CHAPTER 12

Fleas, Knowledge-Making, and the Epidemiology of Plague in British India: Perspectives from the Bombay Epidemic, 1905–1906

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1 Introduction

Between October 1905 and January 1906, in a cluster of houses behind the Parel Laboratory in Bombay, an unusual ecosystem unfolded. Monkeys, guinea pigs, and rats both wild and tame, were released inside perfect replicas of the traditional workers huts (or go-downs) of the city of Bombay. Inside these buildings, these experimental animals awaited a grim fate: some animals, or the fleas they carried with them, had been intentionally infected with virulent *Yersinia pestis*, the causative agent of bubonic plague – and a likely death sentence for its carriers.

These experiments, conducted by the Second Indian Plague Commission in 1905, were part of a larger set of knowledge-gathering activities that arose during the Third Plague Pandemic (1894–1950) designed to examine a key set of controversies on the etiology and epidemiology of plague: "How," as Sanitary Commissioner FHG Hutchinson succinctly inquired, "is plague infection carried from rat to rat, rat to man, or man to man?"[1] While some acceptance of the agency of rats in the plague had gained traction among the imperial scientific community as early as 1903, the question of *how* plague transmitted from rats to humans remained an unsettled question. Experiments by Antoine Yersin (1894), EF Hankin (1897–1898), George Nuttall (1897–1899), Ogata Masanori (1897) and Paul-Louis Simond (1898), all claimed success in instigating transmission through the rat-flea, *Xenopsylla cheopis,* but had achieved at best

1 Captain FHG Hutchinson, IMS, acting Deputy Sanitary Commissioner, Southern Registration District; To The Sanitary Commissioner of Bombay, dated Belgaum, 3rd August 1905. Proceedings, February 1906. Nos. 327–347. Measures for the Prevention of Plague. Calcutta Records 5. Government of India Home Department, Sanitary Branch. National Archives of India, Delhi.

mixed success when replicated.[2] This ambiguity, and widespread imperial competition to contribute to the knowledge-making practices around plague prompted, as medical anthropologist Christos Lynteris has noted, "an epistemologically intense, nuanced and complex era of plague research."[3] It was in this heightened context that the Government of India established a robust research program at the Parel Research Laboratory, Bombay, designed to settle the question of plague transmission dynamics.

Between 1898–1910, Indian Medical Service Officers of the Indian Plague Commission and Bombay Plague Committee drew on the local epidemic and information networks within the city to establish a series of long-term epidemiological and bacteriological experiments and surveillance mechanisms to observe, characterize, and define the boundaries of plague within the city.[4] Keeping with the theme of this edited volume, this chapter will explore how a subset of these experiments, concerning the role of the rat-flea, *Xenopsylla*

2 The Advisory Committee Appointed by the Secretary of State for India, the Royal Society, and the Lister Institute, "Reports on Plague Investigations in India," *The Journal of Hygiene* 6, no. 4 (1906), http://www.jstor.org/stable/3858976.

3 Christos Lynteris, "In Search of Lost Fleas: Reconsidering Paul-Louis Simond's Contribution to the Study of the Propagation of Plague," *Medical History* 66, no. 3 (2022), doi:10.1017/mdh.2022.19.

4 Christos Lynteris, "Pestis Minor: The History of a Contested Plague Pathology," *Bulletin of the History of Medicine* 93, no. 1 (2019); Nicholas H.A. Evans, "Blaming the Rat? Accounting for Plague in Colonial Indian Medicine," *Medicine Anthropology Theory* 5, no. 3 (2018), doi:10.17157/mat.5.3.371; No. 749 P, dated Bombay, the 12th August 1905. From Major WE Jennings, MD, DPH, IMS, Superintendent, Plague Operations, Bombay Presidency; To the Secretary to Government, General Department (Plague). Through The Surgeon-General with the Government of Bombay, pg. 145. National Archives of India, New Delhi, India; J.T.W Leslie, "Question whether it is possible or expedient to adopt generally any organized system for which the extermination of rats in areas which are infected with plague or threatened with an importation of the disease," May 1902, Calcutta Records 3, Sanitary Plague A Branch, Home Department, Government of India, nos. 114–116, pg. 6, National Archives of India; JA Turner, "Executive Health Officer's Report for Bombay, 1910–1925," IOR/V/25/840/24, India Office Records, AAS, BL; TS Weir, Reports of the Health Officer of Bombay, 1896–1909, IOR/V/25/840/23, Asian and African Studies Collection, British Library, London, United Kingdom; R.D. Saigol, IMS, "On Experiments Made to Determine the Jumping Power of Rats," July 10, Simla Records 3, Sanitary Plague A Branch, Home Department, nos. 70–73, pg. 9–10, National Archives of India; "Experiments to test the efficacy of M. Duclaux's cultures for the purpose of destroying rats," March 1901, Calcutta Records 1, Sanitary Plague A Branch, Home Department, Government of India, nos. 55–56, pg. 1–4, National Archives of India; J Dansyz, "Un Microbe Pathogene pour les Rats: Et son application a la destruction de ces animaux," in *Annales de L'Institut Pasteur* 14, no. 4 (1900); Cunningham, Captain J MD, IMS (Bombay Bacteriological Laboratory). *Scientific Memoirs by the Officers of the Medical and Sanitary Departments of the Government of India. The Destruction of Fleas by Exposure to the Sun*0 (Calcutta: Superintendent Government Printing, India, 1911). V/25/850/53. India Office Records, Asian and African Studies Collection, British Library.

