# ABSTRACT


Title of Dissertation: INFECTIOUS DISEASE IN PHILADELPHIA, 1690-1807: AN ECOLOGICAL PERSPECTIVE

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This dissertation examines the multiple factors that influenced the pattern and distribution of infectious disease in Philadelphia between the years 1690 and 1807, and explores the possible reasons for the astonishingly high level of death from disease throughout the city at this time. What emerges from this study is a complex picture of a city undergoing rapid cultural and epidemiological changes. Large-scale immigration supplied a susceptible population group, as international trade, densely packed streets, unsanitary living conditions, and a stagnant and contaminated water

supply combined to create ideal circumstances for the proliferation of both pathogens and vectors, setting the stage for the many public health crises that plagued Philadelphia for more than one hundred years.

This study uses an ecological perspective to understand how disease worked in Philadelphia. The idea that disease is virtually always a result of the interplay of the environment, the genetic and physical make-up of the individual, and the agent of disease is one of the most important *cause and effect* ideas underpinned by epidemiology. This dissertation integrates methods from the health sciences, humanities, and social sciences to demonstrate how disease "emergence" in Philadelphia was a dynamic feature of the interrelationships between people and their socio-cultural and physical environments. Classic epidemiological theory, informed by ecological thinking, is used to revisit the city's reconstructed demographic data, bills of mortality, selected diaries (notably that of Elizabeth Drinker), personal letters, contemporary observations and medical literature.

The emergence and spread of microbial threats was driven by a complex set of factors, the convergence of which lead to consequences of disease much greater than any single factor might have suggested. Although it has been argued that no precondition of disease was more basic than poverty in eighteenth-century Philadelphia, it is shortsighted to assume that impoverishment was a necessary co-factor in the emergence and spread of disease. The urban environment of Philadelphia contained the epidemiological factors necessary for the growth and

propagation of a wide variety of infectious agents, while the social, demographic and behavioral characteristics of the people of the city provided the opportunity for “new” diseases to appear.

# INFECTIOUS DISEASE IN PHILADELPHIA, 1690-1807: AN ECOLOGICAL PERSPECTIVE

by

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Dissertation submitted to the Faculty of the Graduate School of the
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DEDICATION

To Marianne

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# CHAPTER 1

## OVERVIEW AND LITERATURE REVIEW

This research project began as an attempt to resolve a controversy as to whether yellow fever killed Philadelphians selectively, taking an especially heavy toll among the city's "lower sort" (as they were labeled by contemporaries)[1] during the epidemics of the 1790s. Research into contemporary accounts and observations suggested that it had; death rates were reported to be higher among the urban poor.[2] The demographic data[3] compiled by Susan E. Klepp from bills of mortality, newspapers, church records, broadsides and personal papers for the years between 1690 and 1807 hinted at something more significant, however – an astonishingly high level of illness and death from disease throughout the city. These numbers fed my growing hunch that the historian would do better to explain the prevalence of epidemic and endemic disease in Philadelphia than to reaffirm that the city's poor were particularly susceptible to disease, especially during times of crisis. Additionally, the *Diary of Elizabeth Drinker*,[4] a rich and detailed source for the history of medicine in the colonial and early national periods from the years 1758 to 1807, revealed that disease was a constant factor in the lives of all Philadelphians – rich and poor alike.[5] Moreover, the medical reports, bills of mortality and

demographic data made it clear that the city was a particularly unhealthy place in which to live.[6]

This dissertation examines the multiple factors that influenced the pattern and distribution of infectious disease in Philadelphia between the years 1690 and 1807, and explores the possible reasons for the high level of death from disease throughout the city at this time. What emerges from this study is a complex picture of a city undergoing rapid cultural and epidemiological changes. Large-scale immigration supplied a susceptible population group, as international trade, densely packed streets, unsanitary living conditions, and a stagnant and contaminated water supply combined to create ideal circumstances for the proliferation of both pathogens and vectors, setting the stage for the many public health crises that plagued Philadelphia for more than one hundred years.

This study breaks with traditional American Studies methodology, and uses an ecological perspective to understand how disease worked in Philadelphia. The idea that disease is virtually always a result of the interplay of the environment, the genetic and physical make-up of the individual, and the agent of disease is one of the most important *cause and effect* ideas underpinned by epidemiology. This dissertation integrates methods from the health sciences, humanities, and social sciences to demonstrate how disease "emergence" in Philadelphia was a dynamic feature of the interrelationships between people and their socio-cultural and physical environments. Classic epidemiological theory, informed by ecological thinking, is used to revisit the

city's reconstructed demographic data, inventories of diseases listed in the various bills of mortality, selected diaries (notably that of Elizabeth Drinker), personal letters, contemporary observations and medical literature.

The period between 1690 and 1807 has been selected for this study for two reasons. First, it was a time during which the population of Philadelphia underwent a significant transition with respect to its disease environment. This period was characterized by events that considerably changed the risk factors for disease through changes in population levels, changes in pathogens, and changes in human behavior. It was also characterized by intense change in the physical environment of the city. Short-term transitions are often distinguished by the introduction of new pathogens, new therapies, environmental modifications or demographic changes brought about by contact with outside groups.[7] Philadelphia experienced not one but many of these transition factors, and the extraordinarily high levels of morbidity and mortality in the city reflected this. As a result, this period in Philadelphia's history provides an extraordinary opportunity for scholars to study disease "emergence" as a dynamic feature of the interrelationships between people and their socio-cultural and ecological environments. Second, the data for this type of study was readily available thanks to the meticulous compilation and reconstruction of Philadelphia's vital rates by Susan E. Klepp. Deaths from 1690 to 1807 are based on local bills of mortality collected in Klepp, 1991, *"The Swift Progress of Population*," and supplemented by church registers and other records.

It is evident that the years between 1690 and 1807 were a remarkable time in Philadelphia's history, as the city experienced what could arguably be the first series of environmental health crises in theUnited States. The context in which these crises occurred subsequently became the focus of my research. In this light, the yellow fever epidemics of the 1790s assume a new and relatively smaller dimension. Though they remain crucial events in the city's history, they appear here as strands in a much larger web of causation that stretches back to the early days of European settlement. On one level, this work tries to explain why Philadelphian morbidity and mortality were so high during the eighteenth century. On another, it is a synthesis of human actions, having the broad intention of enhancing our understanding of historical processes by emphasizing the complexity of cause and consequence. Introducing particular human interactions into a web of ecological interdependence allows us to see the unfolding of events in Philadelphia from the broadest possible formulation of causality, encompassing the whole system of relationships. Infectious diseases are dynamic, and many factors influence their burden in any population – past or present. I argue that Philadelphia's health crises were the result of the convergence of several social, cultural and environmental processes. The physical environment of the city provided the pool of potential pathogens, while the social, demographic and behavioral characteristics of the people of Philadelphia provided the opportunity for "new" diseases to appear.

## Methodology

This study interprets the historical record with conceptual guidance from the health sciences to set Philadelphia's health crises in an historical framework that shows people interacting *with*, rather than acting *within*, their *total environment.*[8] Any historian working with issues raised by the presence of infection in past populations owes an enormous debt to Alfred Crosby, Ken De Bevoise, William McNeil, Charles E. Rosenberg and others for demonstrating the importance of disease as a critical force in human history. As an interdisciplinary thinker trained in the biological sciences, anthropology, and history, I thought it necessary to fashion an ecological approach to understand the Philadelphia experience, however. Typically, three general forces can affect the burden of infectious diseases in a given population: change in abundance, virulence, or transmissibility of microbes; an increase in probability of exposure of individuals to microorganisms; and an increase in vulnerability of people to infection and to the consequences of infection. A wide range of biological, behavioral, cultural and social factors can influence one or more of these forces. Many are interrelated, and multiple synergies exist.[9] Consequently, an ecological approach allows us to see human health as an outcome of multiple, reciprocal and continuing interactions between pathogens, hosts, and their enveloping environment.

In the introduction (Chapter 2), I describe the public health crises that existed in the city, and in Chapters 3 and 4, I account for their occurrence using the classic

epidemiological model.[10] The model holds that the patterns of disease in any population group depend on factors that determine the probability of contact (Chapter 3) between an agent of disease and a susceptible host (Chapter 4). In a systematic analysis based on disease causation models,[11] attention is deflected from the microorganism as a specific cause, to the environment, host and agent as interacting causes. Models simplify reality and make it easier for the mind to grasp the essence of the issue. Applying this formulation to the historical record allows us to see the Philadelphia crises approaching, and by mapping their journey we can understand why they occurred when they did. The evidence supports the argument that all Philadelphians faced the kinds of challenges that promote vulnerability to disease. If factors linked closely with population growth and mobility were most critical to probability of contact, those responsible for chronic disease, debilitation, malnourishment and poverty determined susceptibility.

In Chapters 5 and 6, I select two disease groups: the epidemic and the endemic diseases.[12] Each group is analyzed within the context of the *total environment*. Since diseases form patterns, and the factors that cause these recognizable patterns are usually complicated interactions between individuals, their physical environment, and their society,[13] this type of multidimensional analysis will help us draw meaning from the complex of relationships. I conclude the study in Chapter 7 with a brief consideration of what the evidence has revealed about how human actions and interventions opened the way for Philadelphia's environmental health crises.

As early as 1940, Macfarland Burnet argued that interaction between human beings and infectious agents is so complex that it can only be understood in the context of their mutual relationship to the global ecosystem.[14] It was René Dubos, however, who conceived of the entire process as taking place within a *total environment*. He explained that "the process of living involves the interplay and integration of two ecological systems." These systems include the community of interdependent cells, body fluids, and tissue structures that make up an organism's internal environment, and all the living and inanimate things with which it comes into contact. Although he did not define it explicitly, the total environment evidently encompasses one's physical, biological, psychological, cultural, political, socioeconomic, and historical universe. As living things ordinarily achieve an unsteady and temporary *ecological* equilibrium sufficient for survival, any change in the "constellation of circumstances" under which the equilibrium evolved can upset the balance. Under these circumstances, it is possible for disease to swamp the host defenses if the change is too sudden for adaptive mechanisms.[15]

Metaphors – such as webs, networks, and intersections – have been used as conceptual tools when trying to comprehend the complex nature of causation.[16] They suggest, among other things, a system of highways, and virologist Stephen S. Morse effectively uses this analogy to analyze the role of human agency in disease causation. He takes the position that human activities, whether through changes in behavior or alteration of the environment, bear substantial responsibility for the emergence of "new" viruses. The social developments that create conditions favoring

rapid viral dissemination he compares to highways complete with "stop" and "go" indicators, and he explains *emergence* as a result of changes in the patterns of "viral traffic." Additionally, he calls for biomedical and social scientists to work closely toward a "science of traffic patterns."[17] This study seeks to participate in this endeavor by trying to understand what cleared the way for epidemic and endemic disease in Philadelphia during the eighteenth century. Since politics was just one of the many factors that shaped the flow of disease, and because the political history of Philadelphia has been written about extensively, I have chosen to underplay that aspect in this study. I have given the years 1690-1807 their own ecological space rather than try to fit them unnaturally into the usual (and implicitly political) organizational scheme that is typically used to describe eighteenth-century Philadelphia. Because I have attempted a history of a past population group that looks at familiar events in a new light, this study consciously and necessarily depends on previous scholarship, building on it as part of a cooperative effort to find new ways of understanding historical processes.

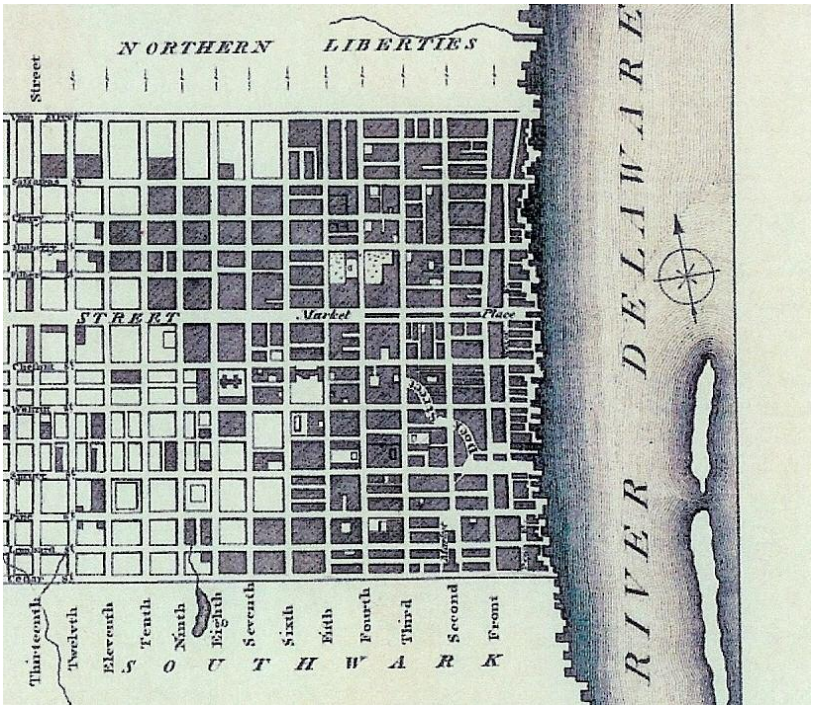
Historical inquiry can bring a valuable perspective to the understanding of disease emergence by focusing on "the consequences of human actions, and the conditions that permit certain developments."[18] The emergence and spread of microbial threats in eighteenth-century Philadelphia were driven by a complex set of factors, the convergence of which led to outcomes of disease much greater than any single factor might have suggested.[19] Human behavior, however, both individual and collective, was perhaps the most significant of all. The eighteenth century was a time

of dynamic growth and change for Philadelphia. The size and mobility of the city's population increased the potential for pathogens to escape their prior geographic boundaries. High levels of immigration, coupled with densely packed city streets, increased both interpersonal contact and contact between people and animals. Domesticated animals such as goats, sheep, cattle, pigs and fowl provided novel reservoirs[20] for zoonoses (disease of animals that can be transmitted to humans). Endemic diseases such as dysentery, malaria and tuberculosis severely weakened their victims, and increased their susceptibility to other infections. And industries such as tanning, sugar refining and milling altered the physical environment and caused ecological disruptions.[21] Infectious disease is a moving target, and as the climate and other sources of natural or anthropogenic change occur in a community, any disease that has an environmentally sensitive stage, reservoir, or vector (any person, animal or microorganism that carries and transmits disease) will be affected.[22] It is quite possible that the sudden and abrupt nature of these changes was sufficient to disturb the delicate ecological equilibrium that existed in the city, and paved the way for disease to take hold.

Consider, for example, the relationship between industrial pollution and an area known as Dock Creek. Dock Creek was an inlet leading from the Delaware River into what became the heart of colonial Philadelphia. The Front Street and Walnut Street areas were the beginning of Philadelphia as a city, with the area around Dock Creek being the early center of the city's commerce (**See Figure 1.1**). Shops, tanneries, stables, taverns, and inns were built on the creek banks. Dock Creek also

served as a manufacturing and materials-processing center for the growing city. By the mid-eighteenth century, environmental problems were a serious domestic challenge for the city, as the Dock and its environs had become the center of pollution and controversy.[23] Citizens increasingly focused their complaints on the tanyards, which by 1730 numbered at least eight.[24] Not only the number of tanneries but also the nature of the tanning process itself made the physical state of the watercourse a central and persistent public health issue throughout the eighteenth century.[25] When yellow fever visited Philadelphia in 1793, Dr. Benjamin Rush found the disease more severe in the neighborhood surrounding Dock Creek. He complained to the City Council about the condition of the Creek, and shortly afterwards they ordered that parts of the Dock be arched and filled. Unfortunately, Philadelphia's dense residential areas contained large numbers of household breeding sites for mosquitoes (containers, clay pots, etc.). Consequently, filling the Dock had little effect on the mosquito population, and yellow fever continued to plague the city well into the nineteenth century.

**Figure 1.1**
**Plan of the City of Philadelphia, 1800**




Source: Map is from W. Birch and Son, 1800, *The City of Philadelphia…As it Appeared in 1800*, Plate 3.

The tanning industry, polluted streams and their role in the appearance of yellow fever well illustrates the complexity of environmental circumstances and the emergence of disease in Philadelphia. Environmental change - which was influenced significantly by population growth, resource consumption, and waste generation - played an important role in the emergence of yellow fever and other infectious diseases in the city. Additionally, urbanization, agricultural expansion and intensification, industry and natural habitat alteration[26] produced changes in Philadelphia's ecological systems, notably in landscapes. These changes in the city's landscapes affected natural communities and, in turn, affected pathogens, animal hosts, and human populations. Therefore, in order to understand how disease worked

in Philadelphia, one has to think ecologically, seeing human health as the product of numerous interactions among pathogens, hosts and the surrounding environment. Such multidirectional reasoning highlights what we often overlook in our search for underlying historical causes – the simple notion that each cause has numerous effects, and each effect is, in turn, the result of numerous causes. The yellow fever epidemic of 1793 was not caused by a polluted stream, nor was it caused by industry along the banks of Dock Creek. The creek, however, provided one breeding area for the mosquito, while the tanning industry provided one group of susceptible workers who, in turn, provided the necessary blood meals for the hungry bugs.[27]

The polluted nature of the Dock also may have contributed to outbreaks of dysentery,[28] which, in turn, may have weakened its victims to such a degree that they were unable to survive a bout with yellow fever. Other endemic diseases, notably tuberculosis,[29] a chronic, debilitating disease constantly present in the city, made its victims particularly susceptible to additional infections. Although tuberculosis is usually transmitted by inhalation or injection of droplets, it can also be transmitted through the products of domestic animals such as milk, hair, skin and dust – animal hair and skin being particularly prevalent in the vicinity of slaughterhouses and tanneries. The bacterium causing tuberculosis was most certainly a causal factor in the existence of the disease, but so too were crowded homes, poverty, debility and cultural attitudes that encouraged sharing a bed with a sick family member. So, the concept of causation becomes increasingly complex as we learn more about the

interaction of environmental processes with the societal and cultural factors that influenced human behavior.

The complexity of cause and consequence clearly dominates the history of health and disease in Philadelphia during the eighteenth century. A debilitated population, crowded living conditions, widespread poverty, rampant population growth in combination with social, cultural and environmental upheaval created a city much in crisis. Narrow thinking based on single causes can mislead epidemiologists into prematurely believing that a problem has been resolved and can severely distort public health action. Similarly, narrow causal thinking can mislead historians into believing that socio-economic status played a much larger role than it did in the emergence of disease in eighteenth-century Philadelphia. This study attempts to lay a foundation for a thoughtful reconsideration of the epidemiological history of the period, and to explore the extent to which Philadelphians helped to create their own disease environment.

## Sources

As the first capital of the United States and one of the largest, most prosperous towns in colonial America, Philadelphia has attracted much scholarly attention throughout the past several decades. Susan E. Klepp's[30] meticulous reconstruction of Philadelphia's crude birth and death rates provides the statistical outline of the process this dissertation will try to explain and elaborate. Additionally, there have

been numerous social, political, and economic studies of early Philadelphia,[31] some environmental studies,[32] various social histories of the yellow fever epidemics of the 1790s[33] and one historical overview of public health in the city during the colonial years.[34] There has been no study of disease patterns in the city, however, nor has there been any discussion of the intimate relationship between environmental circumstances, social conditions, human biology and the occurrence of disease. This dissertation attempts to remedy this oversight. By using the methodology described earlier to revisit the reconstructed demographic data, the inventory of diseases listed in the various bills of mortality, selected diaries (notably the *Diary of Elizabeth Drinker*), personal letters, contemporary observations and medical literature, I show how disease "emergence" in Philadelphia was a dynamic feature of the interrelationships between people and their socio-cultural and physical environments. This type of multidimensional analysis underscores the dependence of human population health upon stocks of natural resources, functioning ecosystems, and cohesive social relations.[35]

Elizabeth Sandwith Drinker had so much to say about so many things in the nearly fifty years that she kept a personal diary. Born in Philadelphia in 1735 to Irish Quaker parents, she began the diary in 1758 after she and her one surviving sibling, Mary, had been orphaned and taken in by another Quaker family. She was knowledgeable, intelligent and witty. More to the point, she was observant. Burdened with childcare, a household to run, and servants to manage, the young Elizabeth Drinker had little time to write more than an occasional sentence or two in

her journal until the 1790s. But as she added years to her life, she added lines to her entries, composing three-fourths of the diary after her fifty-seventh birthday. Eventually, she produced nearly three dozen small manuscript volumes filled with nothing extraordinary, except as a whole, the journal is probably the most remarkable literary work written by a woman in eighteenth-century America. Certainly, it is the most extensive.[36]

The ChristChurch bills of mortality were perhaps the most significant source used by Klepp in her reconstruction of Philadelphia's vital statistics for the years between 1690 and 1860. The bills have been used for a number of purposes, and are one of the best single sources for studying the incidence of disease in colonial Philadelphia.[37] In his famous history of epidemic diseases, the ubiquitous lexicographer Noah Webster used the bills to illustrate his theory on the relationship of atmospheric disturbances and disease.[38] Nineteenth-century historian Burton Konkle used them for his brief description of major epidemics.[39] Roslyn Stone Wolman's collation of "cause of death" and "age at death" data from the bills between 1751 and 1775 provides a valuable backdrop to her discussion of the state of public health in the last years of the colonial period.[40] Gary B. Nash has used these bills and earlier newspaper accounts to estimate the African-American population of the city, under the assumption that black and white Philadelphians had similar mortality rates.[41] Billy G. Smith also used many of the bills for his reconstruction of crude birth and death rates in the city.[42] In this study, the Christ Church bills of

mortality are again used to provide a context for a larger discussion of morbidity and mortality in Philadelphia.

The origins of the Christ Church bills of mortality for Philadelphia are unclear since few of the earliest accounts survive in their original form. According to Klepp, as early as 1721, the clerk of Christ Church parish prepared an account of baptisms and burials by sex for the *American Weekly Mercury*. But it was under the direction of the Reverend Archibald Cummings that the Anglican Church began collecting and publishing its own series of baptisms and burials. He kept a separate account in the registers of the church dating from the beginning of his tenure at Christ Church in 1726 and lasting until 1740, the year before his death. Mr. Cummings' Private Register listed and counted deaths by sex and noted outbreaks of smallpox between 1726 and 1740. Most of the deaths recorded in his register were Anglicans, but some deaths in other denominations were included, especially in the early years of the account.[43]

The first bill of mortality known to have been published by the church was for the year 1737.[44] Christenings and burials were counted in Christ Church parish and burials in four other churches were given. Cause of death among Anglicans was reported under three main headings, "Common Distempers," smallpox, and various casualties. Like the city officials who first authorized the publication of bills of mortality in London, the compiler was primarily interested in the prevalence of epidemic disease, not the incidence of endemic disease. The next bill to survive in its

original form covered 1747. Much more information was recorded. Anglican burials were given by sex, by age, and by cause. Twenty-nine disease categories were listed. Epidemics were no longer the primary concern of the compiler. The total number of deaths in six other churches, in the Strangers' Ground and the Negroes' Ground, and the sum total of all burials was given. Later bills added the number of baptisms in other churches.[45]

While a sustained series of bills survive only for the period 1751-1775, other sources preserve some of the data from the missing years, allowing Klepp to reconstruct the vital statistics for the city. Much of the 1741 bill, for example, was published in the *Mercury*, the burials by church for 1738-1744 appeared in *Poor Richard Improved, 1750*, and Franklin's now missing collection of bills was used by Peter Kalm for his account of total deaths in the city, 1745, 1748-1750. A few other listings survive as well.[46]

If precision is impossible, then the general trends the numbers describe are reliable. It is beyond question that for much of the eighteenth century, epidemic and endemic disease inundated the city. The Christ Church bills of mortality list as causes of death such conditions as "flux," "fever," "decay," "worms," "consumption," "fits" and smallpox year after year. This data in combination with contemporary medical reports, newspaper accounts, city records and personal letters and diaries paint a picture of enormous suffering and distress for the residents of the city. By the middle years of the eighteenth century, Philadelphia was an overcrowded

city of many streets and alleys. Poverty, poor sanitation, and disease were among its defining characteristics. Clustering of illness among the crowded, slum-dwelling poor certainly reinforces the causal link between illness and socio economic status, and some contemporary observers made similar references. Matthew Carey, in particular, made repeated references to the relationship between the urban poor and epidemic disease.[47] What was not considered, however, were alternative explanations for these findings. Eighteenth-century life was characterized by crowded living conditions, poor sanitation, limited access to clean water, rudimentary medical care, poor nutrition, and lack of separation from insects and animals in the environment. These features increase the probability of exposure to infections in such vulnerable populations. The longer and closer the contact between a person with a contagious disease and a susceptible individual, the more likely is transmission. It is a principle that should seem obvious to us now, but it eluded most eighteenth-century observers. Poverty is one marker of a common causal element – close contact. Poverty, however, does not *cause* disease.

While there existed at this time in Philadelphia a practical belief in the relationship of disease and health to environmental conditions, there were few effective responses. Miasmatists[48] like Benjamin Rush, and those who followed him, intimated an understanding of the complexities of causal relationships. Rush observed that there seemed to be a higher incidence of disease as the city grew larger, and thought that perhaps this was linked to the clearing of woods and land. The phenomenon, he noted, was not limited to Philadelphia. He wrote in 1785: "It has

been remarked that intermittents [fevers] on the shores of the Susquehannah have kept an exact pace with the passages which have opened for the propagation of marsh effluvia, by cutting down the wood which formerly grew in the neighborhood." Rush also believed that cultivation was the answer: "draining swamps, destroying weeds, burning brush, and exhaling the unwholesome or superfluous moisture of the earth, by means of frequent crops of grain, grasses and vegetables of all kinds, renders it healthy." He thus saw an environmental balance being restored, much like the "humors of the body" were restored after an illness.[49] And in the early 1750s, a physician named Thomas Bond linked pollution and imbalance in the environment to imbalance in the patient, and urged a campaign to clean up the city. His concerns found strong reinforcement during the 1762 yellow fever epidemic.[50] Although it has been argued that Philadelphia's city government was dreadfully inept[51] during this period in its history, this experience initiated the first comprehensive efforts by the Corporation and the Assembly to organize paving and cleaning of streets, removal of solid wastes, and extension of the city's drainage system. As extensive as these actions were, however, they addressed one small piece of a much larger puzzle. As a result, they had little effect on the city's over-all death rate.

Multiple factors influenced the pattern and distribution of disease in Philadelphia. Mortality levels in the city continued to soar during the 1730s, 1740s and 1750s, and did not begin to decrease until after the mid 1770s. Understanding the mechanisms that underlie newly emerging and reemerging infectious diseases is one of the most difficult scientific problems facing society today.[52] There is an urgent

need to integrate knowledge about infectious diseases with knowledge of climate and environmental change, migration and population growth, demography, and the consequences of conflict. All are inextricably linked and play a part in the changed patterns we are seeing in infectious diseases.[53] There is an equally important need to integrate these factors in studies of disease patterns in past populations. Then, as now, the most important variable in disease emergence is a change in the pathogen-host relationship – a change often associated with anthropogenic activities and increasing human population densities.

## Literature Review: Environment and Health, Environmental History, Disease History and Disease Ecology

### Environment and Health

The study of environmental health is a broad field, encompassing a rich body of literature traversing a variety of disciplines. Until recently, however, the environment has not been a category of analysis in studying the history of disease in society. In order to understand the historical "conditions of possibility" for public health crises like the one that existed in eighteenth-century Philadelphia, an interdisciplinary perspective that incorporates ecology, medicine, anthropology, epidemiology and history is required. In medieval times, Europeans envisioned extensive correspondence between their bodies and the cosmos. More recently, however, the rise of specialist disciplines has tended to sever this connection. As a

result, today's environmental and public health historians face a long tradition of chopping "health" and "environment" into distinct realms of knowledge and practice.[54] To fully appreciate the complexities of disease emergence and transmission in past populations, synthesis, rather than severance, is the more prudent approach.

Although environment and health have long been seen as having separate histories, they do share numerous roots. Many scholars who recently moved into this terrain have been motivated and inspired by social movements to combat the inequitable distribution of, and exposure to, environmental hazards. These movements have emerged as an important faction of worldwide political struggles for justice and equality. In the United States, for example, the environmental justice movement became a major political force in the 1980s and 1990s. Consequently, it shifted the agenda of many environmental groups from land preservation and pollution control to problems of urban and industrial wastelands and a reckoning with the geography of race and poverty.[55]

Interest in environment and health has also been inspired by scholarship arising over the past two decades at some particularly fertile intersections between environmental and medical history. Historians of urban sanitation such as Martin Melosi and Joel Tarr have brought out the continuing importance of infrastructure, engineering, and an environmental focus to modern public health issues. Historians of occupational health have woven together workplace and medical histories in a

variety of ways, from Arthur McEvoy's proposal for an "ecology of the workplace" to Chris Seller's argument about the workplace origins of modern environmental health science.[56] Environmental historians responding to the challenge of environmental justice, too, have turned increasingly to the health dimensions of topics such as pollution and industrial wastes, dimensions often downplayed by earlier historians of conservation and environmentalism.[57]

Environmental and medical historians have only recently begun to explore the manner in which scientific and popular practices, as well as regional economies, were shaped by ideas and experiences resulting from the interplay between health and nature. Although the rise of modern medicine supposedly eclipsed a Hippocratic emphasis on airs, waters, and places by the early twentieth century, there remained a persistence of Hippocratic concerns through this period and beyond.[58] Whether informed by medical geography and climatology in the nineteenth century or by ecological conceptions of community in the twentieth century, many notions of regional disease and health as "ecological" can be found in the literature. Aldo Leopold's extensive references to both the health of the land and to conservation as the "art of land doctoring," for example, offer one indication of the ways in which ecological conceptions of conservation and community borrowed heavily from Hippocratic ideals and "experiential wisdom of the relationships between health of the body and place."[59]

Scholarship on the environmental dimensions of twentieth-century public health has also raised new questions about the variety of "environmental" and "ecological" perspectives that have emerged and the ways they have overlapped, intertwined, or clashed.[60] While American health scientists were more likely to style their work as "environmental," others were influenced by ecological science. In the United States, for example, Rachel Carson's classic *Silent Spring*, which helped make ecology a household word in the 1960s, drew upon the emerging field of ecosystem ecology and the public health discipline of environmental toxicology to trace the health effects of pesticides on humans and wildlife.[61]

Consequently, contemporary environmental problems, new social movements, and past historical scholarship have inspired an emerging body of research on the subject of environment and health. Not just historians of differing agendas and interests, but also anthropologists, sociologists, and geographers, have turned to explore the intersection of place, health, and political economy in diverse settings and unique historical periods. Environmental history, for example, through its penchant for broad historical narratives that give agency to both nature and humans, has offered a compelling yet underutilized model to historians of science interested in connecting their field's preoccupation with local sites of knowledge production to narratives that reach across larger spatial and temporal scales.[62] Similarly, the shift within medical geography and environmental history to more place-centered approaches and more embodied local geographies of health and disease offers an important point of contact

with historians of science similarly engaged with questions about the place-centered position of scientific knowledge.[63]

## Environmental History

The discipline of environmental history is fertile ground for those historians who want to shift their work from the act of simply documenting and explaining past events, to considering how these events inform the present and the future. Environmental historians have long been concerned with suggesting modified values and behaviors that will improve both individual lives and the life of the planet.[64] In the early years of the twenty-first century, urban, suburban and rural America finds itself suffocating under an avalanche of environmental dilemmas. It takes only a quick glance at the daily newspaper to appreciate the enormity of the environmental challenges confronting cities. How do we keep our drinking water free from contamination? How do we go about cleaning the soil of toxic materials dumped indiscriminately by manufacturers over the past two centuries? And the list goes on. It is important to remember that although history will not necessarily provide the solutions, it will certainly enrich our understanding of the social forces that lead us into these predicaments in the first place.

Environmental history comprises a set of approaches to doing history that brings nature into the story. Natural conditions such as climate, rainfall, terrain, vegetation, and animal life create possibilities for the quality of human life.[65] The

false dichotomy between the urban and the natural, so misleading in any analysis of cities and towns, is exacerbated by a misguided impression that there exists a nature in wilderness that has somehow remained untouched by human history. In fact, human beings have shaped the physical environment in both its "natural" wilderness settings and in its urban context. When we think of pure wilderness, we are projecting an idealized notion of what constitutes nature, seeing it as something pristine or separate from ourselves. We ignore that plants, places, and animals have evolved in the context of human activity, if not through deliberate manipulation, then at least through the indirect effects of global ecological changes in which humans have exerted a role. More often than not it has been people living in cities who have been responsible for both constructing our idealized notions of nature and introducing the human element into it.[66]

Most major cities are where they are because of environmental factors: snug harbors, breaking points along rivers, junctures of ecological zones, etc. In addition, the spatial distribution of economic functions and population groups within cities and towns has followed the physical contours of the landscape. It is no coincidence, for example, that in so many cities the wealthiest districts are found on high ground where the air circulates rapidly, vistas are the most pleasing, and the ground is best protected from unforeseen floods. Likewise, factory districts often occupy land adjacent to rivers and harbors where manufacturers have enjoyed easy access to water for transportation, waste disposal, and power. To exclude this dynamic from the history of cities is to miss much of the story.

Putting the environment back into the city and town, or at least into the history of the city and town, necessitates a revision in the familiar arrangement of events, settings, and actors. The rise of social history over the past thirty years has expanded the cast of historical characters to throw light on the contributions that women, laborers, racial minorities, and the poor have made to society. In the process, the setting of historical studies has shifted from the corporate boardroom and the legislative hall to the plantation, the neighborhood street, the factory floor, and the waterfront wharves. Rewriting history from the bottom up has demanded a thorough revision of the way the past, along with key actors and processes, is understood. Consequently, interpretations change when these previously ignored groups or processes enter the picture.[67]

As one might expect, reorganizing any history around the theme of the physical and natural landscape provides yet another set of criteria with which to assess the past. The salubrity of the environment, the sustainability of urban ecological systems, and the degree of equity involved as environmental transformations impinge on diverse social groups are a few examples. An environmental history will, by necessity, bring different episodes and characters to center stage. Events that once may have been considered of secondary importance now emerge as key turning points.

William Cronon details the effect of urban growth on the hinterland of Chicago as it spurred the rise of commercial agriculture and the intensification of

resource extraction in *Nature's Metropolis: Chicago and the Great West*. The book consists of a series of stories, each tracing the path between an urban market and the natural system that supports it. The heart of the author's argument is made in four consecutive chapters on the major industries of the period: railroads, grain, lumber, and meatpacking, and the commodity flows that drive their markets. Grain, lumber, and meat - the commodities that feed, clothe, and shelter us - are our most basic connections to the natural world. According to Cronon, if we wish to take political and moral responsibility for the consequences that resulted from the exploitation of the commodities of our economy, we must reconstruct linkages between these commodities and the resources of our ecosystem. Through a synthesis of Chicago's 19th Century regional economic history, Cronon reconstructs these linkages and concludes that Chicago cannot be understood apart from the environment surrounding it. The perspective presented by the author defines frontier not as the "great expanse" of Frederick Jackson Turner,[68] but rather as an elaborate, interconnected network. Since urban historians rarely looked beyond the city limits, and western and environmental historians usually concentrated on rural areas, *Nature's Metropolis* was significant in that it was one of the first scholarly works to tell the city/country story as a unified narrative.

One of Cronon's most provocative contributions to the field of environmental history, however, is the edited volume *Uncommon Ground: Toward Reinventing Nature*, a collection of essays that originated from an interdisciplinary seminar on "reinventing nature" held in the spring of 1994. The essays incorporate a wide range

of perspectives from the sciences and humanities. While the subtitle of the book is "reinventing nature," the real theme running through these essays is the question of how to reinvent human relationships with nature. The "uncommon" ground upon which the authors stand refers to the refutation of nature as a common, universal identity, as a unified set of relations that are inherently nonhuman. Instead, the authors share a perspective of historical particularity in the idea of nature - that nature is a human construct located in a particular time and place.

"The Trouble with Nature," which is the title of Cronon's lead essay, refers to an ideological position that constructs a separation between the natural world and human beings. He argues, convincingly, that this results not only in a flight from history but in irresponsible environmentalism: there is a dangerous naïveté inherent in the dualism that posits the natural exclusively outside of a human context. Cronon assesses that the challenge is to stop thinking according to a set of bipolar moral scales in which the human and the nonhuman, the unnatural and the natural, the fallen and the unfallen, serve as a conceptual map for understanding and valuing the world. Instead, one needs to embrace the full continuum of a natural landscape that is also cultural, in which the city, the suburb, the pastoral, and the wild each has its place which people permit themselves to celebrate without needlessly denigrating the other.[69] Putting the environment back into towns and cities is one way of breaking the momentum of past behavior.

Environmental historians approach their field from a variety of perspectives. The one most relevant to this dissertation, however, is the focus on biological interactions between humans and the natural world. Animals, plants, pathogens, and people form an ecological complex in any one place that can be sustained or disrupted. When Europeans settled in North America and other temperate regions of the world, they introduced diseases such as smallpox, measles and tuberculosis; livestock, such as horses, cattle, and sheep; grains such as wheat, rye, barley, and oats; and weeds such as plantain and dandelions. These ecological disruptions, especially diseases, devastated the lives of native peoples. While some of the introductions may have been beneficial, the resulting changes in the landscape altered the lives of many.[70]

## Disease History

Public health and disease history would also appeal to environmental and biologically inclined historians, at first through the *Annales* school and then, in the 1970s, through the work of American historians seeking a broad conceptual framework for ambitious surveys. In the *Columbian Exchange*, for example, Alfred W. Crosby expressed a desire to understand man above all as a "biological entity." Inspired by Percy Ashburn's *The Ranks of Death*, a popular book on the contribution of disease to the European conquest of America,[71] Crosby described how European invaders had disrupted the ecological stability of the New World, spreading disease to vulnerable populations. He charted the transfer of plants, animals, and germs

between Europe and the Americas, arguing that this "Columbian exchange has left us with not a richer but a more impoverished genetic pool."[72] A few years later, William H. McNeill applied the theories of Theobald Smith and Hans Zinsser to the historical study of disease outbreaks.[73] McNeill was concerned about human persistence in "tampering with complex ecological relationships." Since World War II, the new generation of disease ecologists, in particular Australian parasitologist F. Macfarlane Burnet and American bacteriologist René Dubos, had been warning of the biological dangers of population growth, biological warfare, and environmental degradation. Like them, McNeill feared that "a sequence of sharp alterations and abrupt oscillations in existing balances between microparasitism and macroparasitism can therefore be expected in the near future as in the recent past."[74]

Alfred Crosby attempts a much broader synthesis of the ecological repercussions of European expansion throughout the past millennium in his seminal work *Ecological Imperialism: The Biological Expansion of Europe, 900-1900*. Many historians from many different perspectives have studied this epoch of expansion, but Crosby focuses attention away from the political and military aspects of European expansion, and refocuses his analysis onto the plants, animals, and microbes that accompanied explorers and colonists. These organisms, argues Crosby, so altered the landscapes of Australasia and the Americas that parts of these regions became areas with ecosystems ideally suited to the European settlers. Crosby contrasts the unsuccessful early European migrations of the Vikings and crusaders with the later successful invasions of the Azores, Madeiras, and Canary Islands. He argues that

Europeans were most successful when they occupied ecosystems capable of sustaining cultigens, domesticated animals, and diseases common to the "Old World." In his analysis of the post-Columbian migrations of the 16th, 17th, and 18th centuries, he claims that these events reproduced Europe's ecological relationships in what he referred to as the "neo-Europes." In parts of North and South America, Australia, and New Zealand, native populations - human, as well as nonhumans - were quickly replaced by European biota. This supports Crosby's thesis that Europeans flourished in areas where the nonhuman biota flourished, and failed where the nonhuman biota failed.

*Ecological Imperialism* reinterprets from a biological perspective the well-known scheme of events surrounding European expansion. By expanding the reasons for the success of "Old World" organisms in "New World" environments, Crosby incorporates a global perspective that challenged readers to see new connections between European invaders, native peoples, and their associated biota. Crosby's analysis continues to mark this work as an exceptional example of interdisciplinary scholarship, and well demonstrates the importance of disease as a critical agent in human history.

Charles Rosenberg continues this theme in his path-breaking and widely influential book, *The Cholera Years: The United States in 1832, 1849, and 1866*. Here, he argues that to understand the nature of antebellum American society, historians necessarily have to understand the implications of epidemic disease. While

not a medical history in the traditional sense, Rosenberg nonetheless does a thorough evaluation of the medical thoughts surrounding cholera and cholera epidemics. He centers his primary attention, however, on the changing social meaning of the disease. By taking such an approach, he provides a window into the workings of nineteenth-century American life.

Rosenberg's insightful choice to make the disease the primary object of his inquiry was a decidedly innovative act at a time when political, institutional and intellectual history dominated the academic landscape. In the analysis, the author constructs what he calls a "natural experiment." During the course of the nineteenth century, epidemic cholera struck New York City three times: 1832, 1849 and 1866. In each instance, the response to the crisis was shaped by a series of historically specific variables: religion, the role of the medical profession, theories of disease and urban demographics. The epidemic of 1832, for example, provided the impetus for religious leaders to call for fasting and repentance, viewing the disease as divine retribution. In 1849, debates centered on the problem of sanitation, yet city services remained meager in the face of urban poverty. The epidemic of 1866 culminated in the creation of the Metropolitan Board of Health – a turning point in governmental response to disease. In charting the responses to cholera, Rosenberg is able to define the shift from a fledging urban society that accounted for disease in essentially religious terms to one that accounted for disease in more proactive, scientific terms.

*The Cholera Years* makes a strong case for the significance of biological forces as historical agents. If cholera was the same disease striking at different times, the changing explanations for its occurrence and the subsequent responses to it would reveal shifts in its social, moral, medical and political meanings. By assessing the religious, medical and political responses to these three health crises – crises separated by important historical changes in both social and cultural life – Rosenberg expands the realm of historical inquiry. It was largely works such as *The Cholera Years* that effectively staked the claim that medicine and science could only be understood within a broad social context.

In his aptly titled book *Agents of Apocalypse: Epidemic Diseases in the Colonial Philippines*, Ken DeBevoise describes the various epidemics that swept through the Philippine Archipelago in the late nineteenth century. Between 1882 and 1903, epidemic diseases ravaged the Philippines. Although this phenomenon had been studied before, DeBevoise's portrayal of the crisis is by far one of the most comprehensive and meticulous. By systematically applying principles from the field of epidemiology in ways not normally undertaken by historians, the author makes a powerful contribution not only to Philippine history, but also to interdisciplinary scholarship as a whole. More importantly, however, he bridges the gap between scientific application and the causality of history, and thus explains how social, economic, military and political influences spread disease.

After providing an overview of the crisis within its historical time frame, DeBevoise discusses how epidemiologists view epidemics and explain their etiology. In five subsequent chapters, he examines STDs, smallpox, beriberi, malaria and cholera, and discusses their impact on both the indigenous peoples and the colonials. While the author assesses each disease individually, he convincingly shows how they often share common causes and interact with one another. For example, he attributes the rise in beriberi deaths to, among other factors, changes in the agricultural economy, urbanization, and to the presence of cholera. Besides colonialism and the introduction of a cash crop economy, he also identifies the decimation of the archipelago's carabao from rinderpest (cattle plague) as a major contributor to human diseases. While each chapter explains the specific illness in terms of its causes and results, the author does an extraordinary job in presenting disease as part of a complex web that was inextricably linked to a changing economy and the harsh warfare from 1896 and 1902.

*Agents of Apocalypse*, which analyzes a demographic crisis on multiple levels, including the health of cattle, the movement of peoples as determined by war and markets, the importation of disease, the role of malnutrition and the bungling of imperial managers, sets a new standard for depth of investigation about the place of disease in society. This book clearly demonstrates that epidemiological questions are not solely relegated to medicine, but are intertwined with ecology, biology and history. As such, it serves as a useful model in studying the interactions between disease, environment and society in past populations.

## Disease Ecology

During the twentieth century, disease ecology emerged as a distinct discipline within infectious disease research. Key figures were Theobald Smith, F. Macfarlane Burnet, René Dubos, and Frank Fenner. They all drew on Darwinian evolutionism to fashion an integrative (but rarely holistic) understanding of disease processes, distinguishing themselves from others in the field. They sought a more complex, biologically informed epidemiology, as their emphasis on competition and mutualism in the natural environment differed from the physical determinism that prevailed in much of the scholarship in both medical geography and environmental health research. The science of disease ecology was derived, in part, from studies of the interaction of organisms, both microscopic and macroscopic, in tropical medicine, veterinary pathology, and immunology. Once a minority interest, disease ecology has attracted more attention since the 1980s for its explanations of disease emergence, antibiotic resistance, bioterrorism, and the health impacts of climate change.[75]

Any discussion of the relationship between environment and health must first distinguish between the assertion of an "environmental" perspective in medical science from an earlier concern with medical geography, and the role of the physical milieu in causing disease. The work of Burnet, in particular, was structured more around a biologically mediated environment, and was derived, in part, from the parasitological tradition in tropical medicine and veterinary pathology – a tradition that was pre-adapted to ecological explanations of this sort, not from older

Hippocratic notions of direct environmental determinism. Unlike most medical geography, disease ecology postulated an evolutionary time scale, models that were integrative and interactive, and a global scope. In general, the spatial imagery of disease ecology was more abstract and biologically animated than medical geography, and the processes it described usually were visible only to experts. As such, the results were not readily discerned or experienced by the general public. That is, the fine pattern of microbial interaction was generally less evident than a change in season or a shift in the wind direction. But the ecological understanding of the global as a site of infectious disease emergence could nonetheless be compelling. It was, after all, Dubos who coined the slogans "Only One Earth," and "Think Globally, Act Locally." Moreover, it would be disease ecology that provided the most plausible explanation for the emergence of "new" diseases in the 1980s.[76]

Ecosystem ecology, meteorology, and environmental engineering are a few of the disciplines to explore the relationships between ecology and human disease. The recent popularity of ecological world history, as evidenced by best-selling books such as Jared Diamond's *Guns, Germs and Steel*, reveals yet another ecology at play, one in which humans and microbes are seen as equal actors in a large-scale evolutionary drama. Diamond's book is indebted to a previous generation of scholarship in environmental history, one that includes William McNeill's *Plagues and Peoples* and Alfred Crosby's *The Columbian Exchange*, which in turn borrowed heavily from the work of Macfarlane Burnet and René Dubos in constructing global ecological narratives.[77]

While Burnet and Dubos are well known for their work on the relationships between environment and health, it was Theobald Smith, a comparative pathologist at Harvard, who became the major advocate of the study of disease as a general biological problem. In an address at the 1904 St. Louis Universal Exposition, he had claimed that the "social and industrial movement of the human race is continually leading to disturbances of equilibrium in nature, one of whose direct or indirect manifestations is augmentation of disease."[78] He described health and disease as consequences of a struggle for existence between living things, both predatory and parasitic. He once wrote that "parasitism may be regarded not as a pathological manifestation, but as a normal condition having its roots in the interdependence of all living organisms."[79] His was a naturalistic and evolutionary understanding of the interactions of organisms, both microscopic and macroscopic, in which human disease was de-centered and the environment, or milieu, became animated.[80] According to Smith, "all that can be postulated is the universal struggle of living things to survive, and in this struggle the fundamental biological reactions gradually range themselves by natural selection under…categories of offense and defense." The environment that mattered most was alive, and any effect of climate on topography would be mediated through the interactions of organisms. Smith reflected on the difference between his dynamic modeling and the work of earlier medical geographers:

> Sanitarians looked to the variations in atmospheric moisture and temperature, the rise and fall of the water in subsoil, great fluctuations in temperature, as favoring causes of epidemics. Today we are inclined to narrow them down to the human and animal world, their intercourse, migrations, the continual

> fluctuations in habits and modes of life, but especially in the increasing susceptibility of populations during the disease-free periods.[81]

Such studies in the biological complexities of host-parasite interaction soon reached a more popular readership who wanted to learn about the latest scientific theories on the place of people in nature. In *Rats, Lice, and History* for example, Hans Zinsser recounted the "biography" of typhus fever, tracing the impact of the disease on the rise and fall of civilization. A similar view could be seen in Percy Ashburn's *The Ranks of Death*. Influenced by Zinsser, Ashburn evoked European migration to the Western Hemisphere as "the greatest mobilization of disease…the most striking example of the influence of disease upon history..." He argued that the native peoples of the Americas had little immunity to the diseases Europeans brought with them. As a result, the preexisting biological equilibrium was upset, and the consequences were devastating.[82]

Influenced by the work of Smith and Zinsser, Macfarland Burnet began to write the *Biological Aspects of Infectious Disease* in 1937, a book designed for the non-specialist reader. He regarded his work as a combination of epidemiology and immunology viewed from a wide biological perspective. Examining a number of common diseases, he sought to provide an evolutionary explanation of the relations between human populations and their parasites. Infectious disease was, he claimed, nothing more or less than "a manifestation of the interaction of living things" in a changing environment. Processes such as migration, urbanization, and general population increase would lead to redistribution of old diseases and the emergence of

"new" diseases. "Wars, internal and external, financial depressions and labor troubles," he wrote, "are all breeders of infectious disease, and the future of disease will depend on the essentially fortuitous circumstances which will let loose or withhold these calamities."[83]

Dubos began to formulate a more complex biological account of host-parasite relationships in the 1940s, drawing on Theobald Smith's previous work and perhaps on Burnet's recent monograph. Although a microbiologist by training, Dubos was gradually moving away from laboratory investigation and refashioning himself as a popular writer and commentator. In a series of books, he would argue for a more integrative social and biological understanding of human disease. The first of these was *The White Plague: Tuberculosis, Man, and Society*[84] which describes the impact of poverty and war on the incidence of this "social disease." He emphasized throughout the book that "the states of health and disease are the expressions of the success or failure of the organism in its effort to respond adaptively to environmental challenges." Like Burnet, he believed "organismic and environmental biology" needed as much attention as "physiochemical biology." He argued that "in comparison with the enormous effort devoted to the components of the body machine, living as a process has hardly been studied by scientific methods."[85] In the *Mirage of Health* and *Man Adapting*, he sought to explore "the complex inter-relationships between man and his physical, chemical and biological environment."[86] Many of his arguments would have been familiar to readers of Burnet's work. Such themes as the "interplay" between organisms reaching an equilibrium, the balance

between parasitism and predation, the determinants of bacterial virulence and host resistance, the impact of increasing population density, and the general evolution of microbial diseases clearly characterized the writings of Burnet. Unlike Burnet, however, Dubos would increasingly focus on the direct physical influences on human health, referring back to Hippocrates and medical geography and pointing specifically to the dangers of environmental pollution.[87]

Dubos continued in much the same manner throughout the 1960s and the 1970s. In 1968, he warned, "[M]an will ultimately destroy himself if he thoughtlessly eliminates the organisms that constitute essential links in the complex and delicate web of life of which he is a part." Increasingly, he represented himself as an heir of the Hippocratic tradition. He stated that "today, as in Hippocrates' time, good medical care implies attention not only to the body but to the whole person and to his total environment."[88] Although he was now calling attention to his "holistic" understanding of disease, Dubos still argued that this general conceptual framework had to be informed by precise laboratory knowledge. Although he tried to resist environmental determinism and hold fast to a more interactive ecological model, he was not always successful. "All natural phenomena," he wrote, "are the result of complex inter-relationships; all manifestations of human disease are the consequences of the interplay between body, mind, and environment."[89] But increasingly, he also wanted to condemn the damage industrial capitalism was doing to "human values," counterposing radical humanism to his ecological sensibility. "Medical problems posed by the environmental stimuli and insults of modern civilization have acquired a

critical urgency," he asserted. Indeed, his main worry had become "the threat to mankind posed by technologies derived from modern physicochemical and biological sciences."[90]

Frank Fenner differed from both Burnet and Dubos in that he provided what was perhaps the most thoroughly "ecological," or least anthropocentric of perspectives on health and disease. He argued that "from the point of view of infectious diseases, the most important features of man's cultural development are the size of the individual communities of men, the number and proximity of such communities, and the extent of movement and interchange between them."[91] Fenner discussed changes in host-parasite interactions in malaria, salmonellosis, cholera, measles, smallpox, yellow fever, and poliomyelitis. He argued that although new viral diseases had been recognized during the century, most of these had been due to human intervention of some sort. Urbanization, human colonization, and air travel seemed especially problematic in the spread of disease. Additionally, Fenner advocated that the best solution to the problem of disease was "the eradication of the sources of infection by the elimination of poverty."[92]

Fenner's prediction in the 1960s of the emergence of "new" diseases in the developing world was fulfilled in the 1980s. Joshua Lederberg was among those who understood the complex role of ecology in the spread of disease. He, along with other scientists, postulated that evolutionary processes operating on a global scale were responsible for the emergence of "new" diseases. As environments changed, and as

urbanization, deforestation, and human mobility increased, so too did disease patterns. As a result, natural selection promoted the proliferation of microbes in new niches. Lederberg argued, however, that "evolutionary equilibrium favors mutualistic rather than parasitic or unilaterally destructive interactions. Natural selection, in the long run, favors host resistance, on the one hand, and temperate virulence and immunogenic masking on the parasite's part on the other." Both Lederberg and Dubos remained concerned, however, that too good a human adaptation to an increasingly degraded environment might yet be detrimental to human values. "In a biological sense, we may achieve new genomic equilibria with these radically altered environments, but the price of natural selection is so high that I doubt we would find it ethically acceptable as it conflicts violently with the nominally infinite worth we place on every individual."[93]

Amplified concern about emerging infectious diseases during the 1990s, along with fears of increasing antibiotic resistance and the health effects of climate change, would boost interest in disease ecology. Stephen S. Morse, a virologist and immunologist at the Rockefeller University, joined Lederberg in arguing that since "most 'new' or 'emerging' viruses are the result of changes in traffic patterns that give viruses new highways," we need "a science of traffic patterns, part biology and part social science."[94] Interest in the emergence of "new" diseases soon led to a proliferation of conferences and symposia, as well as giving rise to numerous reports and popular books, such as Laurie Garrett's *The Coming Plague*.[95] Additionally, the journals *Emerging Infectious Diseases* and *Ecosystem Health* were launched in the

mid-1990s. Clinicians and scientists were also coming to recognize antibiotic resistance as a growing problem, and attributed it to evolutionary processes. According to S. B. Levy, a microbiologist at Tufts, profligate antibiotic use had delivered a selection pressure on microbes "unprecedented in the history of evolution." He adds that "we must somehow find a means to reverse the ecological imbalance that has occurred in terms of resistance and susceptible strains."[96] Others saw us reaping the ecological whirlwind of climate change. Alterations in the abundance and distribution of microorganisms and their vectors might, to a large extent, mediate the influence of climate change and other physical transfigurations. Thus, as mosquitoes extended their range so, too, would malaria, dengue and other supposedly "tropical" pathogens. In *Human Frontiers, Environment and Disease*, A. J. McMichael declared that "as human intervention in the global environment and its life processes intensifies, we need better understanding of the potential consequences of these ecological disruptions for health and disease."[97]

The understanding of disease ecology that emerged at the end of the twentieth century often differed from the earlier theories proposed by Smith, Burnet, and Dubos.[98] There was a common desire among scholars, however, to assemble a more complex and integrative explanatory framework for disease patterns. Proponents of an ecological perspective on infectious diseases seek a means to relate *micro*biological processes to larger environmental or biological forces, as well as a way to describe the interactive, dynamic relationships between host, parasite and environment. Andy Dobson, an ecologist at Princeton University, noted that "when

trying to unravel the infectious diseases of plants and animals, the macroscope of the ecologist can provide just as much information as the microscope of microbiologists, veterinarians and physicians. Infectious diseases exist within an ecological context."[99] Such thinking invokes both an evolutionary time frame as well as a global compass. Consider, for example, the etiology of Lyme disease. The pathogen that transmits infection to humans is regulated by the presence of blacklegged ticks, deer populations, and the diversity of small mammals. Managing infections that have complex lifecycles, where pathogens infect multiple host species, requires an understanding of the ecological conditions that promote or inhibit disease. Since the progression of human society has ushered in unprecedented environmental changes, very few ecosystems can be considered remote or pristine.

