THE UNIVERSITY OF CHICAGO

MICROBIAL EMPIRES: CHANGING ECOLOGIES AND MULTISPECIES EPIDEMICS IN BRITISH IMPERIAL CITIES, 1837-1910

A DISSERTATION SUBMITTED TO

THE FACULTY OF THE DIVISION OF THE SOCIAL SCIENCES

IN CANDIDACY FOR THE DEGREE OF

DOCTOR OF PHILOSOPHY

DEPARTMENT OF HISTORY

BY

EMILY WEBSTER

CHICAGO, ILLINOIS

AUGUST 2021

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Acknowledgements

This project would not have been possible without the help of many people over the years, and it would take a document as long as the dissertation itself to express my gratitude to everyone that contributed to its development. So I will simply say, to each and every one of you: my deep, immeasurable, and profound thanks.

From this formidable group, there are a few persons who deserve special recognition. First and foremost, I would like to thank my advisor and mentor, Fredrik Albritton Jonsson, for his support and insight throughout the graduate school process. Fredrik, thank you for teaching me to take my thoughts, ideas, and goals and refine them into questions worth asking and action worth taking. Perhaps more importantly, thank you for believing in my ability to do so. I wouldn't be the scholar (or person) that I am without you.

I would also like to thank my committee members, Dipesh Chakrabarty and Michael Rossi. Your insights made the project so much richer and more interesting and motivated me to ask questions of my sources in new and enlightening ways.

My gratitude belongs to many wonderful UChicago history, anthropology, and public health faculty I have had the great fortune to work with and take classes with over the years – among them Ken Pomeranz, Emily Osborn, Faith Hillis, Liz Chatterjee, Jan Goldstein, Alice Yao, Diane Lauderdale, and Michael David. Your input shaped not only my research, but the way I think about history and the world generally. Both my work and my brain are immeasurably better for it. I was also extremely fortunate to have a wonderful community of historians at my undergraduate institution, Ohio State, who were central to my development as a historian. David Steigerwald and Peter Mansoor were the first historians to take my interest in pursuing a career in the field seriously, and to show me the world that historical inquiry could reveal. A special thanks belongs to my longtime academic mentor, Chris Otter, who not only guided me through my first primary source research project seven years ago and my many projects since, but modeled the kind of historian, teacher, and mentor I aspired to be.

This project took me to nine cities stretched across three continents. My gratitude belongs to the wonderful staff of the archives I visited in each of these places: The Maharashtra State Archives, the British Library Asia and Africa Studies Collection, National Archives at Kew, National Archives of India, Public Records Office of Northern Ireland, Linen Hall Library, Wellcome Collection, Public Records Office of Victoria, Public Records Office of New South Wales, and the National Library of Australia. This globe-trotting research would not have been possible without funding from the Social Science Research Council's Mellon International Dissertation Research Fellowship, the National Science Foundation's Doctoral Dissertation Research Improvement Grant, the Center for International Social Science Research's Lloyd and Susanne Rudolph Field Research Award, and the Center for South Asian Studies Dissertation Writing Fellowship. I am indebted to the wonderful people at all of these organizations for seeing value in my work – and helping me refine my ideas for wider audiences.

I am also acutely thankful for the many wonderful academic communities that I have had the privilege to participate in over the course of my education and to the wonderful people I have met through them who have taken the time to read my work or share their advice and insights with me. This list could go on for pages, but I will say a special thanks to Jacob Steere-Williams, Edmund Russell, John McNeill, Ruth Morgan, Josh Specht, Sean Connolly, Olwen Purdue, Gregg Mitman, David Harris, Phil Roth, Dagomar Degroot, Emma Moesswilde, Christos Lynteris, my co-editors at Environmental History Now, the students of my Power and Medicine tutorial, the members of the Fishbein Center for the History of Science, the Environmental

Studies Workshop, and the wonderful members of the American Society for Environmental History and the American Association for the History of Medicine.

I am also eternally grateful to the wonderful friends and colleagues who kept me sane throughout graduate school and while away on archival research – Caine Jordan, Greg Valdespino, Alex Herbert, Emma Kitchen, Chris Dunlap, Roland Black, Philip Henry, Christina Flores, Neeraj Agrawal, Shikha Saxena, Thomaz Amancio, Eric Gurevitch, Jillian Foley, Zoya Sameen, Carl Kubler, Spencer and Teresa Stewart, Dan and Julie Knorr, Maura Capps, Rachel George, Kris Palmieri, Elizabeth Hameeteman, Beth Calderon, Justin and Carly Niermeier-Dohoney, Kyle Gardner, Colin Rydell, Lorraine Magill, Jim Gray, and many others. I can't tell you how much I've appreciated your humor, support, and general brilliance these last six years.

My family were my first and best teachers. Mom and Dad, thanks for teaching me that building a life worth living is the most important thing. And Dad, an additional thanks for teaching me to ask, "where does the poop go?" for reasons that will become obvious in this dissertation. Hannah and Mel, thank you for being the best, most supportive sisters a girl could ask for, and the first people to really teach me how to have fun. Thanks to my newly legal family, the Suitses – Joan, Bryan, Jim, Bill, and Lauren – for making my nerdiness feel not only normal, but downright unremarkable.

To Sarada, Lindsay, Markus, Chris, and the Ohio crew, thank you for teaching me that unconditional love doesn't have to come from blood relatives, and reminding me that life exists outside of academic work! To my fuzzy companions, Baldr and Clara, thanks for the same thing.

A special thanks to Robert Gruener, whose thoughtful companionship and many acts of kindness got me through my research and writing years (and the pandemic), and reminded me that family can be found in unexpected places (including housing co-op board meetings).

And finally, to Robert Suits, my spouse, my best friend, my editor, my last-minute map-fixer, my travel companion, and my favorite historian. Our collaborations both professional and personal have been the most important of my life, and your intelligence, wit, humor, insight, and unwavering belief in me continue to astound me. This project is for you.

Abstract

This dissertation explores the emergence of three different epidemic diseases in three British colonial cities at the end of the nineteenth century. At first glance these cities – Melbourne, Australia; Bombay, India; and Belfast, Northern Ireland – appear disparate, and their epidemics – *Mycobacterium tuberculosis* (tuberculosis), *Yersinia pestis* (plague), and *Salmonella enterica serovar typhi* (typhoid), respectively – unremarkable in a period otherwise plagued by any number of epidemic diseases. However, I argue that these epidemics arose because of both local and global ecological and cultural pressures, shaped equally by imperial exchange and unique quotidian patterns of human-environmental interaction. To understand how these epidemics are related and why they were consequential, it is necessary to look beyond the human cultural, political, and economic structures that dictated development, organization, and response, and to engage with the underlying ecologies – and consider how they changed.

This project examines these layered relationships at a key moment in British imperial urbanism, looking to instances of ecosystem disruption in the form of exponential urban growth and improvement projects as opportunities for disease emergence. Historians have long documented British attempts to impose an ideal of "improvement" upon colonial landscapes; settlers and administrators applied theories of climate, disease, urban planning, and infrastructure to their colonial holdings, undertaking massive (and often expensive) schemes to reroute waterways, construct colonial cities, plant crops, and raise livestock, often destroying entire ecosystems in order to perpetuate their ideas of civilization and productivity. The cases explored in this dissertation reveal one of the many ironies of this system of improvement; namely that, while the British attempted to treat the vastly different ecological systems under their control

using uniform ideologies and methodologies, the result was not the development of similar ecological systems, but the emergence of hybrid ecologies that fostered competitive advantage to distinct forms of life.

Accessing changing ecologies in British imperial urban spaces and drawing connections between these seemingly disparate events requires engagement with the life-sustaining activities of both human and nonhuman organisms, and thus requires methods that incorporate their influence on shared environments. The main mechanism by which this project proposes to engage these processes is through the adaptation of Niche Construction Theory (NCT), a prominent theory in ecology and evolution, to the study of environmental history. I demonstrate how the theory's focus on dynamic material change to ecosystems driven by the life-sustaining processes of organisms, both biological and cultural, at local and global scales, provides an ideal framework for the re-integration of humans into their ecosystems while bypassing controversies related to agency dominant in nonhuman studies. I then demonstrate how many of the main mechanisms of niche construction asserted by its major theorists have corollaries in historical and environmental studies methodologies, particularly in the fields' engagements with the idea of "ecological inheritance" and an interest in tracing the intimacies and lifeways of interacting organisms. Given these common goals, combining the overarching framework of Niche Construction Theory with the nuanced methodologies present in this literature would unite disparate methods in environmental studies under a coherent frame. Niche Construction Theory provides opportunities for more dynamic engagement with nonhumans that shape shared ecologies for historians of the environment, while allowing scholars in this field to introduce more nuanced interpretations of cultural and biological processes to NCT, grounded in historical and anthropological methods.

The following chapters explore the utility of this framework in three different epidemics that occurred in diverse ecologies under disparate governance systems in the British Empire at the end of the nineteenth century. In the settler colony of Melbourne, Australia, I examine the role of urban growth and town planning practices on mortality from *Mycobacterium tuberculosis* and *Mycobacterium bovis*, arguing that the "boom town" nature of urban expansion in the city, a style of urban planning fundamentally at odds with the climate and hydrological patterns of the landscape, and the heterogeneity of cultural and sanitary practices allowed under a fragmented board of health together built an ecological niche for the emergence of the two complimentary bacteria, resulting in higher counts of the disease within the city than any other part of the colony.

Examining an infamous outbreak of *Yersinia pestis* in Bombay, India, a city nested within a major interventionist and paternalist colonial structure, the next case study considers how British imperial intervention combined with the unique balance of rat species – *Rattus rattus* and *Rattus norvegicus* – created an ideal ecological niche for the perpetuation of the disease. British patterns of economic investment fostered informal growth in the city, creating a series of haphazard living spaces ideal for colonization by *Rattus rattus*, a black rat species widely known to occupy human homes; meanwhile sanitary improvement plans ill-suited to the monsoon pattern of rainfall provided additional homes for *Rattus norvegicus*, the reservoir species for plague. I draw on Gregg Mitman's concept of "ecologies of injustice" to explore how these structural and social pressures led to uneven experiences of plague among the city's residents, with mortality and loss sustained overwhelmingly by poor and working-class residents.

The final case study explores the burden of typhoid fever mortality in the hybrid imperial-local city of Belfast, Northern Ireland. It argues that the city's imperial economic pattern of growth (as a town that boomed in the wake of the linen and shipping industries moving to the Island from Britain) and its construction of waste-removal infrastructure modeled off British examples interacted with the hydrological features of the city and the ecology of Belfast bay to form a unique ecological niche for *Salmonella enterica typhi,* perpetuated by local cultural practices in shellfish gathering and consumption. Together, these cases demonstrate how the imposition of common British sanitary ideals and the integration of diverse regions into highly specified local economies interacted with local environmental and cultural conditions to establish unique ecological niches for these diseases. Importantly, the project asserts that it is difficult to understand the emergence of these epidemics, or their effect on the historical trajectory of these cities, without taking a Niche Construction Theory approach.

As the project is uniquely concerned with dynamic change in ecosystems and feedback mechanisms that drive environmental change, it explores the ways that the emergence of these epidemics also inspired change in colonial practice, and thus drove further ecological change. In response to each of these epidemic events, I contend, imperial scientific knowledge-making processes and public health intervention changed the relationship of organisms to each other and to their environments. The emergence of the rat-flea theory as the dominant etiology of plague in Bombay, for example, significantly altered the relationship of both *Rattus rattus* and *Rattus norvegicus* to the urban ecology. Poisoning campaigns often led to forced migration, exposing rats and their arthropod and bacterial counterparts into contact with people or groups of people previously uncontacted; trapping campaigns moved rats from a position of vermin to bacteriological examination, and placed them in a position of value (monetary and bacteriological) within sanitary structures. From these interventions, rats grew to occupy a more diverse niche within the urban ecosystem, both dead and alive.

In Melbourne, the growing body of information around tuberculosis, and particularly around bovine tuberculosis, led to the integration of cattle into public health infrastructure, transforming their daily activities, their abattoirs and cowsheds, and their immune systems and bodily functions into sites of government regulation and observations. This visibility and regulation eventually led to the expulsion of cattle from the urban ecology. Humans suffering from tuberculosis became similarly marginalized in urban systems through their documentation in public health structures, as the construction of sanatoriums on the outskirts of the city and the emergence of ideas around "hygienic citizenship" in the wake of bacteriology encouraged their self-isolation.

Meanwhile in Belfast, the isolation of the cause of local typhoid incidence to cockles similarly led to its integration into the public health system. The specific niche of the cockle, as a marine organism living outside the jurisdiction of the city, however, limited the extent to which bacteriologists and medical officers of health engaged with it. While some conducted extensive surveys of cockle beds along the Irish coast and mapped the distance of these beds from major sewage projects to assess risk, and cockles were occasionally sent to the Queen's University Belfast bacteriological laboratory for examination, most of the interventions around reducing typhoid fever mortality in the city focused on altering behavior. Signs were posted along the shores warning of the relationship between cockles and typhoid, and local public health acts were passed to intervene in the hawking of shellfish on the streets, with limited success. The particular ecology of the cockle, I argue, influenced the mitigation strategy attempted by the local government board, orienting it towards risk communication instead of direct intervention.

The ecology of disease was not the only factor that shaped epidemic control, however; the extent to which public health officials and municipal bodies incorporated new forms of knowledge or chose to rely on entrenched methodologies often determined the trajectory and success of their epidemic control measures. Their willingness to incorporate these new forms of knowledge often relied on whether science aligned with the goals of powerful municipal interests. In Melbourne, the incorporation of bacteriological theory into public health practice was key in uniting a fragmented public health infrastructure under a centralized board, which in turn gained the authority to address longstanding concerns over noxious trade industries through a focus on bovine tuberculosis. In Bombay, conversely, the municipally-organized Bombay Improvement Trust ignored the emergence of rat-flea theory in favor of existing contagionist and miasmatic theories, to the detriment of long-term plague control, because these logics aligned with municipal goals of slum clearance and town planning. In Belfast, acceptance of bacteriological theory and the utilization of a risk communication practice through a centralized, representative public health infrastructure promoted behavioral change among city residents, leading to a decline in typhoid incidence through reduced wild shellfish consumption.

In the conclusion, I draw together these case studies to reflect on the importance of historical methods that address ecosystem complexity and dynamic change driven by both humans and nonhumans. I ground reflections in the current epidemic context of COVID-19, emphasizing how examination of past epidemic contexts using ecologically and culturally dynamic lenses reveals the overlapping global and local forces that determine the emergence of a disease, the populations most affected by it, and the lasting changes to institutions and spaces that result. Finally, I suggest other geographic or thematic topics in history that could be equally enriched by a Niche Construction Theory framework and reflect on the implications of this approach for the discipline.

Preface

There were rumors that the pandemic started in China. The expansion of trade networks and peoples into the countryside brought humans and animals into closer and closer contact, allowing the microbe to jump from one welcoming host to another. At first, cases were few, and connected only to the site of first transmission; then, as peoples and goods moved, the microbe moved with them. By the time authorities noticed, it was too late. The disease was already ravaging Chinese cities, and within months, cases were popping up around the world, spreading rapidly along transport lines. Panicked, global leaders convened a series of conferences to decide on preventative measures to slow the spread of the disease, but few were willing to sacrifice trade, and quarantine strategies remained highly controversial. International scientific communities raced to characterize and identify the disease and produce an effective vaccine. Different strains evolved as the disease settled into diverse ecological niches, in South Africa, Australia, and South Asia. The strain most prevalent in India appeared to facilitate increased rates of transmission.[1] India suffered the highest case and fatality rates, with millions of infections and hundreds of thousands of deaths. Public health systems strained under uneven and ineffective governance, and a disease that seemed perfectly suited to spread in the crowded, inequality-ridden commercial cities.

Reading the above description, any person who had lived through the years 2020-2021, would (fairly) assume that the microbe of interest was the SARS-CoV-2 virus, an epizootic that has been linked to expanding urbanization and patterns of consumption in China.[2] The case described above, however, occurred over 100 years before the emergence of SARS-CoV-2; and

[1] L Xu, LC Stige, H Leirs, S Neerinckx, KL Gage, R Yang, Q Liu, B Bramanti, K Dean, H Tang, Z Sun, NC Stenseth, and Z Zhang, "Historical and genomic data reveal the influencing factors on global transmission velocity of plague during the Third Pandemic," *PNAS* 166 (2019): 11833-11838, doi: 10.1073/pnas.1901366116.

[2] Cristina O'Callaghan-Gordo and Joseph M. Anto, "COVID-19: The disease of the anthropocene," *Environmental Research* 187(2020): 109683, doi/10.1016/j.envres.2020.109683.

the disease in question was not an entirely novel virus. *Yersinia pestis* spilled over into human populations frequently; on two occasions previously, ecological, social, political, and economic circumstances aligned to produce the ideal circumstances for the bacteria to spread widely and rapidly, killing millions of people before receding once again, maintaining low-level circulation in sylvatic reservoirs. Its history in the city of Bombay, India, is one of the major case studies of this dissertation.

While haunting similarities exist between the third plague pandemic and the COVID-19 pandemic, the local circumstances leading to their emergence were highly singular. Recent events have brought into sharp focus the relationship between the specific characteristics of a disease – its biology, transmission patterns, interaction with the immune system, and mutation in response to different environmental pressures and ecological niches – and its ability to thrive. As zoonotic pathogen experts are fond of emphasizing, a very specific and highly unlikely cascade of events is necessary for a pathogen to become widespread, let alone become a global pandemic.[3] Despite at least a few identified spillover events every year, it has been nearly forty years since the last pandemic event (HIV/AIDS), and over a hundred years since the 1918 Spanish Influenza epidemic.[4] However, we also know that the speed of these events is increasing, and will likely continue to increase, because of globalized political, economic, and

[3] A few examples of this line of thought within disease ecology include Raina K. Plowright, Colin R. Parrish, Hamish McCallum, Peter J. Hudson, Albert I. Ko, Andrea L. Graham, and James O. Lloyd-Smith, "Pathways to Zoonotic Spillover," *National Review of Microbiology* 15(2017): 502-510, doi: 10.1038/nrmicro.2017.45; Kathleen A. Alexander, Colin J. Carlson, Bryan L. Lewis, Wayne M. Getz, Madhav V. Marathe, Stephen G. Eubank, Claire E. Sanderson, and Jason K. Blackburn, "The Ecology of Pathogen Spillover and Disease Emergence at the Human-Wildlife-Environment Interface," *The Connections Between Ecology and Infectious Disease* 5(2019):267-298, doi: 10.1007/978-3-319-92373-4_8; Christine Kreuder Johnson, Peta L. Hitchens, Tierra Smiley Evans, Tracey Goldstein, Kate Thomas, Andrew Clements, Damien O. Joly, Nathan D. Wolfe, Peter Daszak, William B. Karish, and Jonna K. Mazet, "Spillover and pandemic properties of zoonotic viruses with high host plasticity," *Scientific Reports* 5(2015), https://doi.org/10.1038/srep14830.

[4] The seasonal flu can be thought of as an annual pathogen spillover event, where the recombination of flu virus (usually when two strains simultaneously infect a pig) leads to new strains that require prediction and new vaccines. See DM Morens and AS Fauci, "Emerging Infectious Diseases: Threats to Human Health and Global Stability," *PLoS Pathog* 9(2013): e1003467, https://doi.org/10.1371/journal.ppat.1003467.

social structures that facilitate human-wildlife interactions and provide opportunities for rapid global transmission. While operating on a much faster and larger scale than ever before (a product of what John McNeill and Peter Engelke call the "Great Acceleration"), the systems of power, rapid globalized transport and trade, organized and transnational bureaucracy, and patterns of capitalist development and land use change that facilitated our current pandemic predicament – and in no small part, the Anthropocene – had their origins in the age of imperialism.[5] Thus taking a longer perspective, the circumstances that led to the emergence of the third plague epidemic and the SARS-CoV-2 pandemic are variations of a defined system of human-environmental interaction that, while evolving, has dominated global ecologies for over a hundred years, and forms the basis of contemporary globalized life. The Anthropocene, an epoch ushered in by the highly specific set of Western-dominated, capitalist-oriented structures that drive global land use change, is undoubtedly an epoch of pandemics.

In this dissertation, I take a comparative perspective on epidemic disease during this transformative period, examining how fundamental shifts in the relationships of peoples to their environments in the late nineteenth century carried real biological consequences for the organisms involved. I argue that the structures of imperialism – and in this case specifically, British imperialism – facilitated the widespread dissemination of disease by establishing patterns of human-environmental interaction that, while arguably diverse, resulted in the creation of ecological niches for a variety of infectious diseases. Just as COVID-19 spread due to economic and ecological circumstances that occurred on a global scale but differed in severity based on the unique ecology, cultural characteristics, and social organization of an area, this project contends, the economic and ecological pressures of the British empire scattered a series of diseases widely,

[5] Chris Otter, Alison Bashford, John L. Brooke, Fredrik Albritton Jonsson, and Jason M. Kelly, "Roundtable: The Anthropocene in British History," *Journal of British Studies* 57(2018): 583.

but the success of these diseases was contingent on the highly specific ecological niche of a particular region, city, or neighborhood. Examining the multi-scalar relationships that drove these epidemics through the lenses of biology and history together allows for new insights into these sometimes mundane, sometimes spectacular events – and shows why, despite countless medical innovations, the epoch of pandemics is far from over.

Introduction

In the late nineteenth century, reports flooded into the British metropole from the colonies, many telling what seemed like the same story: twenty years ago, this disease was not a problem; now, it killed thousands annually. In 1870, a medical doctor named William Thomson published a monograph on tuberculosis in Melbourne, Australia, in which he warned of the prevalence of the disease within the city and suburbs. Thomson suggested that "[n]early 1 in 3 of the adult population of Melbourne, between the ages of 20 and 45 years and above 1 in 4 in the whole colony of Victoria, at the same ages, die of phthisis."[1] Across the Empire, the Belfast Health Commission noted a worrying trend of excessive mortality as well, but from an entirely different cause. "Mortality from enteric fever in Belfast has been excessive in almost every year since the time [1872]," the report stated.[2] At nearly the same time as Thomson's investigations into tuberculosis were picked up by the Melbourne Sanitary Commission and the Belfast Health Commission convened to address rates of typhoid fever, reports reached the Bombay Municipal Commissioner of dozens of rats emerging from burrows into the streets in sickly or dying condition. The outbreak of bubonic plague that followed would spread across the country and the world, cause panic and infect millions.[3]

The pattern described in these records runs counter to a prominent historical narrative that labels the late nineteenth century an era of declining infectious disease.[4] The proliferation of novel waste removal and water filtration systems altered the ecology of disease in European

[1] William Thomson, *On Phthisis and the Supposed Influence of Climate*, (Melbourne: Stillwell & Knight, 1870), 5.
[2] Belfast Health Commission. *Report to the Local Government Board for Ireland* (Dublin: Alex. Thom & Co., 1908), 25. Public Records Office of Northern Ireland, LA/7/3/JA/22.
[3] PCH Snow, *Report on the Outbreak of Bubonic Plague in Bombay, 1896-1897. By PCH Snow, Esq, LCS, Municipal Commissioner for the City of Bombay…etc.,* (Bombay: "Times of India" Steam Press, 1897), 9, The British Library, V/27/856/7.
[4] Michael Worboys, *Spreading Germs: Disease Theories and Medical Practice in Britain*, 1865-1900, (Cambridge: Cambridge University Press, 2000); George Rosen, *A History of Public Health*, (Baltimore: Johns Hopkins Press, 1958); David S. Barnes, *The Great Stink of Paris and the Nineteenth-Century Struggle against Filth and Germs*, (Baltimore: Johns Hopkins University Press, 2006).

urban spaces beginning in the 1850s, facilitating a remarkable decline in endemic illnesses. However, these cases point to an opposing phenomenon: that the age of improvement and sanitary development was, for many imperial urban spaces, also an age of virulent epidemic disease. While it is tempting to consider these epidemic events as random, or as an expected occurrence to be attributed to uneven adoption of "modern" sanitary measures, this project contends that these three cases tell a more complex story tied to imperial trends in land use change and ecology. Examining these cities together and through an environmental lens, several important patterns emerge: each city underwent explosive growth just before its documented epidemic; each was a key port for British imperial trade networks; and perhaps most importantly, each experienced an epidemic of a disease previously nonexistent or negligible in that city, which spread at unprecedented rates.

In this dissertation, I argue that these patterns – economic integration into a vast, global empire; subsequent land use and demographic change; and the eruption of a new, challenging epidemic – are representative of a series of complex ecological processes that inextricably linked humans and their environments. Looking not only at dramatic, highly-visible epidemics, but the smaller, quotidian diseases that killed thousands of people on local scales, I assert that the unique interactions of imperial political, economic, and social systems of power with agentive, specific, and overlapping ecosystems created ecological and evolutionary opportunities for microbial diseases to thrive in novel places and populations – and that in order to truly understand the emergence of these microbes, we must think of these systems as interconnected and biologically consequential.

This project draws on these three case studies—Melbourne, Bombay, and Belfast—to explore the relationship between empire-building, environmental management, and epidemic disease. It argues that the expansion of the late-nineteenth century British Empire was characterized by densely populated urban centers, massive reclamation and improvement projects, and deeply stratified systems of inequality, which together formed a unique system of environmental engineering. When applied to the ecosystems that sustained these major colonial cities, all of which represented unique climatic conditions and distinct colonial relationships, these alterations led to an increasingly competitive environment for particular microbes, resulting in the emergence and propagation of diseases unique to particular colonial ecologies. Responses to these epidemics, this project contends, altered the way British subjects interacted with imperial landscapes, and influenced knowledge concerning disease, environment, and urban spaces. In their attempt to engineer climates and ecosystems, the British imperial administrators and settlers destabilized them, which created a new series of challenges and changes to health, livelihood, and the imperial project as a whole.

Imperial and local, biological and socioeconomic, human and nonhuman – the scales of reference implicated in this project are many, and their boundaries are easily blurred. To convey the complexity of these overlapping ecosystems and human and nonhuman pressures, an expansion of historical methodology and theory is necessary. In the wake of many decades of research that demonstrate both the direct and indirect effects of human action on ecosystems near and far, and the many cascading effects of nonhuman life processes on human and nonhuman existence, historians require more rigorous methods for assessing and conveying the plurality of relationships that have always shaped, and continue to shape, our world. While historians and environmental studies scholars have long grappled with this problem (even more so in the wake of the Anthropocene), insights from ecology, which has always seen human activity as grounded in and consequential to environmental processes, hold the key to conveying these relationships. I contend that Niche Construction Theory, an ecological and evolutionary theory that prioritizes the effects of life-sustaining activities of one organism (or group of organisms) on another in a shared ecosystem, can be combined with existing methodologies in environmental studies to fill this epistemological gap.

Material Environments and Microbial Lives: Methods and Historiography

Microbes are, and always have been, the backbone of the biosphere. They are the drivers of planetary processes and the co-inhabitants of our living bodies, the producers of the oxygen we breathe and the consumers of our waste.[5] Microbes are the originators of all multicellular life on Earth and will likely outlive it. As science writer Ed Yong playfully asserts, "It is said that we are now in the Anthropocene; a new geological period characterized by the enormous impact that humans have had on the planet. You could equally argue that we are still living in the Microbiocene: a period that started at the dawn of life itself and will continue to its very end."[6]

It seems fitting that these organisms, which shaped the environments and bodies in which human history occurred, are the focus of a project that seeks to break the boundaries between humans and their ecologies. However, while much of the present enthusiasm around microbes has been concentrated around bacteria that are symbiotic or beneficial to human existence, coevolving through a mutualistic relationship, the focus of this dissertation is on far more insidious bacteria. The microbes that feature so heavily in this work are subject to the same historical privilege as many people: they are here because they are visible. It is estimated that less than 100 species of bacteria cause infectious diseases, a tiny portion of the tens of thousands

[5] Ed Yong, *I Contain Multitudes: The Microbes Within Us and a Grander View of Life*, (London: Penguin Random House, 2016), 8. "Even now," Yong notes, "The photosynthetic bacteria in the oceans produce the oxygen in half the breaths you take, and they lock away equal amounts of carbon dioxide."
[6] Ed Yong, *I Contain Multitudes*, 9.

known to exist worldwide. But the moments in which they meet with human bodies – especially on a large scale – populate the historical record.[7] When Louis Pasteur and Robert Koch first peered through their respective microscopes at *Bacillus anthracis* and *Pasteurella multocida*, they were seeking causes for outbreaks of disease. The subsequent blossoming of a new discipline, bacteriology, was grounded in this assumption – that microbes are disease-causing agents – and the thousands of reports, studies, and inquiries produced by the enterprise line government archives and medical libraries.[8]

Given the extensive documentation of these microbes, it is no surprise that historians have noted the connection between disease-causing organisms and environment across time and space.[9] Many of the foundational texts of environmental history have explored the relationship between environment and disease. In his groundbreaking work, *Ecological Imperialism: The Biological Expansion of Europe*, *1600-1800*, disease was central to how Alfred Crosby theorized the role of ecological niches and biological objects in the colonization of the New World by Europeans, arguing that it was the existence of "empty niches" brought on by the initial

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[7] Ed Yong, *I Contain Multitudes*, 11.

[8] Porter, Roy. *The Greatest Benefit to Mankind: A Medical History of Humanity*, (New York: W.W. Norton Press, 1997), 428-462.

[9] Alfred Crosby, *Ecological Imperialism: The Biological Expansion of Europe, 900-1900,* (Cambridge: Cambridge University Press, 1986); William McNeill, *Plagues and Peoples,* (New York: Anchor Books, 1976); Warwick Anderson, *Colonial Pathologies: American Tropical Medicine, Race, and Hygiene in the Philippines,* (Durham: Duke University Press, 2006); Warwick Anderson, "Natural histories of Infectious Disease: Ecological Vision in Twentieth-Century Biomedical Science," *Osiris* 19(2004): 39-61; Randall Packard, *The Making of a Tropical Disease, a Short History of Malaria*, (Baltimore: Johns Hopkins University Press, 2007); Randall Packard, *White Plague, Black Labor: The Political Economy of Health and Diseases in South Africa*, (Berkeley: University of California Press, 1989); Nancy Tomes, *The Gospel of Germs: Men, Women, and the Microbe in American Life*, (Cambridge: Harvard University Press, 1999); Linda Nash. *Inescapable Ecologies: A History of Environment, Disease, and Knowledge,* (Berkeley: University of California Press, 2007); Gregg Mitman, "In Search of Health: Landscape and Disease in American Environmental History," *Environmental History* 10 (2005): 184-210; Helen Tilley, "Ecologies of Complexity: Tropical Environments, African Trypanosomiasis, and the Science of Disease Control Strategies in British Colonial Africa, 1900-1940," *Osiris* 19(2004): 21-38; there is also an extensive history on toxicity, occupational health, and environment that, while not specifically about the human microbial relationships, brings these ideas together in innovative and revealing ways. For an excellent review of histories of environment and disease, see Linda Nash, "Beyond Virgin Soil Epidemics: Disease as Environmental History," in The Oxford Handbook of Environmental History, Andrew C. Isenberg, ed., (Oxford: Oxford University Press, 2014): 73-106.

ecosystem trauma wrought by contact that eventually allowed European organisms to settle.[10] His earlier work, *The Columbian Exchange*, drew on budding practices of historical demography and biology to posit an explanation for the outbreaks of disease that followed European invasion of the Americas, coining the events (infamously) "virgin soil epidemics."[11] William McNeill, meanwhile, drew on infectious disease as a frame for the rise and fall of civilizations, placing environmental and social drivers of change over time central in his retelling of human history.[12]

While these histories have undoubtedly revealed the importance of disease and environment in shaping human history, they presented a simplified and at times careless interpretation of biology. While Crosby provides an influential model for considerations of biological actors in the process of imperialism, the unidirectional nature of the thesis leaves little room for considerations of how biological objects from the colonies in turn "colonized" Europe, or just as importantly, how indigenous ecosystems reacted to the introduction of new species. More problematically, as many critics have pointed out, both McNeill and Crosby's interpretation of immunity and disease present biologized justifications for colonial conquest. Both works have thus been viewed as perpetuating racialized interpretations of historical events – leading many subsequent generations of historians to shy away from engaging biology.[13] While it has since been argued it was the historical discipline, not disease ecology, that emphasized differences in immunity as a key feature of European conquest, historians have been slow to

[10] Crosby, *Ecological Imperialism*, 83-85.
[11] Crosby, *Ecological Imperialism*, 118.
[12] McNeill, *Plagues and Peoples.*
[13] Linda Nash, "Beyond Virgin Soil Epidemics," 80. David Jones, on the other hand, argues that Crosby actually downplayed the genetic weakness hypothesis and instead emphasized many environmental factors, but argues that subsequent historical work needs to be extremely nuanced in its treatment of the social determinants of disease or risk being interpreted as supporting racial theories. David S. Jones, "Virgin Soil Epidemics," *The William and Mary Quarterly* 60(2003): 703-742.

return to material histories of disease.[14] It is against this unnuanced biologizing that many historians have espoused a less reductionist version of environmental history.

Despite, or perhaps because of, the discipline's early engagements with the biology and ecology of disease, the field has followed the cultural turn in historical study between its formation and the present.[15] The vast majority of environmental histories written in the late twentieth century constructed narratives in which issues of ecology, biology, and climate were crucial, but relegated to a subordinate position in favor of social and cultural perceptions of environment and the role of political structures in shaping environments.[16] While critical to understanding contemporary philosophies of human-environmental interaction and articulating the stakes of human exploitation of the environment, these works largely relegate environment to the position of subject rather than participant, positioning man as the primary actor and environment (either broadly or narrowly defined) as subordinate. Histories of disease largely followed this turn towards the social, focusing less on the ecology of disease and more on what have been broadly described as the social, cultural, and political determinants of health. These stories place historical agency almost exclusively on humans and human systems, largely presenting a static, uncomplicated picture of other organisms and environments; in short, they are

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[14] Linda Nash, "Beyond Virgin Soil Epidemics," 80.

[15] Linda Nash, "Writing Environmental Histories of Disease in the Anthropocene," in Chirs Otter, Nicholas Breyfogle, John L. Brooke, Mari K. Webel, Matthew Klingle, Andrew Price-Smith, Brett L. Walker, and Linda Nash, "Forum: Technology, Ecology, and Human Health Since 1850," *Environmental History* 20 (2015): 710-804.

[16] Some of the key works in this vein include Carolyn Merchant, *The Death of Nature: Women, Ecology, and the Scientific Revolution*, (New York: Harper Collins Press, 1980); William Cronon, "The Trouble with Wilderness: Or, Getting Back to the Wrong Nature," *Environmental history* 1(1996): 7-28; Conevery Bolton Valenčius, *The Health of the Country: How American Settlers Understood Themselves and Their Land* (New York: Basic Books, 2002)*;* Donald Worster, *Nature's Economy: A History of Ecological Ideas,* (San Fracisco: Sierra Club, 1977); Richard Grove, *Green Imperialism: Colonial Expansion, Tropical Island Edens and the Origins of Environmentalism, 1600-1860,* (Cambridge: Cambridge University Press, 1996); Mark Fiege, *Irrigated Eden: The Making of an Agricultural Landscape in the American West,* (Seattle: University of Washingon Press, 1999); Ian Tyrell, *True Garden of the Gods: Californian-Australian Environmental Reform, 1860-1930* (Berkeley: University of California Press, 1999); Linda Nash, "The Changing Experience of Nature: Historical Encounters with a Northwest River," *Journal of American History* 86(2000): 1600-1629; Nancy Langston, *Where Land & Water Meet: A Western Landscape Transformed* (Seattle: University of Washington Press, 2003).

excellent histories, but they are not environmental histories, and microbes are few and far between.

Those studies that continued to focus on the relationship of material environment and disease, while pathbreaking, remained marginal, and were largely subsumed under other types of history – from history of medicine to histories of colonialism or capitalism. [17] Many of these works, with several notable exceptions, continue the early trend of focusing on focuses on moments of geopolitical tension, the traditional "clash of civilizations" drawn from Crosby's *Ecological Imperialism* as flashpoints for epidemics and moments of the assertion of nonhuman disease agency. While these cases certainly open conceptual space for dynamic environmental conditions and more nuanced understandings of disease ecology, they also tend to overwhelmingly focus on vector-borne illness in tropical settings – arboviruses in particular – eschewing diseases common to urban spaces in the northern latitudes. These diseases are instead naturalized and treated uncritically as a cost of human density rather than ecologically-specific. In *Mosquito Empires,* for example, John McNeill claims that the "crowd diseases" common across Europe, "prevailed where populations were dense and interactive, and immunized survivors; they did not depend on the specific environmental conditions."[18] This view not only ignores the ecological complexity of urbanized and industrialized environments (a reality that decades of urban environmental histories have powerfully conveyed), but relegates environment-

[17] Randall Packard, *White Plague, Black Labor.* Christian McMillen, *Discovering Tuberculosis: A Global History 1900 to the Present*, (New Haven: Yale University Books, 2015); Jacques Pepin, *the Origin of AIDS*, (Cambridge: Cambridge University Press, 2012); David Barnes, The Great Stink of Paris and the Nineteenth-Century Struggle Against Filth and Germs, (Baltimore: Johns Hopkins University Press, 2006); Richard Evans, *Death in Hamburg*: *Society and Politics in the Cholera Years, 1830-1910*, (New York: Penguin Books, 2005); Abena Dove Osseo-Asare. *Bitter Roots: The Search for Healing Plants in Africa* (Chicago: University of Chicago Press, 2014); Helen Tilley, *Africa as a Living Laboratory: Empire, Development, and the Problem of Scientific Knowledge, 1870-1950,* (Chicago: University of Chicago Press, 2011).

[18] John McNeill, *Mosquito Empires: Ecology and War in the Greater Caribbean, 1620-1914*, (Cambridge: Cambridge University Press, 2010), 10.

disease interaction to moments of spectacularity; in these tales, disease is not an integral part of an ecology humans share, but an interloper, disrupting plans and designs before vanishing from the historical record.

The emergence of Anthropocene studies in the last decade breathed new life into material histories of environment and disease. As historians have come to reckon with the fragility and consequence of human (especially Western, wealthy) action in earth systems, conceptual spaces have opened for the consideration of biological forces in historical time – but a biology that is a far cry from Crosby's. The posited effects of planetary warming threw into question the survivability of human societies, and rendered clear our dependence on a stable, narrow band of environmental conditions – a "safe operating space" that was now being threatened.[19] Projections of species collapse demonstrated the intertwining of all living species on earth, from bees to algae to the crops we rely on. The microbial revolution, meanwhile, showed that human beings themselves "contained multitudes," further highlighting human dependence on and integration within the complex ecosystems that comprise the biosphere.[20] Grappling with these discoveries has lent increasing power to the rise of a more nuanced biology, an "extended synthesis" that proposes a broader chain of causation and a greater role for complex systems – including social and cultural conditions as well as organismal complexity – in shaping life.[21] Environmental scholars have thus been gradually confronted with the reality that former ideas of human place in

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[19] Johan Rockström, Will Steffen, Kevin Noone, Åsa Persson, F. Stuart Chapin III, Eric F. Lambin, Timothy M. Lenton, Marten Scheffer, Carl Folke, Hans Joachim Schellnhuber, Björn Nykvist, Cynthia A. de Wit, Terry Hughes, Sander van der Leeuw, Henning Rodhe, Sverker Sörlin, Peter K. Snyder, Robert Costanza, Uno Svedin, Malin Falkenmark, Louise Karlberg, Robert W. Corell, Victoria J. Fabry, James Hansen, Brian Walker, Diana Liverman, Katherine Richardson, Paul Crutzen, and Jonathan A. Foley, "A Safe Operating Space for Humanity," *Nature* 461, 472–475 (2009). https://doi.org/10.1038/461472a

[20] Julia Adeney Thomas, "History and Biology in the Anthropocene: Problems of Scale, Problems of Value," *American Historical Review* 119 (2014): 1587-1607.

[21] Gerd B. Muller, "Why an extended evolutionary synthesis is necessary," *Interface Focus* 7(2015):20170015. Evolution: The Extended Synthesis, Massimo Pigliucci and Gerd B. Müller, (Cambrdige: MIT University Press, 2010).

the world "were deeply mistaken," and must reconcile their work in an entirely new "environment" – a dynamic, multi-layered system that they did not act upon, but acted within.[22] Historians like Julia Adeney Thomas and Dipesh Chakrabarty have argued that these realities collapse the boundaries between the human and nonhuman, between biology and history, and prompt the development of novel methods that account for the biogeochemical processes and life-sustaining relationships on which all human activity depends.[23]

The methods historians have suggested to make sense of this newly revealed relationship had become equally dynamic. A successful subset of these methodologies has focused on the impact of human political, cultural, and economic development on far-spanning ecosystems, and the ways in which those ecosystems responded. Linda Nash, for example, argues that humans are both "agents" and "objects" of environmental change in her work *Inescapable Ecologies*, where she emphasizes the impact that anthropogenic land use change in turn held for occupational health in the Central Valley of California.[24] Others have suggested a more radical engagement with scientific methodology. Historians like Edmund Russell and Daniel Lord Smail have begun to consider the role of human-induced evolution in shaping human history, and the coevolution of human and nonhuman populations, while others like Chris Otter, Nicholas Breyfogle, and John Brooke have argued for considerations of human cultural and evolutionary niche construction in our understanding of history.[25] Meanwhile, Otter and Smail suggest the development of a "biological history," in which historians engage with the complexities of living

[22] Timothy J. LeCain, *The Matter of History: How Things Create the Past*, (Cambridge: Cambridge University Press, 2017), 8.
[23] Julia Adeney Thomas, "History and Biology in the Anthropocene: Problems of Scale, Problems of Value," *American Historical Review* 119 (2014): 1587-1607. Dipesh Chakrabarty, "The Climate of History, Four Theses," *Critical Inquiry* 35(2009): 197-222.
[24] Linda Nash, *Inescapable Ecologies.*
[25] Edmund Russell, *Evolutionary History: Uniting History and Biology to Understand Life On Earth,* (New York: Cambridge University Press), 2; Chris Otter et al., "Forum: Technology, Ecology, and Human Health Since 1850," 710–804.

and nonliving influences to historical development.[26] Others, especially climate historians like Dagomar Degroot, Timothy Newfield, and Kyle Harper have begun to draw on paleoclimatology, archaeology, genetics, and linguistic evidence to re-situate the past in environmental and climatological context.[27] These methods engage heavily with "new materialism," an interdisciplinary field of inquiry that asserts that human social, cultural, political, and economic systems emerge "in significant part from a broader world of intelligent and creative animals, plants, metals, and other material things that have made us."[28]

Despite the engagement of many of these projects with disease, and an increasing focus on conveying environmental dynamism, shockingly few histories have attempted to bring these few elements together. Few historians have truly grappled with the fact that, as Chris Otter notes, "all diseases have their own pathogen-ecology system."[29] This gap exposes a far more systematic deficiency in new materialist environmental histories. Individually, these proposed methodologies bring nuance to environmental-human interaction different ways and a convey strong interest in engaging scientific methods directly. However, each approach can only really be seen as part of a whole, a prioritization of a small subset of environmental features or scientific methods that create an incomplete picture of environmental change and human

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[26] Chris Otter, "Planet of Meat: A Biological History," in Tony Bennet, ed., *Challenging (the) Humanities* (Canberra: The Australian Academy of the Humanities, 2013).
[27] Dagomar Degroot, Kevin Anchukaitis, Martin Bauch, Jakob Burnham, Fred Carnegy, Jianxin Cui, Kathryn de Luna, Piotr Guzowski, George Hambrecht, Heli Huhtamaa, Adam Izdebski, Katrin Kleemann, Emma Moesswilde, Naresh Neupane, Timothy Newfield, Qing Pei, Elena Xoplaki, and Natale Zappia, "Towards a rigorous understanding of societal responses to climate change," Nature 591(2021): 539-550; J. Luterbacher, T.P. Newfield, E. Xoplaki, E. Nowatzki, N. Luther, M. Zhang, and N. Khelifi, "Past Pandemics and Climate Variability Across the Mediterranean," *Euro-Mediterranean Journal for Environmental Integration* 5(2020): Kyle Harper, *The Fate of Rome: Climate, Disease, and the End of an Empire*, (Princeton: Princeton University Press, 2018); these innovations have breathed new life into other histories of disease, as well. Recently, Monica Green redefined the temporal and spatial classification of the medieval black plague epidemic using aDNA (ancient DNA) techniques, arguing that the plague moved through Central Eurasia much earlier than previously thought. See Monica Green, "The Four Black Deaths," *The American Historical Review* 125(2020): 1601-1631.
[28] LeCain, *The Matter of History,* 8.
[29] Otter et al., "Forum: Technology, Ecology, and Human Health Since 1850," 712.

integration into environmental systems – drawing on Otter's example, there are pathogens, there are ecologies, but the exploration of the systematic relation between the two is often fundamentally lacking. What is needed is a framework that unites biological, sociocultural, and geochemical methods, one which conveys the dynamism, fuzzy boundaries, and constant motion that we now know characterized both past and present ecosystems. In this project, I suggest that this framework might be adapted from fields that have long focused on describing exactly these relationships and conveying the interconnected nature of humans and environment. Following a tradition of methodological innovation common in environmental and disease history, therefore, I argue that to understand disease in past environments, we must incorporate more critical understandings of ecology and disease by fully engaging the work of ecology and public health science.

Ecology and Public Health Science as Historical Frameworks

While environmental and disease history frame the context and interventions of this project, its scope, lens, and perspective were inspired by a far more interdisciplinary and increasingly political project. Concurrent with the 2003 SARS epidemic, and the publication of Paul Crutzen and Eugene Stoermer's 2000 publication, "The Anthropocene," a large-scale shift occurred in the study of infectious diseases.[30] The theory of "One Health," previously marginal in public health science, came to the forefront of disciplinary discourse as the interconnected nature of human, animal, and ecosystem health, and the stakes of that connection, were made more apparent. A flurry of publications emerged articulating the zoonotic origins of major

[30] Paul Crutzen and Eugene Stoermer, "The Anthropocene," *IGBP Newsletter* 41(2000): 17-18.

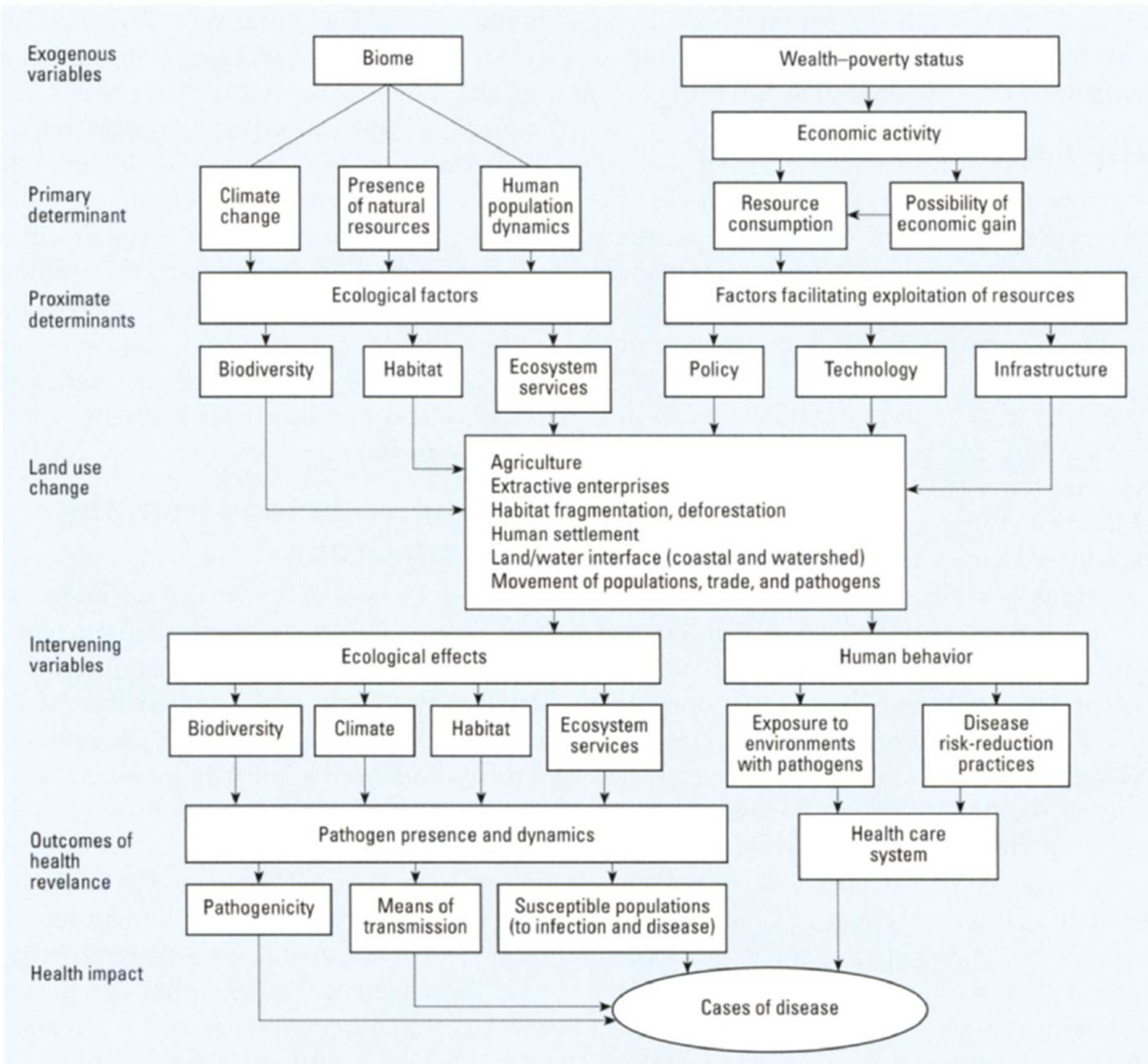
human pathogens – and the likely increase in these transmission events in the twenty-first century.[31]

The convening of the Working Group on Land Use Change and Disease Emergence in 2004 highlighted the extensive scope of the emerging field of inquiry.[32] In an article summarizing the policy recommendations of the working group, Johnathan Patz et al. argued that examining the link between Anthropogenic land use change and infectious disease outbreaks or emergence events was a multi-scalar, multidisciplinary enterprise, and required incorporating human behaviors and systems alongside biological processes. The subsequent expansion of rhetorical space within One Health for qualitative, culturally-centered research provides a powerful example of how to blend biologically and socially centered methodologies under a coherent framework.

[31] P Daszak, AA Cunningham, and AD Hyatt, "Emerging infectious diseases in wildlife – threats to biodiversity and human health," *Science* 287(2000): 443-449; P Daszak, AA Cunningham, and AD Hyatt, "Anthropogenic environmental change and the emergence of infectious diseases in wildlife," *Acta Trop* 78(2001): 103-116. doi: 10.1016/s0001-706x(00)00179-0; Christine Kreuder Johnson, Peta L. Hitchens, Tierra Smiley Evans, Tracey Goldstein, Kate Thomas, Andrew Clements, Damien O. Joly, Nathan D. Wolfe, Peter Daszak, William B. Karesh, and Jonna K. Mazet, "Spillover and pandemic properties of zoonotic viruses with high host plasticity," *Scientific Reports* 5(2015): 14830, doi: 10.1038/srep14830; David M. Morens and Anthony S. Fauci, "Emerging infectious diseases: threats to human health and global stability," *PLoS Pathogens* 9(2013): e1003467. doi: 10.1371/journal.ppat.1003467; Ronald Barrett, Christopher W. Kuzawa, Thomas McDade, George J. Armelagos, "Emerging and Re-Emerging Infectious Diseases: The Third Epidemiologic Transition," *Annual Review of Anthropology* 27(1998): 247-271. doi: 10.1146/annurev.anthro.27.1.247; David M. Morens, Gregory K. Folkers, and Anthony S. Fauci, "The Challenge of Emerging and Re-Emerging Infectious Diseases," *Nature* 430(2004): 242, 249. doi: 10.1038/nature02759.

[32] Johnathan A. Patz, Peter Daszak, Gary M. Tabor, A. Alonso Aguirre, Mary Pearl, Jon Epstein, Nathan D. Wolfe, A. Marm Kilpatrick, Johannes Foufopoulos, David Molyneux, David J. Bradley and the Members of the Working Group on Land Use Change and Disease Emergence, "Unhealthy Landscapes: Policy Recommendations on Land Use Change and Infectious Disease Emergence," *Environmental Health Perspectives* 112 (2004): 1092. doi:10.1289/ehp.6877.

Figure 1. A diagram of factors contributing to land-use driven infectious disease emergence as determined by the Working Group on Land Use Change and Emerging Infectious Diseases.



Source: Johnathan A. Patz, Peter Daszak, Gary M. Tabor, A. Alonso Aguirre, Mary Pearl, Jon Epstein, Nathan D. Wolfe, A. Marm Kilpatrick, Johannes Foufopoulos, David Molyneux, David J. Bradley and the Members of the Working Group on Land Use Change and Disease Emergence, "Unhealthy Landscapes: Policy Recommendations on Land Use Change and Infectious Disease Emergence," *Environmental Health Perspectives* 112 (2004): 1096. doi:10.1289/ehp.6877.

Examining the diagram above (Figure 1), it becomes clear that many of the phenomena central to the consideration of emerging infectious diseases – economic activity, exploitation of resources, human population dynamics, infrastructure, agriculture, and the movement of population, trade, and pathogens, to name a few – are critical domains of inquiry for environmental historians and social scientists. This link has not gone unnoticed in the scientific community, as public health experts like Rosemary McFarlane have emphasized the importance of the historical perspective, noting its ability to provide "a political, socio-economic, and ecological background to infectious disease emergence."[33] The result of this reaching across disciplines has been the development of a series of highly interdisciplinary teams comprised of ecologists, pathologists, and medical anthropologists who draw on cross-disciplinary language to articulate local and global patterns of infectious disease. One Health research subsequently has drawn on methods as diverse as ethnography, education intervention, oral history, statistical modeling, and human and animal pathology, together producing an extensive mixed-methods literature which forms the backbone of discussions of pathogen dynamics and human-animal interfaces in this project.[34] While historians like Linda Nash have also noticed the overlap between the goals of historical disease research and public health research, arguing that "the complex mix of social, biological, and environmental factors driving EIDs …calls out for more historical and anthropological work," it appears that public health scholars have been much more

[33] Rosemary A. McFarlane, Adrian C. Sleigh, and Anthony J. McMichael, "Land Use Change and Infectious Disease on an Island Continent," *Int. J. Environ. Res. Public Health* 10 (2013): 2712-2713. doi: 10.3390/ijerph10072699.

[34] V. Narat, L. Alcayna-Stevens, S. Rupp, T. Giles-Vernick, "Rethinking Human-Nonhuman Primate Contact and Pathogenic Disease Spillover," *Ecohealth* 14(2017):840-850. doi: 10.1007/s10393-017-1283-4; PM David, E Nakouné, and T Giles-Vernick, "Hotspot or blind spot? Historical perspectives on surveillance and response to epidemics in the Central African Republic," *Int J Public Health* 65(2020): 241–248. https://doi.org/10.1007/s00038-020-01338-x; V Narat, KR Amato, N Ranger, M Salmona, S Mercier-Delarue, S Rupp, P Ambata, R Njouom, F Simon, T Giles-Vernick, J LeGoff, "A multi-disciplinary comparison of great ape gut microbiota in a central African forest and European zoo," *Sci Rep* 10(2020):19107. doi: 10.1038/s41598-020-75847-3; G Limon, EG Lewis, YM Chang, H Ruiz, ME Balanza, & J Guitian, "Using mixed methods to investigate factors influencing reporting of livestock diseases: a case study among smallholders in Bolivia," *Preventive Veterinary Medicine*, 113(2014): 185–196. https://doi.org/10.1016/j.prevetmed.2013.11.004; CG Himsworth, Y Bai, MY Kosoy, H Wood, A DiBernardo, R Lindsay, J Bidulka, P Tang, C Jardine, D Patrick, "An investigation of Bartonella spp., Rickettsia typhi, and Seoul hantavirus in rats (Rattus spp.) from an inner-city neighborhood of Vancouver, Canada: is pathogen presence a reflection of global and local rat population structure?" *Vector Borne Zoonotic Disease* 15(2015):21-6. doi: 10.1089/vbz.2014.1657.

inclined to develop methods that take practical steps towards reconciling these methods than historians.

The incorporation of socioeconomic and other anthropogenic drivers of disease in One Health stems in part from the field's strong engagement with disease ecology, and thus with changing methods of ecological and evolutionary practice characteristic of the extended synthesis. Recent studies in ecology have attempted to include contingency and context into ecosystem models, effectively integrating social science research (and historical research) into frameworks for land use management and ecological analysis. Niche Construction Theory (NCT), developed by F. John Odling-Smee, Marcus Feldman, and Kevin Laland in 2003, seeks to incorporate the dynamic agency of organisms acting within the same ecosystem and their ability to shape the evolutionary and ecological roles of one another over time.[35] As a theory that crosses ecosystem ecology, population ecology, and evolution, NCT prioritizes "organism-driven environmental modification," predicated on activity and reactivity of organisms within the same ecological systems. Odling-Smee et al. describe an interest in the relationship between biology and "human cultural processes," broadly conceived, even expanding the boundaries of the term to incorporate human cultural practices that drive environmental modification – a process they refer to as cultural niche construction (for examples, see the righthand side of Figure 1). Much like public health scientists, I argue, historians might find in Niche Construction Theory an ideal theoretical and methodological framework to incorporate biological and social complexity into their research in a way that builds bridges with other scientific and social-scientific disciplines.

[35] F. John Odling-Smee, Kevin Laland, and Marcus Feldman, *Niche Construction: The Neglected Process in Evolution,* (Princeton: Princeton University Press, 2003); Kevin Laland, Blake Matthews, and Marcus W. Feldman, "An Introduction to Niche Construction Theory," *Evolutionary Ecology* 30(2016): 191-202.

While at first glance it may seem that my interest in engaging the discourse around land use change and emerging infectious diseases is to make historians relevant in public health and policy, these fields of inquiry have much to offer the discipline of history, as well. Just as flattening the complexity of human systems weakens the effectiveness of infectious disease inquiry, so the flattening of the left side of Patz et al.'s diagram – pathogen dynamics, means of transmission, habitat, and other biological features – will weaken the claims historians can make about human-environmental interaction and its role in disease emergence. Thus the heart of the project lies in demonstrating that the incorporation of these multidisciplinary frameworks adds immeasurably to our historical understanding of epidemics. Examining the three contemporaneous epidemics featured in this study through the lens of Niche Construction Theory both provides a structural framework for comparison, illuminating networks of trade, migration, and political pressure that influenced the shape and structure of colonial ecologies, and accounts for the quotidian interactions of humans, nonhumans, and environments that allowed for the success of a particular pathogen. Through NCT, historians have the opportunity to convey the richness and dynamism of past environments; and in doing so, come to terms with the central revelation of the Anthropocene: that we cannot be separated from the earth systems in which we live, and from the other organisms with whom we share the planet.

As changing methods in health studies suggest, microbes, and especially disease-causing microbes, present a rich case study in the applicability of Niche Construction Theory. Their often antagonistic relationship to humans and other organisms, rapid reproductive rate, and sensitivity to environmental change at both small and large scales render them both ecologically and socially consequential and highly visible. At the end of the nineteenth century, scientists and government administrators began to document these relationships at a faster pace than ever before, driven by imperial competition for scientific discovery, and the emergence of epidemics that threatened to undermine their authority. The British Empire was a major driving force in this knowledge creation, both as the connecting frame for a series of sites of bacteriological research and as a site of epidemic origin and circulation. It therefore serves as a useful point of reference through which to approach an interspecies history of disease.

Shaping Disease Ecologies: The British Empire as a Unit of Analysis

The British Empire, in the second half of the nineteenth century, exerted considerable global ecological influence. With more than ¼ of the world's population and landmass under its administrative control and many of the world's largest economies oriented towards its systems of trade, the British Empire transformed landscapes at multiple scales. Through far-reaching economic influence and highly differentiated but entrenched systems of governance and political power, the British Empire engineered novel ecosystems, changing the distribution of organisms within those systems and transforming the intimate quotidian relationships within them. The formal and informal processes of Empire were thus a potent form niche construction, exerted on different ecologies; and the resulting changes to landscapes as disparate as the forests of India and the pastures of Australia were unique to the organisms of those ecosystems. While not typically framed in the terms of niche construction, historians have long documented the cultural, economic, and environmental processes that united the British Empire as a (heterogeneous) entity and interrogated their consequences. A history of microbial life and urban ecology in British imperial cities thus inevitably intersects with histories of British imperial networks of expertise, urban planning, histories of medicine and public health, and histories of colonialism, and draws extensively on them to establish scales of analysis and characterize the niche-constructing processes at work. Three major themes within these historical disciplines provide a frame for thinking through the Empire as force for niche construction: the integration of material environments into an imperial economy and the consequences thereof; administrative technologies of empire; and structures of imperial knowledge formation and circulation.

Lotte Hughes and William Beinart argue that the use of the British Empire as a unit of study is made logical through the lens of power – that British power, markets, trade and shipping "knitted" the empire together, with common institutions that provided a framework for the exercise of colonial power.[36] The second half of the nineteenth century was a time of unprecedented expansion of these systems of power, with novel technologies, accelerating production, imperial rivalry, and growing financial and speculative networks fueling the development of colonial infrastructures – and no sites better exemplified this exercise of power than imperial cities.[37] Port cities especially, as historians like James Belich have noted, were central nodes in the imperial network, facilitating the movement of both people and goods. Port cities bloomed at the end of the nineteenth century – or exploded, to use a perhaps more accurately violent metaphor for the trajectory of these spaces – facilitated by regional economic specialization driven by demand for raw materials and the outsourcing of food production in Britain; the establishment of infrastructure through imperial investment networks; and subsequent immigration of peoples, from the local or imperial hinterlands, to reap opportunities for work in expanding industries and institutions.[38]

While volatile in their growth, colonial cities were far from haphazard spaces. Each reflected complex and dynamic relationships that emerged from the clash of power structures, economic systems, and cultures. As economic historians have argued, the integration of

[36] William Beinart and Lotte Hughes *Environment and Empire*, (Oxford: Oxford University Press, 2007), 3-4.
[37] James Belich, *Replenishing the Earth: The Settler Revolution and the Rise of the Anglo World*, 1781-1939, (Oxford: Oxford University Press, 2011); Beinart and Hughes, *Environment and Empire*; PJ Cain and AG Hopkins, *British Imperialism 1688-2015*, 3rd edition, (London: Routledge Press, 2016).
[38] Belich, *Replenishing the Earth,* 175-197.

peripheries into British economic structures dramatically influenced where colonial spaces grew, with major cities often emerging around extractive, raw-material industries that supported the British mainland or other colonial trade networks.[39] Kenneth Pomeranz argues that the use of colonies as "ghost acres" led to dependency-based import-export relationships with the metropole.[40] Cain and Hopkins argue that this relationship was facilitated by shifts in economic relationality through the advancement of "gentlemanly capitalism."[41] They explore the ways in which systems of banking and credit played a significant role in British control and management of Empire, arguing that while the story of Empire does have much to do with extraction, settlement, and civilization, these ends were often directed and shaped by the financial pressure of capitalism in the metropole. These economic pressures, enacted on an imperial scale, changed local landscapes significantly; grasslands were transformed into pastures, forests into plantations, and resident peoples driven out of the ecosystem by agreement or force.[42]

While the resulting urban ecologies were unique, the cultural and administrative structures of the British Empire exerted a diffuse but distinct set of organizational and infrastructural pressures on these vastly different terrains. While Cain and Hopkins consider the influence of this system for the development of vast regions of Australia, India, Canada, and Africa, this process is perhaps best exemplified at the level of the city, where investment from

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[39] Kenneth Pomeranz, *The Great Divergence: China, Europe, and the Making of the Modern World Economy,* (Princeton: Princeton University Press, 2000); Belich, *Replenishing the Earth*; Cain and Hopkins, *British Imperialism*; Robert C. Allen, *The British Industrial Revolution in Global Perspective,* (Cambridge: Cambridge University Press, 2009); Sven Beckert, *Empire of Cotton: A Global History,* (New York: Penguin Random House, 2014).

[40] Kenneth Pomeranz, *The Great Divergence: China, Europe, and the Making of the Modern World Economy,* (Princeton: Princeton University Press, 2000), 12-27.

[41] Cain and Hopkins, 3-5.

[42] Mahesh Rangarajan, *Fencing in the Forest: Conservation and Ecological Change in India's Central Provinces 1860-1914*, (Oxford: Oxford University Press, 1996); "Sheep, Pastures, and Demography in Australia," in *Environment and Empire,* 93-110; Tim Flannery, *The Future Eaters: An Ecological History of the Australian Lands and People*, (London: Secker & Warburg, 1996); Tom Griffiths and Libby Robbin, *Ecology and Empire: Environmental History of Settler Societies* (Edinburgh: Edinburgh University Press, 1997).

the metropole resulted in "explosive colonization" of major urban centers.[43] James Belich, for example, examines the implications of this economic and ecological shift for key imperial port cities, arguing that financial and resource-based dependency in the Settler Empire led to a continuous cycle of boom, bust, and export rescue that characterized many of the central cities of settler colonies.[44] These patterns were a direct result of financial investment by British institutions in combination with environmental factors, "which compressed time and supercharged growth, allowing megacities like Chicago or Melbourne to sprout in a single lifetime."[45] While Belich's work theorizes that these economic patterns extended only as far as the settler colonies, the broad structures of investment, boom-bust cycles, and mass immigration characterized port cities across imperial contexts – and, I argue, created unique opportunities for the emergence of infectious disease in different imperial ecosystems.

British imperialism also derived power and coherence the administrative structures that organized these economic and extractive relationships. Historian Bernard Cohn explores the importance of language and categorization in constructing imperial spaces, as administrators wrote detailed analyses of colonial regions for the purpose of categorizing, surveying, and describing; quantification; and the transformation of objects into anthropological artifacts.[46] The imperial city, as a hub of government organization and the center of vast, dendritic power structures, served an important purpose in the process of integration – and often its shape was

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[43] Cain and Hopkins, 205-240, Belich, 174.

[44] Belich's argument draws inspiration from the methodology and theory of *Nature's Metropolis: Chicago and the Great West*, highlighting the importance of factors like boosterism, railway construction, and financial speculation in allowing urban spaces to boom from muddy, backwater settlements to large and powerful cities in a matter of decades. Chris Otter, meanwhile, argues that systems of economic expansion and resource dependency that defined these relationships not only changed British health and dietary patterns, but also fostered the growth of large agro-food systems that shifted global ecology and bodily compositions. See Chris Otter, *Diet for a Large Planet: Industrial Britain, Food Systems, and World Ecology*, (Chicago: University of Chicago Press, 2020).

[45] Belich, 9.

[46] Bernard S. Cohn, *Colonialism and its Forms of Knowledge: The British in India,* (Princeton: Princeton University Press, 1996).

profoundly influenced by this purpose.[47] While the hierarchy of these governances structures differed between types of colonies – settler colonies often managed their own administrative and bureaucratic affairs, with intervention from the metropole only in the event of crisis or dispute, while paternal colonies included elaborate hierarchies of local, regional, and imperial governance forms often overseen by administrators assigned from the metropole – the information regime was standardized through connection with a central organizational entity.[48] When epidemics emerged – especially urban epidemics, where administrators were most dense and systems of surveillance strongest – they were often counted and conveyed in uniform ways.[49] This is not to suggest that all colonial institutions were the same, but rather that the empire represented an "interlocking set of institutions, practices, and norms that approach populations and peoples with a surveillant gaze."[50] It is through this similarity in surveillance structures that cross-colonial comparison is facilitated.

The empire served not only as a unit of information gathering, but also of knowledge production. The communication of imperial scientists and the development of scientific networks between metropole and colony, as well as between colonies, has been the subject of focus of

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[47] Tristram Hunt, *Cities of Empire: The British Colonies and the Creation of the Urban World*, (London: Metropolitan Books, 2014); Robert Home, *Of Planting and Planning: The Making of British Colonial Cities*, (London: Routledge Press, 2013); Anthony D. King, *Colonial Urban Development: Culture, Social Power, and Environment* (London: Routledge Press, 1976).

[48] The uniformity of information gathering was largely an artifact of what Brewer calls the rise of the fiscal-military state, an expansion of the bureaucracy of governance in Britain that facilitated military expansion at the end of the 18th century. John Brewer, *Sinews of Power: War Money and the English State: 1688-1783,* (Cambridge: Harvard University Press, 1990).

[49] James C. Scott, *Seeing like a State: How Certain Schemes to Improve the Human Condition Have Failed*, (New Haven: Yale University Press, 1999); *Making Surveillance States: Transnational History*, Robert Heynen and Emily van der Muellen, eds., (Toronto: University of Toronto Press, 2019); Michel Foucault, *The Birth of Biopolitics: Lectures at the College of France 1978-1979,* (New York: MacMillan Press, 2010); Ian Hacking, *The Taming of Chance,* (Cambridge: Cambridge University Press, 1990).

[50] *Making Surveillance States*, 24. The varied roles that disease and sanitary structures played in these surveillance systems has been explored at length in histories of imperial medicine, for example, Alison Bashford, Imperial Hygiene: A Critical History of Colonialism, Nationalism, and Public Health, (London: Palgrave Macmillan, 2004); Warwick Anderson, *Colonial Pathologies: American Tropical Medicine, Race, and Hygiene in the Philippines*, (Durham: Duke University Press, 2006); and Randall Packard, White Plague, Black Labor: Tuberculosis and the Political Economy of Health and Labor in South Africa, (Berkeley: University of California Press, 1989).

intellectual historians and historians of Empire.[51] In concert with scholars of infrastructure and technology who have traced the ways that states garnered political power through technological and environmental expertise formed in the metropole and in the field, scholars of Empire have emphasized how knowledge produced in colonial spaces shaped the administration and engineering of empire.[52] Where it concerns environment, this literature has focused overwhelmingly in rural areas, examining agricultural field stations, forests, and other resource-rich areas as sites of information production and transmission across colonies, as well as the sites of ecological and economic consequences of information spread.[53] Helen Tilley's work on Africa as a "living laboratory," for example, demonstrates the importance of local ecology to African disease management and tropical disease knowledge, arguing that that the integration of bioscientific and African ecologies resulted in increasingly ecological theories of disease transmission, highlighting the mutual constitution of knowledge and landscape in imperial settings.[54] Gregory Barton, meanwhile, demonstrates how forest management practices first employed in India became the foundation for forest management practices across the British empire in the nineteenth century.[55] Together, these works demonstrate how imperial networks of knowledge allowed interactions between person and ecosystem in one part of the world to have

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[51] Richard Drayton, *Nature's Government: Science, Imperial Britain, and 'Improvement' of the World*, (New Haven: Yale University Press, 2000); Londa Schiebringer and Claudia Swan, *Colonial Botany: Science, Commerce, and Politics in the Early Modern World,* (Philadelphia: University of Pennsylvania Press, 2005); Daniel Headrick, *Tools of Empire: Technology and European Imperialism in the Nineteenth Century*, (Oxford: Oxford University Press, 1981); Peder Anker, *Imperial Ecology: Environmental Order in the British Empire, 1895-1945*, (Cambridge: Harvard University Press, 2002); CA Bayly, *Empire and Information: Intelligence Gathering and Social Communications in India, 1780-1870*, (Cambridge: Cambridge University Press, 1996); Richard Grove, *Green Imperialism*; Helen Tilley, *Africa as a Living Laboratory*.
[52] Timothy Mitchell, *Rule of Experts: Egypt, Techno-Politics, Modernity,* (Berkeley: University of California press, 2002), 90.
[53] Drayton, *Nature's Government*; Schiebringer and Swan, *Colonial Botany*; Anker, *Imperial Ecology*; Grove, *Green Imperialism*; Fredrik Albritton Jonsson, *Enlightenment's Frontier: The Scottish Enlightenment and the Origins of Environmentalism,* (Yale: Yale University Press, 2013).
[54] Tilley, *Africa as a Living Laboratory*, 169-216.
[55] Gregory Barton, *Empire Forestry and the Origins of Environmentalism*, (Cambridge: Cambridge University Press, 2002).

lasting consequences on human-environmental interaction on other continents – a kind of potent cultural niche construction.

While environmental historians of empire have explicitly drawn the link between knowledge formation and imperial practice in rural spaces, studies on urban areas as sites of information production and transmission have largely occurred in the context of public health and medical histories. Within these frames, sanitary ideologies migrated around the Empire in medical journals and through the work of colonial administrators (often trained in up-to-date sanitary practices through imperial colleges) in attempts to organize, control, and count colonial subjects.[56] Urban spaces, as dense interfaces between colonial officials and residents, and sites of epidemics that challenged colonial authority, became testing grounds for sanitary ideology – but also, equally importantly, sites of knowledge production. As Christos Lynteris, Pratik Chakrabarti, Jacob Steere-Williams, and David Arnold demonstrate, the dense colonial institutions and networks that sprung up around major cities like Bombay were key in the development of theories of disease transmission and management that had profound consequences across imperial spaces, notably in practices like vaccination, disinfection, and rat eradication.[57] In this project, I plan to unite environmental histories of empire with public health histories of empire, by arguing that these systems of surveillance and the circulation of sanitary

[56] Jacob Steere-Williams, "State Surveillance and the Labor of Disinfection", in *Making Surveillance States*, 35-80; *Public Health in the British Empire: Intermediaries, Subordinates, and the Practice of Public Health, 1850-1960*, Ryan Johnson and Amna Khalid eds., (London: Routledge Press, 2012); Mark Harrison, *Public Health in British India: Anglo-Indian Preventive Medicine 1859-1914*, (Cambridge: Cambridge University Press, 1994).
[57] *Plague and the City,* Lukas Engelmann, John Henderson, and Christos Lynteris, eds., (London: Routledge Press, 2019); Christos Lynteris, The Global War Against the Rat and the Epistemic Emergence of Zoonosis, University of St. Andrews, 2020, accessed May 15, 2021, https://wwrat.wp.st-andrews.ac.uk/; Pratik Chakrabarti*, Bacteriology in British India: Laboratory Medicine in the Tropics*, (Rochester: University of Rochester Press, 2012); David Arnold, *Colonizing the Body: State Medicine and Epidemic Disease in Nineteenth-Century India*, (Berkely: University of California Press, 1993).

ideas within urban colonial spaces had profound ecological consequences for human and non-human residents.

The three cases analyzed in this project – typhoid in Belfast, Northern Ireland; plague in Bombay, India; and tuberculosis in Melbourne, Australia – were chosen to represent the heterogeneous colonial structures and diverse ecologies encompassed by the British Empire. While enveloped in the same imperial economic structures and systems of information production and transfer, the political, social, and environmental characteristics of these three colonies differed greatly. By comparing an epidemic "crowd disease" in a temperate settler colony (Melbourne), a spillover epidemic in a semi-tropical paternalist colony (Bombay), and a common food and water borne-epidemic in a quasi-imperial space on the British mainland (Belfast), I hope to demonstrate the utility of Niche Construction Theory and multispecies thinking for exploring the multiple scales of pressure – from imperial to local – that influence events like disease emergence, and fostering new connections between seemingly disparate events.

New Uses for Social History: A Brief Note on Methods

The many thousands of pages that colonial regimes generated through these surveillance structure form the backbone of this project, providing access to important aspects of material ecological changes to urban landscapes and to the nonhuman lives that defined cities. In this way, the project builds on traditional methods and sources prevalent in social history and urban history. Like the works of Asa Briggs, EP Thompson, and more recently Martin Melosi and Richard Evans, this project relies heavily on quantitative sources to access trends in the lives of ordinary people, including sickness, health, occupation, and living patterns.[58] It also follows recent trends in animal studies that expand on these methods, building fuller and more diverse understandings of urban spaces by taking into account animals and other nonhumans rendered visible by these records. As historian Peter Atkins notes, these relationships are key in presenting the city as a socio-ecological system, with its relationships mediated by technological infrastructures. Quotidian human-animal interaction, many historians note, is central to the definition of and character of urbanism in much of the world – and the trajectory of western urbanism, wherein animals and human-animal interactions are marginalized, was in no way inevitable.[59] Ecologies of urbanism in India, for example, include cohabitation (sometimes violent, sometimes symbiotic) between humans, Rhesus Macaque monkeys, street dogs, and cattle, among others.[60] The goal of this project is to offer a new perspective on these methods by incorporating a more dynamic and interdisciplinary analytic framework that not only accounts for the social patterns of humans and the incorporation of animal and nonhuman life in the city

[58] Asa Briggs, *Victorian Cities*, (Berkeley: University of California Press, 1993); EP Thompson, *The Making of the English Working Classes*, (New York: Penguin Random House, 1966); Richard Evans, *Death in Hamburg*; Martin Melosi, *Garbage in the Cities: Refuse, Reform, and the Environment*, (Pittsburgh: University of Pittsburgh Press, 2004); Martin Melosi, *The Sanitary City: Environmental Services in Urban America from Colonial Times to the Present*: *Urban Infrastructure in America from Colonial Times to the Present*, (Baltimore: Johns Hopkins University Press, 2000);Martin Melosi, "The Place of the City in Environmental History," *Environmental History* 17(1993): 1-23.

