peste endemique et bubons climatiques: lymphangite infectieuse de la Réunion et érysipèle de Rio," *Annales de l'Institut Pasteur* 19 (1905): 62–64.

64. Later, in his seminal *Treatise on Plague* (n. 8), 436, Simpson canonized *pestis minor* as a "benign form" of plague, with his only concession being that no agreement exists as to its frequency.

65. *History and Proceedings of the Plague Commission* (n. 27), 3.

66. House of Commons Parliamentary Papers, Cd.141, Indian Plague Commission, 1898–99, Minutes of Evidence Taken by the Indian Plague Commission with Appendices, vol. 3, Evidence Taken from 11th February 1899 to 20th May 1899, 379; Joseph Désiré Tholozan, *Une épidémie de peste en Mésopotamie en 1867* (Paris: Victor Masson et Fils, 1869).

67. In his *Treatise on Plague* (n. 8), Simpson (159) would later claim there was one victim.

68. William John Simpson, "Plague: Its Symptomatology, Pathology, Treatment and Prophylaxis," *Brit. Med. J.* 2, no. 2020 (September 16, 1899): 697–99, quotation on 697.

the Volga).[69] Simpson speculated about the connection of the two events, claiming that it made evident an epidemiological pattern that appeared to be similar to the one concerning the 1896 and 1898 plague-related events in Calcutta.

Three sources on this information, all of them before the bacteriological identification of plague, appear most likely to have been available to Simpson. The first was an 1879 British report on Levantine Plague, which commented on the 1877 outbreak in Astrakhan: "This malady has been since regarded as an abortive form of plague (*peste frustre* of the French physicians who have recently visited the district; *pestis nostras* of the local physicians; *pestis ambulans* as Dr. Arkhangelsky has termed it)."[70] This attenuated form of plague was said to be characterized by fever and submaxillary buboes, with the symptoms being so mild that "the patient rarely took to bed."[71] The report referred to the 1879 paper by G. F. Arkhangelsky, where the Russian doctor drew an extensive discussion of "ambulatory plague" (the term *pestis ambulans* never being used in the article besides its title), describing it is a condition where "patients tolerated the plague, almost without looking up from their usual occupation or, as they say, on the go."[72] Painting a picture of medical knowledge of this "treacherous" form of plague across the centuries, Arkhangelsky stressed that the sequence of the Astrakhan and Vetlyanka outbreaks signaled that ambulatory plague "deserves public attention and that after the appearance of apparently inoccuous forms may suddenly develop an epidemic posing a serious threat to public health."[73] The second and more detailed source was the 1881 article published by Joseph Frank Payne, who in his report on the Vetlyanka outbreak also provided information on the Astrakhan incident.[74] Attributing the notion *of pestis nostras* (that is, "our plague" or

69. Ibid., 697. For historical analyses of the Volga outbreaks, see Hans Heilbronner, "The Russian Plague of 1878–79," *Slavic Rev.* 21, no. 1 (March 1962): 89–112; Maria Pirogovskaya, "The Plague at Vetlyanka, 1878–1879: The Discourses and Practices of Hygiene and the History of Emotions," *Forum Anthropol. Cult.* 10 (2014): 133–64.

70. House of Commons Parliamentary Papers, C.2262, Plague, Papers Relating to the Modern History and Recent Progress of Levantine Plague; Prepared from Time to Time by Direction of the President of the Local Government Board, with Other Papers.

71. Ibid.

72. G. F. Arkhangelsky, "Ambulantnaya forma chumy (*Pestis ambulans*) i ee znachenie v epidemiologii" [The Ambulatory Form of Plague (*Pestis ambulans*) and Its Significance in Epidemiology], *Sbornik sochinenii po sudebnoi meditsine, sudebnoi psikhiatrii, meditsinskoi politsii, obshchestvennoi gigiene, epidemiologii, meditsinskoi geografii i meditsinskoi statistike* 1 (1879): 132–93, 145 (my translation).