More than a century ago Robert Koch presented his famous postulates[100] for ascertaining the cause of infection. Subsequent decades saw the discovery of many infectious microbes, including viruses. One by one, diseases and microbes were matched – and it became clear that determining the cause of disease was not simple. Today we understand that the concept of the microbe as the cause of an infection is inadequate and incomplete because it ignores the influence of the host, the milieu, and the social and physical environment. Yet western medical science still tends to focus on the microbe as the foe, and our response has often been to seek and destroy the invader. A more enlightened understanding, however, would embrace an ecological perspective – one that incorporates the social as well as the physical, chemical, and biological dimensions that characterize the interactions of people with

their natural surroundings. Humans have reached such numbers and have developed such technologies that human activities have a global impact and have changed the earth for all biological life. Humans are part of a vast evolutionary process, and all life is interdependent. For this reason, students of human health, whether examining modern populations or past populations, must look at the health and resilience of the ecosystem, as well as the health and resilience of the individual.[101]

No issue could be a more fundamental measure of sustainability than public health, and the increasing emergence and reemergence of infectious diseases globally is possibly the world's most challenging public health problem today. Yet this problem is incomprehensible without a vastly broadened research perspective, if not an entirely new paradigm.[102] The study of public health and epidemiology has been tracing outbreaks of infectious disease back to their point of origin for a long time (starting, perhaps, with a water pump in Broad Street, London).[103] But the scale of the approach is widening, and a series of new strategies to study complex disease dynamics are being adopted. For example, there is a strong zoonotic skew to emerging infectious diseases (EIDS) in humans. Some of the most significant of these have wildlife reservoir hosts (e.g., HIV/AIDS, Influenza H5N1). Still others cause outbreaks with high case fatality rates and have neither vaccine nor cure (e.g., Ebola virus, Nipah virus, SARS). In classical epidemiology, outbreaks of these diseases are traced back to their wildlife origin, and studies of human contact with wildlife undertaken. But what of the socio-ecological perspective? Take SARS, for example, a disease which has recently been identified as originating in *Rhinolophus*

spp. bats and emerging via the wildlife trade in China.[104] Understanding the process by which SARS emerged may ultimately involve studying the expansion of wildlife trade in China to determine the threshold levels that allowed sufficient contact between bats, civets, and humans to cause pathogen spill-over. It may also involve studying the anthropogenic pressures on these bats: if bats are over-collected and populations thinned, how does this affect transmission dynamics within the wildlife host, pathogen prevalence and, therefore, risk to people? The challenge to researchers here is to break down disciplinary divides between, for example, medicine and ecology, virology and wildlife biology, and sociology and epidemiology to better understand the combined ecological and social dynamics at play.[105] This ambitious goal will not be reached easily and will require science and education initiatives that cross disciplinary as well as institutional, societal, and cultural boundaries.[106]

Historians can contribute to this endeavor by studying disease in past populations where the "global" scope is smaller, the rhythm of life is slower and the variables influencing the emergence of disease are fewer. This dissertation follows the models set forth by Dubos, Burnet, Fenner and others and introduces an ecological perspective[107] to the study of disease in eighteenth-century Philadelphia. Epidemic and endemic disease on the scale experienced by this city is a metaphor for a human society out of harmony with itself and its enveloping environment. Clearly, by appreciating the complex dynamic between social, cultural and ecological processes in the emergence of disease in this eighteenth-century city, we can

potentially gain insights into the underlying causes of the recent upsurge in emerging infectious diseases today.

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[1] See Smith, 1990, p. 4-6 for a definition of the "lower sort."

[2] Matthew Carey noted that the disease was "dreadfully destructive among the poor." He added that "it is very probable that at least seven eights of the number of the dead was of that class." See Carey, 1794, *Short Account*, p. 27. His observation is supported, at least in part, by the disproportionate number of mariners, laborers, artisans, clothes washers, and prostitutes among the victims. Inferior diets, overcrowded housing, and inadequate sanitary conditions intensified the vulnerability of impoverished residents to escape the afflicted area. As one newspaper essayist recognized during a subsequent epidemic, departing the city was impossible for "the poor who have neither places to remove to or funds for their support, as they depend on their daily labour, for daily supplies." See "A Useful Hint," *Mercury Daily Advertiser*, August 19, 1797. The small red flags that, by order of the Board of Health, adorned the doors of houses containing people infected by the fever proliferated in the alleys and lanes occupied by poorer Philadelphians in the city's center. Nearly a third of the residents of Moravian Alley, for example, and half of those in Fetter Lane succumbed to the disease in 1793. Jacob Flake, a tailor living at the end of Moravian Alley, lost six children to the pestilence; "whole families," according to Carey, sank "into one silent, undistinguished grave." See Carey, 1794, *Short Account*, p. 27. Yellow fever deaths in streets, alleys, and lanes are recorded in Hagan,*The Prospect of Philadelphia and Check on the Next Directory,* 1795. For a discussion on the class specificity of the yellow fever virus, see Smith, 1997, p. 150-1. It is important to note, however, that the geography of eighteenth-century Philadelphia was relatively small compared to the modern city. Moravian Alley, for example, was just one block west of Drinker Alley - the home of Elizabeth Drinker, a member of the "better sort." Socio-economic status had less to do with the spread of yellow fever than did the range of flight of the *Aedes aegypti* mosquito; the vector of yellow fever. Note: a vector is any person, animal or microorganism that carries and transmits disease. Mosquitoes, for example, are vectors of malaria and yellow fever, carrying disease-producing parasites. See Rothenberg et al, 2000, p. 580. Many factors influence the burden of infectious disease, but environmental factors change the distribution and impact of infections through a variety of mechanisms. The environment influences the pathogen – its survival, abundance, and dispersal. It affects whether vectors, the agents such as mosquitoes and ticks that carry pathogens from one host to another, can survive in a particular geographic area. The temperature influences whether a pathogen, such as the malaria parasite, inside an insect vector has time to develop to a stage that can be infective for humans. Likewise the environment influences the presence and abundance of intermediate and reservoir hosts. Types of vegetation, also shaped by environmental factors, affect animals and insect populations. The environment also influences the activities and behaviors of humans, and their nutritional status. See Wilson, 2000, p. 7. As a result, socio-economic status, although an important factor in disease susceptibility, is just one of many forces influencing the emergence and spread of infectious diseases.

[3]The Christ Church bills of mortality originally were scattered in repositories including the Huntington Library, the National Library of Medicine, the Library Company of Philadelphia, and the American Philosophical Library, with summaries of most of the missing bills eventually located in newspapers, magazines, city directories, and among the papers of Noah Webster. The statistics of births and deaths in Philadelphia published annually by Zachariah Poulson in the first thirteen issues of his *Town and Country Almanac* (1789-1802) are among the first to be collected systematically after the American Revolution. They provide an unusually detailed record of public health during a decade of dramatic contrasts. See Klepp, "Zachariah Poulson's Bills of Mortality, 1788-1801," in Billy G. Smith, ed., *Life in Early Philadelphia*, 1995, p. 219-220. Poulson's *Almanacs* for 1789 through 1800 are at the Presbyterian Historical Society, Philadelphia. The *Almanacs* for 1801 through 1807 are at the Historical Society of Pennsylvania, Philadelphia. These and all other surviving bills of mortality have been reprinted in Susan E. Klepp, 1991, "*The Swift Progress of Population": A Documentary and Bibliographic Study of Philadelphia's Growth, 1642-1859*.

[4] Crane, 1991, *The Diary of Elizabeth Drinker*, 3 Vols.

[5] Elizabeth Drinker noted that the fever predominated in the poorer sections of town in the early days of the yellow fever epidemic of 1793. As the disease continued to rage throughout the city, however, virtually every neighborhood was affected. Drinker knew many of those who succumbed to the disease, and made particular note of those who died in her own neighborhood. She also observed that other diseases were rampant in the city at this time as well. On August 23rd, she noted that her husband and son "have something of the influensia, which great numbers have at present in Town and Country." See Crane, 1991, p. 495, 508. Excerpts from the *Diary of Elizabeth Drinker*, August 23, 1793 and September 23, 1793.
Drinker's concern with demography and "numeracy" (the collection, use and understanding of factual numerical data) was a hallmark of her reading and writing. Her interest first became evident during the Seven Years War when smallpox once again became epidemic in Philadelphia. She read both medical self-help books and compilations of weather, epidemics and statistics. She also created her own lists and statistics. An avid reader and buyer of books, she owned William Buchan's *Domestic Medicine*, and read Erastus Darwin's *Zoonomia*, and Noah Webster's *A Brief History of Epidemic and Pestilential Diseases*. She copied newspaper statistics on yellow fever and kept track of the weather. Her interest in smallpox and inoculation allows us to view western medicine's first successful attempt to combat this disease. Crane, 1991, p. 880, 890, 895, 1377-8, 1559. Excerpts from the *Diary of Elizabeth Drinker*, January 15, 1797, February 17, 1797, March 3, 1797, January 11, 1801 and September 2, 1802. See Dine, 2001, p. 417-8.
[6] The yellow fever epidemics were certainly dramatic, but they were just one disease factor in a society that would be considered, at least by modern standards, to be in the midst of several public health crises. In 1788 and during the years between 1792 and 1799, for example, yellow fever caused 18 percent of deaths among Episcopalians, but other infectious diseases such as smallpox, whooping cough, and fevers caused 21 percent of deaths, and "consumption" and "decay" (tuberculosis) 28 percent of deaths. Convulsions, diarrhea, and other common causes of infant mortality accounted for another 21 percent of the total. See Christ Church bills of mortality, 1787-8 and 1792-1799 in Klepp, 1991, p. 78-83.
[7] For a discussion of long and short-term transitions, see Swedlund and Armelagos, 1990, p. 5.
[8] The process of living involves the interplay and integration of two ecological systems. On the one hand, the individual organism constitutes a community of interdependent parts – cells, body fluids, and tissue structures - each of which is related to the others through a complex network of balance mechanisms. On the other hand, each organism constantly reacts and competes with all the living and inanimate things with which it comes into contact. Under normal conditions the external environment changes constantly, in an unpredictable manner. In order to survive and continue to function effectively the organism must make adaptive responses to these modifications. René Dubos conceived of this entire process as taking place within a *total environment*, but he does not explicitly use the term. This term was introduced by De Bevoise in his study of the Philippines, and I use it here to describe the human-natural system dynamics of eighteenth-century Philadelphia. See Dubos, 1959, p. 110 for a discussion of the direct and indirect effects of the external environment. See De Bevoise, 1995, p. x for a discussion of the *total environment* concept in relation to his study of the Philippines.
[9] Wilson, 1995, p. 1681-4.
[10] This study takes as its model no better work than that of Ken De Bevoise's study of epidemic disease in the colonial Philippines. His study, which analyzes a demographic crisis on multiple levels, including the health of cattle, the movement of peoples as determined by war and markets, the importation of disease, the role of malnutrition, and the bungling of imperial managers, sets a new standard for depth of investigation about the place of disease in society. See De Bevoise, 1995, p. ix-xii.
[11] There are several well-known disease causation models including the line, the triangle, the wheel and the web. These models help to organize ideas about causes and about strategies to prevent and control disease. See Bhopal, 2002, p. 103-15 for a discussion of models of causation in epidemiology.
[12] The word *epidemic* is from the Greek *epi* (upon), *dēmos* (people). It is defined as the occurrence in a community or region of cases of an illness, specific health-related behavior, or other health-related events clearly in excess of normal expectancy. See Last, 2001, p. 60. Endemic disease, on the other hand, is the constant presence of a disease or infectious agent within a given geographic area or

population group. It may also refer to the usual prevalence of a given disease within such area or group. See Last, 2001, p. 59.
[13] Bhopal, 2002, p. 21.
[14] Burnet, 1940, p. 1-24.
[15] Dubos, 1959, p. 110.
[16] Sontag, 1979, p. 60.
[17] Morse, 1991, p. 387-409.
[18] Morse, 1992, p. 38.
[19] Genetic and biological factors, for example, allow microbes to change, and can make people more or less susceptible to infections. In addition, changes in the physical environment can impact on the ecology of vectors and animal reservoirs, the transmissibility of microbes, and the activities of humans that expose them to certain threats. See Smolinski, Hamburg and Lederberg, 2003, "Executive Summary," p. 2.
[20] A reservoir of infection is any person, animal, arthropod, plant, soil, or substance, or a combination of these, in which an infectious agent normally lives and multiplies, on which it depends primarily for survival, and where it reproduces itself in such a manner that it can be transmitted to a susceptible host. See Last, 2001, p. 158.
[21] Broad environmental changes included climate variation, flooding, drainage, deforestation and biodiversity loss.
[22] Wilcox and Colwell, 2005, p. 246.
[23] The Creek was described in 1760 as being a filthy uncovered sewer, bordered on either side by shabby stables and tanyards. It was so polluted by 1767 that it was declared a health menace and ordered arched and covered with fill from Third Street to Walnut Street. The Provincial Assembly of 1784, impelled by the same reasoning, ordered the remainder of the Creek covered in the same manner. For a discussion of Dock Creek and the tanning industry, see McMahon, 1994, p. 114-147.
[24] McMahon, 1994, p. 121-2.
[25] Slaughterhouses, like the one on the south side of the Dock, were traditionally located near streams, along with cattle pens. Around them, hornworms, chandlers, and soap- and glue-boiling yards made use of the slaughterhouse's by-products. But the largest users of the slaughterhouse leavings were the tanneries. These establishments included not only mills for grinding bark used in tanning but also vats and pits for soaking the hides during the stages of removing the hair, tanning the hide, and giving texture to the leather. The method of curing varied according to the intended use, whether for saddles or for women's gloves. Beyond the yard, then, the tanneries also supported a cluster of linked manufacturing activities, including shoemakers, saddlers, curriers, and glovers. See Van Wagenen, 1953, p. 182-9. Water was needed throughout the processing of the hides. Initial treatment consisted of soaking the hides in lime pits. Measuring, on the average, six feet square, lime vats sometimes were twice that size. Vats usually were wooden structures made to function like "a small water meadow slightly sunk." See Hartley, 1979, p. 254. A retaining dike wall was built around the vat, and, in rural settings, water was flooded in from the adjacent stream. Whether used in a country tanyard or in an urban yard with wells and a nearby tidal cove, the water was always returned to its source along with the various substances that had been added, including acidic liquids resulting from the refuse of cider presses, sour milk, fermented rye, and alkaline solutions made up of buttermilk and some forms of dung. The processing of the hides signified as well the strong connections between the industrial activities around the Dock and the countryside. Breweries, tanneries, slaughterhouses, and distilleries gathered the products of the countryside - animals and grain primarily - processed them, and discarded the unused by-products on the Dock's watershed. See Hartley, 1979, p. 354-5.
[26] Philadelphia experienced urbanization on a grand scale during the eighteenth century. The enormous increase in the city's population meant encroachment into the surrounding wilderness, bringing people into contact with domestic and peri-domestic hosts and vectors. Agricultural intensification in combination with the milling and tanning industries expanded the number of potential breeding sites for mosquitoes. Habitat alteration may have contributed to a hyper-abundance of potential and actual vector and reservoir species. This would have facilitated the emergence and spread of certain infectious diseases.
[27] The mosquito-transmitted arbovirus that causes yellow fever was transported to Philadelphia in the blood of immigrants from areas where yellow fever was endemic. Since infection lasts only a short

time, the virus would not have survived long sea voyages in the blood of its victims. The only way the virus could have survived such voyages was through vector mosquito stowaways on board the ships, laying their eggs in the ships' water casks, and attacking all on board.

[28] Dysentery is an intestinal inflammation caused by bacteria, protozoa, parasites, or chemical irritants. It is marked by abdominal pain, frequent, bloody stools and rectal spasms. See Rothenberg et al, 2000, p. 172.

[29] Tuberculosis is a chronic infection with the bacterium *Mycobacterium tuberculosis*, and is transmitted by inhalation or ingestion of droplets. It usually affects the lungs but may also affect other organs. See Rothenberg et al, 2000, p. 567.

[30] Klepp, 1991, "*The Swift Progress of Population*."

[31] Bridenbaugh, 1938, 1942, 1955; Dunn and Dunn, 1982; Nash, 1979; Salinger, 1995; Schultz, 1993; Smith, 1977, 1990, 1995; Soderland, 1983; Warner, 1968 and 1987; Weigley, 1982.

[32] Cotter, et al, 1992; Liggett, 1971; McMahon, 1994, 1997; Olton, 1974.

[33] Estes and Smith, 1997; Powell, 1949 and 1970; Robinson, 1993; Shannon, 1982; Taylor, 2001.

[34] Wolman, 1974.

[35] McMichael, 2001, p. 6.

[36] Elizabeth Sandwith married the up-and-coming merchant, Henry Drinker, in 1761. She formed a household that included her sister, who remained unmarried throughout her life. Of the nine children to whom Elizabeth gave birth, five survived to adulthood, and four outlived their mother. The description of the diary in the main body of the text is from Crane, 2001, p. 408-9.

[37] Klepp, 1991, p. 10.

[38] Webster, 1799, p. 6-9.

[39] Konkle, 1977, p. 287-9.

[40] Wolman, 1974.

[41] Nash, 1973, p. 223-56. Klepp argues in a later study that enslaved Africans faced substantially higher risk of death in Philadelphia than did free or dependent whites. See Klepp, 1994, "Seasoning and Society: Racial Differences in Mortality in Eighteenth-Century Philadelphia," p. 473-506.

[42] Smith, 1977, p. 863-83; Smith, 1989, p. 328-32; Smith, 1990.

[43] Klepp, 1991, p. 9.

[44] While no bills of mortality were kept in the first decades of the existence of Philadelphia, several sources allow the reconstruction of approximate death rates for the early city. The longest series is the annual number of inventories of estates which began with the founding of the town in 1682. See Ruth Matzkin, 1959, "Inventories of Estates in Philadelphia County, 1682-1710," p. 98. In 1688, the Society of Friends began to record the deaths of members, and between 1692 and 1731 the Friends recorded the deaths of "such as are not friends." See "An account of the Burialls of such as are not friends within the Town of Philadelphia – Taken and Recorded by me William Hudson for the satisfaction of all people whatever their perswation or profession may be." See Philadelphia Monthly Meeting Records, Mss. reaction, p. 412ff.

The inventories of estates reflected most movements in the death rate, but were biased by the fact that only the deaths of propertied adults are included. Other lists of adult deaths, such as that compiled by Gary Nash for merchants or the lists of deaths of Public Friends in Don Yoder et al showed the same pattern caused by the exclusion of children. See Nash, 1986, p. 337-53 and Yoder et al, 1983. Smallpox epidemics, like that of 1701, would not be reflected in these records since most adults likely had immunity to the disease. Other diseases common to children such as dysentery and worms would be missed as well. Conditions affecting the health of the propertyless, the poor, and most servants could not be recovered from these lists, and no account of black deaths can be found before 1722. The inventories of estates are an imperfect guide to mortality conditions but are the only source for the first few years of settlement. See Klepp, 1991, p. 5.

[45] Klepp, 1991, p. 10.

[46] See Vinovskis, ed., 1979, *Studies in American Historical Demography*; Wells, 1975, *The Population of the British Colonies in America before 1776*; Yasuba, 1962, *Birth Rates of the White Population in the United States*. For estimates of Philadelphia's population from 1700 to 1790, see Alexander, 1974, "The Philadelphia Numbers Game: An Analysis of Philadelphia's Eighteenth-Century Population." For estimates of Philadelphia's population from 1693 to 1790, see Nash and Smith, 1975, "The Population of Eighteenth-Century Philadelphia." For estimates of Philadelphia's population between

1769 and 1774, see Salinger and Wetherell, 1985, "A Note on the Population of Pre-Revolutionary Philadelphia." For estimates of Philadelphia's population growth between 1741 and 1838, see Hazard, 1838, *Facts in Relation to the Progressive Increase, Present Condition and Future Prospects, of Philadelphia*. For a discussion of marriage, fertility, and life cycle data for Jewish families in Philadelphia, New York and Charleston between 1725 and 1850, see Cohen, 1976, "Jewish Demography in the Eighteenth Century: London, the West Indies, and Early America." For s discussion of marriage, family size, longevity for top taxpayers in 1775 and 1800, see Gough, 1977, "Towards a Theory of Class and Social Conflict: A Social History of Wealthy Philadelphians, 1775 and 1800."

[47] Carey, *Short Account*, 1794, p. 27.

[48] The miasmatic theory of disease postulated that epidemic outbreaks of disease were caused by the state of the atmosphere. See Rosen, 1958, reprint 1993, p. 264.

[49] See Rush, 1786, "An Inquiry into the Cause of the Increase of Bilious and Intermitting Fevers in Pennsylvania, with Hints at Preventing Them," paper read before the American Philosophical Society on December 16, 1785.

[50] For a discussion of physicians' responses to this event, see Bridenbaugh, 1942, *Rebels and Gentlemen*, p. 295, and chapter 11. Bridenbaugh sites Bond's "Essay on the Utility of Clinical Lectures," delivered in Philadelphia on November 6, 1766.

[51] Warner, 1987, p. 9.

[52] Wilcox and Colwell, 2005, p. 244.

[53] Wilson, 1995, p. 1681-4.

[54] One of the few historical studies to actually break down disciplinary divides is Ken De Bevoise's study of epidemic disease in the Philippines during the late nineteenth century. His work, which analyzes a demographic crisis on multiple levels, including the health of cattle, the movement of peoples as determined by war and markets, the importation of diseases and the accompanying environmental disruption sets a new standard for both synthesis and depth of investigation. See De Bevoise, 1995.

[55] On environmental justice and racism in an American context, see Robert Bullard,1990, *Dumping on Dixie: Race, Class, and Environmental Quality*; Giovanni Di Chiro, 1996, "Nature as Community: The Convergence of Environment and Social Justice," in *Uncommon Ground: Rethinking the Human Place in Nature*, William Cronon, ed., p. 298-321; Michael Egan, 2002, "Subaltern Environmentalism in the United States: A Historiographic Review," *Environment and History* 8, p. 21-41; Daniel Faber, ed., 1998, *The Struggle for Ecological Democracy: Environmental Justice Movements in the United States*; Robert Gottlieb, 1993, *Forcing the Spring: The Transformation of the Environmental Movement*; Dolores Greenberg, 2000, "Reconstructing Race and Protest: Environmental Justice in New York City," *Environmental History* 5, p. 223-50; George Lipsitz, 1995, "The Progressive Investment in Whiteness," *American Quarterly* 47, p. 369-466; Eileen Maura McGurty, 1997, "From NIMBY to Civil Rights: The Origins of the Environmental Justice Movement," *Environmental History* 3, p. 301-23; and Laura Pulido, 1996, *Environmentalism and Economic Justice: Two Chicano Struggles in the Southwes*t.

[56] See Martin Melosi, 2000, *The Sanitary City: Urban Infrastructure in America from Colonial Times to the Present*; Joel Tarr, 1996, *The Search for the Ultimate Sink*; Arthur McEvoy, 1995, "Working Environments: An Ecological Approach to Industrial Health History," *Technology and Culture* 36 (suppl), p. 145-73; Christopher Sellers, 1997, *Hazards on the Job: From Industrial Disease to Environmental Health Science*.

[57] See Andrew Hurley, 1995, *Environmental Inequalities: Class, Race, and Industrial Pollution in Gary, Indiana, 1945-1980*; Craig Colten and Peter N. Skinner, 1996, *The Road to Love Canal*.

[58] The founding text for medical perceptions of the role of the environment in health and disease is part of the Hippocratic Corpus (fifth century). *Airs, Waters, Places* is believed to have been written to enable the Greek peripatetic physicians to anticipate what diseases they were likely to encounter when beginning practice in a new, unfamiliar town. Prognosis and the decision of whether to treat patients were important considerations, and a careful analysis of all factors assisting in this process was requisite for making sound judgments. The title of the work indicates the components of the environment to which it was judged most attention should be paid. *Airs* referred to winds and climatic effects according to season; *Waters* included both spring waters from the ground, and water from rain

and snow; *Places* referred to the location of the town and the character of the site. See Hannaway, 1993, p. 293.

[59] Mitman, Murphy and Sellers, 2004, p. 9. For an exploration of the ways in which notions of community ecology played into twentieth-century biomedicine, see Gregg Mitman, 2003, "Natural History and the Clinic: The Regional Ecology of Allergy in America," *Studies in the History and Philosophy of the Biological and Biomedical Sciences* 34, p. 491-510. See Aldo Leopold, 1949, *A Sand County Almanac*. On the persistence of aerial concerns, see Christopher Sellers, 2003, "The Dearth of the Clinic: Lead, Air, and Agency in Twentieth-Century America," *Journal of the History Medicine and Allied Sciences* 58, p. 255-91.

[60] Wilson, 1995, 2000, 2003; Wilcox and Colwell, 2005.

[61] Mitman, Murphy and Sellers, 2004, p. 8-9.

[62] Overviews of environmental history include Donald Worster, Alfred Crosby, Richard White et al, 1990, "A Roundtable: Environmental History," *Journal of American History* 76, p. 1087-147; Alfred Crosby, 1995, "The Past and Present of Environmental History," *American Historical Review* 100, p. 1177-89; Theodore Steinberg, 2002, "Down to Earth: Nature, Agency, and Power in History," *American Historical Review* 107, p. 798-820; and Richard White, 2001, "Environmental History: Watching a Historical Field Mature," *Pacific Historical Review* 70, p. 103-12.

[63] See Robin Kearns and Wilbert Gesler, 1998, *Putting Health into Place: Landscape, Identity, and Well-Being*; Crosbie Smith and Jon Ager, 1998, *Making Space for Science: Territorial Themes in the Shaping of Knowledge*; Adi Ophir, Steven Shapin, and Simon Schaffer, eds., 1991, "The Place of Knowledge: The Spatial Setting and Its Relation to the Production of Knowledge," *Science in Context* 4, p. 3-218; David Livingstone, 1995 "The Spaces of Knowledge: Contributions Toward a Historical Geography of Science," *Environment and Planning D: Society and Space* 13, p. 5-34; Nicholas Rupke, ed., 2000, *Medical Geography and Historical Perspective*; Gregg Mitman, 2003, "Hay Fever Holiday: Health, Leisure, and Place in Gilded America," *Bulletin of the History of Medicine* 77, p. 600-35; Susan Craddock, 2000, *City of Plagues: Disease, Poverty, and Deviance in San Francisco*; Isabel Dyck, Nancy Davis Lewis, and Sarah McLafferty, eds., 2001, *Geographies of Women's Health*.

[64] Hurley, 1997, p. 11.

[65] Merchant, 2002, p. xiii-xiv.

[66] Hurley, 1997, p. 1.

[67] Hurley, 1997, p 1-3.

[68] Turner, 1920.

[69] Cronon, 1996, p. 89.

[70] Merchant, 2002, p. xv.

[71] Most of the book was finished by 1937.

[72] Crosby, 1972, p. xiii, 219.

[73] It was Smith, a comparative pathologist at Harvard, who became the major advocate of the study of disease as a general biological problem. He was the co-discoverer of the role of a tick in transmitting the parasite that causes Texas cattle fever, and was fond of emphasizing the mutual dependence of host and microorganism, whether in health or disease. Hans Zinsser, in *Rats, Lice, and History*, recounted the "biography" of typhus fever, tracing the impact of the disease on the rise and fall of civilizations. See Anderson, 2004, p. 44-6 for a discussion.

[74] McNeill, 1976, p. 254, 257; Anderson, 2004, p. 46-7.

[75] Anderson, 2004, p. 39.

[76] Anderson, 2004, p. 42.

[77] Mitman, Murphy and Sellers, 2004, p. 9.

[78] Smith, 1904, p. 817-32. Quoted material is on p. 817.

[79] Smith, 1934, p. viii, x.

[80] Anderson, 2004, p. 44-5.

[81] Smith, 1934, p. 162.

[82] Ashburn, 1947, p. 5; Anderson, 2004, p. 46.

[83] Burnet, 1940, p. 4, 307; Anderson, 2004, p. 49.

[84] Dubos and Dubos 1952; reprinted New Brunswick, NJ, 1987.

[85] Dubos, 1965, p. xvii, xix, xx, 333.

[86] Dubos, 1965, p. xxi. See Dubos, *Mirage of Health*, 1959.

[87] Anderson, 2004, p. 53.
[88] Dubos, 1968, p. 9, 61.
[89] Dubos, 1968, p. 61.
[90] Dubos, 1968, p. 88, 111. For a discussion, see Anderson, 2004, p. 53-4.
[91] Fenner, 1970, p. 48-76. Quoted material is on p. 48.
[92] Fenner, 1970, p. 63, 66. Quoted material is on p. 63; Anderson, 2004, p. 57-8.
[93] Lederberg, 1993, p. 4, 8.
[94] Morse, 1991, p. 387-409. Quoted material is on p. 388, 404.
[95] See Garrett, 1994, *The Coming Plague: Newly Emerging Diseases in a World Out of Balance*.
[96] Levy, 1997, p. 1-14. Quoted material is on p. 2, 8.
[97] McMichael, 2001, p. xiv.
[98] Anderson, 2004, p. 61.
[99] Institute of Ecosystem Studies, Press Release, May 7, 2005, Millbrook, NY.
[100] First formulated by F.G. Henle and adapted by Robert Koch in 1877, with elaborations in 1882. Koch stated that these postulates should be met before a causative relationship can be accepted between a particular bacterial parasite or disease agent and the disease in question. First, the agent must be shown to be present in every case of the disease by isolation in pure culture. Second, the agent must not be found in cases of other disease. Third, once isolated, the agent must be capable of reproducing the disease in experimental animals. And fourth, the agent must be recovered from the experimental disease produced. See Last, 2001, p. 84.
[101] Wilson, 1995, p. 1681-4.
[102] Wilcox and Colwell, 2005, p. 254.
[103] John Snow mapped the occurrence of cholera cases in the streets of London from August 19 to September 30, 1854. He also marked the positions of the local water pumps. Snow deduced that water from the Broad Street pump was the source of cholera.
[104] Li et al, 2005, p. 676-9.
[105] Daszak, 2005, p. 239.
[106] See Kaneshiro et al, 2005, p. 349-60.
[107] An ecological perspective enables us to describe the dynamic relationships that existed between the people of Philadelphia, the microorganisms that shared their world and the physical environment that surrounded them.

## CHAPTER 2

## INTRODUCTION: DIMENSIONS OF THE CRISES

Public health in eighteenth-century Philadelphia was influenced by a variety of factors including the introduction of new pathogens, environmental modification and demographic changes brought about by contact with outside groups. Disease was widespread and mortality levels were consistently high. Smallpox, yellow fever and measles were three of the most prevalent epidemic diseases to affect residents,[1] while tuberculosis, malaria and dysentery were endemic[2] in the city, and posed a constant threat to public health. For disease to affect so many people in the community at the same time, there was a shift in the factors that formed Philadelphia's *total environment*. Elizabeth Drinker and others saw disease and epidemics as a routine part of life. Illness was a constant presence in the Drinker household, and the pages of her diary reflect this situation. While disease was certainly a part of the fabric of life in all of the colonies in the eighteenth century, the situation in Philadelphia was particularly dire and the public health crises were quite real. With the exception of the yellow fever epidemics of the 1790s, scholars have tended to overlook these crises for a variety of reasons. First, the demographics show that the population of Philadelphia grew significantly throughout the century. Between 1750 and 1800, for example, Philadelphia's population multiplied more than fivefold, a growth rate

averaging an extremely high 3.4 percent each year.[3] But an overall population increase can hide suffering and death on a monumental scale. Second, attention has long been preempted by the social and political events that so characterized eighteenth-century Philadelphia. The fact that numerous public health crises coincided with such pivotal events is all the more reason to examine them closely. This chapter provides a brief overview of the disease environment of the city between 1690 and 1807.

Pennsylvania had an inauspicious beginning in the 1680s, as floods, crop failures, severe winters, and epidemic diseases began pushing up the crude death rates[4] after the first decade of settlement. Philadelphia began experiencing high rates of mortality in the last years of the seventeenth century.[5] Although the early estimates of mortality are imperfect since they are based on incomplete records, they hint at a possible reason for the absence of official comment on health conditions in early Pennsylvania. The generally favorable public image of the colony might well have been eroded by public knowledge of a precarious demographic situation. For example, many of Benjamin Franklin's most familiar comments on the favorable demographic characteristics of the colonies were made in a pamphlet aimed at convincing England to remove restrictions on colonial expansion.[6] Overly optimistic claims concerning the health and welfare of colonial cities also were made with pragmatic economic considerations in mind, for unhealthy conditions discouraged business and trade. Franklin may well have ceased publishing Philadelphia's

mortality statistics in his newspaper because of his advertisers' concern for the possible adverse effects on Philadelphia's economic affairs.[7]

Numerous references to disease fill the annual bills of mortality after 1720. Fever, flux and fits, along with purging, vomiting, consumption and pleurisy are listed consistently, while smallpox, measles, yellow fever, diphtheria and whooping cough appear during epidemic years. Merging of previously isolated disease pools brought sickness and death to many in the early decades of the century, and illness and epidemics became a routine part of life. Many in the city were undernourished, debilitated and riddled with infection. Although disease classification was often ambiguous in the eighteenth century, some diseases could be identified with some certainty. These included dysentery, smallpox, yellow fever, tuberculosis, measles, whooping cough, and scarlet fever. And, there is little doubt that yellow fever and smallpox were implicated in more deaths than other diseases. Smallpox, recurring every few years, posed a significant danger to Philadelphians during the first three-quarters of the eighteenth century. The "summer complaints" and "fall agues" (most likely dysentery and malaria) visited each year, and the peak influx of immigrants during these seasons helped spread these and other diseases throughout the city. Typhus, a common shipboard affliction, may have been one of the most prevalent fevers in the urban center, although tuberculosis and typhoid fever likewise contributed to high mortality. Yellow fever was a dramatic killer in the 1790s, but other diseases were as deadly. Between 1789 and 1801, yellow fever caused the deaths of 18 percent of Episcopalian decedents, while tuberculosis caused 24 percent

of deaths. Other infectious diseases, including smallpox, whooping cough, measles, diphtheria, and vaguely defined "fevers" accounted for 22 percent of deaths. Convulsions, diarrhea, and other common causes of infant deaths accounted for 18 percent of the total. Deaths of women in childbirth, which are now only 0.02 percent of all deaths in America, comprised about 1.5 percent of the deaths.[8] Venereal disease also ran rampant through the population, killing a great many Philadelphians after years of suffering. A variety of other disorders also took their toll. These included diphtheria, scarlet fever, influenza, malaria and pneumonia.[9] Helminthic (parasitic worms) infections[10] were quite common as well. These infections were serious, and they predisposed individuals to other maladies.

All the colonies experienced high mortality rates at the beginning of settlement. Death rates in Philadelphia, however, remained high throughout much of the eighteenth century **(See Table 2.1)**. The city experienced very high mortality rates between 1690 and 1720, with the death rate exceeding the birth rate. As trade, commerce and immigration introduced recurrent waves of infectious disease, Philadelphia's mortality rate exceeded that of Boston between the years 1720 and 1760. This pattern of frequent epidemics would only begin to moderate in the 1760s and would not disappear until the nineteenth century.

**Table 2.1**
**Comparative Crude Death Rates**
**Philadelphia and Boston, 1700-1770**

| Date | Philadelphia | Boston |
|---|---|---|
| 1700-9 | 42 | 36 |
| 1710-9 | 42 | 38 |
| 1720-9 | 44 | 45 |
| 1730-9 | 47 | 34 |
| 1740-9 | 47 | 39 |
| 1750-9 | 51 | 37 |
| 1760-9 | 46 | 32 |

Sources: Rates for Boston are from John B. Blake, 1959, *Public Health in the Town of Boston, 1630-1822*, p. 247-9. Rates for Philadelphia are from Susan E. Klepp, 1989, "Demography in Early Philadelphia, 1690-1860," p. 104-5.

Few people had more opportunity to observe the course of public health in Philadelphia than Elizabeth Sandwith Drinker (1735-1807). Although her life was not fully representative of the eighteenth century, as women of Drinker's class represented only a small percentage of the population, the extraordinary span and sustained quality of her diary make it a useful document for a variety of historical endeavors. The wealth of information contained in the diary about the ailments in Drinker's family and the remedies that she and local physicians employed make it a particularly fertile source of information on the disease history of the city, as well as the history of drugs and pharmaceuticals in eighteenth-century America.[11]

Drinker was a faithful diarist, and chronicled nearly continuously from 1758 to 1807. Even in the early years of the diary, physicians, medical practitioners, epidemics, and medical procedures played significant roles in her young life. Before her marriage in 1761, she makes known an interest in smallpox, and the preventative measures adopted by physicians to curb its mortality and curtail its spread. Always

the keen observer, Drinker watched the inoculation of James Steel, son of her friend Henry Steel on September 13, 1759. Two months later, she visited her friends Francis and Rebecca Warner Rawle the evening after two of their children had been inoculated for smallpox by Dr. John Redman. Between these two visits to witness inoculation, Drinker also called on Thomas Say, another Quaker friend. She wrote that Say's daughter, Becky, "lays ill, in Small Pox, which she has taken in the Natural way; and to most that take it Naturally (at this time) it proves mortal."[12]

Elizabeth Drinker's interest in this subject reveals that inoculation[13] was sufficiently novel to invite comments and spectators, and, more important, beginning to be widely practiced. She shows a discerning awareness of the differing mortality between those inoculated and those who were not. Smallpox was again on her mind at the end of 1762 when her diary entries for December consisted of newspaper stories and accounts of those who died from the disease. She included lists of those who caught it naturally and died, as well as deaths caused by a mistake made by an apothecary in the medication used to prepare children for inoculation.[14]

Drinker's chronicle details the urbanization of Philadelphia over five decades and contains a rich and detailed account of the epidemic and endemic diseases that inundated the city during this time. Her immediate concern on any given day was the status of her family's health.[15] Constant inquiries and reports containing the most intimate details fill the pages of the journal. Since Drinker, as a woman, was a primary caregiver, the diary is also a useful source for the history of drugs and the

professionalization of medicine in eighteenth-century America.[16] With such a large family, and with disease rampant in the Philadelphia of her day, it is not surprising that Elizabeth Drinker was greatly interested in matters of health and disease, filling the pages of her journal with numerous accounts of illness and medical incidents. Her words tell the story of a constant struggle to keep well, not only for her immediate family and friends, but for acquaintances and townspeople alike. Even if Drinker exaggerated her own disabilities, (and there is no evidence that she did) the dairy offers convincing evidence that physical distress invaded life in Philadelphia to a degree currently unimaginable.[17]

Elizabeth Drinker was not alone in her assessment of public health in the city. The staggering amount of sickness is confirmed in one diary after another, and even mundane details take on greater significance when they are mentioned by more than one person.[18] Jacob Hiltzheimer of Philadelphia, in a diary of the same period, inveighs against a man who had cleaned out a cesspool and then, during the night, emptied the contents of his cart into the street. The cesspools of the day presented a continuous menace to health.[19] Benjamin Rush noted that the city's high death rates were often confined to the "narrow streets, courts and alleys" of the city and southern suburbs.[20] Medical theory of the day blamed the lack of ventilation as the cause of contagious illness. Trapped "miasmal vapors and pestilential airs" were considered to be the cause of many diseases. A more likely scenario would be that the population density in these "narrow streets, courts and alleys" significantly increased the

probability of contact between susceptible groups of people and infectious agents **(See Table 2.2)**.

Rush also made numerous references to climate and its influence on health. Eighteenth-century Philadelphia was centered on the low-lying plain along the banks of the Delaware River. The climate was marshy, hot and humid in the summer, and cold and humid in the winter. To the south, near the confluence of the Delaware and Schuylkill Rivers, was meadow land subject to frequent flooding. Rush noted that these conditions helped to support a large population of flies, mosquitoes and rats that plagued the city and its inhabitants.[21] In addition to the damp environment, ignorance of sanitation made Philadelphia's urban environment[22] particularly unhealthy. The high water-table under the city meant that the wastes in the privies constantly seeped into the hundreds of private wells that supplied the population with water. Intestinal complaints were endemic in the city as a result, with widespread dysentery infections, especially during the summer months. The streams that ran through the city were little more than open sewers where waste of all types was dumped. These streams flowed into the Delaware River, where the docks stretching out into the water prevented the current from sweeping away the pollution deposited there and allowed pools of stagnant water to become ideal breeding grounds for mosquitoes. The clearing of forests for firewood resulted in additional marshy ground around the city so that the suburbs became noted for their unhealthiness in the summer.[23]

**Table 2.2[24]**
**Population Density of Greater Philadelphia, 1790**

| | A | B | C | D | E | F | G | H | I | J | K | L | M | N | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **8** | | | | | | | 37 | 184 | 213 | 176 | | | | | **8** |
| **7** | | | | | | 36 | 130 | 230 | 430 | 116 | | | | | **7** |
| **6** | | | | | | 117 | 86 | 695 | 941 | 451 | 38 | | | | **6** |
| **5** | | | | 44 | 110 | 647 | 378 | 634 | 588 | 687 | 401 | 72 | 18 | 20 | **5** |
| **4** | | | 1115 | 743 | 843 | 652 | 903 | 1229 | 1131 | 1453 | 738 | 230 | 328 | 173 | **4** |
| **3** | | 346 | 509 | 758 | 883 | 1080 | 1405 | 1411 | 1211 | 1052 | 1240 | 354 | 718 | 293 | **3** |
| **2** | 99 | 620 | 776 | 933 | 861 | 619 | 880 | 885 | 1029 | 1038 | 913 | 399 | 514 | 123 | **2** |
| **1** | 107 | 202 | 49 | | | | | | | | | 32 | 24 | | **1** |
| | | | | | | | | | | | | | | | |

**Southwark** **Philadelphia City** **N. Liberties**

## Delaware River

Sources: Population data is from the Federal Census of 1790 and Philadelphia City Directory of 1791. Table is from Mary E. Schweitzer, 1993, "The Spatial Organization of Federalist Philadelphia, 1790" p. 32, 39.

Note: Map of Philadelphia, 1794 with superimposed grids to correspond to city blocks. The Table depicts population levels per city block during the early years of the 1790s. North-south grids are labeled A-N, and east-west grids are numbered 1-8. Philadelphia City is bordered by South Street to the south, Vine Street to the north, 10th Street to the west, and the Delaware River to the east.

Elizabeth Drinker's eye-witness accounts are compelling testimony to the sickly state of the city. Additionally, there exists a great deal of demographic evidence that supports this premise, the most extensive of which are Susan E. Klepp's 1991 reconstructions of population data and compilations of bills of mortality either long inaccessible to scholars or thought lost.[25] "Vital events," particularly births and deaths, are useful for judging the health of a community, past and future rates of growth, and rates of migration. Before disruptions caused by the American Revolution, a few colonial towns, notably Boston and Philadelphia, began to publish

the annual numbers of deaths and births. The sources for the study of the city of Philadelphia's population, however, are fuller than those available for other colonial cities.

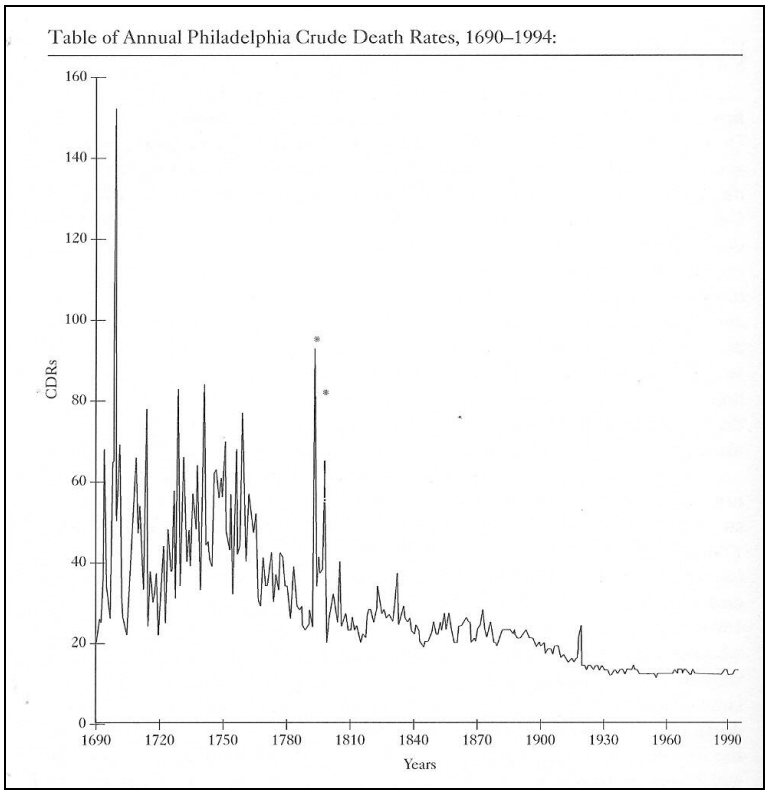
Bills of Mortality provide a basis for insights into the social, demographic, and economic characteristics of the city. Combined with population figures, the data on births and deaths can be used to compute crude birth and death rates. The crude birth rate (CBR) is a measure of fertility that calculates the births per 1,000 population, while the crude death rate (CDR) measures deaths per 1,000 population. These rates are considered crude because they do not take into account the underlying age, sex, and ethnic composition of society. The average Philadelphia CBR for the years between 1760 and 1769, for example, was 56, while the average CDR during these same years was 46 **(See Table 2.3)**. Compared with modern industrialized societies, these rates are quite high. In 2002, for example, the CBR in the state of Pennsylvania was 11.5 and the CDR was 10.5. Eighteenth-century Philadelphia had death rates that are unfavorable even by the standards of war-ravaged and famine-stricken Third World countries today. In 2004, for example, Afghanistan had a CBR of 48 and a CDR of 21, and Somalia had a CBR of 47 and a CDR of 18.[26] Philadelphia was neither war-ravaged[27] nor famine-stricken[28] during this period, yet its death rates in nonepidemic years equaled or surpassed the most dismal of modern rates.

Many in Philadelphia were chronically ill, undernourished, debilitated and riddled with infection. As a result, it was often impossible to ascertain the specific cause of death. Still, the evidence allows the general roster of diseases to be identified with some confidence, if not with anything like statistical precision. There can be little doubt that dysentery, malaria, typhoid fever and other diseases of the gastrointestinal tract, along with tuberculosis, measles, yellow fever and smallpox were implicated in many deaths. In a year when one or more were epidemic, mortality rates could soar. The highest per capita mortality in colonial Philadelphia occurred in 1759 when 77 persons died out of every thousand. Four epidemics were reported that year: smallpox, measles, whooping cough and typhus. None of these was individually the most fatal appearance of its kind but in combination raised the death rate to a level two and one half times that of the healthiest colonial year. In 1755, for example, there were no recorded epidemics, and the death rate was 30 per 1,000.[29]

Smallpox was one of the greatest killers of Philadelphians during the eighteenth century, accounting for most of the annual fluctuations in the death rate before the Revolution, and contributed to the overall downward trend in mortality after 1760 as the disease became endemic in the city.[30] The bills of mortality indicate that smallpox killed a great many Anglicans in 1737, 1751, 1756, 1759, 1762, 1763, 1765, 1769, and 1773, meaning that nearly every peak in the death rates during this forty-year period resulted from an outbreak of the disease (**See Figure 2.1**).[31] Despite efforts at containment, it was difficult to prevent the spread of smallpox to

neighboring towns and colonies with a high percentage of susceptible persons. The movement of people engaged in trade and commerce provided a convenient means of transporting the virus. Moreover, many fled from epidemic areas, despite quarantine measures, and thus facilitated the dissemination of the virus. The war with the French in the 1760s merely exacerbated the problem. Susceptible soldiers – many of whom came from areas untouched by smallpox – were infected and acted as vectors[32] upon their return to their towns and villages.[33]

**Figure 2.1**
**Annual Philadelphia Crude Death Rates, 1690-1994**




Source: Figure is from Susan E. Klepp, 1997, "Appendix I: 'How Many Precious Souls Are Fled'?: The Magnitude of the 1793 Yellow Fever Epidemic," p. 174.

Periodic smallpox and yellow fever epidemics tended to overshadow other diseases that played a much more significant role in shaping population development and increasing risks to life during the eighteenth century. Certain endemic diseases such as dysentery and malaria took a far greater toll on human life, even though their constant presence tended to reduce public fear.[34] Although yellow fever epidemics were particularly spectacular, they were but one disease factor in a society that would be considered, at least by modern standards, in the midst of several epidemiological crises. The decade of the 1790s was not the worst in the city's history despite yellow fever outbreaks in 1793, 1794-5, 1797, 1798 and 1799. From 1690 to 1759, deaths exceeded births, and decadal death rates, bolstered by a number of epidemics of smallpox, measles, typhus, typhoid fever, scarlet fever, whooping cough, diphtheria, influenza, yellow fever and other diseases, surpassed the rates of the 1790s. In the 1790s, the mean crude death rate was approximately 40 per thousand population. But crude death rates had averaged 46 per thousand for the entire 70-year period lasting from 1690 to 1759. Two of those decades experienced average death rates above 50 per 1,000 – the 1690s and the 1750s.[35]

Susan E. Klepp's reconstructed crude birth and death rates for Philadelphia, 1690-1810 **(See Table 2.3)** show that the demographic history of the city falls into three broad periods. The first period between, lasting from 1690 to 1720, is an era marked by rather low crude birth rates and high death rates resulting in negative natural increase. The period between 1720 and 1759 is characterized by high crude birth and death rates, with fewer than 500 births above the 20,000 deaths. After 1760

and continuing until early in the nineteenth century, declining mortality produced a positive rate of natural increase even with the high mortality of the yellow fever epidemics of the 1790s. Fertility, usually high in the eighteenth century, but responsive to war and peace, boom and depression, began to decline rapidly after the 1820s, and ushered in the fourth phase of Philadelphia's development: a period when birth and death rates move toward convergence at even lower levels.[36]

Population and birth-death totals that survive allow us to discern the demographics of the crises, but it is well to recognize the limits of this data too **(See Table 2.4)**.[37] Since crude birth and death rates are particularly sensitive to the age structure of the population, they must be interpreted with care. A population with a high proportion of young married couples, for example, naturally tends to have a higher CBR because it contains more people in their prime childbearing years, while a population with a great many young unmarried people or elderly people has a lower CBR. Similarly, because people are most vulnerable to death in infancy and over the age of sixty, a population with a large number of infants or older people usually has a higher crude death rate than a population comprised mostly of adolescents and adults. As the necessary information on age and sex is often not available to historical demographers, age-specific fertility and mortality rates cannot be computed to correct for these differences. Only crude birth and death rates can be calculated because federal census takers did not collect adequate age or sex data until 1830. A few clues about Philadelphia's age and sex structure, however, can help in interpreting these rates. During the 1790s, for example, Philadelphia had a very young population,

especially when compared with modern population data.[38] This young population was the result of both a high birth rate, which produced a large number of children and a flood of immigrants in the 1780s and 1790s.[39] Philadelphia's youthful population also partially explains the city's high death rates. Infants and young children are particularly vulnerable to death resulting from poor sanitation, limited knowledge of disease, and neglect. Migration also elevated the city's death rates, because migrants were not only more susceptible to new diseases but also introduced and spread infections through the crowded urban environment.[40]

A similar analysis can be made for the early years of settlement. While the data show that during the years between 1690 and 1720 there was an excess of deaths in the city, all of the family reconstruction studies support the claim that there were very high marital fertility rates in early Philadelphia families.[41] Although fertility rates may have been high, they will not produce high birth rates if there are relatively few married women in the population. Many young adults arrived in Philadelphia as indentured servants, unable to marry because they were not free. If the men survived to complete their indenture, their prospects for marriage were slim since only one in ten English servants was a woman in the eighteenth century. The prevalence of men and bound persons in the first decades of the city's existence was certainly a factor preventing the population from reproducing itself.[42] The role of infectious disease, however, cannot be underestimated. It was a significant factor during these early years of settlement, and it contributed greatly to Philadelphia's high mortality rates. Not only did a pattern of frequent epidemics become established at this time, but high

levels of endemic disease also contributed to the tally of deaths in the city. In addition, environmental factors contributed to high mortality, with harsh weather and disastrous harvests bringing death to many in the city in the last decade of the seventeenth century.