[59] AJ Hovorka, "Animal geographies I: Globalizing and decolonizing," *Progress in Human Geography*, 41(2017):382-394. doi:10.1177/0309132516646291. Peter Atkins, *Animal Cities: Beastly Urban Histories*, (London: Routledge Press, 2012); Andrew Robichaud, *Animal City: The Domestication of America*, (Cambridge: Harvard University Press, 2019); Philip Howell and Hilda Kean, "Writing in Animals in History," in *The Routledge Companion to Animal-Human History*, (London: Routledge Press, 2018), 3-27. On the historical contingency of human non-human relationships in the Anthropocene, see Dipesh Chakrabarty, *The Climate of History in a Planetary Age*, (Chicago: University of Chicago Press, 2021).

[60] Maan Barua and Anindya Sinha, "Animating the Urban: An Ethological and Geographical Conversation," *Social and Cultural Geography* 20(2019): 1160-1180, https://doi.org/10.1080/14649365.2017.1409908; Yamini Naryanan, "Street Dogs at the Intersection of Colonialism and Informality: 'Subaltern Animism' as a Posthuman Critique of Indian Cities," *Environment and Planning D: Society and Space* 35(2017): 475-494, doi:10.1177/0263775816672860; Adwait Deshpande, Shreejata Gupta, and Anindya Sinha, "Intentional Communication between Wild Bonnet Macaques and Humans," *Scientific Reports* 8(2018): 5147, https://doi.org/10.1038/s41598-018-22928-z; Chakrabarty, The Climate of History in a Planetary Age, 126-127.

but prioritizes their interactions and the consequences thereof – a move which requires engagement with novel methods emerging from environmental history, public health, and ecology.

Chapter Outline

The first chapter of the dissertation introduces niche construction theory, a concept adapted from ecology and evolutionary biology, as the major methodological intervention of the project. It starts from the premise that human niche construction, which can be briefly defined as human participation in activities that have modified the environments in which they and other organisms live, has altered the survival chances and interactions of other organisms at multiple scales, and that in turn, the niche constructing activities of other organisms have affected the trajectory of human niche construction. I argue that the theory's focus on dynamic material change to ecosystems driven by the life-sustaining processes of organisms, both biological and cultural, at local and global scales, provides an ideal framework for the re-integration of humans into their ecosystems while navigating controversies related to agency dominant in nonhuman studies. Drawing on major works of nonhuman theory in science and technology studies and in history, I examine how scholars have increasingly relied on engagement with scale, practices of tracing intimate relationships between humans and nonhumans, and the construction of lifeways of organisms (in other words, telling stories that engage the daily activities of nonhuman organisms) to tell multispecies stories, but continue to grapple with the problem of developing a theory which allows for the actions of organisms not traditionally included in history. This chapter demonstrates how Niche Construction Theory, when combined with these frameworks, can be adapted to fit this theoretical and simultaneously build bridges between the humanities and the sciences.

The subsequent chapters present cases in this framework. Source material drawn from local and imperial records offices interrogates the nuances of each city's struggle with disease. In the first case study, I explore an epidemic of tuberculosis in Melbourne, Australia. Drawing on the collected publications of Dr. William Thomson, vital statistics, and records of the Central and neighborhood boards of health for the city, I examine the role of immigration patterns, class, and urban structure in propagating insanitary conditions conducive to tuberculosis activation. The chapter uses a combination of geographic and documentary analysis to consider the risk factors for tuberculosis mortality at the scale of the neighborhood.

The second case study is an exploration of the ecology of the third plague pandemic in Bombay. Drawing on Gregg Mitman's framework, "ecologies of injustice," along with niche construction theory, these two chapters explore the classed and racial context of plague incidence and mortality. In the third chapter I examine how human and rat demography changed as a result of the plague epidemic, conducting geospatial, epidemiological, and documentary analysis to explore how sanitary infrastructure, architecture, occupation, and public health ideology and response influenced both human and rat ecology in the city, and subsequently, plague ecology. In the fourth chapter, I explore how the emergence of the rat-flea theory as the dominant epistemology in the early 20th century changed the position of humans, rats, fleas, and *Yersinia pestis* in the city and across India.

The third case study examines epidemic typhoid fever in Belfast, Northern Ireland. Chapter five uses socioecological frameworks to trace the roots of prolonged and elevated typhoid fever incidence in the city (the highest rates in the United Kingdom) and considers the role of bacteriology and social stigma in the decline of shellfish gathering and the shellfish industry. Like previous chapters, it relies on a combination of health statistics, geospatial analysis, and personal accounts to draw connections between the unique demographic and ecological composition of Belfast and its typhoid fever rate.

The final chapter draws these three cases into comparative perspective, exploring how the three differing administrative and urban structures responded to epidemic disease, and how these governments' tendency to embrace or neglect evolving disease etiologies depended to a great degree on local and imperial managerial ideologies. Public health practice developed around hygienism (Bombay), contagionism (Melbourne), and bacteriology (Belfast) carried unique implications for urban ecology and mortality. Together, these cases demonstrate the consequences of human environmental engineering practices in complex and locally specific ecological systems and consider how highly localized ecosystems inform multiscalar knowledge-making practices.

Chapter 1: Towards an Interspecies History of Disease

"Sometimes common entanglements emerge not from human plans but despite them. It is not even the undoing of plans, but rather the unaccounted for in their doing that offers possibilities for the elusive moments of living in common."[1]

Disease, like death, is a unifying experience for all biotic organisms – and an integral part of human existence. A common subject in nearly every subdiscipline of historical study, disease serves as a lens through which to examine some of the most pressing ideas about what it means to be human. It serves as an amplifier of social inequalities and ills; a cultural imaginary; a test of systems of entrenched power; a symbol of globalization; and as a product of environmental change. As members of a field that values its ability to weave stories dependent on context and specificity, time and place, historians have developed a plethora of theoretical frames to explore the many meanings of the "disease" across cultural, geographic, and temporal contexts, and to explore the many ways in which diseases shape our lives and our ecologies.

Our understandings of what it means to be human, and the role of ecology in human history, however, are changing; rising CO_2 levels, species collapse, and increasing environmental toxicity threaten the longevity of our own and billions of other species.[2] In the face of the Anthropocene, an increasingly large cohort of historians have called for a form of historical practice that incorporates a more dynamic materialism. These scholars have made it their business to expose the historical context of problematic divides – nature-culture, mind-body, human-nonhuman – and their role in what might now be considered overly limited

[1]Anna Lowenhaupt Tsing, *The Mushroom at the End of the World: On the possibility of Life in Capitalist Ruins,* (Princeton: Princeton University Press, 2015), 267.

[2] William Steffen, Katherine Richardson, Johan Rockström, Sarah E. Cornell, Ingo Fetzer, Elena M. Bennett, Reinette Biggs, Stephen R. Carpenter, Wim de Vries, Cynthia A. de Wit, Carl Folke, Dieter Gerten, Jens Heinke, Georgina M. Mace, Linn M. Persson, Veerabhadran Ramanathan, Belinda Reyers, Sverker Sörlin, "Planetary Boundaries: Guiding Human Development on a Changing Planet," *Science* 347(2015): 1259855. DOI: 10.1126/science.1259855.

interpretations of human history based in social constructivism and naturalism.[3] Despite this exuberant theorization, however, only a handful of historians have taken up the mantle to incorporate nonhuman dynamism their work.[4] As Tim LeCain notes, "at this early stage, many…self-professed new materialists still seem more comfortable engaging abstract philosophy and theory than the actual stuff of the material world."[5] The goal of this project is to engage this deficit, and to reintegrate human behaviors and events into a dynamic ecology through one of the most highly visible and heavily documented interspecies phenomena present in source material – epidemic disease.

Infectious disease is an often uncomfortable, sometimes deadly, reminder that the human body is not a closed system. It is a break in the dualism of nature and culture, of self and other. It is a moment where the outside world, the distinct "other" invades the "self." It does not just invade the self but *changes* it. The body never truly returns to an equilibrium after its interaction with a disease-causing organism; the interaction is forever recorded in the immune system's T-cells. While we know that this process occurs over and over again in the life cycle of a person – arguably, microbiome research suggests that we would not survive without it, and that perhaps some of these invaders become incorporated into the physical and imaginary "self" – these are microscopic and often historically untraceable interactions. It is difficult to know when one's gut microbiome has shifted in composition, though we sometimes do our best to engineer these

[3] Dipesh Chakrabarty, "The Climate of History: Four Theses," *Critical Inquiry* 35(2008): 197-222; Tim LeCain, *The Matter of History: How Things Create the Past*, (Cambridge: Cambridge University Press, 2017); Julia Adeney Thomas, "History and Biology in the Anthropocene: Problems of Scale, Problems of Value," *The American Historical Review* 119(2014): 1587-1607; Chris Otter, Nicholas Breyfogle, John L. Brooke, Mari K. Webel, Matthew Klingle, Andrew Price-Smith, Brett L. Walker, and Linda Nash, "Forum: Technology, Ecology, and Human Health Since 1850," *Environmental History* 20(2015): 710-804. Arguably, examining these divides has been a longstanding project of environmental history, beginning with Carolyn Merchant, *The Death of Nature: Women, Ecology, and the Scientific Revolution,* (San Francisco: Harper & Row, 1980) and William Cronon, "The Trouble With Wilderness: Or, Getting Back to the Wrong Nature," *Environmental History* 1(1996): 7-28.

[4] An encouraging example of this kind of work can be seen in Otter et al., "Forum: Technology, Ecology, and Human Health Since 1850," *Environmental History* 20 (2015): 710–804

[5] LeCain, *The Matter of History*, 82.

changes through probiotics and prebiotics. Disease-causing microbes, however, leave undeniable evidence of their presence within our bodies. They are an immunological "other," an invader in the landscape of the body, whose actions and processes are at odds with the continued functioning of the whole. The physiological changes that result from this interaction of the non-body microbe and the systems of the body are often of a scale large enough to be noticed, documented, and combatted – and therefore visible to the historian. Infectious disease represents an ideal point from which to incorporate microbial agency into historical study – and to reconnect the body to the ecosystem through interspecies entanglements.

To write a history that centers ecology and human-nonhuman interaction in historical disease events. I argue, we must draw on analytic frameworks that account for the life processes of all organisms and render visible the consequences of these life processes for organisms in shared ecosystems. Looking beyond the humanistic fields, which by nature center the human in analytic frameworks, and towards ecology, which prioritizes systems and networks of living organisms, provides tools for building these analytic frameworks. In this chapter, I propose that Niche Construction Theory, a prominent theory in ecology and evolutionary biology that prioritizes the ways that organisms interact with environments, provides an ideal frame for incorporating nonhumans more actively into environmental history. Niche Construction Theory, by centering the ways in which organisms "interact with environments, take energy and resources from environments, make micro and macrohabitat choices with respect to environments, construct artifacts, emit detritus and die in environments, and by doing all these things, modify at least some of the natural selection pressures present in their own, and in each other's, local environments," provides a broad and adaptable theoretical frame under which to unite existing methods of nonhuman storytelling with biology while bypassing some of the major controversies around agency that often hinder the application of more-than-human theories to historical practice.

In examining three very different disease contexts – tuberculosis in Melbourne, Australia; plague in Bombay, India; and typhoid fever in Belfast, Northern Ireland, this project asserts that disease agency is shaped by environment in nearly every context; and that the conditions that allow for the emergence of disease, both in epidemic and endemic form, are a result of the longer, slower process of people, animals, microbes, and environments "living in common" in a particular time and place. To access the nuances of these assemblages I will draw on Niche Construction Theory to lend dynamic complexity to ecosystems and agency to the diseases, animals, and peoples living within them.[6] In this chapter, therefore, I propose to do three things. First, I will argue for the utility of Niche Construction Theory (NCT) for incorporating interspecies complexity into historical studies of disease. Second, I will engage arguments for agency and materiality in historical inquiry and examine key developments in multispecies storytelling across disciplines to show how this work compliments and is enriched by NCT. Finally, I argue that combining these perspectives with NCT provides a useful framework for historical analysis of more-than-human relationships. In this exercise, I hope to contribute to a small but growing body of work that provides empirical grounding for new materialist theories, and do so by introducing ecosystem dynamism into our understanding of history.

Niche Construction Theory as Historical Method

Imagine a mature deciduous forest, teeming with life. While the term we use to describe the ecosystem – a "forest" – suggests a unified whole, it is, in reality, a complex array of living

[6] "Living in common," is a phrase I borrow from Tsing, *The Mushroom at the End of the World,* 267.

and nonliving processes, the summation of which we characterize as a "forest." Within this system, billions of organisms engage in activities that sustain their life processes. *Acer saccharum*, or the sugar maple tree, spreads its branches in complex patterns, leaves reaching for sunlight to convert into glucose. The nooks and crevasses created by these branches, especially as they rot, provide shelter to *Poecile atricapillus*, or the black-capped chickadee, who build their cup-shaped nest using moss, rabbit fur, and small branches instinctually and by learned behavior.[7] Under the shade of the maple tree, *Convallaria majalis,* or lily of the valley, blooms in spring, relying on adaptations to low levels of light to spread and grow. The sugar maple's shallow, fibrous roots anchor it to the ground, protecting against the pushes and pulls of wind, and drawing in water and nutrients. Sharing these first few inches of soil are the subterranean rhizomes of the lily of the valley. Beneath the surface, earthworms feed on the plant debris and soil, concentrating the nutrients from these materials and releasing it in castings into the soil – a form of waste that holds four times more phosphorous than surface soil. As they burrow, the tunnels that trail behind them allow the lily of the valley and sugar maple's roots to penetrate deeper into the soil, and loosen the soil, creating porous pockets in which water can be absorbed into the soil, and subsequently, into roots.[8] Occasionally, a human seeking remedy for heart failure, irregular heartbeat, a urinary tract infection, leprosy, or some other ailment cuts leaves and flowers from the lily of the valley, disrupting the flow of phytohormones to the shoots, activating the growth of new buds along the hormonal pathway. When years later, a family thousands of miles away wants hardwood flooring in their new house, the sugar maple tree is cut down and its valuable hardwood sold to a multinational furniture corporation. It transfers

[7] "Black-Capped Chickadee Overview, All About Birds, Cornell Lab of Ornithology," Overview, All About Birds, Cornell Lab of Ornithology. Accessed May 1, 2021. https://www.allaboutbirds.org/guide/Blackcapped_Chickadee/overview.

[8] "How Earthworms Can Help Your Soil," NSW Department of Primary Industries, January 1, 1970, https://www.dpi.nsw.gov.au/agriculture/soils/biology/earthworms.

nutrients via a dense network of latticed fungi beneath the soil surface to the oaks, birches, aspens, and maples nearby, increasing their chances of survival with its final chemical signals.[9] The lily of the valley, bared to the full intensity of the sun, continues to survive on the dense nutrients of the soil but produces fewer blooms, and the herbalists who came to harvest the plant for traditional medicine begin to look elsewhere.

Now imagine an urban neighborhood. The brick buildings are on average three stories tall, U-shaped, with a courtyard in the center. The soil on which they are built is primarily loam and sand, and residents plant *Tulipa gesneriana* that blossom in fantastic colors in the spring. The loamy soil is also prone to erosion, however, and with erosion comes sinkage. Gradually the buildings begin to tilt inward towards the courtyard, exposing gaps in the foundation. *Mus musculus*, the house mouse, squeezes through gaps as small as dimes, gathering debris and seeds from garden plants to line its nest, and 19-21 days later, this little gap provides a safe home for its deaf and blind offspring. As the offspring grow and begin to forage for food, they move into the interior of the apartments and out into the garden, feasting on tulip bulbs and causing damage to electrical wiring. The old knob-and-tube wiring, insulated with cloth, ignites, causing a house fire in one of the apartments that does structural damage to the hardwood floors, requiring replacement.

Through these (very) simplified examples, one begins to see how the activities of organisms within the ecosystem, driven by both internal and external forces, alter the lives of others and the abiotic environment. In short, we see Niche Construction Theory at work. In its simplest form, Niche Construction Theory asserts two things: first, that organisms significantly modify both abiotic and biotic environments through their metabolic, physiological, and

[9] Suzanne W Simard and Daniel M Durall, "Mycorrhizal Networks: A Review of their Extent, Function, and Importance," *Canadian Journal of Botany* 82(2004): 1140-1165.

behavioral activities, as well as their choices; and second, that these organism-mediated environmental modification influence selection pressures on a recipient organism.[10] In the first example, we see the life-sustaining activities of the sugar maple facilitate a habitat for birds and plants; the behavior of worms and composition of soil in turn nourish the sugar maple and the lily of the valley; meanwhile, both global and local cultural and economic processes like traditional medicine and supply-chains affect the success and survival chances of organisms within the ecosystem, both directly and indirectly. The growth of these organisms effects the health and social status of humans consuming and using them. One of the major strengths of NCT is its focus on the co-evolution of organisms and their environments – with "environments" being either widely or narrowly conceived. Its analytic framework promotes a dynamic view of ecosystems filled with organisms whose interaction with an environment and with each other are reciprocal in nature, "with selective environments shaping organisms, and organisms shaping selective environments, either relative to themselves or other organisms."[11] By centering changes to the shared environment, it is a theory that is fundamentally concerned with process and consequences at multiple scales and therefore, I argue, an ideal theoretical framework for historians to incorporate environments and nonhumans to stories of change over time.

The process of niche construction was first described by evolutionary biologist Richard Lewontin in 1983. Lewontin argued in a series of papers that organisms "do not passively adapt to conditions in their environment, but actively construct and modify environmental conditions that may influence other environmental sources of selection.[12] "Niche Construction Theory" as a

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[10] John Odling-Smee, Douglas H. Erwin, Eric P. Palkovacs, Marcus W. Feldman, Kevin N. Laland, "A Practical Guide to Niche Construction Theory for Ecologists," *The Quarterly Review of Biology* 88(2013): 3-28. DOI: 10.1086/669266.
[11] Odling-Smee et al., "A Practical Guide," 7.
[12] Kevin Laland, Blake Matthews, and Marcus W. Feldman, "An Introduction to Niche Construction Theory," *Evolutionary Ecology* 30(2016): 191-202.

defined approach rose to prominence in the field of evolutionary biology ten years later with the publication of F. John Odling-Smee, Kevin Laland, and Marcus Feldman's *Niche Construction Theory: The Neglected Process in Evolution.* In this text the authors point out the two major roles that organisms play in evolution – first, carrying genes and passing them on; and second, and more importantly for our purposes, they argue "organisms also interact with environments, take energy and resources from environments, make micro- and macrohabitat choices with respect to environments, construct artifacts, emit detritus and die in environments, and by doing all these things, modify at least some of the natural selection pressures present in their own and each other's local environments."[13]

Niche construction theory's main intervention into the scientific literature – and, I argue, its main potential contribution to historical study – lies in the second of these two categories. Niche construction theory it is a term not concerned with intent but with *process*. By focusing on the effects a particular organism exerts on its surrounding environment – and importantly, how changes in that environment in turn affect other organisms – NCT allows for a more nuanced understanding of agency measured by effect on other members of an ecosystem, a model of agency less focused on intention and more focused on action. As Kevin Laland points out, "Niche construction is a very general process, exhibited by *all* living organisms, and species do not require advanced intellect or sophisticated technology to change their world."[14] By framing the key interventions of biotic organisms in general, processual terms, Niche Construction Theory can connect forces as broadly defined as capitalism, and as narrowly defined as a single interspecies interaction.

[13] F. John Odling-Smee, Kevin Laland, and Marcus W. Feldman, *Niche Construction Theory: The Neglected Process in Evolution*, (Princeton: Princeton University Press, 2003), 1.
[14] Kevin N. Laland and Michael J. O'Brien, "Cultural Niche Construction: An Introduction," *Biological Theory* (2013): 4. DOI 10.1007/s13752-012-0026-6.

To understand the analytic boundaries of the concept, it is useful to examine the four major components that Odling-Smee, Laland, and Feldman argue comprise niche construction theory:[15]

1. Ecosystem Engineering

Niche Construction Theory builds off an earlier theoretical intervention by Jones et al. which proposes the idea of "organisms as ecosystem engineers" – that organisms "directly or indirectly modulate the availability of resources to other species, by causing physical state changes in biotic or abiotic materials. In doing so, they modify, maintain, and create habitats."[16] Particularly important in this theory is not only its assertion of the importance of life-sustaining activities of all living things, but also its explicit statement of relationality. Jones et al. propose that organisms achieve "control via a web of connectivity in ecosystems, an "engineering web…established by the interactions of diverse species of engineering organisms."[17] Through this web, organisms exert "control" over environment by controlling the flow of energy matter and information through ecosystems. Ecosystem engineering is thus a description of action predicated on the assumption that the actions of an individual organism have real consequences for those spatially, temporally, and relationally linked to them. Examining our two imaginary ecosystems, we can see major ecosystem engineering activities present in organisms like worms, who concentrate nutrients and create pathways for root systems in soil, and in humans, who build houses and gardens that are hospitable to tulips and mice.

[15] Odling-Smee et al., *Niche Construction Theory,* 1-23.
[16] Clive G. Jones, John H. Lawton, and Moshe Shachak, "Organisms as Ecosystem Engineers," *Oikos* 69(1994): 373-386.
[17] Odling-Smee et al., *Niche Construction Theory,* 6.

2. Modification of Selection Pressures

The next major requirement of NCT is that the change an organism exerts over an ecosystem must persist long enough to alter natural selection pressures. This tenet incorporates temporality and spatiality into the theory by allowing multiple generations of niche constructing activities to affect survival chances, effects which can radiate outward spatially, and even occur on a global scale depending on the particular action. For example, one might think of generations of cyanobacteria converting CO_2 into O_2, slowly increasing the oxygenation of the atmosphere, and thereby creating a global ecology that selects for organisms that metabolize oxygen. While sexual selection is perhaps the most well-known version of this phenomenon, its biological definition encompasses a wide variety of biological and behavioral processes. Odling-Smee et al. claim that an organism can be said to modify selection pressures if they "modify their environments, and if in addition they affect, and possibly in part control, some of the energy and matter flows in their ecosystems."[18] This modification of selection pressure only becomes an evolutionary force, however, when selection pressures persist over a long enough period of time to have an evolutionary effect. As humans are widely considered to be "niche constructors on a global scale," this criterion will often be met in historical study; the role of nonhuman actors in altering selection pressures should be considered carefully, however, taking into account the role of the organism within the ecosystem and its impact on the survival chances of other organisms. While nonhumans will perhaps exhibit a less traceable pressure on selection in historical context, it is also important to remember that they need not alter the selection pressures of humans directly to be engaging in niche construction – and for that niche construction to have an effect

[18] Odling-Smee et al., *Niche Construction Theory,* 8.

on humans.[19] Furthermore, these criterion need not be met by a single organism; if the niche constructing activities of a generation of organisms persist in the environment beyond their lifespan and modify the selective environments of succeeding generations, this similarly qualifies as evolutionary selective pressure. Generations of worms together may improve the quality of the soil, for example, making the soil nutrient-dense and hospitable to plants that are edible to other organisms, like deer, who then themselves become food and clothing for humans.

3. *Ecological Inheritance*

Describing the material changes that follow the modification of selection pressures, "Ecological inheritance," occurs when the physical consequences of one generation's niche construction are not completely erased in the environments of its descendants but are instead bequeathed, either wholly or in part, from one generation to the next, in the form of legacies of modified natural selection pressures. This is similar to a process often described by historians when we talk about "formal" and "vernacular" landscapes, or when we think about local landscapes reacting differently to imperial land use ideologies. The Anthropocene is our ecological inheritance, and that of our children. Returning to the example of the mouse-infested apartment and the deciduous forest, the nests built by both the mouse and the black-capped chickadee also represent an ecological inheritance, increasing the probability of survival of offspring through environmental modification. It is important to note here that this process

[19] Nicole L. Boivin, Melinda A. Zeder, Dorian Q. Fuller, Alison Crowther, Greger Larson, Jon M. Erlandson, Tim Denham, and Michael D. Petraglia. "Ecological consequences of human niche construction: Examining long-term anthropogenic shaping of global species distributions," *PNAS* 113(21016):6388-6396. While evolutionary consequence is an important aspect of NCT, what constitutes a time period long enough to convey an "evolutionary effect" is difficult to measure. However, the criteria can be met by the collective activities by a group of organisms exerting similar pressures over multiple generations – for example, Odling-Smee et al. note, if generations of spiders build webs in the same place, they may evolve mechanisms over generations that reduce the likelihood of becoming prey to animals present in that environment.

allows for more indirect effects to remain agentive, as "any organism's selective environment is potentially modifiable by any other organism that happens to be a neighbor or that shares, or that has previously shared, some common physical aspect of a mutual environment *or that is capable of exerting an indirect influence by affecting the flow of energy or materials through that environment*." Particularly powerful is the idea that "Ecological and genetic ancestors are not necessarily identical."[20] This definition opens conceptual space for abstract but highly consequential systems, like capitalism, imperialism, and globalization to affect the composition and evolutionary trajectory of an ecosystem without occupying the same material space as organisms within that ecosystem.

4. *Adaptation and natural selection pressures act as feedback systems*.

Finally, if we take seriously the ability of organisms to modify their selection pressures, Odling-Smee et al. claim, then adaptation and natural selection must be considered as two mutually reinforcing, reciprocal processes. Rather than the "classical" definition of natural selection that defines adaptation in reaction to natural selection, which is itself driven by a black-boxed process, niche construction theory allows for adaptation and natural selection to be in constant motion, with the actions of individual members of an ecosystem at the center.[21] As Lewontin notes and Odling-Smee et al. emphasize, "Organisms do not adapt to their environments, they construct them out of bits and pieces of the external world."[22] Together, the push and pull of adaptation and selection form feedback loops that drive change in ecosystems and organisms.

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[20] Odling-Smee et al., *Niche Construction Theory,* 15-16.
[21] Odling-Smee et al., *Niche Constructio Theory,* 16-20.
[22] Odling Smee-et al., *Niche Construction Theory,* 17 and RC Lewontin, "Gene, organism and environment," in *Evolution from molecules to men,* Bendall, ed, *(*Cambridge: Cambridge University Press, 1983).

One of the key examples in ecology used to illustrate these four components of Niche Construction Theory is the process of a beaver building a dam. By meticulously fashioning sticks, logs, and detritus into a home, the beaver alters the riparian ecosystem – this is ecosystem engineering. The beaver's dam forces the river to stagnate, and thus affects nutrient cycling, decomposition dynamics, and flow rate. Oxygen levels decrease; pH neutralizes. By cutting down trees, the beaver might increase the amount of sediment runoff and the amount sunlight reaching the water. These effects ripple out to the other organisms that call the river their home; fish, microbes, and aquatic plants that rely on the flow of the river suddenly find themselves occupying ponds and floodplains – a newly hostile ecosystem changes pressures for their survival. Some organisms, like herons, are able to change their feeding patterns in response to the changing ecology – an example of adaptation. For others, beavers' dams create nurturing ecosystems; many amphibians, small mammals, and species of birds and their offspring thrive in the wake of the beaver's niche constructing activities – an ecological inheritance granted from the beavers to these organisms.

A critical component of niche construction theory is the concept of the ecological niche, which is defined by the organism's ecological requirements and its influence on – and interactions with – the biotic and abiotic environment.[23] Also important to niche construction, however, is the "social niche," which comprises an organism's behavioral associations in a network of interacting social individuals.[24] These two processes often occur together – and the combination thereof is what makes humans particularly powerful ecological agents. For this reason, Laland developed the concept of "cultural niche construction," which argues that human

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[23]Amanda Ellwanger and Joanna Lambert, "Investigating Niche Construction in Dynamic Human-Animal Landscapes: Bridging Ecological and Evolutionary Timescales," *International Journal of Primatology* 39(2018): 799-800. DOI: 10.1007/s10764-018-0033-y.

[24] Ellwanger and Lambert, 800.

ecological inheritance can be determined by either genetic or culturally-ingrained behaviors, and the effects of this ecological inheritance could similarly result in cultural or genetic changes with major ecological consequences.[25] Examination of human niche-constructing activities has been particularly robust in the ecological and social sciences, and in environmental history; arguably, the subfield of environmental history has (somewhat unknowingly) dedicated itself to documenting the specificities of human cultural niche constructing activities. The classic example of human niche construction is dairy farming, which has led to the intense modification of cattle genetics (beginning with their domestication) and similarly allowed for the persistence of the *LCT* gene in humans, which allows for lactase production. However, we have also seen how cultural ideas about milk consumption affect genetics, as individuals from cultures in which milk consumption is less prevalent tend to produce insufficient lactase for milk consumption (an example of epigenetic change, in which environmental pressure alters the expression of genes).[26]

As the span of these examples suggests, Niche Construction Theory is a dynamic theory that incorporates the actions of organisms within an ecosystem into survivability chances for other organisms. The most exciting and novel (at least to the evolutionary sciences) aspect of this framework is its acknowledgement of the importance of organismal life processes and subsequent feedback mechanisms in evolution. For social scientists it provides an enticing framework for a "vibrant" world of dynamic motion that includes both biotic and abiotic components as important in shaping organisms' survivability. It places the emphasis on change and feedback – whether from physiological action, cultural action, reaction, or otherwise – as the primary sites of motion in the ecosystem. In other words, NCT allows for "the full set of

[25] Kevin N. Laland and Michael J. O'Brien, "Cultural Niche Construction: An Introduction," *Biological Theory* (2013): 3. DOI 10.1007/s13752-012-0026-6.

[26] Pascale Gerbault, Anke Liebert, Yuval Itan, Adam Powell, Mathias Currat, Joachim Burger, Dallas M. Swallow, and Mark G. Thomas, "Evolution of Lactase Persistence: An Example of Human Niche Construction," *Philos Trans R Soc Lond B Biol Sci* 366(2011): 863-877.

interactions that occur between biotic and abiotic components in ecosystem and…diverse forms of feedback that contribute to coevolutionary scenarios and ecosystem dynamics."[27]

While Niche Construction Theory is a widely applicable and potentially valuable framework for describing and tracing large-scale ecosystem change and interspecies interactions, the theory is also subject to a number of limitations. Niche construction as a process, its major proponents argue, is valuable because of its wide boundaries, and its ability to incorporate indirect forms of evolutionary and ecological feedback and indirect forms of connection within an ecosystem. However, major debates continue in evolutionary theory as to whether the processes enveloped in niche construction can be considered as "evolutionary." From the perspective of the theory's critics, niche construction is an important driver of environmental change that can itself affect evolution; but to call it an evolutionary process itself extends the boundaries of evolution beyond practical applicability.[28] To environmental scholars working in the post-industrial period, this particular limitation of the theory is less salient, as evidence suggests that humans have altered the survival chances of all other organisms on earth through the alteration of global biogeochemical processes, by either conservative or liberal interpretations of evolutionary theory. Neo-materialist and post-humanist scholars, meanwhile, have worked to complicate the boundaries between organisms within a shared system by documenting human impact on the evolution of nonhumans and pointing to moments when human evolution has been dictated by nonhumans (microbiome theory is a favorite example among scholars of this perspective). Give these trends, an extended definition of what constitutes an evolutionary pressure (or evolution itself) seems a welcome conceptual tool.

[27] Odling-Smee et al, *Niche Construction Theory*, 26.

[28] TC Scott-Phillips, KN Laland, DM Shuker, TE Dickins, & SA West, "The Niche Construction Perspective: a Critical Appraisal," *Evolution; international journal of organic evolution*, *68*(2014): 1231–1243. https://doi.org/10.1111/evo.12332

The theory's focus on evolutionary effects of these niche constructing activities poses other risks to historians, however, as the theory can lead to careless analysis that focuses on genetics and its relationship to environment. If Niche Construction Theory is employed without recognition of the theory's focus on dynamic change and fluidity in ecological systems, and without careful considerations of the boundaries of biological processes, the scholar risks biologizing human behavioral ecology and reducing individual action to evolutionary or survival tendencies. An example of how NCT can be applied problematically emerges from urban studies, in Greg Downey's work on Brazilian street children. While he points out the ways in which niche construction theory benefits urban analysis and uses it to provide a nuanced reading of cities as containing different niches based on economic stratification and social consequence, he comes close to biologizing the survival strategies of street children in Brazil.[29] Therefore, it is critical that historians remain cognizant of the long histories of biologized classism and racism that draw on Darwinian theory to support their logic, and ground questions framed through Niche Construction Theory in anti-racist practice.

While historians have something to gain from adopting Niche Construction Theory as a framework of analysis, I also argue that, given these limitations, they have much to add. Process itself is agentive, contextual, and concrete – if often unquantifiable. Thus, while biologists might use quantified and traditional evolutionary data to ground a processual argument – again, the persistence of the *LCT* gene for human lactase production as the evolutionary result of animal domestication is a good example of this– the lineage of that genetic event is fraught with choices, movements, and uneasy alliances. Here is the point of entry for social scientists and humanists. Historians have the opportunity to add cultural complexity to NCT – drawing the framework

[29] Greg Downey, "Being Human in Cities: Phenotypic Bias from Urban Niche Construction," *Current Anthropology* 57(2016): S52-S54, DOI: 10.1086/685710

away from tendencies to biologize human behavioral ecology and reduce individual action to evolutionary or survival tendencies by providing nuanced contextual explanations based on individual agency within cultural, political, and economic institutions. With the field's focus on context, temporality, and specificity, historians' contributions to NCT could emphasize socially and culturally constructed and highly context-specific activities, avoiding interpretations that neglect deeper causes and individual agency.

In this project, I propose to demonstrate the utility of Niche Construction Theory for history – and the utility of history for Niche Construction Theory – by examining a microcosm of human-nonhuman niche construction in three different, but connected, urban ecologies. Given that human ecological success "is evident at all landscape and ecological scales, from local to global, shaping both abiotic and biotic processes," I argue that the survival chances of these bacteria, as a population, changed the moment they became a part of the imperial urban ecosystem.[30] Drawing on the major theoretical claims of Niche Construction Theory to structure the scale and scope of analysis for each epidemic, I examine both human and nonhuman behaviors that alter their respective ecosystems, improving survival chances for particular bacteria based on their own niche constructing activities. I argue that, when used in conjunction with key methods in environmental history and theoretical approaches from nonhuman studies, Niche Construction Theory can act as a framework through which to access the consequences of multiple species' life-sustaining activities on historical environments, providing an avenue for enriching our understanding of historical epidemics while resolving many of the key limitations inherent in current approaches.

[30] Ellwanger and Lambert, 798.

Agency and Materiality in Historical Context

Informing much of this project's methodology is existing research in STS surrounding the question of nonhuman and multiple agencies. Over the last decade, historians and anthropologists have expanded the ontological space for nonhumans in humanistic studies by drawing increasingly on theoretical work from geographers and anthropologists that create imaginative space for human-nonhuman mutual influence. Meanwhile, recent studies in the sciences – especially in microbiome studies – have taught us that the human-nonhuman dichotomy is much stickier than otherwise imagined.[31] In particular, theories and descriptive categories like assemblages, entanglements and Actor-Network Theory have inspired creative interspecies (or intermaterial) research. Historians have engaged varying degrees with the "nonhuman turn." It is worth outlining the key interventions in humanistic thought to situate this work within a growing body of human-nonhuman research, and crucially, to show how critical arguments within Science and Technology Studies can be leveraged with NCT to promote dynamic interspecies thinking.

While conversations around human-nonhuman interaction have long had a place in social science, Bruno Latour opened avenues of analytic space to consider these relationships with Actor-Network Theory (ANT).[32] While his work is largely concerned with technologies and scientific networks in his spectrum of the nonhuman, Latour also acknowledges the space created by human-animal or even human-microbe assemblages, as demonstrated in his study of Pasteur.[33] It is through humans and nonhumans mediators – actors who "transform, distort, translate, and modify the meaning or the elements that they are supposed to carry" – Latour

[31] Thomas, "History and Biology in the Anthropocene: Problems of Scale, Problems of Value," 1587-1607.
[32] Bruno Latour, *Reassembling the Social: An Introduction to Actor-Network Theory,* (Oxford: Oxford University Press, 2005); Bruno Latour, *Pandora's Hope: Essays on the Reality of Science Studies*, (Cambridge: Harvard University Press, 1999).
[33] Bruno Latour, *The Pasteurization of France*, (Cambridge: Harvard University Press, 1988).

asserts, that meaning emerges. Thus these nonhumans become "full-fledged actors in our collective."[34]

Latour urges scholars to reject the overly-structured "social" frame and instead draw on the assemblages of our narratives to "trace more sturdy relations and discover more revealing patterns by finding a way to register the links between unstable and shifting frames of reference rather than by trying to keep one frame stable."[35] In short, by following the actors themselves and the frames of reference within which they operate, and by learning to move between these frames, historians reveal previously obscured relationalities with real explanatory power. Much like NCT, ANT is concerned with "relations" and "links," tracing the ways that humans and nonhumans shape each other across time and space. Latour insists that scholars "'follow the actors themselves,'… try to catch up with their often wild innovations in order to learn from them what the collective existence has become in their hands, which methods they have elaborated to make it fit together, which accounts could best define the new associations that they have been forced to establish."[36] This language echoes Odling-Smee and Laland's focus on adaptation and pressure, the constant pull of organisms and technologies on each other and their shifting position in shared environments – for Latour the emphemeral "collective," for Odling-Smee et al. the concrete "ecosystem," – and proposes tracing these relationships.

ANT has had wide-spanning effects on the field of animal and nonhuman studies, providing a theoretical basis and analytic lens through which to examine the ways in which humans and nonhumans shape each other amid wider social and cultural contexts. For example, Jane Bennett draws on the language of "agents" and "actants" proposed in Latour's *Pandora's*

[34] Latour, *Pandora's Hope,* 193.
[35] Latour, *Reassembling the Social*, 24.
[36] Latour, *Reassembling the Social*, 12.

Hope, and the earlier concept of "assemblages" developed by Deleuze and Guartari to propose the inclusion of both "matter" and "living things" in a broad agentive analytic. Bennett takes Latour's framework a step further, though, and asserts that material formations carry structural similarities to life, can act in ways "unpredictable" to humans, and often comprise and influence living things in ways that alter their trajectory, thus possessing a "vitality" or "lively powers."[37]

Vibrant Matter is largely written as support and extension to new materialism – incorporating dynamism in nonliving entities that Bennett claims new materialist movements often render static. Bennett gives three reasons why vibrant materialism might be a better analytic frame than environmentalism to conceive of and think with the planet. First, she argues, "materiality is a term that applies more evenly to humans and nonhumans," in contrast to the human/environment divide. Second, she argues, it allows for the emergent properties of biochemical and biochemical-social systems, interrupting the teleological organicism of some ecologists and the machine image of nature governing many of their opponents."[38] Third, "Vital materiality better captures an 'alien' quality of our own flesh, and in so doing reminds humans of the very *radical* character of the (fractious) kinship between the human and the nonhuman…In a world of vibrant matter, it is thus not enough to say that we are 'embodied.' We are, rather *an array of bodies*, many different kinds of them in a nested set of microbiomes."[39] Vibrant Materiality, as Bennett conceives of it, dispenses with distinctions between living and nonliving all together, focusing instead on the consequences of collective interaction.

Tim LeCain incorporates Bennett's "vibrant materiality" into a larger trend he identifies in historical study called "neo-materialism," which is comprised of four basic concepts: first,

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[37] Jane Bennett, *Vibrant Matter: A Political Ecology of Things*, (Durham: Duke University Press, 2010), vi.
[38] Bennett, 112.
[39] Bennett, 113.

“the rejection of any essential distinction between the natural and the anthropogenic”; second, Bennett’s concept of thing-power, “which stresses the creative dynamism of both biotic and abiotic matter”; third, eliminating “conventional distinctions between the material and ideal to emphasize how things help to create humans in all their dimensions, both biological and cultural”; and finally, he argues that “historians and other humanists should therefore develop a much less anthropocentric approach to understanding both their human and nonhuman subjects.”[40] Drawing on examples in the evolutionary history of Longhorn cattle, environmental and cultural drivers of silk production, and the material characteristics of copper, LeCain emphasizes that “insights from across the spectrum of both the sciences and the humanities are telling us that the human body, mind, and culture are even more deeply embedded in our biological and material environments that we had previously imagined,” and that that “this material environment is the very stuff out of which the changing and evolving amalgam we call history emerges.”[41]

The frames of analysis proposed by Latour, Bennett, and LeCain, through engagement with questions of agency and their attempt to diminish the distinction between living and nonliving nonhumans in their work, have invited controversy that has largely drawn attention away from method and into epistemology. Many theorists have launched critiques of post-humanist frameworks through the question of agency, which has itself become something of a metaphor for intentionality.[42] Alf Hornborg demonstrates this ideology in his own analysis of agency, in which he argues for the distinction between “agents” who act and “artifacts,” which have consequences. In opposition to the “distributed” agency he attributes to posthumanism,

[40] LeCain, *The Matter of History*, 20.
[41] LeCain, *The Matter of History*, 7.
[42] A key example of this argument can be found in Langdon Winner, "Upon Opening the Black Box and Finding It Empty : Social Constructivism and the Philosophy of Technology Science, Technology, & Human Values," *Science, Technology, and Human Values* 18 (1993): 362-378.

Hornborg argues that scholars should resist collapsing the distinction between "subject" and "object." He argues that the capacity to act is propelled by purpose, which forms the basis for agency, and that "All living organisms have purposes inscribed in their composition, whether the amoeba's absorption of nutrients from its surroundings, the tree's extension of branches into the sunlight and roots into the soil, or a human preparing and ingesting a meal."[43] Nonliving "artifacts," on the other hand, do not have autonomous agency, but rather become active participants in agentive worlds by acting as constraints, catalysts, being delegated specific functions, or by being "attributed" agency by agentive organisms.[44] While Hornborg allows for the existence of a "hybrid" socioecological process that incorporates both the social and the natural, maintaining this distinction ignores the moments in which symbolism and materiality collapse, forming "feedback loops" of mutual change – moments where, as Edmund Russell claims, historically, "Social forces have been evolutionary forces…Anthropogenic evolution has been a social force."[45]

Actor-Network theory, neo-materialism, and their related theoretical constellations share structural similarities with Niche Construction Theory – painting a picture of ecosystems and materials as constantly in motion. For Feldman and Odling-Smee, this mobility translates to

[43] Alf Hornborg, "Artifacts have Consequences, not Agency," *European Journal of Social Theory* 20(2017): 98.
[44] Hornborg, 99. Hornborg uses this distinction between "reality" and "perception" of agency to incorporate worldviews that include animate nature. Under this definition, Hornborg asserts, "Nature" would include thermodynamics, gravity, and photosynthesis, but "only the non-symbolic aspects of agriculture, markets, or consumption." Meanwhile, "social" refers to objects that do require symbolism. The distinctions so delineated – as between living and nonliving for artifacts and agency, when juxtaposed with a society-nature distinction – seem to challenge themselves. How is it that thermodynamics can underlie biomechanical and chemical processes that mediate living interactions, but be considered an "artifact" rather than an "agent" under Hornborg's own framework? Moreover, this framework becomes immediately problematic when we consider that there are objects that require both an understanding of symbolic importance and of socioecological processes in order to explain their historical significance – for example the corn plant, or for our purposes, the microbe. What would this framework make of organic chemicals like DES, which actively disrupt biological processes and lead to a cascade of (largely still unknown) health consequences for a variety of living organisms?
[45] Edmund Russell, *Evolutionary History: Uniting History and Biology to Understand Life on Earth*, (Cambridge: Cambridge University Press, 2011): 3.

feedback loops – the constant reaction of living organisms to objects in their environment and to each other. Similarly, the "radical empiricism" of ANT, and its exhortations to scholars that they examine the connections between actants to understand the workings of a system is philosophically similar to (and perhaps grounded in) Niche Construction Theory. Where a distinction emerges in the fraught idea of "agency." While their arguments carry strong analytic power, Bennett and LeCain risk expanding the concept beyond an analytic capacity, claiming that everything appears to have at least "quasi agency," claiming action and reaction, capability for inciting change, as key elements of agency. Meanwhile, Hornborg seeks to limit agency to the living-nonliving dichotomy, which, as previously discussed, throws into question the importance of dynamic systems and metabolic (but non-sentient) processes. Niche Construction Theory's utility, perhaps, is its lack of interest in "agency" from a semiotic perspective, and a focus instead on biological and biochemical processes – in other words, regarding metabolism and genetic change as the key drivers of interaction in an ecosystem.

The unique strengths of Niche Construction Theory – its focus on process rather than intent – may provide an opportunity to establish a productive middle ground between post-humanist and neo-materialist theorists and their critics. While Niche Construction Theory does not dispute (and in fact emphasizes) that living and nonliving assemblages have emergent properties (Bennett), Niche Construction Theory emphasizes the role of metabolic organisms in facilitating these emergent properties; as an ecological and evolutionary theory, its focus lies in an organism's ability to interact with an environment independent of a medium. While closer to Hornborg's definition of agency than Latour's in creating a living/nonliving distinction, Niche Construction Theory suggests a system constantly in motion, not unlike Bennett's "vibrant materiality," and finds its strengths in the interconnections between its actors, much like ANT, analyzing action at a multitude of scales constantly shaping other participants in the ecosystem. As a theory bounded by biology, biophysicality, and biochemical processes, NCT incorporates the key assertions of nonhuman theory while assuaging concerns over intentionality.

While Niche Construction Theory may both enliven and provide reasonable boundaries for the analysis of human non-human relationships, the question remains of how to tell stories in which nonhuman action, motion, and change are accounted for and incorporated once a framework is established. For this purpose, historians, anthropologists, and science and technology scholars have learned to rely on salient concepts – "assemblages," "entanglements," and "life-worlds" – which themselves can be combined with a Niche Construction Theory framework to reveal interspecies relationships in historical time. Drawing on examples of how anthropologists, historians, and biologists have designed multispecies and multiobject stories to enliven the ecological systems of which humans are only one part, the next section will demonstrate how key methods of human-environmental and human-nonhuman storytelling compliment NCT, and how together they form a consistent and coherent methodology for interspecies environmental history.

Thinking Through Historical Interspecies Entanglements

While "agency" is controversial in theory, in practice, thinking agentively has opened doors to creative, multiscalar historical thought. In the last decade, environmental scholars in particular have developed narratives that incorporate agency and materiality in their examination of the complex relationships between humans, nonanimal nonhumans, and animal nonhumans, applying ANT and other theories of materiality to multiscalar studies of global capitalism and supply-demand chains, labor histories, material histories, and cultural histories of land and water use. These narratives occasionally engage popular posthumanist tradition seen in the works of Donna Haraway, Anna Tsing, Jane Bennett, and Latour himself, but also a longer tradition of indigenous storytelling and world-building that incorporates nonhumans.[46] In this section, I argue that there are several, often overlapping methodologies embraced by historians and environmental studies scholars that detail the relationships between humans and nonhumans that have parallels in Niche Construction Theory. Together, these methods have allowed historians to reveal the exchanges and transfers inherent in physical and cultural ecosystems and, I argue, provide a bridge between humanistic environmental thought and NCT as a theoretical framework.

Thinking Big: Scaling and Economies of Scale

Questions of scale have increased in popularity as scholars have acknowledged the emergence of a new geological era – the Anthropocene. Historians struggling to come to terms with the implications of defining humans and their activities as geological actors have emphasized the importance of shifting narratives and adjusting imaginative spaces to incorporate what the changing climate and increasing CO_2 levels signify – namely, that our small, daily activities feed into complex systems that alter Earth Systems processes, with profound repercussions for our own activities and survival.[47] As Julia Adeney Thomas notes, "Human-driven processes of many kinds and their synergistic interactions are transforming the planet and its inhabitants on all levels, from the macroscale of planetary warming to the microscale of

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[46] Donna Haraway, A Cyborg Manifesto: Science, Technology, and Socialist-Feminism in the Late Twentieth Century, *Simians, Cyborgs, and Women: The Reinvention of Nature* (New York: Routledge, 1991):149-181; Donna Haraway, *Companion Species Manifesto: Dogs, People, and Significant Otherness*, (Chicago: University of Chicago Press, 2003); Latour, *Reassembling the Social*; Tsing, *Mushroom at the end of the World*.
[47] Steffen "Planetary Boundaries"; Paul J. Crutzen, "Geology of Mankind," *Nature* 415(2002): 23, DOI: 10.1038/415023a; Johan Rockström et al., "Planetary Boundaries: Exploring the Safe Operating Space for Humanity," *Ecology and Society* 14 (2009): 32-65, DOI: 10.1038/461472a.

industrial neurotoxins' effects on fetal development," and importantly, "defining the duration and size of a phenomenon determines much about our understanding of it."[48]

Historians have, in the brief years since engaging with this new phenomenon, already built a rich historiography emphasizing the importance of scale in debates surrounding culpability and impact in the Anthropocene, many of which comprise powerful analyses and critiques of capitalism and imperialism; as Mike Davis, Ramachandra Guha, and Rob Nixon have argued through different means, the entrenchment of the Anthropocene in global capitalism has meant that the consequences of daily activities of those in the so-called developed world are felt disproportionately among those in the so-called developing world.[49] As Nixon in particular reminds us, these are not just questions of spatial scaling between the global and local, but also temporal scaling; the Great Acceleration is counterbalanced with Slow Violence, the "incremental and accretive" effects of environmental destruction in the wake of global development that destroys the health and integrity of communities and individuals – and that falls quickly out of sight in a spectacle-based media and political framework.[50]

By interrogating material environments and practices of extraction, historians and environmental studies scholars have exposed how these forms of relationality often obscure multispecies entanglements, instead alienating and abstracting elements of ecosystems. This practice is, in many ways, entrenched in the practices of environmental history as a field. Alfred Crosby and William Cronon, in works foundational to the field, expose the importance of multispecies relationships in forging imperial and commercial structures across the US. To Crosby, diseases, animals, and "weeds" were enlisted in the imperialist project through their

[48] Adeney Thomas, 1588.
[49] Ramachandra Guha and Joan Martinez-Alier, *Varieties of Environmentalism: Essays North and South*, (London: Earthscan, 1997); Mike Davis, *Planet of Slums* (New York: Verso Press, 2017); Rob Nixon, *Slow Violence and the Environmentalism of the Poor*, (Harvard: Harvard University Press, 2013).
[50] Nixon, *Slow Violence*, x.

relationships with European colonialists; to Cronon, cattle, white pines, and grain, alienated and reframed as "commodities," were central to the explosive growth and economic success of Chicago.[51] Following these examples and inspiring them, rich histories of commodities and imperial environmental change have sought to detail the mechanisms of these relationships and their consequences for both humans and nonhumans, with analytic frameworks that focus on capitalism, imperial land use management, indigenous practice, and global environmental inequality engaging multispecies relationships to varying degrees.[52]

More recently, debates over the stakes of capitalism as a form of relationality have occurred at the global level, with a flurry of activity around exposing the ecological (including human ecologies) consequences of economies of scale emerging out of Anthropocene discourse. The effect of capitalism on the current global order and global ecosystem change have been considered so profound that a number of historians, spearheaded by Jason Moore and Andreas Malm, have argued that the Anthropocene should be re-named the "Capitalocene."[53] Under this

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[51] Alfred Crosby, Ecological Imperialism: The Biological Expansion of Europe, 900-1900 (Cambridge: Cambridge University Press, 2004); William Cronon, *Nature's Metropolis: Chicago and the Great West*, (New York: W.W. Norton & Co., 1991); and William Cronon, *Changes in the Land: Indians, Colonists, and the Ecology of New England*, (New York: Hill & Wang, 1983).

[52] A few notable examples of these histories include Sidney Mintz, *Sweetness and Power: The Place of Sugar in Modern History*, (New York: Penguin Random House, 1986); Richard Tucker and John Richards, *Global Deforestation and the Nineteenth-Century World Economy,* (Durham: Duke University Press, 1983); Edward Mellilo. "The First Green Revolution: Debt Peonage and the Making of the Nitrogen Fertilizer Trade, 1840-1930," *American Historical Review* 117, no. 4 (October 2012): 1028-1060; Kenneth Pomeranz. *The Great Divergence: China, Europe, and the Making of the Modern World Economy,* (Princeton: Princeton University Press, 2001); Fredrik Albritton Jonsson, *Enlightenment's Frontier: The Scottish Enlightenment and the Origins of Environmentalism* (New Haven: Yale University Press, 2013); Mark Elvin, *Retreat of the Elephants: An Environmental History of China*, (New Haven: Yale University Press, 2006). Nixon, *Slow Violence*; Ramachandra Guha, *The Unquiet Woods: Ecological Change and Peasant Resistance in the Himalaya* (Berkeley: University of California Press, 1990); Joshua Specht, *Red Meat Republic: A Hoof-to-Table history of How Beef Changed America,* (Princeton: Princeton University Press, 2019); Prasannan Parthasarathi, *Why Europe Grew Rich and Asia Did Not: Global Economic Divergence, 1600-1850*, (Cambridge: Cambridge University Press, 2011); *Eco-Cultural Networks and the British Empire*: *new Views on Environmental History*, James Beattie, Edward Melillo and Emily O'Gorman, eds., (London: Bloomsbury Press, 2015); Helen Tilley, *Africa as a Living Laboratory: Empire, Development, and the Problem of Scientific Knowledge, 1870-1950,* (Chicago: University of Chicago Press, 2011).

[53] Moore and Malm argue that the central realities of capitalism, including primitive accumulation, alienation, and violent transformation, have meant that earth planetary processes have been subsumed into capitalism; our current

framework, capitalism is seen as the major driver of the Nature/Society divide because it allows for what Moore calls "*real abstractions*" between the structures that (Western, affluent) people live within and the "web of life" within which humans by necessity exist.[54] While these frames have certainly exposed the natural systems that capitalist forms have sought to obscure, the centering of these narratives around capitalism as opposed to nonhuman-human relationships limits the extent to which they re-integrate human behaviors in the environment – in short, the environment is only as deep as the resource as seen by capitalist forces within these frameworks. There are notable exceptions to this limitation, however, with some scholars building stories of capitalist frameworks centered on multispecies relationships, Anna Tsing provides a potent example of this practice in *The Mushroom at the End of the World,* in which she traces the matsutake mushroom from its life in the ground and among pickers (one of the many intimate relationalities in the mushroom-human world), through its abstraction in supply chains and its symbolism within the scope of global elitism and conspicuous consumption.[55]

Other humanists have sought to provide a more locally-grounded portrayal of how economies of scale transform human relationships with ecological systems by focusing on particular iterations and consequences of these economies of scale in global environmental context. This trend is most often visible in environmental histories of imperialism. Mahesh Rangarajan and Ramachandra Guha, for example, have demonstrated how imperial practices shift the relationship between customary users of lands and their ecosystems. Guha looks at the

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epoch is therefore inextractable from capitalism. See Moore, *Capitalism and the Web of Life*; and Andreas Malm, *Fossil Capital: The Rise of Steam Power and the Roots of Global Warming*, (New York: Verso Books, 2016).

[54] Jason W. Moore, "The Capitalocene, Part I: on the nature and origins of our ecological crisis," The Journal of Peasant Studies 44(2017): 598-603. Moore's framework, while an interesting attempt to incorporate nonhumans into capitalist frameworks that have sought to erase them, falls short of his aspirations. Focusing on the "web of life" through the lens of capitalism still necessarily centers humans and human economic systems in environmental thought, and thus reads more as a theory of capitalism than a theory of environment.

[55] Tsing, *The Mushroom at the End of the World*.

divide of Uttarakhand in the mid-19th and early 20th century into two regions, Tehri Garhwal and Kauman, one under a longstanding rajah and the other under British rule and denotes how the introduction of capitalistic structures (particularly the railroad) led to the commercialization of forests as a result, altered peasant rights to common lands and use of forest. Furthermore, Guha argues, the atomizing nature of capitalism meant that the political powers preferred to interact with individual farmers rather than villages that used the land in common, meaning that there was a further reduction in common land. These tensions between commercial use and traditional use sparked many continuing rebellious movements, including the Chipko movement.[56] Rangarajan similarly argues that deforestation was fundamentally different in the colonial period because it was based around capitalist policies of railroad development, revenue, and destroying bandit hideouts, with little consideration for the use practices or necessities of the peasant classes.[57] Histories of bioprospecting also provide a powerful example of this practice of locally-grounded multiscalar storytelling.[58] For example, Abena Dove Osseo-Asare traces how six different plants indigenous to Africa and widely used in traditional medicine were "pharmaceuticalized," and integrated into Western biomedical regimes. All six have since been patented in the U.S., with no acknowledgement to local medical practitioners who first used them.[59]

These trends, in which globalizing supply-demand chains and imperial politics disproportionately affect the relationships of the economically precarious classes to nonhuman

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[56] Ramachandra Guha, *The Unquiet Woods: Ecological Change and Peasant Resistance in the Himalaya* (Berkeley: University of California Press, 1990).
[57] Mahesh Rangarajan, Fencing in the Forest: Conservation and Ecological Change in India's Central Provinces, 1860-1914, (Oxford: Oxford University Press, 1996).
[58] Abena Dove Osseo-Asare, *Bitter Roots: The Search for Healing Plants in Africa,* (Chicago: University of Chicago Press, 2014); *Colonial Botany: Science, Commerce, and Politics in the Early Modern World,* Londa Schiebringer and Claudia Swan, eds., (Philadelphia: University of Pennsylvania Press, 2007).
[59] Abena Dove Osseo-Asare, *Bitter Roots,* 3-7.

environments, inspired Guha's other major conceptualization of human-environmental interaction, which he deems "environmentalisms of the poor."[60] In this framework, the global inequalities driven by imperial and capitalist relationalities are centered, with these systems of power forming an "official landscape" which is then imposed on a "vernacular landscape," fundamentally altering the relationship of people living within a given area to their environment and the other organisms sharing it.[61] The environmentalisms of the poor, according to Guha and Martinez-Alier, are necessarily produced by the violence inherent in this relationship.[62] By conceptualizing multiple environmentalisms and "vernacular landscapes," environmental justice frameworks like Guha and Martinez-Aliers' create analytic space for unique and heterogeneous effects to emerge from these "globalizing" forces.

While useful in illuminating the role of complex power and economic structures in changing ecologies and human-nonhuman relationship, forms of relational thinking through imperialism suffer many of the same limitations as those framed through capitalism. The "environment" largely ends at the scale of the nonhuman directly commoditized by the regime in question. Rarely do the larger ecosystem processes that result from these relationships articulated, and rarely are these ecosystems portrayed as dynamic and changing. As Rob Nixon argues, scholars of the environment as framed through relationalities of imperialism are in need of a theory that situates humans in the biogeochemical as well as and social cycles of existence. He argues that environmental scholars should "ask how directly, how forcefully a given

[60] Guha and Martinez-Alier, *Varieties of Environmentalism: Essays North and South*; Joan Martinez-Alier, *The Environmentalism of the Poor: A Study of Ecological Conflicts and Valuation,* (New Delhi & New York: Oxford University Press, 2005).
[61] Rob Nixon, *Slow Violence and the Environmentalism of the Poor*, 17. In his theorization of the temporal elements of environmental and structural violence in relation to the poor, Nixon draws on a number of examples of how these relationalities alter human-nonhuman relationships. He references, for example, Union Carbide's Bhopal factory gas leak in 1984; Chernobyl and its aftermath; and Ken Saro-Wiwa's advocacy for environmental justice for the Ogoni in Nigeria in the face of oil production.
[62] Guha and Martinez-Alier, xxi.

community is impacted by the cycles of sun and moon; by ebbing and flowing tides; by shifts in the seasons, stars, and planets; by the arrivals and departures of migratory life; and by climate change in ways that are crosshatched with the migratory cycles of transnational capital, electoral cycles (local, national, and foreign), digital time, and the dictates of sweatshop time."[63] In short, scholars of imperialism and environment need a conception of environmental, spatial, and temporal feedback loops that convey the dynamism of environmental systems.

While powerful in conveying the layers of power that drive global environmental change and heterogeneity of responses that emerge from the assemblage of human, economy, and environment, many historians have come to accept the limits of economies of scale as frames of analysis for human-nonhuman studies, emphasizing that the analytic perpetuates euro-centric storytelling practices that are increasingly outdated.[64] Dipesh Chakrabarty, though sympathetic to arguments about the importance of global inequality stoked by capitalism and its relationship to the Anthropocene, emphasizes that capitalism is not a sufficient analytic to explain the current shifts in the planet – that instead we must remember that the conditions for the existence of life "are connected rather to the history of life on this planet, the way different life-forms connect to one another, and the way the mass extinction of one species could spell danger for another."[65] In other words, while capitalism is an important type of multiscalar relationality that transforms global and local actors, it is only one subset of a longer history of relationality that defines the boundaries of life on earth. This observation points to an additional, prominent line of scalar thought focused more broadly on biological history and deep time. While still heavily implicated in Anthropocene studies, these works expand the temporal or spatial scale of historical study

[63] Nixon, *Slow Violence*, 61.
[64] Dipesh Chakrabarty, "The Climate of History, Four Theses," 219-222.
[65] Chakrabarty, *The Climate of History*, 217.

beyond its traditional boundaries, demonstrating the role of climate, ecology, and other biological and geological processes in shaping the fate of human populations. At the forefront of this methodological innovation are scholars like Edmund Russell, John Brooke, and Daniel Lord Smail, who engage temporal and spatial scales of analysis defined by evolution, climate, and neurobiology respectively to de-center economies of scale in historical methods of thinking "big." [66]

Implicated in questions of scale broadly is an important re-imagination of the relationship between the human and natural world as heterogeneous, shaped by a variety of direct and indirect forces, and extremely consequential for the survival of species and entrenched ways of life. These frames thus provide a strong link from environmental humanities to both NCT and posthumanist frameworks. Namely, as Chakrabarty asserts, "Anthropogenic explanations of climate change spell the collapse of the age-old humanist distinction between natural and human history."[67] Once an imaginative space is opened to conceive of humans as geologic and microscopic actors, both geologic and microscopic co-actors populate our understanding of human history. Thus, through scalar thinking, humans are once again incorporated into a dynamic world. Returning once again to our examples of niche construction in action, we can see how facets like ecosystem engineering and ecological inheritance can provide ways of thinking through both spatial and temporal scaling, from the earthworms in the soil to the demand for hardwood flooring that removes trees from the ecosystem, incorporating the actions of the individual into the functioning of the whole. To tell the whole story of Anthropogenic

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[66] Russell, *Evolutionary History*; John McNeill *Something New Under the Sun: An Environmental History of the Twentieth-Century World*, (New York: W.W. Norton Press, 2000); Smail, *On Deep History and the Brain;* Deborah Coen "Big is a Thing of the Past: Climate Change and Methodology in the History of Ideas" *Journal of the History of Ideas* 77(2016): 305-321; John Brooke, *Climate Change and the Course of Human History: A Rough Journey,* (Cambridge: Cambridge University History, 2014).

[67] Chakrabarty, "The Climate of History: Four Theses," 201.

environmental change, however, it is necessary to look not only at the globalizing systems that allow minute interactions to have global consequences – but the quotidian relationships that comprise life in an ecosystem.

Thinking Small to Big: Intimate Relationalities

Often bound up in questions and practices of scale, intimate relationalities comprise the daily interactions of humans with each other and with nonhumans that are highly specific to time and place – the kind that emerge through what Donna Haraway refers to as "vulnerable, on-the-ground work that cobbles together non-harmonious agencies and ways of living" forged between people and nonhumans.[68] These projects trace daily interactions between peoples and landscapes, often exploring how local and global processes unfold in the quotidian – the growth of a coffee plant, for example, as a key component of the livelihoods of residents of a rural village in Mexico, inextricably bound to environment; or the changing nature of human-dog cohabitation as a product of shifting cultural practices and past-times. Anna Tsing and Donna Haraway have produced some of the defining works on this form of relationality used in environmental studies, emphasizing not only the precise combinations of organisms, but their relationships to one another, as singular in time and place, and subsequently consequential. In her recent work, Haraway has taken this focus on relationality to its extreme, arguing that thinking through multispecies entanglements force us to reject the idea of an "Anthropocene" that separates humans from nonhumans. She focuses instead on the constant motions of multispecies connections, the processes of coming together into multispecies assemblages, of "making kin."

[68] Donna Haraway, *Companion Species Manifesto: Dogs, People, and Significant Otherness*, (Chicago: University of Chicago Press, 2003), 7. While Haraway is a useful thinker for multispecies worlds, it is worth noting that her conception of entanglements does not conform to the boundaries I establish in this work – namely, she argues the breakdown of the boundaries between human-animal and machine is a defining feature of posthumanist theory.

She argues instead that we live in the "Chthulucene," a term which instead honors the tentacular nature of our existence – as members of "dynamic ongoing sym-chthonic forces and powers…within which ongoingness is at stake."[69]

Tsing takes a more empirical approach to intimate relationality, demonstrating through ethnographic and archival work what a narrative focused on intimate relationality can bring to our understanding of the layered and complex relationships inherent in multispecies worlds and scalar ecologies. She argues that "The singularity of interspecies gatherings matters; that's why the world remains ecologically heterogeneous despite globe-spanning powers. The intricacies of global coordination also matter; not all connections have the same effects."[70] The *Feral Atlas*, a project of more than 100 social scientists and spearheaded by Tsing, is perhaps the epitomizing work of intimate relationality, tracing the lives and connections of dozens of nonhumans who share ecological and economic spaces with humans, through which it entices readers to "Start with the feral process…it will show you the scale."[71]

Implicit in many works on intimate relationalities, and those of other historians and other social scientists who have sought to weave multispecies environmental stories, are much older indigenous traditions that have captured the constant motion and meaning-making between members of a shared ecosystem. Philippe Descola, for example, documents how different classificatory indicators used by indigenous peoples of the Americas shape their relationship to nonhumans. By his description, the Yukuna developed preferential relationships with animals and plants that implicate protection, using structural relations between humans to inform relationships with nonhumans; meanwhile, he argues, the Yagua classified plants and animals in

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[69] Donna Haraway, "Commentary: Anthropocene, Capitalocene, Plantationocene, Chthulucene: Making Kin," *Environmental Humanities* 6(2015): 160.
[70] Anna Tsing, *The Mushroom at the End of the World,* 213.
[71] *Feral Atlas: The More-than-Human Anthropocene,* Anna L. Tsing, Jennifer Deger, Alder Saxena Keleman, and Feifei Zhou, eds., (Stanford: Stanford University Press, 2020), http://feralatlas.org/.

relation to one another, demonstrating "just how flexible boundaries are in the taxonomy of living beings."[72] Eduardo Viveiros de Castro similarly argues that the "ontological perspectivism" inherent in many indigenous cosmologies resists the Nature-Culture dichotomy, forming instead a "multinaturalism" that facilitates relationality instead of relativity.[73] These ontologies not only comprised perspectives and classificatory systems, but had real, tangible effects on the functioning of the ecosystem – the survival chances of certain animals, plants, soils, and peoples living within them. They also acknowledge, importantly, the role of other organisms within the ecosystem in shaping the livelihoods of their human counterparts – a cosmology symbolized in common practices of metamorphosis and animism.

Scholars focused on water history and borderlands studies have been particularly adept at embracing alternative relationalities brought to the fore through indigenous studies. Josh Reid, for example, in his study of Makah maritime practices, argues that Makah culture was in many ways defined by the "winds, geological features, the circulation of water masses, and marine biology" of the Pacific northwest bioregion and particularly, the entanglement of water, whale, and person. Reid emphasizes, "Unlike Europeans and Euro-Americans who saw marine water as a boundary separating one colonial space from another, Makahs and other indigenous borderlanders continued to experience these waters as a space of connections."[74] Bathsheba Demuth, similarly interested in the intersections of water, whale, and person, notes that Yupikkk, Inupiat, and Chukchi peoples interpreted the material world as an "incorporeal social realm, one in which few things had a permanent form, but most things had souls," and that subsequently, "With no hard line between humans and other persons, land and seas were alive with sentience,

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[72] Philippe Descola, *Beyond Nature and Culture*, (Chicago: University of Chicago Press, 2013): 10.
[73] Eduardo Vivieros de Castro, "Cosmological Deixis and Amerindian Perspectivism," *The Journal of the Royal Anthropological Institute* 4(1998): 469-488.
[74] Josh Reid, *The Sea is My Country: the maritime world of the Makahs, an indigenous borderlands people*, (New Haven: Yale University Press, 2016), 127.

judgment, and perilous whims."[75] These methods do the critical work of revealing the integration of humans into local ecosystems, and the ways that the patterns of life are inextricably bound to each other. By portraying the minute and specific daily interactions of people and their environments and the ways that the nature-culture divide have been culturally and historically constructed, these environmental scholars provide a framework through which to examine the processes of niche construction, ecosystem engineering, and adaptation in specific contexts, revealing the interspecies entanglements that comprise ecosystems.

Especially important in the study of disease, however, is an understanding that "moments of living in common" and intimate relationalities are often less harmonious than the term would suggest. Christos Lynteris emphasizes that microbes in particular show us that moments of interspecies interaction are frequently violent; and when they compromise human populations and institutions, the result is often a very intentional destruction in the form of "culling, stamping out, disinfection, disinfestation, separation, and eradication," which Lynteris asserts is the "sovereign heart" of public health in relation to animal-borne diseases.[76] The way most people interact with a mouse in their house, after all, is through a trap. Disease, perhaps one of the most intimate of all relationalities between human and nonhuman organisms, accentuates this violence – and not only between humans and microbes, but also between any nonhuman perceived to facilitate human-microbe interaction. As Lynteris notes, "all talk of One Health, multispecies relationships and partnerships melts into thin air" any time there is an epidemic crisis.[77] Intimate relationalities document the exchange, sacrifice, and meaning-making that define a vibrant, multi-species world. For Niche Construction Theory, this methodology promises to reveal the

[75] Bathsheba Demuth, *Floating Coast: An Environmental History of the Bering Strait*, (New York: W.W. Norton & Company, 2019), 20.
[76] *Framing Animals as Epidemic Villains: Histories of Non-Human Disease Vectors*, Christos Lynteris ed., (New York: Palgrave Press, 2019), 2.
[77] *Framing Animals as Epidemic Villains*, 2.

quotidian interactions that make up an ecology, describing the very dynamism that the theory seeks to demonstrate.

Thinking with the Individual: Tracing Nonhuman Lifeways

Scalar and intimate relational thinking are methods often grounded firmly in empiricism. Historians and environmental studies scholars build these frames through extensive empirical work grounded primarily in human written or spoken sources. These rigorous methods often maintain an analytic distance from the environment and other nonhumans as a result. The construction of ways of living grounds these practices in the quotidian interactions between individuals, both human and nonhuman, and therefore enriches ecological networks as they integrate into and expand beyond daily interaction. While rarely spoken about it is widely practiced, and I argue, an integral part of re-integrating humans into their environments.

Tracing lifeways is a controversial methodology because it is by nature an imaginative practice, even between individual people; but if we accept the necessity of imaginative practices for history in the Anthropocene (for want of a better term for our current reassessment of human-nonhuman bonds), or for writing about interspecies entanglements, the utility of constructing nonhuman lifeways likewise reveals networks and connections critical to historical understanding. While some caution against "anthropomorphizing" nonhumans in our histories, Jane Bennett argues that "anthropomorphizing" may be essential to revealing the "vibrant materiality" of the world around us – that perhaps, "it is worth running the risks associated with anthropomorphizing (superstition, the divination of nature, romanticism) because it, oddly enough, works against anthropocentrism."[78] Donna Haraway has embraced the practice to

[78] Jane Bennett, *Vibrant Matter*, 121.

illuminate potential for recognizing a mutual constitution of humans and their companion species, emphasizing that "Telling a story of co-habitation, co-evolution, and embodied cross-species sociality…might more fruitfully inform livable politics and ontologies in current life worlds."[79] Meanwhile, Anna Tsing's use of "gatherings" and "happenings" allows for "lifeways – and nonliving ways of being, as well – coming together."[80] By decentralizing the human from action and reaction, we begin to see the ways in which the world forms around us, within us, and because of us. While different in their approaches and in terminology, these theories represent attempts to describe a phenomenon that has long existed in storytelling: namely, grounding our nonhuman actors in their own lifeways, and using what we know of their ways of interacting with the world to form connections.

Perhaps one of the best examples of creative nonhuman studies focused on nonanimal nonhumans – and among the best models for interspecies thinking and writing generally -- comes from Tsing. In *The Mushroom at the End of the World,* Tsing combines ethnography, science and technology studies, and nonhuman and environmental studies to examine the world of the Matsutake mushroom. The mushroom is an non-scalar and yet multiscalar object in Tsing's work, providing a point of entry into worlds as diverse as the Cascades in Oregon, Northern Norway, Tokyo, and Yunnan Province, the life of an itinerant picker, a middle man buyer/seller, a consumer, a spore, a nematode, a tree.[81] Tsing provides theoretical scaffolding for these *multispecies worlds*, as she refers to them, through the works of Haraway, Latour, and other

[79] Haraway, *Companion Species Manifesto*, 4.
[80] Tsing, *Mushroom at the End of the World*, 23.
[81] "Non-scalability", according to Tsing, refers to what multispecies worlds are *not* – they are not homogeneous, not self-contained, not neatly nested, not easily fit within a single analytic frame. To Tsing, stories are nonscalable, and in being nonscalable they are messy and slow – the opposite of economies of scale. According to Tsing, "The main distinguishing feature between scalable and nonscalable projects is not ethical conduct but rather that the latter are more diverse because they are not geared up for expansion." As a historian would say, a nonscalable project or relation is one that is entirely formed and dependent on a particular context.

nonhuman theorists.[82] She draws on the concept of assemblages, but also expands and enriches the idea into "gatherings," and then "happenings," in which these interactions are "greater than the sum of their parts."[83] She moves beyond the simple questions of agency in nonhuman studies, asserting instead that "Making worlds is not limited to humans," and that "world-making projects can overlap, allowing room for more than one species." Within these worldmaking projects, one also sees "multiple temporalities" existing in unison.[84]

Tsing's work shows us an important reality in environmental studies – namely that while debates over decentering humans from history continue to rage in the theoretical arena, the humanities and social sciences have to some extent moved on, and in doing so, accepted the construction of nonhuman lifeways as a powerful tool in multispecies storytelling.[85] Historians have embraced this practice to varying degrees. While humans remain central to most historical narratives, the rise of neo-materialism and the integration of indigenous knowledge forms and storytelling practices have opened conceptual space for the inclusion of more dynamic, detailed descriptions of nonhumans. Bathsheba Demuth provides a striking example of this method in her recent work on the history of the Bering strait, where the rhythms of the ocean and seasons defined the lives of peoples for thousands of years. She opens with a description of the lifeways of the whale, told in the mutual language of labor and nature:

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[82] Anna Tsing, *Mushroom at the End of the World*, 21-22.
[83] Tsing, 22.
[84] Tsing, 22.
[85] Philip Howell, "Animals, agency, and history," in The Routledge Companion to Animal-Human History, Hilda Kean and Philip Howell, eds., (New York: Routledge Press, 2018). The list of works employing this method are almost too numerous to mention. Beyond those already cited numerous times here, a few striking examples include: Alexander Nading, *Mosquito Trails: Ecology, Health, and the Politics of Entanglement*, (Oakland: University of California Press, 2016); Eduardo Kohn, *How Forests Think: Toward an Anthropology Beyond the Human*, (Berkeley, University of California Press, 2013); Urmi Engineer Willoughby, *Yellow Fever, Race, and Ecology in Ninteenth-Century New Orleans* (Baton Rouge: Louisiana State University Press, 2017); Thom Van Dooren, *Flight Ways: Life and Loss at the Edge of Extinction* (New York: Columbia University Press, 2014); James L. Hevia, *Animal Labor and Colonial Warfare* (Chicago: University of Chicago Press, 2018); Cary Wolfe, *Zoontologies: The Question of the Animal*, (Minneapolis: Minnesota University Press, 2003).

> In the long, lazy days with no nights, the mother fed and the calf played: a small breach, then swimming in expanding circles. As summer drew down into September and October, the whales turned again, westward towards the Chukchi Sea, the calf grasping his mother's flippers as they moved. When the chill dark of early winter thickened the pack ice, bringing the danger of sealing the mammals off from oxygen, the whales turned south. Half a year into his life, the calf swam with bolder deeper dives and long gasps at the surface.[86]

Dawn Biehler similarly draws on storytelling elements in the lifeways of pests to draw readers into the ecology of the city in her book, *Pests in the City*:

> *The fly alit upon a cherry, and the miniscule hairs on her feet, akin to taste buds, sensed a viable meal. She vomited digestive fluids onto the cherry's skin, extended her proboscis with its raspy, liplike lobe, and began to mouth and ingest the cherry as it softened. She flitted about the produce cart all day as it made deliveries to markets near the Capitol, some grocers waved her and the other flies away, but others seemed unconcerned about their presence*.[87] [Italics original]

These incorporations of nonhuman perspective through creative storytelling may seem, at first glance, like a simple narrative device designed to draw the attention of wide audiences. While they undoubtedly serve this purpose, I argue that these methods serve an important analytic purpose, as well: they illuminate networks of interaction and means of existence otherwise invisible from an anthropocentric perspective, but necessarily entwined with the ecosystem and sociocultural practices of humans.