73. Ibid., 156, 186.

74. Born in 1840 in Surrey, Joseph Frank Payne was a physician. A fellow of the Royal College of Physicians since 1873, he worked in several London hospitals. At the same time

native plague) to the Russian doctor Janizky (*sic*), Payne supported the idea that the Astrakhan illness that "slumbered for more than a year . . . was the same that re-appeared in the earlier and milder stages of the epidemic in Vetlanka [*sic*]."[75] He concluded that plague could take two forms: a "mild, non-fatal form" affecting the lymphatic system, and a malignant highly transmissible and fatal form. Payne believed that, though harmless in its natural environment, the mild form of plague might become dangerous once introduced "into thickly populated places, its malignity being heightened."[76] This epidemiological reasoning was reflected in the third probable source: the detailed report on Dr. Zuber's medical mission to Vetlyanka. There the French medical envoy noted the use of the term *pestis nostras* by local doctors and their warning that, on account of it, Astrakhan may "soon become the second motherland of plague."[77] Evidently closely read by Payne, Zuber went on to attribute the term *peste frustre* (literarily, "frustrated plague") to Istanbulite doctors, who had been familiar with this form of the disease in "the periphery of plague foyers" or in localities where plague is endemic.[78] To those who objected that "in light of modern science" a disease as powerful as plague could not possibly take such a benign form, Zuber retorted that "*pestes frustres*, are well-established epidemiological facts, which I do not undertake to explicate or to agree with the common doctrines: I confine myself to observing their existence."[79]

Payne's urge to study the transition between the mild, endemic and the malignant, epidemic form of the disease was particularly well reflected in Simpson's *BMJ* article, where he claimed that "the elucidation of *plague ambulans* is of the greatest importance from an epidemiological point of

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he authored historical works on Thomas Sydenham, Galen, and others. Payne maintained a keen interest in plague. He was dispatched to the Volga with Surgeon-Major Colvill, as part of the English commission investigating the Vetlyanka outbreak; T. H. Pennington, "Payne, Joseph Frank (1840–1910)," in *Oxford Dictionary of National Biography* (September 2004), http://www.oxforddnb.com/view/article/35424?docPos=2.

75. Joseph F. Payne, "On Certain Points Connected with the Epidemic of Plague in the Province of Astrakhan, Russia, in the Winter of 1878–79," *Trans. Epidemiol. Soc. London* 4, no. 3 (1880): 362–75, quotation on 374.

76. Ibid., 374.

77. C. Zuber, "Rapport sur une missione médicale en Russie; La peste du gouvernement d'Astrakhan," in *Recueil des travaux du Comité Consultatif d'hygiène publique de France et des actes officiels de l'administration sanitaire* (Paris: A. Lahure, 1880), vol. 9, 140.

78. Ibid., 148.

79. Ibid., 149. A foreign medical envoy to the Vetlyanka outbreak, August Hirsch downplayed the connection between the latter and the ambiguous Astrakhan outbreak; August Hirsch and M. Sommerbrodt, *Mittheilungen über die Pest-Epidemie im Winter 1878–1879 im russischen Gouvernement Astrachan* (Berlin: Carl Heymann's Verlag, 1880).

view."[80] Indeed Simpson doubted that the mild form of the disease was noncommunicable, noting that this is "a pretty theory" but in fact no systematic study had been made on the way it spread.[81] He thus reasoned that "*pestis minor* or *pestis ambulans* is, if anything, more insidious and dangerous to the community at large than the pneumonic, for its mildness produces no sense of danger."[82] For, as such cases "crop up unconnected to one another," doctors were led to ignore this form of the disease, believing it unimportant or harmless; it is thus that "the disease gains a firmer hold on the locality, and may later develop into a more virulent type."[83]