Differential mortality is another important factor when considering the limits of the data. Each immigrant group faced the necessity of adjusting to an environment, a society, and a culture that differed from their place of origin. The complex process of change frequently had dramatic, if unequal, consequences for health.[43] The experiences of German immigrants and African slaves in eighteenth-century Philadelphia are especially suggestive. The difficulties of the former began during the difficult Atlantic crossing where the mortality at sea was about 3.8 percent. Debarkation morbidity was in the same range, and the rate tended to fall over time. Those who settled in Philadelphia, however, remained at a severe disadvantage. From 1738 to 1756 the annual average death rate for first-year German immigrants was 61.4 per 1000, compared with only 37 per thousand for established residents. Native Philadelphians were at a higher risk of dying from smallpox; Germans were at a lower risk, since they came from an area in which the disease was endemic and hence were already immune because they had had the disease in childhood. Yellow fever and malaria, however, took a greater toll among immigrants than among the native population.[44]

In a comparison between English and German settlers, it is worthwhile to note that both suffered from nutritional disorders including beriberi[45] and scurvy.[46] The German settlers, however, experienced the higher mortality rates. Although the diets of the two groups were similar, the English arrived in better health. Their journey across the Atlantic was shorter, and they were provided with a satisfactory diet aboard ship. By the time symptoms of beriberi and scurvy appeared, the first crops were being harvested, thus inhibiting a major outbreak. The Germans, by contrast, had a much longer and arduous voyage. Their diet was inadequate, and conditions aboard ship proved less than satisfactory.[47] Given their more problematic health status upon arrival, it is not surprising that they had a higher mortality rate.

Enslaved Africans in Philadelphia faced quite different circumstances. From 1682 to the 1760s they accounted for perhaps a quarter of the city's workforce. Most came from the Caribbean or southern colonies and had been exposed to semitropical infectious diseases. But they were unaccustomed to the harsher climate that characterized the Northeast. Many were also undernourished and afflicted with a variety of chronic infectious diseases.[48] While there were some similarities between the experiences of the European and African Philadelphians, the process of adjustment for the latter proved both longer and more difficult. Black mortality was about 50 percent higher than that of European immigrants. White mortality tended to peak in the summer, when malaria and enteric diseases were the major causes of death among Europeans unaccustomed to a warm and humid environment. Black

mortality peaked in the winter, when respiratory disorders were the leading causes of death.[49]

Diet played a major role in shaping differential mortality patterns for enslaved Africans. In cold winters with overcast days, the ability to utilize vitamin D is reduced, since it is synthesized by the body as a result of exposure to sunlight. A vitamin D deficiency also increases vulnerability to respiratory disorders. In the harsher northern climate dark-skinned people in particular suffered from this deficiency unless dietary sources compensated for the absence of exposure to sun. The practice of feeding slaves an inferior diet, combined with the harsher winter climate, may have increased their vulnerability to respiratory diseases.[50] Death rates for blacks were likewise higher during epidemics of measles, smallpox, whooping cough, and other diseases.

If exactitude is impossible, the general trends the numbers describe are reliable. Ironically, the population increased rapidly, even as Philadelphian suffered very high levels of mortality. The burials recorded in the city's cemeteries indicate that forty to fifty per thousand inhabitants died each year. These figures suggest that conditions in the city were far worse than those in most contemporary areas. Various diseases, some of them constantly reintroduced and spread by migrants, pushed the city's mortality rates to astonishing heights. Adding to this was the fact that the vast majority of residents lived within an eight by eight block area **(See Table 2.2)**. As the principal American immigrant port during the period, Philadelphia experienced

"the arrival of a trickle of English, approximately 26,000 Irish and Scotch Irish, and nearly 40,000 German newcomers between 1750 and 1775."[51] After pausing during the Revolutionary War, migration resumed as the French and Irish merged with groups leaving the American countryside to create a new influx of migrants to Philadelphia during the last fifteen years of the eighteenth century.[52] Since it was a walking city with no public transportation, every increase in population meant an increase in population density. Houses were built in rows and back alleys were cut into the spacious blocks – especially after 1780, as the population concentrated along the river in ever more congested housing patterns.[53]

While population growth and economic development certainly influenced mortality rates, the evidence suggests that infectious disease remained the major threat to health and longevity. When epidemic disease visited the city, an already high base-line mortality level was exacerbated by epidemic mortality patterns. The consequences of rapid growth, including an inability to adequately quarantine shipping, a dense urban environment, contaminated water, and improper disposal of untreated sewage, all would have contributed to the spread of infectious disease. They would presumably affect immigrants more severely than natives since the over-all health of new arrivals was already compromised. As a result, the first to experience the ravages of infectious epidemic diseases were urban port communities, which brought significant numbers of susceptible individuals into close living conditions. The maritime character of Philadelphia, Boston, New York and Charleston – the most important colonial ports – brought these communities into

contact with each other and, more importantly, with Europe, the Caribbean, and Africa. The ports also tended to be the entry point for both sailors and individuals migrating to the colonies. Such population movements became the means of transporting a variety of pathogens capable of causing disease.

The bills of mortality clearly show that as the eighteenth century progressed, Philadelphia's disease patterns changed. The increase in the size and density of the population, the expansion of internal and external trade and commerce, the development of new forms of agriculture, and transformation of the landscape all combined to alter the complex relationships between pathogens and hosts. In addition to these changes in the physical environment, the people of Philadelphia were changing as well. A high proportion of immune persons in a community generally precluded epidemic diseases, because the capacity to transmit the pathogen from person to person was impaired. In the early stages of settlement, migrants tended to be young adults who had already been exposed to a variety of infections in their native homeland, and were thus immune. Over several generations, however, the increase in the number of susceptible persons in the city created conditions conducive to the spread of infections. By the early nineteenth-century, Philadelphia's *total environment* had changed considerably from that of the early days of settlement. Accommodation to a new environment was an ongoing process, however, and successive generations would continue to face challenges as they moved into new environments that varied in the extreme.[54]

What emerges is a complex picture of a city undergoing rapid cultural and epidemiological changes. The vital statistics that survive are reasonable, although not perfect, estimates of Philadelphia's growth. These, in combination with bills of mortality, diaries, medical literature and other contemporary accounts, well-illustrate the extent to which death and disease pervaded the city. Multiple factors influenced the pattern and distribution of infectious diseases in Philadelphia, but human-initiated changes and activities set the stage for the many public health crises that plagued the city for more than one hundred years. Crude death rates fluctuated greatly throughout the eighteenth century, ranging between 30 and 60 per 1,000. Newly arrived immigrants were the most vulnerable; their mortality rates were nearly double that of native-born residents. Death rates in Philadelphia were high, and for much of the century, the city remained a hotbed for infectious disease. Although fertility was high, Philadelphia's growth was possible only because of the large-scale immigration of younger people.[55]

In order to understand how disease manifested itself in Philadelphia, however, one has to go beyond the crude birth and death rates to see human health as an outcome of multiple, reciprocal, and continuing interactions among pathogens, hosts and the surrounding environment. In the next two chapters, I account for the city's health crises using an epidemiological approach.[56] The idea that disease is always the result of the interplay of the environment, the genetic and physical make-up of the individual, and the agent of disease is one of the most important of the *cause and effect* ideas underpinned by epidemiology. The model maintains that the patterns of

disease in any population group depend on the factors that determine the probability of contact between an infectious agent and a susceptible host. In Chapter 3, I consider the historical process that so dramatically increased the probability of contact between the people of Philadelphia and infection, and in Chapter 4, I take into account those challenges faced by Philadelphians that promoted vulnerability to disease.

**Table 2.3**[57]
**Reconstructed Crude Birth and Death Rates**
**Philadelphia, 1690-1719**

| DATE | POPULATION | BIRTHS | DEATHS | CBR | CDR |
|---|---|---|---|---|---|
| 1690 | 2031 | | 153 | | 75 |
| 1691 | 2045 | | 40 | | 20 |
| 1692 | 2077 | | 55 | | 26 |
| 1693 | 2100 | | 53 | | 25 |
| 1694 | 2123 | | 145 | | 68 |
| 1695 | 2146 | | 73 | | 34 |
| 1696 | 2169 | | 69 | | 32 |
| 1697 | 2192 | | 56 | | 26 |
| 1698 | 2215 | | 123 | | 55 |
| 1699 | 2238 | | 343 | | 153 |
| **Total** | | **888** | **1110** | **41** | **51** |

| DATE | POPULATION | BIRTHS | DEATHS | CBR | CDR |
|---|---|---|---|---|---|
| 1700 | 2261 | | 113 | | 50 |
| 1701 | 2284 | | 157 | | 69 |
| 1702 | 2307 | | 93 | | 40 |
| 1703 | 2330 | | 64 | | 27 |
| 1704 | 2353 | | 59 | | 25 |
| 1705 | 2376 | | 53 | | 22 |
| 1706 | 2399 | | 75 | | 31 |
| 1707 | 2422 | | 91 | | 38 |
| 1708 | 2445 | | 120 | | 49 |
| 1709 | 2464 | | 162 | | 66 |
| **Total** | | **858** | **987** | **36** | **42** |

| DATE | POPULATION | BIRTHS | DEATHS | CBR | CDR |
|---|---|---|---|---|---|
| 1710 | 2684 | | 127 | | 47 |
| 1711 | 2904 | | 156 | | 54 |
| 1712 | 3124 | | 140 | | 45 |
| 1713 | 3344 | | 112 | | 33 |
| 1714 | 3564 | | 277 | | 78 |
| 1715 | 3784 | | 93 | | 24 |
| 1716 | 4004 | | 152 | | 38 |
| 1717 | 4224 | | 127 | | 30 |
| 1718 | 4444 | | 136 | | 31 |
| 1719 | 4664 | | 173 | | 37 |
| **Total** | | **1401** | **1493** | **39** | **42** |

Source: Table is from Susan E. Klepp, 1989, "Demography in Early Philadelphia, 1690-1860," p. 103-7.

**Table 2.3**
**Reconstructed Crude Birth and Death Rates**
**Philadelphia, 1720-1749**

| DATE | POPULATION | BIRTHS | DEATHS | CBR | CDR |
|---|---|---|---|---|---|
| 1720 | 4883 | 316 | 132 | 65 | 27 |
| 1721 | 5102 | 278 | 186 | 54 | 36 |
| 1722 | 5321 | 269 | 241 | 51 | 45 |
| 1723 | 5540 | 219 | 143 | 40 | 26 |
| 1724 | 5759 | 154 | 286 | 27 | 50 |
| 1725 | 5978 | 287 | 236 | 48 | 39 |
| 1726 | 6197 | 347 | 246 | 56 | 40 |
| 1727 | 6416 | 372 | 393 | 58 | 61 |
| 1728 | 6635 | 312 | 214 | 47 | 32 |
| 1729 | 6854 | 465 | 600 | 68 | 87 |
| **Total** | | **3019** | **2677** | **51** | **44** |

| DATE | POPULATION | BIRTHS | DEATHS | CBR | CDR |
|---|---|---|---|---|---|
| 1730 | 7075 | 284 | 254 | 40 | 36 |
| 1731 | 7294 | 486 | 505 | 67 | 69 |
| 1732 | 7514 | 412 | 366 | 55 | 49 |
| 1733 | 7734 | 381 | 317 | 49 | 41 |
| 1734 | 7952 | 484 | 387 | 61 | 49 |
| 1735 | 8372 | 422 | 320 | 50 | 38 |
| 1736 | 8792 | 519 | 477 | 59 | 54 |
| 1737 | 9212 | 442 | 401 | 48 | 44 |
| 1738 | 9631 | 452 | 550 | 47 | 57 |
| 1739 | 9874 | 374 | 350 | 38 | 35 |
| **Total** | | **4256** | **3927** | **51** | **47** |

| DATE | POPULATION | BIRTHS | DEATHS | CBR | CDR |
|---|---|---|---|---|---|
| 1740 | 10117 | 427 | 290 | 42 | 29 |
| 1741 | 10360 | 330 | 745 | 32 | 72 |
| 1742 | 10755 | 427 | 409 | 40 | 38 |
| 1743 | 11150 | 339 | 440 | 30 | 39 |
| 1744 | 11545 | 388 | 410 | 34 | 36 |
| 1745 | 11940 | 509 | 420 | 43 | 35 |
| 1746 | 12336 | 778 | 688 | 63 | 56 |
| 1747 | 12731 | 699 | 732 | 55 | 57 |
| 1748 | 13126 | 723 | 672 | 55 | 51 |
| 1749 | 13521 | 753 | 758 | 56 | 54 |
| **Total** | | **5373** | **5564** | **45** | **47** |

Source: Table is from Susan E. Klepp, 1989, "Demography in Early Philadelphia, 1690-1860," p. 103-7.

**Table 2.3**
**Reconstructed Crude Birth and Death Rates**
**Philadelphia, 1750-1779**

| DATE | POPULATION | BIRTHS | DEATHS | CBR | CDR |
|---|---|---|---|---|---|
| 1750 | 13926 | 465 | 716 | 33 | 51 |
| 1751 | 14330 | 697 | 926 | 49 | 65 |
| 1752 | 14812 | 708 | 673 | 48 | 45 |
| 1753 | 15295 | 700 | 623 | 46 | 41 |
| 1754 | 15901 | 798 | 856 | 50 | 54 |
| 1755 | 16508 | 810 | 502 | 49 | 30 |
| 1756 | 17114 | 942 | 1104 | 55 | 64 |
| 1757 | 17485 | 958 | 714 | 55 | 41 |
| 1758 | 17856 | 1112 | 766 | 62 | 43 |
| 1759 | 18227 | 1190 | 1406 | 65 | 77 |
| **Total** | | **8380** | **8286** | **51** | **51** |

| DATE | POPULATION | BIRTHS | DEATHS | CBR | CDR |
|---|---|---|---|---|---|
| 1760 | 18598 | 1014 | 957 | 54 | 53 |
| 1761 | 19425 | 1146 | 790 | 59 | 41 |
| 1762 | 20251 | 1261 | 1189 | 62 | 59 |
| 1763 | 21078 | 1274 | 1095 | 60 | 52 |
| 1764 | 21904 | 1408 | 1091 | 64 | 50 |
| 1765 | 22731 | 1380 | 1273 | 61 | 56 |
| 1766 | 23557 | 1112 | 990 | 47 | 42 |
| 1767 | 24384 | 1364 | 809 | 56 | 33 |
| 1768 | 25064 | 1287 | 801 | 51 | 32 |
| 1769 | 25744 | 1274 | 1160 | 50 | 45 |
| **Total** | | **12520** | **10155** | **56** | **46** |

| DATE | POPULATION | BIRTHS | DEATHS | CBR | CDR |
|---|---|---|---|---|---|
| 1770 | 26789 | 1263 | 971 | 47 | 36 |
| 1771 | 27833 | 1310 | 1007 | 47 | 36 |
| 1772 | 28878 | 1284 | 1273 | 44 | 44 |
| 1773 | 29547 | 1555 | 1344 | 53 | 45 |
| 1774 | 30216 | 1545 | 1022 | 51 | 34 |
| 1775 | 32073 | 1301 | 1180 | 41 | 37 |
| 1776 | 32073 | 1389 | 1062 | 43 | 33 |
| 1777 | 32073 | 1009 | 1356 | 31 | 42 |
| 1778 | 32073 | 923 | 1305 | 29 | 41 |
| 1779 | 32073 | 1359 | 1095 | 42 | 34 |
| **Total** | | **12938** | **11615** | **43** | **38** |

Source: Table is from Susan E. Klepp, 1989, "Demography in Early Philadelphia, 1690-1860," p. 103-7.

**Table 2.3**
**Reconstructed Crude Birth and Death Rates**
**Philadelphia, 1780-1809**

| DATE | POPULATION | BIRTHS | DEATHS | CBR | CDR |
|---|---|---|---|---|---|
| 1780 | 32073 | 1498 | 1092 | 47 | 34 |
| 1781 | 35435 | 1536 | 1146 | 43 | 32 |
| 1782 | 38798 | 1930 | 1017 | 50 | 26 |
| 1783 | 38890 | 1844 | 1536 | 47 | 39 |
| 1784 | 38982 | 2047 | 1362 | 52 | 35 |
| 1785 | 39074 | 2152 | 1125 | 55 | 29 |
| 1786 | 39166 | 2294 | 1095 | 58 | 28 |
| 1787 | 39258 | 2520 | 1140 | 64 | 29 |
| 1787/8 | 39352 | 2192 | 996 | 56 | 24 |
| 1788/9 | 41724 | 2019 | 996 | 48 | 23 |
| **Total** | | **20032** | **11505** | **52** | **30** |

| DATE | POPULATION | BIRTHS | DEATHS | CBR | CDR |
|---|---|---|---|---|---|
| 1789/90 | 44096 | 2094 | 1035 | 48 | 24 |
| 1790/1 | 46498 | 2256 | 1309 | 49 | 28 |
| 1791/2 | 48840 | 2407 | 1245 | 49 | 26 |
| 1792/3 | 51212 | 2511 | 1497 | 49 | 29 |
| 1793/4 | 53584 | 2379 | 4992 | 44 | 93 |
| 1794/5 | 55956 | 2324 | 1759 | 42 | 31 |
| 1795/6 | 58328 | 2847 | 2283 | 49 | 39 |
| 1796/7 | 60700 | 2694 | 1666 | 44 | 27 |
| 1797/8 | 63072 | 2755 | 2356 | 44 | 37 |
| 1798/9 | 65444 | 2648 | 4463 | 40 | 68 |
| **Total** | | **24915** | **22605** | **46** | **40** |

| DATE | POPULATION | BIRTHS | DEATHS | CBR | CDR |
|---|---|---|---|---|---|
| 1799/00 | 67811 | 2657 | 1762 | 39 | 26 |
| 1800/01 | 69929 | 2189 | 2062 | 31 | 29 |
| 1802 | 72047 | 2688 | 2347 | 37 | 32 |
| 1803 | 74165 | 2581 | 1980 | 35 | 27 |
| 1804 | 76283 | 2619 | 1941 | 34 | 25 |
| 1805 | 78401 | 2518 | 2572 | 32 | 33 |
| 1806 | 80519 | 2893 | 1936 | 36 | 24 |
| 1807 | 82637 | 4230 | 2045 | 51 | 25 |
| 1808 | 84755 | 3342 | 2271 | 39 | 27 |
| 1809 | 86873 | 3372 | 2004 | 39 | 23 |
| **Total** | | **29089** | **20920** | **37** | **27** |

Source: Table is from Susan E. Klepp, 1989, "Demography in Early Philadelphia, 1690-1860," p. 103-7.

**Table 2.4**
**Levels of Confidence**

| Decade | Births | Deaths |
|---|---|---|
| 1690 | 5 | 4 |
| 1700 | 5 | 4 |
| 1710 | 5 | 4 |
| 1720 | 3 | 3 |
| 1730 | 4 | 2 |
| 1740 | 2 | 2 |
| 1750 | 2 | 1 |
| 1760 | 2 | 1 |
| 1770 | 3 | 2 |
| 1780 | 2 | 2 |
| 1790 | 1 | 1 |
| 1800 | 2 | 1 |

Source: Table is from Klepp, 1989, "Demography in Early Philadelphia, 1690-1860," p. 109.

Note: Levels of confidence vary in this reconstruction of vital events. Table indicates the quality of the vital statistics by decade with [1] indicating excellent returns and [5] indicating an estimate that could well be revised when better sources are discovered.

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[1] Epidemic disease is the occurrence in a community or region of cases of an illness in excess of normal expectancy. See Last, 2001, p. 60.
[2] Endemic disease is defined as the constant presence of a disease or an infectious agent within a given geographic area or population. See Last, 2001, p. 59.
[3] Smith, 1990, p. 41-2.
[4] The crude death rate (CDR) measures deaths per 1,000 population. These rates are considered crude because they do not take into account the underlying age, sex, and ethnic composition of society.
[5] The Crude Death Rate for the period between 1690 and 1692 was 40, for the period between 1693 and 1702 it was 55 and for the period between 1704 and 1712 it was 41. Data is from Klepp, 1989, "Demography in Early Philadelphia, 1690-1860," p. 103.
[6] Labaree, 1959-1973, Vol. IX, *The Papers of Benjamin Franklin*, p. 72-4.
[7] See Cassidy, 1969, *Demography in Early America*, p. 124.
[8] Vital rates are taken from Poulson's *Town and Country Almanac, 1789-1802*, while references to specific diseases were taken from the Christ Church Bills of Mortality. There are slight differences in the baptisms and burials recorded by the two sets of records. The Christ Church Bills of Mortality covered one calendar year while Poulson's data generally included the period from September to September, or August to August. Scattered bills for the final years of the century are listed as "An Account of the Births and Burials in the United Churches of Christ-Church and St. Peter's," in Charles Evans, 1903-1959, *American Bibliography: A Chronological Dictionary of all Books, Pamphlets, and Periodical Publications Printed in the United States of America from…1639 down to…1820*. See Klepp, 1995, "Zachariah Poulson's Bills of Mortality, 1788-1801," p. 227.
[9] The epidemic proportions of venereal disease are evident in Daily Occurrences Docket, November 14, 1800, Guardians of the Poor, PCA; Smith, 1990, p. 47.
[10] A helminth is any of various parasitic worms, including flatworms, tapeworms, and roundworms. Some varieties infest humans causing disease. See Rothenberg et al, 2000, p. 255.
[11] Some of the remedies utilized by the Drinkers in their never-ending confrontation with disease were concocted at home from a variety of plants and liquids; others were purchased at the apothecary shop. However obtained, these preparations were not arbitrarily dispensed. Each was prescribed in response

to a particular disease, and some appear to have been moderately effective, while others were notoriously toxic. Carolina pink root, for example, was used in the treatment of intestinal worms. The convulsions that sometimes followed low doses of the drug where minimized by the cathartic or emetic effects of larger doses. No matter the circumstances, eighteenth-century pharmacology was risky business, with the remedy often more deadly than the actual disease. See Crane, 1991, p. xxiii; Estes, 1990, p. 181. Note: a cathartic is a laxative and an emetic is a substance that induces vomiting. See Rothenberg et al, 2000, p. 105, 183.

[12] Crane, 1991, p. 31-2, 39, 36. Excerpts from the *Diary of Elizabeth Drinker*, September 13, 1759, November 13, 1759 and October 24, 1759.

[13] The technique was simple: matter taken from a pustule of someone who had caught the disease naturally was placed on several small cuts made by a needle or a lancet in the arm or leg muscles of the person receiving the inoculation. The method produced a milder, less fatal form of the disease, although the person was contagious while undergoing the procedure.

[14] The entry for December 2, 1762 describes the mistake made by the apothecary in substituting Tartar Emetic for Rochelle Salt (sodium potassium tartrate), a mild cooling cathartic. Tartar Emetic, a preparation of antimony and potassium tartrate, was a popular eighteenth-century emetic, diaphoretic, expectorant, cathartic, and sedative, although it causes poisoning in large doses. See Crane, 1991, p. 98; Dine, 2001, p. 416.

[15] Although Drinker was a member of the elite or "better sort," her diary is, nevertheless, a rich source of information about less affluent Philadelphians, their relationship to the Drinkers, and with each other. She was responsible for the health and well-being of servants in her home, and while her management of them cannot be translated into a generalization for all Philadelphians, certain common customs may be extracted. As members of the Drinker family, servants received much the same medical care as everyone else in the household – treatment that was either advantageous or not, depending on one's assessment of eighteenth-century medicine. Benjamin Rush favored Drinker's servant Sally Dawson with a prescription for castor oil, just as he did his other Drinker patients. Elizabeth's personal physician, Dr. Kuhn, attended daughter Nancy's "black girl" Patience Gibbs and diagnosed Sally Dawson's yellow fever. Crane, 1991, p. 695, 1171, 1688. See the *Diary of Elizabeth Drinker*, June 23, 1795, May 24, 1799 and September 29, 1803. A favorable recommendation from Elizabeth Drinker permitted a servant, former servant, or relative of a servant to obtain free medication from the Philadelphia dispensary, thus extending Drinker's influence over the health of her extended family. Crane, 1994, p. xiii-xiv.

[16] Crane, 1991, p. xx.

[17] Crane, 1991, p. xxxi.

[18] Crane, 1983, p. 25.

[19] Drinker, 1937, p. 24.

[20] Rush, 1815, "An Inquiry into the Cause and Cure of Cholera Infantum," Vol. III, p. 119.

[21] Rush, 1815, "An Account of the Climate of Pennsylvania and its Influence on the Human Body," Vol. II, p. 3-27.

[22] Philadelphia's urban environment was quite small at this time. The vast majority of the population lived within a few blocks of the Delaware River.

[23] Bridenbaugh, 1955, p. 105, 296; Drinker, 1937, p. 22-33; Hawke, 1971, p. 9; Rush, 1815, "Account of the Climate of Pennsylvania and its Influence on the Human Body," Vol. II, p. 27; Klepp, 1989, *Philadelphia in Transition*, p. 225-6.

[24] The Table depicts population levels per city block during the early years of the 1790s. Population density in the center of the city reached a height of 1,411 persons in one-and-a-half city blocks. From there, the density rate declined rapidly. Just three blocks away, at Fifth and Market Streets, density was more than halved. Data is from Schweitzer, 1993, p. 32, 39.

[25] Klepp, 1991, "*The Swift Progress of Population*." The statistics of births and deaths in Philadelphia published annually by Zachariah Poulson in the first thirteen issues of his *Town and Country Almanac* (1789-1802) are among the first to be collected systematically after the American Revolution. They provide an unusually detailed record of public health during a decade of dramatic contrasts. See Klepp, "Zachariah Poulson's Bills of Mortality, 1788-1801," in Billy G. Smith, ed., *Life in Early Philadelphia*, 1995, p. 219-220. Poulson's *Almanacs* for 1789 through 1800 are at the Presbyterian Historical Society, Philadelphia. The *Almanacs* for 1801 through 1807 are at the Historical Society of

Pennsylvania, Philadelphia. These and all other surviving bills of mortality have been reprinted in Susan E. Klepp, ed., 1991, "*The Swift Progress of Population": A Documentary and Bibliographic Study of Philadelphia's Growth, 1642-1859*.

[26] Current Pennsylvania rates are from the Bureau of the Census, Statistical Abstracts of the United States, 2002. The data for Afghanistan and Somalia are from the 2004 World Population Data Sheet of the Population Reference Bureau.

[27] The French and Indian War was raging in the colonies between 1754 and 1763. Consequently, Philadelphia became a city of refugees. There were the surviving soldiers from General Braddock's defeat along the Monongahela. There were civilian refugees from the West, driven from their homes by the aroused Indians, and there were some 450 of the 1000 Acadians whom the English, fearing them loyal to France, expelled from their Nova Scotia homes. This last group was housed in a row of one-story wooden buildings on Pine Street. In addition to the refugees, in late 1756 the city was ordered to house the new Royal American Regiment, British regulars recruited in the colonies. Many of these soldiers were sick with smallpox. The British victory ratified at Paris in 1763 did not spare Philadelphia from further alarms. The city once more filled with refugees from the West. Among them was a small group of "Moravian Indians," who sought succor in the city both from Indians angry at their pacifism and from their white frontiersmen. See Thayer, 1982, p. 105. By the time of the Revolutionary War, Philadelphia may well have been the only place on the continent where smallpox had become endemic. While the continuous circulation of smallpox made Philadelphia a dangerous place for anyone susceptible to the disease, it did not keep the city from becoming the so-called capital of the thirteen states during the Revolutionary War. This posed a particular danger to members of the Continental Congress, many of whom came from states where smallpox was rare and inoculation banned. Nonetheless, the persistent contagion established Philadelphia as a sort of distribution center from which the disease could spread far and wide. In January 1776, the Continental fleet under Commodore Esek Hopkins set sail from the city. Because of ice in the Delaware River, it took more than a month for his ships to reach the Atlantic. By February 17th, when they finally put to sea, smallpox had become rampant among the men. See Fenn, 2001, p. 82-5.

[28] Philadelphians did not appear to have had difficulty in putting food on the table, although whether all classes were well nourished in addition to having sufficient food is another question. See Klepp, 1989, *Philadelphia in Transition*, p. 227-8; Warner, 1987, p. 37-42.

[29] Klepp, 1989, *Philadelphia in Transition*, p. 236; Table 2.3.

[30] Acute infectious diseases which cause permanent immunity, such as measles and smallpox, require populations of 500,000 to 1 million in order to become endemic. Since smaller populations do not have a large enough annual input of susceptible people, the diseases will die out. When reintroduced, people born since the previous epidemic will all be susceptible to infection. So, as population increases, these illnesses tend to become endemic and common in childhood. Smallpox and measles became endemic in Philadelphia in the later decades of the eighteenth century. Evidence of this can be found in the bills of mortality; both diseases were present year after year, but mortality from them grew increasingly less with each passing year.

[31] Figure is from Klepp, 1997, "Appendix I: 'How Many Precious Souls Are Fled'?: The Magnitude of the 1793 Yellow Fever Epidemic," p. 174. Deaths to 1807 based on local bills of mortality collected in Klepp, *"The Swift Progress of Population": Documentary and Bibliographic Study of Philadelphia's Growth, 1642-1859*, and supplemented by church registers and other records. African American deaths are unavailable prior to 1720. From 1807 to the present, deaths have been recorded by the Department of Public Health, under slight changes in name and in various formats. Warner Tillack of the Division of Vital Statistics calculated the 1994 rate for publication of the chart. The population base, prior to the reorganization of the health office in 1860, is the city of Philadelphia and its contiguous, urbanized suburbs. After 1860, the base is Philadelphia County. Before 1790, the calculations of P.M.G. Harris, "The Demographic Development of Colonial Philadelphia in some Comparative Perspective," p. 274, and Billy G. Smith, "Death and Life in a Colonial Immigrant City: A Demographic Analysis of Philadelphia," p. 865, are used as the base population. Asterisks indicate the highest of the recent estimates for annual mortality in 1793 and 1798, while the solid line is based on contemporary reports. Special estimates are based, in part, on the work of Tom W. Smith. See Klepp, 1997, p. 174.

[32] A vector is a person, animal, or microorganism that carries and transmits disease. Mosquitoes, for example, are vectors of malaria and yellow fever, carrying disease-producing parasites. See Rothenberg et al, 2000, p. 580.
[33] Duffy, 1953, p. 55-69.
[34] The Christ Church Bills of Mortality consistently list deaths from "flux, fever, purging and vomiting." Elizabeth Drinker's diary is replete with references to gastrointestinal disorders and summer fevers. Every member of her immediate family suffered from periodic bouts of both dysentery and malaria.
[35] Death rates are taken from Table 2.3.
[36] Klepp, 1989, "Demography in Early Philadelphia," p. 92.
[37] Levels of confidence vary in Klepp's reconstruction of vital events. Table 2.4 indicates the quality of the vital statistics by decade with [1] indicating excellent returns and [5] indicating an estimate that could well be revised when better sources are discovered. Because these guides are by decade they may mask variation within any single decade. In the 1720s, for an unusually complicated example, there is an excellent account of births and deaths for 1722, of deaths alone for 1729, and good accounts for some other years. However, the estimates for 1723-1725 are especially suspect since they are based in part on fragmentary Anglican records. Multipliers developed to account for the missing monthly accounts may be deficient. Taken from Klepp, 1989, "Demography in Early Philadelphia," p. 108-9.
[38] Of the free white males living in Philadelphia County in 1790, some 43 percent were younger than sixteen. In 1990, only 22 percent of the nation's inhabitants were under fifteen. See Klepp, 1995, "Zachariah Poulson's Bills of Mortality, 1788-1801," p. 226.
[39] Klepp, 1995, "Zachariah Poulson's Bills of Mortality, 1788-1801," p. 225-6.
[40] Between 1720 and 1760, the city had established itself as the economic center of a very productive agricultural region and as an important supplier of goods to the West Indies. Immigration from Europe and trade with the Caribbean not only fed the growth of the area but also retarded the rapid increase in the population, since commerce was a constant source for the reinfection of the population with epidemic disease. In particular, tropical diseases that could not withstand the frosts of Philadelphia's winters were reintroduced every summer through trade with the Caribbean. The "seasoning" of new arrivals took a tremendous toll on the people of Philadelphia. As epidemics spread through the town, death rates soared to an average of 47 per 1,000 between 1720 and 1760. See Table 2.3.
[41] Wells, 1971, p. 73-83; Kantrow, 1980, p. 21-30; Klepp, 1989, *Philadelphia in Transition*, p. 142-230; Klepp, 1982, "Five Early Pennsylvania Censuses," p. 491, 495.
[42] Klepp, 1989, "Demography in Early Philadelphia," p. 92-5.
[43] As Klepp points out, differential mortality in colonial Philadelphia was based largely on nativity and less on wealth. During the yellow fever epidemic of 1741, for example, newly arrived German immigrants experienced 95 deaths per 1,000 while Philadelphia residents experienced 55 deaths per 1,000. The native-born, whether rich or poor, were often spared the diseases afflicting new immigrants because of prior exposure to the disease. See Grubb, 1987, p. 565-85; Klepp, 1989, "Demography in Early Philadelphia," p. 95-6.
[44] Grubb, 1987, p. 565-85.
[45] A disease resulting from a deficiency of vitamin B1 (thiamine), characterized by appetite and weight loss, disturbed nerve function, fluid retention, and heart failure. See Rothenberg et al, 2000, p. 68.
[46] Condition caused by a lack of ascorbic acid (vitamin C) in the diet and characterized by anemia, weakness, and spongy, bleeding gums. See Rothenberg et al, 2000, p. 506.
[47] Grubb, 1987, p. 565-85.
[48] Measles, respiratory infections, and whooping cough are diseases that are more fatal to malnourished subjects. See Boserup et al, 1983, p. 305-8; Klepp, 1994, p. 489.
[49] Klepp, 1994, p. 473-506. Sources for epidemics include A.C. Abbott, 1909, "The Development of Public Health Work in Philadelphia: An Historic Sketch," in F.P. Henry, ed., *Founder's Week Memorial Volume*, p. 563-92; John Duffy, 1953, *Epidemics in Colonial America*; Burton R. Konkle, 1977, *The Standard History of the Medical Profession of Philadelphia, Enlarged and Corrected by Lisabeth M. Holloway*; M.A.F. Mansfield, 1947, "Yellow Fever Epidemics in Philadelphia, 1699-1805;" John F. Watson, 1905, Vol. II, *Annals of Philadelphia and Pennsylvania in the Olden Time*, p. 370-3; Roslyn Stone Wolman, 1974, "Some Aspects of Community Health in Colonial Philadelphia;"

William Currie, 1811, *A View of the Diseases Most Prevalent in the Unites States of America, at Different Seasons of the Year*; Suzanne M. Shultz, 1994, "A Catalog of Epidemics in Philadelphia, 1699 to 1799."

[50] Vitamin D deficiency predisposes individuals to respiratory and skin diseases. In northern, less sunny climates, dark-skinned people especially can suffer from vitamin D deficiency unless dietary sources – eggs, tuna, liver, milk, butter – compensate for the body's inability to produce sufficient amounts of the vitamin from the available sunlight. See Kiple and King, 1981, p. 10-1, 91 2. Higher black mortality in Philadelphia coincided with cold winters, so that even if blacks and whites had fared equally well in terms of food, clothing, and shelter, black death rates would have surpassed those of whites in cold, cloudy weather. See Klepp, 1994, p. 479-80.

[51] Smith, 1990, p. 42.

[52] The best analyses of migration to the city are in Marianne Wokeck, 1981, "The Flow and the Composition German Immigration to Philadelphia, 1727-1775," p. 249-78; and Wokeck, 1988, "Irish Immigration to the Delaware Valley before the American Revolution." See also Farley Grubb, 1988, "British Immigration to Philadelphia: The Reconstruction of Ships Passenger Lists from May 1772 to October 1773," p. 118-41; Smith, 1990, p. 42.

[53] Bridenbaugh, 1955, p. 14-5, 224-5.

[54] Infectious diseases continue to represent a major cause of morbidity and mortality worldwide, and will pose an ongoing challenge in recognition, diagnosis, and therapy for health care professionals in the twenty-first century. For a discussion, see Longworth, 2001, p. ix.

[55] Grob, 2002, p. 93-4; Klepp, 1989, "Fragmented Knowledge: Questions in Regional Demographic History," 223-33.

[56] Epidemiology is predominantly an observational and not experimental science. This limitation is shared with many other sciences including demography, geology, evolutionary biology, paleontology, and archaeology. Causal thinking in epidemiology draws upon the principals of other disciplines including philosophy, the laboratory sciences, and the social sciences and is theoretically grounded, though it may not be obvious. Epidemiology has, moreover, contributed new ways of thinking about causality when experiment is not possible. See Bhopal, 2002, p. 116. Consequently, it can be a useful tool when studying disease in past populations.

[57] Klepp, 1989, "Demography in Early Philadelphia," p. 103-7. Population figures, births and deaths should be considered to be representative of the Euro-American population only before 1720; thereafter the Afro-American population, births and deaths are included in the totals. The population includes the suburbs of Southwark and the Northern Liberties. Births were estimated for the following decades: 1690, 1700 and 1710. In each of these decades there was reason to believe that surviving documents underestimated the number of births in the city. For the three decades under consideration here, annual birth rates are not calculated. Years are not constant. In the eighteenth-century reports varied, sometimes measuring the year from Christmas to Christmas, other times measuring from the modern New Year's to New Year's. In the mid-1790s, the most complete source measured the year from September to September [1787-1790], May to May [1798-1800], or August to August [1790-1798, 1801]. Thus the yellow fever epidemics of 1793 and 1798 appear in the totals of the following year. The reports of the Board of Health, 1802+, used the calendar year. Stillbirths are recorded in total deaths. Taken from Klepp, 1989, "Demography in Early Philadelphia," p. 108-9.

# CHAPTER 3

## PROBABILITY OF CONTACT

Classic epidemiological theory maintains that the patterns of infectious disease in any population group depend on factors that determine the probability of contact between an *infectious agent*[1] and a *susceptible host*. In Philadelphia, there were many factors operating to fuel this interaction. Some of the more pervasive included high levels of immigration, a dense settlement pattern, a high volume and velocity of movement both in and out of the city, crude sewage disposal, international commerce and stagnant and contaminated water. At its most fundamental level, this model assists us in understanding the occurrence of the infectious diseases that weighed so heavily on the people of Philadelphia during the eighteenth-century. In order to enhance our understanding, however, we must also include a broadened awareness of critical host factors, such as the immunological state of the host, as well as an appreciation of the *total environment*[2] in which contact took place. Setting aside the susceptibility component of the model for now, this chapter considers the historical processes that so dramatically increased the probability of contact between the people of Philadelphia and infectious disease.[3]

The population of Philadelphia was not at serious demographic risk from wide-spread epidemic disease before the start of the eighteenth century. Basic epidemiological principles tell us that no infection requiring transfers between human hosts for survival can maintain itself for long above the *fade-out threshold*[4] in a static and dispersed population group – a group where the probability of personal interaction is low. Diseases can neither be endemic (constantly present) nor epidemic (excessively present) in a community whose members come in contact too infrequently to keep the chain of transmission alive. Mortality rates from infectious disease among such groups will be relatively low as a consequence. Such was the case in Philadelphia during the last decade of the seventeenth century, when the population density was still quite low and the massive immigrations[5] from Europe and the Caribbean had not yet begun. In the early years of the eighteenth century, however, the increase in the number of susceptible people, in combination with a rapidly changing physical environment, created conditions conducive to the emergence of disease. Virologist Stephen S. Morse suggests that infectious disease emergence can be viewed as a two-step process: 1) introduction of the agent into a new host population (whether the pathogen originated in the environment, possibly in another species, or as a variant of an existing human infection), followed by 2) establishment and further dissemination within the new host population. Whatever the origin, infection "emerges" when it takes hold in a new population. Factors that promote one or both of these steps will, therefore, tend to precipitate disease emergence.[6] As a result of both immigration and a substantial commercial enterprise, Philadelphia was in constant contact with external disease environments,[7] and

infection easily entered into the city. Once established, infectious disease rapidly spread through vulnerable members of the population living in the densely packed streets and alleys.[8]

Relative size and isolation can also determine the extent of infectious disease within a community. Even when population density and interpersonal contact are sufficient to allow rapid diffusion, endemicity of many diseases is impossible in population groups below a critical size. As infection spreads, the remaining pool of susceptibles is quickly reduced to below the level at which transmission can be sustained. Contact with an external population center where the infection is endemic or has been recently introduced is necessary for the reintroduction of the disease, and a new epidemic must await the replenishment of the pool of susceptible hosts. Death rates will be high in small, isolated communities under epidemic conditions, but whether or not mortality is excessive over the longer term depends on the frequency with which the infection is imported. That frequency depends, in turn, on the size of the small community, its distance from the external reservoir, and the efficiency of the transportation network binding them. As a rule, the smaller and more remote a community, the more irregular and discontinuous epidemic waves will be.[9] Overall mortality is likely to be lower as well.[10] Philadelphia was neither small nor isolated at this time in its history, and, as a commercial port city, was a key player in a large, global trade network. As a result, disease was continuously brought into the city, and disease outbreaks were frequent.[11]

While population density was one of the most obvious factors in determining the probability of contact between the people of Philadelphia and infectious disease, other processes played a role as well. They[12] included: ecological changes,[13] including those due to land use, economic development and anomalies in climate;[14] human demographic changes and behavior, including population growth and density, migration and war;[15] international trade and commerce;[16] microbial adaptation and change;[17] and absence of a significant public health infrastructure.[18] Each of these processes is discussed more fully below. Microbial adaptation and change is covered in Chapter 5.

## Ecological Changes

Ecological changes usually precipitate emergence of disease by placing people in contact with a natural reservoir or host for an infectious agent. This can be accomplished either by increasing proximity or by changing conditions so as to favor an increased population of the microbe or its natural host. It can also be accomplished by altering the virulence of the microorganism or changing the susceptibility of the host. Because humans are important agents of ecological and environmental change, many of these factors are anthropogenic. This is not always the case, however, and natural environmental changes, such as climate or weather anomalies, can have the same effect. Whatever the factors involved, these interactions can be complex, with several often working together in sequence. Mosquitoes aboard vessels docking in Philadelphia, for example, found a hospitable

environment in the marshes west of the city and in the Dock Creek area. The vicinity surrounding Dock Creek was the home of several of Philadelphia's tanyards, and water was needed throughout the processing of the hides. The *Aedes aegypti* mosquito, the vector for yellow fever, is well adapted to urban conditions and thrives in water barrels on board ships, rain barrels next to houses, and any place where there is standing water. The mosquito's limited range of a few hundred yards meant that outbreaks of the disease were quite localized. The combination of standing water, mosquitoes and tannery workers made this area surrounding Dock Creek a prime location for outbreaks of yellow fever, which rarely appeared far from the waterfront and almost never in the countryside. Similar to other diseases spread by mosquitoes, yellow fever occurred seasonally, usually beginning in late July or August, peaking in September and October, then ending quickly in November with the first frost of the season.

The physical environment of Philadelphia after the first few decades of the eighteenth century set relatively high limits on the probability of contact with infection from outside reservoirs. The city was then situated entirely between the Delaware and Schuylkill Rivers, with little settlement beyond 7$^{th}$ Street. The proximity of these rivers, together with an abundance of other natural resources, ensured that the region would be as much a magnet for European colonists as it had been for prehistoric people. William Penn founded the city in 1682, and, upon his arrival, there were already ten houses and a tavern on the site and small settlements of Dutch and Swedish farmers nearby. With the arrival of Penn and the Quakers, the

area rapidly became a major settlement. The first to follow Penn to Philadelphia were the British. Other groups included Welsh, Scots, Scotch-Irish and enslaved people from the West Indies and Africa. Germans began arriving in large numbers starting in the 1730s and continuing until the beginning of the Revolutionary War, while the Irish came in two waves that peaked just before and after the War. Other ethnic groups were attracted to the cosmopolitan city as well, and it became the political, administrative and economic center of Pennsylvania. It also dominated the region of southern New Jersey and Delaware. By the middle of the eighteenth century, it was the largest city in British America. Additionally, Philadelphia hosted the Continental Congresses, the Constitutional Convention, and was for ten years the capital of the United States.[19] As a result of its cosmopolitan character, the city housed many transients and visitors - people who could easily "pick up, process, carry and drop off" a wide variety of potentially infectious agents.[20]

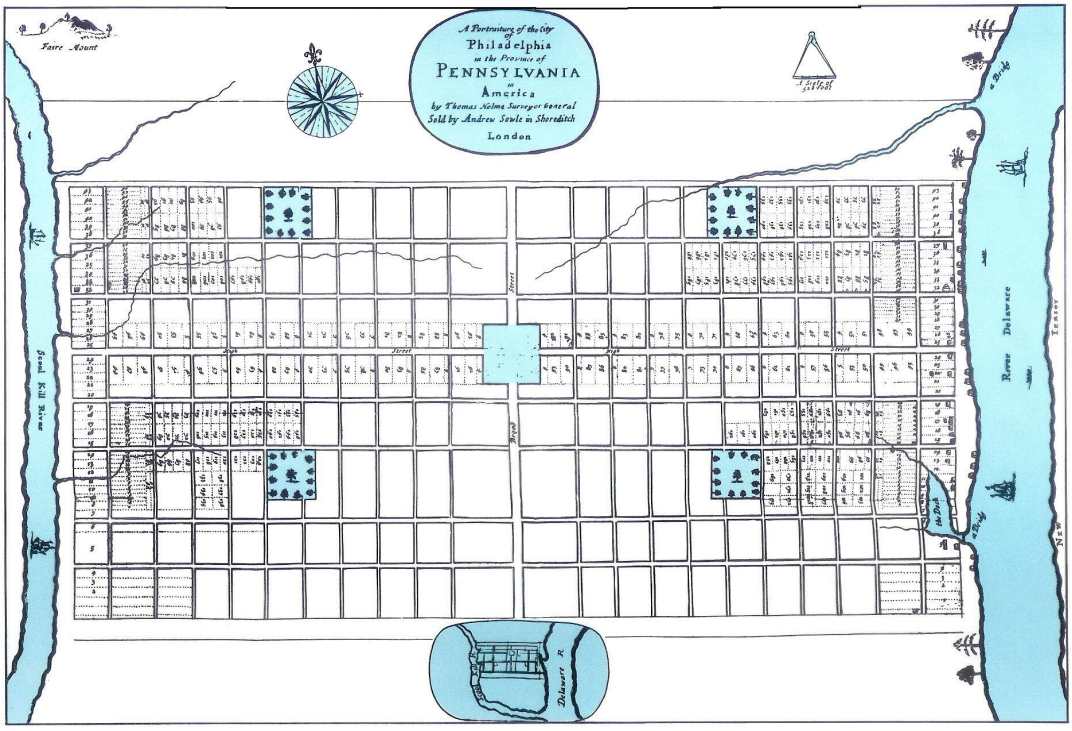
The plan for the town of Philadelphia offered a sharp contrast to the typical, crowded English city. It was to occupy 10,000 acres "in the most convenient place upon the river for health and navigation."[21] The large house lots, some with 800 feet of river frontage and 100 acres of land, were to extend fifteen miles along the Delaware River. With the houses sitting in the middle of their generous plots surrounded on all sides by gardens and orchards, it would be a "green country town, which [would] never be burnt, and always be wholesome." The plan was for a town that would reflect the lifestyle of the landed English gentry – a lifestyle very familiar to William Penn. It was assumed that the owners of these grand urban properties also

would own large country estates beyond the town limits.[22] What actually developed in Philadelphia, however, was quite different.

Crowding and congestion were issues in Philadelphia almost from the beginning. Although Philadelphia had undergone a considerable transformation by the time Penn left Pennsylvania in 1701, it was essentially an outpost where pigs and goats ran freely through muddy, garbage-laden lanes[23] and people lived in caves dug into the riverbank.[24] Philadelphia was much more congested than originally planned. Not only were the lots considerably smaller, but the vast majority of the population (some 2,000 people) lived along the banks of the Delaware. There was virtually no settlement west of Fourth Street. Within its narrow confines, the town had about four hundred houses, a brickyard, three breweries, at least six churches and as many taverns, tanneries, a market of open stalls at Second and High Streets, a ropewalk, four shipyards, and numerous riverfront wharves.[25] In a place so tightly packed, any increase in population meant an increase in population density. This was especially true as more and more houses were built in rows, and alleys were cut into the spacious blocks which William Penn had hoped would keep Philadelphia a "green country town." The population concentrated along the river in ever more congested housing patterns, and the housing codes added to the problem by not providing for adequate sanitation, proper lighting and suitable ventilation. This pattern worsened as the century progressed, and set the stage for the emergence and spread of infectious disease.

The first problem with this plan occurred when Penn's commissioners arrived late in 1681 to arrange for the 10,000 acres of the town. They found Scandinavian, Dutch and English settlers already in possession of most of the land along the Delaware. Having to abandon the original site, they moved further up the Delaware, where the Swansons of Wicaco were willing to sell 300 acres of land with a mile of river frontage between the area now bounded by Vine and South streets. When Penn arrived in the autumn of 1682, he was dissatisfied with this cramped site, and he purchased from two other Swedish farmers a mile of river frontage on the Delaware. The rectangle of 1,200 acres that resulted from this purchase measured one mile from north to south and stretched east and west for two miles across the narrowest point between the two rivers. Within this rectangle, Thomas Holme, Penn's surveyor general, laid out a grid of spacious blocks with an eight-acre public square in each quadrant of the town and a ten-acre central square for civic buildings at the intersection of two main thoroughfares (**Figure 3.1**).[26] Broad Street and High Street (now Market Street) were 100 feet wide, much wider than any street in seventeenth-century London.[27]

**Figure 3.1**
**William Penn's Plan for Philadelphia, 1683**




Source: Reproduced by Historic Urban Plans, Ithaca, New York, from an engraving in Olin Library, Cornell University.

While Philadelphia was larger in size than any other city in the colonies at this time, it was much smaller than Penn had intended. As a result, he was forced to reduce the size of the city lots. Fairly early in the city's history, Holme's spacious blocks were cut by alleys, and lots were divided and subdivided. The narrow workman's houses that were built on these lots gave the town an aspect reminiscent of crowded London. Penn's vision of a public esplanade along the high banks of the Delaware vanished, too, as merchants soon covered the area east of Front Street with warehouses, shops, and dwellings. In addition to these structures, wharves began sprouting out into the river.[28] In order to link the buildings they had erected along the

riverfront, merchants created a street not shown on Holme's plan of the city. This street was the narrow artery known as Water Street, which extended along the banks of the Delaware River.

Philadelphia's idealized design plan was showing signs of stress even before Penn's second visit. In London, Penn had dreamed of city plots hundreds of acres in size, but had settled for one-half or one acre holdings when he actually arrived in his colony. The plan had fitted forty-three lots into the premier stretch along the Delaware's Front Street, but before the 1680s were finished, they had already been subdivided into seventy. By 1703, the number had increased to more than one hundred, with some less than 20 feet wide. As the lots were cut up, a series of alleys and pathways began to develop to link the narrow houses placed on these restricted holdings. By 1698, nine alleys cut through the area between Front and Second Streets alone.[29] The Philadelphia waterfront soon became one of the most congested communities in the colonies. To further complicate the situation, as the city grew, it expanded north and south along the river, rather than westward towards the city center Penn had placed between the two rivers.