These methods also represent entanglements of their own. Constructing the lifeways of another species requires interdisciplinary interaction with science. Few historians would know the geographic patterns of the rat, the migratory and birthing patterns of the whale, or the life cycle of a plant without the work of ecologists, marine biologists, and environmental scientists. The practice of reconstructing the lifeways of nonhumans present within several subdisciplines of the biological sciences provides not only information, but perhaps a promising method for the

[86] Demuth, *Floating Coast*, 16.
[87] Dawn Biehler, *Pests in the City*, 36-37.

humanities and social sciences. As we move into this interdisciplinary spaces, scholars have looked to define theoretical constructs that support the methods described here, namely scaling, relationality, and the construction of lifeways of organisms, and which allows for the actions of organisms to affect others, while still creating boundaries on the scope of these effects that retain the analytic power of storytelling. Niche Construction Theory could provide the theoretical scaffolding to the narrative and analytic impulse to construct lifeways for organisms in a shared ecosystem, bypassing questions of agency by focusing instead on questions of process and consequence.

Thinking with Process: Metabolism

Caught somewhere between methods that engage economies of scale and those that trace the intimate interactions of human and nonhuman organisms are histories that engage the flows of energy and biophysical materials through a system, what can be broadly defined as "metabolism." The term "metabolism" has achieved analytic importance through its metaphorical power, most prominently in its use in social geography to describe the material and energy exchanges between nature and society in the context of the industrial city, as first defined by Marx.[88] In this metaphor, "Urban infrastructure has often been conceived as a functional lattice of different elements which correspond to the different organs of the human body," and metabolism is "interconnected space of flows dependent on external input of energy, materials, and information."[89] Urban metabolism defined in this manner inspired new realms of urban and economic history throughout the late 20th century, with formative effects on the field of

[88] Karl Marx, *Capital,* Vol. III, (New York: International Publishers, 1983), 949-950; Yan Zhang, "Urban Metabolism: A review of research methodologies," Environmental Pollution 178(2013): 463-473.
[89] Matthew Gandy, "Rethinking Urban Metabolism: Water, Space, and the Modern City," *City* 8(2003):363.

environmental history (Cronon's *Nature's Metropolis*, for example, is in part a work on the urban metabolism of Chicago). Importantly, it is through conceiving of the city as a "superorganism" in this way that nonhumans became prominently entangled in narratives of the city, though often as abstracted commodities or facilitators of industry.[90]

However, as Matthew Gandy notes, this traditional definition is not without its problems; he argues, "The metabolic view of the city raises a series of analytical dilemmas concerning the intersection between social and bio-physical dimensions to urban space," reducing a number of historically-specific and agentive interactions to a functionalist model of the city as a self-regulatory system.[91] Recently, the introduction of nonhuman agency into urban metabolic frameworks opened new horizons for the theory by forming what Gandy refers to as a "relational" model of urban hybridity that affords closer attention to entanglements at the local level, allowing for bio-dynamism.[92] Jason Moore has similarly advocated for a relational use of the term "metabolism" in describing human-nonhuman interaction, referencing "metabolic shifts" that occur in the wake of urbanization and industrialization, in which nutrient, mineral, and energy flows were reconfigured towards the city. Moore advocates the term as a replacement for "metabolic shift" mentalities, which he argues perpetuates myth of Cartesian dualism.[93] Urban historians have embraced the "relational" turn, especially in the realm of animal studies, where metabolism has revealed new configurations of nonhuman-human entanglements in the

[90] In addition to Cronon, *Nature's Metropolis*, see Harold Platt, *Shock Cities: The Environmental Transformation of Manchester and Chicago,* (Chicago: University of Chicago Press, 2005); Martin Melosi, "The Place of the City in Environmental History," *Environmental History Review* 17(1993): 1-21. A review of the major literature can be found in *Animal Cities: Beastly Urban Histories,* Peter Atkins, ed., (Burlington: Ashgate Publishing Company, 2012).
[91] Gandy, 374.
[92] *Ibid*.
[93] Jason W. Moore, "Metabolic rift or metabolic shift? Dialectics, nature, and the world-historical method," *Theoretical Sociology* 46(2017): 285-318.

city.[94] Peter Atkins, for example, notes that acknowledgement of the role of animals in forming landscapes and shaping human societies might lead to a reconceptualization of the city as a zoopolis – a space formed in equal measure by humans and by their nonhuman counterparts.[95]

As a term which draws explicitly on the kinds of biogeochemical processes inherent to NCT, metabolism is an important relational framework to connect historical methodology with ecological theory. While this project draws heavily on the forms of relational thinking encouraged by considering “urban metabolisms,” it will largely use the term “metabolism” on a smaller, more conservative scale, defining the term in a strictly biological sense, to describe the biochemical processes by which an organism interacts with its environment and subsequently shapes environments for other cohabiting organisms. In joining a more limited biological interpretation of metabolism with a general Niche Construction Theory frame, I hope to reintroduce some of the “bio-dynamism” often lacking in historical uses of the term.

Conclusion

Thinking through human-nonhuman relationships within the structure of Niche Construction Theory presents a model for a dynamic ecosystem, one that is place-specific and accounts for not only entanglements, but interactions and processes. As a theory that shares methodological and conceptual similarities with many key facets of environmental studies – namely a focus on broad biological and cultural networks that can directly and indirectly influence an ecosystem, altering the relationships therein; the minute daily interactions of organisms and their interactions with their surrounding environments; the life-sustaining

[94] For example, Dawn Biehler, *Pests in the City*; Chris Otter, *Diet for a Large Planet: Industrial Britain, Food Systems, and World Ecology*, (Chicago: University of Chicago Press, 2020); Martin Melosi, “Humans, Cities, and Nature: How Do Cities Fit in the Material World?” *Journal of Urban History* 36(2009): 3-21. doi:10.1177/0096144209349876; Harold Platt, Shock Cities: The Environmental Transformation and Reform of Manchester and Chicago, (Chicago: University of Chicago Press, 2005).
[95] Atkins, *Animal Cities,* 13.

processes that organisms employ that ground them to their environment and connect them to each other; and the energy flows and biophysical processes produced by living and nonliving components of an ecosystem that together shape environments and pressures on other organisms – Niche Construction Theory is uniquely suited to unite diverse frameworks for ecological thinking in the humanities under one coherent theory. The diverse methods employed by environmental scholars to explore these relationships in historical time, meanwhile, provide a set of anchoring practices that contextualize, nuance, and ground Niche Construction Theory. A combination of these elements is particularly important in the context of this project, where I rely on the ecological grounding of Niche Construction Theory, the scalar framework of imperialism, and the intimate relationalities of organisms at the level of the city, neighborhood, and individual to argue that all three cases of epidemic illness are singular interspecies entanglements that result from ecological shocks to the urban landscape driven by imperial economic expansion.

Microbes occupy a uniquely promising position in integrating Niche Construction Theory and methods of conveying interspecies entanglement. Recently, anthropologist Alex Nading notes, scholarship on zoonoses and vector-borne illness has become a lens through which to reconceptualize human-nonhuman relationships as part of a dynamic ecosystem. Anthropological research on these diseases, often grouped into the category emerging infectious diseases (EIDS), has fostered scalar thinking that reveals the chain between the "local" production point and the "global" supply chain; led scholars to question the "porosity of species borders" as they examine how genetic modification transforms mosquitoes from "vectors" into "tools,"; and encouraged interspecies storytelling and ecological thinking to unsettle the prevalent ideas in public health policy that disease ecologies were "knowable, stable, and ground in local 'places.'"[96] The unique position of the microbe at the nexus of human and animal health instills it with a singular power to collapse the human-nature divide – a feature that asserts itself wholeheartedly with the arrival of an epidemic.

This project situates itself at the heart of epidemic crises, taking three different instances of epidemic emergence in urban ecosystems to examine the extent and nature of interspecies entanglements. While there is little doubt that zoonotic epidemic illness led to programs of violence and fundamental changes in the relationship between humans and nonhumans in each of the contexts described here, this project will be careful to consider these moments of interspecies violence not as ruptures in "living in common," but as singular moments of so doing; an epidemic instead becomes an exacerbation of an existing form of relationality – disease or infection – driven by singularities in the social, political, economic, and technological fabric of interspecies life in time and place. In context-dependent examinations of interspecies epidemics, sources reveal moments in which an epidemic not only ruptures the relationship between humans and nonhumans, but makes the relationships between the two more clear than ever by illuminating microbes as important mediators of their interactions – a form of relationality best examined through the lens of Niche Construction Theory.

[96] Alexander Nading, "Humans, Animals, and Health: From Ecology to Entanglement," *Environment and Society: Advances in Research* 4(2013): 68.

Chapter 2: *Mycobacterium tuberculosis* in Melbourne, Australia , 1837-1914

In 1870 the city of Melbourne, Australia did not breathe easily. At least, so argued doctor William Thomson, whose article published that year uncovered an ominous epidemiological trend: a disturbingly high tuberculosis rate. In fact, he argued, Melbourne experienced higher death rates from the disease than any other British colonial city. From 1865—1870, Thomson claimed, 'Nearly 1 in 3 of the adult population of Melbourne, between the ages of 20 and 45 years … die of phthisis'.[1] He mused, 'No other single disease came near to it in fatality. It was at one time thought to be so only in England; but the *English malady* alters not, nor abates its hold upon Englishmen, even here in this favoured climate of Victoria'.[2] Following his discovery, Thompson spent the rest of his career arguing that Melbourne's climate could not cure tuberculosis patients. Despite his vocalizations against medical meteorology and advocacy for sanitary theories of disease, Thomson ultimately failed to provide a firm alternative explanation for Melbourne's elevated tuberculosis rate – especially when other cities in the Empire faced the same sanitary challenges.[3]

Drawing on historical, geographical, epidemiological, and ecological methods, this chapter argues that a combination of environmental factors driven by imperial expansion and local ecology led to the success of tuberculosis in nineteenth century Melbourne.[4] William Cronon argues that cities must be considered as 'places where the products of different

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[1] William Thomson, *On Phthisis and the Supposed Influence of Climate*, (Melbourne: Stillwell & Knight, 1870), 5.
[2] Thomson, *On Phthisis,* 40-41.
[3] Thomson, quoted in Horace Dobell, *Reports on the Progress of Practical and Scientific Medicine in Different Parts of the World,* (London: Longmans, Green, Reader & Dyer, 1871), 484.
[4] Rosemary A. McFarlane, Adrian C. Sleigh, and Anthony J. McMichael, "Land-use change and emerging infectious disease on an island continent," *International Journal of Environmental Research and Public Health* 10 (2013): 2699-2719.

ecosystems, different economies, and different ways of life came together and exchange places'.[5] This rapid confluence of local and imported environmental pressures defined the nineteenth century British boom town. Rapid transformations of landscape occurred as railways, farmland, and urban water supply systems emerged, often modelled on the British mainland.[6] It is thus my contention that radical land use change, driven by the rapid growth of Melbourne and its integration into the British colonial sphere, unbalanced the local ecology. In their settlement of the region, colonists imported infrastructural tools and forms of knowledge that changed the landscape. Through these changes, colonists constructed a cultural niche, which instigated a series of environmental pressures that allowed this non-endemic disease to manifest widely.

By looking beyond traditional historical discussions of tuberculosis, which homogenize environments in which the disease emerges (urban, crowded, poverty-stricken), this project seeks to contribute to global understanding of tuberculosis. It suggests instead that the past environments in which tuberculosis thrived were contingent upon a confluence of specific environmental, social, economic, and political conditions perpetuated by imperial structures. This work necessitates both human and biological models, and therefore expands historical epistemology to accommodate scientific frameworks. The most effective tool for these conditions is the evolutionary and ecological theory of niche construction.[7]

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[5] William Cronon, *Nature's Metropolis: Chicago and the Great West*, (New York: W.W. Norton & Company, 1991), 61.

[6] This pattern of British imperial boom town is best articulated in James Belich, *Replenishing the Earth: The Settler Revolution and the Rise of the Anglo World*, (Oxford: Oxford University Press, 2011), 137-141.

[7] Chris Otter, Nicholas Breyfogle, John L. Brooke, Mari K. Webel, Matthew Klingle, Andrew Price-Smith, Brett L. Walker, and Linda Nash," Forum: technology, ecology, and human health since 1850," *Environmental History* 20 (2015): 710-804.

On Phthisis: The Wicked Problem of Tuberculosis Mortality in Melbourne

As a bacterium that thrives in dark, damp, crowded environments, *Mycobacterium tuberculosis* found the early industrial city a comfortable home. Between the years 1700—1900, tuberculosis was responsible for more than 20 per cent of all human deaths, particularly in Britain, Europe, and New England.[8] In London, a city that experienced near constant epidemics throughout the nineteenth century, the annual death rate reached 716 per 100,000 in the first decade of the nineteenth century and remained above 500 per 100,000 until 1850.[9] Colonialism widened the niche for tuberculosis beyond the European industrial city. Those who embarked from British cities to begin anew in the colonies carried tuberculosis to regions where the disease had waned, or, in the case of Australia, where it had not followed man's initial migration.[10]

Two varieties of tuberculosis can infect humans. Though each has a distinct mode of transmission, once inside the body, they exhibit similar pathogeny. *Mycobacterium tuberculosis* originates in humans and is mainly capable of human-to-human transmission; *Mycobacterium bovis,* or simply bovine tuberculosis, originates in cows but infects humans, sheep, horses, and other livestock. Given livestock raising practices and other characteristics of the built environment, it is likely that both forms were present in the nineteenth century city.[11]

Mycobacterium tuberculosis is contracted (and spread) primarily through the respiratory system. The bacteria emerge from the host in the form of sputum (a product of coughing), and

[8] Thomas M. Daniel, *Captain of Death: The Story of Tuberculosis,* (Rochester: University of Rochester Press, 1997), 16.
[9] Daniel, *Captain of Death*, 30.
[10] H.E. Williams and P.D. Phelan, "The epidemiology, mortality, and morbidity of tuberculosis in Australia: 1850-94," *Journal of Pediatric Child Health* 31 (1995), 496.
[11] Bovine tuberculosis was a larger threat in the nineteenth century than in the twentieth, as pasteurization techniques did not exist. W. Ray Waters, Mayara F. Maggioli, Jodi L. McGill, Konstantin P Lyashchenko, and Mitchell V. Palmer, "Relevance of bovine tuberculosis research to the understanding of human disease: historical perspectives, approaches, and immunologic mechanisms," *Veterinary Immunology and Immunopathology* 159 (2014): 113-132.

may survive in the environment for several days to several weeks.[12] Once inside the lungs, tuberculosis has an exceptional pathogenesis. Unlike most bacteria, it does not immediately sicken the host. Rather, infection begins mundanely; one might develop a slight fever and congestion, akin to a flu virus, which then disappears. The bacteria then fall dormant, quietly circulating in the body, producing no symptoms and no further generations, suppressed by the human immune system. For nearly 92 per cent of cases (today), tuberculosis never progresses beyond this latency phase; however, for 5 to 8 per cent of those infected, the disease manifests. Tuberculosis activation can occur first in the brain, lungs, bone, or kidneys.[13] Before the advent of modern tuberculosis drugs like Isoniazid and Rifampin, active tuberculosis was frequently fatal. It is still a major cause of worldwide mortality today.[14]

Though similar in its pathogenesis to *Mycobacterium tuberculosis,* bovine tuberculosis, or *Mycobacterium bovis*, follows a different pattern of transmission. While bovine tuberculosis can be transmitted through airborne contact like its cousin, as a zoonotic disease, it is notable for its ability to leap from cattle to humans. Transmission often occurs through consumption of unpasteurized dairy products and through direct contact with an infected animal.[15] The bacteria typically enter the circulatory system through the intestine or a wound, and follow an almost

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[12] According to the Public Health Agency of Canada, mycobacterium tuberculosis may last for months on dry surfaces, especially if kept away from sunlight or at a temperature above 4 degrees Celsius. It might linger on carpet for nineteen days, wood for nearly 90 days, in soil for over 30 days, and in the general environment for more than 74 days. For human tuberculosis, however, the most common form of transmission is from particles in the air. See "Mycobacterium tuberculosis complex: pathogen safety data sheet," Public Health Agency of Canada, http://www.phac-aspc.gc.ca/lab-bio/res/psds-ftss/tuber-eng.php/ last accessed December 9, 2016.

[13] Health Academy, "Avoiding tuberculosis: selfstudy program on tuberculosis," The World Health Organization, (Geneva: World Health Organization, 2004), 12.

[14] The Global Burden of Disease Study labels tuberculosis as one of the top ten causes of death in the year 2015. Institute for Health Metrics and Evaluation(IHME), *Rethinking Development and Health: Findings from the Global Burden of Disease Study*, (Seattle: IHME, 2016), 29.

[15] National Center for HIV/AIDS, Viral Hepatitis, STD, and TB Prevention, *Mycobacteruim bovis (Bovine Tuberculosis) in Humans*, (Atlanta: Centers for Disease Control, 2012).

identical pattern of pathogenicity to *Mycobacterium tuberculosis* once inside the body.[16] While pasteurization has rendered bovine tuberculosis uncommon in humans in the twenty-first century, consistent daily contact with cattle heightened its prevalence in the nineteenth century. Children in particular struggled with bovine tuberculosis, often developing tuberculosis of the lymph glands, commonly known as scrofula.[17]

While the epidemiological profile of bovine and human tuberculosis diverge, both thrive in the human body as a result of at least one important host characteristic: lowered immunity, which dramatically increases likelihood of activation. This feature places tuberculosis in the category of both a social and environmental disease, as nutritional status, existing illnesses, and living and working conditions can all dramatically influence an individual's immune strength. Historians have recently developed an interest in immune deficiency as a way of accessing the relationship between human health and the environment in historical time, and thus considerations of poverty, immune strength, and tuberculosis provides an interface between disease, environment, and history.[18]

Tuberculosis is an organism deeply grounded in its environment; its success is closely tied both to its own genetic and environmental niches and those of its hosts. A patient's clinical and exposure history, susceptibility, and concurrent illnesses, when combined with the proper environment, lead to expression of the disease.[19] As latency is associated with a functional immune system encoded with the ability to suppress bacteria, tuberculosis thrives in regions with

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[16] National Center for HIV/AIDS, *Mycobacterium bovis.*
[17] National Center for HIV/AIDS, *Mycobacterium bovis.*
[18] See Gregg Mitman, *Breathing Space: How Allergies Shape Our Lives and Landscapes,* (New Haven: Yale University Press, 2007), 52-53; David S. Jones, "Virgin Soils Revisited," *The William and Mary Quarterly* 60(2003):703-742; and Linda Nash, *Inescapable Ecologies: A History of Environment, Disease, and Knowledge,* (Berkeley: University of California Press, 2007).
[19] E. Schurr, "Is susceptibility to tuberculosis acquired or inherited?" *Journal of Internal Medicine* 261(2007):106-111, and German Center for Infection Research, "Tuberculosis bacteria find their ecological niche," *ScienceDaily*, www.sciencedaily.com/releases/2016/11/161103141408.htm/ last accessed February 17, 2017.

sanitary challenges, where hosts struggle with immune systems weakened by frequent illness and poor hygiene. Importantly, human behaviours like migration might also compromise herd immunity, as new residents of a town predisposed to the disease, previously protected by their home population's high rates of immunity, may have encountered higher rates of infection upon migration.[20]

To consider whether Melbourne, as constructed by settlers, created a niche for tuberculosis, it is important to examine the new city in relation to key environmental factors that the residents and their cultural, political, and economic institutions altered. This includes investigation of the sewage systems and their effectiveness (or lack thereof) as a proxy for drainage or dampness within the city; neighbourhood laws and policies that may have increased interaction between humans and cattle or sheep; examination of housing blueprints to establish style and material; and an examination of census documents related to existing nutritional status, disease, and occupation, which contribute to both the risk of contraction (housemaids, for example, have a much higher risk of developing TB than farmhands) and the risk of expressing the disease.

Tubercular City

Thomson first noted high rates of tuberculosis (specifically phthisis, or pulmonary tuberculosis) in his essay in the *Australasian Medical Journal*, the results of which 'startled' the general population and impressed the scientific community with the 'invulnerable' and

[20] For a definition of herd immunity, see Leon Gordis, *Epidemiology,* (Philadelphia: Elsevier Press, 2013), 26-27.

‘powerfully and eloquently reasoned’ statistics and conclusions.[21] Subsequently in 1870, the Legislative Council of Victoria granted Thomson access to restricted statistics to compile a detailed analysis of the mortality rate from phthisis from 1865—1870.[22] From this study, Thomson concluded that in a five and a half year period, 2,143 people out of a total population of 170,000 (a cause-specific death rate of roughly 14 per thousand) died of tuberculosis, the vast majority of whom were between the ages of 20—35.[23] Over several publications, he revealed characteristics of those who suffered from the illness, including their sex, length of residence in the colony, and place of residence. By Thomson’s calculations, the average sufferer of phthisis resided in the colony for roughly 11 years, obliterating the prevailing theory that active tuberculosis sufferers were primarily immigrants who had arrived already sick with tuberculosis (Table 1). This statistic similarly challenged the prevailing theory that Melbourne’s climate might prevent or cure tuberculosis, as, ‘In deciphering the facts, it plainly appears that an enormous majority [of sufferers] had been many years in the colony before being overtaken by fatal illness’.[24] In his original 1870 publication, *On Phthisis and the Supposed Influence of Climate*, Thomson noted that tuberculosis-specific mortality among those born in the Australian colonies actually tripled between 1865—1870.[25]

[21] “Review: digest of the return ordered by the legislative council of all the deaths (2143) from phthisis in Melbourne and suburbs, during the years 1865-1869, and the first half of 1870,” *The Australian Medical Gazette* 3 (1871): 23.
[22] “Phthisis and Australian climates,” *The Australian Medical Gazette* 2 (1870): 80.
[23] Thomson, *Digest on Phthisis*, 18.
[24] Thomson, *Digest on the Statistics of Phthisis in Victoria*, (Melbourne: Stillwell & Knight, 1871), 8; Thomson, *Third Analysis*, 43.
[25] Thomson, *On Phthisis*, 9.

Table 1. Death Rates from Phthisis among Residents of Victoria Born in the Australian Colonies, 1864 – 1874.

Year	Total Number of Deaths from Phthisis	Born in Australian Colonies (Number)	Percentage of total number of deaths
1865-1870*	4259	233	6
1871	841	91	11
1872	876	112	13
1873	945	147	16
1874	1011	158	16

*First half of year. SOURCE: William Thomson, *On Phthisis and the Supposed Influence of Climate*, Melbourne, 1870, 9

Thomson's explanation for these elevated rates drew on an emerging sanitary ideology. He theorized that aspects of the urban environment in conjunction with population density, may have elevated rates of infection. 'In the lower lying often damp places', where 'land is usually less costly and rents low', Thomson observed sanitary challenges and higher population density.[26] Thomson also noticed particularities in the health profile of the city. In *On Phthisis and the Supposed Influence of Climate*, Thomson cast doubt on citywide density as a risk factor for tuberculosis infection. He compared the rates of tuberculosis in overcrowded London with those of low density Melbourne, and found the two rates nearly identical. [27] This discovery was counterintuitive, as Melbourne's districts covered more land than London (165,000 acres to London's 77,000 acres) and housed a smaller population (only 180,000 to London's 3,000,000). Melbourne had a density of 700 persons per square mile, while the average density of London was 25,000 persons per square mile. '[W]ith all its advantages of space, parks, and widely

[26] Thomson, *On Phthisis*, 9.
[27] M. Lewish and R. MacLeod, "A workingman's paradise? reflections on urban mortality in colonial Australia, 1860-1900," *Medical History* 31 (1987):391; Milton J. Lewis, *The People's Health: Public Health in Australia, 1788—1950*, (London: Praeger Press, 2003), 54.

scattered suburbs and rural districts, it shows as badly as all but the very worst of England's overcrowded cities', Thomson mused.[28]

To reconcile this conundrum, Thomson analysed neighbourhood-level factors, including the occupations, housing conditions, ages, and locations of those who died of tuberculosis over the mid nineteenth century. He concluded tentatively that a difference in diet and built environment may have been to blame for the high mortality rates in particular neighbourhoods, which translated to high citywide rates.[29] Aware that his findings contradicted dominant, climatological theories of disease, Thomson continued to document tuberculosis death rates in the city and its suburbs, which only worsened between 1870—1882.[30] Vital statistics confirm that residents of Melbourne, and particularly residents of a select few neighbourhoods died of tuberculosis at nearly twice the rate of any other residents of the colony of Victoria (Figure 2). To understand why death rates in the colony were remarkably high, we must examine the urban ecology of the city in relation to the bacteria.

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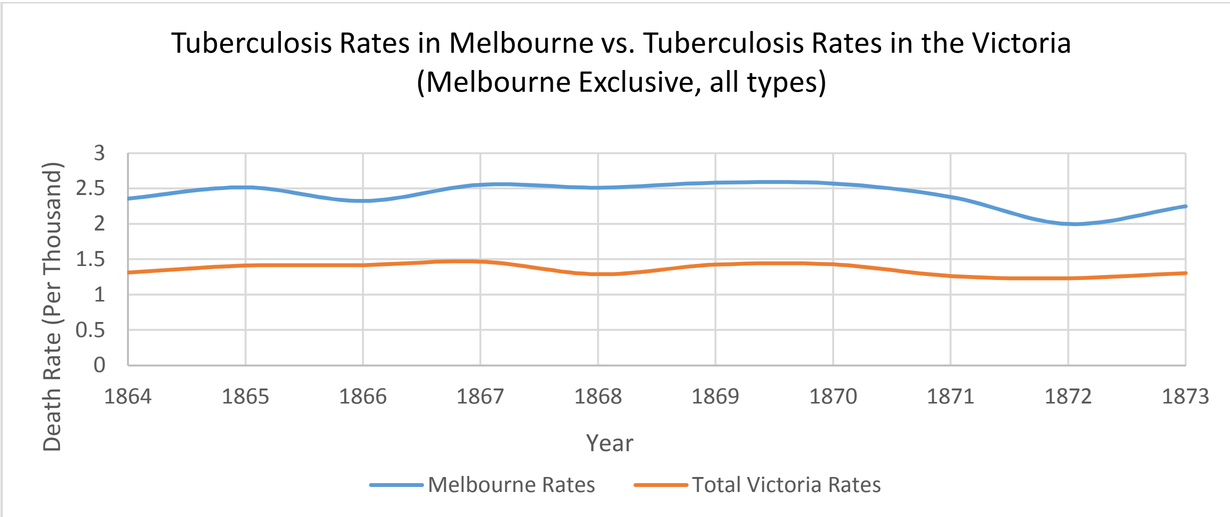
28 Thomson, *On Phthisis*, 44-45.
29 Thomson, *On Phthisis*, 117.
30 Thomson, *The Germ Theory of Phthisis*, 20.

Figure 2. Tuberculosis rates in Melbourne as compared to tuberculosis rates in the rest of Victoria.



SOURCE: William Archer, *Registrar General Statistics for the Colony of Victoria 1873*, *Compiled from Official Records in the Registrar-General's Office,* (Melbourne: Government Press, 1874), 11-24 and 30-32.

Melbourne's Changing Environment

The settlement of Melbourne began as a great many settlements in Australia began: illegally. Though the government of New South Wales launched several expeditions in the early nineteenth century into the Port Phillip region, where the city eventually grew, in hopes of establishing a southern port, it was not until the year 1835 that white Australians attempted settlement. In the summer of 1835, a grazier named John Batman, southbound from the New South Wales and acting as an independent agent, signed a treaty with the native Wurundjeri for the purchase of land along the river Yarra in exchange for blankets, shirts, scissors, looking glasses, and other goods.[31] The government of New South Wales condemned the action and rejected Batman's petition for settlement in the region; he ignored them. By the time the government of New South Wales agreed to send representatives down to Port Phillip to establish

[31] Garryowen, *Chronicles of Early Melbourne*, (Melbourne: Fergusson and Mitchell, 1888), 2.

a formal town in 1837, there were at least 27 men, Batman among them, who resided on the land with thousands of sheep.[32]

Early settlers of Melbourne wrote in detail on the climate, geology, and topography of the region.[33] The climate was often compared to that of London; temperatures ranged between 45—75 degrees Fahrenheit with frequent rainfall. The ecology was coastal in nature, its major flora consisting of banksia, honeysuckle, and grass trees. To the Australian agriculturalist, it was a dream. One observer noted, 'So genial was the climate and so extensive the pasturage, that sheep increased on the average 90 per cent. per annum. From 250,000 sheep in 1839, when they ceased to be imported, they had increased in 1851 to 6,320,000'. It was not long before speculators, builders, and farmers turned to Port Phillip as an ideal site for 'improvement'.[34]

Some of the earliest observations on the region came from Robert Hoddle, the main surveyor of the site and the namesake of the 'Hoddle Grid', Melbourne's urban plan. Hoddle described a climate milder than that of New South Wales, noting the natural drainage systems centred on the Yarra River.[35] Though optimistic about its prospects, he also conveyed concerns over climate and topography, as 'Melbourne, on the river side, is surrounded with a marshy plain, which is frequently inundated; the miasma arising is injurious to the health of the inhabitants, but it might be easily drained and the land made available'.[36]

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[32] Garryowen, 6; Victoria Registrar General's Office, *Statistics for the Colony of Victoria, for the year 1871, Compiled from Official Records in the Registrar-General's Office*, (Melbourne: Government Printer, 1872), 5.
[33] D. Astley Gresswell, *Board of Public Health Report on the Sanitary Condition and Sanitary Administration of Melbourne and Suburbs* (Melbourne: Government Printer, 1890), 5.
[34] Edmund Finn, *A Description of the Province of Victoria: Australia, by the editor of the 'Australian and New Zealand Gazette,'* (London: Algar Street, Colonial Publishers, 1858), 5.
[35] Robert Hoddle, *A Chapter on Port Phillip. Account of the Settlement from its Formation with a Map, by Robert Hoddle, Surveyor, Late in Charge of the Survey Department, — 1840; and now resumed the Charge of the Survey Department,* Thumb Creek: Garravembi, 1991), 8.
[36] Hoddle, *Port Phillip,* 15.

As a native Londoner and a member of the Corps of Royal Military Surveyors and Draftsmen, Hoddle had experience in colonial urban planning.[37] His work in Port Phillip was indicative of his intellectual lineage. Typical of British colonial towns, the original town plan was laid as a 'brisk, matter of fact' grid across the landscape, 2.25 square miles across, with principal streets 99 feet in width (Figure 2).[38] Sewers largely ran above ground, designed to follow the contours of the road until finally flowing into the Yarra and out into the Bay.[39] Though these above ground channels were intended to flow into underground drains, this rarely happened in practice.[40]

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[37] Hoddle, *Port Phillip,* v-x.

[38] Winston Burchett, *East Melbourne, 1837-1977: Peoples, Places, Problems,* (Melbourne: Craftsman Press, 1977), 1; and Andrew Brown-May, *Melbourne Street Life: The Itinerary of Our Days,* (Melbourne: Australian Scholarly Publishing, 1997), 9-10.

[39] Tony Dingle and Carolyn Rasmussen, *Vital Connections: Melbourne and its Board of Works*, (Melbourne: Melbourne and Metropolitan Board of Works Press, 1991), 35-36.

[40] Dingle and Rasmussen, *Vital Connections*, 36.

Figure 3. Map of the City of Melbourne, ca. 1855. Note that all settlement at this point is North of the Yarra River.

SOURCE: David Tulloch, Melbourne and Its Suburbs, 1:10,000, Melbourne, 1855, The University of Melbourne Digitized Collection, https://digitised-collections.unimelb.edu.au/handle/11343/24001.

Melbourne's residents immediately experienced problems with Hoddle's Grid. The somewhat unimaginative pattern ignored the topographical realities of the region, providing inadequate drainage for the volume of rain and discounting particularly flat expanses where water could not drain.[41] Residents wrote of wading through waist-deep flood water following a storm, and how they considered the stagnant water to be a serious health hazard.[42] By 1848, a temporary select committee of the Council wrote with concern on the 'want of drainage, the filthy condition of the narrow streets, courts, and alleys, the prevalence of stagnant pools of water, the habit of slaughtering animals in the city proper and a large unhealthy swamp on the

[41] Burchett, *East Melbourne*, 1.

[42] There are countless accounts of this happening. For examples, see Garryowen, 298; Brown May, *Melbourne Street Life,* 76, and Gresswell, 12-13.

east side of the city known as "Lake Lonsdale"'.[43] Even Hoddle himself complained of the boggy flooding of the streets in 1840, though he blamed the engineers for 'forgetting' to make the necessary drains.[44]

While a lack of proper drainage systems made flooding frequent, the interaction of the delicate river valley ecology with the blooming industrial town might have worsened the situation by increasing water flow into the city. Melbourne's establishment significantly impacted the natural water cycle of the region.[45] Prior to settlement, rainfall was absorbed into the subsoil, used by plants, evaporated, or drained into a system of creeks and rivers flowing towards the Bay. While the central business district (the centre of Hoddle's grid) stood on land that sloped towards the river, the main areas of residence – South Melbourne, Port Melbourne, and West Melbourne – were 'naturally swampy'. [46] More than interrupting the natural system in place, Melbourne's urban plan may have worsened it, as 'many city streets interfered with the natural drainage, increasing the likelihood of stagnation and flooding'.[47] This topographical disorganization resulted in streets so pungent that locals nicknamed the town 'Marvellous Smellbourne'.[48]

To *Mycobacterium tuberculosis* and *bovis*, the rapidly expanding city constituted an ideal habitat. Continually flooded landscapes and poorly constructed drains threatened residents' health, both in creating stagnant water to facilitate water borne illness and damp, molded houses. These structural features combined to foster high levels of infectious disease in a region that, half a century previously, was inhospitable to these organisms. By promoting illness in the human

[43] David Dustan, *Governing the Metropolis: Politics, Technology, and Social Change in an Australian City: Melbourne, 1850-1891*, (Melbourne: Melbourne University Press, 1984), 121.
[44] Hoddle, 8-9.
[45] Dingle and Rasmussen, *Vital Connections*, 32-33.
[46] Dingle and Rasmussen, 32-33.
[47] Dingle and Rasmussen, 32-33.
[48] Dingle and Rasmussen, 32-33.

population of Melbourne, the city structure contributed to thousands of compromised immune systems – exactly the condition needed for tuberculosis to reproduce and thrive.

The challenges of Melbourne's urban structure became more pronounced throughout the nineteenth century. Though already growing at a rapid rate, Melbourne's population skyrocketed upon the discovery of gold in the region in 1851, from 177 in 1836 to 123,000 by 1854.[49] Seemingly overnight, the city transformed from a small, agrarian settlement to a bustling metropolis. Rampant speculation that followed on the heels of the gold rush, resulted in the construction of railways, commercial businesses, and buildings at astounding rates. Locals registered this change with awe. The editor of the Australian and New Zealand Gazette noted,

> A city and rapidly extending suburban population of more than 100,000 souls swarm along the banks of the busy Yarra-Yarra, where quarries, warehouses, and factories have almost sprung out of the soil, twenty-five years ago only trodden by the kangaroo … Here centres a chaos of human happiness and misery – physical wealth and … poverty, distress, and luxury virtue and vice, in extremes unparalleled elsewhere in the British Empire.[50]

Despite the city and suburbs' explosive growth, residents rarely complained of overcrowding; nineteenth century Melbourne was a city of 'notably low density' — an average of twenty-three people to the acre (excluding parks and public gardens), with the most densely populated areas reaching no more than forty people per acre. North Melbourne held thirty-seven people per acre, Fitzroy thirty-five, and Collingwood housed thirty. Compared to London or New York, cities with average densities of over 500 per acre (though with similar rates of tuberculosis), Melbourne may as well could have been a bumbling country town.[51] In the

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[49] William Archer, *Statistics of the Colony of Victoria for the Year 1865, Compiled from Official Records in the Registrar-General's Office*, (Melbourne: Government Press, 1855), 173; and Garryowen, *Chronicles of Early Melbourne,* 492.
[50] Finn, *A Description of the Province of Victoria,* 4.
[51] Dingle and Rasmussen, *Vital Connections*, 28.

construction of this niche, therefore, we might consider that overcrowding alone played a lesser role than previous studies of tuberculosis have emphasized.

Numerous cultural and social practices instated by Melbourne's residents also led to the construction of a niche well suited to respiratory illness. As residential suburbs emerged in the 1850s, it became apparent that Melbourne's early spirit of independent settlement remained strong; rather than growing around a centralized government, Melbourne and each of its suburbs instead maintained local councils (56 in all by the 1890s) that managed major governance decisions.[52] The result was a fragmented city, in which infrastructural development varied widely between municipalities – a weakness only exacerbated by rampant growth.[53] The result was a city with widely varying sanitary systems and building guidelines.

As the population continued to boom in the second half of the nineteenth century, reaching nearly 500,000 by 1893, the challenges of this system became apparent. In an 1890 report on the sanitary condition of Melbourne and its suburbs, a prominent medical officer of health named D. Astley Gresswell described the condition of housing, which 'in not a few localities' rested on ground 'consisting largely of refuse and night-soil'. In many cases, houses were 'lower than the surrounding surface, so that they form a receptacle for stagnant and commonly polluted water'.[54] Likewise, the faulty sewers of the 1840s remained, resulting in pools of waste that frequently overflowed and soaked into the surrounding soil. He observed that most wooden gutters were already rotted through. According to Gresswell, this condition, which existed in most municipalities, could be construed as a failure of local governance systems, as '…[I]t has been stated by local officers that their councils had no power to prevent the building

[52] Dunstan, *Governing the Metropolis,* 3-5 and 65-66.
[53] Dustan, *Governing the Metropolis*, 26.
[54] Gresswell, *Board of Public Health Report*, 6-7.

of houses on undrained land, and that the houses in their districts were being built anywhere and anyhow'.[55]

The effects of these sanitary inadequacies were evident in the city's continuous struggle with epidemic disease – alongside tuberculosis, Melbourne suffered from elevated death rates from dysentery, diarrhoea, typhus, and nutritional illnesses leading to 'atrophy and debility'.[56] In fact, deaths attributed to nutritional deficiency rivalled those from tuberculosis, suggesting consistent undernourishment among Melbourne's residents. This particular epidemiological trend is especially pertinent given the relationship between nutritional deficiency and active tuberculosis.[57] While these waves of illness translated into a relatively average death rate for a nineteenth century British city, they also contributed to a significantly sicklier population.[58]

Beyond the sanitary structure of Melbourne, resident practices contributed to the tubercular environment. Livestock production in particular established an optimal environment for bovine tuberculosis. While the majority of goods that left Port Phillip for the empire came from Melbourne's hinterlands, meat and milk products for the city were produced in the suburbs. It was typical to see herds of cows or sheep being driven down the street; one might later walk to the neighbourhood slaughterhouse to purchase their meat or milk.[59] These corner shops and backyards were often unhygienic, rendering livestock vulnerable to infection by both typhus and tuberculosis. In his sanitary report, Gresswell noted this vile state of affairs, claiming, 'Not one

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[55] Gresswell, 7.
[56] William Archer, *Statistics for the Colony of Victoria, for the year 1863, Compiled from Official Records in the Registrar-General's Office*, 1863-1883, (Melbourne: Government Press, 1864); Archer, *Statistics for the Colony of Victoria, for the year 1873*, (Melbourne: Government Press, 1864); Archer, *Statistics for the Colony of Victoria, for the year 1883,* (Melbourne: Government Press, 1884).
[57] Schurr, "Is susceptibility to tuberculosis acquired or inherited?," 107.
[58] Lewis, *Public Health in Australia*, 54-60.
[59] Finn, *A Description of the Province of Victoria,* 4-6; Bernard Barrett, *The Inner Suburbs*: *The Evolution of an Industrial Area,* (Melbourne: Melbourne University Press, 1971), 123-124; Peter Davies, Mapping commodities at Casselden Place, Melbourne, *International Journal of Historical Archaeology* 10 (2006): 343-355.

of the forty-three milk establishments which I have visited was in a satisfactory condition … Some of the cows in very poor condition, are fed on improper food, and are supplied with water from filthy sources'.[60] Similarly, Gresswell found that once these products had been purchased, they were kept with equal carelessness. He noted, 'Milk and cream were found to be kept in many places where they would be most likely to become contaminated', including living rooms, basements, and bathrooms.[61] The relationship between residents and livestock fostered an environment suited to the transmission of bovine tuberculosis.

From the inception of the original city plan to the blossoming of local districts, Melbourne saw a substantial change in its environment in the mid nineteenth century. This change was not without its negative effects: a poorly conceived grid pattern ill suited to local topography, which promoted damp environments; speculation and rapid growth led to poorly ventilated, closely constructed, unstable housing; and the proliferation of economic and cultural practices like animal keeping and employment in industry left residents vulnerable to infection. Tuberculosis bacteria experienced an increased opportunity for transmission; and once inside the host population, suppressed immune systems allowed the bacteria to multiply, leading to expression of the disease by the host. It is also important, however, to note the specificity of tuberculosis, even within Melbourne. While these pressures existed in the town as a whole, the unique structure of the city and its topographical features left some districts more vulnerable than others. Thus, to relate these ecological and cultural pressures and the emergence of tuberculosis, one must examine where tuberculosis infection rates were highest.

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[60] Gresswell, 21.
[61] Gresswell, 21.

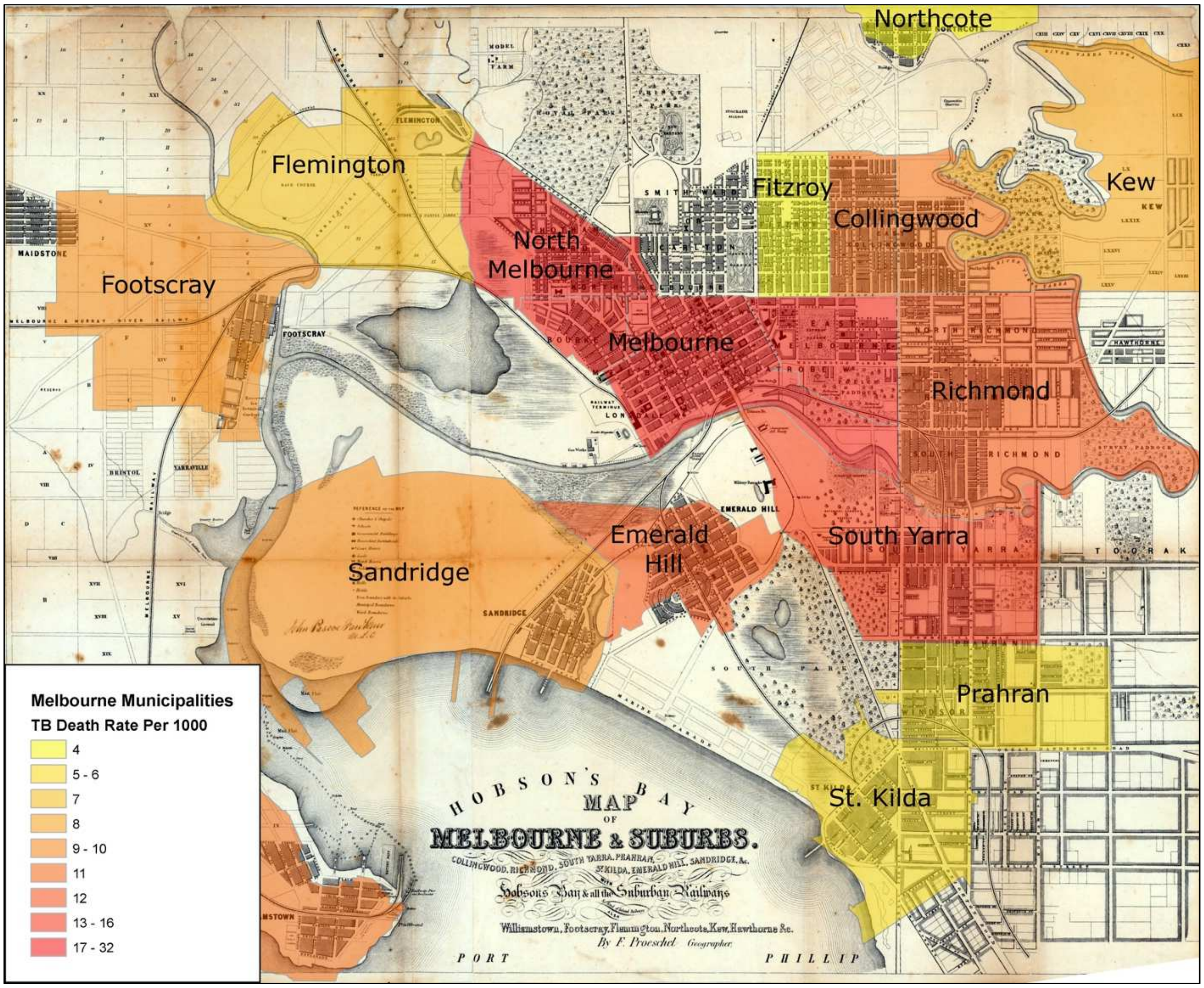
Case Study: Neighborhoods as Ecological Niches

Table 2. Tuberculosis-specific deaths, total number per district, 1865-1870.

Sub District	**Total Deaths from Phthisis**	***Sub District***	**Total Deaths from Phthisis**
Brighton	25	***Malvern***	5
Brunswick	22	***Melbourne North***	352
Caulfield	3	***Melbourne***	824
Cheltenham	7	***Northcote***	49
Collingwood	281	***Pentridge***	17
Emerald Hill	103	***Prahran***	56
Essendon	6	***Richmond***	134
Flemington	10	***Sandridge***	32
Footscray	11	***South Yarra***	47
Hawthorn	37	***St. Kilda***	40
Heidelberg	10	***Toorak***	4
Keilor	2	***Williamstown***	48
Kew	18	***Total***	2143

SOURCE: William Thomson, *Digest of the Return of the Deaths (2143) from Phthisis in Melbourne and Suburbs, During the years 1865-1869, and first half of 1870*, Melbourne, 1871, 18; Thomson's data from William Archer, *Statistics for the Colony of Victoria, for the year 1875, Compiled from Official Records in the Registrar-General's Office*, Melbourne, 1876, 9—36.

Figure 4. Tuberculosis-specific death rates per Thousand in Melbourne and the Inner Suburbs, 1865-1870.



SOURCE: Frederick Proeschel, Map of Melbourne and its Suburbs, Collingwoods, Ritchmond, South Yarra, Prahran, St. Kilda, Emerald Hill, Sandridge, &c. Hobson's Bay & all the Suburban Railways, Williamstown, Footscray, Flemington, Northcote, Kew, Hawthorne, &c., Melbourne, 1851-1869. National Library of Australia TROVE database. Accessed December 8, 2016, https://nla.gov.au/nla.obj-230010454/view. Tuberculosis data from Thomson, Digest, 18. Disease map created by author.

William Thomson conducted the only known analysis of phthisis rates in each of Melbourne's districts and suburbs (Table 2). Most noticeable in the table above, compiled by Thomson himself, are the elevated tuberculosis rates in the districts of Emerald Hill and

Collingwood.[62] The third and fourth most affected regions, respectively, the neighbourhoods had a high death rate of roughly 11 per thousand each. This trend is unexpected at first glance, as the two communities had relatively low population densities and lay on opposite ends of the city (Figure 4). The ecological particularities of these boroughs, and the cultural and economic characteristics that influenced their local environment, however, explain their elevated tuberculosis rates. Their difficult, damp, swampy topography; the rate of housing construction and speculation; inadequate drainage systems and preventative public health measures; and a culture of open—grazing livestock combined to form an ideal niche in both spaces.

Early in Melbourne's settlement, the area south of the River Yarra served largely as grazing ground. Its defining characteristic was a grassy knoll christened Emerald Hill. Local chronicler Edmund Finn (alias Garryowen), cited the local ecology as the main deterrent to early settlement. He noted, 'The Hill, though a picturesque and beautiful place in itself, was surrounded by swamps, and deemed…an unhealthy locality'.[63] Robert Hoddle, who surveyed the area as part of his original plan for Melbourne in 1839, similarly expressed reservations, as the region was largely a 'large marshy plain, subject to inundation, and therefore unsuitable as a town site'.[64] However, proximity to the Yarra River and the town centre proved enticing, and the subdistrict grew to encompass the region directly south of Melbourne proper down to Port Philip Bay (Figure 5).[65]

[62] While at first glance it appears that TB rates are highest in North Melbourne and Melbourne as designated on the map, accuracy of tuberculosis rates in these neighborhoods is difficult to know, as it houses the major hospital for the region; as deaths in the hospital are counted towards death for the district, it may artificially inflate death rates. Thus, the districts with the third and fourth highest tuberculosis rates are considered here. Outer suburbs are omitted from mapping.
[63] Garryowen, *Chronicles of Early Melbourne,* 21; Charles Daley, *The history of South Melbourne: From the Foundation of Settlement at Port Phillip to the Year 1938*, (Melbourne: Robertson and Mullins, 1940), 10.
[64] Daley, *South Melbourne*, 30-31.
[65] Daley, *South Melbourne*, Map, 30-31.

Figure 5. Elevation of Emerald Hill

SOURCE: Map created by author. Elevation data courtesy of Geoscience Australia Digital Elevation Database (ELVIS).

Almost immediately, residents experienced difficulties with flooding. Floods every few years from 1840 to 1860 left Emerald Hill an island, sweeping away the huts and settlements that surrounded the hill.[66] While this ecology made the area very difficult to settle, the discovery of gold in 1851 acted as a great motivator.[67] The population grew so rapidly that until 1855, Emerald Hill was an informal canvas town, as builders struggled to accommodate the increasing demand. In that year, the area housed a large enough resident population to be declared a borough by the city government.[68]

[66] Daley, 14-18.
[67] Daley, 28-29; Garryowen, 249.
[68] Daley, *South Melbourne*, 33.

The trajectory for the neighbourhood of Collingwood resembled that of its southerly neighbour. Much like Emerald Hill, Collingwood was a low lying locality in an otherwise well drained and hilly region. As a result, water often accumulated on its flat surface, resulting in muddiness unparalleled in other parts of the settlement (Figure 6).[69] For this reason, the area was poorly settled until the gold rush, after which rampant speculation and building accommodated a rapidly expanding population.[70] By 1861, the suburb housed a population of 12,653.[71]

Figure 6. Elevation of Collingwood

SOURCE: Elevation data courtesy of Geoscience Australia Digital Elevation Database (ELVIS). Map created by author.

[69] Daley, 33.
[70] Barrett, *The Inner Suburbs,* 22-25.
[71] Barrett, 25.

From the beginning, both neighbourhoods faced structural challenges. Collingwood, for example, was famous as 'a cesspool for refuse which gravitated from higher areas'.[72] Sanitary challenges quickly sullied Collingwood's reputation; early residents expressed alarm over a 'decline in local land values, rents and business and by the district's poor reputation'.[73] Complaining of Melbourne Municipal Council's lack of attention to the needs of the budding community, Collingwood became the first suburb to declare itself an independent municipality in 1855.[74]

Rather than fixing infrastructure, however, the bid for independence created challenges for the new town. Suddenly unable to directly draw money from rates collected by the city, the local authorities were unable to complete the necessary roads and sewers.[75] Though still attached to the Melbourne Municipal government, Emerald Hill fared no better in these early years; the majority of its roads were simply wooden planks.[76] Both neighbourhoods contained mostly wooden houses, as well.[77] While these attributes were not unusual for the nineteenth century boom town, the combination of wooden infrastructure alongside frequent flooding could mean rot, mould, and water absorption – all preferred by tuberculosis bacteria. Furthermore, speculation and sales patterns in the area led to 'unwisely subdivided' blocks that severely congested housing. In a public housing report, a local health officer observed that the houses on Little Ragland Street were built with a 12 feet allotment in front and no room at all in the back, presenting challenges for refuse, night-soil removal, and ventilation.[78]

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[72] Barrett, 9.
[73] Barrett, 9.
[74] Barrett, 37; Daley, 47-53.
[75] Dunstan, *Governing the Metropolis*, 135-138; Barrett, 37.
[76] Daley, *South Melbourne*, 76-77, 112-113.
[77] Barrett, 28-29; William Archer, *Census of Victoria, 1871, General Report and Appendices,* (Melbourne: Government Press, 1871), 7.
[78] Barrett, 123.

An 1889 sanitary report named South Melbourne (which consisted of Emerald Hill and Sandridge) as one of the few boroughs that housed residents in back-to-back buildings that, though properties were 'generally of good dimensions', provided little outdoor space and terrible ventilation. Though this was not unusual for the nineteenth century city, topography exacerbated these conditions.[79] By building houses on natural drainage pathways, residents of Emerald Hill and Collingwood (and the speculators and builders who enabled them) contributed to the creation of uniquely unhealthy environments; damp, sinking houses offered an ideal habitat for *Mycobacterium tuberculosis*, while haphazard construction led to poor air circulation, ensuring that bacteria could live out their maximum lifespan in the air and on surfaces. Though tuberculosis rates cannot be linked solely to sanitary characteristics, these regions showed distressing trends in phthisis mortality: by 1913, tuberculosis had killed more people in South Melbourne than typhoid, diphtheria, and scarlet fever combined (Table 3).[80]

Table 3. Cases and Deaths from Diphtheria, Typhoid, Scarlet Fever, and Phthisis in South Melbourne, 1913.

Disease	Cases	Deaths
Diphtheria	221	2
Typhoid	35	1
Scarlet Fever	4	0
Phthisis	69	28

SOURCE: Report of the Health Officer for Emerald Hill, quoted in Daley, 170.

The economic circumstances of Emerald Hill and Collingwood did nothing to alleviate their unhealthy environments. As the first peripheral aggregations to the blossoming city of Melbourne, both regions became hubs of industrial activity. Collingwood specialized in 'noxious industries', and housed slaughter yards, tanneries, soap and candle works, works for cleaning

[79] Gresswell, 6.
[80] Daley, 170.

sheepskins and wool, brickmaking, brewing, and the night soil trade.[81] Emerald Hill housed similar industries, with a special emphasis on brickmaking.[82] Though they were centres of manufacture, these 'industrial' areas existed in close proximity to the agricultural land from which they drew their materials. Bernard Barrett describes Collingwood as maintaining a 'barnyard atmosphere' for much of its nineteenth century existence, with cattle, sheep, and other livestock wandering the streets freely. In 1871, 71 per cent of Collingwood's householders kept livestock, while 36 per cent kept poultry.[83] This trend appeared to be pervasive within the community, as 'humans in Collingwood were still outnumbered by animals even in 1891'.[84]

The intermixing of livestock and industry caused similar difficulties in Emerald Hill. Residents faced the sanitary challenges of industrialization and complained of improper disposal of manure and refuse, noxious trades and occupations, and nuisances from animals, among other problems.[85] The significance of livestock keeping within the regional boundaries of both Emerald Hill and Collingwood in large numbers (numbers greater than most of the Melbourne metropolitan area) extends beyond questions of manure and refuse. The aetiology of bovine tuberculosis suggests that conditions in both Emerald Hill and Collingwood would suit the spread of this bacterium.[86] Bovine tuberculosis, though virtually indistinguishable from its cousin in its natural history, is spread not only via sputum and direct contact, but also through saliva. Therefore, cattle exposed to the same sources of water as an infected cow are likely to

[81] Barrett, 11.
[82] Garryowen, 663.
[83] Barrett, 124.
[84] Barrett, 124.
[85] Daley, 168; Garryowen, 332.
[86] Anna Rovid Spickler, "Bovine tuberculosis, the Center for Food Security and Public Health," Center for Food Security and Public Health, Iowa State University, 2009, http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php/ last accessed 8 March 2019.

develop the disease.[87] As most residents of Collingwood owned their own livestock, particularly their own cattle, milk and beef likely represented a major portion of the local diet. The neighbourhood also housed a number of abattoirs, which were frequent targets of local nuisance complaints for their uncleanliness.[88] A lack of regulation, combined with the economic practices of Collingwood in particular may have created a unique niche for bovine tuberculosis, as it cost 5s to 20s less to register as a milk vendor in this suburb than in any other part of Melbourne, which led to an increased production (and presumably consumption) of milk.[89]

The combination of poor drainage systems, freely roaming livestock, and pervasive milk sales proved problematic, as 'In the 1890s the council realized belatedly that for years cows had risked infection by drinking diluted sewage in open drains while out grazing or while heading home to be milked'.[90] Cattle exposed to bacteria and refuse swirling in these sewer systems would have risked illness, taxing their immune systems. Compromised immunities in the cattle from this practice may have fostered the development of bovine tuberculosis, which may have subsequently spread to humans via frequent contact. Upon discovery of this deeply unsanitary practice, the council prohibited the grazing of dairy cattle on any unfenced land, began destroying diseased cattle, and in Collingwood, increased the vendor's registration fee from 5s to 20s – level with other municipalities.[91]

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[87] Animal and Plant Health Inspection Service, "Questions and answers: bovine tuberculosis," United States Department of Agriculture, Washington, 2014.
[88] Collingwood nuisances, *Weekly Times*, 20 Mar 1880, NLA Trove; The Collingwood Abattoirs, *The Age*, 18 Mar 1881, NLA Trove. It is worth noting that cattle products would not have been inspected until the late nineteenth or early twentieth century, when literature concerning the relationship between cows and disease pervaded Anglophone public health boards. They would have been even less regulated (if at all) for cattle and pigs owned by individual families. For examples, see Harold C. Ernst, *Infectiousness of Milk: Result of Investigations Made for the Trustees of the Massachusetts Society for Promoting Agriculture,* (Boston: The Society, 1895); and Steve M. Blevins and Michael S. Bronze, "Robert Koch and the 'golden age' of bacteriology," *International Journal of Infectious Diseases,* 14 (2010): 744-751.
[89] Barrett, 137-138.
[90] Barrett, 138.
[91] Barrett, 138.

Social conditions also influenced Emerald Hill and Collingwood's high tuberculosis rates. As smaller communities reduced the amount of random mixing among the city's population, the likelihood of susceptible members of the community encountering infected persons increased dramatically, especially in the case of diseases where common lifestyle or environmental pressures elevated susceptibility.[92] Occupation, which tended to be similar within neighbourhoods, may have also increased risk of tuberculosis contraction or expression, as made evident by the occupational breakdown of residents of Emerald Hill and Collingwood. In his *Digest*, Thomson noted the highest rates of tuberculosis among males occurred within the labouring and artisanal classes, affecting bakers, carpenters, clerks, sailors, and labourers disproportionately, while among women, domestic servants and housewives were most affected.[93] In 1854, nearly 60 per cent of Collingwood residents were categorized as 'Clerks, artisans, and carriers', while South Melbourne housed a wharf and its composite industries, storage houses, and industrial factories.[94] These features denote occupations that include enclosed and often unsanitary workspaces that facilitated the spread of disease. Furthermore, they signify socioeconomic characteristics within these areas which might influence overall health and wellbeing, and therefore increase the likelihood of expression of the disease after initial contact. This relationship was apparent to contemporary public health activists. Dr. J.F. Agnew, Medical Officer of Health for Collingwood, for example, argued in a 1908 report that tuberculosis was the 'most important and imperative question of public health', to the

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[92] Gordis, *Epidemiology*, 25-27. As previously discussed, this is a difficult concept to quantify even by current epidemiologic standards.
[93] Thomson, *Digest*, 32-34.
[94] Barrett, 30-31, and Daley, 322-323.

neighbourhood, and attributed its spread to 'long-continued illness among the working-classes predisposed more or less to poverty'.[95]

The importance of these factors is illustrated in an examination of an inverse case. The suburb of Prahran, southeast of Melbourne proper, had one of the lowest tuberculosis rates in the city.[96] Though its population nearly equalled that of Emerald Hill (in fact, Prahran was slightly larger with a population of 9,886 in 1861 compared to Emerald Hill's 8,822), its tuberculosis rate was nearly one third that of its counterpart. A few key differences in the operation of the suburb may help to explain this disparity.

In its early years, Prahran faced similar ecological problems to Emerald Hill. Home to a diverse swamp, Prahran housed waterfowl, reeds, and a large supply of timber before 1850. Though a favourite spot of the Wurundjeri for hunting and periodic settlement, white settlers eschewed the region as a result of its 'sandy forest, the ground of which was most despised on account of its alleged agricultural inferiority'.[97] Acquired in Batman's original purchase of Melbourne, the area instead served as a missionary station until the 1840s, when the Crown sold off individual allotments within the neighbourhood. This transaction effectively pushed the Wurundjeri off the landscape, opening Prahran to residential development by European settlers.[98] These early settlers fared slightly better than their neighbours at Collingwood and Emerald Hill, as Prahran's topography was much hillier and flood plains south of the river ran further to the west than their northern counterparts (Figure 7). The reputation of the future suburb as a 'sand-blinding, slush-making, and rarely visited region', however, seemed an ill omen for its health and development.

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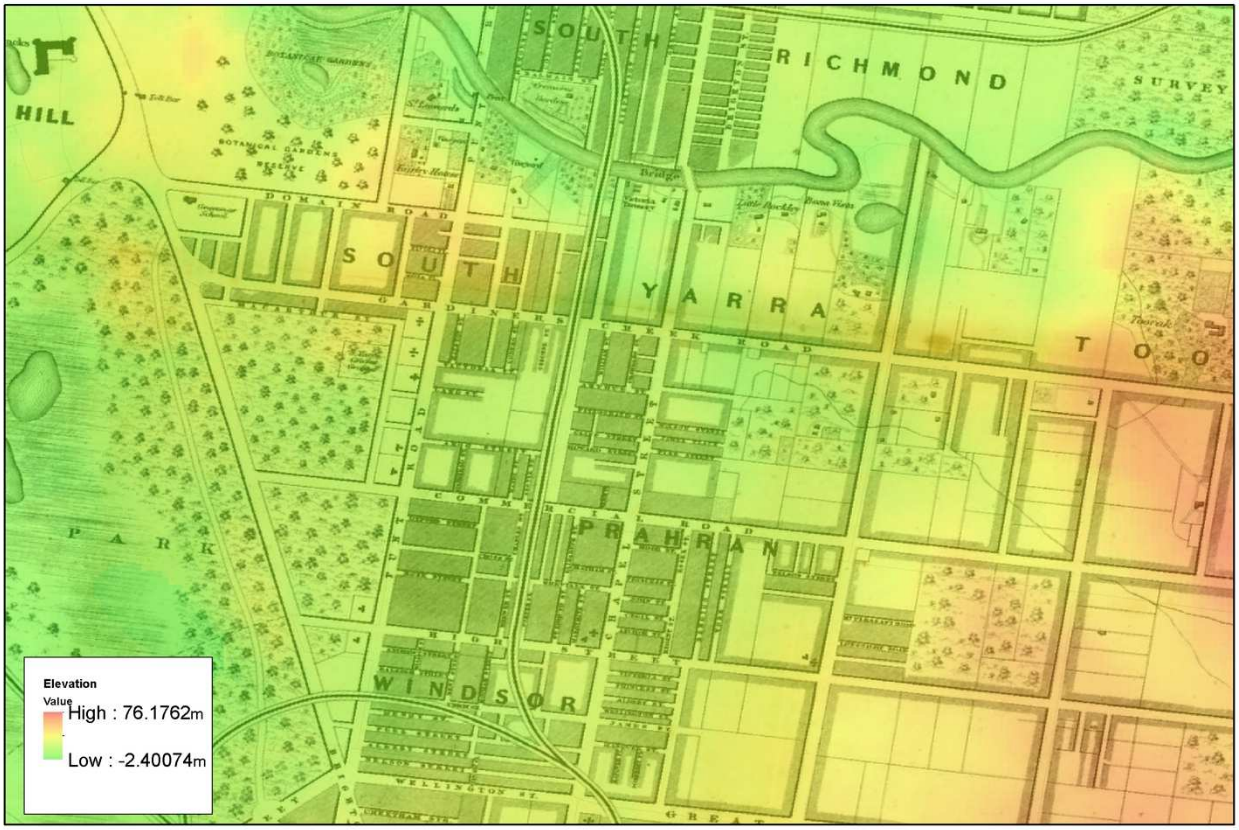
[95] Collingwood sanitation, *The Argus*, 19 May 1908. National Library of Australia Trove.
[96] Thomson, *Digest*, 18.
[97] John Butler Cooper, *The History of Prahran: From Its First Settlement to a City*, (Melbourne: Modern Print Co., 1912), 17-18.
[98] Cooper, *Prahran,* 7-10.

Figure 7. Elevation of Prahran



SOURCE: Elevation data courtesy of Geoscience Australia Digital Elevation Database (ELVIS). Map created by author.

A combination of topographical luck and community organization rescued Prahran from a fate similar to that of Emerald Hill and Collingwood. As the region expanded and residents poured in, an autonomous council formed to attend to local infrastructure. Of particular importance to this council was proper drainage. In 1857, the council urged the Central Road Board to pitch the channels on the north side of Gardener's Creek Road to follow natural water flows, and drew plans to divide the water flow that accumulated in Chapel Street into two courses, to avoid flooding.[99] By building the main drain of Prahran 'along the bed of the old water course', and insisting that drains were pitched, the suburb was able to limit flooding and

[99] Cooper, 153-154. Pitching refers to the practice of building drains on a grade or slope, allowing gravity to pull waste towards the designated areas of disposal.

the accumulation of stagnant water despite its swampy origins.[100] Additionally helpful to sanitary conditions was the favouring of brick houses in the suburb, which were less prone to rotting or dampness than the wooden structures characteristic of Emerald Hill and Collingwood. It is perhaps for this reason that by the 1890s, a relatively small number of houses in Prahran were condemned in comparison to other districts.[101]

The council also attended to other sanitary measures with particular attention. In his report on the sanitary condition of Melbourne, Gresswell noted that Prahran, along with Fitzroy and North Melbourne, removed house refuse multiple times a week while other districts maintained only weekly removal intervals.[102] Furthermore, the district forbade animals from roaming the streets, in stark contrast to Emerald Hill and Collingwood.

Divergent tuberculosis rates and in Collingwood, Emerald Hill, and Prahran demonstrate the extent to which the circumstances of an area as small as a neighbourhood influence the kinds of nonhuman life that thrive there. The environment best suited to tuberculosis – damp, densely packed and poorly ventilated houses, populated by weakened bodies, with close contact between human and bovine life, was localized, but deeply influenced by longer-term settlement patterns and the geographic transformations wrought by explosive growth. While it is difficult to answer a question of causality without rigorous modern medical technology, examining the social, environmental, and political factors that were unique to each of Melbourne's neighbourhoods allows us to understand the kind of habitat this confluence of factors might have constructed, and who (and what) may have benefitted from it.

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[100] Cooper, *History of Prahran*, 120; Gresswell, 19.

[101] Between the years 1884-1890, Gresswell listed 69 properties in Prahran in need of condemnation. See Gresswell, 6.

[102] Gresswell, 10.

Conclusion: Niche Construction and the Ecology of Melbourne

The circumstances surrounding the rise of tuberculosis in Melbourne in the late nineteenth century comprise a unique story. Melbourne was born, boomed, and some would argue, declined, over the course of the nineteenth and early twentieth century. It was a direct product of British colonial planning in the nineteenth century. With lightning-speed change came a host of new pressures upon the local ecology, which suddenly had to adapt to and interact with a series of foreign elements, from the 'portmanteau biota', to use Alfred Crosby's term, to the clearing of forests, swamps, and grasslands, and the construction of gridlike roads in their place. Far from benignly giving way to new systems of organization, Melbourne's local ecology shifted to accommodate and challenge settlers, both human and nonhuman. The result was an ecosystem that acted and reacted in ways colonists did not anticipate.

Above all, this study examines how the expansion of imperialism driven by capitalism has profound effects on biological systems – and how the victims of those biological shifts are often the very humans driving the change. Niche construction theory blends these two fragments, along with other cultural, economic, political, ecological and climatological considerations together into explanations of epidemic disease. As a concept that focuses on the power of both cultural and ecological pressures to alter environments, niche construction also provides a unique opportunity to integrate both materialist and culturalist viewpoints in one coherent framework of analysis. It allows for the specificity of the local within a more generalizing framework of disease analysis. Through it, imperial factors like town design, location, and demographic make-up, mixed with local factors like drainage patterns, lifestyle choices and housing quality, can be analysed in the context of both human and microbial success.

This framework is not, of course, without its flaws. The microbe's invisibility, and furthermore, its inability to write its own history, thoughts, and actions, results in a mix of the theoretical and empirical approaches that assume uniformity over time in microbial behaviour, characteristics of human niche construction, and the interaction thereof. The introduction of niche construction as a framework in the story of Melbourne, however, adds significance to the history of tuberculosis, both in the city and in an imperial context. A framework like niche construction can incorporate environmental pressures from both ecology and culture. Thus, the theory presents a new way of incorporating large and small scale ecological processes for a more nuanced understanding of disease in the past.

Chapter 3: A Plague on the Land, on the Sea, and in the Sewers: Ecologies of *Yersinia pestis* in Bombay City, 1890-1914

On a perfectly ordinary day in 1896, a perfectly ordinary rat went about its usual business. The rat scuttled across its dark, familiar home, and began voraciously chewing on a burlap sack, verifying with its furiously twitching nose that it was full of grain. As it felt the twine of the sack give way, the rat burrowed triumphantly into the grain, munching contentedly and filling its cheeks for later. Suddenly, the bag gave a great heave, and the rat had the sensation of flying, before slamming back down on firm ground. The rat, slightly more careful now, continued to eat. It became aware of a slight swaying around it, back and forth; time passed, and the rat climbed out of the grain bag, aware of new smells of salt, fish, and wood. The swaying stopped, and the rat heard new sounds – and discovered new smells. It scurried tentatively down a rope, onto firm land, and set off in search of food and a new home. Along for the ride, unbeknownst to the rat, was a flea carrying *Yersinia pestis*. The flea fed on the rat and its ship-bound compatriots, filling their blood with the bacteria, so that every flea that bit the rat became infected. And it was only a matter of time before one of those fleas found a new home; and another; and another.

In reality, there are several ways that fleas carrying *Yersinia pestis* might have made their way from Hong Kong to Bombay. They might have moved across several hosts, in several different events, or the bacteria might have infected several generations of flea throughout the voyage (though the average flea lifespan is about two to three months). What is known, however, is that by August of 1896, the first cases of human plague emerged in Mandvi, a mixed-class neighborhood occupied by grain merchants and other port labourers. Within months, it spread like wildfire throughout Bombay and the rest of India, carried forth by railway cars stuffed with fleeing city-dwellers and commerce from the port. By the end of the year, nearly 2,000 people in the city died from the plague; in the following year, over 10,000.[1] By the end of the epidemic, conservative estimates place the total number of deaths in Bombay City at 180,000, or roughly twenty percent of the city's pre-epidemic population.[2] India as a whole suffered over 12,500,000 deaths from the pandemic, while the rest of the world collectively only suffered 2,000,000 – the majority in China.[3] In smaller numbers, deaths and infections were registered in colonial cities like Accra, Lagos, Melbourne, Brisbane, Sydney, Glasgow, London, and Toronto, and in various cities of Europe and the Americas. In no urban center did the plague establish such a lasting and severe impact as it did in Bombay.

In this chapter, I will argue that a combination of ecological factors – namely, the composition of the rat population and its coexistence with human residents, climate, and land use change in the form of vast reclamation projects and sewer schemes that connected the islands; and sociopolitical factors, particularly overcrowding, the perpetuation of systems of inequality, and the imposition of imperial systems of sanitation management, combined to form an ideal ecological niche for *Yersinia pestis* in Bombay.

While the Bombay plague epidemic is a popular topic among historians of nineteenth-century India and colonial medicine, most works have focused on the social and cultural implications of the epidemic. David Arnold's *Colonizing the Body* contains perhaps the best-known analysis of the epidemic and the social ramifications of its management. In his book *Bacteriology in British India,* Pratik Chakrabarti, meanwhile, examines the bacteriological ramifications of the epidemic and the role of Indian doctors and scientists in characterizing the

[1] Estimated number in Ira Klein, "Urban Development and Death: Bombay City, 1870-1914," *Modern Asian Studies* 20(1986): 729; and David Arnold, *Colonizing the Body: State Medicine and Epidemic Disease in Nineteenth-Century India,* (Berkeley: University of California Press, 1993), 201.
[2] Ira Klein, "Plague, Policy, and Popular Unrest in British India," *Modern Asian Studies* 22(1988): 726-727; and Susan Scott & Christopher J. Duncan *Biology of Plagues: Evidence from Historical Populations.* (Cambridge: Cambridge University Press, 2001).
[3] Klein, "Plague, Policy, and Popular Unrest," 726.

plague in India.[4] While these approaches brilliantly dissect the role of disease in challenging or characterizing the colonial regime in India, the focus of these works are largely on the complex dynamics of groups of people and how the plague challenged them, rather than on the disease itself and how it moved in the historical landscape. Ira Klein, perhaps the most well-known historian to perform a demographic and epidemiological analysis of the plague, argues that "Bombay city and the Indian Subcontinent particularly were more susceptible to the plague because Western-style development failed to increase modern protections against the scourge while depriving the city and country of some traditional safeguards against dissemination."[5] Klein emphasizes that local ecological conditions driven by the inequality of housing and lifestyle in Bombay created an ideal environment for rats and fleas. He provides a number of important statistics on mortality from general disease, from plague, broken down by profession, and correlated to crowding. However, his larger analytic frameworks for this historical epidemiology – namely, that a vague "modernization" drove land use change in Bombay, and that the disproportionate mortality of the poor was related to a "Social Darwinism" inherent in Indian society – proves dated at best.

I will argue instead for a more nuanced understanding of structural inequality in Bombay, namely that the urban spaces where the plague exacted the largest mortality and proved most resilient were regions characterized by what Gregg Mitman deems "ecologies of injustice."[6]

[4] David Arnold, *Colonizing the Body*, 200-239; and Pratik Chakrabarty, *Bacteriology in British India: Laboratory Medicine and the Tropics,* (Rochester: University of Rochester Press, 2012).

[5] Ira Klein, "Urban Development and Death," 746.

[6] The term "ecologies of injustice" was coined by Gregg Mitman. Mitman first used the term to describe the disparity in ecological conditions in segregated or poor neighborhoods in mid-twentieth century America as compared with affluent white neighborhoods. Dawn Biehler applies this term specifically to the problem of rats and other "pests," arguing that structural violence in urban spaces create ecologies of injustice, which represent an ideal niche for rats. See Gregg Mitman, *Breathing Space: How Allergies Shape Our Lives and Landscapes,* (New Haven: Yale University Press, 2008), and Dawn Day Biehler, *Pests in the City: Flies, Bedbugs, Cockroaches, and Rats* (Seattle: University of Washington Press, 2013).

Overcrowded and haphazard housing exacerbated by informal, inadequate, or nonexistent public works in these neighborhoods, when combined with the destabilizing social aspects of mass migration into the city, facilitated the outbreak of plague. Meanwhile, once the epidemic took hold, systemic neglect, aggressive and ineffective emergency sanitary tactics, and structural violence propagated by the British colonial government allowed the epidemic to continue for decades and spread between neighborhoods, often by providing ideal environments for the plague's vectors – the brown rat and the black rat, and their parasite, *Xenopsylla cheopis*.

While the state, structure, and organization of Bombay's neighborhoods were often obscured in the colonial record, British methods of plague control rendered these features partially visible. The multiple plague committees that assembled in the wake of the epidemic undertook, as Arnold notes, a "comically thorough" campaign of sanitary intervention aimed at the individual and household.[7] Included with these structural aggressions was a regular invasion of homes by health officers who, upon determining a member of the household displayed any symptom of plague, forcibly removed residents – who, in over 90 percent of cases, never returned.[8] These methods, the result of the aggressive implementation of hygienic modernity in India, were incorporated into the British information regime.[9] Thus, hundreds of documents chronicled the spatial, quantitative, and (occasionally) qualitative features of the plague epidemic. These records provide a multiscalar perspective on the role of plague in the city, revealing the minutiae of bacteriological research, sanitation campaigns, and daily life in many

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[7] Arnold, *Colonizing the Body*, 204.
[8] James Campbell, *Report of the Bombay Plague Committee on the Plague in Bombay for the Period Extending from the 1st July 1897 to the 30th April 1898,* (Bombay: Times of India Press, 1898), 115, Medical History of British India Collection, IP/13/PC.5, National Library of Scotland.
[9] Bernard Cohn, *Colonialism and its Forms of Knowledge: The British in India*, (Princeton: Princeton University Press, 1997), 3-4. Cohn argues that he use of language (i.e., categorizing, surveying, and the description of India in English), quantifying processes, transformation of objects into "artifacts, antiquities, and art," and the meaning of cloth and clothing in communication between administrators and Indians dictated the interaction between imperial administrators and subjects in a way that was totally unique to the British Empire in India.

of Bombay's neighborhoods. While interpretation through these records must be undertaken with caution, when read carefully, they provide insights into the changing ecology of a growing urban center, and a synthesis of British sanitary ideology and local ecology which ultimately nurtured *Yersinia pestis* – and threatened the city's rodent and commensal human populations.