The Indian Plague Commission

It is tempting to see Simpson's effort to safeguard his scientific prestige by defending the category of *pestis minor*, rather than his personal diagnosis of the Calcutta cases, as a last resort tactic by a man attacked from all sides. Yet such approach would be overlooking the importance of shifting between evidential registers on two accounts: first, as regards Simpson's subsequent long and illustrious career as an author who in many ways attempted to "fix" plague both pathologically and epidemiologically for his contemporary audiences; and second, as regards the broader milieu of epidemiological writing at the turn of the century. Rather than simply being a form of rhetorical retreat, this marked the ambivalence of epidemiological evidential hierarchies, a trait most clearly observed in the Indian Plague Commission's own work. As Nicholas Evans has argued, the Fraser Commission followed a process that rendered plague a knowable category through the extraction and categorization of particular forms of speech from British, native, medical, and lay witnesses of the epidemic.[84] There, ethnographic, geological, and climatological information, clinical case studies, etiological theories, and administrative accounts were streamlined in a Q&A extraction of evidence regarding not so much plague's identity, as its epidemiological and pathological possibilities.[85]

80. Simpson, "Plague" (n. 68), 698. Payne's ideas about *pestis minor* were reflected in popular and missionary accounts at the time; see, for example, George Lambert, *India, the Horror-Stricken Empire* (Elkhart, Ind.: Mennonite Publishing, 1898).

81. Simpson, "Plague" (n. 68), 698.

82. Ibid., 697. In his 1905 *Treatise on Plague* (n. 8), Simpson returned to plague on the Volga to stress its importance as an example of plague suddenly acquiring virulence and transforming from a benign to a malignant form.

83. Simpson, "Plague" (n. 68), 697, 698.

84. Evans, "Blaming the Rat?" (n. 59).

85. I am indebted to Nicholas Evans for this observation.

It is from this perspective that what may first appear as a paradoxical conclusion regarding *pestis minor* was reached by the commission. For having demolished the evidential architecture supporting the existence of any form of plague in the Calcutta cases, the commission did not opt to also abandon the notion of *pestis minor* itself, but instead supported it, concluding that "in addition to the three main types of plague . . . abortive form of bubonic plague comes under observation. This is technically known as *pestis minor*, or *pestis ambulans*."[86]

Indeed the Indian Plague Commission sought advice on *pestis minor* from a wide range of witnesses. This included not only the procurement of direct evidence but also thought experiments, such as the one put to the Parsee medical luminary and editor of the *Indian Medico-Chirurgical Review*, Nasarwanji Hormusji Choksy, on February 22, 1899.[87] The commission asked Choksy how he would diagnose a case of *pestis ambulans* among the Shropshire Regiment men. Choksy replied that bacteriological evidence being of no use in this case, he would perform an "inferential" diagnosis by "tak[ing] into consideration whether they have been in localities which have been infected, or whether they have come in contact with plague patients, or whether they have remained in areas which are infected, in which case they will certainly be looked upon as suffering from *pestis ambulans*."[88]

What characterized this pathology, according to the commission, was the presence of the bacillus and, at the same time, its retention in the lymphatic glands, with "the disease stopping short of the septicaemic stage."[89] As a consequence, no deaths were believed to occur. This "phantom form" of plague, as it was coined, was held to be "extremely common among persons who have been much exposed to the infection of plague," with its main symptoms being "sensations of numbness and tingling, or by neuralgic pains, which in many cases are associated with the development of shotty glands in the armpits and the groins."[90] Attributing the

86. House of Commons Parliamentary Papers, Cd.810 (n. 63), 54.

87. On Choksy, see Chakrabarti, *Bacteriology in British India* (n. 26); M. Ramanna, "Nasarwanji Hormusji Choksy (1861–1939), a Pioneer of Controlled Clinical Trials," http://www.jameslindlibrary.org/articles/nasarwanji-hormusji-choksy-1861-1939-a-pioneer-of-controlled-clinical-trials/.

88. House of Commons Parliamentary Papers, Cd.141 (n. 66), 121. Choksy had earlier maintained *pestis minor* and *pestis ambulans* to be two separate forms of plague (out of a total of six types in existence, in his opinion), the difference between them being that *pestis minor* was "mild plague" whereas *pestis ambulans* "a longer continuing but not very fatal form of the disease"; Anon., "The Arthur Road Hospital," *Times of India*, February 19, 1898, 4.