While the population of Philadelphia grew, the geographical boundaries of the city changed only slightly, creating the high density city blocks so characteristic of the city. In 1760, Philadelphia proper consisted of little more than two square miles, with 0.6 square miles actually settled (**Table 3.1**).[30] The development of new blocks in the city did not keep pace with the growth in population. Instead, the already

occupied blocks acquired more and taller structures in place of open yards and gardens. In so doing, they came to resemble those in the historic core of the city of London, which, by 1801, consisted of little more than one square mile of territory which contained more than 128,000 people.[31] In contrast, the wards bordering the Delaware River had densities as high as 93,000 persons per square mile circa 1800, while the outer wards that stretched toward the western boundary of the Schuylkill River were about two-thirds vacant (**Figure 3.2** ).

**Table 3.1**
**Population, Area and Density for Selected United States Cities**

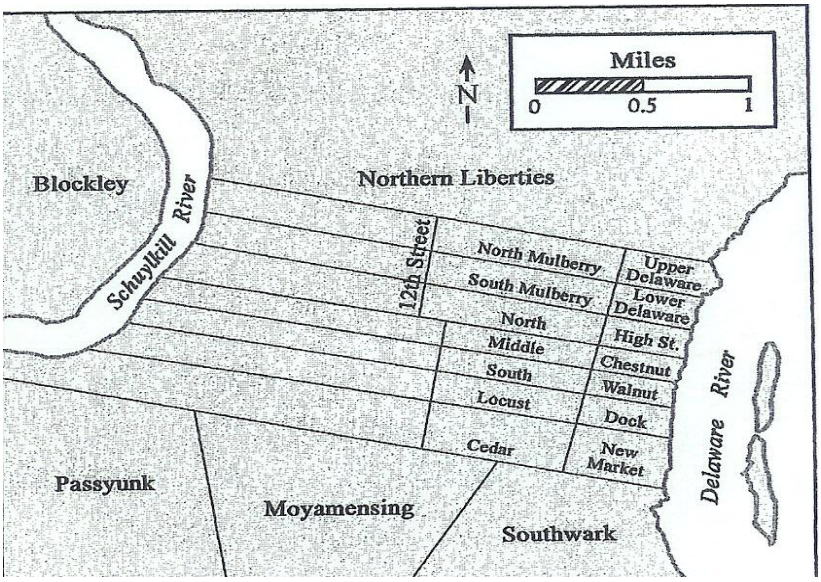
| City | Year | Population | Area | Density |
|---|---|---|---|---|
| | | | | |
| Philadelphia | 1760 | 15,000 | 0.6 sq. mi. | 25,000 |
| Philadelphia | 1790 | 28,522 | 0.7 | 40,746 |
| Philadelphia | 1794 | 32,983 | 0.8 | 41,229 |
| Philadelphia | 1800 | 41,220 | 0.9 | 45,800 |
| | | | | |
| New York | 1771 | 21,863 | 1.1 | 19,875 |
| New York | 1790 | 32,328 | 1.0 | 32,328 |
| New York | 1800 | 60,489 | 1.5 | 40,326 |
| | | | | |
| Baltimore | 1790 | 13,503 | 0.6 | 22,505 |
| Baltimore | 1800 | 26,514 | 0.8 | 33,143 |
| | | | | |
| Boston | 1770 | 15,520 | 1.2 | 12,933 |
| Boston | 1790 | 18,038 | 1.2 | 15,032 |
| Boston | 1800 | 24,937 | 1.2 | 20,781 |

Source: Table is from Carole Shammas, 2000, "The Space Problem in Early United States Cities," p. 507.

Settlement had progressed no further than 12th Street[32] by the early years of the nineteenth century (**Figure 3.2**).[33] Although the population spread along the

banks of the Delaware River beyond the city limits into the Northern Liberties and Southwark, population density had neither declined nor stabilized in the city proper. Instead, proprietors kept subdividing their own lots, creating residential units in alleys behind the large boulevards rather than expanding further out in the direction of the Schuylkill River. From 1760 to 1800, the city added more than 1,000 people and 228 new dwellings annually, yet Philadelphians developed new land to accommodate this increase at a rate of 5.3 acres (about one small city block) per year.[34] In 1798, for example, Philadelphians had allocated only 203 acres for the 5,354 houses and 4,019 outbuildings (mostly kitchens) that made up the domestic, work, and outdoor space for humans and animals.[35] Simple arithmetic produces an average house lot size of 1,652 square feet with 7.1 persons per dwelling. Not only did more people live in each developed block, but more people crowded into city dwellings than did those in the Northern Liberties and Southwark. Philadelphia's back alleys and crowded households produced the density levels observed in Table 3.1.[36]

**Figure 3.2**
**Settled Areas of Philadelphia by Ward, 1800**




Source: Map is from Carole Shammas, 2000, "The Space Problem in Early United States Cities," p. 511n.

Some idea of crowding in Philadelphia can be derived from the 1798 Federal Direct Tax of dwellings. In Philadelphia's High Street Ward, the median house lot was 1,038 square feet. Most of the smaller lots had been carved from backyards, with access provided by alleys and lanes. The median width of the lots fell a few feet below the standard of 20 feet. This unusually small width helps explain the small size of lots in Philadelphia, and the rapid rise of three- and four-story buildings. Houses, with a few exceptions, took up the entire width of lots in this ward, a practice that was becoming common citywide. In High Street Ward, lots had a median length of about 70 feet, with less than half of that amount taken up with the house proper. The median ground floor "footprint" of 455 square feet rose to 1,228 square feet total interior floor space, because of multistory building. A shop usually occupied at least

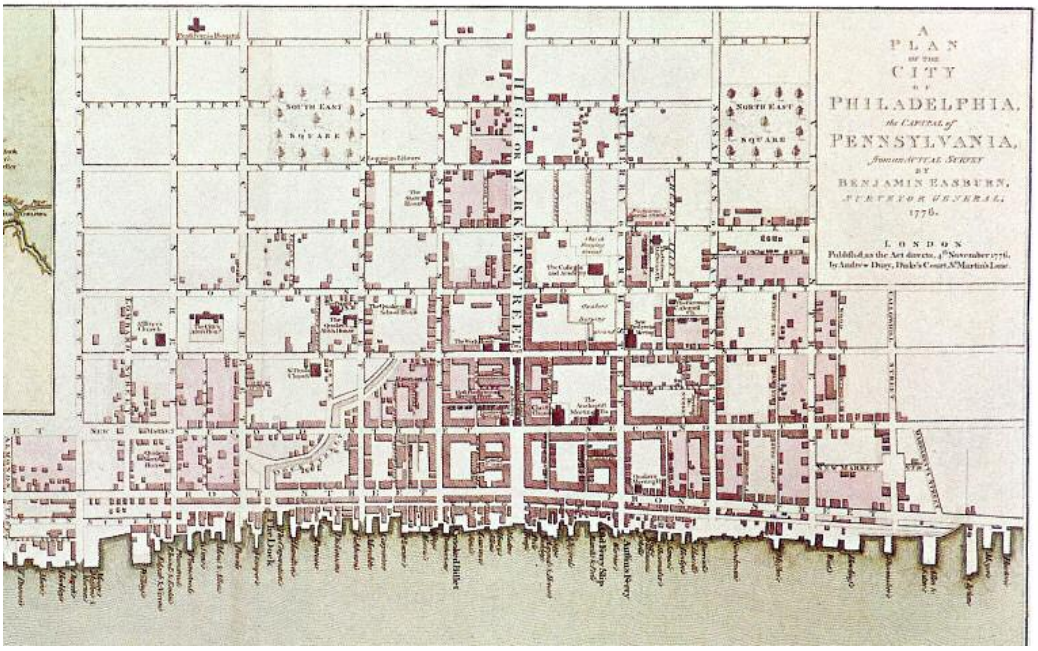
part of the first floor; the rest was used more exclusively as living quarters for the seven to eight people on average who lived in the structure. Almost all houses in the ward were brick. In Philadelphia, the shift to brick construction had been facilitated during the middle years of the eighteenth century by the refusal of insurance companies to underwrite wooden houses.[37]

One or more outbuildings often stood behind the main house. In High Street Ward, for example, 158 house lots supported 212 outbuildings. Approximately three quarters of the house lots had at least one outbuilding. Some of these structures were workshops or buildings associated with the trade of the occupants, while others were coach houses, stables or woodsheds to keep animals and provisions. As Carole Shammas points out in her study of space problems in early U.S. cities, a backyard consisting of 400 square feet may not seem unusually cramped, but most of us today do not conduct an industry or commercial enterprise in our living rooms and patios; keep a horse, cow or pig in the garage; maintain privies, waste dumps, wells, and water pumps outdoors, or dedicate the edge of our lots to an alley providing access to a back rental unit.[38]

In addition to outbuildings, the increased demand for housing forced landowners to build dwellings behind their dwellings. The evolution of Benjamin Franklin's house and lot was typical. Franklin owned a house lot off of Market Street in the second block of the ward where he built three rental houses that faced Market Street. This arrangement turned his home yard into an interior lot.[39] Letitia Court

developed in an almost identical pattern. In 1701, Penn gave his daughter Letitia the northern half of the block from Front Street to Second Street in Chestnut Ward. Initially, Letitia's house well represented Penn's plan for the city – a detached residence surrounded by gardens and orchards. By mid-century, the lots near Letitia Court began to be subdivided. The development moved slowly, but by 1769, the greatly reduced lot contained a series of structures including tenements that fronted on Second Street and Letitia Court, three tenements in the back, and an additional tenement that joined the back building. By 1800, twenty-four lots and house owners replaced the original one, with five of these lots having frontages on a court or alley and two additional ones showing dual frontages on Letitia Court and Second Street.[40] This pattern of development – increasing numbers of alleys providing passage from house to street – became a familiar site in Philadelphia, and greatly increased congestion within the city. Elizabeth Drinker notes that "many worthy persons are pent up in small houses with little or no lotts."[41] Any increase in congestion meant an increase in contact probability (**Figure 3.3**).

**Figure 3.3**
**Plan of the City of Philadelphia, 1776**




Source: This image was scanned from a facsimile printed in Martin P. Snyder, 1975, *City of Philadelphia: Views of Philadelphia before 1800*, p. 67.

Note: The map is entitled *A Plan of the City of Philadelphia, the Capital of Pennsylvania, from an Actual Survey by Benjamin Easburn, Surveyor General; 1776*. It well illustrates the congested city blocks in the center of the city and along the banks of the Delaware River.

The residents of Philadelphia, to judge by their papers and publications, rarely complained about crowding itself. Rather, any mention often related to some specific problem, most often one that appeared to them to be life threatening. The yellow fever epidemics of the 1790s, for example, prompted many of the calls for action against crowded districts. Ironically, density probably killed fewer people through yellow fever than through endemic maladies that people often attributed to poor constitution, not the environment.[42] Yellow fever is transmitted by the bite of an *Aedes aegypti* mosquito infected about fourteen days earlier by feeding on a viremic

person. Since the mosquito has a flying range of a few hundred yards, densely settled cities were necessary for the spread of the disease.[43] Yellow fever epidemics arose when certain conditions prevailed: the presence of the virus, the insect vector, a sufficiently large number of infected and vulnerable people, and a warm and moist climate. Without all of these, density alone would not produce the disease. Nevertheless, population density was a significant factor in the spread of many infectious diseases in Philadelphia. While evacuation, quarantine, and removal of stagnant pools of water were some of the measures that helped to put an end to yellow fever epidemics in Philadelphia, the improvements in crude death rates resulting from these and other measures masked a much more serious problem. Endemic disease, notably tuberculosis, respiratory infections and dysentery[44] routinely killed more urban residents during the eighteenth century[45] than did yellow fever. Research has shown that these diseases are linked to crowded living conditions. A study of mortality in United States cities in 1900, for example, found that density (measured as either persons per acre or persons per dwelling unit or both) is positively correlated with deaths from tuberculosis, pneumonia, and diarrheal diseases.[46] Philadelphia's densely populated city blocks undoubtedly were a contributing factor to the high rates of death associated with these and other diseases.

Climate also played a significant role in the morbidity and mortality patterns in Philadelphia. Those individuals who migrated to America encountered a very different climate than that of Western Europe. The climate of Western Europe was oceanic in nature, with relatively small daily and seasonal fluctuations in temperature,

and adequate rainfall in all seasons. The situation in the eastern portion of North America, however, was quite different. This region had a continental climate, with far greater temperature extremes. Summers were warmer and more humid and winters were colder.[47] The rainfall patterns were less predictable, river and stream levels varied greatly with the season and with cycles of high runoff or rainfall and drought. The streams which ran through the city were little more than open sewers where waste of all types was dumped. The streams flowed into the Delaware River where the docks stretching out into the water prevented the current from sweeping away the pollution deposited there and allowed pools of stagnant water to become ideal breeding grounds for mosquitoes. Consequently, the contaminated water increased the probability of contact between people and water-borne pathogens, while the increase in the mosquito population facilitated the spread of malaria and yellow fever.[48]

The 1750s was a significant period in the city's history as it marked the decade with the highest average crude death rates (CDR) of the century. The period witnessed repeated outbreaks of both smallpox and typhus, along with the constant presence of "fever, flux and fits" as well as tuberculosis and worms.[49] The year 1759 was particularly deadly with epidemic outbreaks of smallpox, measles and typhus. This was a time when animals and livestock still roamed the streets, and some of these streets still had stumps or roots of the original pine trees sticking up. Since there were no paved roads in the city, in wet weather they became small lakes because of lack of drainage. Carts would bog down, and anyone walking might sink

to their calves in the mire.[50] More importantly, the stagnant water created an ideal environment for mosquitoes, which were the vectors for yellow fever and malaria. Although Philadelphia is an unlikely location for widespread malaria in modern times, the disease was, nevertheless, endemic in the city during the eighteenth century. Elizabeth Drinker made almost constant reference to "chills and fever" in her diary, as no member of her family escaped the disease.[51] She writes on August 17, 1785, "we had each of us another fitt of Chill and Fever, worse than the former, sent for Docr. Kuhn, took the bark, stop'd it in me, Nancy [daughter] had a 3d. fitt, but by taking bark in larger quantity stop'd it."[52] Increased population density certainly facilitated the spread of malaria and yellow fever. The migration of infected individuals, and the contemporary ignorance of the roles that mosquitoes and stagnant pools of water played in the transmission of disease, however, permitted widespread infections.

Benjamin Rush observed that there seemed to be a higher incidence of disease as the city grew larger, and thought that perhaps this was linked to the clearing of woods and land. The phenomenon was not limited to Philadelphia, however. He wrote in 1785: "It has been remarked that intermittents [fevers] on the shores of the Susquehannah have kept an exact pace with passages which have opened for the propagation of marsh effluvia, by cutting down the wood which formerly grew in the neighborhood."[53] The principal vector of malaria in the eastern two-thirds of the continent was *Anopheles quadrimaculatus*. This mosquito develops along the edges of permanent pools, lakes and swamps that provide relatively clean, still, sunlit water,

with lush emergent vegetation, marginal brush, or floating debris to provide shade and protection from wave action.[54] The area in and around the settled portions of Philadelphia provided just such an environment for this mosquito to breed.

While the swampy landscape of Philadelphia provided optimal conditions for many species of mosquito, water-borne trade enhanced the economy of the city. The Dock area was both a particularly sodden, and a particularly prosperous, part of town. Dock Creek was an inlet leading off the Delaware River into what became the heart of colonial Philadelphia. The creek served as a manufacturing-processing center for the growing city, and as such, it was the source of much pollution. By 1699, two tanneries were operating there, and by 1739 there were six.[55] As the dock became so dangerously polluted as to be linked with deadly disease, it moved from being the useful and pleasant center of a town to being a center of civic dispute. Tanning, however, was only one of several industries that contributed to pollution levels in Philadelphia. Some of the city's most noteworthy - and most noxious - commercial enterprises included distilling, saltpeter manufacturing,[56] butchering, soap-boiling and sugar refining. Each increased the probability of contact between a susceptible host and an infectious agent by 1) altering the physical environment in such a manner as to make it more hospitable for pathogens and vectors to proliferate, and by 2) bringing workers into close contact with mosquitoes, animals, contaminated water and each other. The paraphernalia used in sugar refining and transport, for example, was an important factor in the nurturance and travel patterns of the yellow fever mosquito.[57] Furthermore, as day feeders, the females in their search for blood meals were

attracted to the lactic acid oxidation products in human sweat. Their most active times coincided with human activities in the early morning and late afternoon, and they hunted their prey anywhere between two inches and three feet above the ground.[58]

Philadelphia, though large for its time, was small by modern standards, and people from all walks of life lived in densely populated blocks in a narrow strip of land along the Delaware River. Workplaces and businesses were small; even the shipyards, ropewalks, and breweries had no more than five to ten people working as a group. The one-man shop was the basic unit of the economy, and it was an experience shared by both artisan and merchant. Artisans either hired themselves out by the job or worked alone in small shops in their homes, where they made and sold their goods.[59] Merchants often worked with the help of one partner and a clerk, with some of the wealthiest living above their counting houses.[60] In addition, there was no residential and commercial zoning at this time. Tanneries and breweries coexisted in the same block with mansions and modest dwellings, many with a shop on the first floor. The mix of land uses certainly increased the probability of contact between residents and potential agents of disease; pathogens, pollutants, chemicals, etc.

Disease patterns in the city were not constant, but changed in response to variations in both people and place. Seen from this perspective, disease in Philadelphia was less an expression of abnormality and pathology and more a mirror of the precarious balance that existed between human beings as biological organisms

and the physical world they inhabited.[61] Smallpox, for example, was the greatest killer of Philadelphians during the third quarter of the eighteenth century, accounting for most of the annual fluctuations in the death rate before the Revolutionary War.[62] The decreasing virulence of smallpox after the 1770s most likely resulted from the disease becoming endemic in the city. As population levels increased, density-dependent diseases like smallpox tended to become endemic in the population and common in childhood. As such they were less lethal than when they afflicted adolescents and young adults. Popular belief notwithstanding, the changes in patterns of this disease (as well as other diseases) at any given time may have had less to do with medical practice, or even conscious decisions by public health authorities, than on environmental factors. As the environment changed, the incidence of specific diseases changed, often radically.[63]

## Changes in Human Demographics and Behavior

Population growth and upheavals caused by migration and war were likely factors in disease emergence in eighteenth-century Philadelphia. Pennsylvania had a specific attraction for Western Europeans who were being persecuted in their own communities, and Philadelphia was their port of entry. Thousands of Germans began to immigrate to Philadelphia every year, starting in 1708. Beginning in 1717, descendents of Scots from the north of Ireland began to pour into the port of Philadelphia as well.[64] As the century progressed, immigration rates increased. The city experienced the arrival of approximately twenty-six thousand Irish and Scotch

Irish and nearly forty thousand German newcomers between 1750 and 1775. The long voyage, lasting from six weeks to six months killed many and left others in a weakened or dying condition on arrival at the city's docks. Disease plagued passengers during many of the voyages as typhus, dysentery and typhoid fever flourished in the over-crowded, unsanitary conditions on the ships.[65] This level of immigration to the tightly packed city blocks of Philadelphia, along with the sickly state of many of the newcomers helped to create a critical level of contact probability in the city.

As a result of this massive immigration into the city, population levels soared. The city's population multiplied by over six and one-half times between 1720 and 1775, a growth rate of 3.5 percent per year. Most of that growth resulted from immigration, although natural increase also became important after 1760.[66] This growth had a profound influence on morbidity and mortality rates in Philadelphia. The extent to which the demise of migrants inflated the city's death rate, however, can only be estimated. Benjamin Franklin considered the burials in Philadelphia's Strangers' Ground during the 1740s to have consisted primarily of immigrants who died from shipboard disease.[67] However, while the graveyard undoubtedly served as the primary immigrant cemetery, burials there included many paupers as well.[68] Historian Billy G. Smith estimates that the deaths of immigrants accounted for about 50 percent of the Strangers' Ground interments or approximately 14 percent of the city's burials during the second half of the century.[69]

Although immigrant deaths added directly to the city's mortality rates, transients carried and disseminated infectious disease throughout Philadelphia. Lice and unsanitary water and sewage disposal systems helped to spread typhus[70] and typhoid fever[71] throughout the city. Smallpox infected practically any susceptible person with whom migrants came into contact.[72] And regardless of attempts at quarantine precautions, infectious immigrants brought disease carrying mosquitoes into the city, as well as fed the resident mosquito population thriving in the nearby marshes, thereby enabling yellow fever and malaria to spread among the inhabitants.[73]

The repercussions from the Seven Years War had a significant, albeit indirect, impact on immigration patterns. The War broke out in 1756, and the ensuing blockade abruptly cut off German immigration to Pennsylvania, although it did not interfere with emigration from Ireland until later. One immediate consequence of this situation was the conscription by the British military of the indentured servants working in the colony. In order to meet the shortage of labor, Philadelphia merchants began importing enslaved people from Africa and the West Indies. Although the Assembly opposed the slave trade, and repeatedly passed laws levying duties on the importation of these people, the trade in human cargo boomed nonetheless.[74] The slave ships on the infamous "Middle Passage" from West Africa to the West Indies or to the colonies directly were notorious death traps, and many on board often carried disease.[75] In the fall of 1762, a severe epidemic of yellow fever broke out in

Philadelphia. John Redman, a prominent physician in the city, believed it spread from ships coming from the West Indies.[76]

Population growth in Philadelphia, along with birth and death rates, were quite extraordinary by eighteenth-century standards. In order to place Philadelphia's demographic characteristics into perspective, historian Billy G. Smith compares Philadelphia's "vital rates" to that of two cities of nearly equal size – Boston, Massachusetts and Nottingham, England – along with the New England Village of Andover.[77] In his analysis, Smith notes that Philadelphia's population increased more rapidly than that of Nottingham, Andover, or Boston. During the third quarter of the eighteenth century, for example, Philadelphia's 3.4 percent annual growth rate was well above the 1.3 percent of Nottingham and the 1.7 percent of Andover, while Boston's population declined slightly. In comparison to Nottingham, both migration and natural increase accounted for Philadelphia's higher growth rate. Because of its low mortality, Andover exceeded Philadelphia in rate of natural increase, but emigration from the New England town created a lower rate of population growth.[78]

Birth rates were also much higher in Philadelphia than in either Andover or Nottingham. This was due, at least in part, to the younger age structure in Philadelphia created by the tremendous amount of immigration into the city. In contrast, Nottingham and Andover had fairly static populations with relatively little in-migration. While Andover's birth rate declined, Nottingham's birth rate increased throughout the century. To further highlight the phenomenal nature of Philadelphia's

"vital rates," death rates were also higher in Philadelphia than in Boston, Nottingham or Andover. Andover's rates were well below those of the cities, reflecting the distinct difference between rural and urban areas. These differences reflect the fact that a high population density was more conducive to the spread of disease, a significant factor in Philadelphia's high death rates. High rates of immigration also swelled Philadelphia's death rates, most notably through deaths of immigrants and by the spread of imported disease to the inhabitants of the city. In contrast, the lower death rates in Nottingham and Boston reflected, at least in part, the relative absence of immigration to these cities. Comparatively speaking, Philadelphia was an extremely hazardous place in which to live, even by eighteenth-century standards. The city was considerably unhealthier than rural areas in the American colonies, and to some degree, unhealthier than many European cities.[79]

Oddly enough, the high mortality rates associated with epidemics that appeared irregularly or seasonally generally had a much smaller impact on population size than did mortality from endemic diseases.[80] Epidemic diseases often made spectacular appearances and, in turn, made distinct and lasting impressions on the people. The history of yellow fever suggests that public fears and apprehensions had little relationship to the actual impact or demographic significance of a specific outbreak. When mortality from endemic diseases was regular and predictable, there was relatively little concern; death was accepted as a part of life. The matter-of-fact manner in which Elizabeth Drinker details the many diseases that plagued her family is strong evidence to support this claim. On July 20, 1772, she wrote "HD. and MS.

[husband Henry and sister Mary] road out this morning with our little Henry, which they have done several times lately, as he has been very unwell, with a vomiting and lax, and much troubled with Worms. MS. went after dinner, George Baker with her to Mt. Hall, to see HS. HD. went for her towards Evening they drank tea there."[81] In contrast, when yellow fever epidemics appeared at irregular or seasonal intervals and resulted in mortality spikes, public fears often reached a fever pitch. On August 27, 1793, Drinker wrote "there is great cause of serious alarm, the yallow-Fever spreads in the City, many are taken of with it and many with other disorders - Jacob Downing[son-in-law] is better, my dear Billy very poorly, I am much distress'd that any of our family continues in town."[82] Under these circumstances, community life was disrupted, and fear could even be "heard" in the words of the usually unflustered Elizabeth Drinker.

Although Philadelphia's population density contributed to the rise in incidence and prevalence of several chronic diseases, it played a particularly significant role in the spread of tuberculosis. In reviewing Philadelphia's bills of mortality, Peter Kalm – a foreign visitor to the colonies – concluded that "consumptions, fevers, convulsions, pleurisies, haemorrages, and dropsies" were the leading causes of mortality in Philadelphia between 1730 and 1750. Another contemporary estimated that 19 percent of all deaths in the city in 1787 were due to this illness.[83] By the late eighteenth century, tuberculosis and other pulmonary disorders may have been the leading causes of death in the new nation. In his history of epidemic diseases, Noah Webster wrote that "pestilence has always been the

peculiar curse of populous cities." Of 200 general plagues, he added, "almost all have been limited to large towns." Webster's concept of crowding, although accurate, was incomplete. Tuberculosis and pulmonary disorders were not confined to more populous communities; they were present in rural communities as well. The critical element was not total population, but household size.

Historian Carole Shammas estimates that a typical dwelling in Philadelphia during the last quarter of the eighteenth century contained 6-7 people.[84] Such crowding facilitated the household transmission of mycobacterium (the bacterium that causes tuberculosis) and other organisms, while other elements merely compounded the risks of contagion. Relatively inefficient heating, for example, led inhabitants to seal doors and windows during cold weather. Physicians added to the problem by advising against opening windows even in warmer weather. In addition, caretakers often slept in the same bed with their patients. As such, these behavioral patterns certainly contributed to the spread of tuberculosis and pulmonary disorders during these decades. Also, migration from England – where the incidence of these diseases had reached unprecedented heights – added to the risk of contagion in the colonies.[85]

One particular condition that provides fascinating insight into the complex relationships between pathogens and humans - and demonstrates the crucial role of environment in shaping morbidity and mortality patterns - is war. The French and Indian War certainly altered immigration patterns during the middle years of the

century, but the Revolutionary War underscored the delicate balance that existed between people and their environment. Although the city was occupied by the British during the years 1777 and 1778, and may very well have been considered a city of refugees, the most significant events may have occurred in the military camps. The War brought large numbers of young males together in crowded quarters that lacked even rudimentary sanitary facilities. The potential for contaminating water supplies from both animal and human wastes was significant. Since many of the recruits came from rural areas, they were never exposed to the endemic diseases common in Philadelphia. In addition, inadequate attention was given to the basic necessities such as food, clothing and shelter. Neither camp nor personal hygiene was given high priority by the military leaders, even though authorities were aware of their significance.[86] It is not surprising, then, that the impact of infectious disease was significantly magnified.

At the beginning of the Revolutionary War, smallpox emerged as a major health problem, and large numbers of previously unexposed soldiers died of the disease. The ravages of the disease drastically impaired military effectiveness. Consequently, military authorities in 1777 took the unprecedented step of ordering the inoculation of all recruits in order to reduce the threat of epidemics. From 1776 through 1778, the combination of cold weather, clothing and food shortages, crude housing, and inadequate sanitation and hygienic practices increased vulnerability to other infections.[87] The vast majority of all deaths were due to disease and not war-related injury. Respiratory disorders and dysentery were the two leading causes of

mortality, with typhus and typhoid fever also present, though less significant. Venereal diseases, scurvy, scabies, and other infectious disorders and inadequate nutrition added to the health burden of the military, and undoubtedly increased vulnerability to other infection. Given the nature of recruitment and the circumstances of military life during the Revolutionary War, it is reasonable to conclude that mortality was overwhelmingly a function of the interaction of environmental conditions, pathogens, and human hosts.[88]

Human behavior can have important effects on disease dissemination. The wars of the eighteenth century were just some of the many factors in a century-long process that realigned the variables determining the probability that the people of Philadelphia would encounter disease. Nevertheless, the city's stunning rates of population increase, levels of urban density and waves of migration, factors profoundly influenced by a variety of human actions, played perhaps the most significant role in shaping the contours of death and disease in the city.

## Commerce

Disease, particularly infectious disease, became more and more significant in Philadelphia as the eighteenth century progressed. This is not to imply that disease was absent in the early years of the century. During these early years, environmental conditions limited the significance and spread of infectious diseases, and prevented them from becoming established in either epidemic or endemic form. Philadelphia

had begun to develop economically by 1720, however, and both domestic and foreign commerce reduced the relative physical isolation of the town. Economic growth was accompanied by rapid population growth and increased contact with European, Caribbean and African ports. As a result, the harsh disease environments so characteristic of a large part of the settled world were partially recreated in Philadelphia. Commercial contacts spread infectious diseases, and a growing population provided a pool of susceptible persons to sustain the chain of infection.[89]

With the possible exception of dysentery, respiratory illnesses were among the leading causes of death in the eighteenth century. Most of these were endemic and seasonal in character. The growth of the population and the expansion of trade began to render the colonies somewhat more vulnerable to influenza epidemics and pandemics, however. At the beginning of the eighteenth century, distance protected the colonies from outbreaks occurring elsewhere in the world. The European epidemic of 1708-09, for example, did not reach the American colonies. In 1732-33, however, influenza was prevalent in the Northeast and Middle Atlantic colonies. It first appeared in New England and reached Philadelphia by November.[90] This may have been a late flare-up of the 1729-30 pandemic that began in Russia and moved westward through Europe.[91] Indigenous influenza outbreaks also occurred in many colonies. An epidemic broke out in Philadelphia in the winter of 1770-71 and recurred the next winter.[92] By the close of the eighteenth century, the newly independent colonies had become part of a larger, global disease pool. During the

years 1781-2 and 1788-9, for example, influenza appeared in pandemic form, affecting millions of people in both Europe and America.[93]

While certainly the unhealthiest urban center on the east coast during the second half of the eighteenth century, Philadelphia was also the wealthiest American urban center. The city's economy rested squarely on the foundation of commerce.[94] Most workers, directly or indirectly, depended on trade with people scattered throughout the Atlantic world, from small farmers and storekeepers in the neighboring countryside to large manufacturers and merchants operating from the West Indies to London. Philadelphia's immigrants both created a market for the imported goods of merchants and produced commodities that the merchants could profitably export. These commodities included wheat, flour, bread, meat, flaxseed, furs, lumber, barrel staves and iron. By the 1750s, the city's import-export trade was booming, and its warehouses along the Delaware were bulging with Irish linens, Portuguese wines and Madeira, West Indian rum and molasses, and fine English woolens, cutlery, and ceramics.[95] Together with merchants and their clerk assistants, the men employed in the commercial sector constituted close to one-third of the free workforce.[96] Many other people relied on trade in some form or another. For example, distillers and sugar boilers used West Indian molasses and sugar; coopers fashioned barrels to hold items bound for the sea; and innkeepers and tavern keepers catered to men who moved merchandise across the roads and the sea. The commercial development opened the way for merchants, shipbuilders, sailors, barrel makers, sailcloth tailors, ropes men, dockworkers, and ship insurance agents to

congregate in the narrow alleys and crowded streets of Philadelphia.[97] This development also opened the way for imported disease to "congregate" in these same narrow alleys and crowded streets.[98]

## Deficiencies in Public Health

Poor sanitation and lack of proper hygiene contributed to the transmission of many infectious diseases in Philadelphia. Historian Michal McMahon argues that there existed in the eighteenth century a practical belief in the relationship of disease and health to environmental conditions. After the outbreak of yellow fever in 1741, for example, the colonial Assembly mandated that doctors visit arriving ships and confine sick passengers (those thought to have carried the disease into the city) in a newly constructed lazaretto on an island in the Delaware River. In the early 1750s, a physician named Thomas Bond linked pollution and imbalance in the environment to imbalance in the patient, and urged a campaign to clean up the city. His concerns found strong reinforcement during the 1762 yellow fever epidemic.[99] This experience initiated the first comprehensive efforts by the Corporation and the Assembly to organize paving and cleaning of streets, removal of wastes, and extension of the city's drainage system. During the 1780s, city officials responded to decades of controversy over the polluted system when they converted the upper third of Dock Creek into a drainage sewer. By that time, the stream served as little more than the collective sump for Philadelphia's wastes.[100]

With the health of the community most clearly on the minds of the city leaders during times of epidemic outbreaks, both internal improvement campaigns and struggles to ameliorate what they believed lay behind the current crisis brought to the forefront a commonly held belief – disease was caused by a filthy urban environment and a dangerously polluted water supply. Yellow fever and diseases like smallpox, malaria and consumption, however, were not directly caused by filthy streets and polluted water. As a result, the city's effectiveness in controlling the spread of these and other infectious diseases was marginal at best. Furthermore, many of these campaigns were blatantly ignored by city residents. Regardless of the city's efforts, pigs, dogs, and rats still roamed freely in 1793 to feed on the garbage in the streets, while residents commonly disposed of their refuse and excrement in the alleys and gutters in front of their homes.[101] The openings in sewers "exhale the most noxious effluvia," according to one contemporary, "for dead animals and every kind of nausea are thrown into them, and there remain till they become putrified."[102] And Dock Creek, a long standing nuisance, continued to be a foul-smelling cesspit in the heart of the city. Not surprisingly, mortality during yellow fever outbreaks was extremely high among people who lived near the creek where mosquitoes thrived.[103]

Classical public health and sanitation measures have long served to minimize dissemination and human exposure to many pathogens spread by traditional routes such as water or preventable by immunization or vector control.[104] Pre-industrial cities like Philadelphia, however, had very little in the way of effective public health measures to combat disease. While the introduction of inoculation and vaccination

undoubtedly contributed to the decline of epidemic smallpox by decreasing the number of susceptible individuals, the combination of public apprehension concerning the procedure, a failure to recognize the need for revaccination, and the large tide of migration from Europe which augmented the pool of susceptibles, tended to facilitate periodic outbreaks. Although terrifying, these and other epidemics were not a major influence on aggregate morbidity and mortality rates in the city.[105] The greatest threat to urban life in eighteenth-century Philadelphia was from endemic infectious disease that flourished in the densely populated and unhygienic environment; tuberculosis being one of the more significant. High housing density, a susceptible population, and the migration of infected individuals from England facilitated the spread of the disease in both rural and urban communities. None of the public health measures adopted in the city served to ameliorate the conditions which were conducive to the spread of this disease. Although Philadelphians attempted to control outbreaks of smallpox and yellow fever by cleaning the city, they neither understood the cause of these infections nor that of endemic disease such as tuberculosis. As a result, genuinely effective public health measures were simply non-existent in eighteenth-century Philadelphia.

The health of any population is primarily a product of ecological circumstance: a product of the interplay of human societies with their wider environment, its various ecosystems and other life-support processes. Ecological interactions can be complex, with several factors often working together or in sequence. Cultural, social, economic and political conditions all played a

fundamental role in modulating the ecological opportunities for infectious disease in Philadelphia. The most important factors, however, were those associated population growth, population density and population mobility. As the principal American port during the eighteenth century, Philadelphia experienced the arrival of several thousand immigrants from Europe, Africa and the West Indies. Still, none of the factors outlined in this chapter would have had epidemiological significance if few of these immigrants had been susceptible to disease. This is discussed more fully in Chapter 4.

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[1] An *agent* is defined as a factor, such as a microorganism or a chemical substance, whose presence, excessive presence, or relative absence is essential for the occurrence of a disease. A disease may have a single agent, a number of independent, alternative agents, or a complex of two or more factors whose combined presence is essential for the development of the disease. See Last, 2001, p. 3.

[2] Philadelphia's total environment included its physical, biological, cultural, political, socioeconomic and historical universe.

[3] The sources of microbes that cause disease in humans are generally another human, an animal or arthropod vector, the soil, tainted food or water. A general estimate of the relative importance of different modes of transmission can be obtained by looking at the breakdown of infections causing death in the world today, as reported by the World Health Organization. In 1996 for example, 65% of infectious diseases causing death were spread from person to person (e.g., influenza, AIDS, tuberculosis, hepatitis B), 22% originated from food, water, or soil (e.g., cholera, salmonellosis), 13% were transmitted by an arthropod vector (e.g., malaria, dengue), and .3% came directly from animals (e.g., rabies). See M.E. Wilson, 2000, p. 7.

[4] Threshold phenomena are events or changes that occur only after a certain level of a characteristic is reached. See Last, 2001, p. 179.

[5] Studies of colonial Philadelphia have shown the high cost in human lives that resulted from crowding European immigrants into one eighteenth-century port city. Africans and Europeans, however, faced dissimilar risks to life and health in Pennsylvania's capital. See Klepp, 1994, 473-4.

[6] Morse, 1995, p. 7.

[7] The consequences of rapid growth, including an inability to adequately quarantine shipping, a dense urban environment, contaminated water, and improper disposal of untreated sewage, all would have contributed to the spread of infectious disease. They presumably would affect immigrants more severely than natives since the over-all health of new arrivals was already compromised. As a result, the first to experience the ravages of infectious epidemic diseases were urban port communities, which brought significant numbers of susceptible individuals into close living conditions. The maritime character of Philadelphia, Boston, New York and Charleston – the most important colonial ports – brought these communities into contact with each other and, more importantly, with Europe, the Caribbean, and Africa. The ports also tended to be the entry point for both sailors and individuals migrating to the colonies. Such population movements became the means of transporting a variety of pathogens capable of causing disease.

[8] In 1754, spurred by the death of over 250 new arrivals, government authorities appointed a team of doctors to visit arriving ships and the places where ailing immigrants lodged. The physicians reported their findings to the governor, criticizing the Assembly for not having "made the necessary regulations to prevent malignant Diseases being generated by these people, after they came into port, where there

is more danger of it than at sea." See Bond, 1912, "A Colonial Health Report of Philadelphia, 1754," p. 479. The Assembly reacted by imposing much tighter restrictions on vessels, since "infectious Distempers have, notwithstanding previous laws, been introduced and spread in this province." See "Minutes of the Provincial Council," Colonial Records of Pennsylvania, Vol. 6, p. 345.

[9] As the principal American immigrant port during the period, Philadelphia experienced "the arrival of a trickle of English, approximately 26,000 Irish and Scotch Irish, and nearly 40,000 German newcomers between 1750 and 1775." After pausing during the Revolutionary War, migration resumed as the French and Irish merged with groups leaving the American countryside to create a new influx of migrants to Philadelphia during the last fifteen years of the eighteenth century. Since it was a walking city with no public transportation, every increase in population meant an increase in population density. Houses were built in rows and back alleys were cut into the spacious blocks – especially after 1780, as the population concentrated along the river in ever more congested housing patterns. See Smith, 1990, p. 42.

[10] Infectious agents vary widely in their ability to survive, and the critical population size necessary to sustain endemicity varies with each infection. Smallpox and measles, for example, require large populations in order to become endemic. Yellow fever, unlike measles and smallpox, is spread by mosquito rather than by direct person-to-person transmission. In its homeland, Africa, as well as in South America it is found in monkeys, which substantially increases the size of the reservoir and chances for transmission. In eighteenth-century North America there was no animal reservoir and the disease was one of urban populations. Large influxes of new susceptibles were required in order for the disease to be maintained. Unlike measles and smallpox, transmission could be interrupted by eliminating the mosquito. See Kunitz, 1984, p. 560.

[11] The decade of the 1790s was not the worst in the city's history despite yellow fever outbreaks in 1793, 1794-5, 1797, 1798 and 1799. From 1690 to 1759, deaths exceeded births, and decadal death rates, bolstered by a number of epidemics of smallpox, measles, typhus, typhoid fever, scarlet fever, whooping cough, diphtheria, influenza, yellow fever and other diseases surpassed the rates of the 1790s.

[12] The convergence of any number of factors can create an environment in which infectious diseases can emerge and become rooted in society. A model was developed to illustrate how the convergence of factors in four domains impacts on the human-microbe interaction and results in infectious disease. Ultimately, the emergence of a microbial threat derives from the convergence of 1) genetic and biological factors; 2) physical environmental factors; 3) ecological factors; and 4) social, political, and economic factors. As individual factors are examined, each can be envisioned as belonging to one or more of these four domains. See Smolinski, Hamburg and Lederberg, 2003, "Executive Summary," p. 4. Although this model has been developed to study the emergence of infectious diseases in the twenty-first century, much is applicable to the study of disease in past populations.

[13] In general, changes in the environment tend to have the greatest influence on the transmission of microbial agents that are waterborne, airborne, food borne, or vector-borne, or have an animal reservoir. See Smolinski, Hamburg and Lederberg, 2003, "Executive Summary," p. 4. In Philadelphia, vector-borne diseases included yellow fever and malaria, food borne diseases included dysentery and typhoid fever, water borne diseases included typhoid fever and possibly dysentery, and airborne diseases included smallpox, influenza, tuberculosis and measles.

[14] Economic development activities can have intended or unintended impacts on the environment, resulting in ecological changes that can alter the replication and transmission patterns of pathogens. A growing number of emerging infectious diseases arise from increased human contact with animal reservoirs as a result of changing land use patterns. See Smolinski, Hamburg and Lederberg, 2003, "Executive Summary," p. 4-5. Slaughterhouses in Philadelphia, like the one on the south side of the Dock, were traditionally located near streams, along with cattle pens. Around them, hornworms, chandlers, and soap- and glue-boiling yards made use of the slaughterhouse's by-products. But the largest users of the slaughterhouse leavings were the tanneries. These establishments included not only mills for grinding bark used in tanning but also vats and pits for soaking the hides during the stages of removing the hair, tanning the hide, and giving texture to the leather. The method of curing varied according to the intended use, whether for saddles or for women's gloves. Beyond the yard, then, the tanneries also supported a cluster of linked manufacturing activities, including shoemakers, saddlers, curriers, and glovers. See Van Wagenen, 1953, p. 182-9. Water was needed throughout the

processing of the hides. Initial treatment consisted of soaking the hides in lime pits. Measuring, on the average, six feet square, lime vats sometimes were twice that size. Vats usually were wooden structures made to function like "a small water meadow slightly sunk." See Hartley, 1979, p. 254. A retaining dike wall was built around the vat, and, in rural settings, water was flooded in from the adjacent stream. Whether used in a country tanyard or in an urban yard with wells and a nearby tidal cove, the water was always returned to its source along with the various substances that had been added, including acidic liquids resulting from the refuse of cider presses, sour milk, fermented rye, and alkaline solutions made up of buttermilk and some forms of dung. The processing of the hides signified as well the strong connections between the industrial activities around the Dock and the countryside. Breweries, tanneries, slaughterhouses, and distilleries gathered the products of the countryside - animals and grain primarily - processed them, and discarded the unused by-products on the Dock's watershed. See Hartley, 1979, p. 354-5.

[15] An infectious disease can result from a behavior that increases an individual's risk of exposure to a pathogen, or from increased probability of exchange of a communicable infectious disease between people as population increases. See Smolinski, Hamburg and Lederberg, 2003, "Executive Summary," p. 4. Human behavior played a large role in the spread of disease in Philadelphia. The sharing of a bed with a sick family member, for example, helped to spread tuberculosis among members of the same household. Additionally, people who worked outdoors were constantly exposed to the bite of mosquitoes, and helped to spread yellow fever and malaria throughout the city.

[16] Immigration from Europe and trade with the Caribbean not only fed the growth of the area but also retarded the rapid increase in the population, since commerce was a constant source for the reinfection of the population with epidemic disease. In particular, tropical diseases that could not withstand the frosts of Philadelphia's winters were reintroduced every summer through trade with the Caribbean.

[17] Microbes continually undergo adaptive evolution under selective pressures for perpetuation. Through structural and functional genetic changes, they can bypass the human immune system and infect human cells. See Smolinski, Hamburg and Lederberg, 2003, "Executive Summary," p. 4. Epidemiological research on measles has found that the mortality rates were higher when the disease was contracted from a member of the same household or a relative than when it was contracted from others in the community. On the basis of these findings, it has been suggested that high death rates in the Americas from European infectious diseases are related to the fact that the virus grown in one host became adapted to the immune system of that individual. When introduced in a genetically similar host, it gained in virulence and resulted in higher mortality rates. See Black, 1992, p. 1739-40; Aaby, 1991, p. 83-116. Note: a separate section on microbial adaptation and change will not be included in this chapter. Rather, it will be discussed in the section on measles in Chapter 5.

[18] A breakdown or absence of public health measures – especially a lack of clean water, unsanitary conditions, and poor hygiene – has had a dramatic effect on the emergence and persistence of infectious diseases throughout the world. The lack of any effective public health measures in eighteenth-century Philadelphia played a significant role in the spread of disease. The city officials believed that disease was caused by a filthy urban environment and a dangerously polluted water supply, so their efforts concentrated on cleaning the city. Yellow fever and diseases like smallpox, malaria and consumption, however, were not directly caused by filthy streets and a polluted water supply. As a result, the city's effectiveness in controlling the spread of these and other infectious diseases was marginal at best. Furthermore, Philadelphia was a city of immigrants. The wretched circumstances with which the immigrants had to cope remained unimproved through much of the century. Pennsylvania's provincial government passed four major Quarantine Acts to regulate conditions on board ships in 1700, 1749, 1765 and 1774. The first was vague and not enforced immediately, the second was easily and widely evaded, and the third was questioned by the Privy Council on the basis that it violated the rights of the British government to regulate trade. The fourth was enacted just before the American Revolution. See Wolman, 1974, p. 13-4.

[19] Klepp, 1989, *Philadelphia in Transition*, p. 7.

[20] M.E. Wilson, 2003, p. 1S.

[21] Soderlund, 1983, p. 72.

[22] Dunn and Dunn, 1982, p. 6-7; Garvan, 1963, p. 189-90; Cotter, et al, 1993, p. 34.

[23] George, Nead and McCamant, eds., 1879, p. 187.

[24] Penn, 1909, p. 303.

[25] Dunn and Dunn, 1982, p. 10-4.
[26] William Penn's plan for the city of Philadelphia was published in London in 1683. The gridiron street pattern, the five large squares, and the size of the city were the features that made this plan unique among all those in the English colonies. Modern reproduction issued by Historic Urban Plans, Ithaca New York, 1965.
[27] Garvan, 1963, p. 193-4; Reps, 1965, p. 161.
[28] Reps, 1965, p. 167-8.
[29] Dunn and Dunn, 1982, p. 15-6.
[30] The population data collected by Shammas from 1789 on are based on decennial federal census numbers or interpolations from census data. For earlier population numbers, see Bureau of the Census, *A Century of Population Growth*, 1909, p. 11. The population in Philadelphia in 1760 is assumed to be 80% of the city and suburb total. Pre-1850 areas are calculated from settled areas shown in maps. Philadelphia 1760, New York 1770, Baltimore 1790, and Boston 1770 are based on maps in *Atlas of Early American History: The Revolutionary Eras, 1760-1790*. See Cappon et al, 1976, p. 9-12. Density levels for Philadelphia in 1794 and 1800 are based on the1794 map known as the *Plan of the City and Suburbs of Philadelphia* and on William Birch, *The City of Philadelphia…1800*. Density was calculated on the basis of settled areas within the city limits rather than the official city limits. Suburban areas were not counted. In these density figures, uninhabitable areas, such as streets, are counted as part of the area. The numbers of people in actual living areas therefore was greater per square mile than indicated here. Streets, for example, can consume as much as a third of the area of a city. See Shammas, 2000, p. 509-10. She also notes that the high-density wards in New York and Philadelphia of 1800 fall into the medium range of tenement crowding by nineteenth-century standards. These numbers are three or more times what is considered medium density today. See Shammas, 2000, p. 507.
[31] Weber, 1899, p. 463-5. In 1801, all of London covered an area of about 37.6 square miles, and had a density of 25,600 per square mile.
[32] Shammas, 2000, p. 513.
[33] Map shows settled areas of Philadelphia by ward. See Shammas, 2000, p. 513. The densities, calculated from a digitized map of the city wards, show Chestnut ward at 92,862 persons per square mile, Lower Delaware at 80,404, Upper Delaware at 76,675, Walnut at 65,727, New Market at 58,614, High Street Ward at 55,840, and Dock Ward at 46,562. The partially settled wards, without a waterfront on the Delaware River, had much lower densities – fewer than 25,000 persons per square mile. See Shammas, 2000, p. 511n.
[34] Population data is from Klepp, 1989, "Demography in Early Philadelphia," p. 103-6; Smith, 1980, p. 51, gives 228 as the number of additional dwellings per year in the city proper, 1790-1810. What constitutes a dwelling is unclear. Additional data is from Shammas, 2000, p. 512; Salinger, 1995, p. 19.
[35] The numbers for 1798 come from the Federal Direct Tax; Shammas, 2000, p. 512.
[36] Shammas, 2000, p. 509-12.
[37] Shammas, 2000, p. 519-20.
[38] Shammas, 2000, p. 521.
[39] Salinger, 1995, p. 15.
[40] Salinger, 1995, p. 15.
[41] Crane, 1991, p. 790. Excerpt from the *Dairy of Elizabeth Drinker*, August 10, 1796.
[42] See Patterson, 1992, p. 857-8.
[43]Mullen and Durden, eds., 2002, p. 229-31.
[44] Dysentery was often fatal and always debilitating, and respected neither class, gender, race, nor age – although the disease posed the greatest risk to infants, children and the elderly. Depending on the invading organism, dysentery can be marked by diarrhea, cramps, fever, sepsis (a toxic condition due to spread of bacteria or their products in the body), and bloody feces. Death from dysentery often follows electrolyte fluid loss and the ensuing dehydration; vascular collapse is also not uncommon. It can be caused by a large variety of bacterial, viral, and parasitological pathogens. Bloody stools generally have bacterial and occasionally amoebic origins and usually do not have a viral cause. Pathogens that cause dysentery are often spread by contact with infected humans as well as by healthy carriers. Environmental factors also play a crucial role as well: the disposal of organic wastes can

cause contamination of water supplies; improperly handled food can encourage microbial replication; and the absence of personal hygiene can create conditions conducive to infection. See Grob, 2002, p. 53. All of these factors were present in abundance in eighteenth-century Philadelphia. Additionally, dysentery tends to peak in warmer months and is more prevalent in southern climates; high temperatures and humidity provide greater opportunity for rapid pathogen proliferation.

[45] Klepp, 1989, *Philadelphia in Transition*, p. 285, 300-1. The Christ Church Bills of Mortality consistently list deaths from "consumption, flux, fever, purging and vomiting." Elizabeth Drinker's diary is replete with references to gastrointestinal disorders and summer fevers. Every member of her immediate family suffered from periodic bouts of both dysentery and malaria, and son William battled tuberculosis for much of his adult life.

[46] Crimmins and Condran, 1983, p. 31-59; Klepp, 1989, *Philadelphia in Transition*, p. 235-8, 256, 264; Shammas, 2000, p. 525.

[47] Kupperman, 1982, p. 1262-89; Grob, 2002, p. 49.

[48] Eighteenth-century Philadelphia was centered on the low-lying plain along the banks of the Delaware River. It was marshy, hot and humid in the summer, and cold and humid in the winter. To the south, near the confluence of the Delaware and Schuylkill Rivers, was meadow land subject to frequent flooding. These conditions helped to support a large population of flies, mosquitoes and rats which plagued the city and its inhabitants. See Rush, 1815, "Account of the Climate of Pennsylvania and Its Influence Upon the Human Body," Vol. II, p. 3-27. In addition to the damp environment, ignorance of sanitation made the Philadelphia urban environment unhealthy. The high water-table under the city meant that the wastes in the privies constantly seeped into the hundreds of private wells which supplied the population with water. Intestinal complaints were endemic in the city as a result, with widespread bacillary dysentery infections, especially during the summer months. The clearing of the forests for firewood resulted in more marshy ground around the city so that the suburbs became noted for their unhealthiness in the summer. Bridenbaugh, 1955, p. 105, 296; Drinker, 1937, p. 22-33; Hawke, 1971, p. 9; Rush, 1815, "Account of the Climate of Pennsylvania and Its Influence Upon the Human Body," Vol. II, p. 27; Klepp, 1989, *Philadelphia in Transition*, p. 225-6.

[49] The Christ Church Bills of Mortality for the year 1756 listed 14 deaths from "consumption," 2 deaths from "flux," 6 deaths from "fever," 8 deaths from "fits," 8 deaths from "nervous fever" (typhus or typhoid), 15 deaths from "purging and vomiting," 112 deaths from "small-pox" and 10 deaths from "teeth and worms." See Klepp, 1991, p. 65.

[50] Bridenbaugh, 1955, p. 30.

[51] Drinker, 1937, p. 32.

[52] Crane, 1991, p. 439. Excerpt from the *Dairy of Elizabeth Drinker*, August 17, 1785.

[53] Rush, 1786, "An Inquiry into the Cause of the Increase of Bilious and Intermitting Fevers in Pennsylvania, with Hints on Preventing Them."

[54] Mullen and Durden, eds, 2002, p. 243.

[55] Bishop, 1864, Vol. I, p. 445.