***Yersinia pestis, Xenopsylla cheopis* and *Rattus* as historical actors**

Yersinia pestis has captured the historical imagination more than any other microbial actor. For decades, if not centuries, historians, archaeologists, and biologists have grappled with the devastating mortality of the Black Death in Europe, tracing its extent, its relation to international trade networks, and its long-term implications for European and global development.[10] Similar projects have emerged in relation to the third pandemic, tracing its economic roots, cultural implications, and role in the development of international sanitary policy.[11] The epidemiology of plague in humans is therefore well-documented. For humans, plague transmission events result from contact with infected organisms – usually rats, but also other rodents like mice, prairie dogs, and cats. Once inside the blood stream, the bacteria replicates and accumulates in the lymph nodes, which become inflamed (hence the "bubo" typical of the bubonic plague). Without proper treatment, bacteria continue to replicate to the point of acute infection, septicemia, and death. Aside from the characteristic bubo, symptoms are

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[10] Monica Green, "The Four Black Deaths," *The American Historical Review* 125(2020): 1601-1631; *Pandemic disease in the Medieval World: Rethinking the Black Death,* Monica Green, ed., (York: Arc Humanities Press, 2015); Michael McCormick, "Rats, Communications, and Plague: Towards an Ecological History," *Journal of Interdisciplinary History* 34(2003): 1-25; Stefan Monecke, Hannelore Monecke, and Jochen Monecke, "Modelling the Black Death. A Historical Case Study and Implications for the Epidemiology of Bubonic Plague," *International Journal of Medical Microbiology* 299(2009): 582-593, DOI: 10.1016/i.ijmm.2009.05.003; McNeill, *Plagues and Peoples.*

[11] Mark Harrison, *Contagion: How Commerce Has Spread Disease*, (New Haven: Yale University Press, 2012); Mark Harrison, *Public Health in British India: Anglo-Indian Preventive Medicine, 1859-1914,* (Cambridge: Cambridge University Press, 2008); Myron Echenberg, *Plague Ports: The Global Urban Impact of plague 1894-1901,* (New York: New York University Press, 2010); Christos Lynteris, "A 'Suitable Soil': Plague's Urban Breeding Grounds at the Dawn of the Third Pandemic," *Medical History* 61(2017): 343-357.

general – fever, chills, weakness, abdominal pain, and possible hemorrhaging. Septicemic plague, in which symptoms occur with no bubo, can also occur. In these cases, plague is spread through contact with infected fluid, with a resultant 50-60 percent case-fatality rate. The third iteration of the disease is the far more dangerous (though rarer) pneumonic plague, which occurs when bacteria is inhaled directly into the lungs from particles in the air, usually the result of contact with an individual who has pneumonic plague. In these cases, respiratory distress and shock are primary symptoms, and without treatment, death occurs in 99-100 percent of cases.[12] From records of the Bombay hospitals and public health workers, it appears that the bubonic form of plague was most prevalent, though there were isolated cases of septicemic and pneumonic plague, as well.

Recently, scholars have turned their attention to the nonhuman actors responsible for the epidemic. Genetic sequencing of *Yersinia pestis* bacteria, anatomical and ecological studies of *Xenopsylla cheopis,* and studies in rat behavior, mobility, and colonization have emerged in several cross-disciplinary settings.[13] The persistence of the bacteria in places like China, Madagascar and the American West has prompted a developing interest in *Yersinia pestis* not only as a disease, but as an organism occupying a niche in a series of diverse ecosystems; it is this ecological research that this chapter seeks to incorporate into historical thinking about the

[12] Scott and Duncan, *Biology of Plagues*, 65-68.

[13] Chelsea Himsworth, "The Vancouver Rat Project," *Canadian Wildlife Health Cooperative.* 2019. http://www.vancouverratproject.com/vancouver_rat_project/home; Matthew Combs, Kaylee A. Byers, Bruno M. Ghersi, Michael J. Blum, Adalgisa Caccone, Federico Costa, Chelsea G. Himsworth**,** Jonathan L. Richardson, Jason Munshi-South, "Urban rat races: spatial population genomics of brown rats (*Rattus norvegicus*) compared across multiple cities," *Proceedings of the Royal Society B* 285 (2018): 1880; LP Angley, MC Combs, C Firth, MJ Frye, I Lipkin, JL Richardson, J Munshi-South, "Spatial variation in the parasite communities and genomic structure of urban rats in New York City," *Zoonoses & Public Health* 65 (2018): 113-123, doi: 10.1111/zph.12418; *Plague and the City,* Christos Lynteris, Lukas Engelmann, and John Henderson, eds., (London and New York: Routledge Press, 2018); *Histories of Post-Mortem Contagion: Infectious Corpses and Contested Burials,* Christos Lynteris and Nicholas Evans, eds., (London: Palgrave Macmillan, 2018); Monica Green, "The Four Black Deaths."

plague, and it is through this research that a vision of the bacteria as a historical actor can be constructed.[14]

The interaction of *Yersinia pestis* and human populations has long historical roots. Archaeologists and historical geneticists have theorized that the bacteria emerged within concentrated human settlements as early as the Neolithic Revolution, hypothesizing that it may have been responsible, among other factors, for the Neolithic decline that began in 4000 BCE.[15] The longevity of the bacteria in the historical record relates to its relationship to its hosts: fleas and rats.[16] Plague is characterized by alternating "quiescent" and "epizootic" periods in its mammalian and arthropod hosts.[17] The multi-host structure of the disease is key in *Yersinia pestis'* success. The bacteria first infect the flea (most often *Xenopsylla cheopis*), which then transmits the bacteria to its rodent host. While all mammals can become infected with *Yersinia pestis*, rodents seem particularly susceptible to the disease, with observed rats developing markedly high concentrations of bacteria in the blood (bacteremia), which increases the risk of septic shock, systemic inflammatory response syndrome, multi-organ failure, and hemorrhaging.[18] The susceptibility of rats and other rodents to high bacterial concentrations of plague facilitates epizootics of plague among rodent communities, and these epizootics often

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[14] See Amy J. Volger, Fabien Chan, Roxanne Nottingham, Genevieve Andersen, Kevin Drees, Stephen M. Beckstron-Sternberg, David M. Wagner, Suzanne Chanteau, Paul Klem, "A Decade of Plague in Mahajanga, Madagascar: Insights into the Global Maritime Spread of Pandemic Plague, *mBio* 4(2012): e00623-12, doi: 10.1128/mBio.00623-12; Michael Walsh and MA Haseeb, "Modeling the ecologic niche of plague in sylvan and domestic animal hosts to delineate sources of human exposure in the western United States, *PeerJ* 3 (2015):e1493, doi: 10.7717/peerj.1493.

[15] Aida Andrades Valtuena, Alissa Mittnik, Felix M. Key, et al., "The Stone Age Plague and Its Persistence in Eurasia," *Current Biology* 27(2017): 2683-2691, doi: 10.1016/j.cub.2017.10.025; Nicolas Rascovan, Karl Goran Srogren, Kristian Kristiansen, et al., "Emergence and Spread of Basal Lineages of *Yersinia Pestis* during the Neolithic Decline," *Cell* 176(2019): 1-11, https://doi.org/10.1016/j.cell.2018.11.005; Nicolas Rascovan, Karl-Goran Srogren, Kristian Kristiansen, et al., "Early Divergent Strains of *Yersinia Pestis* in Europe 5,000 years ago," *Cell* 163(2015): 571-582, https://doi.org/10.1016/j.cell.2015.10.009.

[16] Prairie dogs, present in the steppe of modern-day China, have been identified as a significant reservoir for plague and as a point of origin for many historical plague pandemics. See Scott and Duncan, *Biology of Plagues*, 7, 80.

[17] Rebecca J. Eisen and Kenneth L. Gage, "Adaptive strategies of *Yersinia pestis* to persist during inter-epizootic and epizootic periods," *Journal of Veterinary Research* 40(2009): 1-14, https://dx.doi.org/10.1051/vetres:200803.

[18] Eisen and Gage, "Adaptive Strategies," 2.

lead to significant population reduction in wild colonies (facilitated, in part, because of rats' tendencies to sleep in dense mischiefs).[19]

The virulence of plague in rats reduces the likelihood that the rodent may act as a reservoir for the disease; however, little is known about the mechanisms of inter-epizootic maintenance for *Yersinia pestis* – suggesting that its enzootic and epizootic niche differ.[20] The cyclical nature of outbreaks, modelled in a number of host organisms, including sylvatic rats in Central and South Asia and prairie dogs in the American West, suggests a species that lives in close proximity to the wild rat may act as a reservoir, periodically transferring the disease to coexisting rats.[21]

Rattus rattus, Rattus norvegicus and Yersinia pestis

Until recently, very little ecological research has focused on the secondary vector of plague, the city rat. Though rats are highly visible members of the urban ecosystem, their status as a "pest" organism (and their somewhat unsavory habitats) deterred mammalian ecologists from detailed studies of urban rat behavior. Studies carried out by members of the Baltimore and Boston rat projects in the 1940s and 1950s established a few important facts about the position of rats in the urban ecological system and sponsored preventive campaigns to reduce their threat to homes and peoples, but these projects were largely aimed at mitigation rather than understanding.[22] Increasing human urbanization and interest in "emerging" infectious diseases

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[19] Chelsea G. Himsworth, Kirbee L. Parsons, Claire Jardine, and David M. Patrick, "Rats, Cities, People, and Pathogens: A Systematic Review and Narrative Synthesis of Literature Regarding the Ecology of Rat-Associated Zoonoses in Urban Centers," *Vector-Borne and Zoonotic* Diseases 13(2013): 350, doi: 10.1089/vbz.2012.1195.
[20] Eisen and Gage, 7.
[21] Eisen and Gage, 8.
[22] Biehler, *Pests in the City*, 132-133.

prompted interest in the urban rat, however, and recently, projects like the Vancouver Rat Project and the Munshi-South lab have uncovered the complexities of the urban rat niche.[23]

Two species of rat dominated urban port ecologies at the end of the nineteenth century: *Rattus rattus*, or the black rat, and *Rattus norevegicus*, or the Norway (or brown) rat. True commensal organisms, the brown and black rat have become so well-adapted to anthropogenic landscapes that they are rarely found in environments uninhabited by humans.[24] While both species are thought to have evolved from East or Southeast Asia, archaeological evidence suggests they spread across the world alongside their human counterparts. While the black rat was the only dominant commensal rat species in India for most of recorded history, the advent of steam shipping in the nineteenth century introduced brown rats in large numbers into the country's major port cities. The Norway rat's advantage in size and aggressive behavior often drives black rats out of shared urban spaces; however, in the case of Bombay, studies indicate that the two species coexisted – albeit likely not peacefully – likely due to continued reintroduction of black rats through shipping routes from the East.

The Norway rat, or *Rattus norvegicus,* is thought to have originated in Central Asia thousands of years ago, but became a common European rat as a result of cross-continental trade. Though originally a rural pest, the Norway rat found refuge in the foundations of dense houses, alleys, and soft dirt paths that characterized European cities through the nineteenth century.[25] As

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[23] JL Rothenburger, CG Himsworth, KMD La Perle, FA Leighton, NM Nemeth, PM Treuting, CM Jardine, "Pathology of wild Norway rats in Vancouver, Canada," *J Vet Diagn Invest* 31(2019):184-199, doi: 10.1177/1040638719833436; JL Rothenburger, CG Himsworth, NM Nemeth, DL Pearl, CM Jardine, "Environmental Factors and Zoonotic Pathogen Ecology in Urban Exploiter Species," *Ecohealth* 14(2017): 630-641, doi: 10.1007/s10393-017-1258-5; JL Rothenburger, JD Rousseau, JS Weese, CM Jardine, "Livestock-associated methicillin-resistant *Staphylococcus aureus* and *Clostridium difficile* in wild Norway rats (*Rattus norvegicus*) from Ontario swine farms," *Canadian Journal of Veterinary Research* 82 (2018): 66-69.

[24] Alice Y.T. Feng and Chelsea G. Himsworth, "The Secret Life of the City Rat: A Review of the Ecology of Urban Norway and Black Rats (*Rattus norvegicus* and *Rattus rattus*)," *Urban Ecosystems* 17(2014): 157, https://doi.org/10.1007/s11252-013-0305-4.

[25] Feng and Himsworth, 150.

the Norway rat is primarily a burrowing rat, members of the species tends to occupy sewers and ground-level burrows, entering homes through cracks and holes in foundations and through sewage pipes. They often emerge at night to forage for food across a short distance. The abundance of food made available through concentrated refuse, sewage systems, and improper storage created a veritable cornucopia for this small mammal, and as a result it has become a highly successful urban organism.[26]

Black rats, or *Rattus rattus,* on the other hand, originated in the tropics, and are thought to be indigenous to India. While also responsible for agricultural damage, the black rat is adverse to ground-level harborage, and thus makes its home primarily within built structures, entering homes through holes in the roof or cracks in walls or ceilings. For this reason, they species has earned the moniker "roof rat." Though omnivorous, black rats have shown a preference for nuts, seeds, and fruit, and are frequently found rummaging in kitchens and waste containers. Black rats are slightly smaller and less aggressive than their counterparts, however, which often leads to a decline in their population in cities where the Norway rat has settled. Their slightly differing niches, however, do give an advantage to black rats in hyper-urbanized settings; studies have found that the presence of natural soil is a "strong predictor" of Norway rat populations, and that Norway rats are "uncommon in heavily built up areas of a city where there is little natural soil available for burrowing."[27]

Aside from their slight differences in area of occupation, size, and food preferences, the Norway rat and the black rat share several behavioral characteristics. Much like their human counterparts, both species of rat are classified as generalist organisms, which denotes their ability

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[26] *Ibid*.
[27] Feng and Himsworth, "The Secret Life of the City Rat," 149-162. Regino Cavia, G Reuben Cueto, OV Suarez, "Changes in rodent communities according to the landscape structure in an urban ecosystem," *Landscape and Urban* Planning 90(2009): 11-19, https://doi.org/10.1016/j.landurbplan.2008.10.017.

to adapt to a wide number of climates and circumstances. Though they possess dietary and habitat preferences, the two species are capable of surviving in environments that offer nearly any kind of food (including, occasionally, the feces of other organisms) and any opportunity for cover.[28] Neophobic as rats are, both species will establish small home ranges that are comprised of, at most, a city block, and at least a single house. Both rats are responsible for urban infestations, which frequently lead to structural damage, spoiling of foodstuffs, and, as the case below indicates, the transmission of disease. For both brown and black rats, life appears to be short and violent; average probability of death is thought to be 90-95 percent per year, driven by interspecific competition and resource limitation – though predation accounts for negligible mortality.[29]

Models of epizootic plague reveal an important characteristic of plague ecology that is often ignored in historical studies of the disease. Namely, that while rats are historically associated with the plague because of their visibility they are only secondary vectors for the disease – and often victims of it. Recent studies through the Vancouver Rat Project suggest that epizootics may alter the behavior of rats, leading to their exodus from the burrow and alterations in ranging behavior – a characteristic that may lead to increased transmission events.[30] Epizootics are among a number of factors that might disturb population size and structure in a city, including introduction of a foreign species and the institution of widespread trapping and poisoning

[28] Feng and Himsworth, 149.
[29] Feng and Himsworth, 153.
[30] Feng and Himsworth, 158; Amanda Minter, Chelsea G. Himsworth, Kaylee A. Byers, Jamie E. Childs, Albert I. Ko, and Federico Costa, "Tails of Two Cities: Age and Wounding Are Associated With Carriage of *Leptospira interrogans* by Norway Rats (*Rattus norvegicus*) in Ecologically Distinct Urban Environments," *Frontiers in Ecology and Evolution* 7(2019), https://doi.org/10.3389/fevo.2019.00014

campaigns.[31] In the case of plague, however, transmission events rely not only on the behavior and circumstances of the rat, but on its parasitic companion: *Xenopsylla cheopis*.

Xenopsylla cheopis and *Yersinia pestis*

Xenopsylla cheopis, commonly known as the oriental rat flea, is the primary vector of *Yersinia pestis* and is thought to be the most widespread flea species in nineteenth-century Bombay. [32] Successful in warm climates, the flea typically inhabits tropical or sub-tropical climates, though it is currently distributed worldwide. The rat flea can live for an average of 2-3 months, or, in favorable circumstances, for up to about 1.5 years. Females are remarkably fecund, and can lay up to 25 eggs per day for about 3-4 weeks.[33] *Xenopsylla cheopis* are niculous parasites, meaning they primarily occupy the nests of their host, which is most commonly a member of the genus *Rattus*, but often includes humans and other proximal animals.[34] Adult fleas feed on the blood of their hosts; however, unlike mosquitoes, which feed directly through the blood vessel, the rat flea punctures the skin with maxillary lacunae and feeds from the pool of blood that accumulates.[35]

Fleas first encounter *Yersinia pestis* through the feeding process. Upon entering the digestive tract of the flea, the bacteria will multiply rapidly within the flea's stomach, filling its

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[31] Michael J. Lee, Kaylee A. Byers, Christina M. Donovan, Julie J. Bidulka, Craig Stephen, David M. Patrick, Chelsea G. Himsworth, "Effects of Culling on *Leptospira interrogans* Carriage by Rats," *Emerging Infectious Diseases* 24(2018):356-360, doi: 10.3201/eid2402.171371.
[32] Joseph A. Lewnard and Jeffrey P. Townsend, "Climatic and Evolutionary Drivers of Phase Shifts in the Plague Epidemics of Colonial India," *PNAS* 113(2015), 14601-14608, https://doi.org/10.1073/pnas.1604985113; Caio Graco Zeppelini, Alzira Maria Paiva de Almeida and Pedro Cordeiro-Estrela, "Zoonoses as Ecological Entities: A Case Review of Plague," *PLOS Negl Trop Dis* 10(2016): e0004949, doi: 10.1371/journal.pntd.0004949.
[33] "Fleas," *DPDx: Laboratory Identification of Parasites of Public Health Concern*, Center for Disease Control, last modified Dec 8, 2017, https://www.cdc.gov/dpdx/fleas/index.html.
[34] O.J. Benedictow, "Plague, Historical," *International Encyclopedia of Public Health,* (London: Academic Press, 2017): 110-120.
[35] *Ibid*.

proventriculus.[36] The flea subsequently seeks a new host, and upon feeding, the blood of the new host mixes with the bacteria. The flea then vomits the undigested blood, now containing bacteria, back into the wound, infecting the host.[37]

As a parasitic organism, fleas thrive in spaces occupied by their primary host, and thus succeed in environments ideal for *Rattus*. Density of population amongst hosts, which facilitates ease of movement, and a habitat that remains largely undisturbed – older buildings with hollowed walls and gaps, docks, nests, and tall grass – are ideal. Studies on *Xenopsylla cheopis*, which began in earnest during the third plague pandemic, suggest that the flea exhibits climatic preferences like those of the *Yersinia pestis* bacterium itself; for example, studies show that both *Xenopsylla cheopis* and *Yersinia pestis* reproduce more rapidly in temperatures above 20ºC and below 40ºC, and that the ability of *Xenopsylla cheopis* to transmit *Yersinia pestis* is higher at 30 ºC than at lower temperatures.[38]

As these ecological profiles suggest, the emergence of plague requires a complex assemblage of organisms and environments. Suitable climate, an abundance of commensal rats and host fleas, and structures that encourage their survival are all required to harbor an epizootic, and thereafter, a human epidemic. Before these features become relevant, however, one crucial part of the transmission chain must exist: the reservoir.

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[36] The proventriculus is a thick, muscular part of the esophagus above the stomach of insects and other organisms.
[37] Benedictow, "Plague, Historical," 110-120.
[38] Katharina S Kreppel, Sandra Telfer, Minoarisoa Rajerison, Andy Morse, and Matthew Baylis, "Effect of Temperature and Relative Humidity on the Development Times and Survival of *Synopsyllus fonquerniei* and *Xenopsylla cheopis*, the Flea Vectors of Plague in Madagascar," *Parasit Vectors* 9 (2016), doi:10.1186/s13071-016-1366-z; M. Sharif, "Effects of Constant Temperature and Humidity on the Development of the Larvae and the Pupae of the Three Indian species of *Xenopsylla* (insecta: siphonaptera)," *Philos Trans R Soc Lond B Biol Sci.*, 233(1949):581–633; Anna M. Schotthoefer, Scott W. Bearden, Sara M. Vetter, Jennifer Holmes, John A. Montenieri, Christine B. Graham, Michael E. Woods, Rebecca J. Eisen, Kenneth L. Gage, "Effects of Temperature on Early-Phase Transmission of *Yersina pestis* by the Flea, *Xenopsylla cheopis*, *Journal of Medical Entomology*, Volume 48, Issue 2, 1 March 2011, Pages 411–417, https://doi.org/10.1603/ME10155

The Emergence of the Third Plague Pandemic

The Third Plague Pandemic emerged from the same region that all previous epidemics of plague are thought to have emerged: central Eurasia.[39] Whereas only scattered DNA evidence suggests that the first two epidemics crept along the Silk Road from Central Asia to Europe, the Third Pandemic's insidious spread from Yunnan province was documented in tin and opium routes as early as 1855.[40] The disease took a small number of human lives along major trade routes for nearly thirty years, before eventually erupting in the densely populated city of Canton, from which it spread to Hong Kong.

The protectorate of Hong Kong was the first British imperial port to experience the plague epidemic. Following the Treat of Nanking, the city's role as a foothold for the British East India Company placed it at the center of economic activity in a profitable imperial trade.[41] Subsequently, Hong Kong's population exploded in the mid-nineteenth century as labourers from rural China flooded into the city in search of work, creating an "frontier-town" environment reminiscent of other late nineteenth-century boom towns.[42] While the origins of Hong Kong's trade centered on illegal smuggling, the Taiping Rebellion, along with the promise of free trade, drew Chinese, German, British, and Indian merchants to the city.[43] Economic links between the two British colonies that became the epicenters of the plague epidemic began early in their

[39] Rasmussen et al., "Early Divergent Strains of *Yersinia*," 571-582; Andrades Valtueña, et al., "The Stone Age Plague," 3683-3691.
[40] Echenberg, *Plague Ports*, 17. Also noted in B.W. Brown, "Plague: A Note on the History of the Disease in Hongkong," *Public Health Reports (1896-1970)* 28(1913): 551-557.
[41] John Carroll, *A Concise History of Hong Kong,* (Hong Kong: Hong Kong University Press, 2007), 16.
[42] Carroll, 18.
[43] Carroll, 2-5.

respective histories; by the 1840s, John Carroll notes, nearly a fourth of all businesses in Hong Kong were Indian in origin, established by either Parsees or Muslims.[44]

As with many rapidly expanding cities, overcrowding quickly became a problem. The Hong Kong Sanitary Board noted dramatic population density in Chinese quarters of the city, where residents often occupied substandard housing with improvised roofs, hole-filled walls, and frequently, dirt floors.[45] It was in one of these districts, Taipingshan, that plague cases first came under administrative attention in the city in May 1894. The measures enacted by the Hong Kong sanitary board in the following months, which included an aggressive combination of whitewashing, destruction of property, forced removal, and quarantine, exemplified ingrained contagionist ideologies of British public health practice.[46] Over the subsequent five years, these measures became the foundational model for plague control in other parts of the eastern empire.

While the epidemic in Hong Kong first brought the plague under the attention of the British government and the international shipping community, the disease exhibited much lower mortality in the following year, and attention quickly shifted to the emerging epidemic in Bombay. Bombay was not only the first significant foothold for plague in India, but also the metropolitan space that suffered the highest incidence and mortality from disease. The urban ecology of Bombay shared key similarities with Hong Kong – export-import driven industries centered on European trade; rapid physical and demographic growth sparked by heavy investment by British financial interests; overcrowding, worsened by the physical limitations of island geography; and segregation of local and European populations – however, death rates in Bombay exceeded those in Hong Kong (Table 1) and the disease spread further into India, with

[44] Carroll, 34.
[45] Echenberg, 27-30.
[46] Echenberg, 27-30.

more disastrous consequences, than any other country.[47] The reason for the disparities between these two cities, this chapter will argue, lies in the intersection of Bombay's local ecology and its unique position with the empire, which interacted to form a hybrid ecology that constructed the perfect niche for plague.

Table 4. Annual Mortality Rates from Plague in Hong Kong and Bombay, 1894-1910.

Year	Plague Deaths Hong Kong	Plague Deaths per 1000 Hong Kong	Plague Deaths Bombay	Plague Deaths Per 1000 Bombay
1894	2552	10.53	-	-
1895	36	0.15	-	-
1896	1078	4.42	1936	2.35
1897	19	0.08	11003	13.3
1898	1175	4.67	17631	21.4
1899	1434	5.58	15713	19.12
1900	1022	3.92	14755	18.00
1901	1562	55.45	16887	20.54
1902	559	1.83	14314	17.42
1903	1249	3.92	20484	24.92
1904	493	1.44	13395	16.30
1905	287	0.78	14069	17.12
1906	842	2.56	10672	12.99
1907	198	0.60	6520	7.93
1908	986	2.63	5377	6.54
1909	108	0.25	5203	6.33
1910	23	0.05	3656	4.45
Total	13623	3.06	171615	14.92

SOURCE: Hong Kong Government Gazette, 1846-1990*,* CO 132, Records of the Colonial Office, Commonwealth and Foreign and Commonwealth Office, Empire Marketing Board, and Related Offices, National Archives at Kew.

Bombay as Synthetic Ecology

The rise of Bombay as a commercial and industrial center in the mid-nineteenth century resulted from changing economic currents on the British mainland. First, the port became a critical receiving point for goods moving west from Eastern colonies and trading partners, and

[47] Bombay experienced an overall death rate of roughly 1 in 4, based on the population on the eve of the epidemic; Hong Kong's death rate was closer to 1 in 10. Report on the Health and Sanitary Condition of the Colony of Hongkong, for the Year 1908. 17th July 1908, GA 1908, No. 14, Hong Kong Government Gazette, Hong Kong Government Reports Online, http://sunzi.lib.hku.hk/hkgro/view/G1908/622586.

for the export of manufactured goods from the British mainland to East. The expansion of manufacturing in key cities like Manchester, Liverpool, and Glasgow expanded demand for raw material; and thus, investors opened a series of cotton mills in Bombay in the 1850s. As a result, migrants from the surrounding areas – present-day Maharashtra and Gujarat most prominently – flooded the city in search of work, adding to the burgeoning population employed in the shipping industries on the Island. It is in this extreme technological, social, and physical upheaval that a new ecology, borne of a synthesis of local conditions and British imperial economic pressures – and a haven for both *Rattus rattus* and *Rattus norvegicus* – emerged.

The first major boost to Bombay's economy occurred in 1860 when the outbreak of the American Civil War effectively halted cotton imports to Britain from the United States. Faced with high demand and limited supply, Britain imported cotton from India at an unprecedented rate. Between 1862-1863, the aggregate commerce of Bombay increased 250 percent.[48] The cotton industry exploded; by 1865 there were ten mills on the Island, containing 25,000 spindles and 2,400 looms, consuming and processing more than 40,000 bales of cotton annually, and employing 6,000 workmen.[49] This number continued to increase over the nineteenth century, with a total of 70 mills on the island by 1895.[50] The overall population of the city more than quadrupled in this period, from 180,000 in 1814 to an estimated 816,000 by 1864.[51]

Bombay continued to serve as a major passageway for the shipment of grain from the East to the West throughout the nineteenth century. In the years 1875-1878 alone, India exported nearly 800,000 tons of grain to the United Kingdom, a large portion of it funneling through the

[48] Teresa Albequerque, *Bombay: A History*. (New Delhi: Promilla & Co., 1992), 15.
[49] Albequerque, 9.
[50] Samuel T. Sheppard, *Bombay*. (Bombay: The Times of India Press, 1932), 107.
[51] Albequerque, xiv. Miriam Dossal, *Imperial Designs and Indian Realities: The Planning of Bombay City, 1845-1875*, (Bombay: Oxford University Press, 1991), 22.

port of Bombay.[52] Grain was largely stored in the godowns of the Port Trust Estate, built on reclaimed land and comprising one-eighth of the land of Bombay City and Island.[53] Merchants often lived in dwelling-houses above the godowns on the estate – a feature which would receive much attention twenty years later, when the first cases of plague emerged from these houses.[54]

With the rapid influx of capital and population into the city came new aspirations for urban development, backed by several newly established reclamation companies. Investors scrambled to acquire titles and rights to fill in channels between the Islands and construct causeways to connect the rapidly expanding population to their places of work. These projects amounted to twenty-two square miles of reclaimed land, which increased the size of the island by twenty percent.[55] It is through this reclamation activity that the influence of British imperial environmental and urban management is first visible. The transformation of the seven islands of Bombay into one established a base for a series of important financial and land-based ventures including railroads, factories, and mass housing schemes. Much of this reform happened under the guidance of Sir Bartle Frere, "a keen an ardent sanitary reformer" who was appointed governor of Bombay in 1866.[56] Faced with the prospect of governing the second-largest city in the British Empire, Frere put forth a plan for urban beautification that included a series of waterworks projects, many of the dominant reclamation schemes, and the removal of the walls

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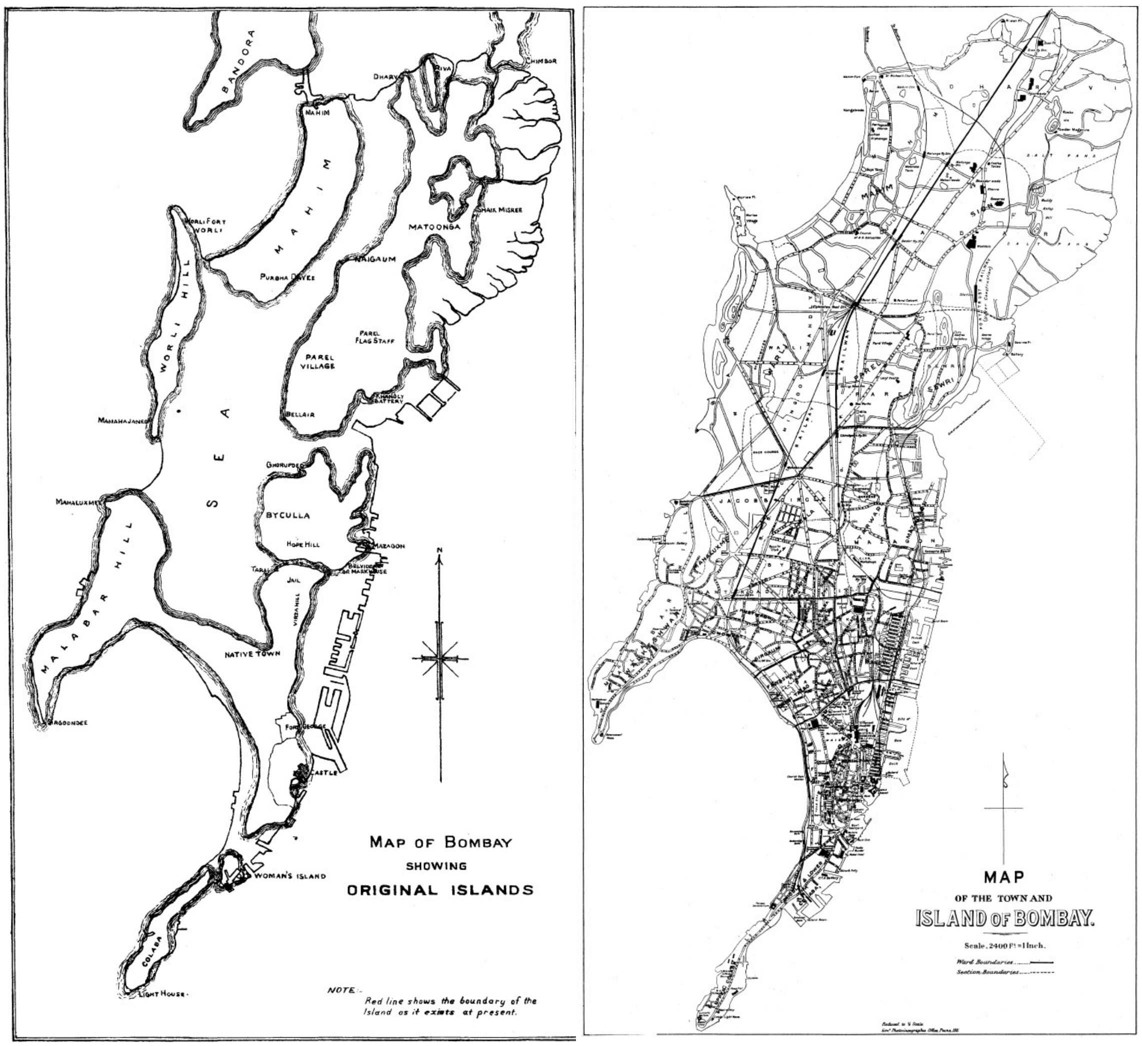
[52] Cornelius Walford, "Famines of the World, Past and Present," *Journal of the Statistical Society of London* 41(1878): 433-535, quoted in Mike Davis, *Late Victorian Holocausts: El Niño Famines and the Making of the Third World,* (London: Verso Books, 2000), 27.
[53] Sheppard, *Bombay*, 76-77; Sharada Dwivedi and Rahu Mehrotra, *Bombay: The Cities Within,* (Bombay: Eminence Designs Pvt. Ltd, 1995): 136.
[54] M.E. Couchman, *Account of the Plague Administration in the Bombay Presidency from September 1896 till May 1897*, 1897, IP/14/PC.4, India Papers Disease Collection. National Library of Scotland, Edinburgh, Scotland, United Kingdom.
[55] Ira Klein, "Urban Development and Death," 727.
[56] Sheppard, *Bombay*, 111.

from the Fort area to allow for the paving of roads.[57] Many of Frere's projects, however, fell victim to an economic crash that followed the conclusion of the American Revolution, and a subsequent dampening of demand on Indian cotton markets.[58] In spite of these economic fluctuations, improvement projects hurtled forward, supported by an increasingly powerful municipal government.

Figure 8. Bombay Island before and after reclamation projects.



[57] Cecil L. Burns, *Catalog of the Collection of Maps Prints and Photographs illustrating the history of the Island and City of Bombay*, (Bombay: The Times Press, 1918) , General Reference Collection, T36243, British Library, London, United Kingdom.
[58] Ibid, 4.

SOURCE: Charles A. Bentley, "Map No. I and Map No. II," Report of an Investigation into the Causes of Malaria in Bombay and the Measures Necessary for its Control, 1911, IP/13/SB.2, Medical History of British India Collection, NLS.

Alongside reclamation projects, the municipal government, with the encouragement of the India Office in London, turned its attention to sanitary infrastructure. By the 1870s, debates raged among colonial administrators and engineering experts over the most effective methods of sanitary management – fueled by the frequent cholera epidemics that struck British holdings in India between 1850-1880.[59] These debates are representative of Bombay's place within the British Empire at large. Sanitation experts both at home and abroad championed Bombay as a city that could represent the most cutting-edge sanitary methods, an improvement on systems promoted by sanitarians like Edwin Chadwick in large British cities in the 1850s and 1860s. These systems included, among others, large-scale, underground sewage networks that facilitated the movement of waste and refuse out of the city via water carriage.[60]

The influence of Britain's sanitary systems on plans for Bombay is evident in a letter written by Edwin Chadwick himself to the Undersecretary of State for India, which details "instructional papers which I prepared with the aid of the special engineering staff of the General Board of Health [England] for the information of the local authorities in England on the drainage of lands and of houses and towns". Chadwick claims that he would be "most happy to aid any Indian officers in such a work," noting that "Indian sanitary officers have of themselves visited some of the towns where new works have been got into action…"[61] What is more, Chadwick notes that his son was at this time under the employ of the Royal Engineers, and had participated

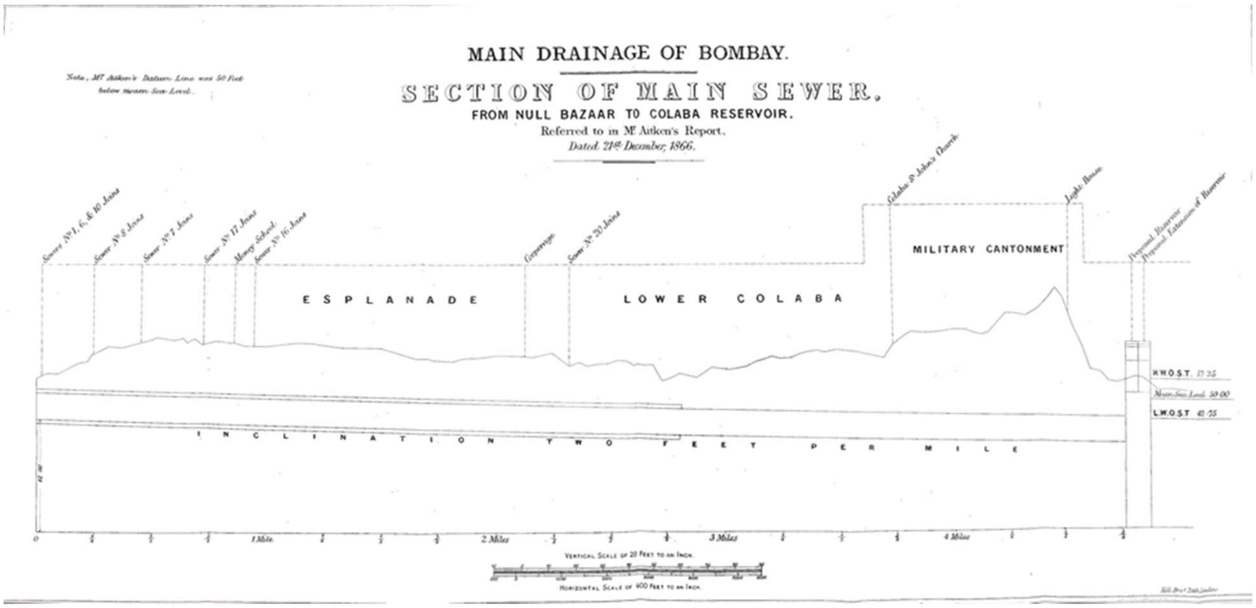
[59] *Report on Sanitary Measures in India in 1875/76 together with miscellaneous information up to June 1877, December 1877*, (London: Eyre & Spottiswoode, 1878), General Reference Collection Tab. 1281.a.2(10), India Office, Sanitary Department, British Library.
[60] Edwin Chadwick, "Sanitary Engineering in India," a Letter from Edwin Chadwick to the Under Secretary of State for India, 15th November 1876, Home Department, Sanitary Branch, No. 41-43, National Archives of India.
[61] Chadwick, "Sanitary Engineering in India," 1.

in sanitary projects across India, including reclamation projects under a Mr. Ormiston in Bombay.[62]

Other publications by sanitary experts consider the merits of individual variations on British sewerage systems for application to Bombay. Major Hector Tulloch of the Royal Engineers, for example, published a work entitled *The Drainage and Sewerage of Bombay*, which outlined a number of projects proposed to the Government of Bombay for separate systems of piped sewerage and drainage in Bombay, four-fifths of which were based directly on the London sewer system.[63] Only one of the projects out of these five suggests that drainage plans should be altered "In order to meet the particular circumstances of Bombay, with its heavy rainfall at certain periods…" advocating a large, open canal running through the middle of the island as opposed to closed, underground sewage systems.[64]

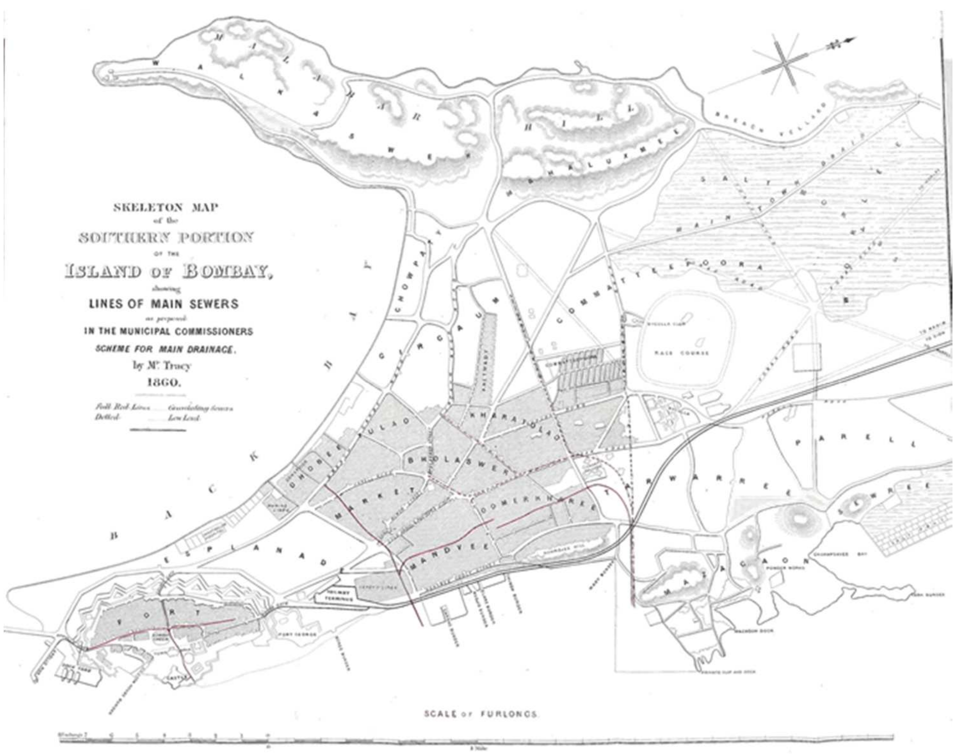
[62] Chadwick, "Sanitary Engineering in India," 1.
[63] Hector Tulloch. *The Drainage and Sewerage of Bombay*, (London: WJ Johnson, 1872), 10, General Collection, Wellcome Library.
[64] Tulloch, *Drainage and Sewerage of Bombay,* 23.

Figure 9. Diagram of the main sewer of Bombay.



SOURCE: Tulloch, *Drainage and Sewerage of Bombay*, 34.

Figure 10. Map of the main sewer lines of Bombay



SOURCE: Tulloch, *Drainage and Sewerage of Bombay*, 28.

The tension between reality and the planners' ideas for sanitation is visible in descriptions of existing sewers, doubtless established during the time of reclamation and town growth in the 1840s and 1850s, which are described as beholden to the cyclic weather patterns of Bombay. Tulloch notes that "Although the main sewer is 20 feet wide and 10 feet high, still, in heavy floods, it is not capable of discharging all the rain which falls on its drainage area. Parts of even the town itself are for many hours underwater. Of course, in the dry weather, just the opposite state of things prevails. The sewer then having to carry away nothing but the sewage of the town, it is enormously too large for the purpose."[65] By suggesting similarly closed systems of sewerage based on the London model, planners showed a continued blindness to the seasonal rain and drainage patterns of Bombay. By advocating the continuation of a closed-pipe sewage system that would run dry in the dry season and flood in the monsoon, urban planners created an unwitting niche for *Rattus norvegicus* in the city; brown rats nested in the sewers during the dry season, and fled them in the wake of monsoon floods.[66]

Debates over proper sewage and drainage systems alluded to a larger problem in the city following its economic boom: overcrowding. The city experienced further growth 1870s and 1880s as widespread famine in Maharashtra brought migrants into the city in search of food and work.[67] Dramatic population growth under constrained conditions invariably lead to crowding and the emergence of informal housing, or chawls, many of which were constructed using informal materials and insufficient space. Housing density averaged over 20 persons per house, and the size of houses – and their proximity – varied widely by community. While the Fort and

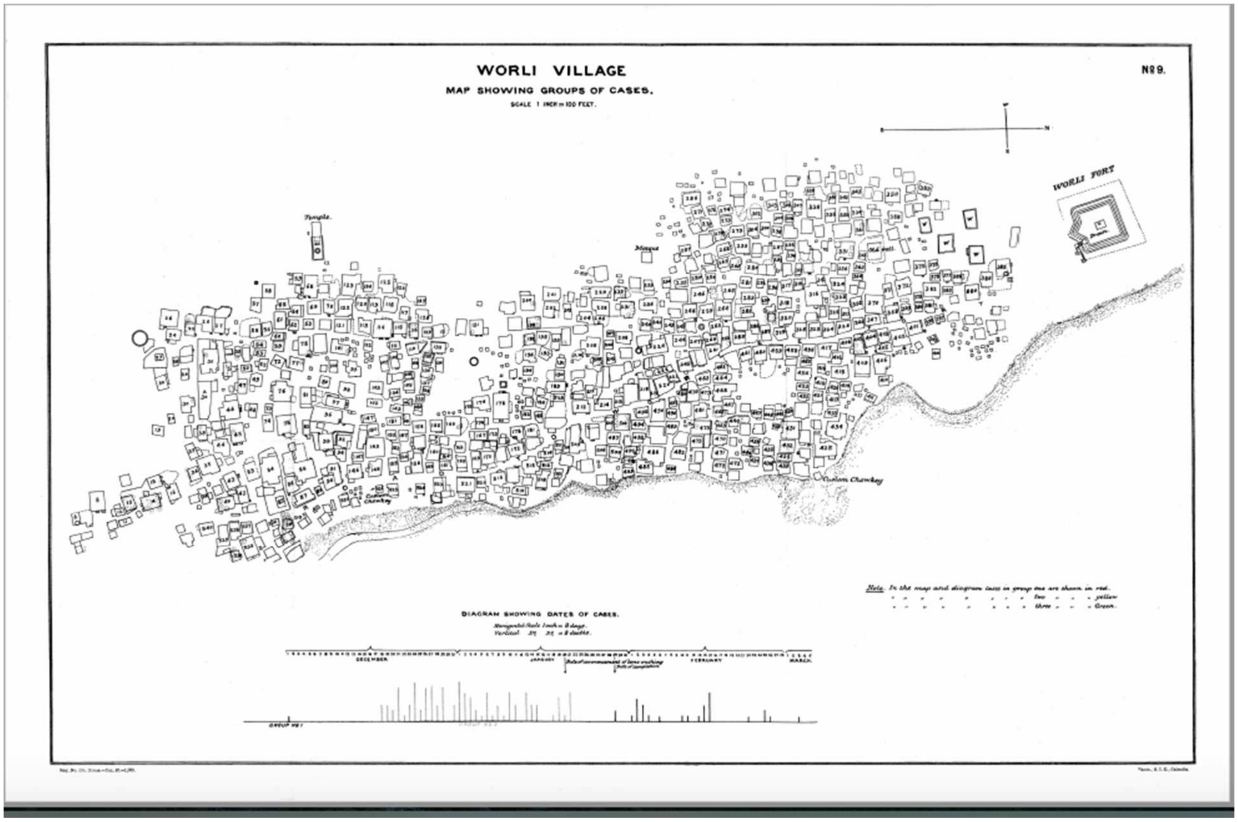
[65] *Ibid*, 10.
[66] "XXII. The Epidemiological Observations made by the Commission in Bombay City," *Epidemiology and Infection* 7(1907): 777. DOI: 10.1017/S0022172400033684.
[67] TS Weir, "Abstract of Report of the Bombay Municipality for 1889-1890 in Report on the Sanitary Measures in India for 1889-1890," 1891, General Department, vol 174, Maharashtra State Archives; Mike Davis, *Late Victorian Holocausts*, 27-28; Aidan Forth, *Barbed Wire Imperialism: Britain's Empire of Camps, 1876-1903*, (Berkeley: University of California Press, 2017), 49-60.

central town were organized on a neat grid pattern, areas beyond the European quarter grew informally. The result was a wide variation in density and organization among different neighborhoods. The Army Sanitary Commissioner, in summarizing the report of the Health Officer of Bombay, claimed, "The house crowding in the city is very great, and the contamination from it terrible."[68] In 1881, the municipal commissioner, TS Weir, also marveled over the city's crowding, noting that "The density of population in the most dense section of London is less than the density in any of the 12 most densely population sections of Bombay…in London the average is about 49 persons to each acre, in Bombay 52; but the extreme pressure in London is 222, whereas in Bombay it rises to 759 persons per acre."[69]

[68] "Memorandum by the Army Sanitary Commission on the Administration Reports of the Commissioners of the Towns of Calcutta, Madras, and Bombay for 1889-1890," in TS Weir, Report on the Sanitary Measures in India for 1889-1890, 1891, General Department, Vol 174, MSA.

[69] TS Weir, *Census of the City and Island of Bombay Taken on the 17th of February 1881* (Bombay: Times of India Press, 1883), 37, 10057.v.2, General Reference Collection, BL.

Figure 11. Worli village, on the North-west side of the city, demonstrates the informal nature of housing.



SOURCE: R Nathan, “No. 9, Worli Village. Map showing groups of cases,” in Plague in India, 1896, 1897, vol. VI, Medical History of British India Collection, National Library of Scotland, Edinburgh, United Kingdom.

With overcrowding and informal growth came systemic neglect, especially in sanitary infrastructure – a condition widely considered to be ideal for rat populations. Despite rabid arguments among sanitary engineers for new sewer systems, by the 1890s few neighborhoods had established sewage or drainage systems, because, as sanitary engineer Henry Conybeare noted, “A proper drainage system in Bombay required that the labyrinth of crooked narrow alleys in the Indian quarter be cleared and straight streets run through it,” which required significant municipal coordination – a characteristic which Bombay lacked.[70] Instead hereditary sweepers were responsible for most municipal waste removal outside of European districts of the city. Until the 1870s, residents of each street or set of streets informally organized sweepers to tend to waste and sewage. Weir, as sanitary commissioner of Bombay in the late 1870s, attempted to organize these workers municipally, with resulting organizational difficulties including an increase in strikes and complaints among the neighborhoods serviced.[71]

While ineffective in the long term, the newly established counts of municipal waste workers makes clear the burden of waste removal in relation to the size of the population. In 1877, when the population had erupted to well beyond 800,000, there were only 1,309 sanitation workers involved in removing the waste of the city, and only 988 involved in removing the solid waste.[72] Thus one person was at best responsible for the removal of the waste of 380 people. The result was a continual accumulation of garbage and waste along the streets – in other words, a rat buffet.

Improvement, Expansion, and Neglect as an Ecological Niche

It is in the attempted sanitary reform of the late nineteenth century, and its fraught interaction with local ecology and economic patterns, that a possible niche for brown and black rats and for *Yersinia pestis* emerges. The connection of the islands may have facilitated continuity among rat populations, establishing points of connection and overlapping territory in otherwise neophobic animals. The informal housing that grew on reclaimed land provided nooks, alleys, and gaps for members of the species *Rattus rattus* to establish their nests – in fact, one

[70] Dossal, 131.
[71] TS Weir, "Tenth Annual Report of the Health Officer to the Municipal Commissioner's Office" in Annual Report of the Bombay Municipality for the year 1875, 1876, vol 4, no. 482, General Department, MSA.
[72] Weir, Annual Report for the year 1875, 1876, 145.

sanitary commissioner argued, "the structure of the houses in this country seems designed to favour the continued existence within them of the black rat," as their elongated roofs offered "ideal places for shelter and breeding."[73] Bombay's role as a major shipping port for grain and for cotton further contributed to this niche; grain godowns were plentiful along the docks, and many grain merchants kept their stores on the ground floor of their homes and slept on the second, providing an easy source of food for cohabiting roof rats.[74] The effects of overcrowding, meanwhile, exacerbated by uncoordinated and often culturally deaf municipal programs, led to consistent problems with refuse and waste removal, providing ample resources for the two rat populations.

The dominant theory of cyclic epidemics draws on the idea that the disease (and rats and fleas carrying the disease) relied on a constant replenishment from shipping. A stochastic model developed by Keeling & Gilligan in 2008 using rat and flea count data from the 1905-1906 Bombay plague epidemic, however, demonstrates that large towns with a variety of subpopulations of rats coexisting across various streets and neighborhoods that are not continuous can harbor endemic plague. Die-off would decrease points of contact between rats in differing burrows, and thus reduce potential incidence for transmission. Once the relatively quick breeding time of rats allowed the population to recover, however, the fleas carrying *Yersinia pestis* would have a greater food supply, a greater opportunity for infection, and the epizootic would once again reach a rate at which humans would be at risk for infection.[75] By focusing on rat population dynamics revealed through the collection of rat infection data in Bombay, Keeling

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[73] WB Bannerman, "Measures for the Prevention of Plague," February 1906, Nos. 327-347, Calcutta Records 5, Home-Sanitary Branch, NAI.
[74] S.M. Edwardes, "Census of Bombay, Town and Island. (Vol X): Part IV History," and "(Vol XI): Part V Report, 1901," (Bombay: Times of India Press, 1901), 1-329, IOR/V/15/69, AAS, BL.
[75] Keeling and Gilligan, "Bubonic Plague," 2226.

& Gilligan conclude that short-lived, local epidemics among rats that likely produced the pattern of infection seen in humans. Through this interpretation, the graph of epizootic counts (Figure 12) is not only a depiction of a rat population cyclically experiencing an epizootic, but also evidence of an ecological relationship between rats and the city.

The major drainage controversies of the mid-nineteenth century that arose alongside improvement schemes might be considered as an important niche in light of this model. Research on rat behavior indicates that *Rattus norvegicus*, a burrowing rat by nature, prefers drier sewers for its habitat.[76] Incidence of heavy rainfall in these environments lead to the expulsion of rats from drainage pipes into environments where they are more likely to interact with humans. As brown rats tend to interact less frequently with surface rats and humans than their house-dwelling counterparts, it is entirely possible that *R. norvegicus* acted as a reservoir for the disease between human outbreaks, passing the bacteria back and forth through the exchange of fleas within and between nests under the surface, until they were flushed out by seasonal monsoon patterns. At this point, increased contact between *Rattus norvegicus* and *Rattus rattus* may have reignited the epizootic. This relationship is further corroborated by evidence from the second Bombay Plague Commission, who, through a number of studies on the behavior and habitat of both rat species, found that the epizootic often occurred in *Rattus norvegicus* up to two weeks prior to its emergence in *Rattus rattus*.[77] Thus while primary human deaths from plague arose in January-March, when lower temperatures (19-30C on average) likely facilitated the growth of *Xenopsylla cheopis* and *Yersinia pestis*, it is possible that the seasonal decline in rat population that occurred

[76] Ann-Charlotte Heiberg, Vincent Sluydts, and Herwing Leirs, "Uncovering the secret lives of sewer rats (*Rattus norvegicus*): movements, distribution and population dynamics revealed by a capture-mark-recapture study," *Wildlife Research* 39(2012): 202-220.
[77] George Lamb, *The Etiology and Epidemiology of Plague: A Summary of the Works of the Plague Commission*, (Calcutta: Superitendent of Government Printing, 1908), 15-20, IP/QA.6, Medical History of British India Collection, National Library of Scotland, Edinburgh, Scotland.

in the late summer related to the flushing of brown rats out of their seasonal habitats during the mid-summer monsoon, as the British-designed sewers and drains were overwhelmed with the extreme rain patterns for which they were poorly adapted.[78]

Plague administrators noted the way that rat behavior changed around the time of the monsoon, as well, often putting rats in direct contact with the human residents of the island. In his administrative report on animals and the plague, for example, IMS officer E.H. Aitken noted that the black rat population changed during the monsoon, claiming, "during the monsoon, they come into houses more than at other times for refuge from the rain. During last monsoon, I caught twenty-two in my house, which is about a quarter of a mile from any other human habitation."[79] Aitken's anecdote illustrates the role of weather patterns in increasing likelihood of interactions between humans and rats. Such an explanation might also shed light on the difference in scale between the Bombay and Hong Kong epidemics; while Bombay had an established sewer system across large sections of the town to provide shelter to a large brown rat population, in Hong Kong, the public sewage project only commenced in 1889, five years before the epidemic, and continued through the end of the nineteenth century.[80]

Given the island-wide infestation of rats and evidence of disease patterns that allowed plague to remain endemic in the city in inter-epizootic periods, the question of human plague re-emergence in Bombay becomes a question not of number, but of opportunities for contact. Given the urban niche and habitat preferences of the black and brown rat, these opportunities for

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[78] Himsworth et al., "Rats, Cities, People, and Pathogens," 353.
[79] E.H. Aitken, "Materials Received for Compiling the Plague Administration Report on Animal and Plague, 1899," vol 711, no 639, General Department, MSA.
[80] Report of the Acting Sanitary Superintendent for 1893, Hong Kong Government Gazette, 21st July 1894, CO132/35, National Archives at Kew, London, England; Report of the Director of Public Works for 1894, Hong Kong Government Gazette, 25th May 1895, CO 132/26, National Archives at Kew.

contact differed across the diverse, haphazard city – a feature of the epidemic evident in the qualitative and quantitative data collected through health surveillance.

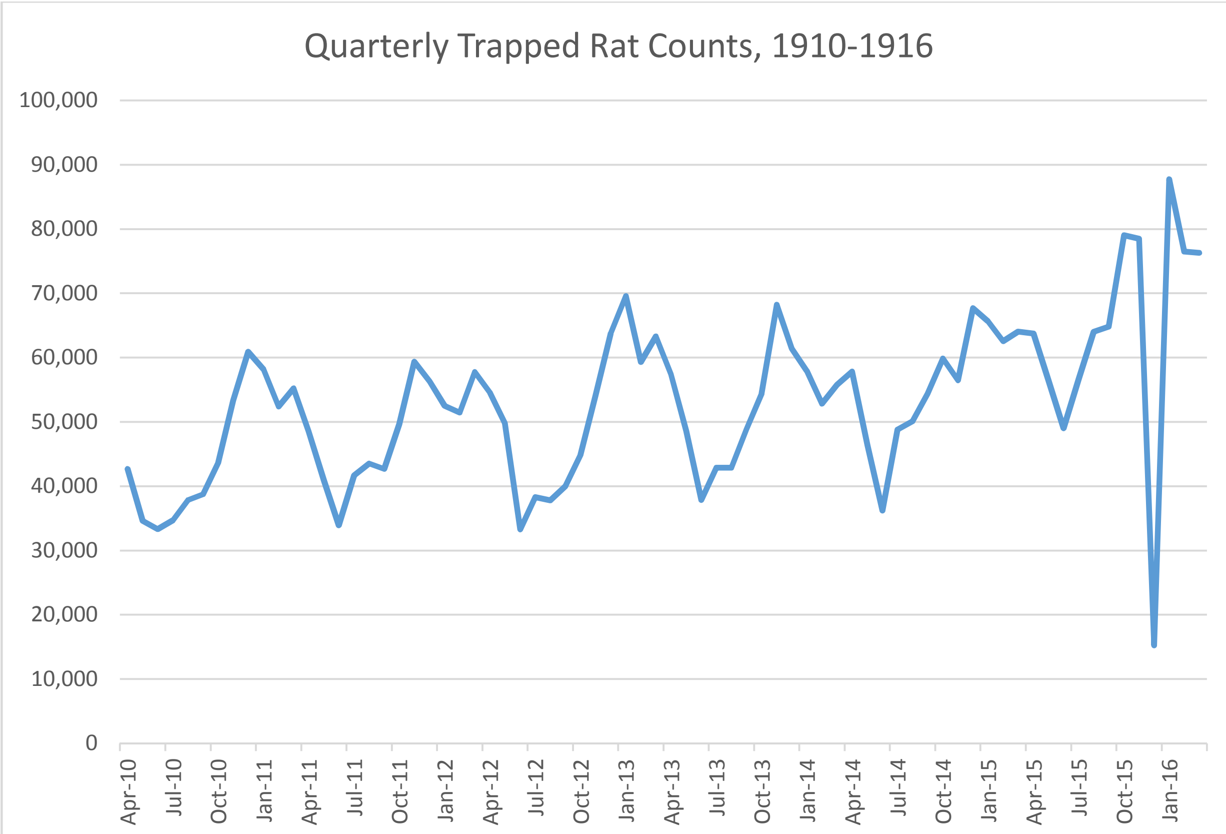
The Third Rat Plague Pandemic

While Bombay seemed like a paradise to the burgeoning rodent population – a large, urban agglomeration with abundant nooks and crannies afforded by informal housing; a steady food supply from numerous grain silos; poor sanitary infrastructure that provided a steady supply of food for those outside the docks; and sewers to scurry within and build nests safely – the city of Bombay quickly turned to a hell for its rodent residents as *Yersinia pestis* arrived. Fleas, an omnipresent annoyance of the species, quickly turned relentless, biting the rats more furiously as bacterial biofilm filled their flea stomachs, leading to starvation and desperation for a blood meal.[81] Within days, affected rats would die of kidney failure or septicemia, the bacterial load in their blood so high that their organs simply ceased to function; the fleas, desperately hungry, would migrate to the nearest host, until entire families of rats were wiped out. While the highly disorganized nature of the imperial port cities was a haven to rats, it also provided salvation to subpopulations; as large swathes of rats died, the many obstacles separating these rodents – roads, sewers, trains, or marshy topography – might have stopped the all-out extinction of the island population, and allowed it time to recover.[82] The strongly cyclical nature of rat counts, visible in the graph below, suggests significant annual die-off of the population. The cycle of recovery and die-off correlates strongly with the re-emergence of human plague, which peaked around late winter – roughly the same time that the rat population appears to decrease.

Figure 12. Annual quarterly trapped rat counts, 1910-1916.

[81] Rebecca J. Eisen and Kenneth L. Gage, "Adaptive Strategies of *Yersinia pestis* to Persist During Inter-Epizootic and Epizootic Periods," *Journal of Veterinary Research* 40(2009): 1-24, doi: 10.1051/vetres:2008039

[82] *Ibid*, 21. MJ Keeling and CA Gilligan, "Bubonic plague: A metapopulation model of a zoonosis," *Proceedings of the Royal Society of London B* 267(2000): 2219, doi:10.1098/rspb.2000.1272.



SOURCE: JA Turner, Executive Health Officer's Report for Bombay, 1910-1925, IOR/V/25/840/24, India Office Records, AAS, BL.

While it is impossible to know the exact carnage of the epizootic among rats, one thing is certain: by the time the first few cases of the plague were identified among human residents of the city of Bombay, it was already too late for the rats. Dr. Accacio Viegas, who reported the first deaths from plague to the Sanitary Committee of the Bombay Municipal Corporation in September, 1896, described observations, by the residents of the *chawl* where the original cases were found, of a great mortality among the rats in the area. The interviewed friends and family, who were employed in grain stores near the docks, described how the rats wandered out of their nests in daylight, dying conspicuously in the streets.[83]

[83] M.E. Couchman, *Account of the Plague Administration in the Bombay Presidency from September 1896 till May 1897*, (Bombay: Government Press, 1897), 130, IP/13/PC. 4, MHBIC, NLS.

While rats were not widely acknowledged as a vector in the plague for several years, members of the Plague Research Committee showed early attention to the visible relationship between the rodents and human plague cases. James Campbell, head of the newly formed Bombay Plague Committee in 1897 mused, "Whether rats bring Plague from infected to uninfected localities, or whether local rats are the first victims to existing local infection, has not been determined. It seems almost certain that, in some instances, rats suffering from plague had moved in numbers to a fresh locality, and have brought Plague among the people there. In other instances the evidence seems to show that local rats sickened in consequence of the introduction of infection by human agency."[84] However, many showed more reluctance to grant legitimacy to the theory of rat-human infection pathway, not least because of its potential to undermine existing sanitary theories of public health.[85] Captain H. Smith of the Indian medical Service, argued that "if rats have a hand in the matter, we may as well capitulate at once; for we cannot…control the movement of rats."[86]

Within three months of the first identified cases of plague in Bombay, the disease had claimed over a thousand human lives, resulting in outright panic and the flight of nearly 390,000 people from the city into the countryside.[87] The Bombay Plague Committee, alongside the newly-established Plague Research Committee, initiated widespread campaigns to discover the extent and demographic patterns of the human epidemic. The breadth of collected sanitary information illusrates the extent to which plague researchers and health officers attempted to fit the newly discovered *bacillus* (Kitasato Shibasibūro and Antoine Yersin had isolated *Yersinia*

[84] RWS Lyons, Bombay Plague Research (Lyons) Committee, 1896-1897: Reports by Members, 1897, IOR/V/26/856/2, AAS, BL.

[85] Nicholas H.A. Evans, "Blaming the Rat? Accounting for plague in colonial Indian medicine," *Medical Anthropology Theory* 5 (2018): 15-42, doi.org/10.17157/mat.5.3.371.

[86] H. Smith, Materials Received for Compiling the Plague Administration Report on Animals and Plague, 1899, vol 711, no. 639, General Department, MSA.

[87] Couchman*, Account of Plague Administration in the Bombay Presidency*, 66-77.

pestis in Hong Kong in 1894) into a sanitarian framework. Frequent attention to light, ventilation, and overcrowding abounded in houses in which the first cases emerged. Prevailing theories for transmission included via personal contact with soil, or contact of diseased or rotten grain with the soil – though General Lyon emphasized that the majority of people suffering from the plague in the early days were not grain merchants (though the first discovered case was employed thus), but dock laborers, in contact with goods moving from China into the port.[88]

The Plague Committee immediately enacted a series of aggressive, top-down sanitary measures on the various neighborhoods of Bombay, including flushing out sewers with seawater, the (often forcible) removal of plague victims to hospitals, and the evacuation and burning of the property of those who had suffered from the plague, among other atrocities.[89] In its reformed iteration (which was still guilty of similarly aggressive policies), the Committee established a more organized approach to documenting plague which included dividing the municipal area into ten districts to be managed by a District Medical Officer of Health.

Detailed statistical reports produced through these entities reveal a thorough attempt at both documentation and explanation. The Bombay Plague Committee (which, unlike the Plague Research Committee, constituted a number of government officials in the general department of the Bombay municipal government) employed hundreds of Indian and European workers to collect information about the location, environment, occupation, and household size of victims. These administrative statistics, though they contained their own biases and misrepresentations, demonstrate a meticulous categorization and quantification that reveals distinct patterns of

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[88] Lyons, *Report of the Bombay Plague Committee*, NLS; The prevalence of soil theory of transmission is explored in Christos Lynteris, "A 'Suitable Soil'," 343-357.

[89] The best treatment of this topic related to imperial biopolitics can be found in David Arnold's *Colonizing the Body*, 200-239.

infection over time. When combined with theoretical frameworks that depict rat, flea, and plague dynamics in urban environments, a characterization of the epidemics and its victims emerges. Quantitative and qualitative analysis of the ten plague districts reveal inequalities in disease burden by neighborhood. At first glance, the five neighborhoods suffering the highest mortality rates from plague appear disparate – they were largely in different wards, had varied population sizes, and somewhat varied population distribution. However, closer scrutiny reveals a textured and more intimate story of who suffered from the plague – and often, narratives that align with theories of ecological injustice.

Table 5. Population size and density per house by ward, (1897).

Ward	Population	No. of Houses	Density per House
A	64,819	3,480	19
B	152,377	5,483	28
C	206,372	7,455	28
D	97,329	6,096	16
E	180,423	7,257	25
F&G	98,402	9,625	10

SOURCE: Campbell, "Report of the Bombay Plague Committee – [Plan No.1]" 245, vol 2, NLS

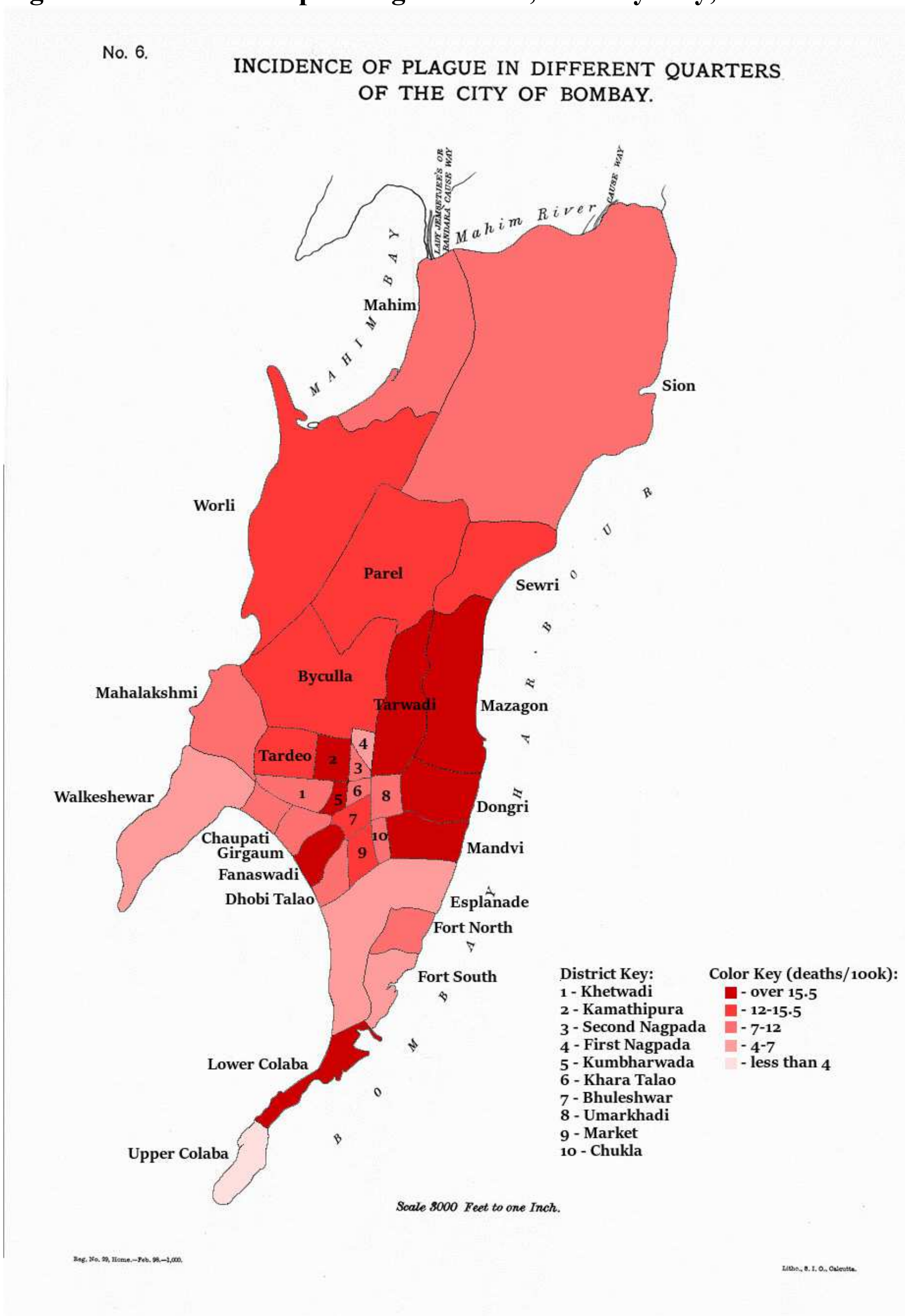
Table 6. Average plague mortality as a percentage of total mortality in the City of Bombay, 1896-1911 (based on data for the years 1896-1899, 1903-1911).

Sections	Ward	Total Death	Total Pop (1901	Average Death Rate per 1000
Upper Colaba	A	213	8956	1.98
Middle & Lower Colaba	A	2606	13023	16.68
Fort, Southern	A	192	3309	4.84
Fort, Northern	A	3360	32047	8.74
Esplanade	A	833	10398	6.68
Mandvi	B	6263	31402	16.62
Chukla	B	3002	24384	10.26
Umarkhadi	B	5750	48481	9.88
Dongri	B	5155	25778	16.66
Market	C	5098	28415	14.95
Dhobi Talao	C	5008	39950	10.45
Fanaswadi	C	3804	16354	19.38
Bhuleshwar	C	5716	38363	12.42
Khara Talao	C	3167	27544	9.58
Kumbharwada	C	5255	23161	18.91
Khetwadi	D	3734	27160	11.46
Girgaum	D	3642	27000	11.24
Chaupati	D	1238	11512	8.96
Walkeshwar	D	847	10563	6.68
Mahalakshmi	D	2264	18092	10.43
Mazagon	E	5449	27933	16.26
Tarwadi	E	3855	18460	17.40
2nd Nagpada	E	2536	18591	11.37
Kamathipura	E	6107	26706	19.06
Tardeo	E	3805	20958	15.13
Byculla	E	10578	57646	15.29
1st Nagpada	E	802	10577	6.32
Parel	FG	5595	33390	13.96
Sewri	FG	1627	9294	14.59
Sion	FG	2770	25433	9.08
Mahim	FG	3442	27386	10.47
Worli	FG	7383	45588	13.50

SOURCE: Reports of the Health Officer of Bombay, 1899-1910, 1897, V/25/840/23, India Office Records, AAS, BL. Census data has been used in place of 1901 census data for five neighborhoods – Fort North, Dhobi Talao, Bhuleshwar, Girgaum, and (Continued on next page)

Chowpatty, due to flight from these neighborhoods in response to plague outbreak at the time of census-taking. See Census of Bombay 1901, Section V – Report, AAS, BL.

Figure 13. Death Rates per Neighborhood, Bombay City, 1896-1911



SOURCE: James Campbell, *Report of the Bombay Plague Committee on the Plague in Bombay for the Period Extending from the 1st July 1897 to the 30th April 1898,* vol. 2, (Bombay: Times of India Press, 1898), IP/13/PC.5, Medical History of British India Collection, NLS.

Plague at the Intersections of Race and Class

The relationship between density, social ecology, and physical geography and the plague is best explored at the level of the individual neighborhood. The five districts in which mortality was highest – Mandvi, Dongri, Khumbarwada, Kamathipura, and Tarwadi demonstrate the complex series of factors that contributed to exposure and death from plague in their similarities and differences. Meanwhile, the European town in Fort South, which was both structurally and epidemiologically distinct from its surrounding wards, provides an inverse case.

Table 7. Ward, death rate, and occupational profile per five highest mortality districts and the European district.

Neighborhood	Ward	Density per House	Death Rate per Thousand	Occupational profile
Fort South	A Ward	8.42	4.85	Domestic S (38)
Mandvi	B Ward	23.09	16.68	Manufacturing & Supply (44)
Dongri	B Ward	28.76	16.62	Unskilled Labour (49)
Khumbarwada	C Ward	42.90	18.91	Manufacturing and Supply (40)
Kamathipura	E Ward	23.42	19.06	Manufacturing and Supply (47)
Tarwadi	E Ward	18.36	17.40	Manufacturing and Supply (48)

SOURCE: Meera Kosambi, *Bombay in Transition,* (Stockholm: Almqvist and Wiskell International, 1986), 72; TS Weir, Reports of the Health Officer of Bombay, 1896-1909, IOR/V/25/840/23, AAS, BL.

A district just north of the European town, Mandvi was one of Bombay's largest shipping centers, where the city's shipping activity was concentrated. The neighborhood was among the most crowded in the city; it housed a population of 37,295 in a total of 1,615 houses on the eve of the epidemic, with documentary descriptions detailing crowded chawls and narrow alleys.[90] It was also among the most important commercial areas in the city; sugar and ghee industries concentrated in the ward, while its Eastern boundary was occupied with the city's main docks, alongside granaries, and major warehouses.[91] The neighborhood's occupational characteristics were largely concentrated around activity on the docks; it housed a number of large grain silos, and many of its residents were involved in grain sales and shipping – the perfect reliable food source for hungry urban rats.[92] Occupational analysis suggests that the neighborhood was of mixed income: 965 were employed as grain and pulse dealers, 1,465 as general merchants, and 751 as indoor servants. Several of these industries may have increased contact between humans and rats – for example, grain and pulse dealers, general merchants selling food, and indoor servants in houses suffering infestations would have risked exposure to rats.[93] Those employed on the docks were also at risk: the first 19 cases notified in the area came from a chawl occupied by 600 people, all of whom were described as "coolies employed in grain stores in the neighborhood of the docks."[94]

Housing appeared to be a key exposure risk to plague in the neighborhood. While there were few chawls, dwelling-houses with godowns represented the one of the most common forms of accommodation. Of 1,269 building types in the neighborhood, 409 of them were dwelling-houses with godowns on the ground floor and occupants on the first or second floor.[95] These

[90] PCH Snow, *Report on the Outbreak of Bubonic Plague in Bombay, 1896-1897*, (Bombay: Times of India Press, 1897), 86, V/27/856/7, India Office Records, Asian and African Studies Collection, British Library.
[91] Dossal, 20.
[92] Edwardes, "Census, Part V - Report" 79-80.
[93] Edwardes, "Census, Part VI - Tables," 140-146.
[94] HM Fernando, "Report on Bubonic Plague in Bombay," April 1897, No 119-120, Simla Records, Home-Sanitary[Plague], NAI.
[95] Edwardes, "Census, VI - Tables," 140-146.

grain storage facilities increased points of contact between humans and rats, as, according to the Bombay Plague Committee, these spaces were largely "store—rooms on the ground floors which are infested with rats."[96] The extent of the epizootic in the neighborhood was such that E.H. Hankin, the President of the Bombay Plague Committee, noted in his epidemiological report that "Dead rats used to be observed in various districts as the disease progressed, but never in such numbers as were seen in Mandvie."[97]

While Mandvi's struggle with plague suggests that industry and caste together may have influenced the success of plague in the neighborhood, examination of wards in which grain shipping did not dominate also suggests that infrastructure and class had a significant impact on mortality from the disease. In the initial years of the epidemic, Mandvi's neighboring district, Dongri, appeared to escape the fate of its counterpart – P.C.H. Snow observed in 1897, "It is remarkable that it was not higher, as the two districts are separated by a narrow road, on the North side of which is Dongri, on the south side of which is Mandvi with high and crowded houses on either side."[98] However, the "poor, Marathi-speaking district," largely populated with unskilled laborers, many of whom were employed on the docks or in the railyard, became one of the major centers of mortality from the epidemic by the turn of the twentieth century.[99] Unlike its neighboring district, Dongri had no stock in grain storage, and did not share Mandvi's godown abundance. In fact, according to the census, the vast majority of residents of the neighborhood lived on the ground floor, and "Buildings with a ground floor only…are indeed so numerous that Dongri easily heads the list of the B ward sections for the highest number of ground-floor

[96] Cambpell, *Report of the Bombay Plague Committee*, 52, NLS.
[97] EH Hankin, "On the Epidemiology of Plague," *Journal of Hygiene* 5(1905): 74.
[98] Snow, *Plague in Bombay*, 150.
[99] Kosambi, *Bombay in Transition*, 76.

dwellings."[100] A primarily residential neighborhood framed by a railyard and the Princess docks, Dongri's land use structure paints the picture of a low-income neighborhood. Between ground-floor dwellings, 59 of which were labeled chawls and 404 designated dwelling-houses. Most residents lived in "humble tenements," and were employed in the category of "general labor." Out of 12,588 documented workers in the neighborhood, 5,481 were thus designated.[101]

Given its proximity to the docks, it is perhaps unsurprising that B ward as a whole also contained by far the highest density of rats based on sampling by the plague committee over a 5.5-year period between 1910 and 1916, when an estimated 1.3 million rats were caught in randomly placed traps.[102] Given the size of the district, this amounted to roughly 1,900 rats per acre – and this was just the number trapped, which can only be a small fraction of the total number of living in the area. These numbers, though they have their limitations, suggest an overwhelming population of rats in the area, and many thousands of opportunities for daily contact between residents and their cohabiting pests.