89. House of Commons Parliamentary Papers, Cd.810 (n. 63), 54.

90. Ibid., 423, 54.

transformation of this condition into "true plague" to a "loss of resisting power" in the afflicted individual, the commission gave for the first time a clear clinical definition of *pestis minor*:

> The patient suffers from headache and sleeplessness, from slight pyrexia, lasting only a few hours, from tenderness and pain over one or more superficial glands, usually those in the groin, which are frequently enlarged, and occasionally from nausea and vomiting. The duration may be as brief as only two or three days, but not infrequently the enlargement of the glands is more persistent, and the duration on this account, or because suppuration has taken place in the glands, is protracted to 10, 20 or even 30 days. All cases terminate in recovery.[91]

What interested the Indian Plague Commission in particular was the question of the infectivity of this type of plague. Choksy, a doctor with considerable experience on plague through his work at the Arthur Road Hospital (Bombay), stressed that in his private practice he had never witnessed this form of plague spreading from human to human.[92] Nonetheless, the commission noted that in one case, from Gobindpur in the Punjab, evidence procured by Captain James of the IMS pointed out that the epidemic in the area had in fact spread from a boy suffering from this attenuated form of the disease.[93] In an effort to explain the incident, the commission argued that though *pestis minor* was not infective while in its benign stage, there might be cases where it became contagious as a result of "suppuration and discharge of pus . . . during convalescence."[94]

The Indian Plague Commission's report thus paradoxically signaled a rebutting of the foundational evidence of *pestis minor* and, at the same time, confirmed its retention as a valid pathological category of potential epidemiological importance. The remote possibility of *pestis minor* infectivity, in combination with "the endemic or epidemic character" of this type of plague, led the commission to propose that, although it was of little importance for individuals afflicted by it, *pestis minor* was very significant when it came to antiplague measures, as "the occurrence [of *pestis minor* cases] in the absence of an epidemic of virulent plague may be the prelude to an ordinary epidemic, while their occurrence at the termination of such an epidemic may serve as an indication of the danger of recrudescence."[95]

91. Ibid., 88, 423. Colonel Fawcett and Captain Morgan also noted other symptoms among Europeans engaged in antiplague work; these "about half an hour after handling plague patients, suffered from painful tingling and numbness in the hands, which extended to the lymph glands of the axilla, neck or groin, the glands becoming extremely painful, but being neither swollen nor tender" (ibid., 424). These were said to then disappear within a few hours.

92. House of Commons Parliamentary Papers, Cd.141 (n. 66), 135.

93. House of Commons Parliamentary Papers, Cd.810 (n. 63), 97.

94. Ibid., 97.

95. Ibid., 424.

This was no less than an explicit and official connection of the question of *pestis minor* with the overarching question of plague recrudescence. Conceived as a larval form of the disease harbored in the human body in an attenuated form that could reacquire virulence and strike later due to a change in (or of) the environment, *pestis minor* was problematized in terms of the pressing question of what made plague disappear and reappear in a given city or neighborhood. Complementing theories regarding true recrudescence in the soil, and yet in contrast to their telluric restrictions, *pestis minor* thus came to evidentialize the reappearance of the disease in a given locus after a long epidemic absence, not as a result of importation, nor however simply in terms of its "breeding grounds," but as an outcome of the fundamentally transformative character of plague.

Attenuation and Transformation

Hollowed out of its foundational evidential bases, *pestis minor* was declared by Fraser's Indian Plague Commission to be bacteriologically undecipherable and epidemiologically undecidable. And yet the category did not perish, but instead persisted in international medical reports and studies well into the final years of the third plague pandemic, in the late 1940s, drawing to itself and in turn generating evidence about plague even after the rat and its flea were conclusively accepted as a key host and vector respectively of the disease (ca. 1905).