cheopis, in plague transmission relied on multispecies relationships and ecologies within the city to develop and test the mechanisms of the rat-flea theory. Looking to a key set of experiments undertaken by the Commission in 1905–1906, it will examine how the urban ecology of Bombay and the highly specific, cyclic presentation of *Yersinia pestis* in the city informed the structure of experiments, allowing imperial medical officials to engineer these epizootics among "wild" rats, fleas, and experimental animals in ways that elucidated the local transmission pathways for plague. Looking to the published reports of the Second Plague Commission from the *Journal of Hygiene* and housed in archival repositories, as well as the sanitary records of the Government of Bombay, evidence suggests that imperial scientists treated the urban ecologies and the multiple species that lived within them as part of an "experimental system," and utilized the locally-specific presentation of plague to drive and design inquiry into the epizootic. While the ecological specificity of the epidemic in Bombay played a significant role in how these experiments were designed, however, the embeddedness of these experiments in a global, multi-scalar knowledge infrastructure meant that the findings of these studies were widely adopted and accepted as indicative of a more general plague etiology and epidemiology – with significant, long-lasting effects on the study of the disease.[5]

The role of colonial spaces as sites of experimentation and knowledge-production has been well-documented and theorized by historians of science and environment. Helen Tilley, Daniel Headrick, and others have demonstrated how, in the first half of the twentieth century, a practice of use of colonial sites as "living laboratories" emerged, in which scientific knowledge-gathering practices around human and nonhuman disease increasingly included controlled "field" experiments, designed to document, describe, and translate the mechanisms of disease transmission.[6] These colonial sites, including their infrastructures, the local and European experts working within them, and their

5 Helen Tilley, *Africa as a Living Laboratory* (Chicago: University of Chicago Press, 2011), 11–12. Tilley notes that while knowledge is "situated," there is too much circulation between metropole and colony, colony and colony, and between nation-states for any knowledge infrastructure to fall neatly into a single category, or be truly localized.

6 Tilley, *Africa as a Living Laboratory*, and Daniel R. Headrick, "Sleeping Sickness Epidemics and Colonial Responses in East and Central Africa, 1900–1940," ed. Philippe Büscher, *PLoS Neglected Tropical Diseases* 8, no. 4 (24 April 2014): e2772, doi:10.1371/journal.pntd.0002772. The persistence of field experimentation in colonial spaces contrasts with the divide that was (imperfectly) drawn between field and laboratory sciences over the 20th century. See Linda Nash, *Inescapable Ecologies: A History of Environment, Disease, and Knowledge* (Berkeley: University of California Press, 2006).

information systems, comprise a unique kind of "experimental system," – to borrow from historian of science Hans-Jörg Rheinberger – which occurred within highly specific environments, which created the conditions for highly impactful knowledge formation.[7]

Looking to the records of the Indian Plague Commission, however, we see an additional, critical aspect of plague's "experimental system": its reliance on material local ecology. Bombay, at the turn of the twentieth century, became a "laboratory" for testing the transmission dynamics of plague. The original relationship delineated by Simond between *Xenopsylla cheopis* and *Rattus* species were made robust; and early links between temperature, *Yersinia pestis* replication, and infectivity among fleas were forged, tested, and theorized.[8] Relying on extensive surveillance infrastructure and the unique, cyclic nature of the epidemic that had taken hold between 1896–1907, members of the Second Indian Plague Commission designed experiments that took into account the ecological conditions of the city and the predictable epizootic among "wild rats" to establish a rigorous course of inquiry into the relationship between rats, fleas, and humans in plague. Experiments conducted by the Commission were designed not to create controlled conditions in which to test plague transmission dynamics; rather, IMS officers embraced the permeability of their experimental systems to the uncontrolled, or "wild" environment. In doing so, these experiments demonstrated an important and often under-considered aspect of experimental systems: that material ecology played an important role in experimental systems and informed the kinds of questions and transmission pathways examined. These multi-species experiments, at once highly locally-driven and designed to test general theories of plague transmission, contributed to a broader etiology of plague that carried lasting impacts on the etiology of the disease.

2 Bombay as an Experimental System

The Third Plague Pandemic is estimated to have caused 15 million deaths globally, from its (hypothesized) emergence in Yunnan province, China, in 1854

7 Hans-Jörg Rheinberger, *Towards a History of Epistemic Things: Synthesizing Proteins in the Test Tube* (Stanford: Stanford University Press, 1997); Hans-Jörg Rheinberger, "Experimental Systems: Historiality, Narration, and Deconstruction," *Science in Context* 7, no. 1 (1994), doi:10.1017/S0269889700001599.