[56] The saltpeter works on Market Street reeked of urine and fermenting animal and vegetable matter, and the pots of evaporating liquor were nauseous to nearby residents. See Raufer, 1998, p. 82. Saltpeter occurs naturally in many parts of the world, but there were no such deposits in the colonies. As a result, it was manufactured in a manner employed for centuries. In this method, vegetable and animal refuse containing nitrogen, the sweepings of slaughterhouses, weeds, etc. were collected into heaps in a shed or a warehouse where they were protected from the rain, and mixed with limestone, old mortar and ashes. The heaps were moistened from time to time with runnings from stables and other urine. When decomposition was complete, the heaps were leached with water, the liquor evaporated, and the saltpeter recrystallized. See Van Gelder and Schlatter, 1927, p. 62. In 1775, the "Committee of the City and Liberties" established a saltpeter works on Market Street, under the superintendence of several prominent Philadelphians, including Benjamin Rush. See Bishop, 1864, Vol. II, p. 24n.

[57] Part of the process of sugar refining required putting partially crystallized sugar in clay pots for a few months to let the molasses drain out. Clay pots and fragments of clay pots caught the rain and made ideal homesteads for *A. Aegypti*. See Goodyear, 1978, p. 5-21.

[58] Ribeiro, 1996, p. 25-33.

[59] Warner, 1987, p. 6.

[60] Cotter, et al, 1993, p. 43. The Drinker family lived for ten years on Water Street, but in 1771 they moved to what became known as Drinker's Big House, on Front Street and Drinker's Alley below Race Street. The wharf of James and Drinker was little more than a block away. See Drinker, 1937, p. 17-8.

[61] The physical environment directly and indirectly influenced the emergence and spread of infectious disease in Philadelphia through impacts on pathogens, vectors, and hosts. Environmental changes affected all classes of human pathogens (viruses, bacteria, protozoa, fungi, helminths), and their many routes of transmission. It also influenced every part of the interaction between pathogen and host, including the survival, abundance, and dispersal of the pathogen, as well as affecting whether vectors could survive in a particular geographic area. The *Aedes aegypti* mosquito, for example, does not bite when the temperature drops below 60 degrees. The temperature can also influence whether a pathogen such as the malaria parasite inside an insect vector, has time to develop to a stage that can be infective for humans. Likewise the environment influences the presence and abundance of intermediate and reservoir hosts.

[62] Smith, 1990, p. 48.

[63] Morbidity and mortality in the colonial period were for the most part independent of therapeutic intervention, medical or otherwise. The introduction of vaccination after 1800 and the continued use of inoculation undoubtedly contributed to the decline of epidemic smallpox by decreasing the number of susceptible individuals. Nevertheless, it is difficult to specify the precise roles of these procedures. The vaccine employed was variable in quality and sometimes failed to induce immunity. Even when effective, vaccination did not confer lifelong immunity, and revaccination had yet to be accepted by the public. Many communities also adopted legislation providing for quarantines during epidemics and regulated occupations that allegedly posed a threat to health. In the aggregate, however, such public health measures had a negligible influence; morbidity and mortality seemed to be influenced by broader environmental forces. See Grob, 1983, p. 10; Grob, 2002, p. 182.

[64] Wolman, 1974, p. 17.

[65] Illness and death were omnipresent threats on board ship. Christopher Sauer, editor of a newspaper in a village outside of Philadelphia, estimated that 2,000 passengers on fifteen ships, due to arrive in 1758, perished en route. Accounts of individual ship disasters are equally appalling: Johann Keppele reported in his diary that 150 of 312 passengers died during the voyage, the *Sea Flower* lost 46 of its 106 travelers, and the *Love and Unity* arrived with only 34 of its 150 journeyers. These accounts are from Diffenderffer, 1900, *The German Immigration into Pennsylvania through the Port of Philadelphia*, p. 260; Hofstadter, 1971, p. 41-2.

[66] Smith, 1977, p. 884.

[67] Labaree et al, 1959-1973, Vol. 3, p. 439.

[68] Zachariah Poulson referred to the Strangers' Ground as "Potter's Field." See Poulson, 1800, *Poulson's Town and Country Almanac*, p. 1.

[69] Smith, 1990, p. 45.

[70] Typhus is caused by one of the rickettsiae microbes that naturally infect small blood-sucking insects. They naturally infected fleas and then rats, and both tolerate the infection without signs of disease. When rickettsiae infect new types of hosts, disease can follow. Rat fleas also bite humans, thus transmitting the rickettsiae that cause human typhus. The disease cannot spread from one human to another on its own, but with the help of another insect that thrives on humans, typhus can spread quickly in filthy, crowded groups. The human louse can become the carrier of the disease as it feeds on infected humans. See Barnes, 2005, p. 251-2.

[71] Human typhoid fever spreads by the oral-fecal route with human fecal-contaminated food or water. Contaminated water provides the major source of transmission. See Barnes, 2005, p. 289-90.

[72] Historian Francis Packard blames the severity of the 1756 smallpox epidemic on the spread of the disease caused by the arrival of troops in Philadelphia during that year. According to Packard, the governor of Pennsylvania was alarmed that "the smallpox is increasing among the soldiers to such a degree that the whole town will soon become a hospital." See Packard, 1963, p. 88.

[73] In 1745 Dr. John Mitchell of Philadelphia attributed the outbreak of two yellow fever epidemics to the arrival of infected immigrants. See Colden, 1919, *The Letters and Papers of Cadwallader Colden*, p. 326.

[74] Wolman, 1974, p. 46.

[75] Colonial Records of Pennsylvania, 1760-1776, Vol. 3, p. 59.
[76] Wolman, 1974, p. 47-8.
[77] Studies of Boston, Nottingham and Andover were done by Blake, 1959, Appendix II; Chambers, 1960, p. 97-125; Greven, 1970, p. 179, 293; analysis in Smith, 1977, p. 885.
[78] Analysis in Smith, 1977, p. 885.
[79] Smith, 1977, p. 888.
[80] Typhus, a common shipboard affliction, may have been one of the most prevalent fevers in the urban center, although tuberculosis and typhoid fever likewise contributed to high mortality. Yellow fever was a dramatic killer in the 1790s, but other diseases were as deadly. Between 1789 and 1801, yellow fever caused the deaths of 18 percent of Episcopalian decedents, while tuberculosis caused 24 percent of deaths. Other infectious diseases, including smallpox, whooping cough, measles, diphtheria, and vaguely defined "fevers" accounted for 22 percent of deaths. Convulsions, diarrhea, and other common causes of infant deaths accounted for 18 percent of the total. See Klepp, 1995, "Zachariah Poulson's Bills of Mortality, 1788-1801," p. 227.
[81] Crane, 1991, p. 178. Excerpt from the *Diary of Elizabeth Drinker*, July 20, 1772.
[82] Crane, 1991, p. 497. Excerpt from the *Diary of Elizabeth Drinker*, August 27, 1793.
[83] Kalm, 1772 reprint, 1972, p. 37; Webster, 1800, Vol. 2, p. 209.
[84] Shammas, 2000, p. 514.
[85] Webster, 1800, Vol. 2, p. 209; Grob, 2002, p. 90.
[86] Grob, 2002, p. 91.
[87] By the middle of August 1776, dysentery, malaria, and "Bilious Putrid Fevers" had begun to supplant smallpox among the American forces, although the threat from smallpox remained real. See Fenn, 2001, p. 78.
[88] Grob, 2002, p. 90; Gibson, 1949, p. 121-7.
[89] See Patterson, 1986, p. 11-28; Caulfield, 1950, p. 21-52.
[90] *American Weekly Mercury*, November 23, 1732.
[91] Patterson, 1986, p. 11-28; Caulfield, 1950, p. 21-52.
[92] Duffy, 1953, p. 199; Wolman, 1974, p. 211.
[93] Grob, 2002, p. 88.
[94] Smith, 1990, p. 64.
[95] Bronner, 1982, p. 37-8.
[96] Smith, 1990, Appendix C.
[97] Rauffer, 1998, p. 23.
[98] Not all geographic regions and populations are equally receptive to the introduction and spread of new infections. Those that are spread directly from person to person (e.g. respiratory, direct contact, sex) or by the fecal-oral route may be influenced by such factors as living conditions (e.g. crowding, ventilation and level of sanitation), population size and density, nutritional status, age structure of the population, immunity from past infection or inoculation, size of the susceptible population, genetics, season of the year, among others. In order for a pathogen to establish itself in a host population, it must have a basic reproductive rate exceeding 1. If it is lower than this, it will die out, though may do so slowly if it is an infection, such as tuberculosis, which can persist in a latent form and can reactivate years or decades after the initial infection. See M.E. Wilson, 2003, p. 3S.
[99] Bridenbaugh, 1942, p. 295 and chapter 11. Bridenbaugh sites Bond's "Essay on the Utility of Clinical Lectures," delivered in Philadelphia on November 6, 1766.
[100] McMahon, 1997, p. 104.
[101] Smith, 1997, p. 158.
[102] *Philadelphia Monthly Magazine*, August 1798, p. 69.
[103] The first cases of yellow fever were usually traced to the vicinity of the docks, giving support to the opinion that the disease was imported. Some physicians and observers maintained, however, that there were specific putrefactive causes at dockside that gave rise to an indigenous disease. In 1793, for example, the origin of the yellow fever epidemic was "traced" to a quantity of damaged coffee which had been thrown upon Mr. Ball's wharf. See Powell, 1949, p. 11-2; Rush, 1815, An Account of the Bilious remitting Yellow Fever, as it Appeared in the City of Philadelphia, in the Year 1793," Vol. 3, p. 37-193. Another observer of the same epidemic suggested, however, that "it first broke out in a part of the city near which ships from those (infected) islands, that had unusual numbers of sick people on

board, discharged their cargoes." See Bordley, 1794, p. 6. In 1798, it was generally agreed that the source of the yellow fever epidemic was imported aboard a vessel that had made stops at Caribbean ports known to be having (or having had recent) yellow fever epidemics. Nevertheless, opinions differed as to the specific cause. One opinion was that the source was the foul air of a cargo hold. Another opinion held that the source was several infected persons who were smuggled into the city. See Condie and Folwell, 1798, p. 27.

[104] Morse, 1995, p. 13.

[105] See Patterson, 1992, p. 857-8.

# CHAPTER 4

# SUSCEPTIBILITY OF HOST

The occurrence of disease in any population group is a function of several major variables: the virulence[1] of the microorganism (i.e., its possession of factors that allow it to cause illness), its mode of transmission (i.e., how it gets to the host), and the susceptibility of the host (i.e., how well the host can defend itself against the microorganism).[2] The human body has many defenses against the entry and multiplication of microbes,[3] and when these defenses function normally, infection is less likely to occur. However, in a weakened host, a microbe is more likely to invade the body and cause disease. Host susceptibility is not a single entity, and many properties of the human body – from its genetic makeup to its innate biological defenses – affect whether a microbe will cause disease. Susceptibility to infection can increase when normal defense mechanisms are altered, when there is no prior exposure to the infectious agent, or when host immunity is otherwise impaired by such factors as genetically inherited traits, malnutrition, poverty, poor hygiene,[4] extremes of age,[5] general debilitation, sex, climate, inadequate physical barriers, ethnicity, preexisting or intercurrent disease, emotional and physical stressors, chronic disease, medical or surgical treatments and a wide range of behavior. Susceptibility in past populations can be difficult to gauge. The immunologic

response to infectious disease is a complex process, and even if measurements of factors such as nutritional intake, microbial burden, and stress had been recorded in Philadelphia, some speculation would still remain. It is apparent, however, that most eighteenth-century Philadelphians faced the kinds of challenges that promote vulnerability to disease. If factors linked closely with population growth and mobility were most critical to probability of contact, those responsible for hygiene and sanitation, chronic disease and debility,[6] malnourishment and poverty determined susceptibility.[7]

Many variables interact to produce illness, and the more we learn about disease, the more complex the concept of causation becomes. Malaria, for example, is a disease that requires several elements to be present in a population for an outbreak to occur. Each contributes in some way to produce the disease in humans, with some more directly related than others. Several of the elements are absolutely necessary – the parasite, *Plasmodium*, for example. Without *Plasmodium* in the bloodstream, there would be no illness. At the same time, without the vector (*Anopheles* mosquitoes), there would be no *Plasmodium* in the bloodstream. And without standing water for breeding and temperatures warm enough to support procreation, there would be no *Anopheles* mosquitoes. Without human reservoirs of parasites, there would be no source for a large supply of the *Plasmodium*. And so it goes. The interlocking components are both extensive and interdependent, allowing the cycle of infection to run its course.[8]

Although it has been argued that no precondition of disease was more basic than poverty in eighteenth-century Philadelphia,[9] it is shortsighted to assume that impoverishment was necessary for disease to occur. Eating tainted food, inhabiting overcrowded and under-heated houses, and living in deplorable sanitary conditions certainly increased the vulnerability of the "lower sort" to many diseases. The poor, however, were not the only residents who were sick, debilitated, malnourished, unclean and living in a filthy environment. Poisons, pests and nuisances affected everyone. There were few physical barriers in the city at this time, and the toxicity of the landscape touched the rich and the poor, the healthy and the sick in every possible way. They drank from the same wells, breathed the same air, walked the same streets, and were bitten by the same insects. Placing excessive emphasis on poverty and the plight of the poor to explain how and why certain diseases emerged in Philadelphia obscures the fact that specific factors precipitating disease emergence can be identified in virtually all cases. These included ecological, environmental, demographic and behavioral factors that either placed people at increased contact with a previously unfamiliar microbe or its natural host, or promoted the dissemination of the microbe within the population.[10]

## Sanitation and Hygiene

Although small by modern standards, eighteenth-century Philadelphia was, for its time, a large, bustling urban community.[11] By the early 1740s, Philadelphia had passed from being a provincial town and began to take on the characteristics of a

small commercial city; its population was about 11,000 people and there were more than 1,500 structures.[12] Most were located in an area east of Fifth Street, and spilled over the city boundaries along the river. A visitor to the city in 1744 presented a picture that was not very flattering: "Att my entering the city, I observed the regularity of the streets, but at the same time the majority of the houses mean and low and much decayed, the streets in general not paved, very dirty, and obstructed with rubbish and lumber."[13] From the earliest days of settlement, the residents kept animals near their homes for food and other uses. This simple husbandry gave rise to serious sanitation and health problems which were not easily solved.[14] As late as the 1750s, animals and livestock still roamed the streets, and some of these streets still had stumps or roots of the original pine trees sticking up.[15]

Philadelphia was a densely populated city, with much of that population concentrated along the Delaware River. Medical commentators noticed that high death rates were confined to the "narrow streets, courts, and alleys of the city and southern suburbs. They also observed that death by cholera infantum (dysentery in children)[16] rarely happened in houses with large and well aired apartments."[17] Medical theory blamed the lack of ventilation which trapped the miasmal vapors and pestilential airs considered to be the cause of contagious disease. Inadequate water supplies and poor public and private sanitation were the real culprits, although crowded quarters would increase contact between the sick and the healthy.[18] The longer and closer the contact between a person with a contagious disease and a susceptible individual, the more likely is transmission. Tubercle bacilli, for example,

are transmitted to human hosts almost exclusively through aerial transmissions. Through talking, coughing, sneezing, spitting, singing, and other respiratory functions, people produce airborne particles, which, if emitted by a tubercular individual, can contain bacilli. Just one bacillus is enough to cause a tuberculosis infection when inhaled. Once airborne in a closed space, these particles disperse, and some remain suspended like tobacco smoke.[19]

Ignorance of sanitation was also a problem, and made the Philadelphia urban environment even unhealthier. The high water-table under the city meant that the waste in the privies (or necessaries, as they were called) constantly seeped into the hundreds of private wells that supplied the population with water. In some areas of town, each house had its own pit, while in others one pit was shared by many houses. These pits were never emptied in the poorer parts of town, and rarely, if ever, emptied in the wealthier parts of town. On March 5, 1779, the Drinker cesspool was cleaned out for the first time in forty years.[20] Elizabeth Drinker makes note of the occasion in her diary.

> "Five Men with two Carts &c. are about a dirty Jobb in our Yard to night. They are removing the offerings from ye temple of Cloacina, which have been 44 years depositing – Jacob, Sarah, Peter and Sally are burning Incense in the Kitchen, pounded rosin in a Chaffing dish of coals, tho there has not yet been any occasion for it, as we have not been offended by any bad smell – as yet."[21]

Contaminated well water was a common problem in the city, and so it is not surprising that intestinal complaints were as numerous as they were. Furthermore, the streams which ran through the city were little more than open sewers where waste of

all kinds was deposited. The streams ran into the Delaware River where the docks, stretching out into the water, prevented the current from sweeping away the debris. As a result, pools of stagnant water formed, becoming ideal breeding grounds for mosquitoes. Several of the city's yellow fever epidemics began in the alleys in this part of town. The clearing of the forests for firewood resulted in the creation of even more marshy ground around the city so that the suburbs became noted for their unhealthiness in the summer.[22]

Water drainage had always been an issue in the city. In the late seventeenth-century, there was already a grand jury bemoaning "the want of a Channell to Convey ye water…along the Front Street," and after numerous citizen complaints the Provincial Council in 1700 passed a law for "Regulating of Streets and Water Courses in Cities and Towns."[23] This law required that the underground culverts be circular in shape and constructed of brick or stone without any mortar in the lower half, so that liquids could leach through the ground. Through private initiatives, there were considerable drainage efforts underway by 1720, but the real push came in the 1760s in conjunction with street paving.[24]

Although the people of Philadelphia were not crowded into urban slums and manufacturing environments as were the poor of London, it is clear that the same sorts of "spoil of dunghills and putrid thaw of nature" were to be found in their littered streets and polluted streams.[25] As there were no zoning restrictions in the city at this time, tanning yards, slaughter houses, soap boilers,[26] saltpeter works, sugar

refineries and distillers all marked the landscape with their own brand of pollutants and potential for disease. Since Philadelphia sits between the confluence of the Schuykill and the Delaware Rivers, many small streams converged in the city, resulting in the perennial health problems associated with drainage. Homeowners were consistently plagued by drainage problems from inadequate sewers, flooded gutters and overflow from a neighbor's necessary house.[27] The area around the Dock Creek, both along its banks and in the low swampy ground stretching westward to Society Hill, was constantly sullied by the refuse of six tanyards. As the tide water sluggishly receded, piles of filth and mud were exposed.[28]

Water initially drained through "watercourses" located in the middle of the streets, and as early as 1727 streets were required to be pitched so that water (especially that from the public pumps) would flow towards the watercourse. By the 1760s, a different design had become accepted, with the streets slightly raised in the middle and convex, draining towards side gutters. It was the responsibility of private property owners to keep the watercourses clear. Since the roadways and streets tended to collect all sorts of mud, wastes, garbage, and other urban detritus, all of this eventually ended up in the watercourses. Although the city banned disposal of human wastes in the sewers of the city, this did not stop cross contamination between water source and waste sink.[29]

Water for personal use was provided by wells that were generally located behind the houses or in the streets. As the city expanded, many of these pumps

became neglected. The wardens of the city in 1756 felt the need not only to dig new wells but to take over privately owned ones and maintain them at public expense. Severe penalties were imposed for tampering with these wells.[30] To protect the wells, the Assembly authorized the street Commissioners in 1769 to limit the depth of "all Vaults, Wells, and Sinks…to be dug for Privies or necessary Houses."[31] Nevertheless, a visitor to the city pointed out that "since into these wells naturally filters the water of the privies, which exist in all the houses, this together with the cellar dwellings inevitably causes the pestilence they have every summer."[32] John Lawrence's well at Sixth Street near Chestnut, particularly recommended by Benjamin Rush for its medicinal qualities, was found ultimately to be one of those so contaminated. This was a popular well. Evidently, Elizabeth Drinker and her friends did their part in helping to exhaust this water source, since in May and July 1773, she speaks of walking to John Lawrence's pump, which was about a half mile from the Drinker house.[33] The Drinker family, like all people in Philadelphia, suffered from their fair share of gastrointestinal complaints. Contaminated water affected everyone, and the resulting enteric diseases were widespread.

Environmental hazards were also present in the city, and they, too, contributed to the general debilitation of the people. Sources of such hazards included the problems associated with insects and vermin. Streets littered with debris and accumulations of stagnant water enhanced the proliferation of these pests. Though most of the houses were built of brick or wood, their cellar floors were usually of dirt – a favorable environment for rodents and their fleas. Additionally, mattresses made

of feathers or straw served as an excellent niche for the breeding of bedbugs and body lice. Typhus is transmitted by the feces of human body lice, but it can also be transmitted by the rat flea.[34] The microbe enters the body through skin lesions and mucous membranes. The disease was a particular problem among new arrivals to the city. Characteristic symptoms of typhus include fever, prostration, aches, and a widespread rash covering trunk and limbs, and it flourishes where people crowd together in unsanitary conditions. It has had many names including "jail distemper," "gaol fever," "ship fever," and "camp fever." These names reflected the poor hygiene that was characteristic of such circumstances.[35]

## Chronic Disease and Debility

Outbreaks of dysentery occurred throughout the colonies, but especially in the more densely populated towns. The disease was endemic in Philadelphia throughout the eighteenth century, but yearly epidemics were not uncommon.[36] On July 6, 1778, Elizabeth Drinker wrote "My dear Henry [husband] unwell with disordered Bowels, he took Rheubarb, as the Flux is about, I am uneasy on his account – Sally [daughter] is also complaining in the same way." On July 8th she wrote "Very warm today: Sally very ill, with the vomiting and Flux, above 30 stools to day, she took a vomit this Morning and I gave her a Clyster this Evening she has a great deal of fever, - HD. [husband] continues disordr'd in his Bowels, I have been realy ill in that way myself this day: and tho not well, am better this evening."[37] Lacking knowledge about the causes of dysentery, Drinker and other Philadelphians were unable to undertake

preventative measures. Infected and healthy carriers transmitted pathogens; crude methods of disposing of organic wastes often contaminated drinking water; the absence of refrigeration permitted the growth of pathogens in food supplies; and prevailing hygienic standards enhanced the risk of exposure. Infants and young children were especially vulnerable, since parents were unaware that dehydration could result in death. Mortality during dysentery outbreaks ranged between 5 and 10 percent, to say nothing about the large numbers of individual deaths during nonepidemic years.[38] Dysentery also contributed to overall mortality in different ways, for it weakened individuals and left them vulnerable to other diseases.

Malaria was also endemic in Philadelphia during much of the eighteenth century. Like dysentery, it severely weakened the afflicted individual, making them susceptible to secondary infection. It was imported into Philadelphia, and quickly spread to interior regions. The clearing of land disrupted drainage patterns and created stagnant bodies of water. The presence of the *Anopheles* mosquito and the transportation of infected persons into the city set the stage for the appearance of malaria. The disease was present along virtually the entire eastern coast of the colonies, and flourished in warm, humid weather. Colder weather diminished its prevalence, but the seasonal decline in new cases did not diminish the burden of disease.

An often ignored group of factors contributing to the overall debility of the population was ingested toxins. Food adulteration was a common practice and some

of the additives were potential poisons. Alum, for example, was continually used as a bleach; potato flour, gypsum, chalk and pipe clay were other adulterants. To flavor beer, "spurious red pepper made of oil cake, common clay and a portion of cayenne pepper sometimes adulterated with red lead…and white pepper was first steeped in seawater and urine."[39] Toxic additives were not limited to bread and beer, however. Teas and wines also contained potentially poisonous components. Wines were especially relished by the "better sort" in Philadelphia, with lead, mercury and arsenic used as common additives to enhance flavor. Additionally, wine bottles were often cleaned with leaden shot, some of which were occasionally left behind through carelessness. Later when these bottles were again filled with wine or beer, lead slowly dissolved and impregnated the liquor.[40]

Rum drinking also posed a significant health threat to the people of Philadelphia. The bulk of the rum consumed in the city was imported from the West Indies or New England. Most of the stills used for the production of both rum and "spirits" were made of copper, and many had copper, tin or pewter "worms" or pipes. Pewter was a mixture of brass, lead and tin. The coiled condensation tube - the "worm" - of an alcohol still was most easily fashioned out of lead. The distillation of fermented sugar products in such a still for rum production caused sufficient lead leaching from the "worm" to render the rum toxic. Epidemics of abdominal colic ("dry bellyache" or "dry gripes") were common in Philadelphia.[41] Upon suggestions from those consumers that their illness might be the result of lead-contaminated New England rum, Massachusetts protected its trade market with remarkable alacrity by

enacting the Massachusetts Bay Law of 1723, prohibiting the use of lead parts in a liquor distillation apparatus. Although the law is often applauded as the first public health law in the colonies, it should be noted there is no evidence of any serious, objective investigation regarding the role of lead in the origin of the rum-drinkers' symptoms, nor does the medical profession seem to have been the principal instigator for the law's passage. It appears the legislators were responding primarily to customer complaints, motivated more by trade than by health concerns.[42]

The evidence strongly supports the belief that the people of Philadelphia were continually exposed to hazardous amounts of lead from environmental sources, and chronic lead poisoning was a health problem. Not only were much of their cider, wine, rum and other spirituous liquors contaminated with traces of lead, they were repeatedly taking in small amounts of lead from cooking and serving utensils, water cisterns, household paints, cosmetics and medications. Pewter house wares were particularly problematic. Plates and goblets were commonly made of pewter in wealthier colonial homes, and perishables and beverages were stored in lead-lined containers. In these homes, almost everything the family ate or drank contained some lead. Colonists were also exposed through their use of lead bottles, funnels, nipple shields, dram cups, candlesticks, lamps, pipes, roof gutters, and other items.[43] Lead poisoning is a serious disease, interfering with almost every body function. The symptom of lead poisoning most commonly encountered in historical literature is abdominal pain. It is usually attributed to intestinal spasm, though the abdominal muscles may undergo the painful, uncontrollable contractions called "colic." Similar

pain is seen in diarrhea, but lead poisoning is instead accompanied by constipation; hence the abdominal pain of lead intoxication is termed "dry bellyache."[44]

In a similar manner, copper – particularly salts of copper - posed a serious health threat to the people of the city.[45] A pharmaceutical and medical guide available in Philadelphia recommended "Verdigris…Copper turn'd into a rust by a corrosion of an acid that is in the grape…very serviceable in those cankerous specks as the nurses call them so apt to be troublesome in children's mouths for which a little of it is mixed with honey and gently rubbed on the parts affected."[46] In addition to the many hazards faced by young children, heavy metal toxicity must be included as a factor in the high infant mortality rate in the city.[47]

Debility was an inevitable consequence of impoverishment, but it can be caused by many things, including malnutrition, chronic disease, parasites, and an accumulation of toxins in the body. Infirmity affected many in the city, leaving them with fewer defenses against disease. The prevention and cure of an infectious process almost always depends upon whether the patient's immune system can mount an effective defense in a reasonable period of time. Any factor that retards or suppresses this response contributes to an otherwise unfavorable outcome. Members of the Drinker household were constantly ill. Henry Drinker was often unwell, usually suffering from some form of gastrointestinal disorder. He constantly subjected himself to treatment with emetics and purges of "dreadful strength and devastating consequence." William, Drinker's fourth child, developed pulmonary tuberculosis in

early adulthood. Although he lived fifty-four years, he was an invalid during much of that time. Every member of the family suffered repeated bouts of malaria,[48] and one daughter contracted yellow fever during one of the more severe epidemics of the 1790s.[49] While poverty certainly played a role in susceptibility to disease, one did not have to be poor in order to be sick. "*Any* diminution in *any* host defense system 'opens the door' to microbial invasion and disease."[50]

Many residents of Philadelphia were undernourished, debilitated and riddled with various infections. There is little doubt that smallpox and yellow fever were implicated in more deaths than other diseases, but it is important not to underestimate intestinal parasitism in any estimate of the burden of infection in this population group.[51] Frequent references to parasitic worms in books and patent medicine advertisements indicate that infestations were both chronic and widespread in Philadelphia during much of the eighteenth century. These worms make their habitat in the human gastrointestinal tract. The four common soil-transmitted roundworms are *Ascaris lumbricoides*, *Trichuria trichiura* (whipworm), *Anclyostoma duodenale* (hookworm), and *Necator americanus* (hookworm). A high prevalence of fecal-oral and fecal-percutaneous worm infections was itself a strong indicator of a poor standard of living and hygiene. Considering the lack of effective sanitation in the city, it is reasonable to conclude that intestinal helminthism and intestinal protozoal infections were almost universal among the people of Philadelphia, especially the children. In her diary, Elizabeth Drinker makes note of her granddaughter's infection. "Elizabeth Skyrin [aged 6] voided a worm 9 ½ inches long, I cut it open

with my Sicers and found several young ones in it – she has taken medicine this even. on that acc."[52] Social class and economic standing played a very minor role in worm infestations among the children of Philadelphia; all children were susceptible.

A distinction needs to be made, however, between the parasitic intestinal diseases themselves, and the far more significant role of parasitism in preparing the way for secondary infection. For example, ascariasis is an infection with ascaris worms, acquired through feces-contaminated water or food. Symptoms may include cough, wheezing and fever or gastrointestinal complaints. Similarly, amebic dysentery is an inflammation of the intestine caused by the ameba *Entamoeba histolytica* which is usually acquired through feces-contaminated food or water, and is characterized by frequent, loose, usually blood-tinged stools.[53] Parasitism as a distinct disease, however, does not necessarily measure the extent of morbidity from intestinal disease accurately, as modern surveys in endemic areas of the world have shown. A parasitic infestation, as opposed to a mere infection, may be necessary to produce disease. Second, some resistance can be acquired against intestinal parasites, so reinfections in an endemic area like Philadelphia usually resulted in milder manifestations than occurred in those initially exposed. Since the level of exposure to intestinal parasites was high, it is reasonable to assume that over time, the general population of the town developed a corresponding level of tolerance.[54] This was not the case for infants and young children, however, where parasitic and intestinal diseases were one of the leading causes of death.[55]

Moderate parasitism of a degree insufficient to produce serious intestinal disease can play an important role in compromising the immune system, however, especially when it operates in conjunction with other debilitating factors, such as concurrent infection, malnutrition, or severe psychological stress. All animal parasites derive nutrients from their host. Although mutual tolerance is achieved in most cases, the host must protect itself with an abnormally high level of blood-forming and tissue-repairing activity. Anything that interferes with that defense mechanism results in a nutritional drain on the host and greater susceptibility to opportunistic infection. As parasitologists Henry Masur and Thomas C. Jones explain, "protozoa and helminthes that cause mild or inapparent infection in normal patients may cause devastating disease in immunodeficient patients." That process has become familiar since the beginning of the AIDS pandemic. Since so many of the residents of Philadelphia had compromised immune systems to one degree or another, it was understandable why many succumbed to disease as readily as they did.[56]

Parasitic infection in Philadelphia was sufficiently extensive to be an important determinant of increased susceptibility. While geofactors like climate, topography, and soil structure determine whether particular helminthiases can exist in an area, human factors determine whether or not the disease becomes endemic in an area. Since the majority of helminthes involve species whose eggs or larvae are passed out with the stool, sanitary conditions as well as behavior are of special significance. Generally speaking, soil-transmitted helminthiases are transmitted by

poor hygiene, food-borne helminthiasis by poor eating habits and food quality, and insect-borne helminthiasis by short distance of dwellings from vector breeding places. In her diary, Elizabeth Drinker writes "The flies are so numerous here, that I set still reading or writing for an hour, I find it necessary to wash my face and hands.[57] The living and environmental conditions of Philadelphia, along with the behavior of the residents favored transmission of all forms of helminthiases. All things considered, conditions in Philadelphia were ideal for the spreading of intestinal parasites.[58]

Philadelphia had undergone rapid and dramatic changes throughout the eighteenth-century. Whether these changes were cultural or biological, they presented conditions that challenged the community's ability to respond to disease. While all colonies experienced extraordinarily high mortality rates at the beginning of settlement, transition to a new and unfamiliar physical and disease environment – also known as "seasoning" – was not a uniform process. Some colonies adjusted quickly, and death rates fell within a few years. In Philadelphia, however, death rates remained high for an extended period of time. This was due, at least in part, to the biological characteristics of those who migrated, but it also grew out of the interactions between climatic, environmental, economic and social forces, as well as from an introduction of new pathogens, environmental modification and demographic changes brought about by contact with outside groups.[59]

Communities that engaged in overseas trade were naturally at a higher risk from imported diseases than their counterparts in the interior. During the second half

of the seventeenth century, smallpox arrived in the colonies as a result of these commercial contacts with other regions. The disease had assumed a more virulent character by this time.[60] This resulted in epidemics with significantly elevated mortality levels. In addition, the density of the population and the susceptibility of inhabitants – most of whom had no prior exposure – facilitated its spread in epidemic form. Smallpox, for example, took a heavy toll on human life in England and on the Continent, but it was largely absent in the colonies before 1700.[61] There are many possible reasons for this, but one obvious reason had to do with the length of the voyage from Europe. A typical Atlantic crossing took two to three months. Given the relatively brief incubation rates of most infectious diseases, it was obvious that even if an epidemic broke out after a vessel had left its home port; it would have run its course before reaching the colonies. If epidemic diseases reached the colonies – as they did toward the end of the seventeenth century – slow communication with the interior and a dispersed population ensured its containment within the town's borders. Smallpox can only maintain itself in social groupings of several thousand persons. In any smaller population, the disease would have quickly killed or immunized all available hosts and would itself have died out. Since infectious diseases remained the single most important cause of mortality during this period, the inability to disperse infectious organisms in America limited their impact.[62]

As the eighteenth century progressed, the factors that would have contained the spread of smallpox were no longer operating in many of the colonies. During the years between 1720 and 1760, Philadelphia established itself as a major port city. As

a port city, the population was exposed to diseases imported from other parts of the world. These diseases not only were carried by intercoastal trade up and down the Atlantic seaboard, but also were carried via transatlantic trade with Europe, Africa and the West Indies. Immigrants arrived with the infections native to their homelands, as well as with typhus and typhoid fevers acquired in the confines of ships. These diseases, though epidemic in nature, killed large portions of the urban population while nearby rural areas were spared high mortality rates.[63] Philadelphia had a large immigrant population, and these recent arrivals came in contact with new diseases without benefit of previous exposure. The large number of deaths from various fevers among those newly arrived, for example, can be attributed in part to their immigrant status and subsequent lack of immunity to local endemic and epidemic disease.

The highest per capita mortality in Philadelphia occurred in 1759, when seventy-seven persons died out of every thousand. Epidemics of smallpox, measles, typhoid fever and whooping cough were reported that year. None of these was the most fatal appearance of its kind, but, in combination, they raised the death rate to a level two and one half times that of the healthiest colonial year - 1755.[64] Insofar as the surviving documents show, epidemics in the colonial period affected all classes equally.[65] The major differentiating factor in survival between persons was the length of time in the area. Native-born adults, having built up immunities to the common diseases, were more likely to survive an epidemic. Immigrants and children were not yet "seasoned" to the disease environment and were highly susceptible.[66]

## Malnutrition and Poverty

Host susceptibility to infection is aggravated by malnutrition. A strong and consistent relationship has been found, for example, between childhood malnutrition and increased risk of death from diarrhea, acute respiratory infection, and possibly malaria.[67] Conversely, infectious processes, especially those associated with diarrhea, drive malnutrition in young children, so that diarrheal illness is both a cause and an effect of malnutrition.[68] Malnutrition diminishes host resistance to infection through a number of mechanisms, and virtually all bodily processes and physical barriers that keep infectious agents from invading the host are affected. These include the skin, mucous membranes, gastric acidity, absorptive capacity, intestinal flora, cell-mediated immunity, phagocyte function, and cytokine production.[69] Although multiple-nutrient deficiencies are much more common than single-nutrient deficiencies, lack of even one vitamin or mineral can impair the immune response. For example, vitamin A deficiency significantly increases the risk of severe illness and death from common childhood infections, such as diarrheal disease and measles, by diminishing the host's resistance to infection.[70]

How the complex connection between inadequate nourishment and disease works is not entirely clear, but we can be certain that it involves a weakening of resistance. Thomas McKeown explains that "response to any infectious disease depends on the state of health of the individual, and that state of health is influenced powerfully by nutrition."[71] His formulation is useful as a general proposition so long

as it is not taken as suggesting that well-nourished persons are always better protected against disease. The relationship is evidently far more complicated. It is more accurate to say that inadequate nutrition increases susceptibility to *some* infective agents and increases the severity of *some* infections once contracted, especially in children. On the one hand, it has been shown that the phagocytic cells (cells which search out and destroy invading microorganisms) of a malnourished host function only at 10 to 30 percent of normal efficiency. On the other hand, it is also true that malnutrition is sometimes antagonistic to certain infections, meaning that the agent is weakened more than the infected host. Viruses, for instance, depend on cellular nutrients for their own survival, so it stands to reason that the better nourished the host, the better the pathogens would thrive.[72] Different viruses behave differently, however, and it is not even likely that the same virus would interact identically with two equally nourished persons. Still, synergism (which results in weakened host resistance) is the norm. Nevin S. Scrimshaw summarizes the evidence as follows: "Malnutrition is almost always synergistic with infectious diseases due to bacteria, rickettsia, intestinal helminthes and intestinal protozoa, but with systemic viral, helminthic or protzoal infections, malnutrition is equally likely to be antagonistic or synergistic."[73]

The importance of nutritional intake in past population groups has received insufficient attention from historians. McKeown's provocative argument that improved nutrition (rather than advances in medical science) was principally responsible for falling mortality rates in the pre-twentieth-century industrialized

world should go far to remedy that neglect.[74] The World Health Organization's experience in developing regions gives credence to his speculation. One WHO expert has pointed out that most children became infected with the measles virus before the vaccine was developed. Death rates, however, were up to three hundred times higher in poorer countries.[75] The explanation, he supposes "was not that the virus was more virulent, nor that there were fewer medical services, but that in poorly nourished communities the microbes attack a host which, because of chronic malnutrition, is less able to resist."[76]

The complex process of acclimating to an environment, society and culture that differed from one's place of origin frequently had dramatic and often unequal consequences for health. In Philadelphia, the experiences of two groups - German immigrants and enslaved Africans - were especially suggestive. The travails of the former began during the difficult Atlantic crossing; surprisingly, mortality at sea was only about 3.8 percent. Debarkation mortality was in the same range, and the rate tended to fall over time. Those who settled in Philadelphia, however, remained at a severe disadvantage. From 1738 to 1756, for example, the annual average death rate[77] for first-year German immigrants was 61.4 per 1000, compared with only 37 (43 if you use Klepp's data) for established residents.[78]

German settlers suffered from nutritional diseases including beriberi[79] and scurvy.[80] Gottlieb Mittelberger described the voyage of a group of German immigrants in 1750 in his missive *Journey to Pennsylvania*. Food on Mittelberger's

journey was particularly poor. Warm food was served only three times per week, and the biscuit rations were full of red worms and spiders' nests. Even the water itself was "often very black, thick and full of worms, so that one cannot drink it without loathing, even with the greatest thirst." These crowded, fetid conditions were conducive to disease, and particularly affected those whose immune systems were not fully developed, or otherwise compromised. "It is a notable fact that children, who have not yet had the measles or small-pocks, generally get them on board ship, and mostly die of them."[81]

Given these dramatic circumstances, it is not surprising that enteric disorders took such a high toll among passengers during the voyage and after their arrival in the colonies. According to a contemporary account, the *St. Andrew*, which docked in Philadelphia on October 27, 1738,

> Lost 160 persons; another that arrived the day before lost over 150; and on that same the day following was said to have had only 13 well persons on board. Meantime another has arrived, in which out of 300, only 50 fares are left. They have mostly died from dysentery, skin sickness and inflammatory fever; likewise some of the captains and many seamen…Many of the survivors die after landing, and thus diseases are brought into the country, so that many inhabitants and landlords become sick, are seized by the epidemic and quickly carried off.[82]

Enslaved Africans faced somewhat different circumstances than immigrants from Western Europe. This group accounted for perhaps a quarter of the city's workforce from 1682 through the 1760s. Most came from the Caribbean or the southern colonies, and had been exposed to a variety of semitropical infectious

diseases. They were unaccustomed, however, to the harsher climate that characterized the Northeast portion of the colonies. Many were undernourished and afflicted with a variety of chronic infectious diseases. As a result, they had much higher mortality rates. Black mortality, for example, was about 50 percent higher than that of European immigrants. Seasonal patterns played an important role in accounting for some differences. White mortality tended to peak in the summer when malaria and enteric diseases were the major causes of death among Europeans unaccustomed to a warm and humid environment, while black mortality peaked in the winter when respiratory disorders were the leading causes of death.[83]

The reasons for the differences in mortality levels are not entirely clear. Native Philadelphians were at a higher risk of dying from smallpox; Germans were at a lower risk since they came from an area in which the disease was endemic and therefore were more likely to be immune. Yellow fever and malaria, however, took a greater toll among immigrants.[84] The complex nature of migration and adjustment precludes simple explanations. It is possible that interpopulation variation in susceptibility to infectious disease reflected underlying genetic differences that might have been under disease-mediated selection. In the case of infectious diseases, however, it was likely that environmental and cultural factors such as climate, living conditions, and sanitation had some effect on the species and densities of parasites and pathogens among the host populations.[85] Variation in culture, nutrition and hygiene, for example, will greatly affect both the rate at which individuals are infected and the course of the disease. According to Susan E. Klepp's study of racial

differences in mortality in Philadelphia, five known outbreaks of measles produced the highest average black death rates in the city between 1722 and 1775. The mean CDR for blacks during measles epidemics was 106, nearly double the white rate of 56.[86] Next in severity were eight reported epidemics of pleurisy and influenza, a finding that suggests a seasonal pattern in the death rates of blacks and a suspicion of vitamin D deficiency in slave diets.[87] Smallpox was a significant threat to all in the city who lacked immunity to the disease. Blacks, however, suffered substantially higher CDRs from the disease than whites; 82 deaths per thousand verses 52 deaths per thousand. Measles, smallpox and whooping cough were three of the four diseases coinciding with the highest black death rates, and were common diseases of children among European Americans. Furthermore, measles, respiratory infections, and whooping cough are diseases that spread more easily and are more fatal to malnourished subjects. As a result, when Africans came in contact with common European diseases, they experienced a proportionately higher death rate.[88]

Individual and community exposure to imported infection, epizootics,[89] micro-parasites, heavy metal toxicity and an assortment of environmental medical hazards affected both rich and poor in eighteenth-century Philadelphia. As such, the role of poverty in this study takes on a somewhat different meaning when linked to susceptibility to disease. While it was certainly true that the greater geographic mobility and the hazards peculiar to their work often meant that laboring Philadelphians generally suffered more illnesses and earlier deaths than did their more affluent neighbors, infectious disease was ubiquitous in the city. Since the

poorer citizens migrated more often than wealthier ones, they were exposed to different epidemiological environments and different health risks. Approximately one out of every four poor patients treated at the Philadelphia Dispensary during the late 1790s, for example, suffered from an infectious disease. These ailments took a high toll both in terms of morbidity and mortality.[90] The city and its immediate environment, however, were rife with diseases that did not discriminate on the basis of class or wealth. The lives and experiences of Elizabeth Drinker's family, in both sickness and health, were a fairly accurate cross-section of the best the times had to offer. Yet death, chronic disease with disfigurement, pain, and outrageously painful therapy fill page after page of her journal.[91]

> July13, 1778: "Billy [aged 11] unwell this even. with pain in his Bowels and bloody Stools we gave him Mollases and Butter."
> September 1, 1807: "My husband's disorder has turn'd on his bowels as usual, he has been very often this day moved and has taken 40 drops liquid ladunun going to bed-"
> September 2, 1807: "My husband very unwell in his bowels, he voids blood in his stools…"
> September 3, 1807: "Dr Rush visited my husband he advised another bleeding which was done, 8 ounces was taken…"[92]

Rich and poor lived side by side in Philadelphia, and they shared many of the same environmental health risks. Elizabeth Drinker and her family shared their alley with "one Dows a sailmaker," as well as with another person who turned out to be a thief. Directly opposite the Drinkers, a neighbor built a "large Soap House" which Mrs. Drinker conceded was "a disagreeable Surcumstance," and which attests to the absence of zoning regulations.[93] "Nearly opposite" the Drinkers in 1793 were Caty Prusia and her husband Christian, the biscuit baker, "Christopher the Barber near ye

corner, and a fringe maker, on the side of him."[94] The Drinkers may not have socialized with the "lower sort," but they were neighbors; living in the same alley, walking the same streets and drinking water from the same wells.

The violent swings in the mortality levels caused by epidemics in the first half of the eighteenth century lessened by the 1760s and, for the most part, disappeared by the nineteenth century. The interplay between human ecology and the microbial world is a complex process, and no one cause for the decreasing virulence of disease can be singled out from the changes in attitudes and practices in the second half of the eighteenth century. Public health measures, personal cleanliness, stricter enforcement of quarantine laws, smallpox inoculation, the natural immunization of an increasingly native-born population, the isolation of the very ill in hospitals, a decline in the crude birth rate all contributed to the smoothing out of the death rate.[95] While establishing habits of cleanliness may have reduced the incidence of certain diseases like typhus and typhoid fever, their effect on parasites, bacteria and viruses remains uncertain. "Some one or more contagious disease, being always more or less prevalent in the city, is one reason why a greater proportion of children die annually in the city than in the country," wrote William Curry in 1792.[96] Isolation was one of the best defenses against disease, but urban life promoted contacts between the sick, the healthy, the rich, the poor, the clean and the unclean. Changes in human culture, technology and environmental incursions nearly always have consequences for health and disease. The "Fourth Horseman"[97] has long followed in the footprints left by humans as they

entered new terrain, disturbed the natural environment, altered their patterns of settlement, and changed their patterns of contact with one another.

Although many factors contributed to the city's high mortality rates of the eighteenth century, the crude death rates (deaths per 1000 population) did decline over the long run. One of the most important factors contributing to this decline was the gradual creation of a global disease pool. Consequently, many infectious diseases became endemic rather than epidemic as the people of Philadelphia developed natural immunities to most common illnesses. The result, in epidemiological terms, was that the probability of contactbetween infectious agents and susceptible hosts had declined.

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[1] Scientists are using evolutionary concepts to explain why some viruses and bacteria are highly virulent and life-threatening, while others reside in their hosts with few, if any, ill effects. In order to understand these concepts, one has to look at infections from the point of view of the microbes: just another population of organisms trying to make a living by exploiting their environment (usually an infected host). A microbe or parasite must balance the amount of harm it does to its host against its ability to transmit itself. The more easily they can spread, the less it matters whether they seriously sicken and kill their hosts. That is the case with the microbe that causes cholera. As a diarrheal parasite, the bacterium spreads easily through bed sheets, clothes, and sewage-contaminated water. Therefore, virulence remains high, and infected people suffer and die. When water is purified and kept clean, the microbes cannot spread as rapidly. As a result, those bacteria that do spread are milder, and do not kill their host. Another example relates to HIV. This virus is more virulent when sexual activity is frequent and involves many partners, favoring a form of the virus that reproduces rapidly. When there is less sexual activity, the virus has fewer chances for transmitting its genes to another host. Under these conditions, natural selection favors forms of the virus that are more latent, allowing their hosts to stay healthy and active. Some scientists suggest that HIV might become less virulent over time, either because it infects so much of the population that there are no further opportunities to spread, or because shifts in behavior patterns cut down opportunities for infection. Seen through the evolutionary lens, it is understandable why certain diseases are so virulent and deadly. Malaria, yellow fever and typhus are spread by biting insects like mosquitoes (and lice). Their hosts do not have to survive long, because mosquitoes transmit the pathogens, so there is no selection pressure for the disease to be mild. Ewald, 2001; Levin, 1996, p. 93-102.

[2] Morris and Potter, 1997, p. 435.

[3] The body's defense mechanisms fall into two general categories: first-line and second-line. First-line defenses include external and mechanical barriers such as the skin, other body organs, and secretions. Intact skin, mucous membranes, certain chemical substances, specialized structures such as cilia, and normal microbial flora can stop pathogens from establishing themselves in the body. If an organism gets past the first-line of defense, white blood cells and the inflammatory response come into play. Because these components respond to any type of injury, their response is termed nonspecific. The

main function of the inflammatory response is to bring phagocytic cells (neutrophils and monocytes) to the inflamed area to destroy the invading microbes and rid the tissue spaces of dead and dying cells so that tissue repair can begin. Furthermore, by raising the body's temperature, fever defends against infection by enabling host defenses to inhibit the growth of pathogens. Certain microbes are unable to replicate at body temperatures above 100.4˚. A pathogen that gets past the body's nonspecific defenses confronts specific immune responses in the form of cell-mediated immunity or humoral immunity. Cell-mediated immunity involves T cells (a type of white blood cell), and humoral immunity is mediated by antibodies. Antibodies produced in response to the infectious agent help fight the infection. See Longworth, 2001, p. 20-1.

[4] Poor hygiene increases the risk of infection because untended skin is more likely to crack and break, allowing microbes to enter. Also, dirty skin harbors transient microbes, and microbial colonization of the skin increases. Removing microbial and fecal accumulation on clothing through frequent laundering is another important step toward controlling infection. See Longworth, 2001, p. 21.

[5] The very young and the very old are at higher risk for infection. The immune system does not fully develop until about age 6 months; at the infant's first exposure to an infectious agent, the infection usually wins out – especially if it is an upper respiratory infection. At the opposite end of the age spectrum, advancing age is associated with declining immune system function as well as with chronic diseases that weaken host defenses. See Longworth, 2001, p. 21.

[6] This section includes a discussion on ingested toxins and other environmental hazards.

[7] Susceptibility is broadly defined here to include exposure and response to disease.

[8] See Gehlbach, 2005, p. 86.

[9] Smith, 1990, p. 62.

[10] Morse, 1995, p. 7.

[11] In such a setting, the spectrum of infectious and parasitic diseases was greatly increased by two main factors. First, human settlements were pervaded by their own accumulated waste and excreta. This situation enabled the recycling of infectious agents, assisted by the proliferating rodents and insects. Second, many new and infectious agents may have been acquired from closely encountered animal populations. See McNeill, 1976, *Plagues and Peoples*. This book provides the classic historical account of infectious disease as an ecological entity, evolving in human populations in response to changes in environment, culture, and patterns of contact. Tuberculosis, for example, is a disease that can spread from animal to person. It can be expressed in various ways, depending on the route of infection and host immune response. Cows can transmit the mycobacterium through their milk, but the mammary glands are involved in only a small percentage of diseased cows. However, when individuals, usually children, consume infective raw milk from diseased cows, they can develop localized tuberculosis lesions within the intestinal tract, with involvement of associated regional lymph nodes. See Barnes, 2005, p. 161.

[12] Klepp, "Demography in Early Philadelphia," 1989, p. 104. The population includes the suburbs of Southwark and the Northern Liberties.

[13] Bridenbaugh, 1973, p. 18; Raufer, 1998, p. 23.

[14] The new opportunity afforded by large numbers of people cohabitating with domesticated and pest animals multiplied the likelihood of occurrence of mutant strains of microbe, some of which crossed the species divide. Epidemiologist A.J. McMichael argues that smallpox arose via a mutant pox virus from cattle; measles from the virus that causes distemper in dogs; leprosy from water buffalo; the common cold from horses, and so on. The list is very long, and the story continues today as we acquire from animal sources such infections as HIV and the Nipah virus. See McMichael, 2001, p. 101.

[15] Bridenbaugh, 1955, p. 30.

[16] Dysentery is an intestinal inflammation caused by bacteria, protozoa, parasites, or chemical irritants. It is marked by abdominal pain, frequent bloody stools, and rectal spasms. See Rothenberg et al, 2000, p. 172.

[17] Rush, 1815, "An Inquiry into the Cause and Cure of Cholera Infantum," Vol. II, p. 215-21.

[18] Klepp, 1989, *Philadelphia in Transition*, p. 238.

[19] Kiple, 2003, p. 337.

[20] Drinker, 1937, p. 26.

[21] Crane, 1991, p. 1142. Excerpt from the *Diary of Elizabeth Drinker*, March 5, 1799.