The supposed fact that *pestis minor* affected individuals ever so mildly that they did not seek medical help and generally escaped medical attention haunted both medical and lay understandings of plague. Fanning medical and colonial anxieties about controlling tropical, subaltern bodies, the concern about this "phantom" form of plague was further fostered by reports of "unrecognized cases or means of infection" lurking in European cities, such as Porto and Glasgow.[96] Yet in the absence of any connection in medical literature between *pestis minor* and debates about carrier states of diseases like typhus or cholera, and indeed of any use of the word "carrier" when referring to the human hosts of this form of attenuated plague, the question arises: can the persistence of this category be simply attributed to the fear of unseen spreaders of known diseases, as famously embodied a few years later by "Typhoid Mary"?[97]

Turning my focus on the properties of the bacillus that allowed for the coexistence and entanglement of the two forms of the disease, I argue here that the productive category of *pestis minor* derived from scientific concerns associated with attenuation. On the one hand, as has been

96. Anon., "Pestis Minor," *J. Amer. Med. Assoc.* 36, no. 19 (May 11, 1901): 1328–29, quotation on 1329.

97. *Pestis minor* did not feature in discussions of natural immunity to plague at the time.

extensively examined by historians of vaccination, attenuation formed a cornerstone of Pasteurian medicine.[98] Pratik Chakrabarti in particular has shown that the ability to produce microorganisms with reduced virulence for the use of vaccines, and thus transform them from pathogenic into prophylactic agents, relied upon, and in turn reinforced, a divide between, on the one hand, "live" microbes, imbued in Pasteurian morality and ideology with "heroic potency," and, on the other hand, "dead" microbes, imbued with notions of "sterilized safety."[99] In the "experimental theatre of vaccines" that was British India, this dialectic was entangled with ideas about the tropics, and in particular with a fear of virulence, which was seen as a microbial property particularly pronounced in tropical climes and the bodies of indigenous subjects.[100] Following Chakrabarti, as "colonial bacteriology developed in the confluence of Victorian imperialism and the Pasteurian revolution," it assumed a particular "moral ascendancy" by "promising to identify and cleanse the germs of the tropics."[101]

On the other hand, the hygienic utopia embodied by humanity's newly acquired ability to harness microbial attenuation—and thus control tropical/native related virulence—by transforming it from a natural phenomenon into a medical technology relied precisely on the idea that, in the first place, attenuation was not merely an artifice, but a natural potentiality of microorganisms instead. As Andrew Mendelsohn has argued, "The attenuation and return to virulence of bacteria was understood as biological variation," with Pasteur himself suggesting that "his method of vaccine-making might hold the key to these vast and ever-perplexing natural historical phenomena of pestilence."[102] In particular, Pasteur held that the transmission of naturally attenuated pathogens through successive hosts might lead to the rebirth of their virulence.[103] Variable virulence was indeed not only "the signature of disease explanation at the Institut Pasteur" but also the exegetic field that allowed Pasteurianism, hygienism, and tropical medicine to function together rather than apart.[104] If

98. Arthur M. Silverstein, *A History of Immunology* (San Diego: Academic Press, 1989); Pauline M. H. Mazumdar, *Species and Specificity: An Interpretation of the History of Immunology* (Cambridge: Cambridge University Press, 1995); Jennifer Keelan, "Risk, Efficacy, and Attenuation in Debates over Smallpox Vaccination in Montreal 1870–1876," in *Crafting Immunity: Working Histories of Clinical Immunology*, ed. Kenton Kroker, Jennifer Keelan, and Pauline M. H. Mazumdar (Abingdon: Ashgate, 2008), 29–53; Anne Marie Moulin, "La métaphore vaccine. De l'inoculation à la vaccinologie," *Hist. & Philos. Life Sci.* 14, no. 2 (1992): 271–97.

99. Chakrabarti, *Bacteriology in British India* (n. 26), 144.

100. Arnold, *Colonizing the Body* (n. 11).

101. Chakrabarti, *Bacteriology in British India* (n. 26), 26, 36.

102. Mendelsohn, "'Like All That Lives'" (n. 53), 4, 8.

103. This was clearly reflected in Cantlie's concerns about rats as catalysts of plague's virulence discussed above.

104. Mendelsohn, "'Like All That Lives'" (n. 53), 9; Michael A. Osborne, *The Emergence of Tropical Medicine in France* (Chicago: University of Chicago Press, 2014).

for doctors at the junction of these medical traditions, natural attenuation was the mechanism through which epidemics waned and ended, *pestis minor* represented a distinctly more pessimistic option: the ability of microorganisms, in this case plague, to attenuate themselves and thus allow plague to escape human detection, by an act of spontaneous and sudden transformation between benign and malignant forms, or *pestis minor* and *pestis major* respectively.