8 J.A. Turner, Esq., Executive Health Officer, Bombay Municipality, to the Municipal Commissioner for the City of Bombay. 8 May 1907. No. P. 1398., Home Department, Sanitary (Plague) Branch A, National Archives of India, New Delhi, India.

to its gradual decline in 1950. While the disease was global in scope, mortality was highly unevenly distributed; 10 of the 15 million deaths occurred in India, between 1896–1930.[9] Within India, no city suffered a higher mortality rate, nor longer-lived epidemic, than Bombay. The Bombay plague epidemic is estimated to have killed over 180,000 people in the city from its arrival in 1896 to its eventual disappearance in the 1930s. Death counts of the epidemic are often given in terms of human mortality, sources indicate that there were other residents of the city who suffered; that there was not one epidemic, but many nested epidemics. Scientific reports produced through the imperial government in Bombay showed millions of deaths, colony collapse, and significant behavioral changes among both *Rattus norvegicus* and *Rattus rattus* as they became infected with *Yersinia pestis*. Their epidemic was the result of another among *Xenopsylla cheopis*, or rat fleas, as the bacteria filled their stomachs with a biofilm and slowly starved them to death. The epizootic, as Plague Commissioner P.C.H. Snow notes, was as obvious as the human epidemic. "From the time the bubonic plague had established itself in Mandvi at the end of September 1896," he notes in his report. "Large numbers of rats were seen running about the streets and coming out of house connection pipes and drains in sickly or dying condition."[10]

As the first city outside of China to suffer cases of plague, Bombay garnered significant administrative and epidemiological attention.[11] The magnitude of the epidemic, and the threats it posed to administrative authority within the Empire's "Second City", made Bombay a concentrated site of scientific activity. Waldemar Haffkine (1860–1930), the renowned bacteriologist, was invited to establish a large bacteriological research laboratory dedicated to the study of plague nearly immediately after the first cases were discovered in the neighborhood of Mandvi in 1896.[12] By March 1897, the Bombay Plague Committee was formed to intervene on the burgeoning epidemic within the city, enacting

9 Myron Echenberg, *Plague Ports: The Global Urban Impact of Bubonic Plague, 1894–1901* (New York: New York University Press, 2007), pg. xii, 17, 50–51.

10 P.C.H. Snow, *Report on the Outbreak of Bubonic Plague in Bombay, 1896–1897* (Bombay: "Times of India" Steam Press, 1897), 9.v/27/856/7, Asian and African Studies Collection, British Library, London, United Kingdom.

11 Estimated number in Ira Klein, "Urban Development and Death: Bombay City, 1870–1914," *Modern Asian Studies* 20, no. 4 (1986): 729; and David Arnold, *Colonizing the Body: State Medicine and Epidemic Disease in Nineteenth-Century India* (Berkeley: University of California Press, 1993), 201.

12 W.B. Bannerman, "The Plague Research Laboratory of the Government of India, Parel, Bombay," *Proceedings of the Royal Society of Edinburgh* 24 (1904), doi:10.1017/S0370164600007781.

mass sanitizing campaigns, constructing plague camps, and establishing surveillance networks across the city.[13] In 1898, the first Indian Plague Commission, referred to as the Fraser commission after its leading physician, was established to pursue the etiology, epidemiology, and cause of the plague epidemic in India, beginning with Bombay; however, it was the Second Commission, established in 1905, that focused its attention most directly on drawing out the nuances and mechanisms of zoonotic transmission of plague – with Bombay as its major experimental site.[14]

There were several reasons that Bombay was chosen as a key site of investigation into the epidemiology and etiology of plague by the Second Indian Plague Commission, as elucidated in their 1906 report in *The Journal of Hygiene*. First, the city housed the laboratory and scientific infrastructure needed to conduct a variety of experiments on plague transmission. The first British Indian research laboratory, established in Bombay in 1884, was a site "in every way and well suited for the requirements of the Commission."[15] The medical laboratory housed at Grant Medical College served as a site of medical and bacteriological training for Indian doctors from its establishment in 1845, and housed Haffkine's initial experiments.[16] The city's municipal and health departments – and the pre-established Bombay Plague Committee – also facilitated surveillance and large-scale coordination of personnel for observation and experiments.

Most important to the Commission, however, was the unique ecology of plague in Bombay. In addition to the sheer magnitude of the epidemic and epizootic, Bombay suffered from plague cyclically, meaning outbreaks could be more or less predicted annually.[17] Seasonal prevalence was also "well-marked", and explaining cyclical seasonal patterns was considered to be a necessary condition for proving any hypothesis about plague transmission. Drawing on the clear relationship between the epidemic and epizootic in the city, the Commission expressed confidence that "[T]he general relationships of the epizootic and epidemic would be obtained from Bombay city, some errors being

13 Report of the Bombay Plague Committee Appointed by Government Resolution No. 1204/720P, on the Plague in Bombay, for the Period Extending from the 1st July 1897 to the 20th April 1896 (Bombay: Times of India Steam Press, 1898), IP/13/PC.5, Medical History of British India Collection, National Library of Scotland. Report of the Bombay Plague Committee, National Library of Scotland.