[22] Bridenbaugh, 1955, p. 105, 296; Drinker, 1937, p. 22-3; Rush, 1815, "Account of the Climate of Pennsylvania and Its Influence Upon the Human Body," Vol. II, p. 3-27; Klepp, 1989, *Philadelphia in Transition*, p. 226. Standing water created an ideal environment for mosquitoes to breed. The presence of the *Anopheles* mosquito allowed for the seasonal presence of malaria both in the city and the suburbs.
[23] See Bridenbaugh, 1938, p. 318.
[24] See Raufer, 1998, p. 60.
[25] Wolman, 1974, p. 247.
[26] The residents of Philadelphia were well acquainted with the environmental impacts of soap production. Like the saltpeter manufacturers, soap boilers had big pots in which various animal parts and drippings were cooked to provide the fatty acids needed for saponification. These facilities were located next to butchers and rendering areas, and were not particularly pleasant places. See Raufer, 1998, p. 83.
[27] Wolman, 1974, p. 259.
[28] Watson, 1891, Vol. I, p. 336-41.
[29] See Bridenbaugh, 1955, p. 239; Raufer, 1998, p. 60.
[30] Statutes at Large, Vol. V, p. 239-40; Wolman, 1974, p. 260.
[31] *Pennsylvania Gazette*, February 23, 1774.
[32] Smith, 1954, p. 82.
[33] Drinker, 1937, p. 25.
[34] Repeated exposure of human populations to infected rat fleas led to a new source of microbe interaction through the human louse. Human victims of rat flea-transmitted typhus managed to infect their own body lice, and the exchange of infection between humans and infected human lice favored strains of rickettsiae responsible for epidemic typhus among humans. See Barnes, 2005, p. 252.
[35] Harden, 2003, p. 352.
[36] The Christ Church Bills of Mortality consistently list deaths from "flux." Elizabeth Drinker's diary is replete with references to gastrointestinal disorders. Every member of her immediate family suffered from frequent bouts of dysentery.
[37] Crane, 1991, p. 314-5. Excerpt from the *Diary of Elizabeth Drinker*, July 6, 1778 and July 8, 1778.
[38] Duffy, 1953, p. 214-22. The Christ Church Bills of Mortality list deaths from "flux, purging and vomiting" on a yearly basis.
[39] Wolman, 1974, p. 270.
[40] Wolman, 1974, p. 278.
[41] Lead poisoning simply means the undesirable health effects induced by lead. Many are "nonspecific" and are similar to effects produced by other causes, and some are so subtle they require laboratory identification. The overt effects apparent upon even casual observation include abdominal colic, muscle paralysis and convulsions. See Aufderheide, 2003, p. 185. Deaths from the "dry gripes" and convulsions were frequently listed in the Christ Church Bills of Mortality.
[42] McCord, 1953, p. 393-9.
[43] See Aufderheide, 2003, p. 188.
[44] Lead also has a destructive effect on the nerves that transmit electrical impulses to muscles, producing muscle paralysis. Muscles raising the wrist or foot are especially affected, causing "wrist drop" (often termed "the dangles") and "foot drop." Behavioral disturbances leading to convulsions, coma, and death are the most severe of lead's effects. Children are notoriously susceptible to such brain toxicity, and even a single episode of convulsions, when not fatal, often causes permanent cerebral damage. Aufderheide, 2003, p. 185.
[45] Acute poisoning resulting from ingestion of excessive amounts of copper salts, most frequently copper sulfate, may produce death. The symptoms are vomiting, sometimes with a blue-green color observed in the vomitus, hematemesis (blood in vomit), melena (dark stools), coma and jaundice. See Goyer, 1996, p. 715.
[46] Alleyne, 1733, p. 11; Advertised in the *Pennsylvania Gazette* on December 30, 1762.
[47] The evidence of the toxicity of lead and copper had been piling up through the eighteenth century. Unfortunately, most Philadelphia physicians had learned to associate "dry gripes" with ingestion of lead from rum. They seemed unaware of the hazards in common household articles and the many

everyday practices of living. Milk, for example, was a constant cause of gastrointestinal complaints. Spoilage was only part of the problem. It is reasonable to assume that the lead compounds both in and on the serving pitchers contributed greatly to heavy metal toxicity in children.

[48] Malaria is a serious infectious illness characterized by recurrent episodes of chills, fever, headache, anemia, muscle ache, and an enlarged spleen. It results from infection by protozoans of the genus *Plasmodium* transmitted from human to human through the bite of an infected Anopheles mosquito or through blood transfusion or infected hypodermic needles. See Rothenberg et al, 2000, p. 336.

[49] Drinker, 1937, p. 9, 15, 33.

[50] How the process works is imperfectly understood even today, but immunologist Philip Y. Patterson has concluded from what we do know that "*any* diminution in *any* host defense system 'opens the door' to microbial invasion and disease." A heavy burden of pathogenic microparasities lowers the resistance of the host and increases susceptibility to secondary infection. Once such an opportunistic infection becomes established in an already immunocompromised host, it can quickly progress and easily disseminate. Disease with a high mortality rate, for example, may produce microorganisms too rapidly for an effective response, and overwhelm the already compromised defenses of the host and result in death. See De Bevoise, 1995, p. 50-1; Mandell et al, 1990, p. 33-146; Patterson, 1980, p. 741. On the immune system and host defense mechanisms, see Mandell et al, p. 33-146. For the quoted statement, see Patterson, p. 741.

[51] Human parasitic infection can be divided into two classes: protozoan and helminthic. Protozoa are one-celled organisms able to multiply within the gastrointestinal tract, while helminthes are multi-cellular organisms with complex life cycles that generally do not multiply within the gastrointestinal tract. Helminths can be divided into nematodes (roundworms), cestodes (tapeworms), and trematodes (flukes). Unlike protozoan parasites, most adult helminthes are incapable of increasing their numbers within their definitive host. As a result, the severity of clinical illness is related to the total number of worms acquired by the host over time. Small worm loads are, in fact, asymptomatic and may not require therapy. Many worms are long-lived, however, and repeated infections can result in very high worm loads with subsequent disability. See Ryan and Ray, 2004, p. 701.

[52] Crane, 1991, p. 1177. Excerpt from the *Diary of Elizabeth Drinker*, June 11, 1799.

[53] Longworth, 2001, p. 36-7.

[54] Schad and Banwell 1984, p. 362; Dammin, 1962, p. 1213-4; De Bevoise, 1995, p. 51.

[55] Deaths from "teeth and worms" were frequently listed in the Christ Church Bills of Mortality.

[56] Chandler and Reed, p. 1962, p. 18-9; Masur and Jones, 1980, p. 406, De Bevoise, 1995, p. 51. For the quoted statement, see Masur and Jones, p. 406.

[57] Crane, 1991, p. 705. Excerpt from the *Diary of Elizabeth Drinker*, July 16, 1795.

[58] House-flies can transmit protozoans including *Entamoeba*, *Cryptosporidium* and *Giardia*. In addition, house-flies can carry eggs of a variety of helminthes including *Ascaris*. See Service, 2004, p. 137.

[59] After an initial stage of "seasoning," new morbidity and mortality patterns generally appeared. They reflected a complex blend of environment, economic, social, and cultural variables. Climate, topography, and geographic location played a critical role in the distribution of human settlements. The same was true for those bacterial and viral organisms that gave rise to infectious diseases. Grob, 2002, p. 49, 59.

[60] Oddly enough, smallpox in Europe before the end of the sixteenth century existed in a relatively mild and non-lethal form. London Bills of Mortality before 1630 indicate that the disease killed less than 1 percent of its victims. Only after 1632 did smallpox become a virulent disease. See Carmichael and Silverstein, 1987, p. 146-68.

[61] Duffy, 1968, p. 27-9, 34-6; Blake, 1959, p. 23-9, 34-6; Rutman and Rutman, 1976, p. 48; Vinovskis, 1976, p. 278-9; Vinovskis, 1972, p. 195-201; Dobson, 1989, p. 259-97.

[62] Grob, 2002, p. 60.

[63] Klepp, 1989, *Philadelphia in Transition*, p. 226; Duffy, 1953, p. 14.

[64] In that year, there were no recorded epidemics and the death rate was 30 per 1000. Klepp, 1989, *Philadelphia in Transition*, p. 236-7.

[65] This was particularly evident in the writings of Elizabeth Drinker.

[66] Duffy, 1953, p. 240-6; Klepp, 1989, *Philadelphia in Transition*, p. 236-7.

[67] See Rice et al, 2000, p. 1207-21.

[68] See Mata, 1992, p. 16-27; Mata et al, 1977, p. 1215-27; Guerrant et al, 1992, p. 28-35.
[69] See Chandra, 1997, p. 460S-63S; Levander, 1997, 948S-50S. Note: cytokines are hormone-like low molecular weight proteins, secreted by many different cell types, which regulate the intensity and duration of immune responses and are involved in cell-to-cell communication. See Spraycar, 1995, *Stedman's Medical Dictionary*, 26th Edition, p. 437.
[70] See Smolinski, Hamburg and Lederberg, 2003, "Factors in Emergence," p. 63-4.
[71] McKeown, 1988, p. 55.
[72] This is debatable, however. Viruses depend on cellular nutrients in an indirect way. Because they are unable to reproduce on their own, they "hijack" the host's genetic machinery. Thus, if hosts are not reproducing, neither is the virus.
[73] Scrimshaw, 1968, p. 1679-81; De Bevoise, 1995, p. 56.
[74] De Bevoise, 1995, p. 56.
[75] For children deficient in vitamin A, the periodic supplying of high-dose vitamin A has reduced mortality by 23 percent overall and by up to 50 percent for those who suffer from acute measles. See Smolinski, Hamburg and Lederberg, 2003, "Factors in Emergence," p. 64. The relative risk of measles mortality in children younger than 2 years of age has been shown to be significantly reduced when the children's diets are supplemented with vitamin A for only 2 days. See Barclay et al, 1987, p. 294-6; West, 2000, S46-54.
[76] On measles see Behar, 1974, p. 29; on malnutrition and infection see Newberne and Williams, 1970, p. 93.
[77] The burials of new arrivals were estimated as a percentage of the burials in the city's "Strangers' Ground." This ground accommodated those who died without belonging to a parish or without colonial relatives who took responsibility for their burial. From 1738 to 1756 these burials consisted largely of newly arrived German immigrants. In order to separate German immigrants from other burials in the Strangers' Ground, the proportion buried in that ground relative to total burials in the city during the years when German immigrants did not arrive (1757-1762) was taken as representative of the proportion of non-German immigrant burials in that ground for the years 1738 to 1756. See the assessment of Franklin in Labaree, 1959-1973, Vol. III, p. 439. The bills of mortality directly stated that "Dutch and Other White People" were buried in this ground up through 1756 when the "Dutch" heading was dropped. The 1754 colonial council minutes also stated that newly arrived German immigrants were buried in the Strangers' Ground. See *Colonial Records of Pennsylvania*, Vol. VI, p. 173. The procedure used to separate German immigrant from other burials may be very accurate, since the number of German immigrant burials estimated for 1754 exactly matched the number reported buried there in 1754 by the colonial council. See Grubb, 1987, p. 580, 580n.
[78] Fourteen German immigrant vessels, which enumerated passenger deaths directly in the ship records, were taken from the Strassburger collection of German ship lists for the port of Philadelphia, and used by Farley Grubb in an effort to derive a less biased estimate of voyage mortality. According to Grubb, the sample had over 1,566 passengers, covered the years 1727 to 1805, and appeared to be relatively representative of the typical immigrant voyage: voluntary, white, civilian immigrants transported by the private shipping market on the North Atlantic route. All fourteen reported on adult men, but only six had mortality enumerated for the separate categories of adult men, adult women, and children. Three ships listed only immigrants and adult men. The overall passage mortality for these 1,566 Germans was 3.8 percent, an average of about 70 percent below the passage mortality for enslaved individuals. The voyage mortality for the 1,153 adult men was slightly above that for the 237 adult women, 3.5 verses 2.5 percent, respectively. The 382 children fared far worse with a passage mortality of over 9 percent or almost three times the adult rate. See Grubb, 1987, p. 570; Klepp, 1994, p. 473-506.
[79] Beriberi is a disease caused by a deficiency of thiamine that is expressed in three major clusters of symptoms, which vary from person to person. It may involve edema, or swelling, of the legs, arms and face. The nerves may be affected, causing first a loss of sensation in the peripheral nerves and later paralysis. The cardiovascular system also may be involved, evidenced by enlargement of the heart and extremely low diastolic blood pressure. In its chronic form, beriberi may result in disability for months or years; or it may be acute and produce death in a few weeks. Until major tissue damage occurs, it is curable and reversible by consumption of thiamine. The populations in which beriberi has been most prevalent have been of two kinds: people confined to institutions such as prisons, asylums and naval

ships, who are limited to monotonous and restricted diets such as bread and water or fish and rice; and people who derive a large portion of their calories from rice from which milling has removed most of the bran in which thiamine is found. See Meade, 2003, p. 44-5.

[80] Scurvy is a dietary deficiency disease, arising from lack of vitamin C (ascorbic acid). It usually occurs in the absence of fruits and vegetables in the diet. The characteristic features of scurvy have been experimentally monitored. At 12 weeks without vitamin C, a feeling of lethargy appears. At 19 weeks, the skin becomes dry and rough, and hair follicles form lumps. Small hemorrhages in the legs begin at 23 weeks; a bit later, fresh wounds will not heal. A classic symptom – swollen, soft, purple gums – appear after 30 weeks. In a mid-twentieth century study, one volunteer developed a tubercular lesion at 26 weeks, and two others suffered apparent cardiac hemorrhages at 36 and 38 weeks. They were clearly near that stage of the disease that killed many thousands of sailors. When volunteers were given large doses of ascorbic acid, all made complete recoveries. Historical reports of symptoms involving far more severe cases add flaccidity of flesh, loosening of teeth, and reopening of wounds to the list of symptoms. See French, 2003, p. 295.

[81] Mittelberger, 1960, p. 15-6.

[82] Westergaard, 1932, p. 12.

[83] This paragraph is based on Klepp, 1994, p. 473-506; Klepp, 1991*, "The Swift Progress of Population": A Documentary and Bibliographic Study of Philadelphia's Growth, 1642-1859*.

[84] Major outbreaks of smallpox were associated with above average mortality among Philadelphians. However, these epidemics were also associated with below average mortality among first year immigrants, most notably for the years 1751, 1752, and 1756. Yellow fever had the opposite impact. Philadelphia experienced a yellow fever outbreak in 1741 and a typhus outbreak in 1754. Both of these years experienced dramatic peaks in immigrant post-voyage mortality. The mortality of resident Philadelphians was above average in 1741 and below average in 1754. Since these epidemiological events affected immigrants and established residents differently, it is reasonable to assume that some level of differential mortality between these two groups existed. See Duffy, 1953, p. 153-5; Grubb, 1987, p. 584.

[85] Ecological changes, including those due to agricultural or economic development, are among the most frequently identified factors in the emergence of infectious disease. Ecological factors usually precipitate emergence by placing people in contact with a natural reservoir or host for an infection hitherto unfamiliar but usually already present (often a zoonotic or arthropod-borne infection), either by increasing proximity or by changing conditions so as to favor an increased population of the microbe or its natural host. The emergence of Lyme disease in the United States, for example, was probably due largely to reforestation, which increased the population of deer and the deer tick, the vector of Lyme disease. The movement of people into these areas placed a larger population in close proximity to the vector. See Morse, 1995, p. 9.

[86] The most generous available estimates of Philadelphia's African American population were used in this study. Black CDRs should therefore be viewed as lower bound estimates for the period before 1788. Deaths are from the surviving Philadelphia bills of mortality originally published in newspapers, almanacs, and broadsides by Philadelphia publishers, churches and the Board of Health. These bills are reproduced and analyzed in Susan E. Klepp, 1981, *"The Swift Progress of Population": A Documentary and Bibliographic Study of Philadelphia's Growth, 1642-1859*. Stillbirths are included in total deaths. See Klepp, 1994, p. 506.

[87] Both blacks and whites fared better in years with moderate winters, but the relative advantage was substantially greater for blacks. Cold winters, as defined here, would be associated with unstable weather patterns with increased cloud cover. One consequence of overcast skies is a reduced ability to utilize vitamin D, which is synthesized by the body through exposure to sunlight. Vitamin D deficiency predisposes individuals to respiratory and skin diseases. In northern, less sunny climates, dark-skinned people especially can suffer from vitamin D deficiency unless dietary sources compensate for the body's inability to produce sufficient amounts of the vitamin from the available sunlight. See Kiple and King, 1981, p. 10-1, 91-2. Higher black mortality in Philadelphia coincided with cold winters, so that even if blacks and whites had fared equally in terms of food, clothing and shelter, black deaths would have surpassed those of whites in cold, cloudy weather. See Klepp, 1994, p. 479-80.

[88] Klepp, 1994, p. 489.

[89] An epizootic is an epidemic outbreak of disease in an animal population, often with the implication that it may also affect human populations. See Last, 2001, p. 62.
[90] Smith, 1990, p. 53.
[91] Drinker, 1937, p. 21. As a member of the elite, Drinker certainly incorporates into her journal some degree of bias, but if we keep in mind that her perceptions were those of a genteel scribe, it is possible to retrieve an impression of the world surrounding her with a fair degree of accuracy. Sometimes a single sentence opens an avenue of thought or confirms what historians have only suspected; other times the continual repletion of an idea or sentence illuminates a way of life long lost to the twenty-first century. Crane, 1991, p. 3-28.
[92] Crane, 1991, p. 316, 2070, 2071. Excerpts from the *Diary of Elizabeth Drinker*, July 13, 1778 and September 1-3, 1807.
[93] Crane, 1991, p. 425. Excerpt from the *Diary of Elizabeth Drinker*, July 20, 1784.
[94] Crane, 1991, p. 499. Excerpt from the *Diary of Elizabeth Drinker*, September 2, 1793.
[95] Klepp, 1989, *Philadelphia in Transition*, p. 236.
[96] Currie, 1792, p. 100.
[97] *Holy Bible* (King James Version). The Revelation of St. John the Divine. Chapter 6, verses 1-8. This graphic text was written while St. John was in political exile on the Greek island of Patamos during 95-97 AD. It contains a vivid fantasy-like account of the opening of the Seven Seals, which give an account of the apocalyptic destiny of mankind. The first four seals reveal four terrible horsemen astride colored horses: white (conquest), red (warfare), black (famine), and pale (death or pestilence). Taken from McMichael, 2001, p. 366.

# CHAPTER 5

# SMALLPOX, MEASLES AND YELLOW FEVER: THE CONSEQUENCES OF DENSE URBAN LIVING

Historians have focused much attention on both the vulnerability of Philadelphia's poor during outbreaks of disease, and the fundamental role of poverty in the emergence and spread of infection in the eighteenth-century city.[1] Using poverty as a primary analytical tool, however, camouflages other relevant issues and obscures a more accurate understanding of how the convergence of any number of factors can create an environment in which infectious diseases emerge and become rooted in society.[2] International trade and commerce, migration, public health policy, ecological changes, unsanitary conditions, poor hygiene, microbial adaptation and disruptions due to war all played a role in disease emergence, but the increase in size and density of the population may have had the most dramatic effect on acute infectious disease. Demographically significant infectious diseases are generally categorized as either acute or chronic. The former have short latency and infectious periods and a short illness followed by either transient or permanent immunity. The latter have slow recovery rates, long periods of infectiousness, and do not result in permanent immunity.[3] Spectacular in their appearance, acute infectious diseases like smallpox, measles and yellow fever[4] were serious threats to health and longevity in

Philadelphia.[5] Each required large populations in order to become endemic (always present), since smaller populations did not have a large enough annual input of susceptible people, and the disease subsequently would died out. When reintroduced, those born since the previous epidemic all were vulnerable to infection. As population increased, these diseases tended to become endemic and common in childhood. Once becoming endemic, they were less lethal than when they afflicted adolescents and young adults.[6]

Population density is essential for the propagation of smallpox, measles and yellow fever, but density plays much less of a role in the epidemiology of other diseases. Specifically, chronic infectious diseases and those acute infectious diseases that do *not* produce solid immunity do not depend on a densely populated community in order to spread. Individuals may be reinfected several times or may maintain the same infection for many years. Such diseases could therefore become well established in relatively small communities, although some could become epidemic at times.[7] Nonetheless, the demographically significant infectious diseases in these categories - acute with transient immunity or chronic - generally assumed an endemic, stable pattern earlier than those that evoked life-long immunity after an initial contact. Once introduced into a community, they could be maintained by relatively fewer individuals. As a result, they did not depend upon the continuous introduction of large numbers of susceptibles. Typhoid fever, dysentery, malaria, and tuberculosis are all in this category, and will be considered in Chapter 6. Although often overshadowed by the extraordinary nature of periodic smallpox and yellow

fever outbreaks, chronic diseases played a far more significant role in shaping morbidity and mortality patterns in Philadelphia.

Since diseases are expressed biologically in individuals, it is often tempting to assume that the causes of disease and the solutions to their control and prevention are also biological and lie at the individual level. Many diseases, however, are caused only by the interaction of individuals within and between populations, and most are profoundly influenced by such interactions.[8] Consequently, the causes of disease are often social.[9] For example, a high proportion of immune persons in a sparsely populated community generally precluded epidemic diseases, because the capacity to transmit the pathogen from person to person was impaired. Whether through immigration or births, the increase in the number of susceptible persons in a community created conditions conducive to the spread of infections.[10] Philadelphia's population grew rapidly, and exceeded 18,000 by 1760.[11] Crowded living conditions,[12] crude sewage disposal, and stagnant and contaminated water characterized the city, and outbreaks of infectious disease were common.[13] Even by contemporary standards, Philadelphia was an unhealthy city, and a dangerous place in which to live.

The general unhealthiness of the city, in combination with precarious medical knowledge and soaring levels of immigration created the right combination of circumstances to drive up the city's death rate. Smallpox epidemics were particularly deadly, and were a frequent source of contagion in the city through the mid 1770s.

Smallpox has often been described as the most infectious human disease known,[14] killing both adults and children and, on occasion, wiping out entire families. During the outbreak of 1762, for example, both Jurg Albrecht and his wife Maria died of smallpox in July, and the last of their five orphaned children succumbed to the disease in November of the same year.[15] Smallpox also decimated the family of George Claypoole in 1731, killing him and four of his children.[16] The fundamental requirement for the establishment and maintenance of smallpox in a population is that the number of susceptible individuals exceeds a threshold density, and nowhere in eighteenth-century North America was smallpox more persistent than in Philadelphia. By the time of the Revolutionary War, the city may well have been the only place on the continent where the disease had actually become endemic. Records indicate that after several serious outbreaks during the French and Indian War (1754-63), smallpox had a constant presence in the city, ebbing and flowing cyclically, as it did in British cities.[17]

## Smallpox

Smallpox is caused by the variola virus, *Orthopoxvirus variola*, and two subgenera of smallpox exist – *variola major* and *variola minor*. It is likely that all smallpox cases in Philadelphia during the eighteenth century were *variola major*. Five clinical types of *variola major* (or classical smallpox) are commonly recognized and are classified according to the nature and development of the rash – hemorrhagic, flat, modified, noneruptive, and ordinary.[18] Data do not exist on the distribution of

cases among the five *variola major* types in the Philadelphia outbreaks. Nor is there ascertainable information on how that distribution changed during periods of massive immigration into the city, concurrent epidemics with other diseases, war, or other stresses inflicted on the population. Modern research suggests, however, that the most severe cases of smallpox[19] seem to correlate with an inadequate immune response. If it could be shown, for example, that case-fatality rates increased as the eighteenth century progressed, there would be compelling evidence of a progressively debilitated population. Unfortunately, this type of analysis is not possible. It is not even clear to modern researchers just what suppresses the immune response to the smallpox virus. Additionally, not much is known about the effect of the various nonspecific mechanisms that form part of the host's defenses. What *is* known, however, is that age is certainly a factor, since immune systems function less effectively in the very young and the very old, and case-fatality rates are accordingly the highest at the extremes of the age spectrum. Hormonal changes are important as well. Pregnant woman have a much greater incidence of hemorrhagic smallpox, for example, due to the elevated levels of a chemical that results in impairment of the immune response.[20] It is also clear that malnutrition interferes with host defenses, but despite a few scattered indications that smallpox and malnourishment may be synergistic, the latter connection has never been proved. Little is known of the effects of multiple concurrent infections, as well as the effects of the psychological stress of wartime conditions.[21] Notwithstanding the lack of empirical evidence, it is reasonable to assume that either susceptibility to or severity of smallpox increased as a consequence of the multitude of eighteenth-century assaults on the host defenses of

the people of Philadelphia. Such assaults included a high level of urban density enabling the virus to easily spread from person to person, a continuous flow of sick and susceptible individuals into the community, chronic disease and debility, and haphazard inoculation procedures that further intensified the emergence and spread of the disease throughout the city.

*Variola* behaves like a "hit and run" pathogen with human-to-human transfer. It thrived in eighteenth-century Philadelphia's large, dense, urban population. Since no carrier state for this virus by asymptomatic human host exists, once it infects someone, it either overcomes the immune system to produce disease, or dies from a fervent immune attack. The virus can only be transmitted to another person when symptoms of the disease develop.[22] It is released through moist droplets from infected upper respiratory passages, from pus-filled skin lesions known as pustules, or from dried scabs falling off healing lesions. Fortunately, with recovery from smallpox comes lifelong immunity to the disease. And when the virus runs out of fresh victims, it dies out from the infected population.[23] Considering that smallpox outbreaks occurred in Philadelphia every few years, lifelong immunity was a significant benefit. Nevertheless, a continuous flow of people through the city constantly replenished the pool of susceptible individuals.

The virus causes infection when it enters the airway passages of the host's lungs.[24] Since it becomes airborne from infectious mouth and throat lesions coughed or sneezed into the air, most smallpox infections are acquired through close contact

with an infective individual. Additionally, dried scabs and draining skin pustules contaminate bed linens and soiled clothing, and handling of these materials easily disperse the virus into the air. Flies, too, have been suspected of transmitting the virus from draining skin pustules of one individual to the nasal passageway of another.[25] However it got dispersed,[26] infection usually required close contact with the virus. Because the virus was sensitive to sunlight and extreme temperatures, it did not travel far on its own outside a host.

Transmission of the disease normally resulted from direct interpersonal contact. Lacking both an animal reservoir and the ability to remain latent within the body, smallpox could exist only as an active infection. Modern investigators in Asia have established that propagation of the infection often occurred as a result of close family contacts, particularly between those who slept in the same room or bed. Transmission between persons living in the same house but who did not share sleeping quarters was the next most frequent method.[27] Sharing a bed with a family member was common practice in eighteenth-century Philadelphia. Unfortunately, prevention of smallpox transmission and epidemic spread required strict isolation of the patient, effective vaccination, surveillance of contacts, proper burial, and disinfection of the premises, bedding, laundry, and personal items. Although quarantine was attempted, and inoculation practiced, other attempts to contain the spread of the disease were inadequate. Consequently, the conditions that prevailed in Philadelphia did much to enhance the spread of smallpox. Congested streets, crowded houses and a continuous flow of susceptible people into the city were only

parts of the problem, however. Nearly every city resident eventually contracted the disease either naturally or through inoculation. Effective as it was, inoculation came at a price. Those who had the procedure actually developed a mild case of smallpox, and, like anyone else sick with the disease, they could pass it on to others in the "natural" way.[28] In the absence of both strict quarantine and a clear understanding of contagion, inoculation could start an epidemic. Because the symptoms could be mild, some who had undergone the procedure felt well enough to circulate in public, and frequently did so. Abigail Adams, for example, who had expressed her own fear of the contagion earlier, "attended publick worship constantly, except one day and a half" while she underwent inoculation in 1776.[29]

While a large number of citizens began to be inoculated after 1760, many strongly resisted variolation, fearing correctly that infected persons could spread the disease among the unprotected residents. As a result, the process had not gained widespread acceptance even two decades after its introduction into the city in 1736. The first inoculation mentioned in Elizabeth Drinker's diary occurred on December 13, 1762, thirty-one years after its introduction.[30] The procedure began in the American colonies in 1721 when Zabdiel Boylston, a Boston physician, learned of the practice from Cotton Mather. When smallpox arrived in Philadelphia in 1731, Benjamin Franklin, who was in Boston during that city's devastating outbreak in 1721, began a tireless campaign to promote the procedure. According to Franklin, only one of about fifty inoculated persons died. In Philadelphia's 1736-7 epidemic, 129 persons were inoculated, again with only one fatality. Franklin reported that by

1752, eight hundred persons had been inoculated, with only four deaths. In the 1750s, publications and lectures by physicians urged Philadelphians to adopt inoculation, and in 1759, a New Jersey physician opened a house in Philadelphia exclusively for that purpose. Elite Philadelphians banded together to found the Society for Inoculation of the Poor in 1774, making the procedure available to a vulnerable part of the population that could not afford the three pounds doctors charged for the preparation and inoculation of patients.[31]

The city quickly became a center of inoculation for all of the British colonies in North America, attracting patients from as far away as the West Indies. In other regions, there was much more opposition to the procedure, and it was barred in several colonies including Connecticut, New York, Virginia and Maryland, and it was not taken up in Massachusetts until later in the century.[32] Historian Elizabeth Fenn argues that the high incidence of smallpox in Philadelphia stemmed, at least in part, from the reluctance of the authorities to regulate inoculation. While officials in other colonies were cautious or opposed to the operation, those in Philadelphia may have been a bit irresponsible with their policy. Quarantines were rare, and restrictions on variolation were virtually nonexistent. As a result, the practice flourished[33] in the city – and so did the disease.

As inoculees circulated through the streets of Philadelphia, they could have passed on the infection in its more dangerous "natural form" to those susceptible to the disease.[34] Smallpox posed a particular danger to members of the Continental

Congress, many of whom came from states where smallpox was rare and inoculation banned. During the congressional sessions in Philadelphia, susceptible delegates were at considerable risk for contracting the infection. To reduce the chance of contagion for vulnerable delegates, Philadelphia physicians agreed to halt all inoculations while the First Continental Congress met in 1774. After 1774, fear of smallpox ran so deep that delegates actually sought out the procedure. Patrick Henry of Virginia went through the process under the care of Benjamin Rush, Matthew Thornton of New Hampshire was inoculated by an unnamed physician, and Connecticut's Samuel Huntington, who had already chanced catching the disease while attending earlier meetings, went through it in the same year.[35]

Despite efforts at containment, it was difficult to prevent the spread of smallpox to neighboring towns and colonies with a high proportion of susceptible persons. If proper quarantine procedures were not followed (as was often the case in Philadelphia), those undergoing inoculation were hastening the start of an epidemic. Additionally, the movement of people engaged in trade and commerce provided a convenient means of transporting the virus. Many fled from epidemic areas despite quarantine measures, and this further facilitated the dissemination of the virus. The war with the French in the 1760s merely exacerbated the problem. Susceptible soldiers – many of whom came from areas untouched by smallpox – were infected and spread the disease upon their return home.[36]

No single group of people was more likely to carry smallpox away from Philadelphia than soldiers in the Continental army. Because of its central location on the Atlantic seaboard, the city was a logical stopping point for newly enlisted southern recruits on their way north to join Washington's army. Southerners were among those most likely to be vulnerable to *variola*.[37] Soldiers not only endangered their own lives, but the lives of those around them. A soldier could pick up smallpox in Philadelphia and then march for nearly two weeks before developing symptoms and infecting fellow soldiers. The authorities were aware of this, and President John Hancock ordered regiments marching from Virginia to New Jersey to go around "Phil. On Acc. Of the Small Pox." Six months later the problem remained, and Virginia troops once again received instructions "to avoid Philadelphia where the Infection now prevails." The contagious city exasperated Washington. "I would wish to have the small Pox entirely out of Philadelphia," he wrote, but acknowledged that it simply could not be done. Only if the troops were immune could they pass through the Quaker city without risk.[38]

While many who suffered from smallpox eventually recovered, Elizabeth Drinker was correct in noting the differences in mortality between those who contracted it naturally and those who were inoculated. "Went this morning to Thos. Says, whose daughter Becky, lays ill, in the Small Pox, which she has taken the natural way; and to most that take it Naturally (at this time) it proves mortal."[39] If given a choice, inoculation was a better route than contracting the disease in "the natural way." The practice of inoculation, however, was different from vaccination,

the much safer procedure that was developed by Edward Jenner in 1796 using the cowpox virus.[40] In inoculation, matter taken from a pustule of someone who had caught the disease naturally was placed in several small cuts made by a needle or a lancet in the arm or leg muscles of the person receiving the inoculation. This method produced a milder, less fatal form of smallpox, but the person was still contagious.[41] Accurate statistics do not exist for Philadelphia, but in the Boston epidemic of 1752 almost 10 percent of those who caught the disease naturally died, and in the 1764 epidemic the figure was close to 18 percent. The comparable mortality figures for those inoculated were 1.4 and 0.9 percent.[42]

Control of an infectious disease through inoculation (or vaccination) does not depend on every last person being immunized. Rather, the objective is to keep the pool of susceptibles permanently below the level at which the virus can sustain the chain of infection. The concept of *herd immunity* explains why this is, and is the theoretical basis for modern eradication programs. Simply stated, herd immunity is a measure of the cumulative resistance of a group, and it varies from disease to disease. Just as individual resistance decreases the probability that a person will contract an infection, so herd immunity reduces the chances that an epidemic will develop in a larger segment of the population. That is because the susceptibility of the group is less than the sum of the individual vulnerabilities of its members. Group resistance is the product of the number of susceptibles and the probability of their exposure to infected persons. Because not every susceptible person comes into contact with an infective agent, the probability of contact is the intervening factor that makes group

resistance greater than would be expected from the total of individual susceptibles. The goal of vaccination is to reduce the number of susceptibles in the population below a critical threshold under which the probability of contact with an infectious agent is so low that epidemic spread is highly unlikely.[43] Although the complexities of herd immunity were unknown to eighteenth-century medicine, inoculation nonetheless helped to contain outbreaks of the disease.

Smallpox continuously changed and evolved as its environment changed and evolved. By the late seventeenth century, smallpox was assuming a more virulent form in Europe. Possibly a lethal strain of *variola major* had been imported from Africa or the Orient, or the virus may have mutated. Nevertheless, the relatively benign form of the disease that existed before 1630 was superseded by a more malignant one. London's large population, however, permitted the disease to exist in endemic form. Between 1731 and 1765 the average number of deaths per year was 2,080 or 9 percent of total mortality.[44] The pattern of smallpox in the colonies was different, however, because no colonial town approached the size of metropolitan London. In the British mainland colonies, an epidemic was succeeded by a period of years in which the disease was absent. During the interval between epidemics the number of susceptible persons gradually increased, and the stage was set for another outbreak. This interval was particularly short in Philadelphia, where a steady flow of immigrants, soldiers, visitors and delegates constantly refreshed the pool of susceptible individuals. Many of these new arrivals crowded into the heavily congested area along the Delaware River.

Philadelphia's first major recorded epidemic was in 1716, extending from August to October.[45] In 1722, both London and Boston experienced severe epidemics, but no mention was made of any outbreak in Philadelphia between 1716 and 1731 by the *American Weekly Mercury* or the *Pennsylvania Gazette*. The *Mercury* did not make a practice of publishing mortality information, but in 1722 it did print monthly statistics on the burials in the city, and these give no evidence of excess smallpox.[46] In 1731, after it had appeared in Boston and just before it became prominent in New York City, virulent smallpox broke out in Philadelphia.[47] Undoubtedly, there were many susceptibles that year, reflecting the long interval since the previous epidemic. Of the more than 500 deaths in 1731, when Philadelphia had a population of approximately 7,000,[48] "the number of those that died of that distemper is exactly 288 and no more."[49] Smallpox reappeared once again in Philadelphia in the fall of 1736[50] and continued for several months. It did not appear again in epidemic form until 1746.[51]

An especially virulent smallpox epidemic struck Philadelphia in 1759, and mortality was high among those not inoculated.[52] It was estimated that five hundred to six hundred deaths were caused by the disease that year. Philadelphians were especially prone to outbreaks of smallpox during the French and Indian War (1756-63) because of the many susceptible soldiers and refugees who streamed through the city. During the war years alone, there were three major outbreaks, and between 1712 and 1773, Philadelphia suffered ten major outbreaks. According to Benjamin

Franklin, there were five "visitations of smallpox" between 1730 and 1752, and Elizabeth Drinker noted several others between 1759 and 1775.[53]

Smallpox was particularly lethal in the city during the third quarter of the eighteenth century, accounting for most of the annual fluctuations in the mortality rate before 1775. Nearly every peak in the death rate during the century's third quarter resulted from an outbreak of the disease. The lethality of the disease lessened as the century progressed, with a corresponding moderation in the city's death rates at the end of the colonial period.[54] The decline of smallpox-induced deaths is evident among Anglicans during the 1760s and early 1770s: the disease caused 26 percent of their burials during the 1750s, 18 percent between 1761 and 1765, and 11 percent from 1765 to the Revolution. At the same time, epidemics became less severe: the outbreaks of 1756 and 1759 accounted for nearly 60 percent of the deaths among Anglicans, whereas the epidemic years between 1766 and 1775 were marked by a high of only 31 percent.[55]

Each disease confers its own measure of actively acquired natural resistance on its survivors, and smallpox grants absolute, lifelong protection against reinfection.[56] That fortunate circumstance reduces the number of susceptibles in the population and makes subsequent outbreaks less devastating than if the virus were introduced into a completely nonresistant group. As a result, the epidemiology of smallpox changes as the virus establishes endemicity within a given population. Resistance acquired by previous infection is vital to a susceptible community because

it assures that epidemics cannot be an annual occurrence. Generally, intervals between contagions will vary according to the volume and frequency of community contact with external reservoirs.[57] In Philadelphia, a variety of circumstances replenished the pool of susceptibles so quickly that the city experienced repeated outbreaks of the disease. The bills of mortality indicate that smallpox killed many in 1751, 1756, 1759, 1762, 1763, 1765, 1769 and 1773.[58] Elizabeth Drinker's diary is filled with references to the disease, and underscores the ubiquity of smallpox both in the city and in the lives of every resident. All of the Drinker children were inoculated, as well as those who worked in the Drinker household. Elizabeth Drinker well understood the importance of inoculation, and regarded smallpox as an inescapable enemy. If children, servants, and retainers were not protected from it, inevitably they would succumb to the disease. As each new servant came to the family, there was the query as to whether or not he or she had had smallpox or had been inoculated; and, if not, there is frequent mention of carrying through the difficult and expensive procedure.[59]

As lethal as the disease was, the common treatment for smallpox was also quite dangerous, and contributed to the general debility in the city. Mercury and mercury-containing compounds were widely used in Philadelphia both to treat the disease and to prepare the body for inoculation. Most of the preparations then in use – cinnabar, liquid mercury, mercurous chloride (calomel), and mercuric chloride (bichloride of mercury) were quite toxic, and once in the body, they could accumulate in and damage the kidneys and brain. Salivation and sore mouth, which some

eighteenth-century physicians regarded as signs that the medications were active, are now known as early signs of mercury poisoning. Other early symptoms of poisoning include loss of appetite, loss of weight, nervous system irritability, and insomnia. More advanced exposure leads to damaged kidneys, muscle cramps, tremor and changes in personality. Benjamin Rush was correct in his observation that mercury as used with smallpox led to glandular swelling, loss of teeth, and a "weak habit of the body" after the disease.[60]

Various inorganic preparations of mercury were also being refined and developed during this period to treat smallpox and other diseases. The most widely used of all mercurial drugs was calomel, or mercurous chloride. It was used as a cathartic (laxative), diuretic, emetic (substance that induces vomiting), expectorant, anthelminthic and antivenereal.[61] In its capacity as a cathartic and emetic, it was commonly used in preparing the patient for smallpox inoculation. On December 16, 1765, Elizabeth Drinker notes in her diary that we "began to Physick Nancy and Polly in order for Innoculation. 16 a dose to purge, 17 a Pill 18 a Pill 19 ditto 20 ditto 21 a Purge 22 a Pill 23 ditto 24 ditto 25 ditto 26 they were Innoculated by [J.] Redman, Docr. Evens Present: took Nothing that Day, 27 took nothing 28, took a Pill, 29 a Purge 30 at night or 31 in the morning Nancy grew Feverish and unwell which seems very early her Leg has been sore since she took the infection, 31 they took cooling Powders, twice.[62]

Other pharmaceuticals were in use in Philadelphia at this time as well. During the smallpox epidemic of 1750-51, the *Pennsylvania Gazette* carried advertisements for numerous patent medicines and home remedies. The one most prominently featured was *Godfrey's cordial*. When this was first advertised, it was called "a great strengthener for the stomach, whether relaxed by bad wine or long sickness, or other causes;" and was recommended for healing of wounds of horses and the sickness of cattle. By 1750, it was "approved for the cholick and all manner of pains in the bowels, fluxes, fevers, small pox, measles, rheumatism, coughs, cold and restlessness in men, women and children and particularly for several ailments incident to child-bearing women and relief of young children breeding their teeth."[63] The chief ingredient of *Godfrey's cordial* was opium; an effective remedy for pain.

Eighteenth-century medicine was, itself, quite dangerous and undoubtedly contributed to the high morbidity and mortality rates in the city. Like mercury, antimony was an ingredient in many of the compounds commonly used to treat smallpox and a variety of other diseases, and it, too, was quite toxic to the body. Antimony is considered a minor toxic metal. It has been used medicinally in the treatment of such diseases as schistosomiasis and leishmaniasis, and its metabolism resembles that of arsenic. It is absorbed slowly through the gastrointestinal tract, and many antimony compounds are gastrointestinal irritants. These compounds may also cause alterations in cardiac function. Modern autopsy studies have shown that cardiac toxicity was often the cause of death in patients treated with antimonial drugs.[64]

Elizabeth Drinker makes note of one particularly tragic story in her diary. It was copied from the December 2, 1762 edition of the *Pennsylvania Gazette*. "Last week were interred in one Grave, three children of one Family in this City, who last Their lives by a most unfortunate accident. It had been proposed, it seems, to prepare their Bodies for the Small-Pox, by giving them some Creme of Tartar, which was accordingly sent for to an Apothecary's Shop; by mistake Tartar Emetic was delivered and administered instead of it which by its Excess of quantity, and violent operation, soon brought on Death. The Grief of the Parents, who have no other Children is inexpressible."[65] One of the substances most often used in preparing the patient for inoculation was Cream of Tartar or powdered potassium bitartrate. This was used as both a laxative and a diuretic. Another was Rochelle salt or sodium potassium tartrate, and it was used as a mild, cooling laxative. Tartar emetic, or antimony potassium tartrate was a popular eighteenth-century remedy used to induce both vomiting and sweating. It was also used as an expectorant, laxative and sedative, and it was known to cause poisoning at high doses.[66] Unfortunately for patients, poisoning was not uncommon and was a frequent by-product of medical treatment. The children mentioned by Drinker were most certainly poisoned by an overdose of tartar emetic; an antimony-containing compound.

Smallpox became less virulent in Philadelphia in the latter portion of the eighteenth-century. No one cause for this can be singled out from the changes in attitudes and practices that existed at the time. Public health measures, personal cleanliness, stricter enforcement of quarantine laws, and inoculation each may have

played a role. More importantly, fewer migrants disembarking at the city's docks during the post-Revolutionary period spread less disease among the residents and, as a result, contributed fewer burials to the cemeteries.[67] One of the most significant reasons for the decline in smallpox mortality in the city, however, was the reduction in the number of susceptible people. As more and more city residents developed immunity to the disease, the potential breeding ground for smallpox contracted steadily.

## Measles

Measles is a viral infection of short duration, and its characteristic symptoms include fever, spotted rash, and a cough. Measles is caused by a species of *Morbillivirus* and is a highly contagious disease. It is transmitted primarily by droplet spread via contact of susceptible individuals with nose and throat secretions from infected persons. Measles has no reservoir other than humans, and needs a continuous chain of susceptible contacts in order to sustain transmission. The period of communicability generally lasts up to four days after the start of the rash, and, like smallpox, has no carrier state. The incubation period from exposure to onset of the rash is approximately fourteen days.[68] It is generally regarded as a disease of childhood, if only because the loss of temporary immunity from the mother renders the infant susceptible at an early age. After an epidemic, the number of immune persons reaches a peak. For another epidemic to occur, several years have to pass before the number of susceptible individuals reaches a critical mass. Although

mortality rates varied, it was not regarded as a particularly lethal disease in Europe during the eighteenth century.[69] The manifestation of the disease in Philadelphia and other colonial cities, however, was quite different.

The checkered history of this disease in colonial America well illustrates the complex relationship between disease patterns and the larger environment. In the seventeenth century, measles appeared only sporadically and had not yet assumed an endemic character. It was, however, a very serious disease, affecting both young and old. The brief incubation period and highly contagious nature of the disease reduced the odds of bringing measles to the colonies; an outbreak at sea would generally run its course before a ship arrived in port. After 1700, however, measles became much more common. The growth of trade and construction of faster ships facilitated the importation of the virus into port communities, and subsequently into interior regions. The disease first appeared in Boston in 1713, and then spread southward into New York, New Jersey and eventually into Pennsylvania.[70]

The first reference to measles in the Philadelphia records was in 1714. The epidemic that started in Boston during the winter of 1713 was widespread in Philadelphia by the spring of 1714.[71] Mortality among those who were infected by a close relative tended to be higher than among those infected in the community at large, as was evident during the epidemic of 1713-1714. In Boston, the epidemic devastated the household of Cotton Mather; his wife came down with the disease, and four of his children and the maid died.[72] Overall mortality from measles was very

high in Philadelphia as well, although some epidemics were more severe than others. The mortality rates in 1747, 1759, and 1772 were particularly devastating and contributed greatly to the overall death rates for these years.[73] In a letter to his mother in October, 1749, Benjamin Franklin rejoiced that his family had remained well despite much illness in town. The "Measles and the flux," he reported had "carried off many children," while a number of adults also had fallen victim to the disease.[74] Measles was often accompanied by what was then termed "pleurisy." The notation of 23 deaths from measles in 1747 was followed by 39 from pleurisy in 1748 in Christ Church parish.[75] It is difficult to determine whether these diseases were independent but concurrent, or whether "pleurisy" was a complication of measles. Complications from secondary infection often include pneumonia and diarrhea. Diarrhea is one of the most important causes of measles-associated mortality in developing countries today.[76] Frequent references to "flux" and "pleurisy" indicated that serious complications accompanied measles outbreaks in Philadelphia, and no doubt contributed to the increased mortality rates.

As for smallpox, Philadelphia's ever-growing population provided a constant pool of susceptible individuals, creating the necessary environment for epidemics of measles to occur. Two of the most severe epidemics occurred in 1759 and 1772. The population of Philadelphia in 1759 was just over 18,000 people. That year, approximately 113 deaths occurred from measles alone. In 1772, the population had grown to approximately 29,000, with 180 deaths from measles.[77] Although the reasons for such high mortality rates are not entirely clear, it is reasonable to assume

that measles, which struck adults as well as children, in combination with other common diseases such as dysentery, influenza, diphtheria, and scarlet fever, caused large numbers of deaths. During the 1759 epidemic, for example, there were concurrent outbreaks of measles, smallpox and typhus.[78] Concurrent infections certainly compromised the hosts, and may have significantly increased over-all mortality rates.

Infectious diseases traditionally associated with infancy and childhood in Europe became more common in the American colonies during the eighteenth century, but here they exhibited very different characteristics. Many, like measles, were not indigenous to the colonies, and when they were imported, young and old alike were susceptible. Although both groups contracted the disease, children were particularly susceptible because of their less developed immune system. Once newborns and infants no longer enjoyed the passive transfer of maternal immunity through breast feeding, they were vulnerable to a host of infections. This resulted in widespread epidemics with relatively high death rates even among the adult population.[79]

Malnutrition and overcrowding have long been implicated in measles mortality rates. One World Health Organization expert has pointed out that until vaccines were developed, most children became infected with the measles virus, but death rates were up to three hundred times higher in poorer countries. He argues that the virus was not more virulent, nor that there were fewer medical services; but in

poorly nourished communities, the microbes attack a host which, because of chronic malnutrition, is less able to resist. P.M Newberne and G. Williams have surveyed the scientific data and conclude that malnutrition influences infection "(1) by effects on the host which facilitate initial invasion of the infectious agent; (2) through an effect on the agent once it is established in the tissues; (3) through an effect on secondary infection; or (4) by retarding convalescence from infection."[80]

Recent findings in genetic and epidemiological research on measles, however, have added a great deal to our understanding of the disease. In studying mortality rates from the disease, Peter Aaby and colleagues found that such host factors as malnutrition and age of infection were not sufficient elements in mortality. They did find, however, that mortality rates were much higher when the disease was contracted from a member of the same household or from a relative than when it was contracted from others in the community. In Africa's Guinea-Bissau, for example, the mortality rate for isolated cases and index cases (the first case contracted outside the home) was 8 percent, as compared with 23 percent for secondary cases in the household. In a study in Senegal that took 1.0 as the mortality rate for cases contracted in the community, Aaby found the rate would be 1.9 if contracted from a cousin, 2.3 from a half-sibling, and 3.8 from a full sibling. Other data demonstrated the absence of any relationship between severity of measles and the dose of the virus to which the individual was exposed. On the basis of these findings, Francis L. Black has suggested that high death rates in the Americas from European infectious diseases are related to the fact that the virus grown in one host became adapted to the immune

response of that individual. When induced in a genetically similar host, it gained in virulence and resulted in higher mortality rates. Genetic homogeneity, in other words, enhanced viral virulence as it passed through ingrown communities and households.[81] This perhaps explains the particular lethality of the disease in the household of Cotton Mather.

Housing arrangements, culture, and tradition further magnified the virulence of measles in Philadelphia.[82] Close living conditions and the absence of an understanding of contagion ensured both the rapid spread of disease and the increase in virulence. Elevated mortality rates may also have stemmed, in part, from the failure of measles to become established in an endemic form. In England and on the Continent, densely populated urban areas always included large numbers of susceptible people. Consequently, many infectious diseases assumed an endemic character, and became less lethal. Epidemics in relatively small eighteenth-century colonial communities, by contrast, reduced the number of susceptible persons to the point where no further outbreak was possible. Over a period of time, the number of susceptibles would increase and the stage would be set for another outbreak of the disease. This cycle was certainly evident in Philadelphia with outbreaks occurring in 1747, 1759, 1772, 1778, 1783, 1789, 1795 and 1796.[83]

Elizabeth Drinker's diary offers convincing evidence that physical distress invaded daily life in the city to a degree currently unimaginable. The measles epidemic of 1772 made its presence known in the Drinker household. Four of

Elizabeth Drinker's children were sick with the disease. At the time Sally was eleven, Nancy eight, Billy five and Henry two. "1772 Octor. 2 the Measels came out on Sally, after being five days very unwell…" "1772 Octor. 15 Nancy has the Measels coming out on her, tho not so kindly as could be wish'd, little Henry they are just appearing on 'tho he has been very unwell for a week past, they are both very poorly." "Octor. 18 First Day: My dear little Henry very much oppress'd, his Fever very high the Mesels have never come out as they should have done; he was let Blood this Evening." "28 Billy very full of the Measels, little Henry continues very poorly."[84] All of the children survived this outbreak. Measles again visited the Drinker household in 1783. In March of that year, Molly Drinker had the disease. She was nine years old at the time. The following month, Elizabeth Drinker's two year old son Charles came down with the disease. While both children recovered, Charles died of diphtheria the following year.[85]

Sporadic epidemics of measles were common throughout the colonies, but the New England and Middle Atlantic regions were the hardest hit. The Chesapeake and southern colonies - areas where population density was lower - were somewhat less affected. In the areas where the disease was more prevalent, there was a steady decline in the intervals between epidemics. The interval in Boston declined from thirty to eleven years between its initial appearance in 1657 and 1772. A similar pattern was evident in Philadelphia, and after 1795 there was not much regularity to measles epidemics. The disease had become endemic in Philadelphia, with a small

number of deaths reported in Christ Church Parish nearly every year. There were two deaths reported in 1795, nine in 1796, one in 1797, one in 1799, and four in 1800.[86]

## Yellow Fever

Smallpox and measles were by no means the only imported diseases. Yellow fever was another, and in many respects, a more terrifying disease. Aside from its higher mortality rate, it was a relatively "new" disease to Europeans and colonials, and consequently aroused greater fears than older but more familiar diseases because of its unpredictable character. The distinctiveness of yellow fever reinforced anxiety and fear. Its symptoms were dramatic; its appearance and disappearance were seemingly random; and it respected neither class, status nor gender. Yet, unlike smallpox and measles, it was not transmitted by infected individuals who moved from an epidemic area; it was transmitted by the mosquito. A dense urban environment was still necessary for its spread, though, since the range of the mosquito was limited.

Yellow fever is an acute viral disease transmitted to humans by various mosquitoes, especially the *Aedes aegypti* (formerly *Stegomyia fasciata*). The disease remains endemic in tropical regions of Africa and the Americas in a sylvan or jungle form, but historically its greatest impact on humans has been in an epidemic or urban form. It presents symptoms ranging from mild to malignant, classically including fever, headache, jaundice, and gastrointestinal hemorrhage (black vomit). High mortality rates were recorded during epidemics (20-70 percent), although today we

know that yellow fever mortality is actually relatively low, suggesting that many cases were mild and undiagnosed.[87]

Yellow fever is normally a disease of nonhuman primates, particularly monkeys. Mosquitoes transmit the disease among them – but species of mosquitoes that do not ordinarily bite humans. This form of the disease is known as jungle yellow fever or sylvan yellow fever; it is enzootic, meaning that the disease is present at low levels at all times in an animal community. In the case of sylvan yellow fever, transmission is from monkey to mosquito to monkey. Humans can become tangential hosts through accidental intrusion into a zoonotic transmission cycle (transmission of a disease to humans from an animal host or reservoir), but are not important in maintaining the sequence of infection.[88] When the virus is carried by an infected human to populated areas where transmission is from human to *A. aegypti* mosquito to human, the disease is termed urban yellow fever.[89] It was urban yellow fever that visited Philadelphia several times in the eighteenth and early nineteenth centuries.