In the following decades, medical experts across the globe would return time and again to this transformative character of plague (both as a bacterium and as a disease) and to the notion of *pestis minor*, procuring and interpreting evidence in accordance to its imagined role in the maintenance and spread of plague. At times, the term would be used in tandem with *pestis ambulans*, as synonyms, while in others a differentiation between the two would be maintained, reflecting Choksy's typology. Rather than being confined to British India, the notion would be adopted and employed across the globe. We thus find that in the course of the Paris plague outbreak of 1920, known as "the plague of ragpickers," 30 percent of cases examined in the Claude Bernard Hospital were diagnosed as "ambulatory plague."[105] In his thesis *Les formes ambulatoires de la peste*, Alfred-Joseph-Auguste Rio discussed his clinical and bacteriological studies of the above-mentioned cases.[106] Describing these as showing a "nearly complete absence of general symptoms," he nonetheless focused his efforts on identifying reliable diagnostic tools for "discovering plague in its ambulatory forms."[107] We equally find, twenty years later and on the other side of the globe, the leading Chilean plague expert Atilio Macchiavello putting together clinical, bacteriological, and epidemiological evidence so as to argue that "ingua de frío" (cold inguinal bubo), as it was reportedly called in Brazil's Minas Gereas State, was a form of ambulatory plague present "generally in children under 15" that "appears sporadically where plague is endemic and tends to disappear when epidemic," with a previous presence among rats.[108] Indeed we should not overlook the fact that, as a capacious and fluid category, *pestis minor* offered itself not only to medical experts but to lay opinion too. In the aftermath of a

105. Alfred-Joseph-Auguste Rio, *Les formes ambulatoires de la peste—Étude clinique et bactériologique* (Angers: Imprimerie Centrale, 1921).

106. Not to be confused with Alejandro del Rio, the Chilean plague expert.

107. Rio, *Les formes ambulatoires de la peste* (n. 105), 15, 22.

108. Atilio Macchiavello, "Some Special Epidemiological and Clinical Features of Plague in Northeastern Brazil," *Public Health Reports (1896–1970)* 56, no. 33 (1941): 1657–61. In *Contribuciones al Estudio de la Peste Bubonica en el Nordeste del Brasil, Oficina Sanitaria Panamericana*, Publicación No. 165 (Washington, D.C., August 1941). See also Atilio Macchiavello, "La ingua de frío o Febre de caroco es una forma de peste ambulatoria" in *Contribuciones al Estudio de la Peste Bubonica en el Nordeste del Brasil*, no. 165 (Washington, D.C.: Pan American Sanitary Bureau, 1941), 230–42.

plague outbreak, the commissioner of public health in Queensland, Dr. Ham, gave a paper at the Medical Congress at Hobart, titled "The Spirit of Hygeia in Australia." Ham wrote in the context of popular interpretations of *pestis minor* as referring to a glandular condition unrelated to plague. This interpretation, following Ham, had led to the contention that "all our plague cases, including fatal pneumonic cases, were only 'pestis minor,'" thus not necessitating antiplague measures. In reaction to this popular use of the notion, Ham mobilized a wide range of evidence from historical and recent cases so as to reassert that the *pestis minor* is different from plague in terms of degree not of kind and to stress that it "is, if anything, more dangerous than plague in its severer aspect, inasmuch as it lulls to a sense of security unjustified by the circumstances."[109]