Khumbarwada, in C ward, shared a similar structural and demographic profile to Dongri. Described in the census as the "chal area *par excellence* of C ward," the area contained 98 chawls and 627 dwelling houses. According to the local census administrator, "The majority of the Kumbharwada population resides on the ground and first floor," with only a small section in which the second-story population exceeds the ground floor population. Much like Dongri, the primary labor form in the district was unspecified general labor, with 4,545 persons employed in the category out of a total laboring population of 16192.[103] In fact, "the mass of the Kumbharwada population is terribly poor. There is not a single community, of which the vast

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[100] Edwardes, "Census VI – Tables," 83, 192.
[101] *Ibid*, 192.
[102] Compiled from Turner, "Quarterly Reports of the Health Officer of Bombay, 1909-1925."
[103] Edwardes, 154.

majority does not live in holdings of a single room; and the percentage of the total population, so domiciled is higher in this section than any other part of C ward," according to Edwardes.[104] Analysis also suggests that crowding within these single-room tenements was high, as the neighborhood's housing density, at over 42 persons per house, was the highest in the city.[105]

Fanaswadi, also located in C ward, tied for highest plague mortality over the twelve-year period examined. While the district held the smallest population in its ward, housing density for the area was relatively high; the neighborhood supported 27 chawls and a number of thatched huts. However, crowding per room was relatively low, with fewer instances of more than 20 persons occupying one room occurring in the neighborhood than in others in the ward. Structurally, residents tended to live overwhelmingly on the ground or first floor, with a total of 11,173 out of 14,199 occupying this position, and half total occupying the ground floor only.[106] This distribution led Stephen Edwardes, the head of the 1901 census, to conclude, "The population resident on upper floors is smaller in Fanaswadi than in any other of the recognized divisions of C ward."[107] While the neighborhood was religiously diverse – it was home to four churches, four masjids and ten temples, and housed Christians, Parsis, Hindus, Jains, and Jews – the majority of the population appeared to be relatively poor. Nearly a quarter of documented workers were employed in unskilled labor, which dominated all forms of employment (1895 out of 9216 workers).[108]

Kamathipura, a district in E ward which shares the highest overall all plague mortality over the twelve-year period examined with Fanaswadi, was also described as a poor neighborhood. A largely residential district, the area was populated with "dwelling-houses of the

[104] Edwardes, 91.
[105] Edwardes, 88.
[106] Edwardes, 220.
[107] Edwardes, 88.
[108] Edwardes, 147.

poorer type," of which nearly forty were considered "unfit for human habitation." [109] Roughly half of the population lived on the ground floor, and "almost entirely" in one-roomed tenements, as, Edwardes notes, "The population of Kamathipura is poor."[110] However, among districts in E ward, the area also had the largest number of people living in second or third floor tenements – though this number amounts to only one-fifth of the population in the entire neighborhood.[111] Occupationally, the neighborhood had a large number of sanitation workers, amounting to 798 persons out of 16,677 documented workers, though the highest employment was in general unskilled labor (3,267 persons) and cotton mill work (1,693 persons).[112] The neighborhood also had the highest density per acre of human residents of all neighborhoods, at 546.7 persons per acre.

Tarwadi, located in E ward, was more textured in its land use. The area housed several cotton mills – more than its surrounding districts of Mazagon and Byculla, and contained dispensaries and four hospitals, as well as a number of stables. Perhaps unsurprisingly, the majority of the population appeared to be employed in the cotton industry, with cotton mill employees making up 3,765 persons out of a total of 10,833 documented workers. General labor formed the second highest proportion of workers, with 1604 persons identified under this heading.[113] This combination of laboring populations suggests a mixed, lower-income neighborhood. Housing was somewhat mixed; like Khumbarwada, Tarwadi held more chawls than any other section in its group, but also contained a number of 'Cadjan' huts, described as "small tile huts" occupied by poorer class, as well as a small number of bungalows and other

[109] Edwardes, 101.
[110] Edwardes, 101.
[111] Edwardes, 222.
[112] Edwardes, 158-163.
[113] Edwardes, 163

dwelling-houses.[114] Like Dongri and Khumbarwada, however, the vast majority of dwellings only consistent of a ground floor, with 909 of 1155 houses listed as ground-floor only. Also similarly, most residents lived in tenements of a single room.[115] However, the neighborhood does seem to include a significant middle class population, with nearly 1500 residents living in tenements with six or more rooms, and average density per house was lower than other high-mortality neighborhoods oat roughly eighteen persons per house.[116]

A comparison of plague rates in these six neighborhoods suggests a series of risk factors that may have influenced plague incidence and mortality rates in differing parts of Bombay. First, ground-floor and first-floor occupancy dominated in Dongri, Khumbarwada, Kamathipura, Fanaswadi and Tarwadi. Norway rats in particular are likely to enter the home on the ground or first floor, given their relative neophobia and limited ranging distances.[117] The relationship between lower-floor occupancy and plague attacks and deaths was noted early on in the plague commission; TS Weir, in the municipal commissioner's Report on the Outbreak of Bubonic Plague in Bombay, noted that in the first two years of the epidemic, 1,062 out of 2,544 attacks and 794 out of 1,936 deaths from plague citywide occurred among ground-floor occupants, and 774 attacks and 612 deaths among first floor occupants.[118] While at the time these observations served as evidence for the soil theory of plague pathology, such spaces also provide easy access to both black and brown rats, and therefore may have heightened exposure to plague. In the neighborhood in which ground-level occupancy did not dominate, Mandvi, housing structure similarly appeared to contribute to points of contact with rats, fleas, and *Yersinia pestis*. In Mandvi, nearly half the residents lived directly above grain storage areas, many of which were

[114] Edwardes, 108-109.
[115] Edwardes, 108-109.
[116] Edwardes, 108-109.
[117] Himsworth et al, "The Secret Life of the City Rat," 156.
[118] Snow, *Report on the Outbreak of Bubonic Plague in Bombay, 1896-97*, 136.

known to be infested with rats. Housing structure may have played a significant role in Fanaswadi's high plague count, as thatched-roof housing provides easier access to roof rats, has been shown to harbor a higher concentration of domestic fleas in some cases of plague.[119]

Tendency towards lower-floor occupancy might also account for difference in plague continuance in Bombay and Hong Kong, despite their concurrent infection timelines. While lower-class residents of Bombay often lived in ground-floor or first-floor housing, Hong Kong's laboring population often occupied chawls of four or more stories.[120] Basement-level occupancy was also evacuated in large parts of the city after the outbreak of plague, with concern that basements were "unhealthy, and more or less infested with rats, and never dry during the rains."[121] Thus, a larger portion of the population in crowded districts living on higher floors, in combination with removal of persons from basements, may have limited contact.

Single-room occupancy was also prevalent in all five wards. In a single-room setting, occupants would likely live in spaces where food was routinely stored, and therefore encounter rats searching for goods – a relationship documented as a risk factor in several other publications on plague transmission.[122] Less clear in its relationship to plague mortality is the question of class; while four of the six neighborhoods with the highest plague mortality (and ten of ten total neighborhoods with plague mortality of greater than 14 per thousand) had general labor as the largest single occupation and manufacturing and shipping disciplines as the largest group of

[119] Anne Laudisoit, Herwig Leirs, Rhodes H. Makundi, Stefan Van Dongen, Stephen Davis, Simon Neerinckx, Jozef Deckers, and Roland Libois, "Plague and the human flea, Tanzania," *Emerging infectious diseases*, *13*(2007): 687–693, doi:10.3201/eid1305.061084.

[120] Osbert Chadwick, and William John Simpson, *Report on the Question of the Housing of the Population of Hongkong*, (Hong Kong, 1902), 9, BS7/46, General Reference Collection, British Library.

[121] Chadwick and Simpson, *Report on Housing of Hongkong*, 7.

[122] Rebecca J. Eisen, Katherine MacMillan, Linda A. Atiku, Joseph T. Mpanga, Emily Zielinski-Gutierrez, Christine B. Graham, Karen A. Boegler, Russell E. Enscore, and Kenneth L. Gage, "Identification of Risk Factors for Plague in the West Nile Region of Uganda," *American Journal of Tropical Medicine and Hygiene,* 90(2014): 1047-1058. doi: 10.4269/ajtmh.14-0035.

occupations, it is unclear where demarcations for class within these laboring groups might lie. For example, census descriptions of those employed in cotton mills, spinners, managers, and owners are all listed in the same category; distribution within this group unclear.[123]

While income and housing circumstances correlate to some degree – multiple-room occupancies correlated to the upper class, densely crowded chawls to the lower – a mixed lower-income group likely occupied single-room houses; for example, grain merchants might be considered closer to a middling class, but still occupy single-tenement houses, and come in contact with rats more often as a result of their occupation. The two poorest districts among the five examined, however, also experienced the highest overall mortality from plague (Table 3), while the three Indian-dominated districts with the lowest mortality rates (Esplanade, Walkeshwar, and 1st Nagpada) were sparsely populated, less crowded per house, and (with the exception of 1st Nagpada) notably more economically prosperous.[124] The qualitative and quantitative evidence from these cases indicates that class and occupation played a nuanced role in plague mortality, but that poverty and plague often coexisted.

Narrative illustrations of the mechanisms behind some of these class-based divides have recently emerged in studies in indigenous medical practice related to the epidemic. Members of the elite or high castes likely drew on a number of mechanisms to avoid infection. Some were inherent in their urban environment – most wealthy Indians lived in Fort South. Walkeshwar and Mahalaxmi districts, which, much like the European town, were much lower density than manufacturing districts and subject to more rigorous sanitary practices as a result of organization and government attention; furthermore, wealthier and middle class community members had the financial means to vacate the city at the start of the epidemic – and more crucially – stay away

[123] Edwardes, "Census," 1-329.
[124] Edwardes, "Census, Part V – Report," 66-117.

for a longer duration. These urbanites could migrate to cities like Delhi, Lahore, or Amritsar for resettlement – while those who had moved to Bombay to join the booming shipping and manufacturing industry would have either remained to continue work, or returned quickly to support themselves and their families.[125] This dynamic is visible in the recovery of Bombay's population within two years of the epidemic from its low of roughly 620,000. This pattern is also referenced in the census of 1901, in which Edwardes notes, "The decrease in the North Fort, Market, Dhobi Talao, Bhuleshwar, Girgaum and Chowpatty, must not be considered other than temporary. They are emphatically the areas from which the annual plague-exodus occurs; and one feels convinced that, if the plague-epidemics were to cease at the close of the year and if a census were taken on March 1st, 1903, the density per acre in each of these sections would be found to have risen very greatly."[126]

While an examination of these neighborhoods illuminates the role of housing structure and class in plague risk, the role of race in plague mortality – or rather, the role of the intersection of class and race – is visible in the trajectory of the epidemic in the "European town." Fort South, on the other hand, suffered remarkably less than other districts from total plague cases, at only 5 per thousand. Several important features distinguished Southern Fort from its surrounding neighborhoods. Perhaps one of the most striking of these features was density. While the average density of Indian-occupied neighborhoods in Bombay ranged from 20-30 per acre, the European town, with its large bungalows and elegant government buildings, averaged 8 persons per acre.[127] The center of imperial Bombay, the European town was the concentrated center of urban planning in the city, and housed the Town Hall, the Admiralty House, the Prince of Wales

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125 Kavita Sivaramakrishnan, "Recasting Disease and Its Environment: Medical Practitioners, the Plague, and Politics in Colonial India, 1898-1910," in *Cultivating the Colonies: Colonial States and Their Environmental Legacies*, (Athens: Ohio University Press, 2011), 205.
126 Edwardes, "Census, Part V – Report," 10.
127 Kosambi, 72.

Museum, and the central business district, all planned on a budding British Town Planning model.[128] Only 49 per cent of the district was residential, and the residences were overwhelmingly single-family homes and bungalows.[129] Its population was primarily European, as indicated by the overwhelmingly Christian (48 percent) and English-speaking (27 percent) population.[130]

For the neighborhood's (primarily Marathi-speaking) Indian residents, average occupation differed from surrounding districts. Most Indian occupants worked as domestic servants to European residents, lowering their risk of interacting with pests. Living within the Southern Fort also precluded residents from many of the sanitary concerns present in the Indian town, as sanitary infrastructure, driven by decades of "improvement" projects, was the best kept in the city.[131]

While these social-ecological statistics provide a firm ground for comparison with plague-stricken districts, not every feature that advantaged the European town can be quantified. The reality of British imperial hegemony during the epidemic, combined with existing racial ideologies of the late nineteenth century, often meant that plague control measures prioritized European safety over Indian mortality.[132] As David Arnold and Mark Harrison argue, the "municipalization" of public health was driven by the reality that most of India's European population lived in urban spaces.[133] Municipal governments' "unspoken responsibility" to protect European residents, as Arnold notes, often led to taxes that burdened local populations

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[128] Sharp, *Cities of Empire: The British Colonies and the Creation of the Urban World*, (New York: Metropolitan Books, 2014), 275-281; Kosambi, 74.
[129] Kosambi, 74.
[130] Kosambi, 74.
[131] Tulloch, *Drainage and Sewerage*, 8-9.
[132] David Arnold, "Disease, Rumor, and Panic in India's Plague and Influenza Epidemics, 1896-1919," in *Empires of Panic: Epidemics and Colonial Anxieties,* Robert Peckham, ed., (Hong Kong: Hong Kong University Press, 2015), 111-129; Aidan Forth, *Barbed Wire Imperialism*, 79-82; Harrison, *Public Health in British India,* 140.
[133] David Arnold, *Colonizing the Body*, 275. Mark Harrison, *Public Health in British India*, 166-167.

and a triaging of public health infrastructure that favored European towns over "Native Towns."[134] The Bombay Improvement Trust, formed in response to the plague emergency, was largely comprised of those representing "European official and commercial interests," including the collector of land revenue for Bombay, a member of the Bombay Chamber of Commerce, and a member of the Millowners association, among others.[135] The majority of their work included the destruction of Indian housing deemed "insanitary," the relocation of people to expensive trust-developed housing, and the "opening up" of crowded areas for traffic (a feature which, as Harrison notes, would likely benefit local business).[136] Meanwhile, more stringent emergency measures, in particular segregation into camps and inspection of persons travelling by rail and sea, were almost exclusively applied to Indian residents.[137]

Analysis of the social ecology of Bombay neighborhoods at the turn of the twentieth century reveals important trends in the relationship between class, race, and plague. As useful as these features are for identifying risk factors and regions of interest, however, it is important to remember that they are proxies for a set of relationships that are more difficult to access: namely, the relationship between environment, people, rats, fleas, and *Yersinia pestis.* This relationship is difficult to articulate because of the constantly changing etiology of plague in the dominant years of the Bombay plague epidemic. When *Yersinia pestis* arrived on the shores of Bombay in 1896, the relationship between rats and the plague was only tentatively established. While the Jean-Paul Simond first posited the rat-flea theory in 1898 and reports narrated rat mortality at the beginning of the epidemic, real efforts in documenting plague among rats did not begin in earnest for nearly a decade. Starting in 1904, the Health Officer of Bombay began a quarterly

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[134] Arnold, *Colonizing the Body*, 275
[135] Harrison, *Public Health in British India*, 184.
[136] Harrison, 184-185.
[137] Forth, *Barbed Wire Imperialism*, 77-83.

count of rats in each of the designated plague wards.[138] Spacing traps at random intervals throughout wards, plague officials collected rats in the thousands which were then brought to the Bombay Bacteriological Laboratory, and a portion of them selected for dissection and examination. The statistics that emerged from these campaigns shocked the scientific community, as is made evident in an article published in *Scientific American* in 1911:

> In Bombay the rat population is an enormous one, *Mus decumanus* (the brown or gray rat) [*Rattus norvegicus*] swarming in the sewers, gullies, and outhouses in the city, and *Mus rattus* [*Rattus rattus*] (the black rat) living in countless numbers in the houses of the people…The severity of the epizootics in the two species will be appreciated when it is stated that during one year the examination of 70, 789 *M. decumanus*, taken from all parts of Bombay city, proved that 13,277 were plague-infected = 18.8 percent, and that out of 46,302 *M. rattus* examined 4,381 were plague-infected = 9.4 percent.[139]

While the data collected by rat campaigns only gives limited reference to the size of the rat population in Bombay – there is no way to know what percentage of rats in these neighborhoods were captured, for example, or whether trends beginning in 1906 are truly representative of earlier trends – ward-by-ward rat captures and analysis might provide a relative idea of the extent of the epizootic in various parts of the city, and the relative size of the rat population in relation to other wards. Through careful analysis of these trends in relation to neighborhood-level characteristics, a link between neighborhood infrastructure, social ecology, and plague incidence and mortality may be more firmly established.

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[138] Turner, "Quarterly Report of the Health Officer of Bombay for the First Quarter of 1906," 12-15, Asian and African Studies Collection, British Library.
[139] GM Petrie, "Rats and Plague," *Nature* 85(191): 15.

Table 8. Rat density per person and per acre in relation to human density and plague mortality.

Ward	Population	Density (persons) per acre	Rats Collected Between April 1910 and December 1916	Rats per Acre	Rats per Person
A	64,819	50	307,486	237	4.75
B	152,377	220	1,314,808	1895	8.63
C	206,372	304	623,391	917	3.02
D	97,329	95	483,560	474	4.97
E	180,423	47	800,041	341	4.43
F&G	98,402	15	334,656	50	3.40

SOURCE: Compiled from Turner, "Quarterly Report of the Health Officer of Bombay, 1910-1916."

Relationships between density of wards and number of rats per ward relate tentatively for some wards (B ward and A ward are striking examples), and heterogeneity in population density per ward makes analysis difficult, especially in F & G ward, where densely populated areas are interspersed with agricultural territory. Despite these limitations, two notable features emerge from the data in Table 6: first, districts involved in the grain trade contained the highest rat population; second, the raw number of rats captured in the period under study correlates to the wards with the highest proportion of elevated mortality – namely, B ward, C ward, and E ward. Beyond giving a sense of the relative sizes of the rat populations in these neighborhoods (or at least, within the trappable radii established), rat-catching experiments indicate a cyclic die-off in rat populations annually with recovery within several months, which supports Keeling and

Gilligan's stochastic model for the continued annual plague epidemics that struck Bombay during this period (Figure 12).[140]

Conclusion: Poverty and Race as an Ecological Niche

As city and neighborhood-level analysis reveals, particular features of Bombay's environment seem to have contributed to the long-term persistence of plague in the city. At the level of the city, its position as an active British port altered Bombay's rat ecology, creating an unusual mix of coexisting Norway and black rat populations. The differing niches of these two rats seems to have allowed for the persistence of *Yersinia pestis*, as the Norway rat acted as a reservoir between epizootics, then passing the disease to its black counterparts, who likely transferred it to humans incidentally. Factors like income, employment, and race acted as risk factors for plague incidence and mortality by altering opportunities for contact with rats and fleas. Lower-income persons were more likely to live in substandard housing, which allowed more opportunities for rats to gain entry into living spaces; employment in grain shipping and storage also provided close contact with the rat populations that swarmed these areas, looking for food; meanwhile, European hegemony provided legal scaffolding to protect white residents of the city and their immediate contacts from contact with plague by instilling more stringent sanitary requirements that often distanced them from those who were infected, whether human or rodent. Together, these features provided an urban ecology in which *Yersinia pestis* could thrive among rodents – and therefore, an environment in which incidental human epidemics continued for decades.

While the numerous sanitary organizations that emerged around the outbreak of plague

[140] Keeling and Gilligan, 2219-2230.

identified several of the risk factors demonstrated in this section by the end of the nineteenth century, interventions targeted at ameliorating some of these conditions did little as the epidemic gained strength. In spite of increasingly interventionist measures of plague control, and a growing number of medical doctors, bacteriologists, and sanitary officials who struggled to understand the disease, mortality rates remained high for the Indian subcontinent, and Bombay in particular. In fact, the next chapter argues, as city-wide plague policies bent to the hegemony of the Bombay Bacteriological Committee, and rats, fleas, and *Yersinia pestis* fell under an imperial gaze, the biopolitical relationships of humans and nonhumans to the imperial government changed – often for the worse.

Chapter 4: Changing Ecologies, Changing Niches – *Yersinia Pestis* and the genus *Rattus* in the Era of Plague Control.

Less than a month after the first case of plague, the Bombay Municipal Government laid siege to city's residents. Accacio Viegas identified the first case of the disease in September 1896; by October 1896, the government had significantly extended its powers to accommodate the epidemic.[1] These measures almost immediately changed the relationship of the imperial government to the private spaces and bodily sovereignty of Bombay's citizenry; municipal officials could order thorough disinfection of the interior of any citizen's home, and upon the discovery of illness, forcibly remove any resident to the hospital.[2] The Epidemic Diseases Act, passed in early 1897, extended these measures further. The Act detailed the measures legally allotted to the newly established Plague Medical Officer and conferred special powers upon local authorities to "control dangerous epidemic diseases." The government was thus granted the right to inspect all persons traveling into or out of the city, and to detain suspected persons – more ominously, the legislation claimed that "the state may take, or require or empower any person to take some measures and by public notice prescribe such temporary regulations to be observed by the public."[3] Within months of the first case, government officials had near total license to alter the relationship of Bombay's residents to their city, in the name of plague.

Historians have gained much traction on the intersection of biopolitical control, colonial medicine, and empire in examinations of the plague in India, and particularly in Bombay. David Arnold labeled the measures enacted in India against plague – most of which were modelled

[1] Papers Regarding Plague Operations in Bombay for the Secretary of State in Connection with the Questions Asked in the House of Commons by Mr. Maclean, March 1898, Nos. 398-411, Simla Records, Home Department, Sanitary – [Plague] Branch, National Archives of India.

[2] M.E. Couchman, *Account of the Plague Administration in the Bombay Presidency from September 1896 till May 1897*, (Bombay: Government Central Press, 1897), 55-56, Medical History of British India Collection, National Library of Scotland.

[3] Couchman, *Account of the Plague Administration*, 55-56.

after the original measures in Bombay – "A new interventionism," marking the increasing invasion of imperial order into the homes and bodies of Bombay's Indian residents.[4] These unprecedented laws reorganized the geography of biopolitics in the city. Before the outbreak of plague, interfaces of the municipal government and the colonial body occurred at an impersonal scale – through the regulation of municipal sanitary practice, workplace conditions, and urban space; after, the individual body became the focus of imperial attention and action. Bombay's lower classes found themselves prodded, examined, detained and inoculated by representatives of sanitary structures.[5] Their skin, flesh, and immune systems became sites of imperial regulation. Geographic changes also occurred across ecological scales, affecting both humans and nonhumans. The reorganization of people on the Island promoted distinct changes in population geography, inciting sanitary challenges; meanwhile, at the local level, cleansing and disinfecting practices coupled with slum clearances destroyed the habitats of both human and nonhuman residents.

While public health campaigns at the local level focused on disinfection and limitation, burgeoning laboratory infrastructures looked to isolate behavior and infection pathways for *Yersinia pestis*, with the goal of waging immunological war on the bacteria. Through the machinations of the Bombay Bacteriological Laboratory and its analogous laboratory spaces, therefore, *Yersinia pestis* gained a new imperial niche. Through microscopic characterization, attempts at identifying transmission methods, and perhaps most importantly, through attempted manipulation of the bacteria to produce a plague vaccine, *Yersinia pestis* developed alternate relationships with humans; no longer only a pathological organism, *Yersinia pestis* became an object of study and, for a brief period, an ally in the continuation of human health. However, as

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[4] Arnold, *Colonizing the Body*, 203.
[5] Arnold, *Colonizing the Body*, 211-214; Forth, *Barbed Wire Imperialism*, 83.

Waldemar Haffkine and Antoine Yersin discovered, *Yersinia pestis* (and the human body) rarely behaved in ways that supported the development of this new immunological niche.

As an etiology of plague that included rats gained popularity among the British medical community, rats also fell under the biopolitical gaze of the colonial government. Across India, experiments arose that subsumed rats into the colonial medical infrastructure and transformed them from commensal nuisances to vectors of disease.[6] Plague measures, furthermore, transformed the position of rats in the city, both symbolically and literally. Sanitary measures resulted in the displacement and destruction of habitat for rats, placing fleas and their resident microbe, *Yersinia pestis*, into closer contact with uninfected humans, and created new habitats that suited rat populations in the form of internment camps. Recent studies on the home range of urban rat species indicate that the range of rats is largely determined by access to food and harborage.[7] Rats range further and into alternate burrows if their principal burrow is compromised or destroyed. Whitewashing and sanitizing practices of the Bombay municipal government, which often included washing down houses and adjacent areas with boiling water and carbolic acid, likely exacerbated this behavior.[8] These sanitary measures would have effectively destroyed rats burrowed in infected houses and flushed them into adjacent homes or neighborhoods, as would motions to "remove or burn any huts or temporary erections in which a case of plague is believed…to have occurred."[9] Plague officials noted these altered special geographies in the wake of forced removals. The Plague Committee, for example, reported in

[6] "Introduction: Infectious Animals and Epidemic Blame," *Framing Animals as Epidemic Villains,* Christos Lynteris, ed., (New York and London: Routledge Press, 2019), 1-25; Nicholas Evans, "Blaming the Rat? Accounting for Plague in Colonial Indian Medicine," *Medical Anthropology Theory* 5(2018): 15-42. doi:10.17157/mat.5.3.371.
[7] Kaylee A. Byers, Michael J. Lee, David M. Patrick, and Chelsea G. Himsworth, "Rats About Town: A Systematic Review of Rat Movement in Urban Ecosystems," *Frontiers in Ecology and Evolution* 7(2019): 3.
[8] "Revised Plague Rules Issued by Local Governments and Administrations," April 1901, Sanitary Plague A Branch, Home Department, Nos 233-247, page 77, National Archives of India.
[9] *Ibid.*

1898 that "[E]xperience had shown that plague passed from house to house, and that neighbours living several doors off a plague house were frequently attacked after the inmates of the plague house and been removed."[10] The routine flushing of sewers with carbolic acid would have also driven out sewer rats from nests and into homes.[11] Other measures, like the suggested punching of holes into the roofs and walls of homes to allow adequate ventilation and sunlight, would have made it easier for rats to enter into homes.[12]

Internment camps, popular in the early years of plague containment, present a clarifying case on the ecology of plague. Beginning in 1897, Municipal Sanitary officers conducted daily house-to house searches throughout Bombay's neighborhoods. Guided overwhelmingly by contagionist doctrines that racialized disease transmission (in spite of governmental focus on bacteriological innovation), these officials concentrated largely on chawls and other crowded spaces housing the city's working populations.[13] If a resident was found to exhibit symptoms associated with plague (though often this could be as nonspecific as a high fever or malaise), the person was removed to the hospital. The other residents of the house (or sometimes block, depending on the discretion of the sanitary officer) were then removed to a plague camp, where they would be kept for a minimum of 10 days. Their property, in the interim, was subjected to whitewashing, or in more extreme cases, outright destruction.[14]

As Aidan Forth argues, camps, often crowded and with haphazard infrastructure, were hotbeds of microbial disease – in particular, cholera, typhus, and fever-related illnesses.[15] Plague internment camps, however, were rarely hotbeds of plague. In fact, the mortality rate of plague

[10] Couchman, *Account of the plague administration*, 101.
[11] *Ibid*, 122; Myron Echenberg, *Plague Ports,* 76.
[12] "Report of the Indian Plague Commission, Chapter VI," 1900, Plague Branch, General Department, Vol 1008, No 628, pg. 327-350, Maharashtra State Archives.
[13] *Ibid.*
[14] *Ibid.*
[15] Forth, *Barbed Wire Imperialism,* 111.

camps was significantly lower than the general mortality rate of the city – about 1.42 percent in total.[16] It is in this disparity that one of the defining feature of plague ecology is most visible: its embeddedness in ecologies of injustice, and its integration into structural violence. Plague's dependent relationship with a series of vectors relies on a suitable environment for those vectors. While originally established to isolate plague victims from healthy residents, increasing focus on plague as a vector-borne disease at the beginning of the twentieth century changed the role of camps. Residents of plague-stricken neighborhoods could move temporarily into plague camps, and thus avoid contact with the increasingly villainized rat. As these camps exemplify, just as plague sanitary measures emerged, therefore, the discovery of mechanisms of infection challenged their use.

Historians of plague have recently highlighted the importance of "the ethical, aesthetic, epistemological and political entanglement of non-human animals with shifting medical perspectives and agendas," in the Third Plague Pandemic, these key considerations have remained largely within the realm of the medical, documenting and theorizing the inclusion of the rat in an imperial (or global) medical regime. In this section, much like the last, I plan to incorporate "non-scalable" elements of the epidemic in India, in Anna Tsing's words, into the existing discussion of "scalable" elements of zoonotic epistemology that emerged out of the Third Plague Pandemic.[17] I will argue that, in addition to changing the rat's relationship to medicine and urban space, through a myriad of behavioral experiments, quantification techniques, and unsuccessful population-limiting campaigns, plague control and prevention measures altered rats' relationship to their landscape, to the parasitic fleas who inhabited them,

[16] Forth, 123.
[17] Anna Tsing, *The Mushroom at the End of the World: On the Possibility of Life in Capitalist Ruins*, (Princeton: Princeton University Press, 2015): 37-43.

and to bacteria that infected them. The result was, much like *Yersinia pestis* a shift in the ecological niche of the rat within the imperial city.

Changing Etiologies of Plague and Shifting Biopolitics of Rats

Research on the etiological pathways of plague emerged within a shifting paradigm of public health epistemology. At the turn of the nineteenth century, the rising hegemony of "Pasteurian science", which combined germ theory and sanitary ideologies, influenced the development of the field of bacteriology.[18] This unique confluence of microbes, morality, and experimentation defined medical science in Europe from the 1870s; it was not long after that bacteriology made inroads into British India.[19] Pratik Chakrabarti argues that the emergence of bacteriology in India developed in the confluence of Victorian imperialism and the Pasteurian revolution. Included in this evolution as the education of a number of Indian Medical Officers in France and Germany at Bacteriological Institutes, and even, as in the case of Dr. Nusserwanji Surveyor, the education of Indian doctors in bacteriology at University College London.[20] The result was the emergence of a number of scattered, often disorganized bacteriological institutes across India – all of which scrambled to provide explanations for the epidemic. Publications positing several different frameworks for the spread of the disease circulated widely, with wildly conflicting results. While many pursued the rat-plague theory from an early stage, others were reluctant to accept animal pathways for the disease. Antoine Yersin, for example, isolated a microbe present in the soil surrounding plague victims' houses, and so asserted that "the microbe can live and thrive in the

[18] Pratik Chakrabarti, *Bacteriology in British India: Laboratory Medicine and the Tropics*, (Rochester: University of Rochester Press, 2012), 27.
[19] Chakrabarti, *Bacteriology in British India*, 27.
[20] Chakrabarty, 32.

soil of infected houses."[21] Others suggested that the disease "can remain alive in sterilised water for many days or even weeks."[22] The relationship between plague and grain merchants prompted studies that suggested that "the microbe can live up to 13 days in certain grains."[23]

Throughout this period the relationship between rats and the plague was colloquially accepted; though bacteriologists observed the rat epizootic as a harbinger of human plague, they were hesitant to establish a direct connection. For example, EF Gordon Tucker, a professor of pathology at Grant Medical College and Captain of the Indian Medical Service, wrote in 1904 that rats certainly suffered from plague, but that "there can be no doubt that [plague's] growth is influenced and promoted by two conditions," namely "a soil saturated with human and animal excreta" and "a congested population living in contact with such a soil, among whom the standard of health is lowered by insufficient or innutritious food, and who are crowded into houses which are deficient in light and ventilation.[24] Often, scientists injected discovered microbes into rats to determine whether they were infectious to the mammal, and thus a possible variation of the plague microbe.[25] However, the ability of rats to spread the plague to humans remained a subject of skepticism, and no definitive theory arose to a position of prominence in sanitary communications. The theory did, however, gain traction in other parts of the Empire prior to its acceptance in Bombay. Ashburton Thompson, Tidswell, Armstrong and Dick in Sydney, for example, fronted ecological theories of plague transmission facilitated by rats, by

[21] EH Hankin, ". Bacteriological Research Life History of the Plague Bacillus in Nature. From Investigations on Plague," 1898, Plague Branch, General Department, vol 479, no 575, pg. 64, Maharashtra State Archives.
[22] Hankin, "Bacteriological Research," 71.
[23] Hankin, "Bacteriological Research," 75.
[24] Ernest Frederick Gordon Tucker, *The Symptoms and Pathology of Plague (With a Note on the Management of a Plague Epidemic)*, (Bombay: Education Society's Press, 1904), 2, T35457, General Collection, British Library; RF Childe and RWS Lyons, *Report(s) of the Plague Research (Lyons) Committee*, IP/13/PC.2, pg. 6, Medical History of British India Collection, National Library of Scotland.
[25] Hankin, "Bacteriological Research," 65.

mapping rat locations and correlating them to outbreaks.[26] Despite their efforts to sway Bombay's bacteriological and sanitary officers, the prominent plague authorities, including Waldemar Haffkine, remained unconvinced of the relationship.[27] Public health officers therefore relied on existing entrenched sanitary frameworks for management and control of the plague.

Near the turn of the century, the rat-flea theory achieved a position of prominence in British Indian sanitary literature and bureaucratic communication. On April 24, 1902, the Sanitary Commissioner for Calcutta drew attention to a paper written in the early Bombay epidemic which claimed that, "where a rat dies of plague fleas forsake him and may carry plague to other rats or to man," and argued that the theory had numerous supporters, despite Waldemar Haffkine's assertion that there was "no scientific evidence that fleas carry the infection from rats to men."[28] Meanwhile, Professor Richard Friedrich Johannes Pfieffer, who travelled to Bombay alongside Robert Koch to investigate the plague outbreak independently, admonished the blindness of the Bacteriological community in India in an address to the German Public Health Society in Berlin that same year where he "ascribed the failure of the drastic measures taken in India to check the first Bombay epidemic by the fact that rats were not, and could not, be regarded as carriers of infection."[29] By 1907, the Sanitary Commissioner for British India, JTW Leslie, had firmly asserted that "the epidemiological inquiries in Bombay and in villages of the Punjab have fully confirmed the view strongly urged by some epidemiologists, that epidemic

[26] Myron Echenberg, *Plague Ports*, 264.
[27] JTW 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.
[28] Leslie, "Exterimation of Rats," 1.
[29] *Ibid.*

human plague is directly consequent upon epidemic plague in rats."[30] With this declaration, Leslie acknowledged the breadth and depth of knowledge formation that surrounded the rat in India since the beginning of the epidemic, and formally shifted the focus of plague operations in India towards the organism.

The emergence of the rat-flea theory as the dominant paradigm of plague etiology catalyzed a shift in sanitary ideology and practice in Bombay. Especially within scientific and medical branches of plague control structures in Bombay and India more generally, focus shifted from human itinerant bodies to center on rat bodies. Bacteriologists conducted experiments to determine the life habits of rats; chemical analyzers tested a series of poisons and biological agents to control the rat population; briefly, the city instituted a rat-killing campaign; and as mentioned in the previous chapter, rats became visible in colonial statistical practices, as quarterly health reports began to detail rat capture and testing campaigns to establish the extent of the populations in wards and neighborhoods as well as the extent of the infection within these populations.[31] In the early twentieth century, therefore, the Bombay Sanitary Committee established a system of control grounded in ecological ideas and epidemiological methods that were largely concentrated on understanding, categorizing, counting, and killing organisms. In other words, the shifting bacteriological theories gave rise to a biopolitics of rats and plague-associated bacteria. Practices of observation, capture, and dissection of rats, and attempts at poisoning, culling, and rat-proofing, members of the Indian Medical Service, not only expanded

[30] JTW Leslie, "Continuance of the investigations of the Advisory Committee for Plague Investigation in India. Extension of the deputation of Captain W.G. Liston, IMS, to England in connection with the investigation into the etiology of plague. Grant of combined leave to Lieutenant-Colonel W.B. Bannerman, IMS, Director of the Bombay Bacteriological Laboratory, and appointment of Captain WG Liston, IMS, to act for him," October 1907, Simla Records 3, Sanitary Plague A Branch, Home Department, nos. 109-123, pg. 11, National Archives of India.
[31] 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, AAS, BL.

rats from "vectors" and "vermin" into "subjects" and "threats," but changed the ecological landscape of Bombay for its occupants.

By the early 1900s, the minutiae of daily life among rats gained an unforeseen importance and integration into imperial experimental science and public health practice. Lieutenant-Colonel Bannerman, the head of the Bombay Bacteriological Laboratory, decentralized man from plague epidemiology; In a 1906 publication on "Conditions Affecting the Origin and Spread of Plague," he claimed, "it becomes more evident as time passes that in studying the etiology of this disease we must regard it not so much from the standpoint of a human disease, but as being essentially a rat epizootic communicable to man."[32] Bannerman demonstrated the extent to which ecological studies of the rat proliferated the Bacteriological Institute in his neat description of the appearance, behavior, and skills of the two rat species in Bombay. "The black rat is a clean, sleek, almost graceful animal and a most nimble climber. When it enters a house it takes up its abode in the roofs and other suitable places above ground." The brown rat, on the other hand, "is a larger heavier animal with coarser hair, often covered with scars of wounds inflicted in the fights that are of daily occurrence, and is altogether a disreputable looking creature. Its habits correspond, for it frequents drains and open gutters, where grabage [sic] of all sorts accumulates, living for the most part in burrows underground and in old sewers and drains."[33] From this behavioral difference, Bannerman argues, "This great difference in their habits necessarily results in the bringing of the black rat into close relation with man, as a domestic animal almost; while the brown rat lives at a distance and is rarely seen by him."[34]

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[32] W.B. Bannerman, "Conditions Affecting the Origin and Spread of Plague," in "Measures for the Prevention of Plague," February 1906, Calcutta Records 5, Sanitary Branch, Home Department, Government of India Home Department, nos. 327-347, pg.106, National Archives of India.
[33] *Ibid*, 106.
[34] *Ibid.*

What is remarkable about Bannerman's analysis is not necessarily the speciated description of rats and their behavior, but the firm grounding of these rats within Bombay's urban ecosystem. This nuance is apparent in his observations related to built environments in India as a whole. He argues, "The structure of the houses in this country seems designed to favour the continued existence within them of the black rat, *e.g.*, the thatched roofs, or flat mud roofs supported on branches and twigs in the mofussil form ideal breeding places for them." Even more remarkable, however, is his highly localized analysis of rat-human interaction. In Bombay, Bannerman notes, "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." Meanwhile, "The bags of grain stored by the grain dealer in his house, and the habit of stabling horses and cattle in the ground floor of dwellings also favours the intercommunication of man and the black rat." He even provides an inverse case in the form of Glasgow, wherein an epizootic of plague occurred among brown rats in 1903, but with only a small number of cases among humans.

Bannerman reframed human plague cases in terms of their likely proximity to or interaction with rats, in terms of occupation and class (he argues that "these facts serve perhaps to explain to some extent the greater incidence of plague among classes, *viz.*, the grain dealers, stable-keepers, and poorer classes generally,") by religion ("while the notorious severity of the disease among the Jains may be due to their dislike for the destruction of animal life in any form, and their encouragement of the rat by feeding,") and by wealth ("The greater immunity from attack of the rich Natives and Europeans is accounted for perhaps by the strongly built houses, roofed with Mangalore tiles or patent stone, which do not afford shelter to rats; and to their habit of removing refuse of all sorts to a distance").[35] Through his work, Bannerman posited a structural epidemiology for plague defined by its primary sufferer, the rat.

In his paper, Bannerman, representative of a large community of medical officials operating in Bombay and across India in the early twentieth century, articulated an altered understanding of plague as a "rat disease" through examples from their shared ecology. To control plague, Bannerman suggested alterations in the shared habitat of humans and their rodent counterparts – either through the "substitution" of the brown rat for the black rat, as, he claims, has happened in Europe, or through an alteration of the style of housing in the city altogether. He advocated, "the erection of substantial dwellings instead of mud houses; the substitution of rat-proof roofs for those of tiles or mud; and stone or concrete floors for earthen ones; the separation of dwelling houses from shops, warehouses, and granaries," alongside the prompt removal of garbage to minimize contact between humans and rats.[36]

Thus to separate humans from the plague, Bannerman focused on the ecological interactions of vector and person, and underlined the need to change the environment for the rat, rather than for the microbe. He touted inoculation as an inexpensive and effective scheme that should also be utilized, and suggests disinfecting of clothing to avoid fleas – and in doing so acknowledges the role of the rat as a sufferer from the disease, rather than simply a vector.[37] However, he emphasized the rat above all, and it is only for the rat that structural change is suggested.

While knowledge-gathering related to rat behavior often relied on ecological studies, occasionally laboratory-based experimentation revealed the minutiae of the rat's niche within the city. In 1901, for example, Captain R.D. Saigol of the Indian medical service gave a "Report on

[35] *Ibid,* 106.
[36] *Ibid*, 108.
[37] *Ibid*, 109-110.

experiments made to determine the jumping power of rats."[38] In his study, Captain Saigol tested a series of different species of rat – *Mus rattus* (*Rattus rattus*), *Mus concolor* (presumably *Rattus norvegicus*), *Nesokia bengalensis* (*Nesokia indica)* and *Mus musculus*. In the first series of experiments, Saigol placed rats inside a two-foot tall drum without food and placed a fish outside the drum. He would then place bricks of gradually higher heights inside the drum until the rat could get out. From this experiment, he deduced that "(1) Rats can jump two feet high. (2) Rats can climb up plain iron-sheeting about two feet, along a corner by pressing their feet on the two walls forming the corner. (3) That rats can take a second side-leap from the point where the first upward leap end and thus rise slightly higher than the first."[39] While the first series of experiments seems like an exercise in pedantry, Saigol then tested a series of "rat shields" hypothesized to prevent rats from entering via the roof – and found that "Rat-guards…projecting six inches out and having their lowest edge two and half feet off the ground appear efficient barriers." Tin bands of ten inches wide could also be used to cover holes in the home, prevented they were more than two feet above the ground.[40] Perhaps valuably, the experiment indicated that rats could not climb straight up brick or plastered wall, except along the corners or along woodwork.[41]

While live experiments were not uncommon, most rats interacted directly with colonial medical infrastructures through death. Surveillance mechanisms dictated that the bacterium inside the rat (or not inside the rat) was far more valuable than the life of the rat itself; subsequently, even rats captured live were often killed, dissected, and destroyed. Dr. Kitasato Shibasaburō, one of the two bacteriologists credited with the discovery of plague, emphasized

[38] Captain 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.
[39] *Ibid,* 10.
[40] *Ibid,* 10.
[41] *Ibid*, 11.

the importance of rat deaths in plague control in a report on plague in Japan in 1906: "The finding of humans victims of the pestilence is almost invariably preceded by the discovery of plague-infected rats. Hence the killing of rats must be resorted to as the first and most important step in the prevention of an epidemic."[42]

Rat-killing took on many forms over the course of the early twentieth century, each with a differing impact on local ecology. Poisoning took place routinely using Common Sense Rat Exterminator, an organic poison produced in Toronto to combat agricultural pests. The poison was spread onto sugared bread, after which it was "laid in the evening in the gullies, between houses, near dustbin carts, and in places where kitchen refuse is thrown out and where rats are likely to report at night time."[43] Dead rats, which were "found over the area treated for three or four days following the lying of baits," were then labelled (with a label identifying the collector), location registered, and sent in tin boxes to the Bacteriological Laboratory for examination.[44] Upon the discovery of infection, death begot death – as "on the following day, these places are treated with 'Pesterine,' which is also poured into all the rat-holes found in the vicinity."[45]

Plague surveillance and prevention measures proved not only fatal to the rats who suffered the epizootic, but to other organisms integrated into Bombay's urban ecology. A report from the Madras Presidency in 1908 on rat destruction measures emphasized, "The Common Sense Rat Exterminator…is harmless or comparatively harmless to human beings, but has been found to be poisonous to domestic animals such as cats, dogs, fowls, and goats."[46] While the poison was liable to be picked up by pets, commensal dogs and cats, and urban livestock, evidence indicated

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[42] WB Bannerman, "Measures for the Destruction of Rats," March 1908, Sanitary A Branch, Home Department, nos. 18-28, pg. 27, National Archives of India.
[43] W Venis, "Accompaniments to Government Resolution, General Department (Plague no. 840-P, dated 21st June 1907), 18th February 1907, no. 9073, pg. 16-19, National Archives of India.
[44] *Ibid*.
[45] *Ibid*.
[46] Bannerman, "Measures for the Destruction of Rats," 7.

that it quickly became ineffective against rats. HD Taylor, Agricultural Secretary to the Government, observed that the rats' keen sense of smell resulted in their avoidance of the substance:

> Experience further tends to show that the Exterminator is successful only during the first few days of its introduction in a particular locality, that the baits have to be very carefully prepared untouched by hand in such a way as to not allow the colour, odour, or luminosity of the poison to be revealed, and that only a small percentage of the baits laid down is taken and that the rats, after a time, seem to avoid the poison instinctively, however carefully the baits may be disguised.[47]

In fact, Taylor emphasized, use of this poison may have forced rat migration, potentially exposing adjacent neighborhoods or streets to the disease, as "the laying down of poison baits in a particular locality has been observed in several cases to lead to a desertion by the rats of that locality."[48] Thus while "Common Sense Rat Exterminator" showed success in laboratory and rural capacities, the density and behavior of city rat, and the specific ecology of the city, rendered the poison controversial.

Other poisons showed perhaps even more limited effectiveness in rat control, often as a result of local ecological specificity rather than outright quackery. Tests by Captain Christophers, superintendent of the King's Institute on "the efficacy of Ratin as shown upon '*Mus rattus*' in Madras," found that the poison was quite ineffective against the "Indian house rat," and thus recommended a discontinuance of its use.[49] Brief flirtations with living poisons in the form of the "Danysz virus" (which was, in fact, a bacterium) proved similarly ineffective. In 1896, the Chemical Examiner and Bacteriologist of the Northwestern provinces (which were overwhelmingly agricultural) secured a sample of the virus from the Pasteur Institute, and

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[47] *Ibid*, 9.
[48] *Ibid*, 9.
[49] SR Christophers, "Report on the efficacy of Ratin as shown upon "Mus Rattus" in Madras," March 1908, Sanitary A Branch, Home Department, nos. 18-28, pg. 12-20, National Archives of India.

conducted experiments on it with rats from the city.[50] These papers referred to the Danysz virus as a "bacterial poison," capable of inciting epidemics among rats. The prevailing theory for its use relied on the pathogenicity of the bacteria for maximum efficiency; rather than a regular poison, which would only kill the rats who had directly consumed the food laced with poison, the Danysz virus appealed to the Sanitary Committee because it carried its own limited agency. A rat who consumed the "virus" (bacteria) would carry the infection back to its nest, transmitting it among its nest and adjacent family units, sparking an epidemic in which about 50 percent of the rat population "disappeared" – though whether disappearance meant death or migration remained to be seen.[51]

Early bacteriological experiments, however, showed limited effectiveness of the "virus." In 1900, the Chemical Examiner concluded experiments on Bombay and Agra rats using the virus – both as food and as an injection – and found the bacteria ineffective.[52] The explanation for this failure can perhaps be located in an observation of Danysz himself, who in the Annales de L'Institute Pasteur, observed effectiveness against "le rat gris," but none at all against "le rat noir."[53] Thus, the particular ecology of rat populations, alongside rat behavioral patterns in India may have rendered the bacteria ineffective.

These mixed results tempered the popularity of poisoning as a method of capture and control, and thus poison remained only one of several attempted strategies in rat control. Live capture remained a consistent alternative throughout the early twentieth century – though a living captured rat was unlikely to stay alive very long. The first formalized rat-catching efforts relied

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[50] "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.
[51] Leslie, "Extermination of Rats," 6; "Experiments to test the efficacy of M. Duclaux's cultures," 4.
[52] "Experiments to test the efficacy of M. Duclaux's cultures" 1-4.
[53] J Dansyz, "Un Microbe Pathogene pour les Rats: Et son application a la destruction de ces animaux," in *Annales de L'Institut Pasteur* (Paris: Masson Press, 1900), 193-204.

on professional rat-catchers – primarily defined as “low-caste Hindus” – employed through the municipal government.[54] Through these measures, increased interaction with potentially disease-carrying rats was increased among a small population of employed sanitary workers. However, quickly after this program was instated, rat-catchers were found to be “unsatisfactory,” and a trapping system quickly set in place. The Municipal Commissioner outlined that traps were “set over night in gullies and houses, with either dried fish or pieces of dried cocoanut as bait and are collected the next morning. When a rat is found in a cage a label is fixed to the cage showing when the rat was caught and the name of the person entitled to payment for the rat,” after which the rat is sent to the Bombay Bacteriological Laboratory for examination.[55] Photographic evidence suggests that bacteriological examination rarely occurred on live rats, and was rarely undertaken by European sanitary workers:

[54] JA Turner, “Report of the Executive Health Officer of Bombay for the 4th Quarter of 1904,” 3, V/25/840/23, India Office Papers, Asian and African Studies Collection, British Library.
[55] Venis, “Accompaniments to Government Resolution,” 19.

Figure 14. Bombay City: Rat Extermination at the Laboratory

SOURCE: "Miscellaneous Photographs: Reproductions of Photos of the Bombay Plague Investigation," SA/LIS/R.198, Box 15, Archives and Manuscripts Collection, Wellcome Library.

Visible in images from the rat examination efforts at the Bombay Bacteriological Laboratory is another facet of the racialized niche for *Yersinia pestis.* Indian sanitary workers were often put in close contact with rats thought to be infected, while Europeans remained safely in the "oversight" category. Incentive-based capture programs (those who found rats in traps were paid 2 pice for a living rat, 1 pice for a dead rat) further exposed civilians to potentially infected rats. This differed significantly from other Indian cities attempting rat-trapping, who often used forced labor or individuals from backwards castes to collect rats. In a report from the Madras presidency, for example, rat capture and poisoning was organized such that "a small menial establishment of coolies, peons or lascars…attend to the distribution of traps and poisons, to the collection of captured or dead rats and other work of menial nature in connection with the operations," suggesting that rat capture, as opposed to rat poisoning, may have aimed less at minimizing points of contact and more on integrating rats, fleas, and plague into imperial surveillance structures. [56] The extent of this incorporation into surveillance is visible in the systematic quantification of rat capture and infection statistics, published quarterly in the Report of the Health Officer of Bombay from 1910 to 1930, far later than the rat-flea theory gained dominance in local sanitary circles, and far longer than plague represented a significant cause of mortality in the city.

The wide variety of knowledge-gathering practices related to rats in the city of Bombay culminated in a detailed and complex understanding of rat ecology and the characteristics of the plague epizootic. The result was a detailed structural and behavioral study of plague that, remarkably, included multispecies narratives of the disease. In a report titled *Etiology and Epidemiology of Plague*, a series of experimental and ecological observations of both the rat and the rat-flea in Bombay and Punjab lead the author to the conclusion that "the conditions which favour this species of rat will be the conditions which favour the spread of the epidemic through the city."[57] Included in this analysis are the description of building structures as "flimsy," with loose foundations, that the system of gullies between the ground floors of buildings and sewers and storm water drains favor the rat – especially the sewers, because "the latter come into complete function only in the rainy season and, therefore, during the greater part of the year are practically dry, and must then offer excellent shelter for rats with habits like *M. decumanus.*[58] Equally to blame were the "innumerable stables and store houses or go downs of all

[56] Leslie, "Measures for the Destruction of Rats," 8.
[57] Lamb, *Etiology and Epidemiology of Plague,* 71.
[58] *Ibid*, 72.

descriptions," which "afford excellent shelter for rats"; and that more broadly, "the food supply for rats is abundant" in the form of waste.[59] However, the Committee claims that "the insanitary conditions which exit in Bombay have no influence which acts directly on the spread of epidemic plague."[60]

The emergence of the rat-flea theory as the dominant etiology of plague significantly altered the relationship of both *Rattus rattus* and *Rattus norvegicus* to the urban ecology of Bombay. Poisoning campaigns often led to forced migration, exposing rats and their arthropod and bacterial counterparts into contact with people or groups of people previously uncontacted; trapping campaigns moved rats from a position of vermin to bacteriological examination, and placed them in a position of value (monetary and bacteriological) within sanitary structures. From these interventions, rats grew to occupy a more diverse niche within the urban ecosystem, both dead and alive. Rat-proofing campaigns demonstrated the extent of the Bombay Plague Committee's fixation on rat mobility in the city, and their willingness to alter urban geography to discourage rat harborage, thus placing a new emphasis on rat behavior and ecology in public health. However, while the focus of many plague control campaigns centered around rats, it was not the rats themselves that comprised the threat, but what nestled in their fur – a reality only acknowledged by a few within the public health infrastructure.

Xenopsylla cheopis and Plague Control

While the rat-flea theory gained acceptance and attention in sanitary circles across both Bombay and India more widely in the early twentieth century, most of these studies focused only on the first – and most visible – actor in this name. Studies circulated in medical journals,

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[59] *Ibid*, 72.
[60] *Ibid*, 71.

government reports, and municipal committees detailing the niche of the brown and black rat in urban spaces, but only a handful of studies attempted to document the agency of the flea as an entity separate from the rat. Much like the rat, studies on the flea occurred in both laboratory and urban settings, and relied heavily on ecological framings to document transmission strategies and risks. From these studies, the flea emerges as a separate urban organism with preferences, patterns, and etiologies.

While studies had supported the rat-flea hypothesis since 1898, experiments into the role of the flea in transmission of plague from rat to man first began in earnest in Bombay in 1904, when a Committee convened by the Secretary of State for India began work at Parel at the Plague Research Laboratory on the epidemiology of plague.[61] Several experimental studies relied on live Bombay wild rats, collected by trapping, and imported English white rats. Common experimental included exposing healthy, flea-free rats to the corpses of rats that had been allowed to die of plague, and the direct transferal of fleas from a rat dead of plague onto a healthy rat.[62]

Observational studies, performed in houses and godowns between 1905-1907, also relied on the parasitic relationship between rats and fleas, or rodents and fleas more broadly. These studies often involved releasing healthy rats or guinea pigs into infected houses, recapturing them, combing them for fleas, and noting the infection rate. The primary focus of these experiments was not the flea as an organism unto itself, but as a vector, as the Committee attempted to construct a model of the epizootic among *Mus decumanus*, *Mus rattus,* and then humans to determine patterns. While largely focused on the flea as a parasite of rats, rather than

[61] Lamb, *The Etiology and Epidemiology of Plague*, i-iv.
[62] "Progress Report Submitted by Major G. Lamb, IMS, Senior Member of the Plague Research Commission to India, to the Advisory Committee in England," August 1906, Calcutta Records 2, Sanitary Plague A Proceedings, Home Department, no. 99, pg. 1-8, National Archives of India; Lamb, *The Etiology and Epidemiology of Plague,* 43-59.

as a separate organism, the committee observed an unexpected phenomenon in flea behavior – namely, that when trapped rats were combed for fleas, it was most often found that *Mus decumanus* carried twice as many fleas as *Mus rattus*, suggesting to the committee that *Mus decumanus* acted as the reservoir for the disease. Furthermore, epizootics measured between 1905-1907 often occurred in *Mus decumanus* first, moving to *Mus rattus* within several days. While this result was tentative at best (*Mus decumanus* is frequently more easily trapped because of its ground-level harborage and epizootic realities may have altered flea numerosity, for example), it is possible that this result indicates a differing ecological niche for *Xenopsylla cheopis* in Bombay. Whereas studies indicate that *Rattus rattus* is often the preferred host for the flea, the presence of both species of rat in abundance may have led to cyclic preference for the sewer rat.[63]

As experimental evidence implicated the flea in transmission of the plague from rat to rat, and even rat to human, bacteriologists separated the agency of the flea from the agency of the rat more consistently, building a behavioral profile for the organism. Committee employees conducted a systematic flea census, "with the object of ascertaining if there was any definite seasonal prevalence of these insects."[64] Observation revealed the homogeneity of the flea in Bombay – "practically the only species of flea to be found on Bombay rats was *Pulex cheopis*."[65] Starvation experiments also revealed that while *Xenopsylla cheopis* showed a strong preference for rats over humans, though, "they have no aversion to such when hungry (as they become when their natural hosts die or are driven from their runs in houses)."[66]

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[63] Lamb, *The Etiology and Epidemiology of Plague*, 10.
[64] G Lamb, "Fourth Progress Report of the Plague Research Commission," June 1907, Simla Records 3, Sanitary Plague A Branch, Home Department, Government of India, no. 189, pg. 4, National Archives of India.
[65] Lamb, *Etiology and Epidemiology*, 36.
[66] WE Jennings, "Information Collected Since the Publication of the Report of the Plague Commission," 12th August 1905, Calcutta Records 5, Sanitary Branch, Government of India, no. 327-347, pg. 144, National Archives of India.

Figure 15. Flea catchers in Bombay City

SOURCE: "Miscellaneous Photographs: Reproductions of Photos of the Bombay Plague Investigation," SA/LIS/R.197, Box 15, Archives and Manuscripts Collection, Wellcome Library.

Furthermore, observation revealed *Xenopsylla cheopis*' strong seasonal preference. It was found in these observations that "the plague epidemic season corresponds with the greatest prevalence of these fleas, while in the months when plague is at its minimum the fleas are fewest in number."[67] By 1907, the Committee established a reasonable working theory of larval stages of the flea and the role of temperature in generation of the egg, fertilization, and larval

[67] Lamb, 88-92.

development, concluding that the flea thrives at a temperature under 85º F.[68] Experimental dissections of fleas also revealed characteristics of the niche of *Yersinia pestis* within the body of the flea. Dissection illustrated the ability of the bacteria to multiply in the stomach of the flea, while live observation revealed the mechanism of regurgitation as the primary mode of infection.[69] Strikingly, Lamb observed the parallel (and likely evolved) seasonal preferences of both *Xenopsylla cheopis* and *Yersinia pestis*, noting that "A high mean temperature, *i.e.* 85º F and over, affects the fate of the plague bacillus in the stomach of the flea. At this temperature, fewer successful transmissions from animal to animal are obtained and besides, the flea does not retain its power of infecting nearly so long as it does at a lower temperature, *i.e.*, 70 º F."[70]

Though the Bombay Plague Committee's experiments resulted in detailed and accurate descriptions of the mechanism of flea transmission and even the ecological niche of the flea within Bombay, these experiments largely remained useful in the realm of imperial knowledge-making, rather than practice. Mitigation efforts targeting the flea were limited at best, relying on original plague measures like the disinfection of clothing and bedsheets in infected houses and among travelers. The rat loomed much larger in the imperial imaginary as the culprit of plague, as rat-proofing and rat-poisoning campaigns indicate. However, the two did sometimes intersect; Common Sense Rat Exterminator, for example, was promoted for use in India because it advertised as killing not only rats, but fleas: "The object to be aimed at therefore isn ot merely the destruction of the rat (in itself sufficiently difficult) but also the destruction of the fleas with which they are infested. This can be accomplished by the use of a special compound of Canadian manufacture which not only kills the rat and eats it quite up without any smell whatever but also

[68] *Ibid*.
[69] Jennings, "Information Collected Since the Publication of the Report of the Plague Commission," 145.
[70] Lamb, 88.

kills the fleas the moment the rat eats it as by an electric shock."[71] However, the inutility of the poison for rats precluded its efficiency on fleas, and few other poison production companies mentioned the flea at all.

In fact, rather than mitigating flea-human interaction, evidence suggests that investigations into the plague by the Bombay Plague Committee may have led to the transmission of plague-infected fleas into areas that otherwise experienced limited plague mortality. In a general summary of the work of the plague commission in Bombay, Major Lamb, the senior member of the commission, claims that "during the visits of the Commission to plague houses in Bombay the members and their assistance often carried away fleas on their persons and clothing, some of which proved to be rat-fleas." More directly, he notes that "In Wadhala village the first plague-infected rat was found outside a house in which the coolies who worked for the Commission lived. All these men worked amongst plague-infected rats and fleas at the laboratory and two of them likewise assisted in the work in Sion, which at that time was badly infected."[72] Given the stated preference of *Xenopsylla cheopis* for rats rather than humans, it is possible that commission employees carrying fleas may have endangered commensal rats and sparked a localized epidemic. Studies on the plague might therefore be considered to facilitate the movement of fleas, subsequently endangering unaffected rat populations and their human counterparts.

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[71] "'Common Sense Rat Extermination," October 1903, Sanitary Plague B Branch, Home Department, nos. 49-50, pg. 12, National Archives of India.
[72] Lamb, 82-83.

***Yersinia Pestis* Gains a New Imperial Niche**

Much as the rat-flea theory altered the relationship between humans, rats, and fleas, the isolation of the plague bacillus by Yersin, Kitasato, and then in India by Surveyor and Haffkine initiated a change in the relationship between humans and *Yersinia pestis*. While the natural history of *Yersinia pestis* placed it in contact with humans most often through distinct urban and rural ecologies, the discovery and isolation of the bacteria in the lab constructed a new niche for *Yersinia pestis.* Through experiments on mice and rats; replication in assays; and the development and distribution of Haffkine's live vaccine, *Yersinia pestis* began to occupy entirely new spaces and roles in Bombay. While for rats and fleas these changes occurred on through ecological and bacteriological mechanisms, *Yersinia pestis*'s interaction with human and nonhuman counterparts also occurred at a molecular and immunological level.

One of the first alterations in the relationship between the plague bacteria and the human body emerged almost immediately upon its discovery. Just two decades previously, Pasteur's innovation of the first laboratory-developed vaccine for chicken cholera (*Pasteurella multocida*) opened a new realm of possibility for interactions between human and microbe. No longer solely an infectious agent, bacteria were transformed from sickness into medicine through chemical transformation and interaction with the immune system. This innovation spurred the creation of bacteriological institutes globally, as Pratik Chakrabarti notes, with institutes appearing in colonial spaces almost simultaneously with European and northern American spaces.[73] By 1894, the year plague emerged in the British Empire, Waldemar Haffkine had already established a bacteriological laboratory and instituted vaccination campaigns in Calcutta.[74] With Yersin and Kitasato's near-simultaneous identification of *Yersinia pestis*, the combined vaccine optimism

[73] Chakrabarti, *Bacteriology in British India*, 2-3.
[74] Chakrabarti, 1.

and colonial infrastructure comprised the perfect climate for the bacteriological transformation of the microbe.

Whereas the established niche for *Yersinia pestis* as an invasive organism often rendered it antagonistic to the humans, live and killed vaccines established a benign, if not beneficial, relationship between bacteria and the body. While live *Yersinia* pestis enter the body and replicate quickly through a variety of anti-inflammatory mechanisms that prevent their discovery by the immune system, and simultaneously suppress host innate immune responses, the heat-killed bacteria from the vaccine mounts none of these mechanisms. Instead, recognized as a foreign object quickly by the body, instigating immune response without fighting back.[75] Previously pathological agents, when brought into the imperial laboratory, were transformed into a commensal companion species, warped, altered, and integrated to be sacrificed to the human immune system.[76] As Chakrabarti notes, Latour warns, and Tsing emphasizes, though, organisms in an assemblage – whether in the laboratory, the streets, or some blurred line between the two – shape each other, and can rarely be expected to behave in ways logical to scientific (or bacteriological) practice.[77] The unique interaction of the human immune system and the *Yersinia pestis* bacteria in its new iteration was complex, differing from body to body and vaccine to vaccine. Thus, while heeded with a tremendous amount of optimism, iterations of the plague vaccine were relatively ineffective in the prevention or control of plague as a disease. In other

[75] Zogmin Du and Xiaoyi Wang, "Pathology and Pathogenesis of *Yersinia Pestis*," in R Yang and A Anisimov, eds., *Yersinia Pestis*: *Retrospective and Perspective* (New York: Springer, 2016), 193-222.

[76] Alison Bashford has a slightly different interpretation of this relationships, arguing for "vaccination as a kind of colonial contagion, as the deliberate circulation and proliferation of contagious matter along the imperial lines, and across the colonial borders of trade, travel and migration…an all too real cutaneous introduction of a foreign body into the self." Alison Bashford, *Imperial Hygiene*, 15.

[77] Anna Tsing, *The Mushroom at the End of the World,* 20-26; Bruno Latour, *The Pasteurization of France,* (Cambridge: Harvard University Press, 1988), 74-75, 122-123.

words, attempts to integrate *Yersinia pestis* into imperial biopolitics rarely went according to plan.

Two major attempts at the development and distribution of a plague vaccine garnered attention from the plague committee throughout the epidemic. The first was an antitoxin that migrated from Hong Kong to Bombay in a series of vials, carried by Alexandre Yersin. Drawing on novel methods developed by Kitasato and Emil Behring in their diphtheria research, Yersin attempted to develop an antitoxin for the plague from a serum of heat-killed *Yersinia pestis.*[78] While heralded with enthusiasm, the serum proved ineffective relatively quickly, as Surgeon-Captain Thomson, the Medical Officer in charge of the Parel Plague Hospital, reported that of 23 cases of plague in which the patient was treated with the antitoxic serum, 14 or 60.83 percent died.[79] Doctors and other bacteriologists noted local conditions as a major explanatory mechanism for the failure of the vaccine, relying on popular sanitary frameworks. Dr. Khan Bahadur N.H. Choksy, the head of the Arthur Road Infectious Diseases and Maratha Plague Hospitals, for example, noted that "The virulence of plague and the unfavourable conditions existing in India were not taken into account. It is therefore not surprising that the results obtained by Yersin when he went to Bombay in 1897 caused great disappointment."[80] From Choksy's perspective, the specificity of the disease in Bombay rendered the vaccine, developed in Hong Kong, ineffective.

In spite of its less-than-promising first trial in Bombay, members of the bacteriological committee remained fierce advocates for its use and development in the face of the epidemic. Chief among these advocates was Waldemar Haffkine, who emphasized the importance of

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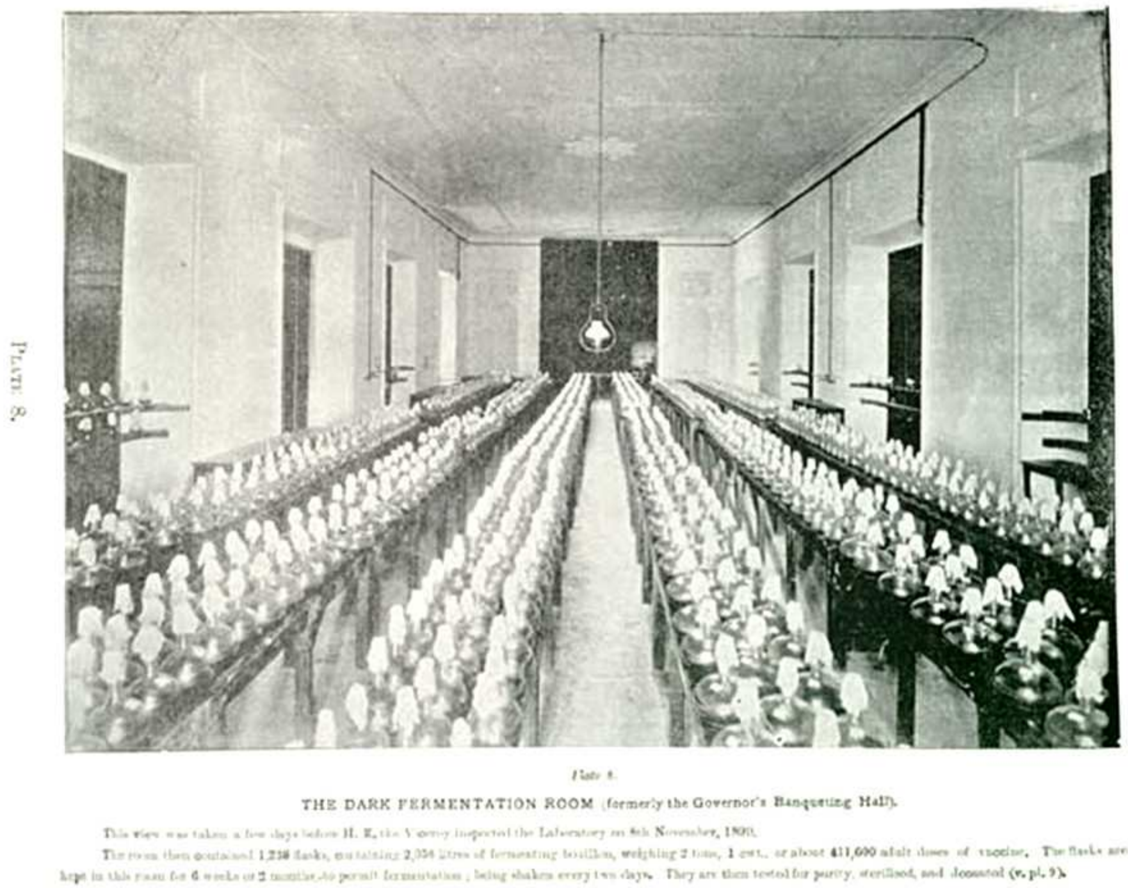
[78] Lyons, *Report(s) of the Plague Research (Lyons) Committee*, 14.
[79] Lyons, *Report(s),* 14.
[80] Khan Bahadur N.H. Choksy, "On Recent Progress in the Serum-Therapy of Plague," *The British Medical Journal* 1 (1908): 1282.

Yersin's antitoxin in the initial report of the plague committee, in which he neglected to sign off on the report because it recommended solely a continuation of existing sanitary procedures. In his addendum to the report, Haffkine wrote, "The preparation of an antitoxin serum, similar to that which is being experimented upon by Dr. Yersin, is a correct attempt."[81] Haffkine's interest in grounding prophylactic vaccines as a method of plague control had much to do with his own work. In October 1896, he was charged with the development of a plague antitoxin by the Plague Research Committee, alongside a characterization of the microbe. By December 1896, Haffkine had successfully developed his own live vaccine for plague and confirmed its efficacy by testing the vaccine on himself.[82] Despite misgivings by the Indian Plague Commission on the efficacy of the vaccine, the Commission moved Haffkine's Bacteriological laboratory to a larger space – Government House, Parel – and allowed for the mass manufacture of the vaccine, in hopes that it would confer immunity and reduce likelihood of death upon contraction of the disease.[83] The result of Haffkine's advocacy and influence was the integration of *Yersinia pestis* into a governmental laboratory space. While the bacteria replicated inside the bodies of fleas, humans, and rats on the streets of Bombay, in the laboratory it replicated under the watchful gaze of bacteriologists and their assistance, stewing in a solution of broth or ghee.

[81] Lyons, *Report(s)*, 27.
[82] Campbell, "Report of the Bombay Plague Committee," 207-208.
[83] J. Taylor, *Haffkine's Plague Vaccine*, (Calcutta: Indian Research Fund, 1933), 20-25.

Figure 16. Plate VII. The Dark Fermentation Room (formerly the Governor's Banquet Hall).



SOURCE: JK Condon, *Bombay Plague, Being a History of the Progress of plague in the Bombay Presidency from September 1896 to June 1899*, (Bombay: Education Society Steam Press, 1900), 119, IP/13/PC.6, Medical History of British India Collection, National Library of Scotland.

Bacteriological experiments and transformations of *Yersinia pestis* not only altered its relationship to the human immune system, but also altered its interactions with other organisms – in several cases, expanding the range of the bacteria into previously unaffected species. In his comments, Haffkine claims, "Since several weeks, horses, cows, goats, and sheep are being immunized at the Government lazaretto at Sewri."[84] Through these experiments, animals otherwise non-susceptible to the live bacteria were exposed to the heat-killed version in large quantities. Among those organisms that typically suffered from plague – namely, rats, mice, and guinea pigs (as well as humans), vaccination changed the immunological relationship of the bacteria to the animal. Experimental inoculations on monkeys, rats, and guinea pigs were thought to confer immunity at least for a short time (up to a month). However, these inoculation experiments were largely aimed at testing the effectiveness of the vaccine on humans, with no mass inoculation scheme proposed for wild rats – suggesting, again, as Lynteris et al. claim, that rats had been villainized as carriers of the disease.[85]

While the bacteria themselves came under the organizational gaze of the British government through bacteriological laboratories, once integrated into a vaccine, they also became a tool of biopolitical control for British sanitary regimes on the general population. With Foucauldian irony, the first experimental cases for the vaccine occurred among Bombay's incarcerated population. In December 1896, plague broke out at the Byculla House of Correction; of the 321 adult prisoners, 148 "submitted to inoculation"; of those, 14 cases of plague developed. Of the twelve non-inoculated cases, six died. Neither of the two inoculated cases died, and thus, "This result served as an inducement to many persons in Bombay, and the room in the laboratory became crowded with applicants for inoculation."[86] However, studies continued to indicate that the vaccine conferred less than the promised "full immunity," especially among those whose already shared their bodies with the live vaccine. Choksy found that in tests of the eight total iterations of the vaccine, including Haffkine's and Yersin's, case

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[84] Couchman, "Account of the plague administration," 17.
[85] Calmette, "The Prophylaxis of Plague by Preventive Inoculation," *The British Medical Journal* 2(1900): 1256.
[86]Condon, *Bombay Plague*, 113.

mortality still remained above sixty percent (except Kitasato's vaccine, which showed twenty-five percent mortality).[87] However, anecdotal evidence of the success of the Haffkine vaccine, combined with its relative ease of control (unlike rats or fleas) rendered immunization the most popular mitigation policy among the plague commission.[88] By December 1900, 1,628,696 doses of the vaccine had been distributed around India and thousands of vials around the world, marking another peculiarity – the widespread circulation of an infectious bacteria without an infectious pathway.

Conclusion

While the experiments on rats, fleas, and *Yersinia pestis* that occurred in Bombay were only a single part of a larger global effort to identify the etiology and epidemiology of plague, the scope and depth of experiments, which combined bacteriological and ecological interpretations of disease, reveal an urban ecology in flux. Through sanitary interventions, human migration, and bacteriological developments, the role of *Rattus rattus, Rattus norvegicus, Xenopsylla cheopis,* and *Yersinia pestis* changed within the urban ecosystem. Rats gained a laboratory and experimental niche that centered on both their life and death for knowledge formation; poisons altered their harborage and behavioral patterns, while trapping experiments put them in closer, more consistent, and often more threatening contact with their human counterparts. *Xenopsylla cheopis* similarly gained an experimental niche, while frequent contact with commission investigators increased its range of feeding; and *Yersinia pestis*, like its rat counterpart often valued for its death as often as life, gained both a new experimental and new

[87] Choksy, "Serumtherapy of Plague," 1282.
[88] WB Bannerman, "Compilation of Statistics of Inoculation with Haffkine's Anti-Plague Vaccine," March 1901, Calcutta Records 4, Sanitary Plague A Branch, Home Department, Government of India, nos. 187-189, pg. 8, National Archives of India.

immunological niche. Through the development of the Haffkine vaccine (and other attempted vaccines), *Yersinia pestis* transitioned from pathogen to cure – at least in theory.

While these experiments did little to reduce mortality from plague in the city or across India as a whole – attempts at rat control were limited and unsuccessful, while vaccination results revealed questionable efficacy – they carried lasting effects for the development of epidemiology. The integration of bacteriological research into a public health infrastructure during a local epidemic led to the incorporation of a complex, socio-ecological framework of disease that factors in human behavior, animal behavior, local ecological conditions. Experimental support of the flea-climate connection drew together ecological and bacteriological theories of disease to produce productive explanations for regional prevalence and persistence of the disease – an explanation still in use today when discussing the ecology of plague. Meanwhile, incorporation of animals into ecological and bacteriological frameworks of disease as vectors and as victims of disease afforded the agency of other organisms in imperial epidemiological practice, therefore contributing to a larger emerging epidemiological framework for vector-borne illness.