It is now generally accepted that the cradle of yellow fever was West Africa. If so, both virus and vector had to be imported, and the many challenges facing the pair in making a transatlantic voyage and taking root in the Americas probably account for the relatively late debut of yellow fever in the Caribbean. This migration most likely occurred within the last 400 years, and it is reasonable to assume that huge numbers of *A. aegypti* arrived in the West Indies with early trading and slaving voyages. A ship in tropical waters with many individuals in a confined space, and

water casks close at hand provided the migrating insects with a nearly ideal habitat. The yellow fever virus also made many early voyages, although not as easily as its mosquito vector. Presumably, the virus would have boarded a ship in the bodies of non-immune sailors or slaves (most likely the very young) during the first three to five days of illness when that virus was still in the blood, and yellow fever was virtually symptomless. It was also during this early period of viremia (presence of virus particles in the blood) that one or more of the female *A. aegypti* mosquitoes aboard would have had to have bitten the victims, for the patient is no longer infective after the virus leaves the blood and yellow fever's distinctive features of hemorrhage, high fever, jaundice, and black vomit appear. The infected mosquitoes would then have had to survive the generally requisite 9 to 12 days necessary to incubate the virus (although the period could be as long as 30 days depending on the temperature) before they could pass it along to another human on board. After the incubation period, the mosquito remains infectious for whatever remains of a lifetime that generally lasts a month, although life spans of three to four months have been recorded.[90]

Philadelphia was the center of government and the center for mercantile trade and shipbuilding, making it both the economic and political heart of the colonies. These factors combined to draw large numbers of visitors, immigrants and migrants to the city, dramatically increasing the population of the city as the century progressed. This steady influx of new arrivals, rather than birth rate alone, was largely responsible for this growth. Immigrants and migrants converged on

Philadelphia from three directions in the 1790s.[91] European immigrants, mostly Irish and Scots-Irish, arrived after a long and often grueling sea voyage – a voyage that left many sick and malnourished. Refugees escaping the Haitian Revolution also arrived in the city. Unlike their European counterparts, West Indian refugees were often merchants, doctors, lawyers, and planters.[92] Arriving in large numbers in the summer of 1793 from an area where yellow fever was endemic, this group most likely served as a viral reservoir for local *A. aegypti* mosquitoes, thereby initiating the epidemic of 1793.[93] Accompanying these refugees on their journey were countless infected mosquitoes: stowaways on board ships. They, too, could have transported the disease into the city. The final group to converge on Philadelphia consisted of migrants from the countryside in search of better prospects, many quite vulnerable to infectious disease.[94]

## The Vector

It is conceivable that the growth of the sugar industry in both the West Indies and the colonies assisted the *A. aegypti* in becoming acclimated to its new surroundings.[95] In addition to blood meals, the mosquito is attracted to and nourished by sweet fluids. Adult mosquitoes of both sexes of most species regularly feed on plant sugar throughout life, but only females feed on vertebrate blood.[96] The boiling of cane juices, the reboiling of molasses, and the piling of waste stalks around plantations certainly would have made a great deal of sucrose available to mosquitoes on a year-round basis. In addition, other features of sugar production would have

contributed to mosquito proliferation, especially the use of artificial containers such as clay pots, which, when discarded or simply left outdoors to be filled with rain water, became ideal breeding vessels.[97]

Philadelphia was a major distribution center for imported sugars and molasses, and it was no coincidence that the city's several visitations of yellow fever all were attributed to contact with the West Indies. The epidemic of 1762, in particular, was traced to a West Indies ship docked at Sugar House Wharf below South Street.[98] The city later became an important sugar refining center, with the first refinery being built in 1777, and three more in operation before the close of the century. The industry demanded large importations of raw sugar from the Caribbean. The city had extensive commercial links to the Sugar Islands, with the "bulk of sugar consumed in the United States before 1789 coming from Philadelphia refineries."[99] By providing exceptional feeding opportunities, imported sugars may have helped to temporarily increase mosquito population in an area not suited to year-round infestation. This could contribute to an epidemic of yellow fever in a non-sugar growing locale such as Philadelphia.

While the sugar industry may have played a role in mosquito proliferation in the city, the habits of the female *A. aegypti* had much to do with shaping the characteristics of an epidemic. It is a domestic mosquito, living close to humans, depending on them for blood meals, and breeding in nearby loci of water. *A. aegypti* often breeds in pots and water storage jars placed either inside or outside houses. Its

range is short, at most a few hundred yards meaning that it requires a fairly dense human population to spread disease. Because *A. aegypti* can survive only days without water and requires water in which to breed (although eggs can survive for years in dehydrated form), adequate rainfall is a prerequisite for epidemic yellow fever. Warm weather is another prerequisite. *A. aegypti* does not bite in temperatures below 62˚F and hibernates in extended chilly weather.[100] Adults of most *Aedes* species bite mainly during the day or early evening.[101] This particular mosquito was well suited to Philadelphia's densely packed urban environment and flourished in the city during the summer months.

The host-finding behavior of mosquitoes also plays a role in the ecology of yellow fever. This behavior involves the use of volatile chemicals (those that evaporate rapidly) to locate vertebrate hosts; carbon dioxide and lactic acid are among the best-documented *host attractants*. Other skin emanations also are important. Fatty acids produced by normal bacteria flora of the skin, for example, are particularly effective in attracting the malaria vector *Anopheles gambiae* to human feet. Mixtures of these fatty acids probably play a role in attracting most mosquitoes. Subtle differences in the odors of different host species, and even different individuals, undoubtedly play a role in host preference. These odors commonly have a combined effective range of 7-30 m, but the range can be up to 50 m for some species. Vision also is important in orienting to hosts, particularly for diurnal species and especially in an open environment and at intermediate or close ranges. Dark, contrasting, and moving objects are particularly attractive. As the female approaches

to within 1-2 m of a potential host, chemical and visual cues are still important, but convective heat and humidity surrounding the body also come into play. Odor, carbon dioxide, heat and humidity all are detected by sensilla on the antennae and palps of the mosquito.[102] Exposed skin, sweat, and the lack of personal hygiene among the residents of Philadelphia may have played a role in attracting mosquitoes. Mosquitoes can feed from a variety of skin surfaces, and they can penetrate mucus, matted hair, light layers of feathers, and heavy cloth, provided it is not thicker than the length of the proboscis.[103] It stands to reason that those who spent a great deal of time outdoors were more likely to be bitten by the insects.[104] Also, certain types of clothing may have allowed for more exposed areas of skin, thus creating a larger surface area for the mosquito to target. This could explain, at least in part, why more men contracted the disease than women.

All things considered, it is not surprising that the mosquito thrived in eighteenth-century Philadelphia. Near the waterfront, many people were crowded into a complex maze of dirty, dark alleys and densely packed buildings. During the 1793 yellow fever epidemic, Samuel Jackson described the neighborhood near the waterfront in the following manner: "Those infected were scattered over the City in every quarter; many in Water Street, in various narrow & uncleanly alleys, & in small crowded & ill-ventilated dwellings."[105] Mosquitoes aboard vessels docking in Philadelphia found a hospitable environment near the waterfront, in the marshes west of the city and in the vicinity of Dock Creek, a stream that "barely oozed from the swamps through the city's heart to the Delaware River."[106]

## The Virus

It is evident that much was needed for the mosquito to survive and thrive in Philadelphia, but the yellow fever virus also had some distinctive requirements for transmission – a process in which humans are best thought of as the site where the virus changes mosquitoes. This exchange can take place only during the first 3-6 days of infection of the yellow fever victim, while the virus still remains in the blood (viremia); after the virus has entered the mosquito, it must incubate for another 9-18 days before it can be transmitted to another human. After this period of extrinsic incubation, however, the virus is transmissible for the life of the mosquito, which can be upward of 180 days, although generally the lifespan of the female *A*. aegypti is closer to a month or two.[107] Yellow fever does not harm the mosquito, but the consequences of infection for the susceptible human host are somewhat variable. Some develop illness of varying severity with viremia, whereas others may have long-term viremia without clinical disease. These hosts are then a source of further spread of the virus, since non-infected mosquitoes can feed on them and acquire the virus, thereby increasing the risks of transmission.[108]

The season-to-season survival of the virus is also complex and has multiple mechanisms. Mosquitoes are rarely present during all seasons in a locale as far north as Philadelphia. The question then arises as to how the arboviruses[109] survive between the time the vector disappears and the time it reappears in subsequent years. Several mechanisms can operate to sustain the virus between transmission periods

(often referred to as overwintering). One such mechanism is sustained viremia in lower vertebrates such as small mammals, birds, and snakes, from which newly mature mosquitoes can be infected when taking a blood meal. Another is the hibernation of adult insects that survive from one season to the next. A third mechanism, and one most associated with *A. aegypti*, is transovarial transmission, whereby the infected female transmits virus to its progeny.[110]

Transovarial transmission is more often associated with tick-borne diseases than with mosquitoes, but studies indicate that viruses can survive through dry seasons in eggs deposited by infected females.[111] Specifically, the yellow fever virus may be transovarially transmitted in *Aedes* species. This means that both the eggs and the larvae that emerge from them already are infected with the virus. Venereal transmission within the mosquito population is also possible when the virus is passed from congenitally infected males to females during mating.[112] The importance of these routes of infection remains unclear, but such mechanisms, especially transovarial transmission, may explain the virulence of the back-to-back outbreaks in Philadelphia during the summers of 1797, 1798, and 1799. The newly hatched larvae in combination with a group of susceptible individuals may have provided the necessary ingredients for an outbreak of the disease.

Unlike smallpox and measles, yellow fever is intimately related to climate and geography. Since temperature and moisture were critical to the lifecycle of the mosquito, the disease could not become endemic in Philadelphia because the

mosquito could not survive the winter temperatures. Typically, the virus had to be reintroduced for an epidemic to begin again. The expansion of foreign and domestic commerce, in combination with urban growth, made possible the introduction of many different pathogens into the city. During the eighteenth century, yellow fever was a disease confined to port cities with commercial ties to tropical areas, as well as to communities located on rivers that flowed into these ports. Philadelphia's brisk trade with the Caribbean greatly contributed to the frequency of yellow fever in the city, with epidemics in 1741, 1747, 1762, 1793, 1794, 1797, 1798, 1799, 1800, 1802, 1803 and 1805. While the gap in epidemics between 1762 and 1793 may have been due to the interruption in trade and immigration that occurred during the Revolutionary crisis, the absence of a native *Aedes* mosquito population certainly played a role in the ultimate disappearance of the disease in the city, with the last recorded outbreak in 1822.

The many factors associated with the pathogenesis of yellow fever highlight the complex relationships that existed in Philadelphia between pathogens, insect vectors, humans, and their environment. The virus, for example, must establish a cycle that allows indefinite transfer from mosquito to human host to mosquito. This requires a large number of mosquitoes. Without them, the virus cannot move from person to person rapidly enough: people have the disease only 7 to 10 days, and their blood is infective for only 3 to 6 days. The cycle also needs a favorable ratio of nonimmune to immune people available for the mosquito to bite. The mosquito only lives a few weeks, and immune people are virus-killers. So, in order to perpetuate the

cycle, a sufficient quantity of infected *A. aegypti* must quickly find a sufficient quantity of susceptible hosts. The cycle of transmission is broken when mosquitoes inject the virus only into immunized bloodstreams.[113] Philadelphia's densely populated city streets, along with standing water and a constant influx of susceptible individuals and disease-carrying mosquitoes "set the table" very nicely for the yellow fever virus.

## The Disease

Yellow fever was a relatively new disease in the Americas, and was absent in the mainland colonies for most of the seventeenth century. The disease first struck Barbados in 1647, where it resulted in an estimated 5,000 deaths. Ultimately, it spread to other Caribbean islands as well as Central and South America. The first epidemic in the colonies occurred in Boston in 1693 following the arrival of British vessels from Barbados. Six years later, yellow fever appeared in Charleston and Philadelphia. Yellow fever had a long history in Philadelphia, with the first outbreak in 1699 perhaps being the most lethal. The population of the city at the time was just over 2,200, and more than 300 deaths were attributed to the disease.[114] While many fled the city during the outbreak, those who remained had no immunity to the disease and were extremely susceptible to infection. Most of what is known about this epidemic comes from letters written by residents. According to one witness, the disease was introduced into Philadelphia by "a ship from Barbados whose cargo consisted of cotton in bags which were landed at a wharf between Market Street and

the draw-bridge and there stored for sale."[115] It broke out first in this neighborhood and then spread gradually through the city with "great mortality." Another spectator ascribed its origin to the stench of the pits from the two tanyards on Dock Street fronting the river. Lambert, owner of one of these yards, died within two days from a violent attack, and, soon afterward, many families in the area had the infection.[116] As with later epidemics, the disease intensified in late August and early September and did not subside until after cooler weather prevailed.

The fever was absent from the city for forty-two years, and did not reappear until 1741. This outbreak, while not as lethal as the one in 1699, lasted from early June through early October. The disease followed its characteristic pattern of attacking those new to the city. It was known as "the palatine fever" since it prevailed among recently arrived German immigrants who undoubtedly had no prior exposure to the virus and were therefore susceptible to the disease. Additionally, newly arrived Irish immigrants also were susceptible to the disease and were blamed as the source of the infection in several accounts of the epidemic. Dr. Phineas Bond, for example, claimed the disease originated among "a number of convicts from the Dublin jail."[117] Once in the city, the fever appeared along the waterfront, attacking those who had no prior exposure to the virus.

In the wake of this outbreak, serious attempts were made on the part of Philadelphia's medical community to ascertain the source of the contagion. All of the surviving medical documents indicate that the medical community was convinced the

disease occurred as a result of importation. None of the accounts give any hint of domestic origin for the disease. Dr. James Lind, for example, claimed the disease arrived in the city in "a trunk of wearing apparel belonging to a gentleman who had died of the fever in Barbados, and that it proved mortal to more than two hundred of the inhabitants."[118] The constant reference to the Caribbean as a source of the disease was quite telling. Epidemics of yellow fever were common in the Caribbean during the period between 1690 and 1770, when the proportion of non-immunes in the population there was at its highest. These outbreaks tended to appear in areas where there was rapid development.[119] In the 1730s and 1740s, these areas included Barbados, Martinique and Saint Domingue.[120]

The fever next visited Philadelphia in 1747, arriving in late June and remaining through the beginning of October. The CDR reached a high of 57 per thousand that year, with epidemics of yellow fever, measles, malaria and influenza all raging in the city.[121] The disease followed its usual pattern, appearing first along the wharves on the Delaware River and confining itself to the southern part of town. The fever was particularly lethal in the neighborhood near the Dock, which, in 1747, was a muddy stream that crossed three major city streets. The Dock was used widely as a garbage dump, and was the focus of several legislative attempts to clean up the city. During the 1790s, the city finally allocated funds to cover portions of the creek. Although this was a significant measure to combat disease, those living in the vicinity of Dock Street still experienced significant mortality during subsequent outbreaks of yellow fever.[122]

Yellow fever appeared again in Philadelphia in August of 1762 and did not subside until October of that year. This was the first outbreak of the disease to occur during the period of Elizabeth Drinker's diary. Little mention of the disease was made in the diary, except for the brief entry in mid-September where Drinker notes "A Sickley time in Phila. many Persons are taken down, with Something very like the Yallow-Feaver."[123] The Drinkers were out of town at their summer home in Frankfort during most of the period of the epidemic, although Henry Drinker and Elizabeth's sister Mary made frequent visits to the city to conduct business. Yellow fever was only one of three epidemics to visit Philadelphia that year. Smallpox and typhoid fever also were present. With three concurrent epidemics raging in the city, it is not surprising that the CDR reached a high of 59 deaths per thousand that year.[124]

Yellow fever was believed to have been brought into the city by a ship from the West Indies which docked at Sugar House Wharf below South Street. As Dr. Redman tells the story, "three of the men who landed there died of a contagious fever." The first patient was a sick sailor smuggled ashore from a vessel coming from "Havannah," where the disease raged. The owner of the house who received the sailor "with most of his family and many others in that court soon after fell a sacrifice to the distemper; and from thence it spread rapidly, first affecting the houses nearest."[125] Although the fever initially appeared in the low, wet ground south of the city, it rapidly moved north. By late September, physicians were treating more than twenty-five patients a day, with no relief in sight. Once in the city, yellow fever

remained close to the waterfront and to small tenements and back alleys where sailors lived.[126]

Yellow fever returned again to Philadelphia during the summer of 1793, after a thirty-year break. It is possible that the lull from 1762-1793 was directly linked to the absence of yellow fever in the Lesser Antilles during this period.[127] The series of yellow fever epidemics to visit Philadelphia and other port communities beginning in 1793 were undoubtedly the result of the dramatic upsurge in the incidence of the disease in the West Indies. The escalation can be linked to a variety of factors, including the Anglo-French wars, the Haitian revolt, and the large-scale introduction of susceptible European troops into the region.[128]

The disease appeared in the city in eight out of twelve subsequent years, killing some ten thousand citizens over that period. It arrived in devastating form in 1793, tripling the annual CDR rates of the preceding five years. According to Susan E. Klepp's assessment of the best available evidence, death rates among Philadelphia's original inhabitants averaged between 64 and 98 per thousand of those infected during the epidemic.[129] As devastating as it was, not all deaths resulted directly from yellow fever. Dysentery, tuberculosis and other endemic diseases severely compromised the health of many residents, making them particularly vulnerable to yellow fever. Fatality was considerably higher among those who did not flee the city; as many as one in five died. Although yellow fever reappeared in Philadelphia and other cities during the summer and fall months for at least the next

dozen years, death rates gradually fell as more and more of the population developed immunity to the disease.

Yellow fever epidemics arose where certain conditions prevailed: the presence of the virus, the insect vector, and a sufficiently large number of both infected and vulnerable people. As extensive commercial links were established between Philadelphia and regions of the world where yellow fever was endemic, and the susceptibility of the population increased due to constant waves of immigration and migration, the possibility of an outbreak always was present. The epidemic of 1793 was particularly devastating for the city. The virus most likely was imported from Santo Domingo, which was then experiencing a slave rebellion. It is believed that 2,000 refugees came to Philadelphia from the strife-torn island, and some, no doubt, were ill from the disease. The *A. aegypti* mosquito most likely accompanied the refugees on their journey. A hot and humid summer provided ideal conditions for the proliferation of the mosquito population, and by August the city faced an epidemic of catastrophic proportions. "It was indeed melancholy," one resident subsequently recalled, "to walk the streets, which were completely deserted, except by carts having bells attached to the horses' heads, on hearing which the dead bodies were put outside on the pavements and placed in the carts by the negroes, who conveyed their charge to the first grave yard, when they returned for another load."[130]

A significant percentage of the population fled during the outbreak; approximately half of the residents left the city, and of those that remained, between 9

and 12 percent died. Not all residents of the city were equally vulnerable when yellow fever struck, however. Adults had a much higher death rate than children, African Americans much lower rates. Poor people were at greater risk of dying, but only because they lacked the resources to flee and often were immunologically naïve newcomers to the city. The disease took its greatest toll in areas adjacent to the wharves; it diminished considerably in outlying areas and never extended beyond the city's boundaries. Although the epidemic had few lasting effects, it symbolized the vulnerability of densely populated urban communities where commercial contacts spread infectious diseases, and a growing population provided a pool of susceptible persons to sustain the infectious pathogens.[131]

Elizabeth Drinker and her family often left the city during the summer months. Early in the summer of 1793, the family moved to their summer home in Germantown. She made note in her diary of trouble in the city, and on August 16, 1793, she wrote that "John Gillenham was bury'd on second day last -'tis a sickly time now in philada. And there has been an unusual number of funerals lately here."[132] Although the family was situated away from the epidemic, it was not unusual for Henry Drinker and various members of the family to travel to and from Philadelphia on business. Because Henry Drinker was a merchant, much of his business was conducted in town. Elizabeth Drinker was well aware of the situation in the city and was fearful for the safety of her family. On August 19th she wrote, "Henry stays with us to night, 'tis seldom any one of the Family comes to stay a night with us, but they bring an account of the death of one or more of our Citeicnes, Henry

informs of the death of Richard Blackham and Peter Aston son of Peter Aston."[133] On the 21st, she notes that "8 or 10 persons bury'd out of water street, between Race and Arch Streets, many sick in our Neighborhood, and in y City generally."[134]

At this time, the Drinker town house, which was located on Front Street and Drinker's Alley below Race Street, in the heart of the epidemic area, was open and under the care of Elizabeth's sister Mary Sandwith. This was for the convenience of family members who had business in town, since the trip to Germantown required several hours of travel.[135] It is curious to note that both Elizabeth's husband and son were unwell during much of the month of August. She wrote that "my husband and son William have something of the influensia, which great numbers have at present in Town and Country."[136] Although influenza was present in Philadelphia at this time, it is certainly possible that both Henry Sr. and William had mild cases of yellow fever. Sub-clinical cases of the disease had few symptoms, and could have been easily confused with influenza. It is believed that many in the city had mild or sub-clinical cases of yellow fever, and as a result, often went undiagnosed. Whether one survived a mild or severe case of yellow fever, the result was the same: life-long immunity from the disease.

The people of Philadelphia had barely recovered from the 1793 epidemic when yellow fever struck again in 1794. The epidemic was not as severe as the previous year, with a CDR of only 31 per thousand.[137] It was during this outbreak that Elizabeth Drinker's second daughter (Nancy) came down with the disease. On

December 24th, she writes "I have been led to think, I may say to conclude, on reading Docr. Rush's acc. Of the Yallow fever, that my daughter Nancy had it towards the later end of October last, at Clearfield – and do suppose that Doc. Kuhn, who attended her, knowing that we would steadily attend her, be it what it would, kindly endeavourd to conceal it from us – he say's it was the Jaundice and some thing of the fall fever – it is possible it may be so, - but it has pleas'd kind providence to restore her, I intend at a sutable oppertunity to tell the Doctor my opinion of the matter, and I have no doubt of his candour on the occasion, - I suspected it while nurseing her by many of the symptoms, and finding many others in Dr. R.s book, seems a confirmation."[138] Why Doctor Kuhn concealed the true nature of Nancy's illness remains a mystery. Possibly his intense disagreements with Benjamin Rush over the nature and cause of yellow fever were at issue here. Doctor Kuhn insisted that Nancy's symptoms were that of jaundice, while Rush's description of yellow fever clearly indicated that Nancy had contracted the disease.

During the course of the summer and autumn of 1795 and 1796, the fever appeared sporadically in the city, but no wide-spread outbreak occurred. The disease again assumed the character of an epidemic in 1797. In a letter to Governor Thomas Mifflin of Pennsylvania, the Academy of Medicine called attention to the putrid exhalations from the gutters, streets, ponds, and marshy grounds in the vicinity of the waterfront. The disease was prevalent in the area of Pine Street Wharf near Water and Penn Streets and in the suburbs of Southwark and Kensington.[139] The CDR for

1797 was 37 per thousand,[140] indicating a moderate spike in the number of deaths in the city.

Yellow fever returned to Philadelphia in devastating form in 1798. This epidemic was considered by many contemporary observers, as well as some modern historians, as the most destructive to occur in Philadelphia between 1790 and 1800.[141] The CDR for this year was particularly high at 68 deaths per thousand.[142] Although not all deaths were directly attributed to yellow fever, the dramatic spike in the CDR indicated the presence of epidemic disease in the city. Despite the early and extensive evacuation of the city, approximately 3,500 people died in a period of four months.[143] Such a high rate of evacuation, which began during the first weeks of the epidemic, had not occurred in earlier outbreaks. While the over-all mortality was not greater than that of 1793, it was greater in proportion to the number of individuals "attacked," or those who "continued [to be] exposed to the infection."[144]

The sanitary conditions in the city had not changed significantly since the 1793 epidemic, and proponents of environmental theories of illness had a great deal to worry about in Philadelphia. Eyewitness accounts of the epidemic made particular note of numerous insects in the city. "Many tribes of insects were uncommonly numerous; as mosquitoes, ants, crickets, cockroaches…"[145] The city also lacked an efficient sewage system, and outhouses and industrial wastes continued to pollute the water supply. Noxious fumes filled the air from tanneries, distilleries, soap manufacturers and other industries. Animal carcasses lay rotting on the banks of the

Delaware River and in public streets, particularly in the market area along High Street.[146] Adding to (or resulting from) the general unhealthy state of the city was a severe outbreak of dysentery in July. Young children were particularly vulnerable to the disease and often died from it. Following closely on its heels was a return of yellow fever in August. Given that the health of many in the city may have been compromised by chronic disease, it is not surprising that yellow fever was particularly devastating that year.

City officials made efforts to check the spread of the infection. Attempts were made to quarantine ships arriving in port from May through October, but there are indications that there was considerable interaction between townspeople and those on board.[147] It also has been observed that the flight distances of the *A. aegypti* over water are considerable, and an infective vector could easily have reached the town from an infected but quarantined ship.[148] Experiments to determine the flight capabilities of *A. aegypti* indicate that a flight of more than 300 meters is not exceptional in an urban setting, and longer distances are common over water.[149] This could explain why the city's attempts to quarantine ships were largely ineffective in containing outbreaks of the disease. Although sporadic outbreaks continued to occur in the early years of the nineteenth century, the CDR steadily dropped as the population grew more and more immune to the disease. The CDR exceeded 30 deaths per thousand only twice – in 1802 and in 1805.[150] Measles and scarlet fever may have been present in the city in 1802, and this may have inflated mortality rates.[151] Elizabeth Drinker's last intimate contact with yellow fever, as far as the

Diary recounts it, came in 1803 when Sally Dawson, a favorite servant, died from the illness.[152]

The history of yellow fever in Philadelphia suggests that public fears and apprehensions bear little or no relationship to the actual impact or demographic significance of a specific disease. When mortality from common diseases was regular and predictable, there was relatively little concern; death was accepted as a part of life. When uncommon epidemics appeared at irregular intervals and resulted in mortality spikes in an already unhealthy community, public fears intensified. Disease was commonplace in Philadelphia, but the spectacular nature of yellow fever epidemics disrupted community life on a grand scale. Oddly enough, the high mortality rates associated with these epidemics had a smaller total impact on population size than did the mortality from the many diseases that were constantly present in the city.[153]

The emergence and spread of microbial threats in Philadelphia were driven by a complex set of factors, the convergence of which lead to consequences of disease much greater than any single factor might have suggested. Genetic and biological factors, for example, allowed the measles virus to adapt and change, increasing its virulence as it passed through households. Changes in the physical environment of the city positively impacted the ecology of mosquitoes by creating standing pools of water, while densely packed city streets enhanced the transmission of smallpox and measles by bringing susceptible people closer together, and ignorance of quarantine

laws pointlessly exposed residents to infection. Human behavior, both individual and collective, was perhaps the most complex factor in the emergence of disease in the city. Circulation in public (while undergoing inoculation for smallpox) was, for example, a potential way to start an epidemic. Emergence was further complicated by social, political, and economic factors including massive immigration, international trade and commerce, and poverty. Collectively, these factors ensured the continuing presence of infectious diseases in the city.[154]

The people of Philadelphia were fearful of such diseases as smallpox, yellow fever and measles if only because of their visibility and dramatic nature of symptoms. Despite the high tolls in lives, however, these diseases were by no means the most significant determinants of morbidity and mortality. Certain endemic diseases – notably dysentery, tuberculosis and malaria – took a far higher toll even though their omnipresent nature tended to reduce public fear. Although they often did not kill their victims directly, these diseases weakened them and reduced their resistance to other fatal disorders. Dysentery was widespread in Philadelphia throughout the eighteenth century, and consisted of a number of causal agents including shigella and salmonella, as well as certain disease-causing ameba; each posing a potentially serious threat to health.[155] What is important about all of them, as well as tuberculosis and malaria, is that they do not evoke life-long immunity. As a result, re-infection was probable and they could become endemic in smaller populations than were required to support measles and smallpox, which only became endemic childhood diseases at the end of the colonial period.[156] By the end of the eighteenth

century, a new equilibrium had been established in Philadelphia. The density dependent epidemic diseases declined, leaving behind a relatively stable group of endemic diseases coupled with a lower mortality rate. If disease and environment are mirror images of each other, it stands to reason that a change in one will merely reflect a change in the other. As the physical, political, cultural and economic environment of the city changed in the course of the century, so, too, did its disease environment.

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[1] See Smith, 1990, *The "Lower Sort;"* Smith, 1977, "Death and Life in a Colonial Immigrant City;" Klepp, 1994, "Seasoning and Society;" Klepp, 1989, *Philadelphia in Transition*; Klepp, 1989, "Demography in Early Philadelphia."

[2] Wealthy residents like Elizabeth Drinker and her family had available to them the best eighteenth-century medicine could provide, yet disease was constantly present in the Drinker household. The entire family experienced repeated bouts of dysentery and malaria. Several of the Drinker children died in infancy, all contracted measles, one died of diphtheria, and one lived with chronic tuberculosis. Although not all people experienced disease equally, viruses and bacteria did not distinguish between the wealthy and the poor; susceptibility to disease was the product of a much more complex array of circumstances.

[3] Cvetanović, 1982; Kunitz, 1984, p. 560.

[4] Yellow fever is another acute infectious disease which causes life-long immunity and was a significant public health threat in Philadelphia during the latter part of the century. Unlike measles and smallpox, however, it is spread by a mosquito rather than by direct person-to-person transmission. In its homeland, Africa, as well as South America, it is found in monkeys, which substantially increases both the size of the reservoir of infection and the chances for transmission. With no known animal reservoir in North America at this time, the disease existed as one of urban populations. Large influxes of new susceptibles were required in order for the disease to be maintained in the population. Unlike measles and smallpox, transmission could be interrupted by eliminating the mosquito vector.

[5] From first settlement through the end of the eighteenth century, high levels of base-line mortality from endemic diseases like dysentery and malaria had superimposed upon them epidemics caused by disease agents that require large populations in order to become endemic. Smallpox, measles and yellow fever were three of the more significant diseases in this category.

[6] Kunitz, 1984, p. 560.

[7] Cholera is an example of an acute infectious disease that does *not* invoke solid immunity and was introduced in several epidemic waves in the nineteenth century. And tuberculosis among certain American Indian tribes assumed the character of an acute, epidemic infectious disease. See Kunitz, 1984, p. 560-1.

[8] The maritime character of Philadelphia brought residents into contact not only with other colonial ports, but also with Europe, the Caribbean, and Africa. More importantly, movements of goods and people became the means of transporting a variety of pathogens capable of causing epidemic outbreaks.

[9] Bhopal, 2002, p. 22.

[10] Philadelphia's vital demographic rates were remarkable by contemporary standards: the population grew quickly, mortality levels were high, births were numerous, and successive waves of immigrants poured into and through the city. Klepp, 1989, "Demography in Early Philadelphia," p. 92. See Figure 1.

[11] Klepp, 1989, "Demography in Early Philadelphia," p. 104-5. See Table 2.

[12] Increases in population brought significant numbers of people into close living conditions. As a result, the probability of contact between an infectious agent and a susceptible host was fairly high. The merging of previously isolated disease pools through immigration brought a pattern of frequent epidemics to the city – a pattern that would only begin to moderate by the third quarter of the eighteenth century. Klepp, 1989, "Demography in Early Philadelphia," p. 94.
[13] Descriptions of colonial urban life can be found in Carl Bridenbaugh's *Cities in the Wilderness* and *Cities in Revolt*, as well as in John Duffy's *A History of Public Health in New York City*.
[14] In 1967, smallpox existed in every continent except for North America and Europe, and it was estimated that from 10 million to 15 million people contracted the disease annually. By 1972, it was gone from South America. By 1974, it was restricted to India, Ethiopia and Somalia. In October 1975, the last case of smallpox in Asia occurred, and in October 1977 in Somalia came the last case of naturally occurring smallpox in the world. The virus is now believed to exist only in deep-freeze lockers in Atlanta and Moscow, and is no longer an active infection. See Crosby, 2003, p. 303.
[15] Klepp, 1989, *Philadelphia in Transition*, p. 233.
[16] Labaree, *Papers of Benjamin Franklin*, Vol. 1, p. 200-1.
[17] Fenn, 2001, p. 83; Duffy, 1953, p. 100; Blake, 1959, p. 112.
[18] Case-fatality rates vary from nearly 100 percent in the hemorrhagic and flat types to almost zero with the modified and noneruptive types. The great majority of cases, however, are of the ordinary type, with its characteristic raised pustules. In his 1972 study of hospitalized smallpox patients in Madras, India, A.R. Rao found that 88.8 percent of cases were of the ordinary type. Rao found that case-fatality rates among its three subtypes varied markedly – 62 percent for confluent, 37 percent for semiconfluent, and 9.3 percent for discrete – with an average of 30.2 percent for all cases of ordinary type smallpox. For the types of smallpox and case-fatality rates, see Fenner et al, 1988, p. 4-40.
[19] The most severe cases of smallpox are generally hemorrhagic, flat, and ordinary confluent. See Fenner et al, 1988, p. 4-40.
[20] The serous membranes covering the placenta attract the virus. Infection during the early months of pregnancy often causes spontaneous abortion, and about half of pregnant women infected during the later part of their pregnancies lose their babies. See discussion in Barnes, 2005, p. 226. Note: serous membranes are smooth, transparent membranes containing fibrous connective tissue that line many large cavities of the body. See Rothenberg et al, 2000, p. 510.
[21] On the immunology of smallpox, see Fenner et al, 1988, p. 32, 38, 146-66; De Bevoise, 1995, p. 100.
[22] Kempe, 1975, p. 206-11.
[23] See Barnes, 2005, p. 222.
[24] The virus then multiplies within the mucous membranes of the upper respiratory tract, after which it spreads to the reticuloendothelial cells throughout the body and continues to multiply during the nine-to-fifteen-day incubation period. At the end of that time, the virus escapes into the bloodstream and infects the skin and mucous membranes. As the epidermal cells are invaded, the characteristic lesions are produced on the skin, and the virus is shed from them. At the same time, lesions form and ulceration occurs in the mouth, releasing the virus into the saliva. Note: the *reticuloendothelial system* (RES) is a unit of the body functioning in immune response to infection and in ridding the body of cellular debris. See Rothenberg et al, 2000, p. 489.
[25] See Dixon, 1962; Barnes, 2005, p. 223.
[26] Clothing and bales of cotton contaminated with smallpox scabs also played a role in the dissemination of the virus. Philadelphia was home to many tailors, and those infected with the disease had a novel way of spreading it. Both the habit of joining broken cotton fibers by moistening hands with saliva, and the presence of sloughing scabs from workers recovering from the disease, contaminated cotton and cloth. See Barnes, 2005, p. 230. Since tailors often shared cramped quarters with several other people, those who became infected with smallpox could have spread the disease to other members of their household, and to their many patrons. Laborers, mariners, cordwainers, and tailors generally lived with only four or five people in the mid-1770s, but with between six and eight persons during the 1790s. See Smith, 1990, p. 180. It is believed that the smallpox virus can survive in suspended animation within the dust of dried scabs for years, perhaps even centuries, if left away from sunlight and extreme temperatures. See Henig, 1993; Barnes, 2005, p. 222-3. If so, the virus

could have survived in a dry state for months or years in Philadelphia's warehouses and in the hulls of ships.
[27] De Bevoise, 1995, p. 101; For a discussion of transmission, see Fenner et al, 1988, p. 191; Manson-Bahr and Apted, 1982, p. 275, 278; Christie, 1974, p. 213, 248-9.
[28] In theory, the inoculated virus could be spread and could create epidemics where there were none before. This may not have occurred, however. For reasons still not certain, the virus acquired by implantation was less harmful than when acquired by the usual respiratory route. Introduction through the skin dampened its destructive powers. Most illness that resulted was limited to pustules around the inoculated site. Signs of serious systemic spread were infrequent, and the quantity of virus present in the respiratory tract available to be dispersed by cough or sneeze was most likely low. As a result, the fear that inoculation could propagate or create new epidemics was probably overdrawn. See Gehlbach, 2005, p. 33.
[29] Abigail Adams to John Adams, August 5, 1776. See Butterfield et al, 2002, p. 150-1.
[30] Crane, 1991, p. 98.
[31] Blake, 1959, chapter 2; Silverman, 1984, p. 339-40; Labaree, *Papers of Benjamin Franklin*, Vol. 4, p. 341-2; Toner, 1865, p. 163-82; Wolman, 1978, p. 338-47; Crane, 1991, p. 32.
[32] Wolman, 1978, p. 342.
[33] Medical practitioners had long urged the public to adopt inoculation, and affluent Americans seeking immunity flocked to Philadelphia from other locales where the procedure came under closer regulation. Thomas Jefferson, for example, underwent inoculation in Philadelphia in 1766, and ten years later, George Washington's wife, Martha, did the same, even as her husband grappled with the news of the smallpox's ravages to his Northern Army. See Fenn, 2001, p. 83.
[34] Fenn, 2001, p. 83.
[35] Fenn, 2001, p. 84-5.
[36] Grob, 2002, p. 73-4.
[37] The southern colonies had neither the population density nor the transportation networks needed to sustain the ongoing, endemic presence of the virus. As a result, years could pass between outbreaks, allowing the number of susceptible individuals to increase.
[38] Fenn, 2001, p. 86; For Washington's frustration , see GW to William Shippen, Jr., Morristown, January 28, 1777, in *Writings of George Washington*, Vol. 7, p. 75-6.
[39] Crane, 1991, p. 36. Excerpt from the *Diary of Elizabeth Drinker*, October 24, 1759.
[40] *Cowpox* is a mild disease characterized by a pustular rash and caused by vaccinia virus transmitted to humans from infected cattle. Cowpox infection confers immunity to smallpox, which is caused by a similar virus. See Rothenberg et al, 2000, p. 141.
[41] Rothenberg et al, 2000, p. 290.
[42] Hopkins, 1983, p. 41; Crane, 1991, p. 36.
[43] De Bevoise, 1995, p. 102; On herd immunity, see Susser, 1973, p. 61-2; Bhopal, 2002, p. 2.
[44] Grob, 2002, p. 73; Carmichael and Silverstein, 1987, p. 147-68; Duffy, 1953, p. 21-2; For a chronology of epidemic disease and mortality in seventeenth- and eighteenth-century England, see Dobson, 1997, p. 383-449.
[45] Wolman, 1974, p. 61; Jarcho, 1956, p. 195-212.
[46] Wolman, 1974, p. 62-3.
[47] Duffy, 1953, p. 78.
[48] Klepp, 1989,"Demography in Early Philadelphia," p. 104.
[49] *Pennsylvania Gazette*, July 8, 1731; Wolman, 1974, p. 70.
[50] The Christ Church Bills of Mortality for 1737 list 61 deaths from smallpox. See Klepp, 1991, p. 61.
[51] Wolman, 1978, p. 340.
[52] The Christ Church Bills of Mortality for 1759 list 106 deaths from smallpox, with "3 only inoculated." See Klepp, 1991, p. 67.
[53] Toner, 1865; Wolman, 1978; Hopkins, 1983, p. 257; Benjamin Franklin to John Perkins, August 13, 1752, in Labaree, *Papers of Benjamin Franklin*, Vol. 4, p 340-1; Crane, 1991.
[54] Klepp, 1989, *Philadelphia in Transition*, p. 233. See Table 4.1.
[55] Smith, 1990, p. 48.
[56] Scientists are now saying that smallpox vaccine may not be fully protective throughout life. This issue has come up in light of recent bioterrorism threats.

[57] For lifelong protection, see Manson-Bahr and Apted, 1982, p. 276.
[58] The Christ Church Bills of Mortality list 47 deaths in 1751, 112 deaths in 1756, 106 deaths in 1759, 30 deaths in 1762, 56 deaths in 1763, 57 deaths in 1765, 39 deaths in 1769 and 44 deaths in 1773. See Klepp, 1991, p. 47, 65, 67-70, 73, 75.
[59] Drinker, 1937, p. 91.
[60] Wolman, 1974, p. 168-9.
[61] An *anthelminthic* is an agent that expels worms from the intestinal canal, because of the roughness of the drug particles, its cathartic action, or, in some cases, because of a specific toxic effect on the worms themselves. See Estes, 1990, p. 12 for definition of anthelminthic; p. 34 for definition of calomel.
[62] Crane, 1991, p. 125. Excerpt from the *Diary of Elizabeth Drinker*, December 16, 1765.
[63] *Pennsylvania Gazette*, June-September 1732; Wolman, 1974, p. 102.
[64] Winship, 1987, p. 67-90.
[65]Crane, 1991, p. 98. Excerpt from the *Diary of Elizabeth Drinker*, December 2, 1762.
[66] Estes, 1990, p. 14, 55; Estes, 1980, p. 365, 371, 378.
[67] Smith, 1990, p. 50.
[68] Kim-Farley, 2003, p. 212.
[69] Grob, 2002, p. 32.
[70] Grob, 2002, p. 78.
[71] Wolman, 1974, p. 217.
[72] Caulfield, 1943, p. 531-8.
[73] The Christ Church Bills of Mortality list 23 deaths from measles in 1747, 22 deaths in 1759 and 17 deaths in 1772. See Klepp, 1991, p. 62, 67, 75.
[74] Duffy, 1953, p. 174; Sparks, 1839-47, Vol. 7, p. 41.
[75] The Christ Church Bills of Mortality for 1747 list 23 deaths from measles and 4 deaths from pleurisy. There were 39 deaths from pleurisy recorded the following year. See Klepp, 1991, p. 62.
[76] Kim-Farley, 2003, p. 212.
[77] Population data is taken from Klepp, 1989, "Demography in Early Philadelphia," p. 105. Deaths are from the surviving Philadelphia bills of mortality originally published in newspapers, almanacs, and broadsides by Philadelphia publishers, churches, and the Board of Health. These bills are reproduced and analyzed in Klepp, 1991, *"The Swift Progress of Population."*
[78] The Christ Church Bills of Mortality alone list 106 deaths from smallpox, 22 from measles, and 9 from "nervous fever" (typhus or typhoid). See Klepp, 1991, p. 67. Nervous fever was generally associated with typhus, after William Cullen, an eighteenth-century British physician famed for his classification of diseases, so described it. The symptoms included a quick, low pulse, chilliness and flushing in turn, giddiness and pain in the head, nausea, and vomiting. There were, however, many descriptions of nervous fevers, not all of which were typhus, and there were also illnesses that presented certain nervous symptoms but were not considered the nervous fever. See Cullen, 1791, Vol. I, p. 109-15; Rush, 1815, "Outlines of the Phenomena of Fever," Vol. III, p. 3-36; Crane, 1991, p. 21. Note: e*pidemic typhus* is an acute rickettsial disease transmitted by lice. Characteristic symptoms include fever, prostration, aches, and a widespread rash covering trunks and limbs. See Harden, 2003, p. 352.
[79] Grob, 2002, p. 78.
[80] On malnutrition and infection, see Newberne and Williams, 1970, p. 93.
[81] This paragraph is based on Francis L. Black, "Why Did They Die?," 1992, p. 1739-40 and "An Explanation of High Death Rates among New World Peoples When in Contact with Old World Diseases," 1994, p. 292-307. Peter Aaby's work can be followed in Aaby et al, "Overcrowding and Intensive Exposure as Determinants of Measles Mortality," 1984, p. 49-63; Aaby, "Malnutrition Overcrowding/Intensive Exposure in Severe Measles Infection: Review of Community Studies," 1988, p. 478-91 and "Determinants of Measles Mortality: Host or Transmission Factors?,"1991, p. 83-116.
[82] According to Susan E. Klepp's study of racial differences in mortality in Philadelphia, five known outbreaks of measles produced the highest average black death rates in the city between 1722 and 1775. The mean CDR for blacks during measles epidemics was 106, nearly double the white rate of 56. See Klepp, 1994, p. 506.

[83] Elizabeth Drinker mentions outbreaks of measles in 1759, 1772, 1778, 1783, 1795 and 1796. Benjamin Rush discusses an outbreak in 1789. See Rush, 1815, *An Account of the Measles, as they appeared in Philadelphia in the Spring of 1789*, Vol. II, p. 255-61.
[84] Crane, 1991, p. 180. Excerpts from the *Diary of Elizabeth Drinker*, October 2, 1772, October 15, 1772, and October 18, 1772.
[85] It is clear from reading her diary that Elizabeth Drinker's immediate concern on any given day was status of her family's health. Constant inquiries and reports containing the most intimate details fill the journal pages, and it is curious that family members, separated by no more than a dozen blocks in any direction, felt impelled to inquire (and in Drinker's case, record) about the progression of each headache, toothache, sore throat and cough. From a modern perspective, these short-term afflictions are presumably of no consequence. In the eighteenth century, however, they could portend a life-threatening situation. She mentions measles on more than thirty occasions in her diary, and was well aware of the seriousness of the disease. See Crane, 1991, p. xxx.
[86] Caufield, 1943, p. 548. These bills are reproduced and analyzed in Klepp, 1991, *"The Swift Progress of Population,"* p. 81-3.
[87] Cooper and Kiple, 2003, p. 365.
[88] Ryan and Ray, 2004, p. 588.
[89] Cooper and Kiple, 2003, p. 365.
[90] Kiple and Higgins, 1992, p. 239-40.
[91] Three important characteristics distinguish the nature of migration during the 1790s from that of the previous four decades. First, the Irish and Scotch Irish dominated among overseas immigrants during the last dozen years of the century. Like earlier immigrants, they came primarily from the poor and middling ranks of society. Second, a large group of French settled in Philadelphia during the 1790s, refugees from revolutions in their own country and in the West Indies. Third, a substantial proportion of the migrants during the last decade of the century arrived from the nearby countryside. In particular, hundreds of slaves who had acquired their freedom during the Revolution flocked to the city in search of economic opportunities. See Smith, 1990, p. 60-1.
[92] The influx of the French and the Irish is evident in the growth of their interments in the city's Catholic cemeteries, as recorded in the Bills of Mortality. Of the immigrants arriving in Philadelphia between 1789 and 1793, 53 percent were from Ireland and 27 percent from France or the French West Indies, as cataloged in Health Officer's Register. The economic characteristics of the French refugees are discussed in Powell, *Bring Out Your Dead*, 1949, p. 6-7.
[93] *Transient viremia* is a feature of many infections in hosts other than their reservoir; those affected, including humans and higher vertebrates are often referred to as blind-end hosts. In contrast, if viremia is sustained for longer periods of time, the vertebrate host becomes highly important as a reservoir for continuing transmission. Viremia may last a week or more in human dengue and yellow fever infections, and humans may then serve as a reservoir in urban disease. See Ryan and Ray, 2004, p. 587-8.
[94] Smith, 1990, p. 60-1.
[95] One particularly ambitious analysis is James Goodyear's study of yellow fever and its relation to the sugar trade. He argues persuasively that an important factor in the nurturance and travel patterns of the yellow fever mosquito was the paraphernalia of sugar syrup refining and transport. The need for slave labor in the newly established sugar cane, molasses and rum economy of the Caribbean region undoubtedly promoted movement of both vector and virus. See Goodyear, 1978, p. 5-21.
[96] Mullen and Durden, 2002, p. 219.
[97] Kiple and Higgins, 1992, p. 241.
[98] LaRoche, 1855, Vol. I, p. 62.
[99] Vogt, 1908, p. 7.
[100] Cooper and Kiple, 2003, p. 365.
[101] Service, 2004, p. 58.
[102] Mullen and Durden, 2002, p. 219-20.
[103] Mullen and Durden, 2002, p. 220.
[104] One does not need to be outdoors to be bitten by the *A. aegypti* mosquito since it readily enters and typically rests in houses. See Mullen and Darden, 2002, p. 229. More to the point, there were no window screens in eighteenth-century Philadelphia.

[105] Letter to Nicolas Chervin regarding yellow fever, May 22, 1821. Nicolas Chervin Collection, College of Physicians of Philadelphia, p. 83-6.
[106] Klepp, 1995, p. 221.
[107] Cooper and Kiple, 2003, p. 365.
[108] Ryan and Ray, 2004, p. 587.
[109] The term is taken from *ar*thropod-*bo*rne *virus* This is a virus that multiplies in a blood -sucking arthropod and is principally transmitted by the bite of arthropods to vertebrate hosts, e.g. yellow fever virus. See Service, 2004, p. 261.
[110] Ryan and Ray, 2004, p. 588.
[111] Service, 2004, p. 67; WHO, 1986, *Prevention and Control of Yellow Fever in Africa.*
[112] Service, 2004, p. 67.
[113] See McNeill, 1999, p. 177.
[114] Klepp, 1989, "Demography in Early Philadelphia," p. 103.
[115] Letter from James Pemberton to Doctor Casper Wistar, Jr., October 10, 1802. See *Additional Facts and Observations Relative to the Nature and Origins of the Pestilential Fever*, 1806 (7). In 1806, the College of Physicians of Philadelphia published this pamphlet summarizing the results of its inquiries as to the nature and history of yellow fever in Philadelphia. The investigation was made because of the repetitive and virulent epidemics of the preceding few years; specifically, 1793, 1797, 1798 and 1799; Taken from Wolman, 1974, p. 172.
[116] See Hazard, *Register of Pennsylvania*, 1828-1835, Vol. 9, p. 240.
[117] LaRoche, 1855, p. 56; Currie, 1800, p. 39-40.
[118] LaRoche, 1855, p. 58; Currie, 1800, p. 43.
[119] Some historians have been interested in applying current knowledge to past epidemics in an attempt to sort out "what really happened." They seek to elucidate what environmental, social, epidemiological, or economic factors favored the appearance and spread of yellow fever. David Geggus, for example, has analyzed the yellow fever epidemics in Santo Domingo in the 1790s with the ambition of understanding the severity and mortality of those especially epidemic years in the Caribbean. He finds his answers in the societal havoc of war, excessive movements of people from one island to another, and the presence of large numbers of susceptible British troops. See Geggus, 1979, p. 38-58.
[120] Geggus, 1979, p. 41.
[121] CDR is from Klepp, 1989, "Demography in Early Philadelphia," p. 104. The Christ Church Bills of Mortality for 1747 list 23 deaths from measles and 42 deaths from fever. The Christ Church Bills of Mortality for 1748 list 39 deaths from pleurisy and 33 deaths from fever. See Klepp, 1991, p. 62.
[122] Dock Creek continued to be a stagnant sludge of refuse in the heart of the center of the city. Not surprisingly, mortality during yellow fever outbreaks was extremely high among people who lived near the creek where mosquitoes thrived. See Smith, 1997, p. 158.
[123] Crane, 1991, p. 96. Excerpt from the *Diary of Elizabeth Drinker*, September 1762.
[124] Klepp, 1989, "Demography in Early Philadelphia," p. 105. The Christ Church Bills of Mortality for 1762 list 13 deaths from consumption (tuberculosis), 26 deaths from decay (tuberculosis), 20 deaths from fits, 16 deaths from fever, 9 deaths from nervous fever (typhus or typhoid), 22 deaths from purging and vomiting, 30 deaths from smallpox and 16 deaths from yellow fever. See Klepp, 1991, p. 68. In addition to epidemic smallpox, yellow fever and nervous fever (typhus or typhoid), chronic diseases like tuberculosis and dysentery added significantly to the mortality rate.
[125] Redman, 1793, p. 13.
[126] LaRoche, 1855, p. 61.
[127] Blake, 1968, p. 675-6.
[128] Geggus, 1979, p. 38-53.
[129] Klepp, 1997, p. 164-5.
[130] The theme of isolation runs through many of the fever narratives. Many years after the epidemic, Robert Simpson recalled this scene. See Simpson, 1949, p. 50 and Simpson, *Letterbook*, 1788-1807.
[131] Data on the epidemic of 1793 can be found in Estes and Smith, *A Melancholy Scene of Devastation*, Powell, *Bring Out Your Dead*, Pernick, "Politics, Parties and Pestilence," and Kiple and Kiple, "Black Yellow Fever Immunities."
[132] Crane, 1991, p. 494. Excerpt from the *Diary of Elizabeth Drinker*, August 19, 1793.

[133] Crane, 1991, p. 494. Excerpt from the *Diary of Elizabeth Drinker*, August 19, 1793.
[134] Crane, 1991, p. 495. Excerpt from the *Diary of Elizabeth Drinker*, August 21, 1793.
[135] Drinker, 1937, p. 114.
[136]Crane, 1991, p. 495. Excerpt from the *Diary of Elizabeth Drinker*, August 23, 1793.
[137] Klepp, 1989, "Demography in Early Philadelphia," p. 105.
[138] Crane, 1991, p. 633. Excerpt from the *Diary of Elizabeth Drinker*, December 24, 1794; The "book" is Benjamin Rush's *An Account of the Bilious Remitting Yellow Fever, as it Appeared in the City of Philadelphia, in the Year 1793*.
[139] Mansfield, 1949, p. 63-4.
[140] Klepp, 1989, "Demography in Early Philadelphia," p. 105.
[141] DeClue and Smith, 1998, p. 243-4; Middleton, 1964, p. 497-515; Pascalis-Ouvière, 1798.
[142] Klepp, 1989, "Demography in Early Philadelphia," p. 105.
[143] Shannon and Cromley, 1982, p. 355.
[144] LaRoche, 1855, p. 85.
[145] Condie and Folwell, 1798, p. 13.
[146] Foster, Jenkins, and Toogood, 1998, p. 89.
[147] Condie and Folwell, 1798, p. 31.
[148] Taylor, 1951, p. 442.
[149] Shannon, Burke, and Davis, 1930, p. 151-6.
[150] Klepp, 1989, "Demography in Early Philadelphia," p. 106.
[151] Mansfield, 1949, p. 100. The Christ Church Bills of Mortality for 1802 list 11 deaths from measles, 9 deaths from scarlet fever, 11 deaths from consumption (tuberculosis) and 24 deaths from decay (tuberculosis). See Klepp, 1991, p. 86.
[152] Crane, 1991, p. 1687-91. As members of the Drinker family, servants received much the same medical treatment as everyone else in the household; treatment that was either advantageous or not, depending on one's assessment of eighteenth-century medicine. A favorable recommendation from Elizabeth Drinker permitted a servant, former servant, or relative of a servant to obtain free medication from the Philadelphia dispensary, thus extending Drinker's influence over the health of her extended family and reinforcing the deferential nature of eighteenth-century society.
[153] The Christ Church Bills of Mortality consistently list numerous deaths from consumption, decay, fever, fits, flux, purging and vomiting.
[154] See Smolinski, Hamburg and Lederberg, 2003, "Executive Summary," p. 2.
[155] *Dysentery* is an inflammation of the large intestine characterized by loose stools containing blood and mucus. Diarrhea, marked by frequent production of watery stools, may be confused with dysentery in historical accounts, but references to "bloody flux" refer to true dysentery. The condition may be caused by an ameba, *Entamoeba histolytica*, or by several species of bacteria, including the genera *Shigella* and *Salmonella*. See Patterson, 2003, p. 105, 43.
[156] Kunitz, 1984, p. 564; Duffy, 1953, p. 178.