The examples are numerous; however none makes so clear why *pestis minor* remained such a capacious and productive category, allowing doctors and public health experts to explore, reason about, and debate plague's supposedly transformative nature, as its reemergence in relation to the epidemiological problematization of plague in Hong Kong. There, following the initial outbreak of 1894, the disease continued to afflict the colony on nearly annual bases until 1923. The annual circle of plague in the Crown Colony, appearing in spring and disappearing in the late summer, had already been the object of much concern and study by 1903, when the colony's governor Henry A. Blake, an official with an intense interest in the disease, published his memorandum on plague.[110] There, Blake stated in no ambiguous terms that what carries the disease over the winter is nothing less than dormant human cases suffering from a form of chronic plague. To support this opinion, the governor referred to Simpson's observation on chronic plague among animals, in combination with the discovery of cases where, upon death resulting from an accident, bacteriological examination of the otherwise hitherto healthy men's blood the latter was found to contain plague bacilli. To give but a brief a description of one of the many cases cited, when a worker was killed

109. Anon., "Plague and Its History" (n. 55), 1370.

110. House of Commons Parliamentary Papers, Cd.1821, Colonial Reports—Miscellaneous, no. 25, Hong Kong, Bubonic Plague, Memorandum on the Treatment of Patients in Their Own Homes and in Local Hospitals, December 1903. For studies of Blake's other interventions in the field of plague, see Robert Peckham, "Matshed Laboratory: Colonies, Cultures, and Bacteriology," in *Imperial Contagions: Medicine, Hygiene, and Cultures of Planning in Asia*, ed. Robert Peckham and David M. Pomfret (Hong Kong: Hong Kong University Press, 2013), 123–47; Christos Lynteris, "Suspicious Corpses: Body Dumping and Plague in Colonial Hong Kong," in *Histories of Post-mortem Contagion: Infectious Corpses and Contested Burials*, ed. Christos Lynteris and Nicholas H. Evans (London: Palgrave Macmillan, 2018), 109–33.

by a bag of sugar falling on him in the Tal Koo sugar plant, postmortem examination showed his blood to contain the bacillus. These incidents led Blake to order Inspector Gidley to collect random samples of blood from 1,110 individuals between June 23 and July 10, 1903. Upon examination, the government bacteriologist, William Hunter, reported back that five samples unambiguously contained the plague bacillus. Blake reasoned that, this accounting for 4.54 percent of the overall tested sample, the number of infected but dormant individuals among the colony's "working coolie population alone" stood at 8,172 persons. Rather than taking this to be in itself a scientifically valid deduction, however, the governor used it to urge further scientific investigation, especially in light of the consequences the existence of such form of the disease could have on the effectiveness of quarantine.

Blake's challenge was indeed taken up, yet not by scientists in Hong Kong but by those in Manila instead. There, Maximilian Herzog and Charles Hare sought to replicate Blake's test, using bacterial cultures rather than simple microscopic examination.[111] Acknowledging that whereas plague appeared to be endemic in Manila, it was of a much lower intensity than in Hong Kong (in 1903 the former had 198 cases whereas Hong Kong had 1,415), they proceeded to test 195 Filipino and 50 Chinese "apparently healthy" individuals from infected districts of the city.[112] None of these tested positive for plague, leading Herzog and Hare to conclude that Blake's hypothesis was untenable.

And yet the two bacteriologists noted that while their investigation showed that no such thing as latent or dormant plague existed, it did confirm the existence of so-called ambulatory plague. Claiming that this should not be mistaken as a "latent" type of the disease, in the strict sense of the term, they nonetheless conceded that, as previous studies had shown, this was a form of the disease that "escape[s] detection in the absence of subjective symptoms."[113] The case they procured as proof of

111. M. Herzog and C. B. Hare, "Latent and Ambulatory Plague," *J. Amer. Med. Assoc.* 42, no. 24 (December 10, 1904): 1781–88.

112. In Herzog and Hare's calculation the plague cases in Hong Kong in 1903 numbered 1,135. I derived the corrected number from Hong Kong Government Administrative Reports, AR 1929, Medical and Sanitary. Warwick Anderson notes that ambulatory plague had been a concern in the Philippines since April 1901, as a condition especially affecting the Chinese; Warwick Anderson, *Colonial Pathologies: American Tropical Medicine, Race, and Hygiene in the Philippines* (Durham, N.C.: Duke University Press, 2006). On the Philippines medical authorities' opinion that Chinese individuals were more prone to ambulatory plague, see Office Board of Health, "The Ambulatory Type of Plague; Manila, P. I., April 8, 1901," *Pub. Health Rep.* (1896–1970) 16, no. 32 (August 9, 1901): 1838.