Chapter 5: *Salmonella enterica serovar Typhi* in Belfast, Northern Ireland, 1897-1910.

I. Introduction

In Dublin's fair city
Where the girls are so pretty
I first set my eyes on sweet Molly Malone
As she wheeled her wheelbarrow
Through the streets broad and narrow
Crying 'Cockles and mussels, alive, alive, oh'

Alive, alive, oh
Alive, alive, oh
Crying 'Cockles and mussels, alive, alive, oh'

She was a fishmonger
And sure t'was no wonder
For so were here mother and father before
And they wheeled their wheelbarrow
Through the streets broad and narrow
Crying 'cockles and mussels, alive, alive, oh'

Alive, alive, oh
Alive, alive, oh
Crying 'cockles and mussels, alive, alive, oh'

She died of a fever
And no one could save her
And that was the end of sweet Molly Malone
Now her ghost wheels her barrow
Through the streets broad and narrow
Crying 'cockles and mussels, alive, alive, oh'

Alive, alive, oh
Alive, alive, oh
Crying 'cockles and mussels, alive, alive, oh.[1]

Often referred to as the "unofficial song" of Dublin, the popular tune "Molly Malone" is a fine starting point for the final case study of this project: typhoid fever in Belfast, Northern

[1] The oldest known version of the song Molly Malone is dated to the 18th century, and currently located in the Dublin Writers Museum. This version of the song is attributed to James Yorkston, a scot, and was first published in the US in 1883.

Ireland. The reader may have discerned, of course, that Molly Malone is the song of *Dublin*, not of Belfast, and that the titular character seems to have lived and died in that city. However, the lyrics to Molly Malone reveal an important aspect of food culture in both cities at the turn of the nineteenth century. Namely, that shellfish were widely consumed, often in their raw "alive, alive" state; that street vendors wheeled their barrows "through the streets broad and narrow" to sell them to working class customers; and that those who consumed the shellfish sold by street vendors like Molly Malone might have "died of a fever."[2]

Though considered "the unofficial song of Dublin," one might thus consider Molly Malone to represent the experience of those living in a very particular set of Irish urban ecologies which, not long after the song was first recorded, gradually came under public and administrative attention because of a strange epidemiological phenomenon in Belfast: that the annual rates of typhoid fever in the city were higher than any other urban environment in the United Kingdom. It is possible that the epidemic, which struck the city in the years 1897-1901 and led to over 3200 cases annually, represented the largest outbreak of typhoid fever in the United Kingdom – and that the practice of shellfish consumption was central to its development and persistence. [3]

Typhoid fever has long occupied an important position in the history of public health in the United Kingdom. Attempts to identify and contain outbreaks shaped nascent public health systems, food safety policy, and imperial practice, as a rich historiography suggests. Scholars like Anne Hardy and Jacob Steere-Williams have noted the role of typhoid fever in the evolution of epidemiology and public health in the nineteenth century, and through their work provided a more nuanced consideration of how features like water contamination, sewage outfalls, and

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[2] A few other historians have concluded that Molly Malone likely died of typhoid fever. See Ann Hardy, "Exorcising Molly Malone: Typhoid and Shellfish Consumption in Urban Britain, 1860-1960," *Workshop Journal* 55(2003): 72-90.

[3] Henry Whitaker, Report on the Health of the City of Belfast for the Year 1903, Local Authority Records, LA/7/9/DA/13, Public Records Office Northern Ireland (PRONI).

infected foods at multiple scales mediated urban typhoid epidemics. These works provide valuable insight into the trajectory of typhoid fever in epidemiology, bacteriology, and public health in England and Wales, demonstrating how connections to national and international scientific networks often encouraged material changes to urban ecologies that lowered typhoid incidence. Through examinations of early nineteenth-century epidemics of typhoid, Hardy and Steere-Williams show how the link forged between water and typhoid led to "outbreak investigation" style epidemiological practices. These practices, they argue, often included a detailed ecological examination, which culminated in the isolation of a single case or cause.[4] It is through this style of investigation that local ecologies, and particularly their nonhuman inhabitants, become visible.

Other historical works employ a more generalist framework to consider typhoid in local and global perspective. In *Typhoid Fever: A History*, Richard Adler and Elise Mara argue that historically, typhoid fever epidemics correlated to two events: 1. Urban areas outgrowing their sources of clean water, and 2. The movement of armies.[5] Public health literature, until recently, has been largely guilty of similar generalization. Aside from case-by-case studies where a number of broad risk factors are examined in the wake of an outbreak, most public health analyses of *Salmonella typhi* stress lack of access to clean water, poor oral or food hygiene, or other direct routes of fecal-oral contamination.[6] The unique position of *Salmonella enterica* as an

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[4] Anne Hardy, *Salmonella Infections, Networks of Knowledge, and Public Health in Britain, 1880-1975,* (Oxford: Oxford University Press, 2015), 21-30; Anne Hardy, "Exorcizing Molly Malone: Typhoid and Shellfish Consumption in Urban Britain, 1860-1960," 55(2003): 72-90; Jacob Steere-Williams, "The Perfect Food and the Filth Disease: Milk-Borne Typhoid and Epidemiological Practice in late Victorian Britain," *Journal of the History of Medicine and Allied Sciences* 65(2010): 514-545; Jacob Steere-Williams, *The Filth Disease: Typhoid Fever and the Practices of Epidemiology in Victorian England* (Rochester: University of Rochester Press, 2020).

[5] Richard Adler and Elise Mara, *Typhoid Fever, A History*, Jefferson, North Carolina: McFarland & Company, 2016, 11-12.

[6] Only a handful of public health scholars to date have taken a socio-ecological approach to typhoid fever. The subject has been treated most thoroughly by A Aaron Jenkins, *A Nested Environmental Approach to Typhoid*

organism that is only pathological in humans certainly lends itself to readings at these scales. Statistical records and historiographical accounts suggest that interventions at the scale of the city or country – the completion of a water-based carriage system in London in 1855, replicated in other cities quickly thereafter; the introduction of stringent food safety laws in the early twentieth century across America, Britain, and a number of British colonies; and the introduction of more stringent sanitary practices in military campaigns and cantonments following the Anglo-Boer war, to name a few – had a significant impact on the rates of typhoid fever in historical urban and military spaces.[7] In these stories the presence of typhoid, much like the presence of tuberculosis, acts as a metonymy for poor sanitation. However, recent studies on endemic typhoid fever in Fiji suggest that examinations of the localized niche of *Salmonella enterica* Serovar *typhi* as an organism reveal distinct patterns of survivability that occur not only at the scale of the city, but at the scale of the ecosystem. Features like topography, soil composition, and temperature often combine with urban structures to influence the scope of an epidemic by influencing the life chances of the bacterium – a revelation which has the potential to cast historical typhoid epidemics in a new light.[8]

The problem of typhoid fever in Belfast provides an opportunity for novel analysis, as it was unusual in both scale and cause in the nineteenth century United Kingdom. Much to the

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Epidemiology in Central Division, Fiji, Doctoral Thesis, Edith Cowan University, retrieved from https://ro.ecu.edu.au/theses/1992 on July 12, 2019.

[7] Anne Hardy, *Salmonella Infections, Networks of Knowledge, and Public Health in Britain,* (Oxford: Oxford University Press, 2015), 21-63; Anne Hardy, "Exorcising Molly Malone: Typhoid and Shellfish Consumption in Urban Britain 1860-1960," *History Workshop Journal* 55(2003): 72-90; Jacob Steere-Williams, *The Filth Disease: Typhoid Fever and the Practices of Epidemiology in Victorian England* (Rochester: University of Rochester Press, 2020).

[8] Jenkins, *A Nested Environmental Approach to Typhoid Epidemiology.* AP Jenkins AP, S Jupiter, U Mueller, A Jenney, G Vosaki, V Rosa, A Naucukidi, K Mulholland, R Strugnell, M Kama, and P Horwitz, "Health at the Sub-catchment Scale: Typhoid and Its Environmental Determinants in Central Division, Fiji," *EcoHealth, 13*(2016): 633-51, doi: 10.1007/s10393-016-1152-6; R de Alwis, C Watson, B Nikolay, JH Lowry, NTV Thieu, TT Van, DTT Ngoc, K Rawalai, M Taufa, J Coriakula, CL Lau, EJ Nilles, WJ Edmunds, M Kama, S Baker, and J Cano, "Role of Environmental Factors in Shaping Spatial Distribution of Salmonella enterica Serovar Typhi, Fiji," *Emerg Infect Dis* 24(2018):284-293. doi: 10.3201/eid2402.170704.

frustration of public health administrators, water in Belfast was filtered "very carefully," and the water was of "very good character."[9] And yet, public health officials noted, "The mortality from enteric fever during the past 25 years in Belfast had been so great that no other city or town of the United Kingdom had even approached it. During the years 1900, 1901, and 1902 the annual death rate from this disease was 0.72 per 1,000 of population in Belfast as compared with 0.34 in Dublin, 0.13 in Manchester, and 0.15 in England and Wales."[10] By the early twentieth century, the unusual elevation in typhoid fever rates in Belfast had been traced (controversially) to widespread local practices of consuming raw shellfish collected from Belfast Lough. James Lorrain Smith, medical officer of health in Belfast, confirmed the widespread presence of typhoid in shellfish using newly minted bacteriological practices.[11] While public health officials occasionally traced outbreaks or small epidemics (under 100 people) of typhoid in other spaces in the United Kingdom to shellfish, never had outbreaks as widespread or continuous as Belfast's been traced to local fauna contaminated with the bacteria.

This chapter will explore the social, ecological, and economic dynamics that led to Belfast's elevated typhoid fever rates. Drawing a comparative lens between the two largest (and most ecologically similar) cities in Ireland, Dublin and Belfast, it will argue that the typhoid epidemic that struck Belfast in 1897, less than a decade after the introduction of a "modern" water carriage system of sewage disposal, and despite the absence of traditional risk factors like

[9] "Belfast Water Bill: Proceedings in Select Committee. Stoneyford Opposition Withdrawn. The Typhoid Question," *Northern Whig* (Belfast, Ireland), Thursday 04 May 1899.

[10] "Great Britain and Ireland: Report from Belfast. Conclusions of Commission Relative to Prevalence of Enteric Fever. Unsanitary Shellfish as Cause of Disease. Mortality from Tuberculosis," *Public Health Reports (1896-19070)* 23(1908): 995-996.

[11] James Lorrain Smith, "An Investigation into the Conditions Affecting the Occurrence of Typhoid Fever in Belfast," *Journal of Hygiene* 4(1904): 407-433; James Lorrain Smith, "A Study of the Epidemic of Typhoid Fever in Belfast, 1898," *The British Medical Journal* 1 (1899): 193-197; Charles A. Cameron, "Sir Charles Cameron's Report," *The British Medical Journal* 2(1891): 1167-1168; Charles Cameron, "Report on the State of Public Health in Dublin and the Sanitary Work Performed Therein," 1881-1889), Dublin Corporation & c. Records, Dublin City Archives.

overcrowding, poverty, or generalized "poor sanitation", can only be understood by regarding the city as an ecosystem – and the economic, political, and social forces of an industrial "boom town" as interacting with and shaping that ecosystem. I will argue that through this comparison, typhoid becomes complex, dynamic and place-specific; its occurrence in Belfast was the result of a confluence of unique factors, including ecological characteristics like topography, rainfall, and river basin ecology; and social and urban environmental characteristics such as urban growth fueled by imperial connections, housing placement, water systems, sewage systems, class, industry, and local dietary practices. Through this unique confluence of features, *Salmonella enterica typhi* found a niche in Belfast.

Urban Systems and Sub-catchment Ecologies* – Salmonella enterica serovar Typhi *in Environmental Perspective

Salmonella enterica Serovar *typhi* is among the oldest bacteria known to be pathogenic to man. Recent paleoarcheological work traces the bacteria (or at least, its direct ancestor) as far back as the Plague of Athens in 430 BC, corroborated by a description from Thucydides, with estimates for its ancestral strain going back as far as 50,000 years.[12] Despite its anecdotal prevalence, identification of typhoid epidemics using solely historical sources was nearly impossible until the isolation of the bacteria in Karl Eberth's laboratory in 1884 because of its relatively common symptoms – fever, rash, and stomach pain, with either constipation or

[12] Manolis J. Papagrigorakis, Christos Yapijakis, Philippos N. Synodinos, and Effie Baziotopoulou-Valavani , "DNA examination of ancient dental pulp incriminates typhoid fever as a probable cause of the Plague of Athens," Int J Infect Dis. 10(2006): 206-14. Published online 2006 Jan 18; Manolis J. Papagrigorakis, Philippos N. Synodinos, and Christos Yapijakis, "Ancient typhoid epidemic reveals possible ancestral strain of *Salmonella enterica* serovar Typhi," *Infection, Genetics and Evolution* 7(2007): 126-127.

diarrhea, presenting 1-2 weeks after ingesting the bacteria and lasting continuously for 3-4 weeks.[13]

Unlike plague and tuberculosis, both of which infect other organisms (who in turn may act as vectors for the disease), *Salmonella typhi* is solely pathogenic in humans.[14] In order to develop typhoid fever, a potential case must come into contact with the bodily fluid of an infected person – most frequently fecal matter or urine.[15] Once ingested, *Salmonella typhi* moves through the human digestive system and is thought to pass through the mucosal membrane of the intestine and into the reticuloendothelial system and gall bladder. The organism then enters the bloodstream relatively quickly, where it incubates for an average of 8-14 days.[16] Cases then present with several non-specific symptoms of bacteremia for several weeks. In the case of typhoid carriers, who often present no symptoms but shed the bacteria in their excretions, it is thought that the bacteria remain in the gall bladder and do not enter the bloodstream in the same manner as clinical typhoid.[17]

Two important features of clinical typhoid fever are important to its epidemic progression. First, most people who become infected with typhoid will develop an immunity to the disease. Second, contact with a relatively high number of organisms is required to achieve pathogenesis, with an average of 10^4 organisms required to induce illness. However, higher

[13] "Typhoid Factsheet," World Health Organization, Last updated 31 January 2018. https://www.who.int/news-room/fact-sheets/detail/typhoid.
[14] Evolutionary evidence suggests that the ancestor of present-day Salmonella enterica serovar Typhi may have been capable of infecting both humans and animals. See Papagrigorakis et al., "Ancient typhoid epidemic," 126-127.
[15] Denise M. Monack, "*Salmonella* persistence and transmission strategies," *Current Opinion in Microbiology* 15(2012): 100-107; Aaron Peter Jenkins, Stacy Jupiter, Ute Mueller, Adam Jenney, Gandercillar Vosaki, Varanisese Rosa, Alanieta Naucukidi, Kim Mulholland, Richard Strugnell, Mike Kama, and Pierre Horwitz, "Health at the Sub-catchment Scale: Typhoid and Its Environmental Determinants in Central Division, Fiji," *EcoHealth* 13(2016): 633-651.
[16] Myron M. Levine, Carol O. Tacket, and Marcelo B. Sztein, "Host-*Salmonella* interaction: human trials," 1271.
[17] Monack, "*Salmonella* persistence and transmission strategies," 100-107.

doses tend to shorten the median incubation period by about 1.5 days per 10^2 organisms.[18] This feature is particularly interesting when considering large-scale outbreaks or epidemics of typhoid fever. Methods of indirect transmission can be as diverse as contamination of food with fecal matter (which would likely lead to a high concentration of organisms in a single location) and contamination of water sources (in which organisms from a single source would be more diluted). The pathogenicity of typhoid therefore may rely on a number of ecological and environmental factors that allow or prevent high concentrations of the bacteria.

The challenges presented by distinct environments have led some epidemiologists to consider typhoid as a an organism that thrives best as a result of a combination of social and ecological features. [19] These characteristics have been divided into distinct modes of infection, referred to as "short cycle" and "long cycle" transmission.[20] "Short-cycle" transmission focus largely on behavioral and socio-cultural characteristics, and operates at the scale of an individual, a household, or a cluster of households. These cases often occur when an infected member (or an asymptomatic carrier) contaminates food or water, which then results in the infection of those

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[18] Levine et al., "Host-*Salmonella* interaction: human trials," 1271; and Jenkins, "A nested environmental approach to Typhoid Fever," 3.

[19] Jenkins, "A Nested Environmental Approach to Typhoid Epidemiology in Central Division, Fiji,"; M Ahern, RS Kovats, P Wilkinson, R Few, and F. Matthies," Global Health Impacts of Floods: Epidemiological Evidence," *Epidemiology Review* 27(2005): 36-46; A Akullian, E Ng'eno, AI Matheson, L Cosmas, D Macharia, B Fields, G Bigogo, M Mugoh, G John-Stewart, JL Walson, and J Wakefield, "Environmental Transmission of Typhoid Fever in an Urban Slum," *PLoS Neglected Tropical Diseases,* 9(2005): e00004212. J Gonzalez-Guzmán, "An Epidemiological Model for Direct and Indirect Transmission of Typhoid Fever," *Journal of Mathematic Biosciences* 96(1989): 33-46; Jenkins, "A Nested Environmental Approach," 3-4. VE Pitzer, CC Bowles, S Baker, et al., "Predicting the Impact of Vaccination on the Transmission Dynamics of Typhoid in South Asia: A Mathematical Modeling Study," *PLoS Neglected Tropical Diseases* 8(2014): e2642. Jonathan A. Polonsky, Isabel Martinez-Pino, Fabienne Nackers, et al., "Descriptive Epidemiology of Typhoid Fever during an Epidemic in Harare, Zimbabwe, 2012," *PLoS One*, 9(2014): e114702. Doi: 10.1371/journal.pone.0114702.

[20] Jonathan A. Polonsky, Isabel Martínez-Pino, Fabienne Nackers, Prosper Chonzi, Portia Manangazira, Michel Van Herp, Peter Maes, Klaudia Porten, and Francisco J. Luquero, "Descriptive Epidemiology of Typhoid Fever During an Epidemic in Harare, Zimbabwe, 2012,"*PLoS One* 9(12): e114702. doi:10.1371/journal.pone.0114702; Aaron P. Jenkins, Stacy D. Jupiter, Adam Jenney, Varanisese Rosa, Alanieta Naucukidi, Namrata Prasad, Gandercillar Vosaki, Kim Mulholland, Richard Strugnell, Mike Kama, John A. Crump, and Pierre Horwitz, "Environmental Foundations of Typhoid Fever in the Fijian Residential Setting," *International Journal of Environmental Research and Public Health* 16(2019): 2407-2427. Doi: 10.3390/ijerph16132407.

sharing common food or water sources.[21] Studies of short-cycle factors often reveal features of isolated epidemics and person-to-person transmission dynamics, which can influence local public health intervention and education. While studies reveal variation in local transmission, the most commonly implicated risk factors for typhoid include a lack of handwashing or, euphemistically "poor personal hygiene," alongside poor microbial controls on food and water sources, crowding, low socio-economic status, and poor localized sanitation in the form of waste removal, housing structure, and use of latrines instead of water closets.[22] Municipal factors like water contamination or food regulation are often implicated in these analyses, and become the target scale of intervention.[23]

In areas suffering endemic or cyclic typhoid fever, studies designed around short-cycle transmission can ignore structural and ecological features that contribute to recurrence or persistence of the bacteria by providing a suitable ecological niche. Recent analysis of typhoid fever epidemics in high-risk areas have turned to spatio-temporal analysis to explain "long-cycle" risk factors for the epidemics.[24] These studies expand the scale of analysis for typhoid transmission to consider the importance of landscape morphology, presence of watershed or

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[21] Amruta Radhakrishnan, Daina Als, Eric D. Mintz, et al., "Introductory Article on Global Burden and Epidemiology of Typhoid Fever," *American Journal of Tropical Medicine and Hygiene* 99(2018): supplement 4-9, doi: 10.4269/ajtmh.18-0032; R Leon Ochiai, Camilo J Acosta, M Carolina Danovaro-Holliday, et al., "A Study of Typhoid Fever in Five Asian Countries: Disease Burden and Implications for Controls," *Bulletin of the World Health Organization* 86(2008): 260-268; V Mogasale, B Maskery, RL Ochiai, et al., "Burden of Typhoid Fever in Low-Income and Middle-Income Countries: A Systematic, Literature-Based Update with Risk Factor Adjustment," *Lancet Global Health* 2(2014): e570-80, doi: 10.1016/S2214-109X(14)70301-8.

[22]Jonathan A. Polonsky, et al., "Descriptive Epidemiology of Typhoid Fever During an Epidemic in Harare, Zimbabwe, 2012,"; Aaron P. Jenkins, Stacy D. Jupiter, Adam Jenney, Varanisese Rosa, Alanieta Naucukidi, Namrata Prasad, Gandercillar Vosaki, Kim Mulholland, Richard Strugnell, Mike Kama, John A. Crump, and Pierre Horwitz, "Environmental Foundations of Typhoid Fever in the Fijian Residential Setting," *International Journal of Environmental Research and Public Health* 16(2019): 2407-2427. Doi: 10.3390/ijerph16132407;

[23] Aaron Jenkins, *A Nested Environmental Approach to Typhoid Epidemiology in Central Division, Fiji,* Doctoral Thesis, Edith Cowan University, retrieved from https://ro.ecu.edu.au/theses/1992.

[24] Jenkins, *A Nested Environmental Approach,*1-17.

catchment areas, river characteristics, and other "upstream" drivers of determinants of health.[25] In a study on the ecological determinants of typhoid morbidity in Central Division, Fiji, Aaron Jenkins et al. found that the complex ecology and drainage networks of sub-catchment areas often fostered the spread of typhoid fever across landscapes and even influenced *Salmonella typhi* pathology. In a large sub-catchment area, Jenkins et al. concluded, the centralization of water drainage into a single, often large and meandering, river allowed pollutants to accumulate with water runoff, which increased nutrient concentrations, which in turn fostered the growth of *Salmonella typhi* and faecal coliform bacteria.[26] Jenkins et al. suggest that pollutant particles may contribute to a "micro niche" for *Salmonella* survival and replication, "providing shelter from grazing protozoans" while simultaneously providing a higher concentration of free amino acids and sugars than clean water.[27] Once within water sources, the high sedimentation common in sub-catchment systems reduces oxygen content in water. This low-oxygen environment may then "trigger increased pathogenincity" in *Salmonella* bacteria.[28]

In addition to fostering an ideal aquatic environment for the survival of *Salmonella* typhi bacteria, sub-catchment ecologies proved challenging for sanitary infrastructure, with their soft, eroded soil often inadequately supporting concrete septic tanks and sewers.[29] Meanwhile, the soils themselves may contribute to the survival of the bacteria outside of its human hosts. According to McGinnis and Dewalle, *S. typhi* has been found to live at least 42 days in sodden

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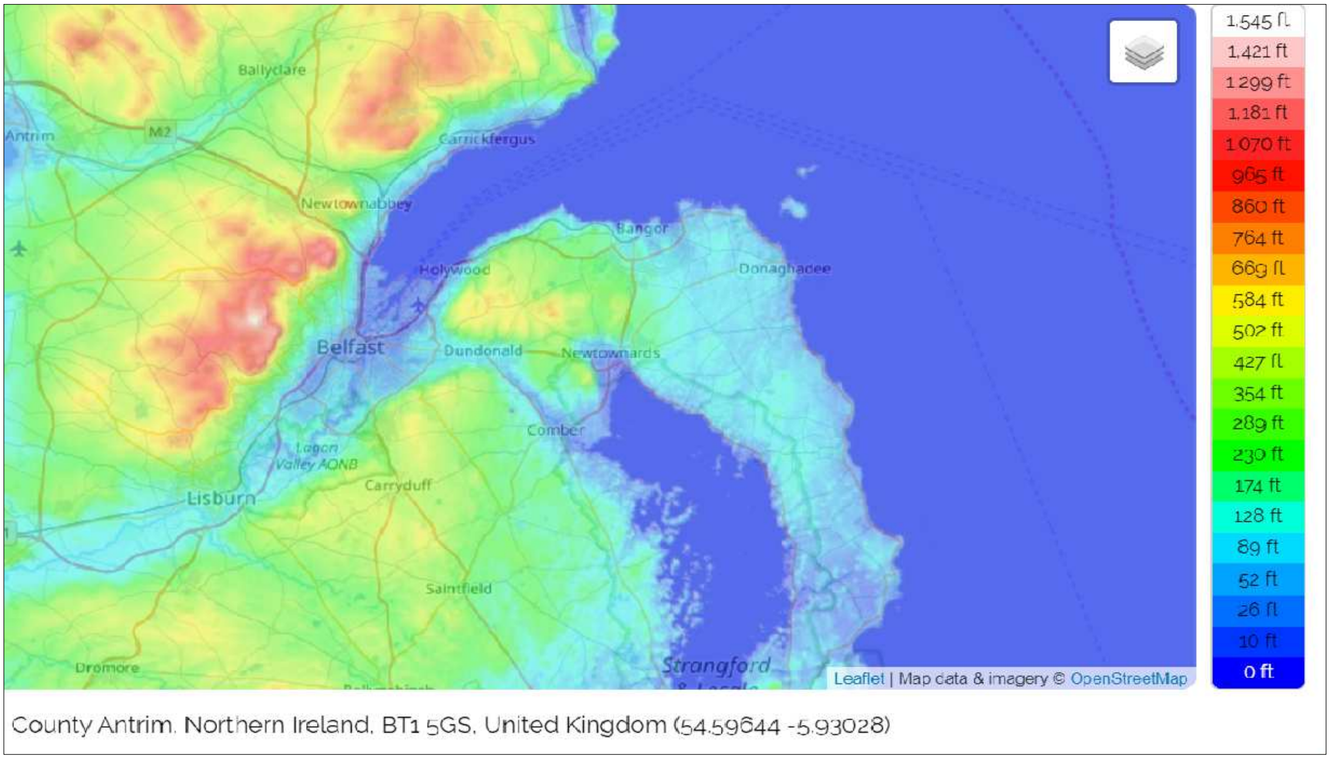
[25] Jenkins, *A Nested Environmental Approach,* 9.
[26] Jenkins et al., "Health at the Subcatchement Scale," 644.
[27] James A. McGinnis and Foppe DeWalle, "The Movement of Typhoid Organisms in Saturated, Permeable Soil," *Journal of the American Water Works Association* 75(1983): 266-271; Duane F. Berry and Charles Hagedorn, "Soil and Groundwater Transport of Microorganisms," in *Assessing Ecological Risks of Biotechnology,*(Stoneham: Butterworth-Heinemann, 1991): 57-73
[28] Jenkins, "Health at the Subcatchment Scale," 647; Mollie D. Winfield and Eduardo A. Groisman, "Minireview: Role of Nonhost Environments in the Lifestyles of *Salmonella* and *Escherichia coli*," *Applied and Environmental Microbiology* 69(2003): 3687-3694.
[29] Jenkins, "Health at the Subcatchment Scale," 645.

soil, and that the bacteria could be transported as far as 61 meters to the nearest riverbed.[30] The combination of hospitable soils, nutrient-dense waters, and sanitary challenges inherent in catchment basin structure suggest that sub-catchment basins – and urban settlements on sub-catchment basins in particular –represent a unique niche for typhoid fever.

It is in these sub-catchment level dynamics that Belfast differs from other UK cities that suffered typhoid epidemics in the nineteenth century. The name Belfast itself denotes its position in the landscape, as the name is derived from the Irish *Beal Feirste, Beal* meaning "mouth" and *Fierste* roughly meaning "sand bank ford."[31] As landscapes carved from glacial retreat, the east and northeast of Ireland are formed of basalt hills and drumlins, and covered with arable loams, and sits at the mouth of the slow, meandering Lagan river (Figure 17).

Figure 17. Elevation Map of the Lagan River Valley, demonstrating the position of Belfast and its environs.

[30] Winfield and Groisman, 3690.
[31] "The Gaelic Placenames of Belfast," Belfast City Council, https://www.belfastcity.gov.uk/nmsruntime/saveasdialog.aspx?lID=2910&sID=2272



SOURCE: “Belfast topographic map, relief map, elevations map.” https://en-gb.topographic-map.com/maps/0r/Belfast/. Map data and imagery © OpenStreetMap

The position of Belfast at the lowest elevation point in a subcatchment basin, the composition of the city’s soils, and its high average rainfall suggest the city may support a number of “long-cycle” risk factors for typhoid fever; in other words, the wider landscape ecology in which the city was formed may provide increased survival and replication chances for typhoid fever, and therefore form part of an explanation for consistently high typhoid fever rates. Typhoid transmission, however, still requires individual points of contact – which in the case of Belfast’s epidemic, involved other, nonhuman organisms that thrived within its estuarial ecology.

Shallow-sea vectors?

While *Salmonella typhi* is not pathogenic to nonhumans, other organisms, particularly aquatic organisms, act as carriers of the bacteria. The most well-known (and most pertinent to this study) carrier is class *Bivalvia*, which thrives in coastal estuarial ecosystems and under cultivation by humans. Bivalves are filter-feeders, who procure sustenance by inhaling nutrient-laden water through the inhalant siphon. The water is then passed over a set of gills and food is extracted, before the remaining water is expelled through the exhalant siphon.[32] This feature, ideal for accumulation of particles, along with bivalves' tendency to burrow into the substrate of coastal regions, has made bivalves a common marker for environmental contamination in recent years – and a marker for pollution as early as 1880.[33]

The relationship of bivalve shellfish to typhoid fever transmission is a complex one, and dependent on the position of bivalve shellfish not only to the human food chain, but to the ecosystem. It is therefore important to consider how land use changes around burgeoning port cities, including the rerouting of water systems, the building of sewers (which often empty into coastal areas), and the management of docks and waterways for shipping have a distinct impact on shellfish and other features of coastal ecology. These changes can alter the habitat of shellfish and change the nature of the organic material filtered by these organisms. To incite an epidemic of typhoid, at least one human (but likely more, given the concentration of typhoid microbes required to induce illness) must secrete typhoid bacteria; the secretion must make its way into an estuarial habitat, often through a sewage outfall, flooding, or other drainage; and it must be absorbed by the shellfish in sufficient concentration to induce illness once the organism is selected and eaten. Studies indicate that typhoid bacteria do not replicate inside shellfish once absorbed, which places its status as a vector (in the technical definition of the term) in question –

[32] Elizabeth Gosling, *Bivalve Molluscs: Biology, Ecology and Culture* (Oxford: Fishing New Books (Blackwell Press), 2003): 4-5.
[33] Gosling, *Bivalve Molluscs*, 11. Charles Cameron, "On Sewage in Oysters, presented at the Forty-Eighth Annual Meeting of the British Medical Association," *British Medical Journal*, 2(1880): 465-480.

but that the organism's position as a filter feeder allows it to magnify public health problems through bacterial or viral accumulation.[34]

Speculations as to the relationship between typhoid fever incidence and cockle consumption in Belfast emerged in parallel with a wider British epidemiological trend linking shellfish and *Salmonella typhi*.[35] This widely-publicized connection, which Anne Hardy argues may have generated the "first documented food scares," in England and Wales largely concentrated around oyster consumption, but occasionally extended to "the plebian cockle, the coster periwinkle, and the hooligan mussel."[36] Oysters remained foremost in the national imagination, however, and an inquiry into the conditions of the English coastal oyster industry emerged in 1893.[37] It was not the oyster that drew the attention of the Public Health Committee in Belfast, however, nor of the Royal Commission established to investigate the high death rate of Belfast in 1907 – but the "plebian" cockle.[38]

The cockle is widely distributed along the coasts of Britain and Ireland, largely inhabiting coastal estuaries and sandy bays. A distinctly North Atlantic organism, the cockle ranges in habitat from Northern Norway to the coast of Senegal. Unlike many other bivalves, it is a shallow burrower, often no more than 5 cm below surface sediment – a feature which lends it to leisurely predation by humans and a number of other organisms.[39] While cockles are often commercially fished, individual gathering as a supplement to diet has been its most common

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[34] Scott R. Rippey, "Infectious Diseases Associated with Molluscan Shellfish Consumption," *Clinical Microbiology Reviews,* 7(1994): 419-425.
[35] Anne Hardy, "Exorcizing Molly Malone,"; Anne Hardy, *Salmonella Infections*, 43-63.
[36] Sir James Crichton-Browne, 'Dangers in our Food', Sanitary Journal 8 (1902): 185.
[37] "Report from the Select Committee on Sea Fisheries; together with the proceedings of the committee, minutes of evidence, appendix and index," 1893-94, House of Commons Papers, Reports of Committees, British Parliamentary Papers Online, accessed 27 February 2020.
[38] Ann Hardy, *Salmonella Infections*, 45.
[39] H Tyler-Walters, "*Cerastoderma edule* Common Cockle," in *Marine Life information Network: Biology and Sensitivity Key Information Reviews* [online], H Tyler-Walters and K Hiscock (eds.), (Plymouth: Marine Biological Association of the United Kingdom, 2007), https://www.marlin.ac.uk/species/detail/1384.

mode of interaction with humans throughout history.[40] In both Dublin and Belfast, as the song suggests, consumption of the cockle was thought to be widespread.

While estuarial ecology and cultural practice play a large role in shellfish-related typhoid transmission, the historical role of the cockle and other shellfish in *Salmonella typhi* outbreaks cannot be separated from the construction of urban ports at the end of the nineteenth century. As historian Harold Platt has argued, industrial cities must be considered not only as shocking to social sensibilities and processes, but as a series of shocks to the landscape – and to the ecosystem present within that landscape.[41] The rapid demographic growth, extractive practices, disorganization and social inequality that characterized the mid-nineteenth century industrial city changed the relationship of people – and different groups of people – to the land, air, and water; and subsequently, corrective practices, including water carriage systems, housing improvement schemes, and food safety measures altered that relationship further.

As the only industrial city in Ireland, Belfast occupied a peculiar position within the United Kingdom and within the British Empire. Plagued with religious and partisan tensions that remain artefacts of an earlier phase of colonial conquest, Belfast – and Ireland as a whole – became very consciously incorporated into the United Kingdom at the beginning of the nineteenth century. As the rise of Peelism and the events of the Irish Potato Famine illustrated, however, the extractive and pseudo-colonial relationship between the two islands continued well beyond the point of unification.[42] Belfast occupied a complicated and often contested position within this political ecology. Intellectually, often politically, and somewhat economically, Belfast was incorporated into the British metropole; however, the trajectory of the city in the late

[40] H Tyler Walters, "*Cerastoderma edule* Common Cockle," xx-xxx.
[41] Harold Platt, *Shock Cities: The Environmental Transformation and Reform of Manchester and Chicago,* (Chicago: University of Chicago Press, 2005), 12.
[42] David Nally, *Human Encumbrances: Political Violence and the Great Irish Famine*, (Notre Dame: Notre Dame Press, 2011), 129-175.

nineteenth century aligns with several key characteristics of what Jim Belich has attributed to settler colonialism: a boom-bust economic cycle, driven by specialization in shipping and production intertwined with metropolitan interests; development driven by financial investment and speculation; growth based on proximity to oceanic shipping routes; and explosive population growth.[43] Thus, while it might be unwise to categorize Belfast as a settler colonial city, or a colonial city generally, I will argue that its integration into the British metropole – and more importantly, the city government's eagerness to be included in this categorization – fostered these "shocks" to the landscape, facilitating land use changes that, when combined with local ecology, widened the niche for microbes like *Salmonella enterica typhi.*

Industrial Belfast and the* Salmonella typhi *Niche

Belfast, from its earliest recorded days to the present, has occupied a dynamic and often significant role in the British Empire.[44] Coveted as a strategic location in the crossing of the Lagan and in its proximity to England, Belfast was labelled "one of the high water marks of English settlement in Ireland" as far back as the thirteenth century.[45] The settlement was certainly an area of contention between the Irish kings and English invaders; ownership passed back and forth between the O'Neill clan, competing Gaelic families, and the occasional English planter from the 14th through 17th centuries.[46]

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[43] Jim Belich, *Replenishing the Earth: The Settler Revolution and the Rise of the Anglo-World, 1783-1939,* (Oxford: Oxford University Press, 2011), 15-41.
[44] Charles B. Brett, Notes on the Topography of Old Belfast, D271/10, Leases and Correspondence Records Relating to Property in Belfast, PRONI.
[45] Sean Connolly, *Belfast 400: People, Places, Ideas,* 19.
[46] Brett, Topography of Old Belfast, 4-12.

Despite its strategic location at the mouth of the Lagan and contested history, sources suggest that by the year 1690, Belfast was a settlement of no more than 300 houses. It was not until the mid-nineteenth century that the town achieved economic importance – perhaps a result of tensions between the area's geographical advantages and topographical challenges. Historian Sean Connolly described the inlet of Belfast Lough as "Initially unpromising." The bay was silt-covered, turbid, and shallow; it held no significant deposits of iron or coal.[47] The Lagan was "a misfit stream, draining rather ineffectually a grand valley fashioned not by its own waters but by glacial action."[48] Nevertheless, the geographical importance of the site for English commercialism won out, and exports of butter and beef, soon replaced by a rising export trade in linen, grew the town throughout the 18th century. While still behind Cork and Dublin as sites of commercial importance, by the early 18th century Belfast had become the third-busiest port in Ireland.[49] As early as 1823, a local historian drew comparison between Belfast and other nascent industrializing cities of the British Isles: "Belfast is to Ireland what Glasgow and Liverpool are to their respective kingdoms, and it has actually been compared to these places by travelers as well as in its general appearance, as in the manners and sentiments of its inhabitants."[50]

As the importance of the port grew for shipping and manufacturing, its topographical challenges became an unnecessary hindrance. Like the rest of Britain, parliamentary reform in 1832 drastically changed the political landscape of Belfast, transferring power from the hands of the Donegall family and the Belfast Corporation to an elected mayor and council by the end of

[47] Connolly, *Belfast 400*, 14-15
[48] Stephen Royle, "Workshop of the Empire," 203.
[49] Connolly, *Belfast 400*, 20.
[50] The History of the Town of Belfast, with an accurate account of its former and present state: to which are added a statistical survey of the Parish of Belfast, and a description of some remarkable antiquities in its neighborhood, 1823, pg. 131, BPB1823.2, BPB Collection, Linen Hall Library.

the decade.[51] Eager to flex its new powers, the town council embarked immediately on a series of improvement projects, erecting a major thoroughfare in the center of town, purchasing land between the town and the Lagan for cattle sales, and establishing a number of cultural institutions and banks.[52] Thus, in 1832, plans for reclaiming "the sloblands", or the shores of Belfast bay, and engineering the bay for deep-water ships, were underway. While from 1810 onward a series of local engineers had proposed methods for deepening the bay, much like in Bombay and Melbourne, the plans of a London engineer were selected and enacted over those of Belfast residents. James Walker's plan to "dredge deepwater channels" into the harbour commenced a few years later, the sludge from which was piled to create Queen's Island – the location of Harland and Wolff's shipyard later in the century.[53] The city's position as a major port for Ireland was solidified by the completion of a rail network in the 1840s.[54] It is perhaps unsurprising, therefore, that when the Famine struck in 1848, sufferers from rural Ulster migrated into the city in search of employment in its booming linen industry. By 1851, the city had become the second largest urban center in Ireland, surpassing Cork.[55]

While Belfast grew steadily in population and economic importance over the course of the nineteenth century, the town's true "boom" occurred with the simultaneous expansion of the linen industry and construction of its first shipyard in 1851.[56] Its growth largely relied on

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[51] Connolly, "Belfast: The Rise and Fall of a Civic Culture?", in *Belfast: The Emerging City*, ed. Olwen Purdue, 31. This break was not as radical as it might seem, Connolly notes, as one of Lord Donegall's sons was elected to the new governing body.
[52] Connolly, "Belfast: The Rise and Fall of a Civic Culture?", 38-39.
[53] Royle, *Belfast 400*, 203. Sir David J. Owen, *A Short History of the Port of Belfast* (Belfast: Mayne Boyd, 1917), 38.
[54] A.C. Hepburn, *A Past Apart: Studies in the History of Catholic Belfast, 1850-1950,* (Belfast: Ulster Historical Foundation, 1996), pg. 3, Irish Studies Collection, Linen Hall Library.
[55] Hepburn, *A Past Apart,* 3-5.
[56] CD Purdon, The Sanitary State of the Belfast Factory District, During Ten Years (1864-1873 inclusive), under various aspects (1877) (Belfast: H. Adair, 1877), pg. 3, Pamphlets, Linen Hall Library; Michael Moss and John R. Hume, *Shipbuilders to the World: 125 Years of Harland and Wolff, Belfast, 1861-1986*, (Belfast: Blackstaff Press, 1986):

overseas markets, the largest of which was America through the early twentieth century, but also included France, Romania, Spain, Greece, Australia, Russia, and the West Indies, among others.[57] The boosterism of the Linen Merchants' Association played a large role in the industry's growth, as well, establishing firm political and technological links with major trading partners.[58] This unique combination of economic and political interests proved effective, as by 1894, Belfast mills employed 28,000 women and 6,000 men, minding 724,000 flax spinning spindles, spinning 644 million miles of yarn annually.[59]

The shipyard became the site of the Harland & Wolff shipping company, one of the primary employers in Belfast from 1890 onward, and by the mid-twentieth century, the largest dry dock in the world.[60] Often on the forefront of shipping technology (Harland and Wolff are credited with pioneering a "new, technically advanced tonnage" for passenger ships and early adoption of triple-expansion engines), the shipyard built the majority of the White Star Line fleet, which provided passenger and cargo services between British imperial port towns - particularly between Liverpool, Australia, and New Zealand (often via Cape Town) and between the British Empire and America – most often Liverpool to New York or Boston, and by the early twentieth century Liverpool to Montreal or Quebec City.[61] The most infamous of this line was the *Titanic*, completed at the Harland and Wolff shipyard in 1912 – and by no means the only ship of the

[57]Aiken and Royle, "Markets and Messages," 4.
[58] Aiken and Royle, "Markets and Messages," 8-14.
[59] Report of the Belfast Health Committee to the Local Government Board of Ireland, 1908, pg. 1, LA/7/3/JA/22, Local Authority Records of the Belfast Corporation and County Borough Council, PRONI; Emily Boyle, "'Linenopolis': the rise of the textile industry," in *Belfast: The Making of the City*, J.C. Beckett ed., (Belfast: The Appletree Press, 1988), 46-47; Edward James Aiken and Stephen A. Royle, "Markets and Messages: Linenopolis Meets the World," in *Belfast: The Emerging City,* ed. Olwen Purdue, (Dublin: Irish Academic Press, 2013), 1-23.
[60] Michael McCaughan, *The Birth of the Titanic* (Montreal: McGill-Queen's University Press, 1998), 4.
[61]"White Star Line/Oceanic Steamship Company/White Star Line of Boston Packets," Ships List Database, accessed 7 September 2019, http://www.theshipslist.com/ships/lines/whitestar.shtml.

White Star Line to end its commission at the bottom of the ocean.[62] This relationship led to the humorous (if partisan) adage common in Belfast today: "The Irish built the ships, and the British sank them." Activity around the shipyard was a major source of employment for men in Belfast, with 15,000 men involved directly in the production of ships, and thousands more employed in complementary industries like ropemaking, iron casting, and glass production. Between Harland and Wolff and Workman, Clark, and Co., and MacIlwaine and McColl, the shipyards produced nearly 80,000 tons annually by 1890.[63]

By 1870, Belfast had become the fastest growing city in the United Kingdom, outpacing its industrialized rivals, Manchester, Liverpool, and Glasgow.[64] While other towns in Ireland stagnated and industry declined, Belfast continued to grow, fueled by British investors keen on profit and proximity to industrial giants like Liverpool and Manchester.[65] By 1890, Belfast's population had blossomed to over 230,000, nearly twelve times its 1800 size, surpassing Dublin as the largest city in Ireland.[66]

The Sanitary State of Belfast

Despite its rapid growth, Belfast was notable for its relatively high quality of life. The availability of employment for both men and women (and therefore the possibility of a multiple wage household), clean water supply, and widely available housing stock meant that "Belfast's laboring classes had the potential to enjoy a better standard of living than might be experienced

[62] Michael McCaughan, *Steel & Iron, Ships & Men: Shipbuilding in Belfast, 1894-1912* (Belfast: The Friar's Bush Press, 1989).
[63] "Report of the Belfast Health Committee," 1; Robin Sweetman, "The development of the port," in in *Belfast: The Making of the City*, J.C. Beckett ed., (Belfast: The Appletree Press, 1988): 68-69.
[64] AC Hepburn, *A Past Apart: Studies in the History of Catholic Belfast, 1850-1950.* (Ulster Historical Foundation, 1996), 48.
[65] *Ibid*, 14-15.
[66] H.W. Bailie, Report on the Health of the County Borough of Belfast, for the Year 1909, pg. 20, LA7/9DA/19, Local Authority Records of the Belfast Corporation and County Borough Council, PRONI.

in other shipbuilding cities across the Irish Sea."[67] Whereas other large industrial cities housed much of their working population in dilapidated and overcrowded tenements, the majority of Belfast residents lived in terraced housing. The average arrangement consisted of a single family occupying one floor of a two-floor, semi-detached house.[68] Only 0.09 percent of the population of Belfast lived in spaces designated as "overcrowded" (5 persons or more in a single-room house) while Glasgow and Dublin housed as many as 4.28 and 8.69 percent of residents in such quarters.[69] While terraced houses were occasionally targeted for sanitary improvement, Belfast was widely regarded as having the best quality of housing of any large industrial town in the United Kingdom.

The city council showed great interest in developing a reliable water source, undertaking two major projects in the second half of the nineteenth century to provide water from catchment areas in Belfast's environs. The Belfast Water Act 1840 established the Belfast Water Commission, which was responsible for securing a safe water source for the growing town.[70] Under the Belfast Water Act 1865, the Commission began constructing the Woodburn Water Works, a series of reservoirs just northeast of Belfast that supplied water to one-half of the city through 1903.[71] The Stoneyford Water Works, responsible for the other half of the water supply to the city, were constructed in 1884, from a catchment area ten miles to the south-west of Belfast.[72] The hilly topography, combined with frequent rainfall and low population in these sub-catchment areas provided a relatively clean water source to the city, improved further by the implementation of

[67] Olwen Purdue, "Introduction," *Belfast: The Emerging City*, xxi.
[68] Belfast Health Commission Report and Minutes of Evidence, 1907, pg. 18, WAT/1/3H/2/4, Records of the Belfast Water Authority, PRONI.
[69]Belfast Health Commission Report and Minutes of Evidence, 1907, pg. 18, WAT/1/3H/2/4, Records of the Belfast Water Authority, PRONI.
[70] F.W. McCullough, *A Brief History of the Belfast Water Supply*, (Belfast: WG Baird, Ltd, 1923), 6-8.
[71] *Ibid*, 12.
[72] *Ibid*, 17.

filtration technology. Emulating the "slow sand filtration systems" of other large towns in the United Kingdom, in 1889, the Belfast Water Commission established filtration beds at both the Stoneyford and Woodburn water works.[73] Public confidence in the water supply was such that the *Evening Telegraph* bragged, "Fortunately Belfast is, happily, supplied with an abundance of pellucid water... almost utterly free of possibility of sewage contamination."[74] The region's distaste for the use of night soil in agriculture further reduced the likelihood of fecal contamination in water sources.[75]

While water supply to the city accorded with modern sanitary science, localized public health infrastructure occasionally lagged behind - a feature often attributed to a lack of municipal coordination.[76] Henry Whitaker, Chief Medical Officer of Health for Belfast, referred to some of the older sewers in the town as "little more than elongated cesspools," in his annual report, while noting that many houses containing a privy or ashpit had "no back passage to remove the filth, which must be carried out through the rooms in which the people live."[77] However, this was a minority problem in the city; Dr. James King Kerr, Alderman of the city of Belfast, notes that in 1897, there were 40,859 houses with water closets, and 26,620 houses with privies. By 1902, the number of houses with water closets had increased to 67,788, while the number with privies had decreased to 10,000.[78]

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[73] *Ibid*, 20-21.
[74] Belfast Evening Telegraph, 16th January 1893, in Newspaper Cuttings from the Water Board, WAT1/3F/3/1, Records of the Belfast Water Authority, PRONI.
[75] Belfast Health Commission Report and Minutes of Evidence, 1907, pg. 18, WAT/1/3H/2/4, Records of the Belfast Water Authority, PRONI
[76] Henry Whitaker, Report of the Belfast Health Commissioner for the Year 1896, 4. PRONI. While the Public Health Act 1848 created the position of Medical Officer of Health across the United Kingdom, Belfast did not appoint a Medical Officer of Health until 1878. Furthermore, no standardized infectious disease reporting system existed until the Infectious Diseases Notification (1897) Act.
[77] Whitaker, 23.
[78] "Minutes of Evidence, Tuesday, March 5, 1907 (Day 1)," WAT/1/3H/2/4 – Belfast Health Commission Report and Minutes of Evidence, 1907, 47-48.

Also problematic for sanitation was the fact that much of the city sat only a few feet above high tide.[79] The city's position at the center of a major floodway, combined with the fact that "large tracts of the low flat shore have been reclaimed from the sea by artificial means," made Belfast particularly prone to flooding. In a report on the causes of flooding in Belfast as late as 1908, City Surveyor Henry A. Cutler noted the "inadequacy of the existing provision for carrying off stormwater in time of heavy rainfall, especially about the period of high tide, when the discharging capacities of all the streams are at a minimum, and the storm overflows from most of the sewers are inoperative."[80] Unsurprisingly, the areas most prone to flooding were in the "low-lying" portion of town – precisely where elevated rates of typhoid occurred consistently from 1897-1901.[81] This spatial pattern (which will be explored further in the next section) is consistent with evidence that suggests that *Salmonella enterica Typhi* thrives in high flood-risk areas, propelled to new carriers through water and dislodged from sewers and soil in which it lay dormant (Figure 3, part I).[82]

Sanitary concerns, exacerbated by the burgeoning population of the town, led to a series of improvement projects beginning in 1870. A main drainage scheme was completed in 1896, replacing previous practices of dumping sewage into the Lagan.[83] While at the time of its completion, the drainage system was lauded as an ideal solution for sewage pollution in the river, it quickly became clear that the English model was not suited for the Northern Irish landscape.

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[79] C.J. Clibborn, "Belfast: Report on the Sanitary Circumstances and Administration of the County Borough," in *Sanitary Conditions and Administration in Cities and Towns, and the Precautionary measures Taken Against Plague.* Enhanced British Parliamentary Papers On Ireland Database. http://www.dippam.ac.uk/eppi/documents/20233/page/557043

[80] Henry A. Cutler, *Report on the Causes of Flooding in the City of Belfast and Proposed Remedies*, (Belfast: W& G Baird, 1908): 1. PRONI.

[81] Belfast Health Commission Report and Minutes of Evidence, 1907, WAT/1/3H/2/4, Records of the Belfast Water Authority, PRONI.

[82] Jenkins, "Health at the Sub-Catchment Scale," 634-651.

[83] Belfast Health Commission Report and Minutes of Evidence, 1907, pg. 47-48, WAT/1/3H/2/4, PRONI.

Modelled after Joseph Bazalgette's sewage system in London, which diverted sewage from the Thames through a series of intercepting sewers and into the Thames estuary far downstream of the city, Belfast sewers diverted waste from the Lagan and into Belfast Lough, with a chute pushing sewage further away from the city.[84] However, the topography of Belfast and London were substantially different in relation to their respective tidal estuaries. London sits within the Thames tideway, roughly 3.81 miles west of the Thames estuary; the movement of the river is therefore subjected to tidal pulls, and the river often moves swiftly out into the estuary. This characteristic meant that the eventual sewage outfall, located in the estuary, posed little risk to the city's residents. The Belfast Lough, on the other hand, is relatively still, possessing no tidal pulls. The city surrounds the Lough estuary, with 5 out of 14 dispensary districts occupying the shoreline.[85]

Estuarial Challenges to the Sanitary Ideal

Beyond its immediate proximity to the city, the currents of the Belfast Lough proved problematic for the quick removal of sewage. Shallow and sluggish by nature, the Lough failed to carry sewage out to sea efficiently, with much of the polluted matter instead sinking to the sedimented bottom. While originally a chute was designed to mitigate this problem by dispelling sewage further out in the Lough, where faster currents might pull the sewage matter away, the chute (installed in 1893) was found to be broken in 1897 – the same year the typhoid epidemic

[84] L.L. Macassey, *Belfast City and District Water Report on the Quality of the Water Supplied to the City and District, and its Effects Upon the Health of the Consumers, with a Reference to Other Causes of Disease which Operate within the City*, 1900, 1900.3 BPB Collection, Linen Hall Library.
[85] Matthew Smallman-Raynor and Andrew Clifford, "Featured Graphic. Enteric Fever in Belfast," *Environment and Planning A* 46 (2014): 1267-1269.

began.[86] In his evidence to the Belfast Health Committee, Professor E.A. Letts of Queen's University Belfast noted, "The main tide makes a sweep through the outer half of the Lough, leaving only a very slight current in the upper reaches, so that it is doubtful if the Belfast sewage really gets out of the Lough, and from float experiments I am convinced that it does not do so in one tide. The upper reaches of Belfast Lough are therefore more or less a cesspool."[87]

The changes to the ecology of the Lough in the wake of the city's improvement schemes were noted – and debated – by residents; Richard Patterson, a lifetime Belfast resident, lamented, "When I was a lad I frequently waded over the banks and I found sea urchins, different species of sea urchins, star fish, little star and common star…None of these things exist now. Banks are covered in mussels now."[88] He claims, "Gradually as Belfast increased, and with the enormous amount of crude sewage matter that was poured into the Lough, it altered the character of the seaweed, and the grass seaweed ceased gradually, very gradually ceased to exist and its place was taken by what we now know is *ulva latissima*…it is now accounted, I think, by all experts that it is a sewage plant."[89] Another city resident noted that while he thought the animal life of the bay had not declined in the wake of sewage outposts, that mussels had increased in number, and *ulva latissima* had appeared.[90]

The pollution of Belfast Lough might have constituted nothing more than unfortunate nuisance not for the deeply entwined relationship between the city's residents and the bay. Belfast Bay's estuarial ecosystem supported a wide variety of shellfish, including periwinkles, cockles, crabs, lobsters, mussels, and (occasionally) oysters. This abundance fostered a

[86] Darra L.W. Mair, Addendum to the Report of the Belfast Health Commission: Enteric or Typhoid Fever in Belfast, WAT/1/3H/2/4, PRONI.
[87] Testimony for the Sewage of Belfast Investigation of Mr. E. A. Letts, Professor of Chemistry at Queen's College, Belfast, pg. 461-462, D2682/2/20, Robert F. Blake Papers, PRONI.
[88] Report of the Belfast Health Commission, 1574
[89] Report of the Belfast Health Commission, 1574.
[90] Report of the Belfast Health Commission, 1580.

successful shellfish industry in the city, in which professional scavengers collected shellfish – largely periwinkles for bait, but also larger shellfish like lobsters and crabs for consumption – for export to England, Scotland, and Wales.[91] Many others developed a more informal relationship with the shellfish of Belfast Lough, fostered by a lack of regulation around shellfish collection by the Belfast Corporation. Professional gatherers or "hawkers" represented an important industry in the city itself, concentrating their efforts on the collection of cockles and mussels (alive, alive ho), of which they collected an estimated 700 to 800 quarts per day and sold in the streets.[92] In a report on the shellfish layings on the Irish Coast, Dr. Thomas Browne noted of cockles and mussels in Belfast, "They are hawked in the evenings through the working class districts, sold to the residents and in public-houses. Rag gatherers frequently carry them and exchange them to children for rags and bones."[93] Meanwhile, among laypersons, it was considered "quite a common thing to go down on Saturday afternoons and Sundays to the foreshore and bring home handkerchiefs full."[94] The numbers of commercial shellfish taken, even excluding local sales and other informal gathering practices, suggests a vast industry in unregulated waters (Table 1).

[91] TJ Browne, Report on the Shell-Fish Layings on the Irish Coast, As Respects their Liability to Sewage Contamination, (Dublin: Alexander Thom & Co., 1904), pg. 60, I.361/GREA, Irish Closed Access, Linen Hall Library.
[92] Mair, Addendum to the Report of the Committee, 132.
[93] TJ Browne, Report on the Shell-Fish Layings on the Irish Coast, As Respects their Liability to Sewage Contamination, (Dublin: Alexander Thom & Co., 1904), pg. 6, I.361/GREA, Irish Closed Access, Linen Hall Library.
[94] Report of the Belfast Health Commission, 1637.

Table 9. Shell-Fish Taken from Belfast Lough, 1896-1902.

Year	Periwinkles (tons/qrts)	Cockles (Gallons)	Crabs (no.)	Lobsters (no.)	Oysters (hundreds)	Mussels (tons)
1896	26/6	16,600	7,800	3,816	510	150
1897	29/9	19,000	7,200	2,040	400	130
1898	0/10	10,000	6,500	2,550	240	100
1899	9/10	1,728	5,000	1,899	240	90
1900	34/11	14,000	4,000	2,250	180	-
1901	56/14	2,510	3,000	2,100	120	1,397
1902	59/5	1,500	2,000	21,694	-	1,029

SOURCE: TJ Browne, Report on the Shell-Fish Layings on the Irish Coast, 6.

The combination of sewage improvement schemes, bay ecology, and local practices represent a fertile niche for the growth and survival of *Salmonella typhi*. As studies on typhoid ecology suggest, turbid water with high fecal content may provide the nutrient content to allow *Salmonella typhi* to thrive; meanwhile, the slow, meandering waters of the estuary allowed dense sewage matter to sink to the floor of the lough, where numerous filtering shellfish collected the microbes in their soft, dense bodies. The bacteria then lay dormant until their carriers were picked up by day-trippers and hawkers, cracked open, and eaten. Given the endemic nature of the disease in the British Isles, local use of the shoreline, and the weaknesses of novel sanitary infrastructures, it was simply a question of when a typhoid epidemic might strike in Belfast – and perhaps more importantly, who sickened.

Typhoid in Belfast, 1897

Through its rapid population growth and numerous improvement projects, "Belfast in its heyday seemed, to many, a miracle of human achievement against the environmental odds."[95] However, by the end of the nineteenth century, it became clear that this "miracle," promoted by boosterism and progressivism, disguised a dark underbelly. In April 1897, a special meeting of the Belfast Public Health Committee convened to discuss typhoid fever cases in the city, "of which there appeared to be many."[96] It was not until October, however, that the typhoid rates had become something of national comment. That month, *The South Wales Daily News* described the epidemic as "causing serious alarm," with 354 cases notified within the previous four weeks.[97] By the end of the year, 3,269 cases of typhoid had been notified to the Medical Officer of Health Henry Whitaker, and 336 deaths.[98] While the epidemic abated in the winter months, typhoid returned even more severely in spring 1898. In the decade that followed, Belfast's health officials fought an annual battle with the disease, resulting in nearly 20,000 cases and 2600 deaths (Table 10).[99]

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[95] Connolly, *Belfast 400*, 14-15.
[96] "Minutes of the Public Health Committee, 19th March 1896-24th August 1899," LA7/9/AA/3, Local Authority Records of the Belfast Corporation and County Borough Council, Public Records Office of Northern Ireland.
[97] "Typhoid Scourge," *South Wales Daily News*, Wednesday 27 October 1897, 5. British Newspaper Archive. Accessed 17 September 2019.
[98] Belfast Health Commission Report and Minutes of Evidence, 1907, pg. 298, WAT/1/3H/2/4, Public Records Office Northern Ireland (PRONI).
[99] Compiled from the Reports of the Belfast Health Commissioner for the Year 1903-1909. PRONI.

Table 10. Typhoid Cases, Deaths, and Rates Per 1000, 1897-1908.

Year	Population	Typhoid Fever Deaths	Typhoid Fever Cases	Deaths per 1000	Cases per 1000
1897	310000	336	3269	1.08	10.55
1898	340000	640	5136	1.88	15.11
1899	350000	263	1598	0.75	4.57
1900	359000	261	1777	0.73	4.95
1901	350862	341	2530	0.97	7.21
1902	360000	169	1044	0.47	2.90
1903	360000	136	842	0.38	2.34
1904	360000	111	530	0.31	1.47
1905	360000	128	631	0.36	1.75
1906	366220	90	551	0.25	1.50
1907	370163	82	356	0.22	0.96
1908	380344	57	274	0.15	0.72

SOURCE: Compiled from the Reports of the Belfast Health Commissioner for the Year 1903-1909, PRONI.

While it is unclear whether typhoid rates were higher in the years before 1897 (typhoid only became a compulsory notified disease in the year 1897), local and national commentary suggests that the epidemic was unusual. The *Edinburgh Evening News* noted in August 1898 that the typhoid epidemic in Belfast was spreading at an "alarming pace," and that extra doctors and nurses were being sought by the Board of Guardians for the city, while the *Daily Telegraph and*

Courier noted that the disease had reached "formidable dimensions," causing "great anxiety."[100] By August 1898, the Public Health Committee called on Dr. James Lorrain Smith, an eminent bacteriologist, to discern the cause of the epidemic.[101] The report, and the ensuing decade of investigations, provide detailed evidence that the typhoid epidemic in Belfast diverged from other epidemics on the British mainland both in extent and nature.

Lorrain Smith's original suspicions regarding the source of the typhoid outbreak aligned with major epidemiological trends present in British medical circles at the end of the nineteenth century. His report consisted of broad descriptions of death rate and terrain, but focused largely on water supply, sewage, and drainage.[102] Drawing on methods detailed in other British urban outbreaks, Lorrain Smith concentrated his investigation on the catchment areas at Stoneyford and Woodburn, which supplied water for the city. Through his investigation, Lorrain Smith became convinced that Stoneyford water supply must have been the source of the 1897 epidemic in Belfast; in the collection of farms surrounding the catchment area, he noted "an almost total lack of ordinary sanitary arrangements," and found that in 1897, one of the houses in the area reported four cases of typhoid fever. In 1901, when infection and mortality once again increased rapidly, he found two cases of typhoid had occurred in the area. As Lorrain Smith hunted for a source of fecal contamination in the water supply, several years of regular bacteriological analysis instead

[100] Edinburgh Evening News, "General News" Wednesday 24 August 1898, pg.6, British Newspaper Archive; "Typhoid in Belfast," *Daily Telegraph and Courier (London)*, Thursday 25 August 1898, pg. 5, British Newspaper Archive.

[101] J. Lorrain Smith and John Tennant, "A Study of the Epidemic of Typhoid Fever in Belfast, 1898," *The British Medical Journal* 1(1899): 193-197.

[102] Anne Hardy describe this methodical but broad-spanning nature of investigation by Medical Officers of Health, which she classifies as "late Victorian observational epidemiology." See Anne Hardy, *Salmonella Infections, Networks of Knowledge, and Public Health in Britain, 1880-1975,* (Oxford: Oxford University Press, 2015), 22.

revealed oddity to the Public Health Committee: “In examining the water...no typhoid bacilli were isolated.”[103]

An uncontaminated water carriage system was not the only epidemiological oddity surrounding the epidemic. Further investigations into the demographics, geography, and severity of the epidemic defied again and again major existing theories of typhoid transmission.[104]Annual cases did not cluster in particular localities of the city, but seemed to spread widely throughout; cases could not be traced to a common water source, common career, or common milk source.[105] Privy use declined in the city, and did not correlate to typhoid cases – nor did ineffective drains in individual houses.[106] In fact, Lorrain Smith found in a study of total cases between 1898-1903 that 85 percent of cases of typhoid fever occurred in houses in which there was no second case[107]

Only one group of residents in the city seemed impervious to the epidemic; in 1900, Sir Otto Jaffe, lord mayor of Belfast from 1899-1900 and 1904-1905 and president of the Hebrew community in Belfast, noted in an opinion written to *The Lancet* that among a population of “about 700 Jews, mostly of Russian origin, and poor, who all strictly adhere to the Mosaic code of diet,” no cases of typhoid fever occurred throughout the epidemic.[108] From this observation, and following the failure of Lorrain Smith’s investigation into the water, Jaffe concluded, “that

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[103] Smith and Tennant, “A Study of the Epidemic of Typhoid Fever in Belfast, 1898,” 193. *Bacillus coli communis* has since been renamed *Escheria coli*. While its presence in water may indicate human fecal contamination, its widespread presence in the colon of warm-blooded mammals generally suggests that it could be related to any number of nonhuman sources as well. It is furthermore worth noting that E. coli infection and typhoid infection do not present with similar symptoms.

[104] J Lorrain Smith, “An Investigation into the Conditions Affecting the Occurrence of Typhoid Fever in Belfast,” *Journal of Hygiene* 4(1904): 407-433; Mair, Addendum to the Report of the Belfast Health Commission, 105-132, PRONI.

[105] Minutes of Evidence and Report relative to the Water Supply, Belfast Health Commission 1907, 607, WAT1/3H/2/5.

[106] *Ibid,* 605.

[107] Lorrain Smith, “Investigation into the Conditions Affecting the Occurrence of Typhoid Fever in Belfast,” 420.

[108] Otto Jaffe, “Shellfish and Typhoid Fever,” *The Lancet* 1(1900): 353.

the sale of cockles, collected on the shores of estuaries, contaminated by sewage, is perhaps one of the sources which would account for the prevalence of typhoid fever in Belfast."[109]

Cockles, Mussels, and The Belfast Health Commission

Continually high rates of typhoid fever throughout the early twentieth century drew attention from the Local Government Board of Ireland, and eventually from London; suspicions of sanitary mismanagement by the Belfast Corporation thus culminated in the appointment of a Royal Commission to inquire into the health of Belfast in 1907.[110] The commission, comprised of a number of medical officers from Local Government Boards around England and Ireland (but notably not from Belfast), conducted a thorough, thirty-one day investigation into the possible causes of elevated mortality in Belfast, focused on typhoid fever. From these reports emerge a thorough picture of the state of the water source, bacteriological practice, sanitation, public health practice, and even quotidian urban life in Belfast at the time of the epidemic.

Similar to Belfast's own public health committee, and consistent with British and European epidemiological thought at the end of the nineteenth century, the commission focused on the water supply, returning to the topic again and again throughout their investigation. After extensive evidence and bacteriological analysis of the two major water sources for the city of Belfast at the time of the original epidemic, Stoneyford and Woodburn Reservoirs, and an examination of the newly completed Mourne scheme, however, the Commission were forced to conclude, much like Lorrain Smith, that "it is inconsistent with the facts to hold the Belfast water supply responsible for fever in Belfast since 1897."[111] They noted the particular lack of an

[109] Jaffe, "Shellfish and Typhoid," 353.
[110] Report of the Belfast Health Commission, 1907, PRONI.
[111] Belfast Health Commission Report, 27.

"'explosion' of the disease on the scale which has been characteristic of epidemics of fever which have been disseminated by public water services infected at their sources," exhibited at Maidstone, Worthing, and Lincoln.[112] Furthermore, in line with initial studies into the epidemic, the commission was forced to conclude that "unsanitary circumstances" were also likely not the cause of excessive fever, as "it cannot be contended that, in this respect, Belfast is on an altogether lower plane than other cities and towns in the United Kingdom."[113]

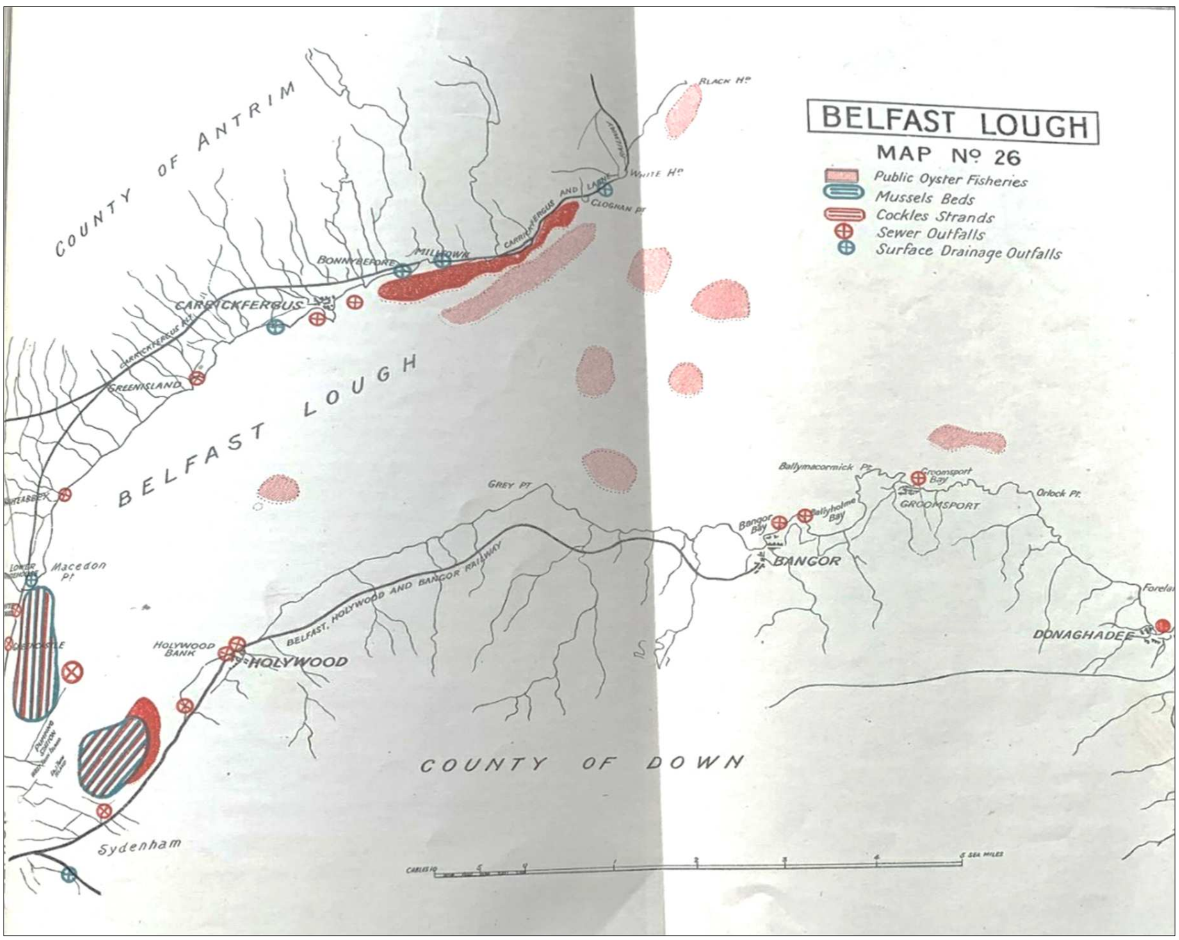
The shellfish connection had been considered tentatively in Belfast since the early twentieth century; the Belfast Corporation erected signs warning of the risks of collecting shellfish from Belfast Lough as early as 1901, and Dr. Lorrain Smith had confirmed the presence of sewage in shellfish samples in his bacteriological laboratory in July 1902.[114] However, between the initial reports and the 1907 commission, the shellfish theory of typhoid transmission gained a significant foothold in the British epidemiological imaginary. This change in opinion was the result of a number of eminent medical officers and physicians drawing the link between shellfish and typhoid outbreaks in England in 1894 and 1895, corroborated by bacteriological evidence.[115] Thus, once questioning related to the water sources was exhausted, the commission, and particularly Dr. Ludovic William Darra Mair, a Medical Inspector for the Local Government Board for England, turned to investigating practices of shellfish collection and consumption in Belfast.

Immediately clear from anecdotes and reports collected by the commission concerning shellfish gathering was the probable extent of pollution among the beds – and the continued popularity of these beds despite contamination. At Greencastle, one of the major coastal areas of the city and a

[112] Belfast Health Commission Report, 27.
[113] Belfast Health Commission Report, 26.
[114]Minutes of the Public Health Committee of Belfast, 10th July 1902, pg. 205, LA7/9AA7, Local Authority Records of the Belfast Corporation and County Borough Council, PRONI.
[115] Hardy, "Exorcizing Molly Malone," 81.

major cockle collection site for hawkers, a resident noted “between two and three hundred” people on the shores collecting shellfish, despite the appearance of foul-smelling seaweed in the area. Bacteriological examinations revealed multiple bacteria present in cockle samples – including *Salmonella enterica typhi.*[116] Greencastle, like the majority of shellfish beds in the region, stood less than two miles from the nearest outfall sewer – a feature illuminated in Dr. TJ Browne’s report on the shellfish layings of Ireland (Figure 18).

Figure 18. Position of the Mussel and Cockle Beds of Belfast in Relation to Sewer Outfalls



SOURCE: TJ Browne, Report on the Shellfish Layings of Ireland, 59.

[116] Minutes of Evidence for the Belfast Health Commission, 1580.

Bacteriological investigations revealed the contamination of shellfish to be extensive. Dr. Symmers, Professor of Pathology at Queen's University, "examined samples taken as far as Carrick, and as far as Bangor, and in each case he found evidence of sewage contamination in the cockles."[117] The harbor commissioner similarly noted in 1907 that he had in the past witnessed people collecting there "almost every day, more particularly at the end of the week," from land where "the sewage flows over it and settles down upon it."[118] Dr. Browne provided similar observations in his 1904 studies of the shellfish layings of Ireland:

> I may mention here that when visiting the mussel and cockle strands in Belfast Lough, I saw several people gathering these shell-fish only a few hundred yards from the sewer outfalls, and, on questioning some of them, found they were workmen from the shipbuilding yards who were taking a holiday. They informed me that it was a common practice for the workmen to collect shell-fish in the same locality and to bring them home for their families to eat. The shell-fish are generally eaten cooked, but sometimes in the raw state. On further inquiry I was informed that several members of the workmen's families who had partaken of the shell-fish had subsequently developed enteric fever, but that whether the disease was caused by the shell-fish was not ascertained.[119]

While reported as a widespread practice, evidence suggests that cockle consumption was often (but not always) classed. While quantitative statistics on informal (and eventually illicit) practices like shellfish consumption are not available, extensive anecdotal evidence suggests that shellfish consumption was widespread among the working class, which, according to the public health committee, represented "by far the greater portion" of the population of the town.[120] Mair notes in his summary of the commission, "there is much evidence to the effect that the consumption of shellfish by the working classes in Belfast has been very large indeed. A witness, himself engaged in the shellfish trade, informed the Commission that the professional gathering

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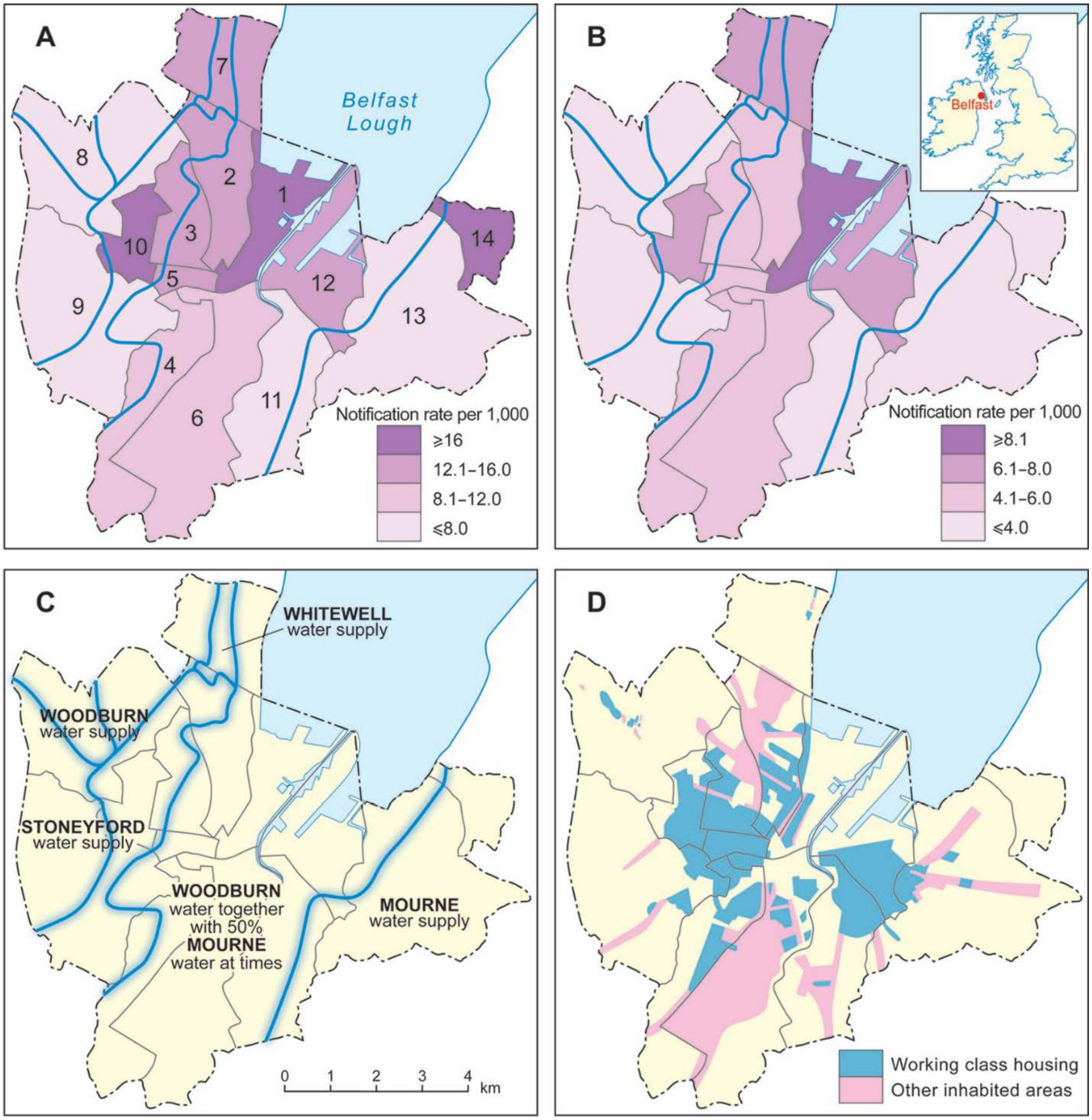
[117] Minutes of the Belfast Health Committee, 1330.
[118] Testimony of David Bennet, Minutes of the Belfast Health Committee, 1614-1615.
[119] Report on the Shellfish Layings, 62.
[120] "Bacteriological Examination", Minutes of the Public Health Committee of Belfast, 10th October 1901 – 6th May 1904, pg. 133, LA7/9AA/6, Local Authority Records of the Belfast Corporation and County Borough Council, PRONI.

of shellfish from the shores of the Lough had been an important Belfast industry for “more than half a century.”[121] These observations are corroborated by spatial analysis of typhoid rates in the city, spatial analysis, conducted at the time of the epidemic investigation and reproduced in present GIS forms. Spatial patterning indicates a higher concentration of disease around working-class housing – and especially working-class housing located close to the Lough (Figure 19).