14 Evans, "Blaming the Rat?," 21–28.

15 "XXII. The Epidemiological Observations Made by the Commission in Bombay City," *The Journal of Hygiene* 7, no. 6 (1907): 725, doi:10.1017/S0022172400033684.

16 Lynteris, "Pestis Minor," 61; Pratik Chakrabarti, *Bacteriology in British* India (Cambridge: Cambridge University Press, 2011), 26–60.

17 Reports of the Indian Plague Commission, 530.

corrected by the very large number of plague rats and of human cases dealt with …"[18] Far from being considered a limitation of the city-as-experimental-site, its unique qualities were seen as integral to elucidating the relationship between rats, rat-fleas, environments, humans, and *Yersinia pestis.*

3 Urban Structure as Experimental System

Beginning in 1904, the Bombay Plague Committee began a program of rat capture and counting to monitor the epizootic within the city. Included in this process was the systematic trapping of rats from key locations around the city, combing of the rats for fleas, and dissection of rat and rat-flea for signs of *Yersinia pestis* infection.[19] These robust surveillance infrastructures, combined with existing experiments by members of the Government Laboratory (in particular WB Bannerman (1858–1924), director of the Plague Committee), had already raised a series of questions about the relationship between urban structure and ecology and the plague epizootic, and identified regions where urban structure may play a role in transmission.[20] Bannerman notes in his own 1906 experiments that different roof and housing structure types appeared to be more or less conducive to rats, observing,

> The structure of the houses in this country seems designed to favour the continued existence within them of the black rat … in Bombay the roofs of round country tiles and the curious shelf-like projections found in almost every room in the chawls, where firewood and dung cakes are stored, afford them ideal places for shelter and breeding.[21]

Indian Medical Service (IMS) officers also drew on the unique seasonality of the plague epizootic in the city to conduct studies on the role of climate on flea transmission. Disparities between Nuttall and Simond's findings in 1898 in the role of temperature in the activity of fleas inspired studies like those conducted in *The Fourth Progress Report of the Plague Research Commission*, in which "A large number of observations on the effect of temperature on the transmission of plague by fleas have been carried out in specially constructed

18 "Epidemiological Observations," 725.

19 "Epidemiological Observations," 728–752.

20 W.B. Bannerman, "Conditions Affecting the Origin and Spread of Plague," in "Measures for the Prevention of Plague," February 1906, Calcutta Records 5. Government of India Home Department. Sanitary Branch. Proceedings, February 1906. Nos. 327–347, 106. National Archives of India.

21 Bannerman, "Conditions Affecting the Origin and Spread of Plague," 106.

rooms both below and above the ordinary room temperature." While some were optimistic that the results of these experiments "have thrown considerable light on the problem of the seasonal prevalence of the disease" the Commission remained cautious about the conclusiveness of the findings.[22] It was precisely these questions that the Commission sough to settle and hoped that the cyclicality of Bombay's epidemic would facilitate.

In 1907, The Commission designed a series of ecological experiments to test the transmission pathways of plague in urban environments, grounded in the construction of environments that would foster an epizootic among rodent communities, using observations of the plague in the city to construct the experimental system. Experiments concerning "the relative importance of the Indian rat flea, *Xenopsylla cheopis*, and of actual close contact in the absence of fleas, in the dissemination of plague from animal to animal" highlight the focus on interconnected ecologies, porous experimental boundaries, and multispecies etiologies in these experiments.

The studies began with the construction of replica "go-downs" designed by Lt. Col. Bannerman and Captain Liston (1872–1950) (who had already begun to experiment with the transmission dynamics of the rat-flea in 1905) of the Bombay Bacteriological laboratory.

The body of the buildings were constructed to be "rat-proof", with 9-inch walls built of brick and mortar and concrete floors on top of a concrete slab. Inside, an "inspection chamber" made of wire netting about 3.25 by 3 feet wide was connected to wire netting covering the inside of the roof but separated from the rest of the hut, to prevent anything that might settle in the roof from entering the hut itself. The object of this structure was to allow wild rats to colonize the roofs, but to limit contact between "wild" rats and experimental animals, and thus controlling for the possibility of direct rat-to-rat transmission but allowing fleas to move from wild animals to experimental animals. (see Figure 13.1) [23]

Once identical huts had been constructed, an environment was engineered in which "wild" Bombay rats (and their resident fleas) would colonize the roofs. Drawing on observation (and presumably available materials) from the city, Bannerman and Liston instructed that there be three different types of roofing material across the six huts: the first two huts (1 and 2) were furnished with "country tile"; the second two (3 and 4) with "Mangalore tiles" (a red clay tile commonly used for roofing across India); and the third two (5 and 6) of corrugated steel.[24]

22 Bannerman, "Conditions Affecting the Origin and Spread of Plague," 106.

23 "Reports on the Plague Investigations in India," 450.