113. Herzog and Hare, "Latent and Ambulatory Plague" (n. 111), 1786.

this was a seventeen-year-old Filipino boy, employed at the soda water factory of the city, who was apparently unaffected by any disease or ailment with the exception of developing a pale contour and not sleeping well. Having spent the evening playing on the streets with his friends, he woke up at midnight with what was diagnosed postmortem as embolism of the pulmonary artery, which led to his death two hours later. Taking smears from his organs and swollen glands, cultures of "typical plague bacilli" were developed.[114] It was thus that once again the pathological category of *pestis minor* or *pestis ambulans* was salvaged to continue its evidential productivity as an index of plague's transformative nature.

Conclusion

As medical anthropologist Charles Briggs has recently argued, "Epidemics in which the production of evidence resists transformation into a diagnosis are particularly interesting for exploring ecologies of evidence."[115] *Pestis minor* is a paradigmatic historical case where the entanglement of different types of evidence in an inconclusive flux of proof and disproof institutes a pathological category that, though only ever tentatively diagnosed, continued to inform epidemiological reasoning for over four decades.

By comparison to the soil, which at the time attracted considerable attention as a locus of the disease's supposed attenuation, ideas about *pestis minor* did not lead to major public health interventions. Nor can the notion be said to have contributed to the development of experimental systems in a manner witnessed in relation to the soil in India or Hong Kong.[116] And yet *pestis minor* managed to persist as a productive field of epidemiological reasoning long after the soil and its problematization had been abandoned by the majority of plague experts. On the one hand, this "success" was the result of *pestis minor* being focused on the human body, and particularly that of native subjects in the colonial tropics. This left the particular category unaffected when public health attention on plague shifted from the soil to the rat. And, at the same time, it sustained a colonial suspicion of native bodies as the organic medium where, to

114. Ibid., 1788. A shortened reprint of the article omitted the discussion of ambulatory plague embolism, perhaps accounting for Anderson's reading of the two scientists' work as dismissing of the notion of *pestis ambulans* (Anderson, *Colonial Pathologies* [n. 112], 256); M. Herzog and C. B. Hare, "Does Latent or Dormant Plague Exist Where the Disease Is Endemic?," *Department of the Interior Bureau of Government Laboratories* 24 (October 1904): 5–20.

115. Charles L. Briggs, "Ecologies of Evidence in a Mysterious Epidemic," *Med. Anthrop. Theory* 3, no. 2 (September 2016): 149–62, quotation on 151.

116. See Lynteris, "A Suitable Soil" (n. 3).

return to Cantlie's argument, "the bacillus of the benign variety attains malignancy."[117] On the other hand, while reaffirming the centrality of the native body as a site of virulence for colonial medicine, the persistence of *pestis minor* as a category also relied on a pervasive belief in the transformative character of the disease. Dating back to prebacteriological times, this configuration of plague as a protean entity productively allowed for a fertile entanglement and exchange between evidential regimes and practices in the examination of plague's origins, its "breeding grounds," its interepidemic ecology, and, ultimately, its "true nature."

Defying bacteriologically led hierarchies of evidence, *pestis minor* was not, however, simply turned into epidemiological fodder, some contingent or vague reserve category where unsorted data could be temporarily set aside without causing epistemic embarrassment. Instead, being a hotly contentious notion, it pitched leading medical experts against each other, emerging out of such scientific battles time and again evidentially debunked but categorically alive. And, at the same time, long after studies had conclusively shown the role of rats in maintaining plague in urban settings and of wild rodents as natural reservoirs of the disease, *pestis minor* maintained epidemiologists' interest in the human body as a potential transformative locus of plague.

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117. Cantlie, "Lecture on the Spread of Plague" (n. 33), 91.