[121] Mair, Addendum to the Report of the Belfast Health Commission, 132.

Figure 19. Maps of enteric fever notification rates per registration district, and in relation to type of housing and water supply scheme. (Map A shows Enteric Fever Notifications for the year 1901, while Map B shows Enteric Fever Notification for 1901-1905).



SOURCE: Matthew Smallman-Raynor and Andrew D. Cliff, "Featured Graphic. Enteric Fever in Belfast," Environment and Planning A 46(2014):1267-1269.

While water supply, localized sanitary patterns, and milk source showed no correlation to typhoid case, visual evidence indicates some spatial clustering of the epidemic. Beyond the correlation between coastal and working-class neighborhoods and typhoid rates, perhaps the most striking pattern demonstrates that the regions with the highest case notification appear to follow the drainage pathway of the catchment basin, with the lowest lying areas appearing to suffer the highest case rates (Figure 5, Panel A and B). Testimonies suggest that the main drainage scheme, in addition to polluting the foreshore and shellfish beds, was unequipped to deal with the rainfall patterns of Belfast, resulting in frequent flooding. In 1902, more than a decade after the completion of the town's main drainage scheme, Belfast suffered catastrophic flooding, particularly in the low-lying areas of the city. Mill owners present at the chamber of commerce following the flood asserted that "the bridges and culverts at present spanning the Blackstaff River are wholly insufficient to vent the volume of water comprised in the united Blackstaff, Clowney, and Pound Burn streams in time of rain and storm, such as we have experienced in November last year and September of this year."[122] The City Surveyor's observation that "the obstructions and contractions of rivers and streams to enable the water to be used for manufacturing purposes," which combined with the "insufficient capacity of the main sewers when proper relief cannot be obtained from storm overflows," also contributed to the flooding suggests that a combination of ecology and industrial pressure led to frequent flooding patterns along riverbeds, which also represented the lowest points in the sub-catchment basin – and therefore places where typhoid fever rates were consistently elevated.[123]

While less prominent in contemporary public health literature, it is important to consider how immunological dynamics of *Salmonella enterica typhi* may have played a role in typhoid morbidity and mortality in Belfast. Studies suggest that nearly 1 in 20 persons infected with

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[122] Appendix, Meeting held on 6th November, 1902, to Belfast Chamber of Commerce, Robt Thompson, Esq, JP presiding, Evidence of the Belfast Health Committee, 593.
[123] Report of City Surveyor on The Causes of Flooding in the City of Belfast, and Proposed Remedies, July 1908, pg.1, D2964/XB/2, McConnell & Co. Papers, PRONI.

typhoid become asymptomatic carriers, shedding high loads of the bacteria in feces and urine intermittently for years.[124] While the statistics of the Health Committee of Belfast demonstrate that multiple cases rarely occurred in single homes, it is likely that carriers may have shed the bacteria into the sewage system continuously, maintaining the high concentration of typhoid fever in Belfast Lough and in its bivalve population.

Population growth may have also provided opportunities for transmission and maintained consistent levels of endemic typhoid in the city. As survivors of typhoid fever develop immunity to the bacteria, a large and continuous epidemic would require consistent access to a vulnerable population by *Salmonella enterica typhi.* At the time of the epidemic, Belfast was the fastest growing city in the United Kingdom, with a constant influx of new workers for the booming linen and shipping industries. While most immigrants came from Belfast's hinterlands, immigration statistics also show immigration from Scotland and the English Midlands.[125] During the second year of the epidemic, 1898, bankruptcies around the Linen industry, including the Belfast Flax Spinning Company, the Ligoniel Spinning Company, the Co. Down Flax Spinning and Weaving Company and the Congry Flax Spinning Company also drove more women seeking work out of the hinterlands and into the city. As companies in Limavady, Keady, Castleblayney, Buncrana, Coleraine, Ballymoney and Emyvale went into liquidation, the industry concentrated more than ever in the greater Belfast area.[126] Thus, just as the epidemic began, an influx of new and vulnerable people into the city may have provided new opportunities for *Salmonella enterica typhi* to spread.

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[124] Denise M. Monack, "*Salmonella* persistence and transmission strategies," *Current Opinion in Microbiology* 15(2012): 100-107.
[125] S.J. Connolly and Gillian McIntosh, "Whose City? Belonging and Exclusion in the Nineteenth-Century Urban World," in *Belfast 400*, 265-269.
[126] *Belfast, The Making of the City*, 49.

Through a combination of local topography, sewage and drainage systems, estuarial ecology, and local food practices, Belfast at the end of the nineteenth century became an ideal environment for the typhoid bacteria. The dynamics of ecology in the perpetuation of typhoid fever, and the success of the *Salmonella typhi* bacteria, is perhaps best summarized in the report of the Belfast Health Commission at its conclusion:

> It does not, of course, follow that shellfish have been the universal or almost universal direct cause of attacks of fever in Belfast. The correct interpretation appears to be that shellfish have been the means of keeping the disease constantly alive, as it were, in Belfast, and that not only have they been the 'additional fever agency' referred to when discussing the influence of insanitary conditions, but that this additional fever-agency has largely controlled the course of events, not necessarily as the direct cause of a greater number of cases of fever than all other agencies, but by repeatedly furnishing opportunities for these other agencies to come into operation.[127]

Typhoidal Neighborhoods and Typhoidal Towns – Assessing Typhoid Risk in Belfast and Dublin

In the other two case studies examined in this project, neighborhood-level analysis revealed characteristics at the scale of the home, individual, and community that perpetuated disease – revealing infrastructural and social disparities that aligned with Mitman's conception of "ecologies of injustice." In the case of the Belfast typhoid epidemic, however, neighborhood-level analysis is complicated by the diverse ways of living subsumed under the dispensary district structure. Additional challenges emerge from the scale of evidence: while topographical placement may have influenced typhoid epidemiology, other habits, like shellfish collection and consumption, cannot be traced quantitatively at the level examined by public health infrastructure in the nineteenth century. However, comparison of social, ecological, and sanitary characteristics of the epidemic across differing neighborhoods may reveal possible avenues for

[127] Report of the Belfast Health Commission, 28.

infection – while comparison with Dublin, a city of vastly different demographic and housing characteristics, suggest that others may have been of less importance than often assumed.

Table 11. Belfast Typhoid Cases Notified by District and cases per thousand, 1897-1906.

Dispensary District No	Location	Typhoid Notifications	Notification Per 1000
1	Dock	2377	16.13
2	Duncairn	5202	11.13
3	Shankhill	6375	13.50
4	Workhouse	5360	14.34
5	Millfield	2167	12.46
6	College	4872	10.12
7	Greencastle	166	10.89
8	Ligoniel	428	8.46
9	Falls	2095	11.13
10	Woodvale	4405	19.10
11	Ravenhill	3408	9.79
12	Newtonards Road	5084	15.14
13	Ballyhackmore	1065	7.10
14	Ballymagan	4	1.67
Total		**43008**	**12.51**

SOURCE: Compiled from the Reports of the Belfast Health Commissioner for the Year 1897-1906, PRONI.

The three neighborhoods that suffered the highest overall morbidity from typhoid fever at first glance have little in common. District No. 1, which encompassed the area around the docks, had a relatively small population related to other urban districts, and lay "very low," with a collection of "very old" houses. By 1903, the district had been subject to improvements as the result of a burst of civic action. According to Whitaker, "unsanitary courts and lanes have been closed, whilst hundreds of houses have been pulled down in Carrick Hill, under the Housing of the Working Classes Act, and a great sanitary improvement effected thereby." The area had also undergone structural change, as "The splendid and spacious thoroughfare of Royal Avenue has replaced the old and congested districts of Hercules Street and John Street, with their unsavoury and unhealthy porlieus with narrow lanes and entries, some of which were a disgrace to the community."[128] District No.10, meanwhile, was situated at an "elevated position," and "the great majority of the houses have been erected during the past few years, under the surveillance of your officers, and with due regard to sanitation."[129] The streets were considered "wide and spacious."[130] Most of the population of District No. 12 occupied land that "lies very low," but occupied by "workmen's houses" that were "generally in very good order."[131] The three districts had widely different sanitary features, and not commonly poor sanitary infrastructure – sanitary operations undertaken in each dispensary district annually varied, and the areas were rarely the extreme of any given feature: none were considered to be the most or least unsanitary district by public health officials; none housed the largest number of cows (and therefore no firm connection to milk-borne typhoid); and none contained an unusually high number of faulty drains or privies.[132]

A closer look at the demography and topography of the districts, however, suggest several similar characteristics that may have influenced morbidity. Problems surrounding elevation and flood risk dominated in both District No. 1 and District No. 12 at the end of the nineteenth century. In his examination of the factory districts of Belfast, Dr. CD Purdon noted that the No. 1 dispensary district "runs for a considerable distance along the quays. Consequently during winter, when high tides and rains are very prevalent, the low lying streets are frequently flooded, especially as the drainage is very insufficient."[133] Whitaker similarly described the land

[128] Henry Whitaker, "Report on the Health of the City of Belfast for 1903," pg. 11, LA/7/9/DA/13, Public Records Office of Northern Ireland.
[129] Whitaker, "Report on the Health of the City of Belfast for 1903," 13.
[130] Ibid, 13.
[131] Whitaker, "Report on the Health of the City of Belfast for 1903," 14.
[132] Bailie, "Report on the Health of Belfast for the year 1909," 197.
[133] CD Purdon, *The Sanitary State of the Factory Districts of Belfast,* (Belfast: H. Adair, 11 & 13, Arthur Street, 1877) 21, Pamphlet Collection, Linen Hall Library.

in District No.12 as " always damp and wet."[134] As areas prone to frequent wetting and flooding, and located further along the drainage path for the catchment basin, it is possible that these districts may have been further prone to typhoid as the bacteria accumulated along drainage pathways and multiplied in fertile conditions. Frequent flooding may have allowed sewage to overflow onto the subsoil, and thus settle in the environs and housing foundations of these neighborhoods, increasing chances of contact.[135]

The characteristics of District No. 10, however, considered alongside District No. 1 and District No. 12, suggest that while elevation and flood risk may have increased chance of typhoid ambiently, class played perhaps the most significant role in its transmission. Local knowledge of shellfish consumption patterns suggested again the link between working-class economic status and cockle consumption. Unlike the other two districts, District No. 10 occupied elevated ground and had nearly no houses considered to be in poor condition. Eighty-seven percent of the houses had water closets instead of privy systems.[136] However, the neighborhood was also described as "a population mainly composed of the working classes,"[137] a characteristic shared with both District No. 1 and 12. Purdon describes Dispensary District No 1. as occupied primarily "by the Factory and labouring classes," and by Lorrain Smith as occupied by "the artisan and labouring classes"; meanwhile, Whitaker describes that in District 12, "A large number of workmen's houses has been built upon this ground. The occupiers are, to a large extent, strong able-bodied men employed chiefly in the iron shipbuilding trade, with their families at the ropeworks and other industries of the district."[138]

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[134] Lorrain Smith, "Investigation into the Conditions of Belfast," 14.
[135] This relationship between sewage overflow and typhoid increase is also examined in contemporary context. See Jenkins et al., "Health at the Sub-Catchment Scale," 633-651.
[136] Lorrain Smith, "Investigations," 412.
[137] Whitaker, "Report on the Health of Belfast, 1903," 13.
[138] Whitaker, "Report on the Health of Belfast, 1903," 14.

The link between socioeconomic status and typhoid risk in these neighborhoods, despite sanitary disparities, supports the relationship between disease and shellfish consumption. Evidence suggests that the working classes represented the primary collectors of cockles and mussels, and the primary consumers of hawked goods. Shellfish represented an important supplement to diet for workers, both men and women, who supported families. While a relatively prosperous industrial town, Belfast could be a challenging place in which to earn a living wage. As historian of Belfast Sean Connolly notes, "In 1855, when a four pound loaf of bread cost 7 ½ d, a pound of meat cost 5 ½ d and coal was 7 12d per hundredweight, women and girls in the industry earned an average of 5/6d, lads 3/3d and unskilled men 10/6d….Even so it has been estimated that unskilled men could only just have earned enough to keep a family of two adults and two children with 'minimum comfort'."[139] As a witness for the Health Commission noted, shellfish thus represented a free and plentiful food source for the working classes of the city. When considered alongside the temporal correlation – that the chute expelling sewage further into the bay broke in the same year of the epidemic – and that typhoid cases concentrated in neighborhoods with prevalent working-class populations, geographic and medical evidence suggests that local practices of shellfish consumption may have initiated the spike in typhoid fever cases in 1897.

Further evidence of this link is how intervention into shellfish consumption practices aligned with the decrease of the typhoid epidemic in Belfast. Despite the controversy around the shellfish question, the Belfast corporation took several steps to discourage shellfish consumption in the city. Pamphlets were posted along the seashore; it was made illegal to hawk cockles and periwinkles in the city, enforced by food inspectors. By 1907, there were palpable changes in the

[139] Connolly, *Belfast 400*, 53-54

practice within the city. A shellfish gatherer called for evidence in the Health Commission, a Mr. Henry McCauley, noted that "A few years ago a great amount of [cockles and periwinkles] was consumed, but lately not so much; it is getting less." Whereas before they were "hawked and shouted through the streets and sold, and bought by the public," now "they are less sold now, because the caution given out by the Corporation some time ago has taken effect, and people do not go for them; they are frightened to eat them now, you know."[140]

While the reduction in cockle consumption clearly related to the decrease in typhoid fever incidence, some features of improvement in housing might also account for changes in typhoid rates in low-lying areas, as well. In District No. 12, for example, Medical Officer of Health Joseph Martin observed in 1907 that "some [houses] are modern and some are old. In some portions of the district they are new, in other portions the houses are old" (1893). It can be so uneven that "In cases, say like Gertrude street, where one finds houses well kept and occupied on the side, and on the other side many of them are empty," with many people vacating the houses for those being built on "higher ground, with sandy sub-soil."[141] Thus, a combination of reduced shellfish consumption and alterations to housing patterns in the city, in addition to growing attention to improving municipal drainage schemes, likely reduced typhoid rates in the city by 1914.

Ecologies of typhoid in Dublin

One of the strongest cases for the correlation between shellfish consumption and higher-than-average typhoid fever morbidity arises through comparison of Dublin and Belfast. Charles Cameron, Chief Medical Officer of Health for the city of Dublin, first noted elevated rates of

[140] Evidence of the Belfast Health Committee, 1783-1784.
[141] Evidence of the Belfast Health Commission, 1893.

typhoid infection and mortality in the city in 1880. In his annual report on the sanitary state of Dublin for 1892, Cameron drew a comparative table of typhoid fever deaths per 10,000 in fifty towns and cities in the United Kingdom from 1887-1891. From his statistical analysis, he concluded, "The table shows that in Dublin [5.04 per 10,000], typhoid fever causes a greater waste of life than in any English town than St. Helens [5.22], and in Ireland is only exceeded by Belfast [5.36]."[142] Much like Belfast, Dublin also drew its water from a nearby elevated catchment area; and much like its neighbor, the water was often lauded as superbly clean (Figure 6). Cameron himself claimed, upon examination of the Wicklow mountain scheme, "I believe there are few cities in the world supplied with such good water as Dublin fortunately possesses."[143] He notes the generally unremarkable nature of other sanitary aspects of the city as well, claiming that while the city did not possess a main drainage scheme, "Its street sewers are as good as those in the English towns."[144]

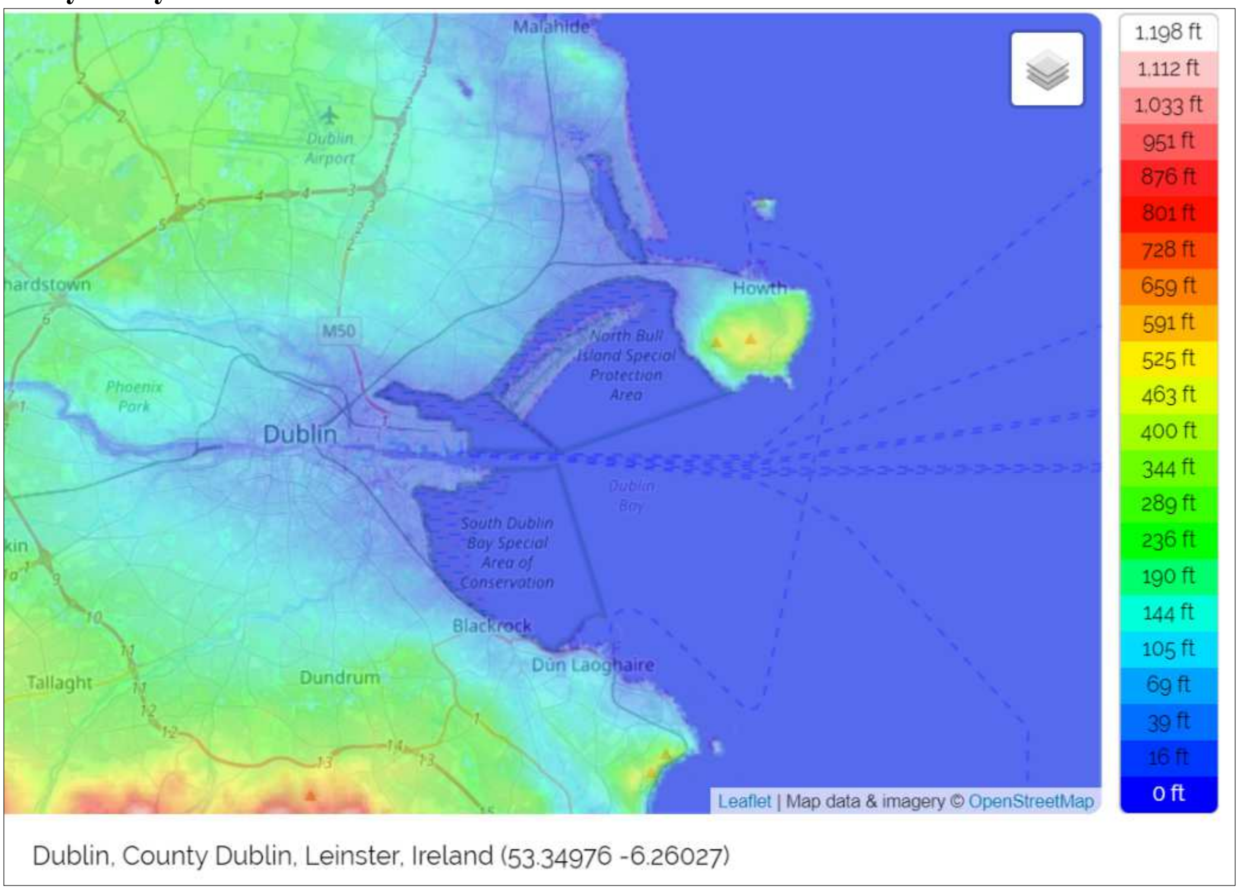
At first glance, there were few commonalities in the socioeconomic structure of nineteenth-century Dublin and Belfast. The seat of commerce and government, and the largest city in the south of Ireland, Dublin supported (or rather, failed to support) a high proportion of poor residents in infamously derelict, overcrowded tenement houses. Most families shared a single water closet with multiple other occupants.[145] Belfast was the booming seat of industry, populated largely by working and middling class who occupied single-family terraced homes. Each home had its own waste disposal system, whether a flush toilet (two-thirds of cases at the

[142] Charles Cameron, Report Upon the State of Public Health, 1897, *Dublin Corporation Reports & c.*, vol. 2, no. 97, pg. 713, Dublin City Library and Archive.
[143] Cameron, Report Upon the State of Public Health, 1897, 723.
[144] Cameron, Report Upon the State of Public Health, 1897, 724.
[145] Charles A. Cameron, Report upon the State of Public Health and the Sanitary Work Performed in Dublin during the Year 1880; also, Nineteenth Annual Report Upon the Analysis and Inspection of Food, &c., in Report and Printed Documents of the Corporation of Dublin, 1881, vol. 2, (Dublin: Dollard Printer, 1881), 50-82, Dublin City Library and Archives.

end of the nineteenth century) or a privy (one-third of cases). However, each struggled with an elevated typhoid fever rate at the end of the nineteenth century (albeit at different scales).

While these disparities suggest two very different urban structures, Cameron's examination of the sanitary state of Dublin reveals key commonalities between the two cities. First, he noted that "Both Dublin and Belfast are rather low-lying cities, situated upon the estuaries of rivers, and at present are badly drained."[146]

Figure 20. Elevation Map of Dublin, Ireland, demonstrating position of the city in the River Liffey valley.



SOURCE: "Dublin Topographic Map, Relief Map, Elevations Map," https://en-gb.topographic-map.com/maps/lpw4/County-Dublin/. Map data and imagery © OpenStreetMap.

[146] Cameron, Report Upon the State of Public Health, 1897, 713.

Second, he observed that shellfish gathering was common among the poor and working classes of Dublin Bay: "Oysters, cockles, mussels, and other lamellibranchiate molluscs are often eaten uncooked, and their shells enclose a liquid which is also often drank raw," voicing a concern that "at low water, sewage trickling down the shore is likely to find its way into the interior of the oyster and other shell fish."[147] This combination of estuarial ecology, drainage, and local shellfish consumption became a preoccupation of Cameron's throughout his time as Chief Medical Officer of Health in Dublin - often to the detriment of his reputation.[148] For while anecdotal connections between typhoid and shellfish consumption emerged in the late nineteenth century, the hegemonic theory for typhoid transmission remained grounded in its connection to contaminated water supplies. It was nearly twenty years after Cameron first posited the connection that the shellfish theory of transmission was considered among the British medical community – an epistemological transition that was only strengthened when, in 1897, Belfast experienced an unexplainable typhoid epidemic.

At the time of its own typhoid epidemic, Dublin lacked a main drainage scheme, and areas of the neighborhood at lower elevation were prone to regular flooding. According to Cameron, the areas of the city that lay upon gravel (which represented the lower elevation parts of the city, located closer to Dublin Bay), were "water-logged for a large portion of the twenty-four hours," and that during high tide, "sewage entering the sewers from houses and other places remains

[147] Cameron, Report Upon the State of Public Health, 1897, 728.
[148] Charles A. Cameron, "Oysters and typhoid" in *The British Medical Journal* 2(1880):471. Cameron was, in fact, the first medical officer of health in Britain to posit a connection between typhoid fever and shellfish. In the year 1880, just as Karl Eberth distinguished *Salmonella enterica Typhi* for the first time, Cameron gave a speech entitled "Sewage and Oysters," that nearly got him laughed out of the annual meeting of the British Medical Association. At the conclusion of his speech, the head of the Association, knowing Cameron to be a somewhat lighthearted man, inquired as to whether he was making a joke.

therein, except in the case of some of the easterly sewers which are connected with pumping stations," occasionally flooding areas surrounding the Liffey.[149]

While Cameron remained convinced of the soil theory of transmission well into the 1890s, only considering shellfish transmission as a marginal addition to existing typhoid rates in the city, observations of the patterns of infection – and its seasonality – suggest that shellfish contributed significantly to typhoid rates in the city. In his report on the Sanitary State of Dublin, Surgeon-Colonel D. Edgar Flinn, a medical inspector for the Local Government Board of Ireland, noted that cockle gathering and cockle hawking were both widespread in the city, and that "At low water during the spring, summer, and autumn months, the strands on the southern shores of Dublin Bay are sometimes thickly crowded with cockle gatherers."[150] He estimated from interviews with hawkers that, "It is probable that from about seventy to eighty thousand quarts of cockles are eaten in Dublin annually."

The most popular area for cockle gathering, Clontarf, also served as the major sewage discharge point for the city of Dublin. According to Flinn, "The foreshore at Clontarf is always laid dry at low water, and the sewage of a large and populous district is discharged directly on to the foreshore there. The entire estuary receives the crude sewage of the City of Dublin as well as that from the recently added Urban Districts of Clontarf, Drumcondra, and Glasnevin."[151]While the socioeconomic makeup of Dublin differed from that of Belfast, Flinn noted that similarly, "The cheapness of cockles as an article of dietary brings them within the reach of the poorer classes, hence their consumption is considerable, and a large number of people gather them in

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[149] TM Grimshaw, "Appendix: The Distribution of Enteric Fever in the City of Dublin," in *Dublin Corporation Reports &c.,* 2(1889): 374, Dublin City Library and Archives.
[150] D. Edgar Flinn, "Report on the Sanitary State of Dublin," "Enclosed – Official Report on the Sanitary Circumstances and Administration of the City of Dublin, with Special Reference to the Causes of the High Death-Rate," 1906, CSO/RP/IR/5294, General Administration, National Archives of Ireland.
[151] Report on the Shellfish Layings, 72.

addition to those who earn a livelihood by selling them."[152] Cameron noted the connection between the profession of shellfish gathering and typhoid, as well, when in 1888 he found that of 168 deaths from typhoid that year, 68 of them (or 40 percent) occurred among those classed as "Hawkers, Porters, Labourers, &c."[153] Geographic data corroborated the shellfish theory of transmission as well. In an inquiry into the distribution of enteric fever in the city of Dublin, Charles Cameron and Thomas Grimshaw found that the highest death rates also occurred in Clontarf:

Table 12. Average Death Rates from Enteric Fever in Dublin Registration District, 1881-1887.

Neighborhood	Population	Total Deaths, 1881-1887	Death Rate per 10,000
Clontarf	3868	14	5.2
Coolock	8190	25	4.4
Finglas	3994	5	1.8
Palmerston	6305	16	3.6
Rathmines	24834	65	3.7
Donnybrook	24,703	54	3.1
Blackrock	8,054	11	2.0
Kingstown	20,085	38	2.7
City Centre	250,981	683	3.9
Total	351,014	911	3.7

SOURCE: Compiled from Charles Cameron, Reports Upon the State of Public Health and Sanitary Work Performed in Dublin, 1880-1890, Dublin City Corporation Records & C., Dublin City Library and Archives.

[152] Flinn, "Report on the Sanitary State of Dublin," 29.
[153] Charles Cameron, *Dublin Corporation Reports &c for the Year 1889*, vol. 2, pg. 620.

Another indication of the role of shellfish in the distribution of typhoid in Dublin – and the role of local custom in its spread – was annual variation in typhoid rates in the city. While in Belfast shellfish were thought to be collected year-round, in Dublin, shellfish collection had a distinct seasonality. Between 1887-1891, most deaths from typhoid fever in the city occurred between August and October, precisely when, Flinn notes, there was a marked increase in cockle gathering in the city.[154] From these circumstances, Flinn concluded,

> Shellfish gathered from polluted sources, such as existed, and still exist, in the vicinity of Dublin, must be regarded with suspicion, and it seems probable that the ingestion of shellfish from these tainted sources has played no inconsiderable part in the history of Enteric Fever in Dublin, as large quantities of oysters and cockles were taken daily from Clontarf and Dollymount district, and were gathered from highly-polluted sources, and consumed in the city.[155]

Belfast's industrialization and position as the fastest growing city in the United Kingdom is perhaps its major distinguishing feature from Dublin. This growth, both economic and demographic, provided encouragement for burst of sanitary improvement – which resulted in the positioning of a modern sewage disposal system directly over local shellfish beds. Population growth, unique to Belfast at the time, likely also influenced the persistence of the epidemic. As most people develop immunity to typhoid once they have encountered the bacteria, an epidemic would require a continuous vulnerable population. Thus Belfast's position of importance in the United Kingdom (and by extension, the British Empire), exposed an entirely new and vulnerable population to an ecology suited to typhoid perpetuation. Alternately, Dublin's population growth slowed at the end of the nineteenth century, and while Belfast's modern drainage system directed sewage to the main cockle beds of the estuary, evidence suggests that the new drainage scheme enacted in Dublin in 1891 and completed in 1906 diverted the flow of sewage away from the

[154] Browne, Report on Shellfish Layings, 72.
[155] Flinn, "Report on the Sanitary State of Dublin," 29.

beds in question, both of which, alongside growing public awareness of the connection between shellfish and typhoid fever, may have accounted for the gradual decline of cases towards the beginning of the twentieth century.[156]

A comparison of the role of shellfish in Dublin and Belfast, two socioeconomically disparate cities integrated into ecological similar landscapes, reveals the potential importance of environmental features in typhoid transmission. While it is likely that a number of features contributed to the transmission of typhoid, including complex and often untraceable individual sanitary practices, the existence of carriers, and other iterations of direct person-to-person transmission, it is unlikely that these practices should have been more conducive to the spread of *Salmonella enterica typhi* in Belfast than in other cities in the United Kingdom. The similarities between Dublin and Belfast in cultural practice and ecological position, combined with their socioeconomic and infrastructural disparities, suggest instead a confluence of ecological and social features unique to these cities may have contributed to a niche for *Salmonella typhi*

Conclusion

Belfast represents a fascinating case in the role of imperial pressure and local ecology in the transmission of disease. While endemic to the United Kingdom, typhoid fever had retreated to an incidental disease in Britain by the end of the nineteenth century; but in the "Workshop of Empire," as Belfast was known, typhoid fever proved a greater nuisance than ever. As a microbe solely pathogenic in humans, and reliant on fecal-oral pathways, *Salmonella enterica typhi* has been traditionally thought to transmit as a result of poor sanitary practice, lack of access to water closets, and poor food hygiene practices. While these risk factors certainly account for many

[156] *Ibid*, 10. Though it should be noted that a small typhoid outbreak occurred in Clontarf again in 1908.

historical and contemporary typhoid cases, recent research into the ecology and niche of *Salmonella enterica typhi* has, much like recent research in *Vibrio cholerae*, revealed that ecology may contribute significantly to its survival, replication, and transmission. Topographical features like sub-catchment basins, rainfall level, soil type, and local fauna have all been recorded to contribute to typhoid transmission. The unique relationship of typhoid fever to the human immune system, however, also limits its spread in stagnant populations, in which cohabiting humans develop immunity to the disease. It is therefore a unique confluence of these characteristics – ecology, topography, and economic and demographic features – that contributed to the unusually severe and lengthy epidemic of typhoid fever in Belfast at the end of the nineteenth century.

In its attempt to emulate London's sanitary scheme in the 1880s, Belfast's local authority ignored local landscape, local ecology, and local cultural practices, polluting areas within the bay from which large sections of the population supplemented their diet. While the results of bacteriological research, performed by Drs. Lorrain Smith and Symmers, allowed public health officials to isolate the bacteria in various sources, the hegemony of existing transmission theories – namely, that typhoid must be connected to the water source of the city – also led to a belated recognition of local contributions to the disease. However, as spatial, quantitative, and colloquial evidence continued to contradict preformed theories of transmission, the extensive inquiry launched to isolate the cause of the epidemic revealed a complex urban ecology in the city, and gave some indications of how that ecology changed in the wake of the epidemic, collapsing the shellfish industry in Belfast, changing the local relationship with the bay, and motivating a number of changes to urban structure and spatial use – a subject which will be explored in the next chapter.

While the typhoid question remained prevalent through the early twentieth century in the city, the 1910s initiated a radical break in Belfast's history. Belfast continued to grow – albeit more slowly – throughout World War I. In 1919 the town, then the eighth largest in the United Kingdom, was swept into the instability of the Irish Civil War. The Home Rule question, which dominated politics from the 1880s, engulfed Ireland in the postwar period. As the largest town in the heavily Protestant north of Ireland, Belfast served as the stronghold first of the anti-separatist movement and then, after the Government of Ireland Act 1920, the capital city of newly formed Northern Ireland. The topography of Belfast within the British Empire – as an industrial town and economic center, especially – was radically altered by the political violence of the twentieth century, as conflict within the country, combined with the Great Depression, contributed to the decline of the city's industry. While little about the city of Belfast remained the same throughout the twentieth century, it is worth noting still, perhaps, that the occasional shellfish warning appears on the shores of the Lough to this day.

Chapter 6: Urban Biopolitics of Disease: Human-Nonhuman Relationships and Urban Public Health Infrastructure

If one were to walk the streets of Bombay, Belfast, and Melbourne today, each city would hardly resemble its form at the end of the nineteenth century. Sleek, bustling, and modern, each represents the pinnacle of contemporary global urbanism in its own way; they are sites of international investment, increasingly diverse populations, and cutting-edge development plans. However, if we look more closely, we might uncover many traces of the epidemiological past. Walking the shores of Belfast harbor today, one might come across signs cautioning against the consumption of shellfish from the bay; would inevitably notice wide thoroughfares cut from one side of Bombay island to the other, while rats still scurry between holes and cracks in the sidewalks and buildings; and in the Melbourne neighborhoods of Collingwood or Fitzroy, would observe that factories that formerly processed animal products or housed brickmaking industries still stand, converted into industrial-chique apartments and coffee shops (with not a cow in sight).

These structures are not simply echoes of epidemics past, but evidence of response to these epidemics. At the beginning of the twentieth century, Belfast, Bombay, and Melbourne grappled with the effects of their respective epidemics. While previously, theories of disease dictated a broad sanitary regimen as the best method of epidemic control, the changing landscape of public health management lent a new specificity to disease. Each city now confronted a specific microbe, with a specific etiology, which resulted in specific threats to governmental authority, trade, and urban management. The plague in India, for example, was no longer simply a cluster of symptoms and mortalities, but increasingly understood to be the visible result of a complex interaction of microbe, rat, flea, and person, rendering initial plague control measures even more questionable. How each city incorporated bacteriological knowledge into their mitigation efforts, and the subsequent mitigation efforts chosen, this chapter argues, relied not only on the unique ecology of disease, but on the alignment of disease ecology with powerful municipal interests. The effects of mitigation efforts, I argue, had lasting effects on the shape of the city and on the livelihoods of human and nonhuman occupants.

The public health landscape on both the imperial and local levels underwent a critical shift at the end of the nineteenth century. The integration of bacteriology into public health practice lent new authority to local and municipal health regimes, allowing for the isolation of infection to a person, animal, or environment. Through this newfound analytic, uncovering these assemblages became a primary concern of public health; medical officers of health and public health inspectors dedicated much of their time to describing the relationships between germs and other participants in the urban ecosystem, documented in extensive sanitary reports and other vestiges of "state medicine."[1] Contemporaneous outbreaks of infectious disease among cattle and other livestock, including the 1865 rinderpest epidemic that swept Europe, drew veterinary professionals into the constellation of public health expertise, giving rise to what Joanna Swabe has termed the "veterinary public health regime" – though, as this chapter will explore, the adoption of veterinary experts into public health often varied by location.

What emerged from this shift in public health practice was an increased focus on the mechanisms of disease transmission that rendered the minutiae of daily urban life both visible and important. The ecological specificity of disease afforded by the synthesis of bacteriology and outbreak investigation at the end of the 19th century emphasized the unique conditions required

[1] David Arnold, *Colonizing the Body: State Medicine and Epidemic Disease in Nineteenth-Century India*, (Berkeley: University of California, 1993).

for each outbreak.[2] Bacteriological investigation revealed that epidemics required a unique set of ecological and social characteristics – whether it be leaky sewers erected near water supplies, milkers neglecting to wash their hands before tending to cattle, or narrow gaps in buildings admitting disease-infected rats – to establish itself at a particular time and location.

Often, however, this extensive documentation of disease dynamics resulted in very little change to public health practice. Outbreak investigation was, by nature, retrospective, and the intervention into the structure of urban space was often dictated by municipal bodies that may or may not have accepted bacteriological theory. Histories of the sanitary-bacteriological regime note this conservatism, suggest that the discovery of "germs" resulted in a uniform system of sanitary practice across urban spaces, transforming them in similar ways.[3] Historian Peter Baldwin, for example, argues that the evolution of disease etiology in the nineteenth century had little discernible impact on the practice of public health in Europe. "Knowledge of disease etiology, whether scientific in the modern sense or not, was no more than the background against which prophylactic strategies were decided upon," he argues. "It provided an overall map to guide broadly the authorities in their preventive ambitions but did not determine in any precise sense the measures chosen."[4] When restrictive measures were defined for a particular disease, literature suggests, it was often to assert the dominance of the state over colonial subjects, or to protect international or imperial networks of trade.[5]

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[2] Jacob Steere-Williams, *The Filth Disease: Typhoid Fever and the Practices of Epidemiology in Victorian England*, (Rochester: University of Rochester, 2020), 172-224.

[3] David Barnes, The Great Stink of Paris and the Nineteenth-Century Struggle Against Filth and Germs, (Baltimore: Johns Hopkins University Press, 2006): 6-8. Peter Baldwin, *Contagion and the State in Europe, 1830-1930*, (Cambridge: Cambridge University Press, 2004), 527.

[4] Peter Baldwin, *Contagion and the State in Europe, 1830-1930*, 527.

[5] Arnold, *Colonizing the Body*; Mark Harrison, *Disease and the Modern World*: *1500 to the Present Day*, (Cambridge: Cambridge University Press, 2004); Mark Harrison, *Contagion: How Commerce Has Spread Disease*, (New Haven: Yale University Press, 2012); *Public Health and the British Empire: Intermediaries, Subordinates, and the Practice of Public Health, 1850-1960*, eds. Ryan Johnson and Amna Khalid, (New York: Routledge University Press, 2012).

However, the response to disease by municipal officials in the three cities examined in this dissertation suggest there is another crucial circumstance in which bacteriological methods informed urban intervention: when their findings aligned with urban improvement schemes. At the end of the nineteenth century, urban improvement became a central preoccupation of colonial cities.[6] Echoing improvement projects modeled on the works of Edwin Chadwick and Patrick Geddes, municipal administrators attempted to modernize their cities defined by British standards, advocating programs of slum clearance, ostracization of animal industries, and the introduction of wide, straight boulevards. While social and medical historians have long argued that pathologization of these features of the urban ecosystem was key in their removal, few have considered the role of disease ecologies in facilitating this pathologization.[7] Far from a "background" to a proscribed set of sanitary interventions, I argue that the ecological specificity of disease was extremely consequential to urban action, and that knowledge of specific disease form and etiology was a powerful tool wielded by municipal interests to drive improvement projects unique to their urban structure. In Melbourne, Bombay, and Belfast, recognition of the ecological and bacteriological specificity of each disease allowed municipal interests to focus on specific improvement projects, justifying action or inaction through etiology. In each case, the recognition of disease lent authority to municipal public health and urban improvement organizations. The extent to which interventions (and claims to authority) succeeded or failed, this chapter argues, was determined heavily by how well action aligned with disease ecology.

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[6] Tristram Sharp, *Cities of Empire: The British Colonies and the Creation of the Modern World*, (New York: Metropolitan Books, 2014).

[7] Barnes, *The Great Stink of Paris*; Bruno Latour, *The Pasteurization of France*, Allen Sheridan and John Law trans., (Cambridge: Harvard University Press, 1984); Baldwin, *Contagion and the* State; and Nayan Shah, *Contagious Divides: Epidemics and Race in San Francisco's Chinatown*, (Berkeley: University of California Press, 2001) are just a few examples of this kind of work.

By identifying and tracing pathogens through urban space, public health officials made visible additional vital actors in the urban ecosystem. However, much as bacteriological investigation elucidated different features of the urban landscape in relation to each epidemic, the implications of this discovery – the existence of *Yersinia pestis* in rats, *Mycobacterium tuberculosis* in people and cattle, and *Salmonella enterica typhi* in shellfish – differed dramatically by location. In each city, the unique assemblage of government agencies, urban social and material structure, and disease ecology resulted in a different form of public health intervention. Through these acts, I argue, disease became entwined in the urban imaginary in Belfast, Bombay, and Melbourne, and together drove the reorganization of the respective cities, transforming the relationship of peoples to their environments and co-inhabitants.

Table 13. Summary of Governance Structure, Public Health Ideology and Intervention, and Outcome by City.

City	Major Governance Structure	Disease	Dominant Etiology	Major Intervention	Outcome
Melbourne	Local/Imperial (independent)	*Mycobacterium tuberculosis* and *Mycobacterium bovis*	Germ theory/Vector borne illness (cattle)	Policy/Regulation-Oriented: Integration of veterinary pathology into public health; inspection; removal of tuberculous bodies from urban spaces.	Centralized public health board, reduction in TB rates
Bombay	Municipal/Imperial (paternalist)	*Yersinia pestis*	Divided: Miasma theory for local government, germ theory/vector-borne illness (rats) for imperial government	Physical intervention: Forced Removal through slum clearance.	Dissolution of Bombay Improvement Trust; persistent plague through 1930.
Belfast	Municipal/Local (independent)	*Salmonella enterica serovar typhi*	Germ theory/vector-borne illness (shellfish)	Risk Communication: public awareness campaign around shellfish as vectors.	Continuity in public health structure; reduced typhoid rates

Ecologies of Tuberculosis and Public Health in Melbourne

In Chapter 2, we examined how patterns of urban social and infrastructural growth in late nineteenth-century Melbourne interacted with local ecology to produce a unique niche for *Mycobacterium tuberculosis.* Through a detailed study of tuberculosis rates within the city, Doctor William Thomson discovered that tuberculosis mortality was highest among residents of packed, poorly ventilated houses, located in the neighborhoods most strongly associated with industrial activity and noxious trades. Far from being a town with a "salubrious" climate for tuberculosis suffers, Thomson argued, Melbourne suffered a higher mortality rate from the disease than any other place in Australia.

While Thomson's findings on tuberculosis caused a temporary stir in the public health community, the nature of public health response in the colony resulted in relative inaction throughout the 1870s and 1880s. Unlike Bombay, where an organized and hierarchical, paternal, and racist system of colonial control resulted in rapid, often draconian, top-down public health response to epidemics, the mixed governance style of booming settler colonies like Melbourne – with power concentrated at the level of the colony or at the local (neighborhood) level, but rarely at the municipal level – often resulted in disorganized and ineffective public health systems.[8] The Central Board of Health frequently complained of non-compliant local health officials, citing neglect to turn in annual reports on the condition of their localities, or even concealment of cases of disease.[9] The result was highly inconsistent sanitary regulation and regulation of noxious trades across the city, largely dominated by special interest groups and private citizens.[10] Local health officers for each neighborhood or district (56 in all by 1890) maintained relative

[8] David Dunstan treats this argument at length in his work, *Governing the Metropolis: Politics, Technology, and Social Change in a Victorian City, Melbourne, 1850-1891*, in which he argues that Melbourne, as a site of economic growth for the Australian colonies and as a rapidly expanding city often relied on "bureaucratic managerialism" rather than elected political representation to manage its resources, a system of fragmented and loosely aligned local political institutions heavily influenced by business. The result, Dunstan argues, was a complete inability to handle issues like sanitation or water which occupied the liminal space between these two governing structures. See David Dunstan, *Governing the Metropolis: Politics, Technology, and Social Change in a Victorian City, Melbourne 1850-1891,* (Carlton: Melbourne University Press, 1984), 42-73, 121-151.

[9] Report of the Central Board of Health for the period from 1st June, 1887 to 31st May, 1888, VPRS 3253/P0000/710, item 143, pg. 5, Original Papers Tabled in the Legislative Assembly, Public Records Office of Victoria, Melbourne, Australia.

[10] John Lack, "'Worst Smellbourne': Melbourne's Noxious Trades," in *The Outcasts of Melbourne: Essays in Social History*, Graeme Davison, David Dunstan, and Chris McConville, eds, (Sydney: Allen & Unwin Press, 1985), 197-200.

autonomy from the centralized organization, and often neglected to report morbidity and mortality statistics, or even to provide annual reports to the board of their activities.

The city of Melbourne, therefore, represents an oddity in the history of late-nineteenth century British urbanism. While the cities of the United Kingdom, Canada, the United States, and many outlying colonies relied more heavily on municipal structures to dictate and shape their expansion, Melbourne remained highly decentralized, with the few corporations holding power across the Greater Melbourne Area often at the mercy of local authorities. Historians of Melbourne have attributed this characteristic to the unusual pattern of urban growth in the city – first as an informal city only retroactively granted authority by the disorganized Australian colonial government, and then as a town subject to boom-bust patterns of growth and contraction based around financial speculation and gold rushes.[11] Fragmented public health infrastructure was one symptom of a broader characteristic decentralization, with uneven urban infrastructure, poor enforcement of sanitary regulations, and a lack of systematic or organized information about the burden of disease within the city as a consequence.

TB thrived in the ecological niche constructed by decentralization, its creeping nature and long pathology particularly suited to subverting disorganized bureaucracies. Disease seemed to strike almost at random, with symptoms emerging so long after initial infection that methods of contact tracing and investigation, otherwise gaining popularity in public health structures across the Empire, were nearly impossible. Local authorities had little incentive nor the tools to tackle the tuberculosis problem, instead choosing to focus resources on explosive epidemics that clearly related to urban infrastructure – particularly typhoid and cholera (though it is worth noting, the latter generally avoided the city due to the long travel distance between other imperial ports and

[11] Dunstan, *Governing the Metropolis*, 14.

Australia).[12] Until the 1880s, therefore, health authorities focused on broad sanitarian reforms for the city – relying on miasmatic and pythogenic theories of disease, primarily deploying these logics to condemn the living conditions of the city's working class, to protect middle and upper class neighborhoods from incursion by the lower classes and noxious trades, and to advocate a centralized water system.[13] Thomson's reports faded quickly to oblivion without an organizational entity to address the city-wide problem.

While fragmented, idiosyncratic, and laissez-faire to the extreme, Melbourne's infrastructure was anything but inconsequential. As the primary port through which sheep and coal from New South Wales, beef from Queensland, and timber through New Zealand circulated through the British Empire, Melbourne was the very model of imperial expansion and specialization.[14] However, through several market crashes and unregulated expansions, the town had equally become a site of local and international embarrassment. Well-positioned to be an exemplary city of the British Empire, Melbourne instead acquired a reputation for sanitary crises and filthy environs.[15] The unregulated growth of animal trades in the inner suburbs of the city and along waterways, combined with fragmented sanitary infrastructure driven largely by local interest led to extreme pollution within the city, resulting in the convening of a number of Royal Commission to address threats throughout the nineteenth century and the informal rechristening

[12] Report on the Sanitary Condition of Melbourne: Final Report of the Royal Commission to Inquire into and Report Upon the Sanitary Condition of Melbourne, VPRS 3253/P0000/740, pg. 10, unit 10, Original Papers Tabled in the Legislative Assembly, Public Records Office of Victoria, Melbourne, Australia; William Thomson, *On Typhoid Fever*, (Melbourne: Stillwell & Knight, 1874).
[13] John Lack, "'Worst Smellbourne': Melbourne's Noxious Trades," in *The Outcasts of Melbourne: Essays in Social History*, Graeme Davison, David Dunstan, and Chris McConville, eds, (Sydney: Allen & Unwin Press, 1985), 172-173
[14] James Belich, *Replenishing the Earth: The Settler Revolution and the Rise of the Angloworld*, (Oxford: Oxford University Press, 2009), 356-358.
[15] Tony Dingle and Carolyn Rasmussen, *Vital Connections: Melbourne and its Board of Works*, Melbourne: Ringwood, 1991), 32-33; and Tristram Hunt, *Cities of Empire: The British Colonies and the Creation of the Urban World*, (New York: Metropolitan Books, 2014), 330.

of the town as “Smellbourne.”[16] Public health officials and town councilors looked outward for the tools to solve these sanitary crises, engaging with imperial medical communities to develop theories and interventions – a trend visible in Thomson himself.

The widespread acceptance of germ theory in imperial health practice beginning in the 1880s therefore changed the relationship of public health to the city’s many neighborhoods and breathed new life into Melbourne’s tuberculosis control measures. Bacteriological analysis revealed that *Mycobacterium tuberculosis* was not the only disease to thrive in Melbourne’s unique ecology; with the rise of tuberculin testing in both humans and livestock in the 1880s, medical professionals (and veterinarians in particular) found its twin, *Mycobacterium bovis*, to be pervasive among cattle. As a major economic resource and an organism bred to be nearly entirely dependent on human care, cattle were more easily surveilled than their human counterparts and their environments more easily controlled, providing an ideal demographic for observation, theorization, and control of tuberculosis.

Over the course of the early twentieth century, therefore, Melbourne’s public health infrastructure united around the investigation and control of bovine tuberculosis, thus drawing bacteriology, veterinary pathology, and human health together in a centralized system. By providing justification for large-scale regulation of the cattle industry on the grounds of both economy and human health, the recognition of Koch’s bacillus lent new authority to centralized public health structures and validated longstanding goals for the sequestration of noxious trade industries to the outskirts of the city and regulation of the meat industry. By embracing germ theory and aligning themselves with major urban improvement goals vocalized by the city’s wealthy residents, the Central Board of Health (and the Board of Public Health thereafter)

[16] Lack, “’Worst Smellbourne,’” 170-180.

simultaneously became a center of bacteriological expertise and action and altered the ecological niche for tuberculosis within the city.

Tuberculosis bacillus and The Ecology of Neglect

In 1870, the same year that Thomson first published *On Phthisis*, the governor appointed a royal commission to inquire into the public health nuisances of Melbourne's "noxious trades."[17] The Commission had been a long time coming; as early as 1855 private citizens and local health authorities had called for regulation of the city's private and public abattoirs, citing putrid miasmas and carcasses in the streets and sewers, resulting in the (largely ineffective) Yarra Pollution Act of 1855, which prohibited slaughterhouses and tanneries from dumping refuse into the river (but, it should be noted, not into peripheral rivers in the city). Concentrated at Collingwood, Port Melbourne, South Melbourne (otherwise known as Emerald Hill) and Fitzroy, the abattoirs were beacons of Melbourne's economic prosperity; largely unregulated and highly productive, the abattoirs were, as historian John Lack noted, testimony "to the worship of economic progress through unfettered private enterprise, and to the influence of imported British traditions of industrial management."[18]

However, the Commission revealed that British slaughtering practices did not hold up well to the Australian climate. Products quickly putrefied in the warm weather, and as Lack notes, pelts from slaughter yards and abattoirs were frequently rotten before transported to their place of sale.[19] In its initial report, the Commission found, "As a rule, the sites were bad, the arrangements for the approach and keeping of stock for slaughter defective, and the

[17] Lack, "'Worst Smellbourne'," 172-173.
[18] *Ibid.*
[19] Lack, "Worst Smellbourne," 177.

accommodation insufficient; the drainage and the mode of disposal of offal were most unsatisfactory, and general cleanliness was imperfect or neglected."[20] They recommended the immediate closure of abattoirs in South Melbourne, Port Melbourne, Collingwood, and Footscray. However, it took nearly twenty years (and the recognition of *Mycobacterium bovis* as a major health threat) before these abattoirs were closed or relocated.

Evidence suggests that bovine tuberculosis was a significant problem among stock in Australia before it received governmental attention. Veterinarian Graham Mitchell, for example, wrote in an 1882 issue of the *Australasian Veterinary Journal* that he "first drew attention to the prevalence of this disease in Australia in 1871, through the press," and that "since then it has been annually on the increase; and, as it is not included in the schedule of diseases in the Diseases in Stock Act, it is likely to cause great mortality amongst our herds and seriously endanger human life." He argued that much of human tuberculosis mortality was likely attributable to the disease, and especially that the "high death rate amongst children reared on the bottle is attributable in great measure to the use of milk from tuberculous cows."[21] Despite their timeliness, these arguments largely remained sequestered in veterinary journals, and were therefore omitted from medically-centered public health discourse.

The publication of Koch's study, "The Etiology of Tuberculosis," in *the Australian Medical Journal* in 1882, however, assigned new relevance to this body of work. As awareness of the potential threat of meat and milk products to human health burgeoned within European scientific and medical networks, the Department of Agriculture appointed a Board to Inquire into the

[20] Final Report of the Royal Commission to Inquire into and Report Upon the Sanitary Condition of Melbourne, 6.
[21] Graham Mitchell, "Diseases of Stock," *Australasian Veterinary Journal* 1(1882): 112.

"Existence and Extent in Victoria of the Disease in Cattle known as Tuberculosis."[22] The annual reports produced by the Board reveal the shifting nature of bovine tuberculosis etiology. Continued reports from the board document a solidification of bovine tuberculosis etiology and pathology and revealed the extent of bovine tuberculosis in the city. In their report, the Board concluded, "Inspection indicates that about 5-6 percent of cattle slaughtered are found to have tuberculosis, and that 95 percent of those found to have tuberculosis are then allowed to be sold for consumption."[23]

Table 14. Return of the Numbers of Cattle Slaughtered and Inspected at the City Abattoirs, July 1884-June 1885.

Period	Number of Cattle Slaughtered and Inspected	Number with TB	Number with TB Condemned	Number with TB not Condemned
July 1884-Dec 1884	16,780	1194	74	1123
Dec 1884-June 1885	18,722	780	39	741
Total	35502	1974	113	1864

SOURCE: Report on the Existence and Extent in Victoria of the Disease in Cattle Known as Tuberculosis, v.

While the report shed light on the lack of control and inspection of tuberculous meat, it also renewed attention to the state of Melbourne's abattoirs. Detailed descriptions of abattoir conditions suggest that they formed an ecological niche for *Mycobacterium bovis*. The board observed that in the city abattoirs, "the paddocks are not levelled nor properly drained, and,

[22] GL Singleton, "Human and Cattle Diseases in Australia," *The Australasian*, 1(1891): 7; Report of the Board Appointed to Inquire Relative to the Existence and Extent in Victoria of the Disease in Cattle Known as Tuberculosis, Whether its Existence is Likely to Be Detrimental to the Public Health, and What Preventive Means Should be Adopted, together with Minutes of Evidence, presented to both houses of parliament by his excellency's command, V/AA/145/04/03, No. 871, Original Papers Tabled in the Legislative Assembly, Public Records Office of Victoria, Melbourne, Australia.

[23] Report on the Existence and Extent in Victoria of the Disease in Cattle Known as Tuberculosis, vi.

therefore, in parts become swampy in wet weather; that the site of the abattoir buildings is not so levelled as to secure effective flow in all the drains within and between the buildings; that the floors of the various compartments are too little removed from the water in the subsoil," that little cover exists for the animals to protect from weather in the paddocks and pens, "that the ventilation of many of the compartments is insufficient," and that "there is not that universal regard for cleanliness which should be enforced."[24] These conditions – damp, dilapidated housing, low circulation of air, and poor sanitary conditions for residents – echoed the environments that facilitated the spread of tuberculosis in the city at large, and thus likely facilitated the transfer of bovine tuberculosis between cattle.

In the suburban abattoirs, the conditions were far worse, and furthermore, there was "practically no official inspection of the meat, with regards to its fitness for human food." Once again local health officers were of little help, as "in some instances it has been made certain that the local boards of health and their officers are either not congisant [sic] of, or will not recognise, the evil conditions hereinbefore stated."[25] Close quarters, lack of ventilation, lack of regulation, and general noxious conditions thus provided the perfect environment for bovine tuberculosis. It is perhaps no surprise, then, that by the turn of the century, Veterinary Pathologist Samuel Sherwen Cameron noted that tuberculosis was "the most common [disease among cattle], the least easy to recognize, and the most dangerous, both as regards cattle and man."[26]

Integrating Nonhumans into Public Health: The Board of Health, Tuberculosis, and Veterinary Pathology

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[24] Sanitary Condition of Melbourne: First Progress Report of the Royal Commission to Inquire into and Report Upon the Sanitary Condition of Melbourne, VPRS 8609/P/0001, unit 18, pg. xxvi, Original Papers Tabled in the Legislative Assembly, Public Records Office of Victoria, Melbourne, Australia.
[25] First Progress Report of the Sanitary Commission, xxvii.
[26] Samuel Sherwen Cameron, Dairy Farms, Their Sanitary Arrangements and Management: Report to the Board of Public Health, Victoria, 1901, H2013/6023, pg. 1, State Library of New South Wales, Sydney, Australia.

The Tuberculosis in Cattle Board not only revived interest in abattoirs as sites of TB transmission but acted as a catalyst for the centralization of public health authority in Melbourne around the burgeoning authority of veterinary pathology.[27] As veterinary pathologists capitulated on the contagious nature of bovine tuberculosis, public health officials saw the opportunity to strengthen its own authority by drawing on the expertise of the complimentary field. The result was an increasing integration of veterinary pathology into public health science, a phenomenon which rendered cattle increasingly visible in the public health record. Furthermore, tendencies among veterinary professionals employed by the public health board to use a combination of bacteriology and comparative pathology to establish characteristics of a novel bacteria resulted in constant comparison between human and bovine tuberculosis.[28] Human and cattle health became increasingly intertwined in the public health record, resulting in a series of regulations around bovine TB that strengthened the centralized health authority and consolidated governmental power over abattoirs. Reorienting public health discourse around bovine tuberculosis allowed the Central Board of Health to pass legislation that standardized practices of inspection, accomplished the longstanding sanitary goal of banishing abattoirs from the city.

One of the most striking aspects of veterinary science that facilitated the link between human and animal health was the practice of comparative pathology. The Tuberculosis Board drew on homology to characterize tuberculosis in their initial report outlining the infective nature of bovine TB, frequently drawing comparisons between human and cattle:

> Ordinary phthisis in man and the tuberculosis of bovines both usually commence insidiously with languor, loss of strength, want of appetite; in both, the earliest distinct

[27] GL Singleton, "Human and Cattle Diseases in Australia," 7.
[28] Peter Atkins notes similar developments in London, where comparative pathology emerged as a "new research frontier." He emphasizes that in the United Kingdom, veterinarians were gaining authority into the 1880s through their proven ability to eliminate diseases like bovine pleuro-pneumonia from some states. Peter J. Atkins, " 'Your Enemy the Cow': Actors, Networks, and Competitive Interests in the Medical Veterinary and Administrative Debate about Bovine Tuberculosis in Britain, circa 1800-1964," unpublished ms., 2006a.

> symptom is usually a dry cough; in both, progressive emaciation occurs in the great majority of cases; the cough becomes moist, the expectorated matter being usually swallowed by cattle until a late period of the disease; diarrhea is frequently profuse in the latter stages of both; and death takes place from exhaustion, or from some intercurrent complication. In both, when cavities form, there are marked symptoms of hectic fever. In both, the disease may run a very rapid course, or may be very chronic, extending through a long term of years.[29]

The relationship was undeniable, according to the Board, as exhibited through a series of similarities, including first, "similarity in the symptoms, progress, and history of the two diseases," including a dry cough, progressive emaciation followed by a wet cough, and eventually death from exhaustion either rapidly or over the course of years; second, "similarity in their predisposing causes" including dampness of soil, improper food, bad ventilation and drainage, cold, and "depressing and debilitating agencies"; third, "similarity in the microscopic characters of the elemental structures formed in both diseases"; fourth, "the constant presence of a specific form of bacillus in the products of both diseases"; and fifth, "the unfailing production of the same disease, acute disseminated miliary tuberculosis, in rabbits and guinea-pigs, by the inoculation of tubercle bacilli cultivated either from human or from bovine tubercle."[30] By drawing attention to the similarities in presentation of the disease within the patient, the environments in which disease was most often formed, the pathology of the disease, and its verified contagiousness (via Koch's postulates) in model animals, veterinary pathologists argued that both bovine *and* human tuberculosis were contagious diseases spread through similar environments and caused by a specific bacteria – an argument far from settled in the medical discourse.[31]

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[29] Report of the Board Appointed to Inquire Relative to the Existence and Extent in Victoria of the Disease in Cattle Known as Tuberculosis, xxix.
[30] Report of the board, xxix.
[31] Atkins, "Your Enemy the Cow," 1-21.

Equally striking in the report was the comparative bacteriology of the two microbes. The Board noted that, regarding their similarity, "On this point there is no contention. Thus, in both human and bovine tubercle we find giant cells with many nuclei, usually arranged round the periphery of the cell; epithelioid elements, which, like the giant cells, stain in a constant manner with dyes; and around these larger elements, in young tubercles, a multitude of small round cells. In both the human and the bovine disease the tubercle may organize or degenerate…"[32] In the isolation of both bacteria, the comparative pathology, and the comparative bacteriology, veterinary pathologists left little doubt that the two diseases were intertwined.

As the epidemiology of bovine TB and human TB solidified, sympathetic public health officials began to see solidarity between the two animals as key to the eradication of tuberculosis. The importance of the strength of the comparison between human and bovine tuberculosis for establishing authority for the germ theory of human tuberculosis was not lost on Dr. Astley Gresswell, medical inspector of the Victorian Board of Public Health, who in his address at a meeting to discuss the necessity of forming an Association for the Prevention and Cure of Tuberculosis, brought together humans and cattle as mutual sufferers of tuberculosis, arguing that "*Tuberculosis is the greatest of all Plagues of Man and of Cattle*,"[33] and that efforts at control and eradication should be focused on "the whole of the vertebrate sub-kingdom." He asserted, "It is *communicable* from man to man, from man to animals, from animals to man, and from animals to animals – not by the breath nor by the perspiration, but by discharges from invaded parts of the system, discharges which swarm with the bacillus."[34] The extent to which the two diseases formed one public health imaginary is visible in his recommendations for

[32] Report of the board, xxx.
[33] D. Astley Gresswell and Victoria Board of Public Health, *Address on Tuberculosis and its Prevention,* (Melbourne: R.S. Brain, Govt Printer, 1899), 10.
[34] Gresswell, 8.

tuberculosis control, which proposed similar interventions for people and cows: isolation, treatment with open air, and notification.

The relationship between these two interests was codified in legislation at the end of the decade. Written into the Public Health Bill 1889, which officially abolished the central board of health and established a Department of Public Health, were lengthy sections on the regulation of the meat industry. The bill established a board of two appointed members and seven representatives of the Municipal Councils to administer public health, in contrast to original format of nine government-appointed members who had little to do with the municipalities. The Board "was empowered to make regulations" related to dairy farms and milk stores, including regular inspection by municipal officers and compulsory registration of cattle and dairy farmers across Victoria.[35] The newly established Board emphasized that the reports furnished by the Tuberculosis board "showed the absolute necessity" of closer supervision of dairies, "especially taking into account the frequency with which disease is spread through the agency of unwholesome milk, and the extensive use of milk as an article of food," a sentiment echoed widely among the Australian Medical and Veterinary communities.

The development of interventions, theories, and preventative measures targeting bovine tuberculosis brought cattle under biopolitical control by an increasingly powerful and centralized public health authority. While this phenomenon can be observed across the British Empire, the role of veterinary pathologists in Melbourne's public health structure placed the relationship between cattle and tuberculosis front and center in public health reports and action. In the first decade of the 20th century, Samuel Sherwen Cameron, appointed veterinary inspector to the

[35] "The Public Health Act 1889", Friday April 25, 1890, Victorian Government Gazette, No. 38, pg. 1527, Victoria Government Gazette Online Archive 1836-1997, State Library of Victoria, Melbourne, Australia. http://gazette.slv.vic.gov.au/

Board of Public Health, circulated a series of pamphlets ordered by the board to the owners of cow-byres and milksheds describing in detail the appropriate sanitary conditions for the prevention of bovine tuberculosis. He notes, "The importance of the dairying industry, so far as milk and milk products are concerned, both from a public health and from a commercial point of view, has increased enormously during recent years," asserting that bacteriology has revealed the importance of proper sanitary arrangements for the health of the public – beginning with the health of cattle themselves.[36] Sherwen argues that "building should be constructed for the health and comfort of both cattle and employees," advocating sophisticated drainage systems for the elimination of waste, adequate lighting, ventilation that allows for the "ample exchange of air," and adequate space for each cow of at least 600 cubic feet.[37] Cameron also took great pains to describe the general health habits of cattle, including their respiration, gait, and eating habits, and looked for signs of disease based on the alteration of these habits.

Sherwen Cameron integrated novel methods for detection and prevention into his recommendations to industry through quarantines and regular tuberculin testing, once again reinforcing the cruciality of veterinary pathologists to public health infrastructure: "All dairymen and stock-owners should have this test applied to their cattle," he notes, "not merely for their own protection, but also in the interests of the health of their customers. It should be applied only by a qualified veterinary surgeon thoroughly experienced in the use of it, or by a pathologist having such experience."[38]

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[36] Cameron, Dairy Farms, their Sanitary Arrangements and Management, 3-4.
[37] Cameron, Dairy Farms, 3-4.
[38] Cameron, Dairy Farms, 16-17. The centrality of tuberculin testing in Cattle was echoed by none other than J. Ashburton Thompson, President of the Board of Health of New South Wales, and central proponent of the rat-flea theory of plague. In his own publication on Bovine TB, Ashburton Thompson notes, "Our interest in the prevention of tuberculosis among oxen is twofold. We share in the commercial interest which prevention of that disease has for this pastoral country. Tuberculosis is a disease which in those districts where it is prevalent inflicts very great losses on the breeder…Secondly, we know that tuberculosis in man is a too prevalent disease; or, to put it in another way,

In addition to the documentation of their immunological lives in the Public health record, bovine tuberculosis encouraged the documentation and surveillance of the minutiae of daily life for cattle. Cameron, for example, documented extensively the daily behavior of cattle, and justified this detail by arguing, "Observation of the attitude of a patient is always of material assistance in forming an opinion as to the nature of the disease." He noted that a healthy cow "stands firmly, with weight equally disposed on all four limbs…generally engaged in either feeding or ruminating (chewing the cud)," and noted that "the turning out of the elbows and the fixing of the ribs along with distressed breathing, are characteristic and pathognomic."[39] He recommends clipping of hairs around the udders, constant surveillance for unusual behavior and for scabs, chaps, blisters, or ulcers. Cattle that demonstrated unusual behavior were to be quarantined or their death quickened. Through this detailed observation, cattle were transformed into "patients," their suffering subsumed under the sanitary-medical regime.

The new biopolitics of cattle in Victoria were reinforced by a further cascade of public health regulations, starting with the Meat Supervision Act of 1900, requiring inspection of cattle at time of slaughter and adopting the recommendations of the Tuberculosis Board for regulation of dairies, butchers' shops, and abattoirs, followed by the Milk and Dairy Supervision Act and the Pure Food Act of 1905.[40] In short, the monitoring of cattle for bovine tuberculosis created a space for cattle in the public health record and in urban governance as complex, living organisms with individual behaviors, habits, and best practices; through disease, cattle transform from

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we now know that tuberculosis in man is a preventable disease, which yet is far from being effectually prevented." See J. Ashburton Thompson, "On the Application of the Tuberculin Test to the Ox," *Australian Medical Gazette* 16(1897): 582-585.

[39] Cameron, 8.

[40] 142 Regulation: Pure Food Act, VPRS 3253/P000/1016, Original Papers Tabled in the Legislative Assembly, Public Records Office Victoria, Melbourne, Australia; and James Lewis Milton, *The People's Health: Public Health in Australia, 1788-1950,* (London: Praeger Press, 2003), 138.

commodity to co-inhabitant, a living member of the urban system that requires specificities from its environment to maintain health and well-being.

Tuberculosis on the margins

With shifting theories of transmission and public health structures came a shift in the biopolitics of tuberculosis for both human and non-human occupants of the city. Increasing awareness of the death rate of tuberculosis, spurred by the work of Thomson and veterinary pathologists like Cameron, changed the quotidian behaviors and interactions of residents of the city in evolving ways over the late nineteenth century. Medical and veterinary officials, upon concluding that bovine tuberculosis was communicable to humans, and might be responsible for a large proportion of tuberculosis infections, often encouraged legislation that transformed the cow from a commensal animal into a vector. More often than not, cattle interacted with the state in the form of their deaths, with public health and economic records most often conveying numbers of cattle killed or sides of beef sold. This grim arithmetic became even more pronounced in the hunt for *Mycobacterium bovis*. Slaughtering in abattoirs became not only an economic activity, but a public health activity, as officials examined each carcass for signs of tuberculosis, concluding that 5-7 percent of the animals killed in the city's abattoirs suffered acutely from the disease (though, it should be noted, this rarely stopped their meat from being sold). Those found to be tuberculous before death suffered the same fate: In 1887, the Central Board of Health agreed to rules for inspectors dealing with suspected tuberculosis in cattle that included "condemning all wasted animals in which even a moderate amount of tubercle was found…no matter what their condition" and burning of their carcasses.[41]

One of the most important effects of the transformation of cows into vectors, and the centralization of the public health board around this health risk, was the elimination of livestock in urban ecology. Emerging concerns over bovine tuberculosis not only reorganized the lives of cattle within the city but played a role in the reorganization of the city as a whole. As John Lack notes, concerns over the "noxiousness" of abattoirs led to continuous concerted efforts for their banishment beyond city limits as early as 1855.[42] Bovine TB concerns aligned with burgeoning complaints over noxious industry and abattoir sanitation (which had led to a renewal of a royal commission to inquire into the sanitary condition of Melbourne and its suburbs in 1888-1889). and acted a catalyst, alongside concerns over typhoid fever, for mass sanitary reform efforts, and then eventually closures of abattoirs and the relocation of dairies, slaughterhouses, and butchers outside the city limits. In the year of the report, abattoirs were closed in St. Kilda, Collingwood, and Port Melbourne, and notices issued to several other suburban abattoirs. Those that remained fell under strict government regulation under the Public Health Act of 1889, and even further regulation was suggested in the report to take care of "nuisance" in the abattoirs. Under section 32 of the Public Health Act of 1889, trades labelled as "noxious" were made extremely vulnerable to removal, as it authorized "persons, whether resident in the district or not, to object to the continuance or extension of any such trade," and required the consent of Local Authorities

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[41] Treatment of Carcasses of Tuberculous Cattle, Friday, June 17th 1887, Victoria Government Gazette 53, pg. 1839, Victoria Government Gazette Online Archive 1836-1997, State Library of Victoria, Melbourne, Australia. http://gazette.slv.vic.gov.au/

[42] Lack, 183.

for the establishment of those trades in their neighborhoods.[43] The Sanitary Commission also openly advocated establishing "country-killed meat" industries like those present in Sydney.[44]

The result of this strict regulation was not only the sequestration of cattle to the suburbs and the margins of the urban ecosystem, but also the destruction of working-class livelihoods within the city. Historian Andrea Gaynor notes that livestock and poultry were often owned by the working classes, and that selling milk was a way to recoup losses of ₤10-12 on purchase of a cow. Initial regulations around milk may have in fact worsened bovine tuberculosis incidence in the city, because selling milk required registration with local or central boards of health and therefore paying an annual fee of between 10s-₤1, resulting in many householders selling their milk "on the sly." As restrictions tightened, Gaynor notes that "the many poor households in inner-city areas were often the first affected by regulations which banned livestock altogether."[45] For those whose livelihoods were deemed "noxious" by the Public Health Board, compensation was rare, and restricted to payment "only for actual loss sustained in removal."[46] As the trades and mutualisms that defined working class livelihoods were pushed out of the city and into a handful of inner suburbs, class lines hardened within the city, and working class residents found themselves surrounded by a greater concentration of noxious trade industries and fewer opportunities for subsistence through individual household ecosystems.[47] The consolidation of public health legislation around cattle drove what Peter Atkins has called The Great Separation.[48]

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[43] Final Report of the Royal Commission to Inquire into and Report Upon the Sanitary Condition of Melbourne, VPRS 3253/P0000/740, unit 10, pg. 6, Original Papers Tabled in the Legislative Assembly, Public Records Office Victoria, Melbourne, Australia

[44] *Ibid.*

[45] Andrea Gaynor, *Harvesting the Suburbs: An Environmental History of Growing Food in Australian Cities*, (Perth: University of Western Australia, 2006), 45-47.

[46] Final Report of the Royal Commission to Inquire into and Report Upon the Sanitary Condition of Melbourne, 5.

[47] John Lack, 198-199; Andrea Gaynor, "Fowls and the Contested Productive Spaces of Suburbia, 1890-1990," in *Animal Cities*, 205-219.

[48] Peter Atkins, "Animal Wastes and Nuisances in Nineteenth Century London" in *Animal City*, 46-51.

The bacteriological transition in Melbourne, driven by bovine tuberculosis and the eruption of several typhoid fever epidemics in the 1890s, thus consolidated public health authority at the municipal level and catalyzed the destruction of an urban niche for livestock.

Increasingly bacteriological characterizations of human tuberculosis, fostered by comparison with bovine tuberculosis, provided justification for the sequestration of humans as well as animals. Gresswell himself noted, in his address to the newly formed tuberculosis association, that "the isolation of and the cure of persons and animals affected with this disease will, of course, prove preventive measures."[49] Central to this isolation was the construction of sanatoria on the outskirts of the city. In the early twentieth century, the State Government of Victoria constructed four tuberculosis sanatoria on the outskirts of Melbourne's urban area: Greenvale (1905-1956), Number 1 Military Sanatorium (1916-1960), Janefield (1920-1925), and Gresswell (1933-1970). Unlike many other tuberculosis sanatoria of the time, these sites were not located on the sides of mountains or in rural areas, but near the city, and directly accessible by railroad. Historian Rebecca Le Get argues that this clustering of sanatoria can be attributed to attempts by the Victorian government to make state-run sanatoria accessible to the (primarily working-class) consumptives who lived there, and to allow for the sharing of resources, like experts and supplies, between sanatoria.[50]

Isolation of human tuberculosis sufferers, however, was expected to be voluntary, participating in a form of social and economic self-sacrifice rather than bodily sacrifice. At the same time that tuberculosis control measures were implemented in both humans and cattle, Alison Bashford notes, the gradual acceptance of tuberculosis communicability emerged in

[49] Gresswell, 7.
[50] Rebecca Le Get, "More than just 'Peaceful and Picturesque': How Tuberculosis Control Measures Have Preserved Ecologically significant Land in Melbourne," *Victorian Historical Journal* 89 (2018): 67-87.

concert with exclusionary sanitary ideologies that defined Australia as a white settler colony. While many infectious diseases were characterized as external, brought to the Australian colonies by nonwhite settlers, tuberculosis was seen as a "'great white plague'…a disease originating and belonging to 'civilized man'.[51] Thus, it became the responsibility of individual persons within the civic body to maintain the health of the whole, a "hygienic citizenship."[52] It was a sense of moral and political responsibility that drove consumptives to the margins of the city, and likely kept them returning to sanatoria long after antibiotics rendered the disease curable.

The creation of these sites had long-term impacts on the relationship of Melbournians to their environment lasting well into the present. As tuberculosis epidemiology promoted the idea of isolation and modification of behavior for consumptives, it led to the construction of sanatoria, which changed the relationship of Melbourne residents to each other and to the city. Rebecca Le Get argues that the construction of these sites had an interesting and unintentional effect on the local ecosystems: by acting as areas of isolation in the greater Melbourne area and occupying crown land, they became prime locations for the creation of biodiversity areas and conservation parks after sanatoria closed. The construction of "salubrious" environments for consumptives outside the city therefore preserved the remnants of grassy eucalypt woodlands otherwise removed by Melbourne's urban development and serve as a "cultural heritage" sites for nature appreciation.[53] Thus while tuberculosis control measures removed a niche for cattle within the city of Melbourne, they also preserved an ecological niche for many of Australia's unique species near the city.

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[51] Alison Bashford, *Imperial Hygiene: A Critical History of Colonialism, Nationalism, and Public Health,* (London: Palgrave MacMillan, 2004), 78-79.

[52] Bashford, 78-79.

[53] Le Get, 81.