24 "Reports on the Plague Investigations in India," 451–453.

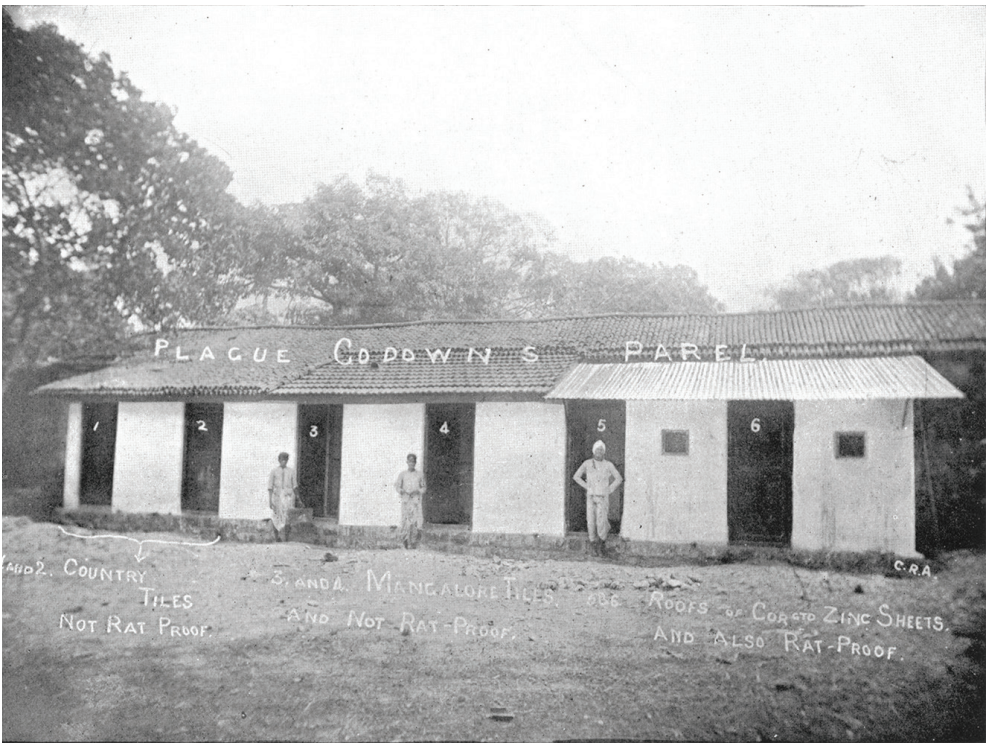

FIGURE 13.1 Photograph of the six "plague go-downs" constructed at the Plague Research Laboratory in Parel, Bombay, designed to mimic different urban structural conditions known to be more or less conducive to plague
SOURCE: "REPORTS ON PLAGUE INVESTIGATIONS IN INDIA." *THE JOURNAL OF HYGIENE* 6(1906): 450–451

Choices in roofing were based on ecological observations of rat density and plague mortality within the city: "in the case of go-downs Nos. 1 and 2," the report notes,

> the roofs of which offer good protection and shelter to the wild rat of Bombay, the flea supply is abundant and regular; in the case of go-downs Nos. 3 and 4, the roofs of which offer only poor protection to rats, the flea supply is more or less scanty; while in go-downs Nos. 5 and 6, the roofs of which are absolutely impervious to rats, no fleas should be able to gain access unless carried through the door on the experimental animals themselves, or by the attendant when feeding these animals.[25]

25 "Reports on the Plague Investigations in India," 453.

Once the requisite ecologies had been established in each hut, a series of experiments were designed to test the role of the rat-flea in transmission. In the first round of experiments, three guinea pigs were released in each hut for six days, then combed daily for fleas, to demonstrate the varied porousness of experimental environments to the broader urban ecology – and they noted that "the number of fleas varies in each instance with the accessibility of the roof to rats."[26] The experiments that followed focused on manipulating the number of fleas present in each go-down and the duration of exposure to fleas by experimental animals to establish the link more firmly between infected rat-fleas and epizootic plague. Some experiments relied on the existing epizootic to jump to the experimental guinea pigs, and observation of the trajectory of the epizootic among inoculated and uninoculated guinea pigs once it commenced. In the first round of experiments, several guinea pigs were inoculated with virulent *Yersinia pestis* and released to live alongside healthy guinea pigs in experimental huts 5 and 6 (devoid of fleas). In these experiments, it was found that the inoculated guinea pigs died, while uninoculated guinea pigs remained healthy. The experiment was then repeated in hut 2, where "rats had taken up their abode under the tiles" and thus "these go-downs were kept supplied with rat fleas, for as the insects left their hosts they fell down into the go-downs."[27] In these experiments, uninoculated animals died from plague in varying numbers; experiments were conducted during different periods, dependent on the pervasiveness of the epizootic within the city as a whole – and mortality rates among guinea pigs responded accordingly.[28] When conducted in November, during the cyclic increase in the urban epizootic, the experimental hut experienced an "epizootic of the most rapid description," with 115 fleas isolated from the last five animals to die of the disease – nearly ten times the typical number.[29]

The second set of experiments relied solely on transmission between experimental animals using fleas continuously replenished from the "wild" environment. Fleas isolated from other huts, in which epizootics were active among experimental guinea pigs, were then transferred to huts with healthy guinea pigs, and consistently added additional fleas over a number of days. The experiment showed that when the flea population was consistently replenished, the epizootic continued until all guinea pigs had died.[30]

26 "Reports on the Plague Investigations in India," 453.
27 "Reports on the Plague Investigations in India," 456.
28 "Reports on the Plague Investigations in India," 456–457.
29 "Reports on the Plague Investigations in India," 457.
30 "Reports on the Plague Investigations in India," 460.

While the controlled conditions were ultimately compromised – an "abundant supply of fleas" were found in go-down No. 6, for reasons unknown to experimenters – the existing hypotheses for why each would be useful in allowing or disallowing wild urban rats to colonize the space demonstrate a use of local urban ecology to experiment with and induce epizootics. In each case, the porousness of the experimental system was central to experimental design; *Xenopsylla cheopis* was isolated from the urban environment and conditions established for its breeding. What is more, Bannerman and Liston used the distinct seasonality of Bombay's epidemic to time their experiments. The first three experiments were undertaken when plague cases were sporadic among the city's wild rats (in June and July), and served as "control" experiments; the remaining three, tested against these original three, occurred in November, "during the period where the epizootic was just commencing."[31]

In keeping with Rheinberger's assertion that experiments often intentionally or unintentionally test multiple hypotheses, we might also look to how experiments controlled for, tested, and probed competing theories of plague transmission.[32] For example, in hut No. 1 and No. 3, a "certain amount of light" was allowed to penetrate through a small glass window in the tiles, and a small ventilation hole established in the housing, testing the role of sunlight and air flow on the spread of the microbe (in line with hygienic and miasmatic theories of disease). In multiple experiments, guinea pigs or chimpanzees were suspended in their own cages above the floor to avoid possible interaction with feces, urine, or infected soil in each hut, or set in cages in which fly paper was placed around the bottom six inches of the cage to prevent fleas jumping in (while simultaneously verifying existing findings from Egypt that fleas could not jump higher than six inches).[33] In another experiment, pregnant guinea pigs were removed from epizootic conditions and combed for fleas, and those already infected with plague were not found to transmit the disease to their offspring. Looking to these experiments, and the ways they used their environments to test and define the parameters of plague transmission, it is clear that the particular ecology of plague in Bombay had a direct effect on their conditions, observations, and outcomes; and the material environment in which the experiments were conducted was made inseparable from its findings.

31 "Reports on the Plague Investigations in India," 456.

32 Rheinberger, *Epistemic Things*, 76. Rheinberger argues that in experiments, "there at every step what is about to take shape creates unforeseen alternative directions for the next step to be taken."

33 "Reports on the Plague Investigations in India," 464–466.

This relationship becomes even more explicit in another set of studies which eschewed the controlled environments of the constructed huts altogether and instead loosed guinea pigs in houses recently impacted by plague. In a series of studies, guinea pigs were either allowed to run free in houses where plague had broken out or left in these houses in cages. The guinea pigs were then re-captured and combed for rat fleas. The Commission found that there were up to 40 rat-fleas on each of them, at least 40 percent of which were infected with the bacteria.[34] Perhaps most damningly, the same result occurred when guinea pigs were allowed to run free in houses that had been disinfected with sulfuric acid after the outbreak of plague, to much the same result – which indicated that British sanitary disinfection practices were not effective in removing fleas from housing and therefore were likely ineffective in preventing the re-emergence of plague.[35]

Engagement with the material urban environment in the design of rat-flea experiments carried significant implications for understanding of both plague etiology and understanding the legitimacy of plague control. The Commission engineered an environment in which the multispecies assemblages of the local plague epidemic could be tested, described, and (they believed) generalized. The resultant studies offered one of the most nuanced and detailed descriptions of plague dynamics to date, and provided support to existing theories of plague transmission while throwing others into question. Perhaps most strikingly, they undermined arguably the most controversial public health intervention enacted during the epidemic.[36]

4 Continuing Legacies: the Rat-Flea Hypothesis, Epidemiological Modeling, and Plague

From these experiments in which epizootics were manufactured within controlled environments – or observed in the broader urban ecology – a number of claims on the mechanisms of the transmission of plagues were posited and solidified.[37] As the results presented and the design of their experiments suggest, the transmission pathways elucidated by the Commission were quite ecologically specific. However, as historian Christos Lynteris argues in his

34 "Transmission of Plague by Fleas," Tables I–IV, in "Reports on the Plague Investigations in India," 482–483.

35 "Transmission of Plague by Fleas," Tables I–IV, in "Reports on the Plague Investigations in India," 481–482.