Plague, Displacement, and the Bombay Improvement Trust

In Chapter 3 and 4, we examined how plague came to occupy an ecological niche in Bombay, and how plague control measures through public health infrastructure altered the relationships of humans, nonhumans, and plague. The rise of bacteriology led to a focus on the daily life of the rat among imperial public health officials. Colonel Bannerman reframed human plague cases in terms of their likely proximity to or interaction with rats, in terms of occupation and class (for example, he argues that the rat-flea theory "explain[s] to some extent the greater incidence of plague among classes, *viz.*, the grain dealers, stable-keepers, and poorer classes generally,") by religion ("while the notorious severity of the disease among the Jains may be due to their dislike for the destruction of animal life in any form, and their encouragement of the rat by feeding,") and by wealth ("The greater immunity from attack of the rich Natives and Europeans is accounted for perhaps by the strongly built houses, roofed with Mangalore tiles or patent stone, which do not afford shelter to rats; and to their habit of removing refuse of all sorts to a distance").[54] Through his work, Bannerman posited a structural epidemiology for plague defined by its primary sufferer, the rat. His work, published in the *Journal of Hygiene* and widely circulated in British medical journals, laid the groundwork for a global project of "building out the rat," which dozens of port cities undertook in the early 20th century. Bombay, however, was not among them.

While specialists in Melbourne largely relied on bacteriological methods developed elsewhere in the Empire (especially mainland England) to justify changes to public health structure and legislation, Bombay housed the premier site of bacteriological research on plague and served as the primary site at which the rat-flea theory of disease was solidified, detailed, and

[54] "XXII. The Epidemiological Observations made by the Commission in Bombay City," *Epidemiology and Infection* 7(1907): 724-798. DOI: 10.1017/S0022172400033684.

brought to the epistemological forefront of medical science. However, the city suffered a fragmentation of local government and imperial scientific body that prevented these major bacteriological discoveries from translating to effective municipal work. In Melbourne, attempts to mitigate fragmentation of public health infrastructure led to the adoption of bacteriological epistemology, which facilitated the transfer of power from local authorities to the municipal government. Conversely, conflicting sanitary goals amongst municipal authorities and Bombay Bacteriological Laboratory scientists in Bombay resulted in municipal authorities eschewing bacteriological innovation, much to the detriment of the city's plague-stricken residents and the municipal government itself. While interventions from the Melbourne Board of Public Health removed the urban niche for cattle and tuberculosis sufferers, I argue in this section that interventions from the Bombay municipal authorities that relied on a saprophytic and miasmatic imaginary likely exacerbated plague conditions by shifting (but not eliminating) the ecological niche for rats within the city.

While campaigns emerged on a small scale to control points of access between humans and rats, including short-term use of the Danysz virus, experiments on the jumping power of rats, and the compilation of reports detailing possible rat-guards and housing structures that might prevent the entrance of rats into homes, enthusiasm for these measures died in translation. The Government of Bombay instead established the Bombay Improvement Trust in 1898 in response to the plague, with the goal of enacting "a comprehensive scheme for the improvement of the City of Bombay, more especially in respect to the better ventilation of densely inhabited parts, the removal of insanitary dwellings, and the prevention of overcrowding."[55] The Trust set out with five major goals:

> *[F]irstly*, the improvement 'en bloc' of areas declared to be insanitary and incapable of any less drastic measure, *secondly*, the creation of new Streets for the purpose of affording efficient ventilation to the more crowded parts of the City and of increasing and improving the means of inter-communication, *thirdly,* the improvement and development of available areas in the Island and the reclaiming of land from the Sea to provide Building-sites for the expansion of the City, *fourthly,* in connection with the foregoing, the provision of sanitary accommodation for the poorer and working classes who are displaced by those operations, and *fifthly*, also in connection with Street and Improvement Schemes, accommodation for the Police.[56]

While initially these plans aligned with contemporary theories of plague transmission, bacteriological inquiry quickly identified the rat as the primary culprit in disease transmission. Ignoring Bannerman's recommendations for rat control, the Trust continued to rely on miasmatic and hygienic theories of disease, which together formed what historian Prashant Kidambi referred to as "contingent contagionism," to guide their building standards.[57] This approach pathologized the built environment of poor communities of Bombay as sites of transmission, and furthermore, pathologized the residents of these communities as potential carriers and transmitters of plague. While Kidambi argues that the continuation of contingent contagionism, even as the rat-flea theory gained acceptance, was largely the result of skepticism on the part of municipal officials for new scientific theory, I argue that the Trust relied on an outdated etiology of plague because it aligned with plans for urban renewal that benefitted the millowners and landowners elected or appointed by the corporation.[58]

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[55] Bombay Improvement Trust, Administration Report for the Year Ending 31st March 1901, Bombay Improvement Trust Administration Report 1898-1899 to 1904-05, pg.3, GP_00133648, Royal Asiatic Society, Mumbai, India.
[56] G Owen Dunn, "A Lecture Delivered at the Health Section of the Bombay Industrial Exhibition in January 1905 by G. Owen Dunn, Esq, Chairman of the Bombay Improvement Trust," in J.P Orr, *The Bombay City Improvement Trust from 1898 to 1909*, (Bombay: Times Press, 1911), 5-6.
[57] Prashant Kidambi, "'An infection of locality': plague, pythogenesis and the poor in Bombay, c. 1896-1905," *Urban History* 31(2004): 249-252.
[58] Kidambi, "An infection of locality," 252-255.

The effect of these projects on rat ecology was likely complex. In a recent review, Byers et al. have noted that habitat modification alters rat movement, resulting in migration of colonies.[59] Migration due to construction and control efforts (as discussed in Chapter IV) often lead to recolonization by rats of vacated areas due to migration from surrounding areas, and re-infestation of sewer systems.[60] When rats range into already colonized areas, studies indicate that the invading individuals have difficulty establishing harborage, and thus extend their range further for appropriate shelter.[61]

The role of urban renewal projects in facilitating the movement of rats, and particularly in displacing rodent populations, has been studied extensively, with overwhelming evidence that these projects increase instances for rat-human contact.[62] Mass rodent displacement likely complimented mass human displacement, as rats living in these neighborhoods would have fled to nearby neighborhoods, leading to population mixing – and chances for transmission. A report by GF Petrie and Ronald Todd on plague in Cairo in 1923, however, found that rat populations were less likely to traverse wide streets than narrow streets.[63] Thus, the widening of roadways as part of the Trust's beautification projects may have broken up rat territories, reducing their range, and may have even reduced transmission of plague between rats. Continued cyclical transmission of plague and elevated mortality rates as late as the 1930s, however, as well as the aforementioned deadly outbreaks of plague within chawls built by the Trust, suggest a failure to

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[59] Kaylee Byers, Michael J. Lee, David M. Patrick, and Chelsea G. Himsworth, "Rats About Town: A Systematic Review of Rat Movement in Urban Ecosystems, *Frontiers in Ecology and Evolution* 7(2019): 7. DOI: 10.3389/fevo.2019.00013.
[60] Byers et al., 7.
[61] Byers et al., 7.
[62] Byers et al., 8; SA Battersby, R Parsons, and JP Webster, "Urban Rat Infestations and the Risk to Public Health, *J. Environ. Health. Res.* 1(2002): 4-12; Matthew Combs, E.E. Puckett, J. Richardson, D. Mims, and J. Munshi-South, "Spatial Population Genomics of the Brown Rat (*Rattus norvegicus*) in New York City. *Mol. Ecol.* 27(2018b): 83-98, DOI: 10.1111/mec.14437.
[63] G.F. Petrie and Ronald E. Todd, *A Report on Plague Investigations in Egypt*, (Cairo: Government Press, 1923): 125-129; Byers et al., 2-6.

alleviate the disease.[64] Plagued with mass expenditure, snail-like progress, and overly ambitious, culturally- and ecologically-deaf schemes, therefore, the Improvement Trust's greatest legacies were controversy, plague exacerbation, and the mass displacement of the city's working class between 1898-1930. By ignoring the disease dynamics explicitly outlined by the Bombay Bacteriological Laboratory in favor of an imaginary that aligned with the city's beautification goals, the Trust "began to not only exacerbate the problems that [they] had set out to solve but also create new ones."[65]

Logics of Slum Clearance

At the Bombay Industrial Exhibition in January 1905, G. Owen Dunn, then Chairman of the Bombay City Improvement Trust, reflected on the legacy of the organization. It is perhaps telling that he began this talk by quoting a satirical poem in a local newspaper, in which the fictional speaker hailed from Karachi:

> "We may not have a Ballard Pier and Passengers Galore,
> "We May not have a 'Taj Mahal' upon our sandy shore,
> "We may not have to live in an Apollo Bunder Flat,
> "But we haven't an Improvement Trust and thank the Lord for that!"[66]

This quote is perhaps the most lyrical of complaints leveraged against the Bombay Improvement Trust – but it was no means solitary. Between 1898-1910, the Bombay Improvement Trust undertook major slum clearance projects focused on labouring districts in the city, displacing thousands of residents with insufficient plans to rehouse them. The method of

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[64] Epidemiological Observations Made by the Bombay Commission, 782.
[65] Prashant Kidambi, "Housing the Poor in a Colonial City: The Bombay Improvement Trust, 1898-1918," *Studies in History* 17(2001): 60, DOI:10.1177/025764300101700103; Caroline Arnold, "The Bombay Improvement Trust, Bombay Millowners and the Debate over Housing Bombay's Millworkers, 1896-1918" *Essays in Economic and Business History* (2012): 106. G Owen Dunn, "A Lecture Delivered at the Health Section of the Bombay Industrial Exhibition in January 1905 by G. Owen Dunn, Esq, Chairman of the Bombay Improvement Trust," 1-18.
[66] Dunn, "A lecture delivered at the Health Section of the Bombay Industrial Exhibition," 1.

slum clearance, and the miasma-centered logic employed to justify it, are best exemplified in the schemes at First Nagpada and Mandvi.

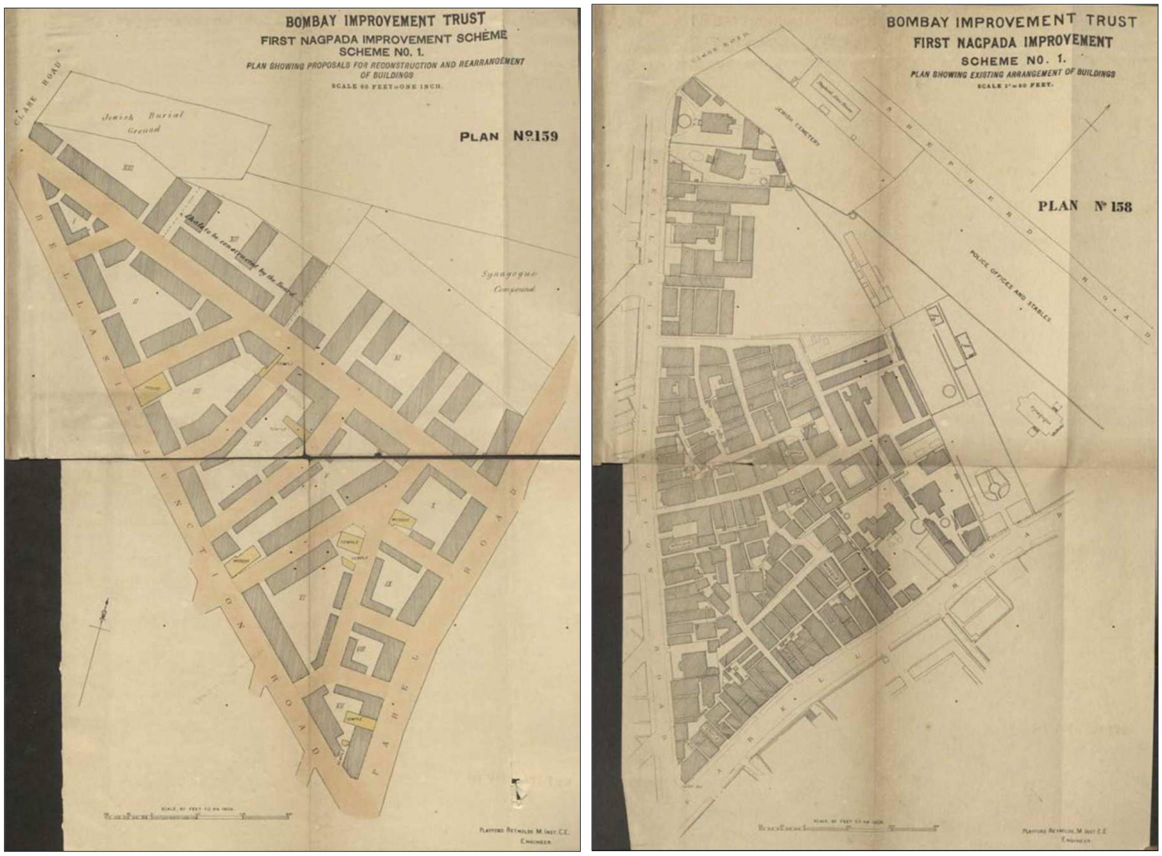
The first scheme cleared First Nagpada, a district described by Edwardes in the census as a combination of single and multi-story housing, 39 of which were classified as chawls. The neighborhood was known for its overcrowding, with most buildings subdivided into 10 to 80 tenements, with an average of 50 persons per house (with one notable instance in which a single house was let to "over 140 rent-payers conjointly").[67] The majority of these houses (94 percent, estimated Edwardes) were let to "those of the humblest class."[68] James Orr, the Chair of the Bombay Improvement Trust, characterized the neighborhood rather more dramatically, as filled with "properties crowded together without regard to ventilation and served only by narrow tortuous passages," a feature not mentioned by Edwardes, and filled with "miserable structures [constructed at] minimum cost."[69] He argued that the area "had the unenviable notoriety of being the unhealthiest are in Bombay," citing deaths per 1000 for the area between 1896-1901. In the year 1900-1901, he claimed, 1,373 out of 11,000 residents became sick with plague.[70]

The improvement scheme, begun in 1899, resulted in a total 1,240 buildings destroyed, and 2,600 people displaced.[71] The new scheme, the according to the improvement trust, restricted the density of the population to 500 persons per acre, restricted the height of chawls or houses to three stories, and "wherever possible the buildings have been arranged to form the sides of a quadrangle with an interior courtyard. In such cases openings are left to provide amply for

[67] S.M. Edwardes, "Census of Bombay, Town and Island. (Vol X): Part IV History," and "(Vol XI): Part V Report, 1901," (Bombay: Times of India Press, 1901), 124, IOR/V/15/69, AAS, BL.
[68] Edwardes, "Census of Bombay," 14.
[69] Orr, *The Bombay City Improvement Trust from 1898-1909*, 9.
[70] Orr, 9-10.
[71] Bombay Improvement Trust, "Administrative Report for the Year 1898-1899, 7.

thorough ventilation."[72] Nearly 1700 of the 11,000 residents in the neighborhood were relocated to a planned estate at Agripada, where residents continued to be plagued with high death rates (Table 15).

Figure 21. Bombay Improvement Trust Scheme for the First Nagpada Neighborhood, and the existing arrangement of buildings before the improvement scheme.



SOURCE: BIT, Administration Report 1899, 15-16.

Examination of data collected by the Bombay Plague Committee suggest that the choice of First Nagpada for the first "Improvement" project based on plague mortality was questionable, at best. Between 1896-1910, the average death rate per 1000 of the neighborhood was 6.32 per 1000, among the lowest rates of any neighborhood cited (see Chapter III, Table 3). However, the neighborhood was the site of a heavy concentration of labouring class housing, located near the

[72] Bombay Improvement Trust, Administration Report for the Year Ending 31st March 1899, Bombay Improvement Trust Administration Report 1898-1899 to 1904-05, pg.7, GP_00133648, Royal Asiatic Society, Mumbai, India.

mills and other points of profit in the city. The scheme included displacement of nearly all 10,577 of the neighborhood's residents, replacing worker's accommodating with chawls of only two stories high and replacing much of the workers housing with police accommodation.[73] The eventual scheme was estimated by the Trust to displace a at least 2600 persons.[74]

The other neighborhood-wide scheme (Scheme VIII) took place at Mandvi Koliwada, "where Plague was first detected and where it has persistently remained ever since."[75] The project targeted "the insanitary area in which plague was worst in its earliest days and in which the annual recrudescence of plague always occurred first".[76] While the 36,000 square yard area was first earmarked for destruction in 1898, by 1906 prolonged legal battles with residents meant that the project had only just acquired the majority of buildings. Plans entailed the demolition of all housing in the area, with plans for widened streets and "buildings constructed on sanitary principles" to "occupy the frontages." The scheme displaced residents of 52 buildings on 7,910 square yards of land, with plans to rehouse only a "portion" of them.[77]

Rehousing plans perpetuated some of the key structural issues of the neighborhood, namely, constructing housing which contained "shops and godowns on the ground floor and one-room tenements on the upper floors," demonstrating the Trust's general disregard for plague etiology, despite the clear understanding of the epizootic nature of the disease indicated in their investment in Common Sense Rat Exterminator during the same year.[78] Improvement schemes, while also

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[73] Bombay Improvement Trust, Administration Report for the Year Ending 31st March 1901, Bombay Improvement Trust Administration Report 1898-1899 to 1904-05, pg. 8, GP_00133648, Royal Asiatic Society, Mumbai, India.
[74] City of Bombay Improvement Trust, Administration Reports of the Bombay Improvement Trust 1898/99-1907/08, V/24/2791, pg. 7, Asian and African Studies Collection, British Library, London, UK.
[75] Dunn, 10.
[76] Orr, 11.
[77] Bombay Improvement Trust, "Administration Report for the Year Ending 31st March 1907," Bombay Improvement Trust Administration Report 1906-1907, pg. xii, GP_00133649A, Royal Asiatic Society, Mumbai, India.
[78] There is some amount of interest in the rat-flea theory, but marginal compared to other methods (gets only Rs. 7000 from a total expenditure of Rs. 3,509,177). The report states, "Being a possessor of a very large house property

removing “unsanitary” housing from one of the highest plague mortality areas in the city, focused on expanding a “main artery” through the city that terminated at the G.I.P Railway.[79] The project thus served the purpose of both demolishing a neighborhood that had become a symbol of plague in the city and opening up a clear pathway from the manufacturing districts (centrally located within the city) to the railway and shipping districts.

Other Trust projects dropped the pretense of providing sanitary housing for the city’s residents altogether, instead opting to demolish densely populated areas and build “handsome thoroughfares” through them in the style of English and Scottish Improvement schemes.[80] In these projects, the Trust demonstrated the flexibility of their etiology of disease, relying largely on miasmatic logic to justify improvement schemes despite earlier fidelity to the rat-flea theory. While clearly improvement-minded, the Trust still used the language of sanitarianism to justify beautification projects through allusions to the relationships between overcrowding and filth. The Sandhurst Road Street Scheme, for example, was designed to “clear out considerable portions of the terribly overbuilt and insanitary areas in Kumbarwada, Umarkhadi, Mahomedkhan Pakadi, Old Nagpada and Nawroji Hill” in order to “give clear passage to the air from Back Bay to the Elphinstone Bridge.”[81] Of the twenty-two proposed schemes in 1906, thirteen explicitly concerned widening throughways and major roads in the city that connected commercial centers

in the City, the Board were also interested in the campaign against rats in connection with measures for the extermination of plague in the City, and accordingly sanctioned a contribution of Rs. 7000 towards the extensive employment of Common Sense Rat Exterminator in the districts covered by their Schemes. It is not possible however to express satisfaction with the comparatively meagre results that have been obtained.” BIT, 1906-1907, v.

[79] BIT, 1898-1899, 6-7.

[80] Dunn, 19; BIT, 1901-1902, 15.

[81] Dunn, 11.

with areas of transport, resulting in the displacement of residents of the "overcrowded" and "insanitary" housing lining the existing streets.[82]

The Ecologies of Improvement: Displacement, Dispossession, and Slum Clearance Schemes

These plans, undertaken often in the name of mitigating high plague mortality, were widely criticized by other members of the municipal body (including the municipal commissioner) and common citizens for their disorganization and violence. Across improvement schemes, the Trust overwhelmingly engaged in forced removal of residents and homeowners from their properties by law, suggesting widespread resistance to the schemes. The Bombay Improvement Trust Act of 1898 empowered the board to acquire land for the Trust's schemes either "by agreement or by compulsory acquisition."[83] Records suggest the latter approach was overwhelmingly employed: In 1907, of the 1,188 properties acquired by the Bombay Improvement Trust, 769 were acquired through the courts while only 419 were acquired through "amicable settlement."[84] This divide widened in 1910, when only 46 cases were settled amicably and 813 were "fought out."[85]

Residents' reluctance to relinquish their properties was no doubt amplified by the lack of alternative housing. The Trust's failure to rehouse the estimated three to four hundred thousand people displaced by its improvement schemes remained a consistent point of controversy. In 1915, Executive Health Officer J.A. Turner noted that over ten years previously, "the City Improvement Trust…had already demolished a large number of houses, without any serious

[82] Bombay Improvement Trust, Administration Report for the Year Ending 31st March 1901, Bombay Improvement Trust Administration Report 1898-1899 to 1904-05, pg. 89, GP_00133648, Royal Asiatic Society, Mumbai, India.
[83] Kidambi, "'An infection of locality,'" 60.
[84] BIT, Admin Report 1906-1907, pg. iv.
[85] BIT, Admin Report 1909-1910, pg. 3.

attempt having been made till then to provide accommodation for those displaced."[86] Semi-permanent accommodations were only provided for a total of 14-15,000 people by 1909, "possibly less than 1/20th of what is really required."[87] The Trust instead looked to *laissez-faire* principles to solve the housing shortage created by its slum clearance projects. To house the over 14,000 residents displaced by slum clearance schemes they asserted, "'Cheap dwellings should be provided by all employers of labour, where possible, near the work of the labourer. Large employers of labour owe to the City and to their employees a certain responsibility for the housing and care of their work people and, until this responsibility is recognized and immediate steps are taken to provide it, the health of the City must suffer, and, with it, its trade, commerce and prosperity.'"[88] While some cotton mills did opt to build workers housing, Caroline Arnold notes, only 4,497 rooms in total were built, which thus had the capacity to accommodate only 8.5 percent of the 52,283-person work force.[89] As a result, tenants and homeowners removed from their properties often faced a scarcity of housing and an increased cost of rent. The Executive Health Officer noted that these displaced persons often moved into accommodations shared with others, thereby actually increasing the problem of overcrowding in the city – an ideal circumstance for the plague and its vectors.[90]

Conditions were hardly better for those who found temporary or permanent accommodation through the Trust. The series of huts constructed at Agripada estate and along the Kennedy Sea Face to accommodate persons displaced by the Improvement Trust were also heavily criticized, often dubbed "insanitary" and "inhumane", a feature Orr tries to dispute in his lecture on the

[86] *Annual Report of the Executive Health Officer for 1914, in Administration Report of the Municipal Commissioner for the City of Bombay for the Year 1914-1915, Vol II*, (Bombay: Times Press, 1915), 66.
[87] Orr, 14.
[88] Kidambi, "Housing the Poor in the Colonial City," 66.
[89] Caroline Arnold, "The Bombay Improvement Trust," 106.
[90] Kidambi, "Housing the Poor," 64.

progress of the Bombay Improvement Trust, arguing that "sufficiently rapid progress" could not be made "without the clearance of insanitary areas."[91] However, the camps continued to see a relatively high death rate – 23 per thousand – only surpassed by the death rate of those lucky enough to actually secure "sanitary" accommodation from the Trust (Table 3).[92]

Table 15. Population and Death Rate on the Bombay Improvement Trust's Schemes, 1908-1910.

Name of Chawl	Population in 1909-1910 (Average)	Death Rate per 1000	Population in 1908-1909 (Average)	Death Rate per 1000
Agripada	4427	22.58	4342	26.78
Nagpada	494	18.21	462	20.83
Chandanwadi	2224	31.47	2534	34.82
Imamwada	2370	32.06	965	36.65
Princess Street	251	43.82	-	-
Semi-permanent sheds and buildings Scheme III	992	19.15	1082	22.11
Semi-permanent sheds and buildings Paltan Road	1226	39.96	1063	63.48
Total	12147		10,175	

SOURCE: Bombay Improvement Trust, Admin Report for the Year 1909-1910, 8.

Studies by the Bombay Bacteriological Laboratory suggest that these improvement schemes continued to be hot spots for plague transmission – with one chawl constructed by the Trust abandoned in 1906 because of a severe outbreak of plague. Bannerman notes that "the buildings in themselves offer no shelter to rats," with brick walls, concrete floors, and Mangalore tile roofs, with hallways made of concrete and large passageways, and yet "In spite of this *Mus*

[91] Orr, 15.
[92] BIT, Admin Report for the Year 1909-1910, 8.

rattus is common in the houses."[93] These estates also seem to have constructed an ecological niche for another deadly disease in Bombay: malaria. In the Trust's report for 1909-1910, they detailed "the training of all inspectors and peons employed on Trust Estates to find out mosquito breeding places and destroy larvae," noting that "The Trust's Gamdevi Estate has suffered in the past from the evil reputation of the adjacent Gowalia Tank as a source of malaria."[94] Orr also attributes a decrease in the death rates present in Trust estates during the year 1909-1910, particularly at Palton Road, "due no doubt in part to the extermination of several mosquito breeding places from the compound."[95]

Contrasting this case with the intended and unintended consequences of disease mitigation in Melbourne, the importance of etiology, bacterial specificity, and public health structure in influencing ecological change is highly visible. While both forms of intervention overwhelmingly placed the burden of urban environmental change on the poor to varying degrees (with the paternalist structure of Bombay's government inflicting more direct and often more extreme violence on poor residents than Melbourne's more indirect regulatory system), the nature of disease and conceptualization of mitigation strategies resulted in the production of very different ecological niches. In the case of Melbourne, bacteriologically-based tuberculosis control techniques aimed at working class residents resulted in the preservation of eucalyptus forests near the city and the removal of cattle and other livestock from the urban environment, and ultimately fostered a decline in incidence of bovine tuberculosis through regulatory practice; in Bombay, plague control measures undertaken by the Improvement Trust (and, as noted in a previous chapter, by the Indian Medical Service before it) that relied heavily on miasmatic

[93] The Epidemiological Observations made by the Commission in Bombay City, 782.
[94] BIT, Administration Report for 1909-1910, 7-8.
[95] BIT, Administration Report for 1909-1910, 7-8.

theory built urban niches for *Plasmodium falciparum* (malaria) and its vector, and likely continued to encourage a niche for *Yersinia pestis*.

Etiologies of improvement

The forced removal of residents, combined with the speed with which the Trust acquired property before organizing appropriate alternate accommodation and a noted lack of improvement in plague mortality rates, lends credence to a common argument among historians of Bombay that land acquisition by the Trust was driven more by interest in driving speculation and investment from other parts of the Empire than by an interest in improving the health of the population.[96] Shabnum Tejani, for example, notes that much of the land in question sat near the mill district, and thus "had 'an inherent value for every possible purpose connected to the mills'," and thus was "'extremely desirable from an investor's point of view.'"[97] Most schemes did not result in the Trust purchasing the entirety of the land in a neighborhood, as occurred in Mandvi and Nagpada, but instead targeted so-called "overcrowded" areas in other neighborhoods as sites of new urban infrastructure – railroads, large streets connecting important commercial zones of the city – that would be highly attractive to investors.

Also suggestive of the Trust's economic motives was its continued reliance on outdated logics of disease to justify areas of focus for improvement projects. The isolation of the plague bacillus in 1894 left little doubt the disease's cause, though its etiology and ecology remained largely unknown. International consensus (visible in the convening of the 1897 International

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[96] Shabnum Tejani, "Disputing 'market value': the Bombay Improvement Trust and the reshaping of a speculative land market in early twentieth-century Bombay," *Urban History* (2020): 1-18. DOI: 10.1017/S0963926820000565; Sandip Hazareesingh, "Colonial Modernism and the Flawed Paradignsm of Urban Renewal: Uneven Development in Bombay, 1900-25," *Urban History* 28(2001): 235-255, Accessed January 7, 2021, http://www.jstor.org/stable/44613237; and Kidambi, "Housing the Poor," 61.
[97] Tejani, 9.

Sanitary Conference in Venice) firmly rested on contagion theory as the primary mode of transmission for plague, shifting to the rat-flea theory between 1903 (when this etiology was endorsed by the International Sanitary Conference in Paris) and 1906.[98] However, among the Trust, miasmatic and saprophytic logics of disease were often deployed as justification for improvement projects long after both theories had been largely disproven. For example, in 1905, the chair of the Bombay Improvement Trust noted that "In many quarters the houses are jammed so closely together that free circulation of air is prevented, the Sun can barely get lower down that the roofs and the atmosphere is thick with foul odors."[99] Even far beyond when the rat-flea theory of transmission was not only accepted but under heavy investigation by the Bombay Bacteriological Laboratory in 1909 (and after, it should be noted, the Trust itself attempted a small-scale experiment with Common Sense Rat Exterminator), the Chair of the Trust argues that the purpose of a scheme for road construction from one side of Bombay to the other "is to remedy the defective ventilation of the parts of the City traversed by it" and to reconstruct the areas "with a sufficiency of air space and on sanitary principles."[100]

Focus on issues of light, ventilation, and foul odor, while not in keeping with the major etiologies of plague dominant at the turn of the century, did serve an important purpose to the Trust: it provided a logical basis for the destruction of slums and interventions that echoed late nineteenth-century English and Scottish town planning models. While the initial growth of Bombay was largely informal, with an influx of laborers into the emerging cotton and shipping markets occupying unregulated and inexpensive accommodation near sites of employment, the Trust looked to English models for clearance of these areas and "beautification" projects within

[98] Harrison, *Contagion*, 184-196.
[99] Dunn, 5.
[100] Orr, 3.

the city. A direct quote from the Housing of the English Working Classes Act 1890 in the Trust's 1913 report suggest that the organization modeled its legislation and its compensation scheme for displaced residents directly off English and Scottish town planning.[101] Section 49 of the Bombay Town Planning Act, "under which the Board are empowered to acquire insanitary houses on specially low terms," was "itself a rescript of the English Acts relating to the provision of dwellings."[102]

This model, however, was quickly recognized as a mistake. The executive health officer noted in 1901, for example, that "the standard laid down in England for guidance in erecting dwellings for the working people was not whole applicable here." Meanwhile, one of the elected members of the Board of Trustees, Dinsha Edulji Wacha, delivered a blistering condemnation of the Town Planning Act, noting that "The measure is confiscatory in its character and calculated to destroy all just and fair rights and interests in private property while provoking the greatest discontent in the City and Presidency of Bombay."[103] The failures of the program are perhaps best summarized by historian Sandip Hazareesingh, who noted that in their attempt to restructure Bombay using the same methods employed in the United Kingdom – namely, relying on slum clearance as a primary mechanism of sanitary reform, "the new body passed over a unique opportunity to draw on lessons of decades of British urban 'improvement' schemes whose neglect of housing provision had generally resulted in the shifting, rather than the abolition, of overcrowding."[104] In failing to learn from midcentury slum clearance schemes in the United

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[101] Bombay Improvement Trust, "Administration Report for the Year Ending 31st March 1913," Bombay Improvement Trust Administration Report 1912-1913, GP_00133651, pg. 144, Royal Asiatic Society, Mumbai, India.
[102] BIT, Administration Report for 1898-1899, 15.
[103] BIT, Administration Report for 1909-1910, 103.
[104] Hazareesingh, 240.

Kingdom, the Bombay Improvement Trust not only angered the citizenry and municipal government of Bombay, but also worsened conditions of plague in the city.

Plague thus became an excuse to begin improvement projects that aligned strongly with Bombay's self-perception as an "imperial city," creating profitable investment projects for circulating British capital. While the Trust used the plague epidemic as justification for action, these projects were by no means focused on eradicating plague from the city or on the health of its Indian residents – a feature visible in the violence of displacement and marginalization, the use of miasmatic theory to justify projects, and the likely worsening of conditions for plague by ecological disruption. Unlike Melbourne, where the bacteriological and ecological characterization of tuberculosis contributed to the consolidation of public health authority at the municipal level, the Improvement Trust's reliance on miasmatic theory and general ignorance of bacteriological authority fostered wide criticism. It is perhaps no surprise, then, that after several independent investigations and numerous polarizing complaints, the Bombay Improvement Trust was replaced by the Bombay Development Directorate in 1920 after "failing to improve the housing and sanitary conditions" of the city – and indeed, for failing to curb the continued spread of plague.[105]

The Changing Ecological Niches of Cockles and **Salmonella Enterica Typhi.**

Just as the Belfast Public Health Department drew plans to prepare for local outbreaks of pandemic plague, the Executive Sanitary Officer developed a parallel plan to address an epidemic already raging within the city. The typhoid epidemic was brought to the attention of the Public Health Committee by 1897 following the report of James Lorrain Smith, and

[105] Tejani, 13.

bacteriological examination incorporated into the surveillance of the disease by 1898.[106] While contact tracing, bacteriological testing, and outbreak investigation formed the bulk of the Public Health Committee's information-gathering methods – very much in keeping with and modeled on similar methods employed across England and Wales since the 1870s, as Jacob Steere-Williams notes – their chosen form of public-facing intervention differed from those employed in Melbourne and Bombay. While in the case of plague and bovine tuberculosis, public health departments sought to regulate hygiene and sanitary conditions of organisms deemed "pathological," – both human and nonhuman – in Belfast, the relationship of bacteria and host was perceived quite differently. Unlike cattle, rats, and infected humans in Bombay and Melbourne, shellfish in Belfast were often regarded as "victims" of poor urban planning, and their connection to typhoid related to the leisure activities of the population. The source of infection lay in informal shellfish collection, facilitated by residents who "trawled the shores of Belfast Lough for cockles on spring and summer afternoons and evenings," and street traders who "hawked in the working-class districts in the evenings, selling to residents and the pubs."[107] In Belfast, there was, Dr. Bullstrode noted, an "entrenched history of shellfish-borne typhoid."[108] The source of infection was built into the sanitary, social, and ecological landscape of the city.

Thus, when the Belfast Health Commission concluded that it was the very sanitary updates recently undertaken in the city combined with local practices in shellfish consumption that fueled

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[106] Belfast Corporation General Sewerage Committee Minute Book, 28th January 1868 to 24th November 1874, Local Authority Records, LA/7/9AC/1, pg. 243, Public Records Office Northern Ireland, Belfast, Northern Ireland, United Kingdom.

[107] Anne Hardy, "Exorcising Molly Malone: Typhoid and Shellfish Consumption in Urban Britain 1860-1960," *History Workshop Journal* 55(2003): 78-79, accessed August 21, 2017, http://www.jstor.org/stable/4289828.

[108] H. Timbrell Bulstrode, "Supplement in Continuation of the Report of the Medical Officer of the Board for 1909-10, containing a Report on Shellfish Other Than Oysters in Relation to Disease, by H. Timbrell Bulstrode, MD, with an introduction by the medical officer of the board; a report on the conditions under which certain Shellfish other than Oysters are grown, collected, cleansed and stored, and the relation of such treatment to the prevalence of Enteric Fever and other Illness," Thirty Ninth Annual Report of the Local Government Board, 1909-910, Local Authority Records, LA7/9AA7, pg. 727, PRONI, Belfast, United Kingdom.

the typhoid epidemic, health officers calibrated intervention accordingly. As such, public health practice relied on risk communication methods and burgeoning sanitary authority to curb the epidemic – resulting in behavioral modification rather than structural modification. By the turn of the century, "notices, cautioning the public against eating shellfish gathered on the shores of the Lough, had been printed and published," an approach which resulted in "The marked decline" of typhoid fever incidence in the city.[109]

"The Mollusk was only the unwilling vehicle of infection": the Cockle as victim.

As discussed in Chapter V, the first attempts to isolate the cause of typhoid in Belfast relied on the epidemiological methods developed in the greater United Kingdom, including investigation, bacteriological analysis, and regulation of milk and meat industries. However, it soon became clear that the nature of the epidemic in Belfast was something entirely different. In examination after examination of local milk and water samples, Dr. Symmers, a bacteriologist at Queen's university Belfast, reported the absence of the bacilli, with only very rare exceptions.[110] Similar conclusions were reached by medical officers of health undertaking inspection of drinking water reservoirs around the city; The Public Health Committee concluded in 1901 that water filtration methods "removes some 95 per cent of the germs present in the unfiltered water," noting that Professor Frankland, "who is one of the highest living authorities on such matters, examines the Belfast water periodically, and has advised the Commissioners that their system of storage and filtration afford very real protection to the water consumers."[111] Local regulations similarly kept cattle in a somewhat sanitary condition – or at least, under government

[109] Bulstrode, "Supplement," 727.
[110] Special Meeting of the Belfast Public Health Committee, 15th October 1906, Local Authority Records, LA7/9AA7, pg. 636-654, PRONI.
[111] The Water Supply & Typhoid, Meeting of the Public Health Committee, 8th August 1901, Local Authority Records, LA7/9AA7, pg. 580, PRONI.

observation. In the Inquiry into the Health of Belfast conducted in 1907, the board questioned Dr. Bailie (then the Chief Medical Officer of Health) about the salubrity of keeping cows within the city limits, to which Dr. Bailie responded, "Considering the state of the law, I think if they were outside it would be worse," referencing the strict regulation of cattle industries within the city and the lax regulations without.[112] Unlike Melbourne, the biopolitical transformation of cattle had already occurred in Belfast by the time of the typhoid epidemic – allowing Dr. Bailie to conclude that cattle were not the source of the city's epidemic.[113]

Having exonerated the cattle industry and water supply, sanitary officers turned to the shellfish hypothesis as the next possible source of the epidemic. The nature of the shellfish-typhoid hypothesis was unsettled in British medical circles at the turn of the twentieth century – medical societies largely ignored Charles Cameron's convictions in 1880, only admitting that the connection had been drawn persuasively by Arthur Newsholme in 1893.[114] The slow development of medical consensus on the relationship between typhoid and shellfish meant Belfast was among the first major urban areas in the United Kingdom to assess the mechanisms of the infection pathway. The Belfast Health Commission and Royal Sanitary Committee, in solidifying this connection within the city, thus brought the cockle under the eye of the city public health apparatus.

The informal nature of shellfish collection confounded attempts to measure the extent of infection among shellfish and the causal pathway of infection in humans. Given the proliferation of shellfish along the shoreline and the deregulated nature of their collection and distribution, public health officials were unable to anticipate outbreaks. At the occurrence of an outbreak,

[112] Belfast Health Commission Report and Minutes of Evidence, 1907, pg. 102, WAT/1/3H/2/4, Records of the Belfast Water Authority, PRONI.
[113] Belfast Health Commission Report and Minutes of Evidence, 1907, pg. 194, WAT/1/3H/2/4, Records of the Belfast Water Authority, PRONI.
[114] Hardy, "Exorcising Molly Malone," 83.

sanitary officers conducted interviews of affected families; if the family admitted to a history of gathering cockles in preceding weeks or to purchasing cockles from street hawkers, sanitary officers were dispatched to collect cockles from the area or vendor for bacteriological examination. By the time cockles underwent bacteriological examination, however, the offending bacteria may have filtered out, and the sample of shellfish no longer represented true extent of contamination. In their 1904 report on the shellfish-layings of Ireland, Dr. Browne and Dr. McWeeney highlight the ability of contaminated shellfish to evade even the most novel public health interventions, noting,

> As the law at present stands, there does not appear to be any adequate means of protecting the public against the sale of polluted shell-fish. Unlike other forms of food which, on account of their external appearances may be seized and condemned by the Sanitary Inspector, shell-fish, unless they are decomposed, cannot be dealt with, as there are no obvious external signs by which it can be determined whether they are capable of producing disease or not, and although the Sanitary Inspector may know that they have come from polluted layings, he has no evidence upon which he would be justified in seizing a particular consignment, nor, if he did so, any ready means of proving that the shell-fish are unfit for human food. In this sense, even bacteriological testing is relative, not an absolute, criterion, of danger, in the absence of isolation of specific human-disease-producing microbes.[115]

The minutes of the Royal Sanitary Commission on Enteric Fever in Belfast further demonstrated the difficulties of surveillance and control. In numerous interviews with representatives of the public, the medical profession, and the university, the Commission failed to find consistent answers on the extent or nature of shellfish consumption in the city, only that, "There is no doubt it is very general. It is quite a common thing to go down on Saturday afternoons and Sundays to the foreshore and bring home handkerchiefs full."[116] The testimony of Henry Reynolds, a sanitary sub-officer for the city, also demonstrates the importance of

[115] TJ Browne, *Report on the Shell-Fish Layings on the Irish Coast, As Respects their Liability to Sewage Contamination*, (Dublin: Alexander Thom & Co., 1904), pg. vii, I.361/GREA, Irish Closed Access, Linen Hall Library.
[116] Belfast Health Commission Report and Minutes of Evidence, pg. 1641, WAT/1/3H/2/4, PRONI.

unlicensed hawkers in perpetuating typhoid, noting that, "In February 1906 I investigated 15 cases of typhoid fever in the northern part of the city, and out of the 15 cases 7 told me they had been eating cockles…I asked them where they got the cockles, and they said they had got them from hawkers in the street."[117]

While focus largely remained on the behaviors and practices of the city's residents, reports also assigned shellfish an agentive role in the spread of typhoid fever – perhaps more so in Belfast than in any other city in the United Kingdom, as Dr. Mair, the major proponent of the shellfish theory of typhoid in Belfast, noted.[118] In the conclusion of the Royal Sanitary Commission's inquiry into the typhoid rate in Belfast, Mair highlights the agency of the cockle, noting that "the inference to be drawn from the facts appears to be that if this shellfish agency had been absent, the history of fever in Belfast would have been vastly different from what it has been."[119] "It may be said," he continued, "that the fact that the exceptional increase of fever referred to followed so closely upon the increased opportunities of gross contamination of the principal sources of shellfish by vast volumes of sewage, together with the fact that there is an absence of other adequate explanation of this increase of fever, affords in itself presumption that Lough shellfish did take an important part in the maintenance of fever in Belfast."[120] In drawing out the agency of shellfish in typhoid fever, Mair also assigns agency to the environment and to the microbe responsible, noting, "Experience has shown that the manifestations of this disease vary considerably according to the different agencies which have been mainly responsible for its spread, and that much may be gleaned for what may be called the behaviour of the disease, as illustrated by the manner of its incidence, as well as from its distribution, in elucidating the

[117] Belfast Health Commission Report, 1641.
[118] Mair, Addendum to the Report of the Belfast Health Commission, pg. 103, WAT/1/3H/2/4, PRONI.
[119] Mair, Addendum to the Report of the Belfast Health Commission, pg. 137, WAT/1/3H/2/4, PRONI.
[120] Mair, Addendum to the report, 135.

problem of its causation."[121] Shellfish, similarly to rats and cattle in other parts of the Empire, also came under as much observation as possible given their position outside the formal economic or urban structures of the city, with attempts to document their scope and ecological characteristics emerging in public health literature. By 1906, before shellfish were conclusively assigned a role in the typhoid epidemic, Dr. King-Kerr highlighted, "we began this systematic examination of cockles all along the foreshore, and then we mapped out these danger areas."[122] Dr. Browne, as discussed in a previous chapter, also made an extensive study of the shellfish beds of Ireland and consumption patterns surrounding them as early as 1903.[123]

While in the case of Bombay and Melbourne the inclusion of agentive language around non-human vectors resulted in tightening biopolitical control around the organism in question, facilitated by public health legislation aimed at preventing zoonotic disease, the pathological relationship of shellfish to typhoid further complicated its relationship to the public health apparatus. Unlike cattle or rats, shellfish were not vectors who themselves produce disease, but instead carriers of a pathogen excreted by humans. Villainization, so common in the case of the rat and even among cattle, was virtually absent in the context of shellfish. As incidental carriers of disease, harboring typhoid because of their practices of filter-feeding, shellfish were instead treated as victims in the follies of people, that "the mollusk was only the unwilling vehicle of infection."[124] In Belfast, contaminated cockles were instead seen as a symptom of pollution caused by recent ambitious sanitary projects (the other being the far more sensorially invasive *ulva latissimi,* a smelly seaweed known to thrive near sewage outlets).[125] The emergence of the typhoid epidemic and estuarial pollution in the wake of the completion of the main drainage

[121] Mair, Addendum to the report, 107.
[122] Belfast Health Commission Report and Minutes of Evidence, pg. 1331, WAT/1/3H/2/4, PRONI.
[123] Browne, *Report on the Shellfish Layings of Ireland*, 1-62.
[124] Hardy, "Exorcising Molly Malone," 49.
[125] Belfast Health Commission Report and Minutes of Evidence, pg. 1556-1557, WAT/1/3H/2/4, PRONI

scheme was thus, as Professor Letts noted in his cross-examination by the sanitary committee, a case of "the engineer being hoist with his own petard."[126]

Communicating Risk: Behavioral Intervention and Regulation

While the shellfish hypothesis for typhoid transmission was not fully accepted among Belfast's bacteriological and sanitary authorities until 1906 – when, according to the Belfast Corporation, "Having exonerated the water supply and the sanitary condition of the City, the Commissioners have been obliged to look for some other cause of the alleged high death-rate, and have adopted 'the shellfish hypothesis as the only one available'"[127] – methods for mitigation of the threat began much earlier and focused almost exclusively on voluntary behavioral regulation. Dr. Herbert Timbrell Bullstrode noted in the 1909-1910 report of the Local Government Board, that notices began to be posted along the shorelines as early as 1902, attributing the beginning of this public health awareness campaign to a marked decline in typhoid fever rates.[128] The posting of notices along the shoreline warning people off consumption of contaminated shellfish, with mentions of continued printing and posting of notices from the Public Health Committee of the Local Government Board in 1905, 1906, and in the Royal Sanitary Commission Reports suggesting that this intervention was both consistent and widespread.[129]

[126] Belfast Health Commission Report and Minutes of Evidence, pg. 1587, WAT/1/3H/2/4, PRONI

[127] Report of the Joint Public Health, Improvement, Works, and Market Committees of the Belfast Corporation upon the Report of the Belfast Health Commission, 1907-1908, pg. 11, LA7/9DB/1, Local Authority Records, Public Records Office Northern Ireland, Belfast, UK.

[128] H.T. Bullstrode and the Local Government Board, *Supplement in Continuation of the Report of the Medical Officer of the Board for 1909-1910, Containing a Report on Shellfish Other than Oysters in Relation to Disease*, (London: Darling and Son Ltd., 1911), 727.

[129] Minutes of the Public Health Committee of Belfast, "Meeting of the Public Health Committee, 28th June 1906 – "Shellfish and Typhoid Fever"", pg. 589, LA7/9AA7, PRONI; Minutes of the Public Health Committee of Belfast, "25th May 1905: Shell fish and Typhoid Fever," pg. 294 LA7/9AA7, PRONI; Belfast Health Commission Report and Minutes of Evidence, pg. 304, WAT/1/3H/2/4, PRONI.

Interviews with members of the public carried out over the course of the Royal Sanitary Commission meetings emphasize the effectiveness of public health communication as a strategy for mitigation of the typhoid epidemic. Henry McCauley, a self-described "gatherer of shellfish" interviewed by the sanitary commission noted, when asked about cockle consumption, that "There are less sold now, because the caution given out by the Corporation some time ago has taken effect, and people do not go for them; they are frightened now to eat them, you know…It has had the effect of stopping the thing nearly altogether."[130] John Spence, a shipyard worker, similarly observed that "the Corporation have taken every steps to prevent [the consumption of raw cockles] by posters calling attention to the matter," though he presented greater ambivalence about their extreme success, noting that "I suppose when people are hungry they are not particular about what they eat."[131] A remarkable aspect of the ubiquity of notices was not just that citizens comment on them – but that they appeared to be an effective public health intervention. Bullstrode noted a decline in typhoid rates starting in 1903, and "the acceleration of such decline since 1905, when more active steps in the prevention of shellfish eating were undertaken… A widespread distrust of shellfish has been created, and this distrust has led to a very great reduction in their consumption."[132] By 1909, the rate of typhoid fever in Belfast had fallen to or below that of the majority of large English towns.

[130] Belfast Health Commission Report and Minutes of Evidence, pg. 1785, WAT/1/3H/2/4, PRONI.
[131] Belfast Health Commission Report and Minutes of Evidence, pg. 276, WAT/1/3H/2/4, PRONI.
[132] Bullstrode, 658.

Table 16. Death Rate per Five Year Interval, Belfast 1891-1910.

Year	Belfast Death Rate (per 1000)	Liverpool Death Rate (per 1000)
1891-1895	0.51	0.29
1896-1900	0.99	0.29
1901-1905	0.49	0.21
1906-1910	0.14	0.21

SOURCE: H.W. Bailie, Report on the Health of the County Borough of Belfast, for the Year 1910, pg. 42, LA7/9DA/20, Local Authority Records, PRONI; and Mair, Addendum to the Report of the Belfast Health Commission, 103.

While attempts at legal regulation followed the erection of notices, their effect was mixed at best. The Public Health (Regulations as to Food) Act, 1907, which allowed health officials to seize shellfish intended for sale for human consumption, was often employed against street hawkers with mixed results. Sanitary sub-officer Henry Reynolds, for example, notes in his cross-examination that since January 1906 the sanitary officers had made a practice of seizing shellfish. He claimed that he "got orders from the Medical Officer owing to the number of cases of typhoid, and the patients having all stated that a fortnight or so previous they had eaten these shellfish, if I came across any shellfish being retailed which were polluted with sewage to seize and destroy them, which I did." Following similar leads, he claims to have made at least fourteen seizures since January 1907 "of shellfish being retailed by hucksters.[133] However, the seizures did not result in any prosecution of the hawkers, and Reynolds noted, "I have never been able to make a seizure of the same person twice," which suggests that hawkers became particularly

[133] Belfast Health Commission Report and Minutes of Evidence, pg. 1329, WAT/1/3H/2/4, PRONI.

adept at avoiding detection by the sanitary sub officers.[134] Reynolds did observe a decrease in sales in the city – though of course, this could be a sign of the movement of the trade away from his watchful eyes.

Cultivating Effective Public Health Response

The story of typhoid in Belfast stands in stark contrast with that of plague in Bombay or tuberculosis in Melbourne. The source of the epidemic was traced; the nature of the vector and its challenges acknowledged; and a synchronized and continuous response implemented that affected lasting change to the typhoid rates of the city. So, what was unique about Belfast? Arguably, the three cases in question have more differences than similarities; as discussed at length in other chapters, each city contained its own unique governance structure, relationship to the British metropole, and hierarchies of class and race that shaped their ecologies and the structure of their public health systems. Focusing on the intersection of environment, public health infrastructure, and the nature of the epidemics of these three cities, however, provides some insight. First, unlike Melbourne, Belfast possessed a centralized board of health with thorough coverage of the city's many industries and neighborhoods. The appointment of numerous sanitary sub officers to monitor the health conditions of the city was well in place by the time of the epidemic. Unlike Bombay, a city which also possessed a centralized health authority, sanitary officers were appointed with consideration of the nature of power dynamics and cultural specificity of the city. Female sanitary sub officers were often appointed to positions like factory and workshop inspection (where many of the workers themselves would have been female) and infectious disease inspection, especially as the latter often required entering homes

[134] Belfast Health Commission Report and Minutes of Evidence, pg. 1329, WAT/1/3H/2/4, PRONI.

and instruction of mothers as to the feeding of infants; in fact, as the Public Health Committee boasted, "No similar city in the kingdom has so large a staff of Female Sanitary Officers."[135] The structure of the Central Board of Health thus built trust between the community and authority, paving the way for an amicable reception of public health intervention. Though of course, it should be noted, that amicability had its limits, with small sections of the population continuing to collect and hawk shellfish for several years beyond the erection of notices, and defying attempts at the enforcement of legislation.

Another important distinction between Belfast, Melbourne, and Bombay was the nature of the bacteria and vector themselves, and their position in the emerging culture of bacteriology. *Yersinia pestis* and the two *Rattus* subspecies served an important role in the development of vector pathology in India, and attempts at observation and nominally, control, provided justification for the removal of poorer classes from the center of the city, and therefore advancement of the agenda of the Bombay Improvement Trust. In Melbourne, the isolation of *Mycobacterium bovis* provided justification for the sequestration of cattle and their associated noxious industries to the outskirts of the city, supporting a decades-long fight against these industries by middle-and-upper class residents of the city. In Belfast, the position of shellfish as filter-feeders contaminated by human activity removed some of their culpability from infection, instead focusing attention on the shortcomings of sanitary improvement and sewage removal in the city. If anything, public interests were against the removal or extermination of shellfish (were it even possible, which seems ecologically unlikely), because of the city's role as an exporter of

[135] Report of the Joint Public Health, Improvement, Works, and Market committees of the Belfast Corporation upon the Report of the Belfast Health Commission, 1907-1908, pg. 15, LA7/9DB/1, Local Authority Records, PRONI.

periwinkles, molluscs, and other shellfish to the English mainland.[136] In short, the nature of the disease, and the nature of its interaction with public interests, played an important role in public health response, and in the changing urban ecology that resulted from this singularity.

Conclusion

As these three cases demonstrate, in the context of the British imperial state, disease etiology proved much more than just a backdrop to public health intervention. In Bombay, Belfast, and Melbourne, the interaction of disease – whether it be *Yersinia pestis*, *Mycobacterium bovis*, or *Salmonella enterica* – its vector or carrier, and the public health apparatus differed greatly, dependent on both local and imperial governmental forms, and importantly, on the human and non-human life present in the city. The specific interventions undertaken by governments to mitigate each of these epidemics were often dictated by the complex interaction of government and citizen interests, disease ecology and etiology, and public health infrastructure; in short, municipal action towards an epidemic often depended on whether the ecology of eradication aligned with powerful interests. In Melbourne, bovine tuberculosis took on a greater role in the public health imaginary than human tuberculosis because recommendations for its mitigation aligned with the interest of upper-and-middle class citizens for the marginalization of noxious trade industries (and the working-class citizens employed in them) to the outskirts of the city. In Bombay, the fragmentation of imperial public health and municipal governance meant that municipal interests rarely relied on public health recommendations for so-called "sanitary improvements," and instead upheld the specter of plague to justify urban improvement and slum clearance schemes in ways that contradicted or ignored recommendations of the Bombay

[136] Belfast Harbour Statistical Information for 24 December 1890 to 1 October 1898, pg. 30, HAR1/H/4/1, Belfast Harbour Commissioners, PRONI.

Bacteriological Laboratory, but furthered their own interests in beautification. While in Belfast, the ecology of typhoid and the integrated nature of public health and municipal interests promoted focus on behavioral rather than infrastructural change, with a centralized board of health undertaking massive communication campaigns to encourage residents to curb consumption of raw cockles. While examination of the long-term effects of these epidemics suggests that responses to all three resulted in lasting effects to urban life, the assemblage of governance forms, human and nonhuman actors, and urban structure dictated the trajectory of these changes. The unique niche constructed by each urban ecosystem, and the diseases that emerged within those niches, resulted in unique forms of sanitary improvement and structural change, the effects of which are apparent on the landscape of each city to this day.

Conclusion: Lessons from Epidemic History in the Age of COVID-19

When I first began this dissertation, pandemic disease had become nearly unimaginable in global political and social spheres. Soothed by technological optimism, narratives of scientific and medical progress, and the long interval between the last pandemic (at least, the last pandemic that affected non-marginalized groups) and the present, the US government dismantled its pandemic response team and froze hiring within the CDC and the HHS; the UK found itself embroiled in debates over the validity of the NHS; and many national medical systems contented themselves with operating on for-profit models that provided razor-thin margins. Pandemics became a thing of horror stories, interwoven into tales of mythical or realist apocalypse, only brought out for consideration on a Friday night with a bowl of popcorn, or talked about with a flashlight under the chin around the campfire.

This division was not representative of a reality, but of an imagined space resulting from the sequestration of activities related to epidemic monitoring and global biosecurity preparedness. As the eerily prescient documentary series *Pandemic* illustrated, hidden networks of epidemiologists, doctors, animal scientists, and others monitor contact points between nonhuman animals and humans that could lead to potential spillovers – all with the understanding that not every interaction could be monitored, and that the next event was not a question of if, but when.[1]

The species crossover event that spurred the development of COVID-19 occurred because of cultural and ecological forces specific to a time and place. And yet, we can speak of

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[1] Syra Madad and Dennis Carroll, directors, *Pandemic: How to Prevent an Outbreak*. *Netflix Official Site*, 22 Jan. 2020, www.netflix.com/title/81026143.

the next pandemic with an air of certainty, because the multi-scalar systems around which 21st century life (and twenty-first century global development) are structured encourage a set of human-environmental relationships that make spillover events – and their amplification – more likely. It is precisely these systems and their consequences that have come under scrutiny during the COVID-19 pandemic: global and local structures of inequality that alter survival chances and change the landscape of risk for people within and between countries; accelerating patterns of land use change – driven by agriculture, urbanization, and increasingly land-intensive lifestyles – that bring people and wild animals into closer and more frequent contact; and fragmented and inefficient governance systems that consistently fail to establish comprehensive health management and preparedness protocols for their populations. The fundamental failures of COVID-19 response indicate that these issues will define the next pandemic, as well. As this dissertation has argued, they have already defined the past – a reality which only becomes apparent when we take an ecological view of the history of disease and imperialism.

The eerie similarities between the three cases presented in this dissertation and the current pandemic crisis are no coincidence. The systems of power, economic exchange, and sociocultural organization that connect the three epidemics explored here are the predecessors of the multi-scalar systems that continue to shape disease ecologies.[2] Through 19th century imperialism and capitalist expansion, human-environmental relationships were solidified which, while heterogeneously experienced, formed ecologies uniquely suited to the emergence and perpetuation of epidemic diseases. By examining each of these epidemic diseases in both their

[2] Mark Harrison, *Contagion: How Commerce Has Spread Disease*, (New Haven: Yale University Press, 2012), 277-282; Jim Belich, Replenishing the Earth, The Settler Revolution and the Rise of the Anglo-World, 1783-1939, (Oxford: Oxford University Press, 2009), 551-559; Chris Otter, *Diet for a Large Planet: Industrial Britain, Food Systems, and World Ecology*, (Chicago: University of Chicago Press, 2020), 272; Robert Home, Of Planting and Planning: The Making of British Colonial Cities, (London: chapman & Hall, 1997), 219-220; Animal Cities: Beastly Urban Histories, Peter Atkins, ed., (London: Routledge Press, 2012); Randall M. Packard, *A History of Global Health: Interventions into the Lives of Other Peoples*, (Baltimore: Johns Hopkins University Press, 2016).

local context and as a function of their position within an imperial infrastructure, a set of key observations emerge that have significant implications for the study of history and for our approach to global pandemics.

1. ***The confluence of local and global environmental pressures often drives where and when diseases emerge.***

The three case studies examined in this project relate three distinct epidemic circumstances in three vastly different ecologies, subsumed under three very different forms of British colonial management. While these differences are enough to confound comparison, all three port cities were subject to a series of economic, structural, and managerial pressures inherent to their integration into British imperialism that drove changes to their local ecologies. Through explosive urban expansion fostered by financial speculation, economic specialization in material industries (wool and meat in Australia; shipping in Belfast; and cotton and grain in Bombay); integration into the British information regime, which encouraged urban and sanitary development around similar principles and designs; and stratified systems of inequality perpetuated by British administrative systems and industrialized societies, these cities faced a series of interrelated pressures to their unique ecologies.

While these cultural and physical pressures to the environment within the British Empire made the emergence of epidemic disease in each locality more likely, the differences between these cities defined their epidemic trajectory. In Bombay, the composition of the local rat population, the segregation of neighborhoods and homes based on class and religion, the economic specialization of the city in the grain trade, and British managerial response to the epidemic combined to foster a niche for *Yersinia pestis*. Melbourne's grid pattern and boomtown infrastructure promoted the expression and spread of both *Mycobacterium tuberculosis* and *Mycobacterium bovis* in its citizens in part because of its position in landscape ecology and its lack of regulation, which drove uneven sanitary development, and cultures of animal-keeping; while in Belfast, *Salmonella enterica* serovar *Typhi* exacted unusually high mortality because of the city's position on the Bay and the relationship of its abundant working-class population to the shellfish therein. Heterogeneous experience of disease mortality exhibited in all three of the cases shows that multiple overlapping ecologies can form a niche for a disease, but that there is much to be gained by thinking about those ecologies in both local and global context.

The intersection of local and global selection pressures for the emergence of disease is also visible in the COVID-19 pandemic. While the pathways of transmission and origin of the virus remain inconclusive in the wake of the World Health Organization 2021 report, the most likely scenario for transmission is thought to be introduction of the virus from a reservoir species to a host species, followed by zoonotic amplification and spillover to a human host.[3] The report concludes that the most likely scenario is therefore contact between a wild animal host (most likely bats or pangolins) and a mustelid (ie, mink) or felid (ie, cats) "farmed in sufficient densities to allow potential for enzootic circulation," noting that "high-density farming…includes many livestock species as well as farmed species" worldwide.[4]

Recent patterns of rapid urbanization and land use development for agriculture in China in the last several decades that facilitated this kind of virus transmission chain are to some extent local – a function of endemic bat populations, demand for wild animals and livestock as meat

[3] World Health Organization, *WHO-convened Global Study of Origins of SARS-CoV-2: China Part, 1*, Joint WHO-China Study, Geneva: World Health Organization, 2021, https://www.who.int/publications/i/item/who-convened-global-study-of-origins-of-sars-cov-2-china-part. The lab-origin theory of COVID-19 has received some attention lately, despite the WHO report's conclusion that such a pathway is "extremely unlikely," and numerous reputable scientific publications demonstrating the homology of the COVID-19's genetic sequence to other coronaviruses that have emerged through zoonotic transmission. However, we might think of the prominence of this argument in popular culture as a sign of cultural inclinations towards discrete, simplified explanations in place of dynamic, complex, and difficult to convey explanations for disease emergence. In short, undue attention given to the lab-origin theory demonstrates the necessity for historical interpretations of disease that show ecological complexity now more than ever.

[4] World Health Organization, 115.

consumption and standards of living rise, and practices in large-scale agriculture – but equally related to cross-cultural practices of factory farming, economic expansion, and patterns of land use facilitated by globalization. To understand the emergence of COVID-19, therefore, it is necessary to examine both the local ecological and social context and the globalized supply chains and financial institutions that foster opportunities for animal reservoirs for diseases with zoonotic potential to move closer to points of human contact.[5] Histories that apply a similarly multi-scalar approach to past epidemics have the opportunity to open imaginative space for consideration of these complex and overlapping ecological pressures.

2. ***Who is affected by disease is driven by the intersection of cultural, economic, and environmental dynamics.***

The emergence of an epidemic in a particular location and time is due to the confluence of multiple overlapping ecological pressures ranging from local to global. However, examination of neighborhood and demographic characteristics at the "local" level demonstrate that ecologies within these smaller scales are equally heterogeneous. As historian Gregg Mitman argues, structural violence perpetuated by systems of inequality have direct environmental consequences for marginalized groups and can produce vastly different disease landscapes for people living just a few miles from each other. "Ecologies of injustice," as Mitman terms these heterogeneous spaces, can be observed across cultural contexts, and in the case of this project, across epidemic environments.

In all three cities examined in this project, marginalized communities encountered significantly different risks of epidemic transmission fostered by their relationship to the built and natural environment. In Bombay, lower class residents of the city were more likely to

[5] Aaron Jenkins, Stacy D Jupiter, Anthony Capon, Pierre Horwitz, and Joel Negin, "Nested ecology and emergence in pandemics," The Lancet Planetary health, 4(2020): e302-e303. DOI: https://doi.org/10.1016/S2542-5196(20)30165-0.

encounter rats harboring plague infection through occupation, housing structure, and sanitary infrastructure (or lack thereof); in Belfast, cultural practices of shellfish gathering prevalent among the working class facilitated typhoid transmission, while location of working-class housing along floodplains facilitated sewer dysfunction and therefore increased opportunities for fecal-oral contact; while in Melbourne, lack of sanitary infrastructure, occupational dynamics, and patterns of livestock keeping among the working classes amplified the effects of environmental mismanagement and facilitated tuberculosis transmission and expression. These social, political, and economic determinants of health, as public health literature often labels them, are rooted in real environmental differences on the micro-scale.

Disparities in morbidity and mortality risk among marginalized communities and wage workers during the COVID-19 pandemic have rendered these textured local transmission dynamics extremely visible in the present. Early outbreaks among Amazon warehouse workers and rural meatpacking industry employees revealed the way that supply-demand chains shaped occupational risks for disease. As community transmission rates climbed, workers classed as "essential", often performing wage work for international corporations, placed themselves at increased risk for disease so that the privileged could remain indoors. Comorbidities that increased likelihood of severe disease were unevenly distributed, with heavy burdens on black and poor communities, denoting several environmental features – increased indoor and outdoor air pollution leading to asthma; food deserts that promoted obesity, diabetes, and heart disease; and a lack of infrastructure, both physical and metaphorical, that supports physical and emotional health.

The risk of COVID among these communities is also longer-lived. A striking map that shows vaccine availability and density of COVID-19 cases in the city of Chicago resembles a checkerboard, with wealthy communities with lowest incident rates receiving vaccine shipments first.[6] Meanwhile, cultures of vaccine skepticism and entrenched anti-science political ideologies worldwide constructed environments in which the virus could thrive – though often those espousing these ideologies and those suffering occupy very disparate ecologies. Thinking through the niche of the microbe itself and the multiple ecologies constructed by human cultural processes provides context for these disparities in health beyond statistical observation and makes clear the long history of these relationships.

3. *Government response is a complex feedback mechanism that can either perpetuate or mitigate an epidemic disease – and success or failure is often dependent on government's engagement with the unique ecology of a disease.*

The effects of local and global human structures on microbes were anything but one-sided. Microbes and their vectors responded to their changing environments, with adaptations themselves often inspiring further change. These constantly evolving relationships often formed feedback loops, both positive (with interventions in epidemics furthering the spread of the epidemic) and negative (with interventions reducing habitable environments for microbes and thus dampening the epidemic). In each of the epidemics examined in this project, attempts by local and imperial governments to address disease resulted in changes to urban ecologies, and altered the niches of microbes and organisms implicated in their spread. The ability of a government to address the epidemic tested their authority, and in many cases, undermined it. In Melbourne, the incompetence of the Central Board of Health exposed in part through veterinary and medical investigations into tuberculosis mortality contributed to a complete reorganization of the health structure of the municipal government, consolidating authority of the otherwise

[6] Nada Hassanein, Grace Hauck, Jayme Frazer, and Aleszu Bajak, "Map: COVID-19 Vaccination Rates are Highest in Chicago's Whitest Zip Codes," in "'Just not equal at all': Vaccine rollout in Chicago a microcosm of racial disparities nationwide," *USA Today* February 12, 2021, https://www.u'/satoday.com/in- depth/news/health/2021/02/12/data-analysis-chicago-vaccine-rollout-reflects-us-racial-disparities/4418978001/.

laissez-faire municipal authority. Through this consolidated power, long-term community interests in the removal of noxious trades in the city were realized, and cattle were expelled from the urban environment. Environmental change sparked by urban policy resulted in changes to urban governance, which in turn had a dampening effect on the spread of the microbe – a negative feedback system, predicated on the integration of medical expertise and attention to the ecology of tuberculosis in the city.

In Bombay, initial top-down government responses to *Yersinia pestis* resulted in governmental crisis, with draconian and culturally-deaf measures sparking riots among the residents of the city. Adjustments to plague control focused primarily on broad contagionist ideals, even as the Bombay Bacteriological Laboratory and the city Plague Committee undertook mass studies on rat and flea dynamics. The resulting destruction of property, razing of city blocks, and patchwork interventions targeted at poor communities both increased opportunities for human-rat contact through the destruction of rat burrows and created pockets for rat populations to rebound, ensuring new hosts for *Xenopsylla cheopis* and *Yersinia pestis*. This positive feedback loop between government intervention and disease ecology facilitated the continuation of plague in the city for decades. It is possible that initial lack of adequate plague control measures may have facilitated the traceable evolution of the bacteria; in 1898, the 1.ORI3 variant of *Yersinia pestis* was isolated by the Bombay bacteriological laboratory (genetically characterized and sequenced in the twenty-first century), and historical genomics reveals that it is this variant that was spread to Madagascar and continues to fuel their current cycles of epidemic plague.[7]

[7] G Morelli, Y Song, C Mazzoni, M Eppinger, P Roumagnac, D Wagner, M Feldkamp, B Kuseck, A Vogler, Y Li, Y Cui, N Thomson, T Jombart, R Leblois, P Lichtner, L Rahalison, J Petersen, F Balloux, P Keim, T Wirth, J Ravel,

Meanwhile in Belfast, the public health response calibrated to the specific ecology of typhoid in the city arguably resulted in a negative feedback loop with minimal effect to the authority of the Local Board of Health. By focusing on the specific ecology of typhoid in the city, turning attention away from the oft-suspected water catchment systems and towards the local shellfish population and its interaction with sanitary systems, the Public Health Committee was able to design an effective health intervention that reduced incidence of typhoid in the city.

These brief summaries cannot begin to capture the complexities of each government structure and its relationship to its population, tempered by imperial politics, constructions of race, local interest groups, and countless other administrative and social differences. However, examined together, they convey realities of epidemic response that have implications for current government forms which still grapple with the legacies of these bureaucratic systems. In the COVID-19 pandemic, the success or failure of a government to implement precautions that considered the confluence of the ecology of the SARS-CoV-2 virus and social, economic, and cultural practices. This often manifested in a lack of attention to risk communication, the prioritizing of global economic patterns over local disease incidence, or the outright denunciation of control measures that explicitly thwarted disease transmission dynamics (like masking and social distancing), resulted in poorly controlled epidemics in countries like the United States, Britain, India, and Brazil. These missteps often spelled governmental crisis for each of these administrations and resulted in the establishment of multiple heterogeneous niches for COVID-19 that encouraged the development of variants that threaten higher mortality, higher

R Yang, E Carniel, and M Achtman, "*Yersinia pestis* genome sequencing identifies patterns of global phylogenetic diversity," *Nat Genet* 42(2010): 1140–1143, https://doi.org/10.1038/ng.705

transmissibility, or at worst, vaccine escape.[8] Looking to both the historical and current epidemic context, it appears that government response is itself a factor in evolutionary development.

4. *Infectious disease serves as a potent reminder that humans are embedded in their environments. Through epidemics, our role in ecologies becomes inescapable – but only if we choose to remember it.*

Moments of bodily infection, whether latent or experienced as "disease," are moments where the imperfect borders of the body are breached and we are (quite visibly) forced to reckon with our environments. The air, water, and commensal organisms take on new significance as facilitators of interspecies interaction between person and microbe, eroding the nature-culture divide. In each of the three imperial cities examined in this project, the emergence of an epidemic and the isolation of the causative microbe drew new scrutiny to and encouraged visibility of non-human and ecological dynamics like air circulation, hydrology, cattle immunity, and rat behavior. While ecological features (particularly air and soil) have a long history of integration into scientific and public health epistemologies, the isolation of the microbe as a living, causative agent of disease opened new avenues for the conceptualization of disease-as-ecology, resulting in greater awareness of the role of other organisms and their life processes and biogeochemical cycles in facilitating human infection. In all three cases, this moment of ecological awareness resulted in lasting changes to the urban environment, whether through the banishment of animals from urban centers, the displacement of peoples through slum clearance, or the decline of a particular cultural practice like shellfish gathering.

[8] "About Variants of the Virus That Causes COVID-19," *Centers for Disease Control and Prevention*, Centers for Disease Control and Prevention, www.cdc.gov/coronavirus/2019-ncov/variants/variant.html.

As the first chapter of this project argues and the subsequent case studies demonstrate, even the diseases that are frequently thought of as generalist or crowd-based are strongly influenced by their environments, both built and natural. Both typhoid and tuberculosis, both considered to be relatively common afflictions of 19th century cities, presented challenges to their respective cities on an unusual scale because of the unique confluence of ecology, culture, and built environment. In isolating the causative microbes of each epidemic and grounding those microbes in their respective ecosystems, public health experts rendered importance of ecological dynamics and human integration therein visible – and frequently noted this integration themselves.

However, much as in contemporary epidemic crises, this momentary ecological awareness was often largely forgotten once the epidemic subsided, either through constructed cures or chance. Moments of biomedical optimism, whether through the development of vaccines (especially for typhoid and plague) or, a few decades later, the discovery of antibiotics, were heralded as indications of human capacity to break free from the cycles of nature – a narrative that has persisted in the face of new emerging epidemics throughout the 20th and 21st centuries that have their root in human-environmental interaction.

As countless public health experts and historians have warned in the last several months, we are in danger of suffering the same techno-optimism in the wake of COVID-19 vaccination, even as similarly destructive epidemic events become more likely than ever.[9] Op-eds reflecting on the Spanish Influenza, AIDS, and Zika virus epidemics have argued that we will likely forget the COVID-19 epidemic sooner than expected – in part because we will be eager to put the

[9] Kelly McGuire, "COVID-19, *Contagion,* and Vaccine Optimism," *J Med Humanit* 42(2021): 51–62. https://doi.org/10.1007/s10912-021-09677-3; Heyd T. Covid-19 and climate change in the times of the Anthropocene. *The Anthropocene Review*. 2021;8(1):21-36. doi:10.1177/2053019620961799

trauma of the event behind us, but also because we do not have the appropriate mechanisms or culture of remembrance necessary to recall these events.[10] This project argues that by writing histories that integrate epidemics – and the humans who suffer them – into dynamic ecologies, we can render legible the longer-term challenges of infectious disease that do not disappear at the end of a single epidemic. And to do this, I argue, we need new theoretical models for historical analysis.

5. ***Understanding why diseases occur in time and place requires a more-than-human perspective.***

Amid the COVID-19 epidemic and the epistemological crises spurred by the Anthropocene, a moment of unusual clarity has emerged around the co-constitution of humans and their ecosystems. It has become increasingly clear that the frameworks previously used to conceptualize non-human life processes and ecological systems are at best shallow and static. While disciplines like One Health and Planetary Health have emerged in recent years to correct for this deficiency, un-learning the compartmentalization of human disease and ecology requires a complete re-orientation of storytelling and collective memory that foregrounds these relationships. To understand an epidemic like plague, COVID-19, typhoid, or tuberculosis, in a particular time and place, we must acknowledge the unique life processes of the organisms involved, whether it be microbe, mink, bat, rat, or person, trace the environmental and cultural pressures on their survival, and – crucially – understand how the confluence of those processes and pressures affect other organisms in a shared environment. By incorporating a more-than-human perspective in our explanations of infectious disease emergence, we can begin to adopt

[10] Nina Burleigh, "Why Do We Forget Pandemics?", *The Nation.* April 26, 2021, accessed May 22, 2021, https://www.thenation.com/article/society/spanish-flu-pandemic/; Jonathan Freedland, "history suggests we may forget the pandemic sooner than we think," *The Guardian* Friday January 29, 2021, accessed May 22, 2021, https://www.theguardian.com/commentisfree/2021/jan/29/history-forget-pandemic-spanish-flu-covid/.

the kind of complex systems perspective that is required to expose the "processes of collective self-organisation, adaptation, and evolution," occurring across ecological scales that allow these epidemics to emerge.[11]

In this project, I have posited Niche Construction Theory, as a theory designed to render these relationships legible, as the ideal framework for historians to utilize to begin the work of re-telling and re-defining these stories. While the British Empire served as the connecting scale of reference for niche construction in this dissertation, the expansion of this framework necessarily relies on historians from a wide range of geographic and subdisciplinary backgrounds engaging NCT. Within the scope of histories of disease and imperialism, an expansion of this framework into other imperial settings, building on the extensive literature focused on environmental change driven by French and German Empires, Comanche Empires, Spanish Empires, or Han China, would almost certainly test the robustness of the theory's application to history and, with luck, demonstrate the heterogeneity of ecological contexts that can facilitate infectious disease emergence. Projects focused on smaller, localized scales of reference that detail specific instances of cultural niche construction and its relationship to environment and disease emergence would also provide welcome, nuanced case studies in the utility of more-than-human thought.

Beyond histories of disease, it is my hope that this framework, when combined with existing historical methodologies for nonhuman storytelling, will open new avenues of inquiry in environmental studies broadly. By incorporating the biophysical processes of other organisms into studies of human-environmental interactions, Niche Construction Theory and more-than-human thought generally allows for critical and dynamic analysis of the varying scales of

[11] Jenkins et al., e302.

relationships, both human and nonhuman. This rethinking of environmental-human relationships through richer biological perspective presents the possibility of discovering connections that were previously obscured – just as three seemingly disparate epidemics are connected by imperial niche constructing activities in this study.

The emergence of COVID-19 is just one of a series of events and discoveries over the last two decades that have signaled a need for a deeper and richer understanding of how humans fit within the dynamic worlds in which we live. From the emergence of multiple epidemics to wildfires that threaten human and animal lives and infrastructures on opposite sides of the Pacific, moments of crisis have shattered the imagined divide between humans and the natural world —denoting a precariousness that is conspicuously absent from the stories we have chosen to tell. As panicked stories of the "next pandemic" splash across popular media, it is clear that these narrative absences have left us feeling vulnerable, suddenly exposed to the patterns and movements of a world from which we had assumed submission. These moments are not just physical crises, but existential ones, derived from the false dichotomies that we are guilty of establishing between humans and our natural world. Historical studies of disease are an integral part of rehabilitating that divide. As the current moment and these historical examples show, nonhuman organisms have changed the patterns of human life for as long as we have cohabited and catalyzed change in historical events on both small and large scales. I argue in this project that historians can no longer ignore the specific biology of the organisms with whom we share the planet. These relationships have always existed – and it is time for history to reflect them.

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