36 Evans, "Blaming the Rat?," 26–28; Arnold, *Colonizing the Body*, 200–239.

37 A.W. Bacot and C.J. Martin, "LXVII. Observations on the Mechanism of the Transmission of Plague by Fleas," *Journal of Hygiene* 13 (1914).

study of Paul-Louis Simond, the experiments also occurred in a different kind of "ecology" – what Anthropologist Charles Briggs calls a set of "ecologies of evidence."[38] In other words, these experiments were part of "broader assemblages" of networks of knowledge production that allowed certain types of evidence to be privileged, expanded upon, and mobilized over others.[39] Findings from the Second Plague Commission circulated widely across the Empire, and its experiments on the rat-flea cited widely in English-language medical journals throughout the 20th century. Articles in professional journals as widespread as *The British Medical Journal*, *the Journal of Hygiene*, and *the Journal of Infectious Diseases*, claimed that the study "established very conclusively the fact that the flea is the most important factor in the transmission of plague from rat to man."[40]

The rat-flea experiments conducted in Bombay were thus taken by imperial scientists to constitute a "representative" model for plague transmission – and one that definitively affirmed the rat-flea theory of disease. However, the findings conferred by the rat-flea experiments and the model of transmission they proposed, while often aligned with observations in other plague-cities, were constructed and affirmed under highly specific circumstances, which presented only a limited and partial view of plague dynamics. Following from the work of philosopher of science Nancy Cartwright, we might see the central slippage here as being in model interpretation: a representative model (a model that represented a particular phenomenon in the world) was taken by the wider scientific community to be an interpretive model (a model that could be linked to an abstract theory) in an instance where such a leap was dubious – if useful for integrating the study's results into existing "ecologies of evidence."[41] By relying heavily on the local ecology and its natural mechanisms to engineer a laboratory setting, what the experiments ultimately showed was

38 Charles L. Briggs, "Ecologies of Evidence in a Mysterious Epidemic," *Medicine Anthropology Theory* 3, no. 2 (2016), doi:10.17157/mat.3.2.430.

39 Briggs, "Ecologies of Evidence," 151.

40 Bacot and Martin, "Transmission of Plague by Fleas"; T.L. Anderson and J. Burton Cleland, "The Transmission of Plague," *The British Medical Journal* 1, no. 2414 (1907): 838; "Fleas and Plague: Recent Additions to Our Knowledge of the Mechanism by Which Fleas Probably Spread the Disease," *Public Health Reports (1896–1970)* 29, no. 19 (1914); Wheeler, C.M. and J.R. Douglas, "Sylvatic Plague Studies: V. The Determination of Vector Efficiency," *The Journal of Infectious Diseases* 77, no. 1 (1945), http://www.jstor.org/stable/30061611; Cole, LaMont C., "The Effect of Temperature on the Sex Ratio of Xenopsylla Cheopis Recovered from Live Rats," *Public Health Reports (1896–1970)* 60, no. 45 (1945), doi:10.2307/4585454.; W.M. Frazer, "Rats and Vermin and Their Role in the Spread of Disease," *The Journal of State Medicine (1912–1937)* 40, no. 12 (1932).

41 Stephan Hartmann, Carl Hoefer, and Luc Bovens (eds.), *Nancy Cartwright's Philosophy of Science*, Routledge Studies in the Philosophy of Science 3 (New York: Routledge, Taylor &

a detailed picture of the ecology of the Bombay epidemic itself and the role of rat-fleas within it.

While this model of plague transmission related to existing observations and proposed theoretical frameworks for plague transmission, including those listed above, historians of the plague (and indeed historical actors) have pointed out the ways that the Bombay epidemic was itself highly unusual. Features like housing type, urban structure, and rat ecology were highly specific to Bombay, and the epidemic itself, in its cyclicality, endemicity, and mortality rates did not easily map on to other plague-affected areas, and yet had a significant effect on transmission dynamics.[42] In being highly locally grounded, therefore, the study sacrificed its generalizability – but was nevertheless treated as generalizable evidence because of its adherence to basic mechanisms of scientific validity and seeming support of prevalent theoretical claims constructed by scientists across the British Empire. Meanwhile, the more robust, locally-specific results were largely ignored, and slum clearance and sanitation projects continued in spite of evidence of their ineffectiveness.[43]

The effects of this confluence of study design and broader imperial ecologies of knowledge carried long-term effects for the etiology of plague. Looking to several key studies in mathematical biology and epidemiology in the last twenty years, we can see how the cyclic nature of the epidemic in Bombay – and once again, fleas and findings on fleas from studies conducted during this time – are used to construct an ecology of plague that can be tested and verified. The fit of Kermack and McKendrick's SIR model (1927) with the data from the 1905–1906 Bombay epidemic has been referred to as the "most reproduced figure in books discussing mathematical epidemiology," and was taken as evidence of the model's strength as a predictor of epidemic structures.[44]

Francis Group, 2008), 24–25; Nancy Cartwright, *The Dappled World: A Study of the Boundaries of Science* (Cambridge: Cambridge University Press, 1999).

42 Emily Webster, "Plague in Bombay, 1896," in *Epidemic Urbanisms: Contagious Diseases in Global Cities*, ed. Mohammad Gharipour and Caitlin DeClerq (Bristol: Intellect Press, 2021); Ira Klein, "Urban Development and Death"; Prashant Kidambi, "'An Infection of Locality': Plague, Pythogenesis and the Poor in Bombay, *c*.1896–1905," *Urban History* 31, no. 2 (2004); Myron Echenberg, "Pestis Redux: The Initial Years of the Third Bubonic Plague Pandemic, 1894–1901," *Journal of World History* 13, no. 2 (2002); Arnold, *Colonizing the Body*, 200–239.

43 Sandeep Hazareesingh, "Colonial Modernism and the Flawed Paradigm of Urban Renewal: Uneven Development in Bombay, 1900–1925," *Urban History* 28, no. 2 (2001).

44 The SIR model estimates the trajectory of an epidemic within a population based on the number of susceptible (S), Infected (I), and Recovered (R) members of that population. It has become a cornerstone in epidemiological modelling since it was first posted in 1927.

However, this modeling structure was questioned by Bacaër in 2012, who pointed out the "remarkable seasonal pattern" of the epidemic undermined the original model, and Bacaër presents instead a seasonal model that explicitly used flea ecology described by the Commission to establish parameters with very different results.[45] Similarly, Keeling and Gilligan's widely-cited metapopulation model of zoonotic plague, relies on estimates of number of fleas per rat, flea life cycle and death rate, that are all derived from the structure of plague in India, and also cite the 1906–1907 Plague Commission explicitly in their studies.[46]

The observations taken from the 1906–1907 plague experiments were therefore used as evidence as to the epidemiology of plague generally – and yet, the study site itself was chosen because of its unique ecology. This tension has been pointed out by plague historians like Ann Carmichael, who posits the importance of marmots in the Alps as reservoirs of plague; and Matheus Alves Duarte da Silva, who examines the emergence of the concept of sylvatic plague and research on its circulation among wild rodents in the 20th century – a mechanism that remains highly uncertain and under-researched to this day.[47] These disparities and discontinuities carry real-time implications for plague research, as well; the ongoing outbreak of plague in Madagascar, which began in 1898 with the Third Plague Pandemic, is poorly understood in its ecological dynamics in part because it relies on some of these understudied transmission mechanisms.[48]

William Ogilvy Kermac and A.G. McKendrick, "A Contribution to the Mathematical Theory of Epidemics," *Proceedings of the Royal Society of London. Series A, Containing Papers of a Mathematical and Physical Character* 115, no. 772 (August 1927): 700–721, doi:10.1098/rspa.1927.0118.

45 Nicolas Bacaër, "The Model of Kermack and McKendrick for the Plague Epidemic in Bombay and the Type Reproduction Number with Seasonality," *Journal of Mathematical Biology*, 64 (2012), doi:10.1007/s00285-011-0417-5.

46 M.J. Keeling and C.A. Gilligan, "Bubonic Plague: A Metapopulation Model of a Zoonosis," *Proceedings of the Royal Society of London. Series B: Biological Sciences* 267 (2000).

47 Matheus Alves Duarte Da Silva, "Between Deserts and Jungles: The Emergence and Circulation of Sylvatic Plague (1920–1950)," *Medical Anthropology* 42, no. 4 (2023), doi:10.1080/01459740.2023.2189110.

48 Jennifer Alderson et al., "Factors Influencing the Re-Emergence of Plague in Madagascar," *Emerging Topics in Life Sciences* 4, no. 4 (2020), doi:10.1042/ETLS20200334; Voahangy Andrianaivoarimanana et al., "Understanding the Persistence of Plague Foci in Madagascar," *PLoS Neglected Tropical Diseases* 7, no. 11 (2013), doi:10.1371/journal.pntd.0002382.

5 Conclusion: Etiologies and Epidemiology of Time and Place

While much has been written on both experimental systems and field experimentation, this chapter has argued that there are three key insights to be gained from looking at the role of rat-flea ecologies in the design of field experiments in imperial epidemics. First, that by treating the urban space as a "experimental system" of its own – and attempting to utilize the locally-specific urban ecologies and porosity between controlled and uncontrolled environments to drive and design inquiry – the Indian Plague Commission was able to both "settle" major epistemic controversies and generate new hypotheses about plague transmission that were highly consequential. Second, the way that the specific ecology of plague in the city shaped the structure of experiments – with scientists utilizing both the cyclical nature of the epidemic and its sheer scale to both justify its use as an experimental site and to inform the structure of experiments – played a key role in the contributions of the Commission. Finally, that understanding this specificity allows us to think more critically about the multi-scalar imperial knowledge systems that allowed the findings of these studies to be widely adopted and accepted within broader plague etiology and epidemiology, despite continued emphasis on the unique ecology of the epidemic in the urban environment. Looking to the epizootics engineered by the Second Indian Plague Commission in Bombay, we can see how the ecological specificity of the epidemic in time and place allowed for particular experimental designs that themselves influenced the way knowledge was gathered, generalized, and legitimized – an epistemic lineage we are still discovering the ramifications of to this day.

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