# CHAPTER 6

# DYSENTERY, MALARIA, TUBERCULOSIS AND TYPHOID FEVER: OMNIPRESENT MENACES

Many elements of the physical environment collectively influence the host directly, determine the survival of agents that exist outside the host, and mediate the transmission of agents between hosts, including the movement from animal to human hosts. Viewed in this light, the physical environment takes on considerable importance in determining the epidemiology of infectious disease.[1] The interactions among vectors, animal reservoirs, microbes, and humans presented many opportunities for changes in the physical environment to influence the transmission of infectious disease in Philadelphia. Many of the factors that affected the abundance, survival, activity, or feeding behavior of vectors (mosquitoes, flies, fleas and lice) also affected the reproduction, survival, and abundance of animal reservoirs. For example, elevated rainfall and clearing of the forests for firewood created new breeding habits for mosquitoes, leading to an increase in mosquito population density. The presence of the *Anopheles* mosquito allowed for the seasonal presence of malaria both in the city and the suburbs. Increased levels of precipitation also led to the overflow of sewers, adding to the already profuse amount of wastes in the streets. Likewise, these same factors affected human behavior and exposure to infection by

impacting outdoor activities, housing, and the quality and quantity of food and water. Streets littered with debris and accumulations of stagnant water, for example, enhanced the proliferation of insects and vermin. Though most of the houses were built of brick or wood, their cellar floors were usually of dirt – a favorable environment for rodents and their fleas.[2]

> The weather, during the first three weeks of the month of May, was dry and temperate, with now and then a cold day and night. The strawberries were ripe on the 15th, and cherries on the 22nd, day of the month, and in several city gardens. A shower of hail fell in the afternoon of the 22nd, which broke the glass windows of many houses. A single stone of this hail was found to weigh two drachms. Several people collected a quantity of it, and preserved it till the next day in their cellars, when they used it for the purpose of cooling their wine.
>
> The weather, after this hail storm, was rainy during the remaining part of the month. The diseases were still inflammatory. Many people were afflicted with a sore mouth in this month. The weather in June was pleasant and temperate. Several intermittents [fevers] and two very acute pleurisies, occurred in my practice during this month. The intermittents were uncommonly obstinate, and would not yield to the largest doses of bark (Benjamin Rush, 1794).[3]

These were the words of Benjamin Rush as he began his observations on diseases that beset Philadelphia in 1794. As he did during the yellow fever epidemic of 1793, Rush took great care to observe and catalog weather conditions. His intention was to learn more about disease patterns from clues provided by meteorological factors. He was convinced that environmental factors played a pivotal role in the emergence of infectious disease – and he was correct. He proclaimed hot, humid weather was unhealthy, noted the summer was the time when fluxes (diarrhea) and fevers (malaria and typhoid fever) were rampant, and he praised the cold rains

that occurred in mid-October of 1793 as salubrious because they cleansed the air of miasma.[4]

The convergence of any number of factors can create an environment in which infectious diseases can emerge and become rooted in a community.[5] Although population density was essential for the propagation of smallpox, measles and yellow fever in Philadelphia, it played much less of a role in the epidemiology of chronic infectious diseases such as dysentery, malaria, tuberculosis and typhoid fever. These diseases were influenced more by environmental factors such as climate and weather,[6] contaminated water, poor hygiene, migration, crowded houses and changing ecosystems.[7] While dysentery and malaria may have been particularly widespread in Philadelphia, tuberculosis and typhoid fever added considerably to the city's mortality rates.[8] All had a constant presence, with periods marked by intense outbreaks. Together, they lowered the population's ability to cope with its environment.

While most urban centers were unhealthy to some degree, the situation in eighteenth-century Philadelphia had become particularly dire. Environmentally, the city had reached an impasse by the middle years of the century. It had invested heavily in infrastructure, paving roads and lighting streets, but was still struggling to deal with the wastes accumulating throughout the city. Much of this filth was either covered over in some manner or simply ignored. Privies were a particular problem as they were rarely cleaned and often overflowing. The sights and smells of human

wastes, backyard debris, and basement pits were never far from view. Adding to this was the fact that the city had become a *focus of infection*[9] for a variety of illnesses. As such, it contained the epidemiologic factors needed for the transmission of disease: a susceptible group of people, numerous pathogens and parasites, plenty of insects to spread disease, and a crowded, filthy, hot and humid environment.

High death rates from endemic diseases were common in the city. Once introduced into a community, dysentery, tuberculosis, malaria and typhoid fever could be maintained by relatively fewer individuals, since they did not produce solid immunity after infection and did not depend upon a densely populated community in order to spread. Consequently, individuals likely were re-infected several times or maintained the same infection for many years. Although often overshadowed by the extraordinary nature of periodic smallpox and yellow fever outbreaks, these diseases played a far more significant role in shaping morbidity and mortality patterns in Philadelphia.[10]

## Dysentery

Broadly defined, dysentery is an inflammation of the large intestine characterized by loose stools containing blood and mucus, and by painful and unproductive attempts to defecate. Diarrhea, marked by frequent production of watery stools, may be confused with dysentery in historical accounts, but references to "bloody flux" generally referred to true dysentery.[11] It is usually spread through

ingestion of food and direct contact with items contaminated by excrement of infected individuals, with flies and mosquitoes often serving as mechanical vectors. Dysentery has multiple causes and symptoms, was sometimes fatal, always debilitating, and respected neither class, gender, nor age. It did, however, pose a serious health threat to infants and young children. Outbreaks were common in all of the colonies, but densely populated towns were particularly vulnerable. Yearly epidemics were not uncommon in Philadelphia, and rarely was there as much as a five-year interval between them. Lacking knowledge about the causes of dysentery, colonial Americans were unable to undertake effective preventative measures. Infected individuals and healthy carriers transmitted pathogens, crude methods of disposing of organic wastes contaminated drinking water, absence of refrigeration permitted the growth of bacteria in food supplies, and existing hygienic standards greatly enhanced the risk of exposure. It tended to peak in warmer months when high temperatures and humidity provided greater opportunity for rapid pathogen proliferation. Infants and young children were particularly vulnerable to infection, and often died from dehydration. The prevailing system of health care actually magnified the dangers posed by gastrointestinal disorders through lack of understanding that dehydration could result in death. Data reveal that during an epidemic, perhaps half of the community's population would become infected, and of these, one of every six or seven would die. Mortality rates during such an outbreak ranged between 5 and 10 percent, to say nothing about the large number of individuals affected during non-epidemic years. Dysentery contributed to over all

mortality in indirect ways as well, by weakening individuals and leaving them vulnerable to other infections.[12]

Intestinal disorders were widespread in the city at this time, and were caused by a variety of pathogens. Lacking the tools of modern bacteriology, Philadelphia physicians relied largely on description when identifying an illness. These included such terms as "cholera infantum," "bloody flux," vomiting, diarrhea, dysentery, teething, and worms. The assorted diagnoses of intestinal disorders had several features in common, however. Frequent and watery stools, general fatigue, and rapid weight loss were some of the more common. Whatever the specific cause, the typical mode of communication was by water or food that had been contaminated by inappropriate hygiene or spoiled from lack of refrigeration. Elizabeth Drinker writes on June 17, 1773 "Sally very unwell in the Night with a lax and Vomiting, she had eat to much Fruit…"[13] It is very likely that the fruit consumed by the child was contaminated by organic wastes. The preparation and storage of food in such environments also posed serious problems, as did the bacterial transmission from feces and flies. The circumstances of urban life, particularly crowding and inadequate sanitation that led to high mortality from other infectious diseases, played an equally significant role in enteric disorders. Given that dehydration was often a consequence of severe diarrhea, and the risks associated with this were not clearly understood at the time, it was inevitable that intestinal disorders became a leading cause of death in Philadelphia.

The descriptive terms "vomiting" and "dysentery" were implicated overwhelmingly in the deaths of children from the age of about ten months though the second birthday.[14] Contemporary opinion blamed the weaning process itself for the large number of deaths in late infancy, and there were a number of superstitious practices designed to ease the transition from breast feeding to solid foods. It was most likely the contaminated milk, food and the germ-laden water from Philadelphia's wells that most affected these children. Dr. William Currie admitted in 1792 that Philadelphia water was "very disagreeable to the palates of most people who reside at a distance from the city, and is sometimes offensive to their stomachs and bowels. But custom had rendered it both agreeable and wholesome to the inhabitants."[15] Water offensive to adults could be fatal to children, however. Those children who survived their early childhood faced a much smaller chance of death in later childhood and in adolescence.[16]

Dysentery can be caused by a variety of bacterial, viral, and parasitic agents. Bloody stools generally have bacterial and occasionally amebic origins, but almost never have a viral cause. Waterborne epidemics of amebic dysentery[17] are not as frequent as those of bacillary dysentery[18] in temperate climates, but the former do occur when sewage contaminates wells – a constant problem in Philadelphia. The high water-table under the city meant that the waste from privies constantly seeped into hundreds of private wells which supplied the population with water. In March and July of 1773, Elizabeth Drinker speaks of walking to John Lawrence's pump, which was about a half mile from the Drinker house.[19] The popularity of this well

may have been the result of the "medicinal properties" attributed to it by Benjamin Rush. He described these "medicinal properties" in a paper he read before the American Philosophical Society on June 18, 1773.[20] In actuality, the well was contaminated by a neighboring "necessary." It was this contamination that gave the water its "strong ferruginous taste." Intestinal complaints were so common in the Drinker household that an almost constant reference to them is made throughout the Diary.

Benjamin Rush indicated in his 1789 description of the "bloody flux," that this disease, also known as cholera infantum,[21] "prevailes in most large towns of the United States." Infants and children under two years old were particularly vulnerable. The Philadelphia outbreaks typically lasted from mid-June to mid-September with "frequency and danger always in proportion to the heat of the weather." According to Rush, the initial stage was often gradual "but it more frequently comes on with a violent purging and vomiting with a high fever…the stools are sometimes slimy and bloody." He also noted that worms were frequently discharged with the stools. The children, in a feverish state, often became delirious. Death, preceded by convulsions, could result in two days or after a prolonged illness of 6 weeks to 2 months. He strongly advocated cleanliness, daily cold baths, fresh country air, and salted meat and wine for such victims.[22]

The climate of Philadelphia was such that outbreaks of dysentery were most likely of bacterial origin. Amebic dysentery occurs mainly in the tropics, has a slow

and insidious onset, and lasts for years with spontaneous improvements and relapses. The bacillary form, on the other hand, is prevalent in hot weather and has an explosive onset of symptoms. Outbreaks occur when hygienic conditions are unsanitary, with the bacilli being spread by personal contact, contaminated food, and polluted water; all of which were commonplace in Philadelphia. To further complicate the situation, the bacteria can live in the intestines of apparently recovered patents, and give rise to sudden flare-ups when host immunity is otherwise compromised.[23] Benjamin Rush commented that although sporadic cases of dysentery were common, he had never seen an epidemic of that disease in Philadelphia between 1760 and 1766.[24] Although epidemic outbreaks of dysentery may not have been common during these years, the endemic nature of the disease made it a constant threat to public health in the city.

High death rates among Philadelphia's infants were the result of a whole host of circumstances including inadequate maternal nutrition, poverty and even neglect. One of the most likely factors, however, was the general lack of understanding that mortality from enteric disorders could be limited by preventative measures to minimize contamination of water, milk, and food and by ensuring re-hydration during acute episodes. The synergy that exists between malnutrition and diarrhea may have also aggravated an already compromised situation. A bout of diarrhea, for example, can decrease caloric intake between 20 and 60 percent, while malnutrition can contribute to more severe or prolonged diarrhea.[25] The use of cow's milk exposed infants and toddlers to a variety of bacterial and other agents of dysentery and

diarrhea. Because milk is an excellent growth medium for *Shigella*[26] and many other pathogens, contaminated milk and lack of refrigeration contributed to the especially high death rates in hot weather.[27] Milk-borne shigellosis may well have been a significant contributor to the "summer complaint," which took many young lives in Philadelphia. Those elements traditionally associated with urban environments certainly played a role, especially crowding, lack of sanitation, and poverty. Inadequacies in the prevailing systems of infant care, however, cannot be overlooked. Many infants perished because parents lacked the knowledge to deal effectively with intestinal disorders.[28]

Although the people of Philadelphia did not know the true causes of their intestinal disorders, they treated them nonetheless. The City's newspapers and almanacs, both English and German, carried frequent advertisements and testimonials on the treatment of "fluxes." John Bartram, for example, proposed "the true Indian Physic" or ipecacuanha for treatment.[29] Since ipecac has been used in the treatment of amebic dysentery, the continued use of this drug in a temperate climate suggests that amebic dysentery may have been present in Philadelphia; the parasites were, perhaps, carried into the city by slaves from both Africa and the Caribbean.[30] The most popular nostrums, however, were *Bateman's Pectoral Drops* and *Godfrey's Cordial.* Each of these had opium as a main ingredient.[31] Antimony mixtures, clarified butter, chamomile, rosemary, Bole armenic and mixtures of bark and rum were also used.[32] Germans resorted to such herbs as Jobs Tears, Cinquefoil, Speedwell, Shepherd's Purse, Pepper Grass, Slippery Elm, Flaxseed, Catnip and

Cranesbill, all brewed as teas for use against dysentery. On occasion, they drank mutton fat or huckleberries mixed with wine.[33] Whether these remedies alleviated or aggravated symptoms is not certain. Modern treatment of bacterial dysentery includes enteric precautions, low-residue diet and, most important, replacement of fluids and electrolytes. Antibiotics are of questionable value but may be used in an attempt to eliminate the microbe and therefore prevent further spread of the disease.[34]

Outbreaks of dysentery were especially common in port communities like Philadelphia, Boston, New York and Charleston. These cities were the entry points for ships that brought thousands of immigrants to the colonies. Conditions aboard ships were particularly conducive to outbreaks of the disease, and it was relatively easy for infected immigrants to serve as *reservoirs of infection* upon their arrival. A reservoir of infection can be any person, animal, insect, plant, soil, or substance, or a combination of these, in which an infectious agent normally lives and multiplies, on which it depends primarily for survival, and where it reproduces itself in such a manner that it can be transmitted to a susceptible host.[35] Under such conditions, it is not surprising that intestinal disorders took such a high toll among passengers during the voyage and after their arrival in the colonies.

Infections caused by a variety of parasitic worms, including flatworms, tapeworms, and roundworms were also quite common in Philadelphia, and further complicated the plethora of intestinal disorders. Ascariasis, also known as roundworm infection, was one particular variety, and was caused by the parasitic

worm *Ascaris lumbricoides*; a large roundworm resembling an earthworm. It is transmitted to humans by feces-contaminated water or food. Mild intestinal ascariasis may cause only vague stomach discomfort. The first clue may be vomiting a worm or passing one or more in the stool. Severe disease, however, causes stomach pain, vomiting, restlessness, disturbed sleep, and, in extreme cases, intestinal obstruction.[36] These giant intestinal worms can be 6-14 inches long, and they typically reside in the lumen of the small intestine. If vomited into the oral cavity, they may exit from the host's mouth or nostrils. Female worms produce up to 200,000 fertilized eggs daily, which are passed in the feces. Eggs incubate in soil for at least 2-3 weeks to produce an infective larval stage. The eggs are resistant to chemical, desiccation, and extreme temperatures, but mature or "embryonate" most rapidly in warm, moist, shady conditions in clay soils.[37] Ascariasis never passes directly from person to person.

People became infected by eating embryonated eggs in contaminated food or water, while toddlers often were infected by direct ingestion of eggs with dirt. Philadelphia's poor sanitation practices obviously favored transmission, and worm infestation was a common, but serious, ailment among the children of the city. There are over two dozen references to worms in Elizabeth Drinker's diary. Intestinal complaints were an almost daily occurrence in the Drinker household, and Drinker's children and grandchildren were often infested with a variety of intestinal worms. On August 16, 1765, Drinker writes "Nancy [age 1] very unwell to Day. Found several little worms in her Clout about the Eighth of an inch long…" On July 29, 1772 she writes My little Henry [age 2] has voided nine worms this Day. 20 since he came

home from Nurse. He has taken the Caro. Pink-Root;[38] Rheubarb, and Bark for disordered Bowels." On August 20 or 21, 1777 she writes "our dear little Henry [age 7] was taken ill with vomiting and disordered Bowels…he voided in the course of his Sickness, (which turnd out to be an inviterate Bloody and white Flux) 3 large Worms, and vomited one alive – for 12 Days he eat nothing- and is now Sepr. the 6 in a very poor way, reduced almost to a Skelaton with a constant fever hanging about him…"[39]

A variety of social, cultural and environmental factors facilitated the spread of diarrheal diseases in Philadelphia, especially among infants and young children. Dominating the landscape of the city were open sewers, stagnant mill ponds, contaminated wells, flooded clay pits, and sinkholes. Along the riverfront, ships dumped their daily accumulation of excreta into the Delaware, while the wharves cut the flow of the river'scurrent . Waste material was allowed to accumulate in the mud, where flies carried microbes to nearby dwellings. The absence of enforced regulations permitted basement cesspools to be emptied into the streets and dead animals and garbage to be hurled into sinkholes originally dug to receive gutter runoff. The cumulative effect was to aggravate the already seriously inadequate waste disposal system, which, in turn exacerbated the spread of enteric disease.

The sanitary conditions in Philadelphia were poor by most contemporary standards, and interest in public sanitation became politically important in the 1760s as voters and taxpayers demanded that the city provide a cleaner environment. Street paving received renewed interest at this time, while installations of sewers and gutters

helped to improve over-all cleanliness in the city. Garbage collection began in the late 1789s when the city hired scavengers to clear the streets. It was also at this time that laws requiring individuals to maintain their own property began to be enforced. The city also started to regulate butchers and tanners whose offal was most offensive to those living in the immediate vicinity. It was not until 1786, however, that the most noisome open sewer, Dock Creek, was covered.[40] Although these improvements were a "good faith" attempt to curtail the emergence and spread of infectious disease, they had little effect against enteric disorders.[41] William Currie wrote in 1792 that "some one or more contagious disease, being always more or less prevalent in the city, is one reason why a greater proportion of children die annually in the city than in the country."[42] Isolation was the best defense against disease, but the urban environment of Philadelphia was such that the probability of contact between infectious agents and susceptible hosts was consistently great.

## Malaria

Located on the low-lying plain along the banks of the Delaware River, Philadelphia was a marshy place - the climate was hot and humid in the summer and cold and humid in the winter. Stagnant pools of water made for an ideal breeding ground for mosquitoes, while the garbage in the streets aided in the proliferation of flies. While mosquitoes served as the vector for both yellow fever and malaria, flies had a formidable role as vectors for a variety of agents including helminths, fecal

bacteria, protozoans and viruses contributing to the spread of enteric diseases such as dysentery and typhoid fever.

Since Philadelphia was an international port, residents were exposed to imported disease from around the world, in addition to those afflictions native to the city. The infrequent appearance and elevated mortality rates associated with imported epidemic diseases only served to strengthen public fear and apprehension. The anxiety that pervaded the city during such outbreaks was quite evident in the pages of Elizabeth Drinker's diary, as was Drinker's own fear for the safety of her family. During the yellow fever epidemic of 1797, for example, husband Henry and sister Mary were in the city, while Elizabeth and other members of the family were at North Bank on the Delaware River, and away from the heart of the epidemic. On September 9th, Elizabeth wrote to her husband urging him to leave the city. "We have been looking over Careys account of 93. and were frightened at the great increase that took place after this date – let me repeat, that it is my Opinion the longer you stay in the City, the more difficult thee will think it to come away – with dear love to Sister I conclude at present thy E. Drinker – please, if convenient, send when opportunity offer, half pound pale bark."[43]

Imported epidemic disease may have been dramatic in its appearance, but it was those diseases that were constantly present that were the most problematic to the health of the city. Although Elizabeth Drinker's family often left Philadelphia during the summer months to escape the heat and the general unhealthiness of the port, there

was no escaping malaria. Drinker's mention of *pale bark*[44] in the above mentioned letter to her husband made an indirect reference to the presence of this disease.[45] No member of Drinker's immediate family escaped infection, and her diary is replete with references to the disease. During the summer of 1768, for example, the Drinker family was at their summer home in low-lying Frankfort. At the time, Drinker's two young daughters (Sally, age 7 and Nancy, age 4) were suffering from malaria. She wrote that Dr. John Redman and Dr. Cadwalder Evans had identified Sally's fever and condition as "a double tourchen [tertian]; every other day it comes on at about 2 or 3 oclock and every other at 7 or 8 in the Evening – I sat up with her."[46] The physicians prescribed cinchona bark for both Sally and her equally ill sister Nancy, and the symptoms were arrested within a few weeks.[47] Unlike smallpox and yellow fever, an attack of malaria does not confer lifelong immunity. Sally and Nancy would suffer from repeated attacks of the disease throughout their lives.

Benjamin Rush attributed the growing incidence of malaria in Pennsylvania to "the establishment and increase of mill-ponds" and to "the clearing of woodlands without draining and cultivating them."[48] He also noted that the removal of trees between the Schuylkill and Delaware Rivers resulted in an increase in fevers, and that as needed grist mills spread throughout the colony, fever invariably accompanied them. Fevers that were once largely confined to the banks of the Delaware River now ventured eight to ten miles inland. Although Rush correctly surmised the association between fever and stagnant water, he did not understand the role the mosquito played in the transmission of malaria. Rush hoped that a program of filth removal would

eliminate miasma, commonly assumed to be the cause of these fevers.[49] Such a program would have been a great benefit to the city, but would have had no direct effect on the spread of malaria.

The more we learn about diseases, the more complex the concept of causation becomes. The sustainability of malaria in Philadelphia, for example, was dependent on the complicated interplay of numerous factors associated with host (both human and mosquito), agent (the plasmodium), and the environment,[50] and they operated simultaneously and at different levels in order to produce the disease in humans.[51] While some were more directly related than others, several of the elements were absolutely necessary for transmission to occur. Without the parasite *Plasmodium* in the bloodstream, for example, there would be no illness. At the same time, without *Anopheles* mosquitoes, there would be no *Plasmodium* in the bloodstream, and without standing water for breeding and temperatures warm enough to support procreation, there would be no *Anopheles* mosquito. In turn, without human reservoirs of parasites, there would be no source for a large supply of the *Plasmodium*. And so the cycle continued, with extensive interlocking components operating throughout the entire process.[52]

## The Host

An individual's prior immunologic state largely determines the course and severity of the disease. Though an attack of malaria confers only limited and varying

degrees of protection on its survivors, the resistance thus acquired is a significant transmission factor - the higher the resistance, the higher the level of endemicity in the city. When the disease is constantly present in an area, infants experience their first attacks at four to six months of age. Subsequent infections become more severe until the third to fifth year of life, when those who survive begin to develop a homologous resistance (resistance to the same strain of parasite) that progressively reduces the effects of later attacks – assuming that the victims do not migrate from the region. A low level of heterologous resistance to other strains within the same species (but not to other species) results, and repeated infection with several strains can produce heterologous resistance to all of them. Nevertheless, it is obvious that the strain-specificity is a major limitation on the acquired resistance to malaria. Thus individuals from a highly endemic area can develop severe infections when exposed to different strains in or from another community.[53] The city's massive waves of immigration and migration guaranteed the spread of the disease throughout the city and suburbs. Individuals with chronic infections carried their parasites with them when they migrated to and from Philadelphia, and those susceptible to the disease became fresh hosts.

Debilitating diseases like malaria do not kill their victims directly. Instead, they weaken them and reduced their resistance to other disorders. This was especially true for new arrivals to the city. The date that malaria made its first appearance in Philadelphia is not certain, but John Bartram spoke of an epidemic of "Dumb Ague" in the summer of 1746,[54] a time of intense immigration. The Christ Church Bills of

Mortality for the years 1747 and 1748 show severe epidemics with 47 and 35 deaths, respectively, from "fever and ague." Additionally, the Billsof Mortality record annual deaths from the disease for the next thirty years, indicating that it was constantly present in the city.[55] Environmental conditions undoubtedly promoted its spread, since settlement was located primarily along the Delaware River, where there existed a healthy mosquito population. As both infected and susceptible people moved into the city, the cycle of malarial infection was maintained.[56]

## The Agent

Norman Taylor has written that "man, the mosquito, and malaria are mere incidents in the life history of an organism that needs our blood for food, uses the stomach of the mosquito to complete its sex life, and in the process causes the most devastating disease known to science."[57] Seen from this perspective, malaria is simply the background, while it is the plasmodium that is the principal actor. When one considers that all human-infecting strains of four species of malarial parasites are transmitted globally by about sixty species of *Anopheles* mosquito in a cycle in which insect and human act as successive hosts, the primary role of the parasite becomes quite clear. The female adult mosquito requires blood meals to produce fertile eggs, and if the meal is taken by biting an infective human, it may also ingest malarial plasmodia. The parasites migrate throughout the mosquito's body, and those that reach the salivary gland are injected into another human host when the mosquito takes its next human blood meal. There they eventually enter the blood stream, and

as the infected red blood cells rupture (causing malaria's characteristic febrile response in humans), the parasites move to other red blood cells where some of them continue their maturation and await ingestion by a mosquito, thus beginning another round of the process. Others remain in the blood stream, proliferating until their life cycle is broken by the infected person's immune response, antimalarial drugs, or the death of the human host.[58]

Malaria is not a single disease in humans but is actually a family of four different diseases caused by four different parasites.[59] The parasites are similar in appearance, all belonging to the genus *Plasmodia*. The four species names are *falciparum*, *vivax*, *malariae*, and *ovale*. The fevers caused by these four organisms vary enough in their clinical presentations that different labels evolved for their symptom complexes. The two dominant organisms in Philadelphia were most likely *falciparum* and *vivax*. *Falciparum* causes the most severe form of malaria. The victim suffers from intermittent high fevers, with severe headache, parched throat, and severe body aches. Mortality can range from 20 to 40 percent, especially in a host who has never seen the infection before and has no acquired immunity. *Vivax* malaria causes a much more benign disease but a highly unpleasant one nonetheless. It can kill up to 5 percent of its victims, although mortality is usually much lower. Like *falciparum*, it causes high fever,[60] headache and icy pains throughout the skeleton. In the case of both types of malaria, a sequence of chills, fever and sweating happens at about forty-eight-hour intervals, reflecting the life cycle of the parasite itself. This creates a pattern of fever on day one and then day three,

generating the label *tertian* for this cycle. The intermittent pattern helped to distinguish malaria from, say, typhoid fever, which presented with a more continuous fever.[61]

## The Environment

Environmental factors, migration and the clearing of land all converged to create an ideal setting for malaria to become established in Philadelphia. Sequential changes to the landscape, in particular, had a direct impact on the *Anopheles* mosquito. Before the land was cleared, the deeply shaded woodland was inhospitable for breeding. *Anopheles* mosquitoes prefer to lay their eggs in water where they receive sunlight. Once the trees were felled, existing ponds and pools became exposed. Moreover, as the city became more settled, new ponds were built, and water collected in depressions in the landscape created by clearing rocks and stumps. This kind of activity created new habitat, not just for people, but for numerous insects. A nineteenth-century observer noted that "the first breaking up of the soil appears, from a variety of observation, scattered through our topographical descriptions, to be frequently followed by autumnal fever; and, on the other hand, long-continued cultivation is accompanied by diminution of that disease."[62] The pattern was observed so commonly that people accepted the predicted fluctuations in health as part of the cost of settlement.[63] They even named the strange phenomenon *seasoning the land*.[64]

The disease flourished in warm and humid weather, while colder temperatures diminished its prevalence. The seasonal decline in new cases, however, did not diminish the *burden of disease*. Malaria severely weakened its victims, making them susceptible to other infections. Among people who were poorly nourished or had immunity that was depleted from other conditions, the disease could linger for months, adding burden to an already weakened system. In late autumn and winter, for example, respiratory disorders emerged as a major cause of mortality in the city. Those who were already debilitated from repeated bouts of malaria often succumbed to these infections. Although respiratory diseases took a heavy toll on the entire community, they were particularly devastating among the very young and the elderly.

## The Disease

Malaria,[65] also known as "ague"[66] or "autumnal fever," was unlike outbreaks of smallpox and yellow fever. It did not sweep the city in waves, and then vanish, only to appear years later. It was an endemic rather than epidemic disease, and its presence was constantly felt. Smallpox and yellow fever produced dramatic episodes of brief duration. After several weeks of battling the disease, the outcome would be determined – the patient either recovered or died. Such was not the case with malaria, although at first it seemed similar to so many other fevers. Patients complained of headache, increasing fatigue, muscle pains, nausea, and fever – as with the "flu" or a multitude of intestinal afflictions. These episodes were brief. After several days, body temperature would return to normal and recovery appeared at

hand. Elizabeth Drinker describes a particularly severe episode in which three of her children were quite sick from the disease. On September 11, 1768 she wrote "Sally very bad, Nancy [taken] again very bad this afternoon it has turn'd out in her, a regular intermitting fever; several here today." On the 12th she wrote "Sally very ill, light head'd in the night, Sister and Betsy Jervis set up with her, Nancy better." And on September 13th she wrote "Sally and Nancy both very ill this Afternoon, high fevers and delirious Billy but poorly." She continued with daily progress reports for the better part of two weeks, when on September 23rd she finally wrote "The Children through Mercy are now on the recovery; tho Sally's very weak."[67]

Unfortunately for those suffering from the disease, malaria returns and recurs not only with greater force but in repeating episodes. Three stages characterize each recurrence. The first, a cold stage, begins with a sudden chill, a feeling of intense cold and uncontrolled shivering. This stage often subsides within an hour. A hot stage follows, during which the chill is replaced by intense heat. The face grows flushed, the skin dry and burning. A racing pulse and a severe headache are common. The temperature may rise to 106˚F or more. The hot stage can last up to six hours, and is, in turn, followed by the sweating stage. The total duration of the attack is relatively short. Symptoms often begin in the afternoon and the entire cycle is completed within twelve hours. Within twenty-four hours, victims frequently are returned to a reasonably normal condition. Within two or three days, however, the ordeal begins again, with exactly the same sequence: cold stage, hot stage, and a sweating stage. Recurrences can continue with regularity for weeks, months, even

years. At times, the illness may appear to abate, only to return months later when the cycle begins again.[68]

The extent to which malaria is present in a population group is inversely proportional to the stability of various interconnected factors in the total environment. Anything that alters even slightly the ecological balance between human, mosquito, and parasite can dramatically increase malaria in a community. Philadelphia was undergoing considerable transformations through much of the eighteenth century. Within eighty years of its founding, a small village on the banks of the Delaware River became the largest city in the American colonies, and capital of a new nation. Its population grew from 4,000 in 1700 to over 40,000 in 1775. Much of this growth can be attributed to a constant flow of immigrants from Europe, but a rural to urban demographic shift was also taking place in and around Philadelphia, dramatically increasing the numbers of susceptible people living in the city. The destabilized environment that resulted from the massive influx of infectious and susceptible people to the region, in combination with standing water, poor hygiene, malnutrition,[69] crowded houses, and the urbanization of the physical landscape of the city undoubtedly favored the transmission of malaria.

The intensity of malarial transmission in an endemic area like Philadelphia depended upon the density and feeding habits of suitable mosquito vectors and the prevalence of infected humans who served as parasite reservoirs. Malaria can be maintained within an endemic area as long as people receive a sufficient number of

mosquito bites each night to keep the plasmodia within the human population.[70] Tipping the balance between numbers of biting mosquitoes and numbers of human hosts can lead to a gradual reduction and demise of the malarial parasites.[71] Long-lasting climate changes, such as cold or dry spells affecting mosquito numbers; environmental or manmade changes affecting mosquito breeding places; and cultural practices, such as the use of window screens can shift the paradigm. In hyperendemic areas (areas where more than half of the population is parasitemic), transmission usually was constant, and disease manifestations were moderated by the development of immunity. Mortality was largely restricted to infants and to nonimmune adults who migrated into the region. When the prevalence of the disease was lower, as it was in Philadelphia, transmission typically was intermittent.[72] In this situation, solid immunity did not develop and the population suffered repeated, often seasonal, epidemics - the impact of which was shared by people of all ages.[73]

## Tuberculosis

The greatest threats to life in Philadelphia during the eighteenth century were endemic infectious diseases that flourished in densely populated and unsanitary urban centers, and tuberculosis[74] was among the more significant of these diseases.[75] A chronic, infectious disease caused by the bacterium *Mycobacterium tuberculosis*, it, too, was particularly responsive to environmental cofactors[76] such as crowded living conditions, harsh working conditions, and a host of cultural and socioeconomic factors that enhanced the spread of the disease. With the exception of the bovine

form of the disease, which is transmitted through contaminated milk, *M. tuberculosis* is transmitted overwhelmingly by droplet infection. Upon entering the human body, the organism can lie dormant for long periods of time. The lungs are the most frequently infected organ, although tuberculosis can infect any part of the body. As was the case in William Drinker's battle with the disease, its course was often irregular and unpredictable, with episodic attacks alternating with periodic remissions.[77] Those in the active stage deteriorated slowly, and the process of dying could span many months or years.

Tuberculosis, although possessing some features common to epidemic diseases, frequently manifested itself in slow-moving cycles. It peaked in England about 1650, declined until 1715, and surged to even higher peaks in the eighteenth and early nineteenth centuries. In the colonies, the incidence of tuberculosis increased significantly during the course of the eighteenth century. High housing density, a susceptible population, and the migration of infected individuals from England facilitated the spread of the disease in both rural and urban areas. There is general agreement that the incidence and prevalence of tuberculosis and other pulmonary disorders are influenced by population density, nutrition, and occupation, and that a synergistic relationship between these variables may exist. Of the three, population density – particularly housing density – may well have played the most important role in Philadelphia. The significance of diet in the city's tuberculosis morbidity and mortality, however, is less clear. Protein deficiency can increase vulnerability to tuberculosis as well as to other infectious diseases,[78] but there is little

evidence to suggest that severe malnutrition was common in Philadelphia, even among the urban poor.

Disease classification was more art than science during the eighteenth century, with relatively few diseases identified as separate entities. Bloody flux (dysentery), consumption (tuberculosis of the lungs), nervous fever (typhoid fever or typhus), remitting and intermittent fevers (malaria), yellow fever (bilious, putrid or malignant fever), smallpox, scarlet fever, measles, and whooping cough nearly exhaust the list of diseases that can be identified with some certainty.[79] The remaining "diseases" were commonly descriptions of external symptoms or manner of death. Illnesses that were in fact descriptive of the manner of death are frequently found in the bills of mortality. The most common of these was decay, also called debility or atrophy. Considered a specific disease, decay meant any wasting illness, usually lingering over a period of weeks or months and involving loss of flesh. It could have been almost any disease; worms or other parasites, lead poisoning, diabetes, and cancer are a few possibilities. It would seem from a few lengthy descriptions, however, that decay was often an undiagnosed case of tuberculosis.[80]

The term "consumption" was often and aptly used to describe tuberculosis. Unlike the rapidly acting, decisive diseases like smallpox and yellow fever, consumption came on gradually, beginning as an apparently inconsequential respiratory infection or "cold" that lingered on as persistent fevers, disquieting sweats, and a seeming dissolution of the body. Victims were indeed "consumed" as

appetite declined, weight disappeared, and the skin took on a pale, almost transparent appearance. As the disease progressed, there were incapacitating and often violent attacks of coughing. These turned most ominous when the resulting phlegm became first tinged with blood and then bright with it.[81] Elizabeth Drinker often wrote of her son's battle with the disease. "He went on Horse-back towards New England – but was stop'd at a place call'd Rye 30 miles beyond New York, with a fever and spitting of blood…brought up two quarts of blood from his lungs in 3 days."[82]

With the possible exception of dysentery, respiratory illnesses were among the leading causes of death in Philadelphia.[83] This was particularly true for African Americans. White mortality was highest in the summer and fall as malaria and diarrheal diseases adversely affected European Americans unaccustomed to Philadelphia's semitropical summer environment. Late fall and winter were most deadly to African Americans, with respiratory diseases the most likely culprit.[84] The prevailing system of health care, such as it was, undoubtedly exacerbated the problems posed by these disorders. While cold indoor temperatures were a characteristic feature of eighteenth-century housing, and certainly antagonistic to health and well being, failure to provide sufficient fluid intake in times of illness may have posed one of the most serious risks to health. Moreover, certain diseases often had symbiotic relationships with each other. The severity of respiratory disorders, for example, was magnified when they occurred in association with other ailments. Vitamin D deficiency, for example, predisposes individuals to these diseases.

John Tennent noted that "pleurisy," a term often used to describe respiratory disorders, was "the most fatal Disease that affects the Constitution of the Inhabitants of this Country."[85] Peter Kalm – a foreign visitor to the colonies – concluded that "consumptions, fevers, convulsions, pleurisies, haemorrhages, and dropsies" were the leading causes of mortality in Philadelphia between 1730 and 1750. Kalm could not assess accurately the population of Philadelphia at mid-century because the "bills of mortality" were "not kept regularly by all churches." Nonetheless, in reviewing these bills, Kalm lists "consumptions" first in "those diseases which were then most fatal in Philadelphia."[86] A contemporary of Kalm writing in the *American Museum* estimated that 19 percent of all deaths in Philadelphia during 1787 were due to consumption. Elizabeth Drinker frequently mentioned influenza and pleurisy in her diary, as the entire family suffered from repeated bouts of both diseases. It was after one such bout of influenza in 1788 when William Drinker's tuberculosis became active.[87]

Most eighteenth-century respiratory disorders were endemic and seasonal in character. But the growth in population and expansion of trade began to render Philadelphia and other colonial cities somewhat more vulnerable to influenza epidemics and pandemics. At the beginning of the eighteenth century, distance protected the colonies from outbreaks occurring elsewhere in the world. The European epidemic of 1708-9, for example, did not reach the American colonies. Influenza was prevalent in the Northeast and Middle Atlantic colonies, however, during the years 1732-33. This may have been a late flare-up of the 1729-30 pandemic that began in Russia and moved westward through Europe. In addition,

indigenous influenza outbreaks occurred periodically in many areas, as the newly independent colonies became part of the larger disease pool. Influenza appeared in pandemic form in 1781-82 and again in 1788-89, and it affected millions of people in both Europe and America. Case fatality rates associated with influenza, however, remained low. With the exception of the risks to elderly and chronically ill persons, influenza was not usually a lethal disease in the eighteenth century.[88] It did, however, leave its victim in a weakened state, and subsequently more susceptible to other infections.

Tuberculosis was a major killer of those between the ages of twenty and forty. Although most Philadelphians had been exposed to tuberculosis in childhood, children were often able to hold the disease in check.[89] After age twenty, however, resistance decreased, possibly because of the added stress of hard labor among men and pregnancy among women.[90] What accounts for the rise in incidence of this disease is not clear, but factors related to increasing urbanization certainly played a role. In his history of epidemic diseases, Noah Webster wrote that "pestilence has always been the *peculiar curse of populous cities*." Of 200 general plagues, he added, "almost all have been limited to large towns." Webster's concept of crowding, however accurate, was too limited. Tuberculosis and other pulmonary disorders were not confined to more populous communities; they were present in rural areas as well. The critical element was not total population, but household size. Benjamin Rush pointed out that individuals who lead active, outdoor lives do not get the disease. It is more common, however, among woman who lead a quiet, indoor life.[91] Eighteenth-

century data reveal that, irrespective of town size, the typical dwelling (which was relatively small compared to modern structures) contained from 7 to 10 inhabitants. Elizabeth Drinker's household was no exception. In addition to her children (a total of nine, with four dying in infancy or early childhood), she lived with her husband, her sister Mary, and the many visitors, servants and boarders who frequented the Drinker home. Such crowding facilitated the household transmission of mycobacteria[92] and other organisms.

Other elements merely compounded the risks of contagion. Relatively inefficient heating led inhabitants to seal doors and windows during colder weather, and eighteenth-century physicians advised against opening windows even in warmer weather. Moreover, caretakers often slept in the same bed with their patients. This custom was frequently mentioned in Elizabeth Drinker's diary, particularly in connection with William's illness. In a letter dated October 18, 1791, she writes to her husband "our dear son has just gone to bed after setting up above 6 hours, he thinks he coughs less sitting up than lyeing…Nancy or myself lay in the same bed by him, ye other in a Cot close by…"[93] These types of behavioral patterns contributed to the spread of tuberculosis and pulmonary disorders in general. Migration from England, where the incidence of these diseases had reached unprecedented heights, also added to the risk of transmission in the city.[94]

The tubercle bacilli reach human hosts almost exclusively through aerial transmission. Talking, coughing sneezing, spitting, singing, and other respiratory

actions produce airborne particles called droplet nuclei; one is enough to establish an infection if inhaled. Once airborne in a closed space, droplets disperse, and some remain suspended, like smoke. Larger ones fall, presenting little threat, although dry tubercle bacilli are capable of surviving for months. After entering the body, they are remarkably durable, and can remain viable throughout the host's lifetime. The bacilli can exist in a state of dormancy until resistance fails, whereupon they can cause disease even if they never did so when first entering the body. Whether or not tubercular bacilli cause immediate disease upon infection depends on several factors such as age, gender, and genetics. In addition, environmental factors such as crowding and nutritional status can increase the over-all susceptibility of the host to infection.[95] Since tuberculosis is acquired almost exclusively by the respiratory route, infection requires exposure to someone who is actively transmitting the bacteria. Although the disease is communicable,[96] it is not highly so. Transmission is most likely when there is prolonged, close contact between individuals – most typically in households. Even then, infection may occur in 50% or fewer of household contacts. The extent of close contact and the quantity of organisms produced by the cough or sneeze are the two factors most related to the likelihood of infection.[97]

The tubercle bacillus does not itself cause damage to the body. Rather, cellular and tissue damage arises from an allergic reaction or hypersensitivity to the bacillus; after the body has become allergic to invading tubercle bacilli, the immune system destroys them. The process, however, releases proteins and fatty substances that in turn cause inflammation and can damage surrounding tissue and cells. The

same process also creates the illness's distinctive tubercles. Individual resistance to tuberculosis fluctuates markedly. Quiescent infections rekindle when resistance is depressed, only to subside when resistance returns. Acquired resistance to the tubercle bacillus confers no durable protection as in measles, smallpox and yellow fever. Rather, it can assist the development of active disease.[98]

William Drinker's tuberculosis became active in 1788 when he was twenty-one years old. The entries in Elizabeth Drinker's journal for 1786 are irregular and the volumes for 1787 and 1788 were burned, so that there is no systematic record of the first years of the illness.[99] William's illness was of great concern to his mother, and she frequently expressed these worries in her diary. In one such entry on July 3, 1789, she wrote that William Drinker and Ben Wilson sailed in the ship *Mary* for Baltimore, a voyage for William's health, with Ben as a companion. Eighteen days later, "received a Letter this evening from our dear Son, which was a great satisfaction to me, - but there is no joy, – his being indispos'd after his Arrival makes me desirous of soon hearing from him again."[100] Drinker again writes of her son's illness on December 31, 1793.

> "Our dear William, who has been in a low state of health for upwards of four years, is at present as well as could be expected considering what he has been through, it is four years this last fall since he had the Epidemic cold, call'd the Influensia, and had been drooping for a twelve month before, all our family had it, and I believe every family in the City, and Country also, more or less, William was the worst of us, and longer getting better, he had another attack the spring following – was poorly all summer. The fall after, he went into the beach woods, was from home about 6 weeks, lay out in the woods, one night, and but little better many other nights – he return'd home much recover'd – but on the 9th of Novemr. Was taken with a sore throat, he has smok'd a Cagar

> the evening before in the store in water-street with some young men, not being us'd to it, found himself sick, and in a sweat, went out in the cold air, and walk'd as far as walnut street – the next day the 9th. he went with his father to visit R James, Abel James very lately dead. – had a chilld in the Evening the next day we sent for Dr. Kuhn, who said he had a touch of the Quinsey, but it turn'd out much worse than the Doctor expected, he was many days that he could not speak, - and when the disorder in his throat was better he lost the use of his limbs, so as not to be able to walk alone, or to button his Jacket, he was four months confin'd to his Chamber, - in the spring he rode out in a Close Carriage, seem'd to gether strength in the Summer, in the fall 91, he went on Horse-back towards New England – but was stop'd at a place call'd Rye 30 miles beyond New York, with a fever and spitting of blood – hir'd a Chaise and Man to bring him back to N-Y – where he was ill at Henry Haydocks, brought up two quarts of blood from his lungs in 3 days, Dr. Jones was sent for, who call'd in Dr. John Bard; Robert Browne wrote an account to HD. of Billys having broke a blood vessel, and his dangerous situation, he and self, set off the next day for NY…I fully expected to have found my dear Boy a Corps on our arrival at NY – the journey to me was in truth an anxious one – we walk'd from the Elizath. Town ferry, and on approaching HHs house I look'd up to the Chambers windows which I found was rais'd up, as I expected, supposing, my son was laying in the front Chamber, on crossing the Street, we were mett by HH junr. Who coming up to me, said 'friend Drinker Billy is better'…William who expected our coming, had fortified himself all in his power, to see us, the discharge of blood had ceas'd for near 24 hours, he had been twice bled in his Arm, before we came, and twice after upon a small appearance of a return of the complaint, he had a hard cough, which was much against him, he keep'd his bed for 3 weeks by Dr. Bards direction – by that means and other judicious [percrecptions] the cough wore off, and he gradually became better – tho amazingly week;…We left H Haydocks on the third of Decemr. And arriv'd at our own dwelling on the 6th. after dinner, I believe I may say with thankful hearts. he was confin'd to the house most of the winter, very poorly in the spring, but re[]uted again in the Summer - in the fall 92, he caught cold staying too late in the evening at Frankford – was 8 or 10 days ill of pleurisy – after that something of the fall fever, so that what little health he geather'd during the summer, was generally overturn'd by a cold in the fall – he is now, the many pullbacks consider'd, as well as might be expected."[101]

William Drinker's tuberculosis was undoubtedly active during this time, with the tubercle bacilli in his sputum. As was often the custom during serious illness for family members to lie in the bed of the patient, William often slept with his mother, father or one of his sisters, so that he could be attended to on a moment's notice.

Other members of the family may have contracted tuberculosis, but no one other than William had an active case of the disease. During the remaining years of the Diary, it is evident that William lived the life of an invalid, never able to tolerate exposure to the elements. He often complained of cough and soreness in his "breast." After 1775, there was no more blood spitting and he gradually became stronger, this in spite of "much bloodletting and violent medication."[102]

The onset of active tuberculosis often coincides with lowered resistance. Today, this type of lowered resistance is associated with immune systems compromised by disease, drug addiction, etc. Susan Klepp argues that in the early national period, lower class men were more than twice as likely to die of tuberculosis as upper class men. Hard manual labor, poor nutrition, housing and sanitation are the probable explanations for the lower resistance of the men of the lower class.[103] William Drinker was among the fortunate; he lived until 1821, and was fifty-four at the time of his death. The cause of his death is not known, but it most likely was not tuberculosis since he was reasonably well at this time and had not suffered from an active form of the disease since 1807. He was evidently quite resistant to tuberculosis, and, in many ways, was well treated. The family resources permitted change and travel, and allowed him to regulate his employment as his health permitted.[104]

Among the women of Philadelphia, there was no great difference in the incidence of tuberculosis. Twenty-five percent of upper class women and twenty-

nine percent of lower class women died of consumption.[105] Tuberculosis was often thought of as a "household epidemic" because there were many chances for the bacillus to spread in the intimate family circle. The confinement of women to the home contributed to higher rates of tuberculosis, even while it spared them from other infectious diseases and accidents. Overcrowded living conditions were common among both upper and lower class households and certainly played a role in the spread of the disease. Elizabeth Drinker's home, although large by contemporary standards, was still quite crowded, and illness spread through the family with relative ease.

Wherever tuberculosis is rampant, nearly everyone becomes exposed, but only susceptible individuals develop the disease. The very young, sick, poorly nourished, and fragile elderly with poor immune responses become easy targets for tuberculosis. Some individuals can become infected without overt signs of the disease, developing tuberculosis only later in life. This happens when the back-up inflammatory immune response fails to restrain the microbes within their capsulated prisons, allowing the mycobacteria to reactivate and spread. Such a failure occurs when the immune system has been disturbed by another disease, by nutritional disturbance, or by other stresses on the body.[106] The case rate of tuberculosis was similar for upper and lower class women in Philadelphia, but disparities in age at death indicate that resistance to the disease was an important factor among woman as well as men. Women of the upper classes were, on average, eleven years older than lower class women when they died of tuberculosis. A life of labor coupled with lower standards of living may have

contributed to the inability of poorer women to recover their strength and survive the disease.[107]

Tuberculosis is a complex disease, and many epidemiological factors influenced its prevalence in Philadelphia. These included environmental features,[108] differences in the biology of the bacillus,[109] and variations in the host.[110] Tuberculosis was and is a world-wide disease that can flourish in almost any climate. Reservoirs of the principal mycobacterial species included not only humans and animals, but also soil and water. Lung-infected people were the main source of infection by *M. tuberculosis*, but many animals could have become infected by this organism as well, most likely contracting the disease from an infected human. While animal-to-human transmission probably occurred, it did so primarily through consumption of unpasteurized milk from animals with infected udders, though lung-infected cows could have transmitted the disease to their handlers by aerosolization.[111]

As population levels in the city increased, it became difficult for families to keep their own cows in the city. As a result, milk had to be purchased from commercial dairy farms. It was here where cows were housed in the densely crowded, unhygienic conditions that were ideal for animal-to-animal (as well as human-to-animal and animal-to-human) transmission of tuberculosis. Both domestic herd animals and humans serve as reservoirs for the tubercle bacillus. As a result, larger herds of dairy cattle could have facilitated the spread of bovine tuberculosis

through contaminated milk.[112] Although bovine bacilli are ordinarily ingested through the digestive tract via milk or milk products and usually cause intestinal disease, they infrequently lead to pulmonary tuberculosis.[113]

The profound impact of host factors on the prevalence of tuberculosis in Philadelphia was extensive. Poor nutrition, compromised immune systems, and densely crowded living conditions were of prime importance in human exposure *to* and invasion *by* mycobacteria. As a result of the interplay of these and other variables, both the form and the frequency of human tuberculosis show great variations through time and space. Abrupt, severe demographic and environmental upheavals that usually accompany any major transition in a population are almost predictably accompanied by an equally abrupt and marked rise in tuberculosis infections within that population.[114] Philadelphia was experiencing rapid population growth, large-scale immigration, high levels of commerce, war, and a rural to urban demographic shift. As was the case for many of the diseases that plagued the city at this time, the probability of contact between an infectious agent and a susceptible host was consistently great, and a key factor in the transmission of tuberculosis.

## Typhoid Fever

Typhoid fever and dysentery were particularly prevalent in Philadelphia and the most likely causes of high mortality during the summer months. Typhoid fever is caused by the bacterium *Salmonella typhi*, and its symptoms include fever, intestinal

hemorrhaging, skin rash, enlargement of the spleen, and a low white blood count. Because it is spread by contaminated food and water, it can appear in epidemic form. An asymptomatic healthy carrier can also disseminate the disease. The disease occurs most commonly in the late summer and early fall, and can have a mortality rate of 30 percent or higher.[115] The symptoms of typhoid fever, however, are similar to those of dysentery, and the information necessary for a discrete classification of the disease is simply not discernible from the historical data of this period. A number of different bacteria and viruses can cause similar signs and symptoms, each running a highly variable course. Consequently, only descriptive terminology can be applied here, and not definitive diagnoses. Given the environmental conditions that prevailed in Philadelphia at this time, it is likely that a combination of dysentery and typhoid fever was responsible for high summer mortality rates.

Typhoid was another disease that was endemic in Philadelphia during the eighteenth century. Large urban areas like Philadelphia served as centers of trade and commerce, and were magnets for rural residents as well as for European immigrants. The movement of large numbers of people into the city magnified a variety of health-related problems. Rural migration, for example, had the unforeseen consequence of bringing a steady supply of susceptible people into the city – people who had never before been exposed to many infectious diseases. Additionally, many immigrants arrived not only with the infections of their land, but with typhus and typhoid fevers acquired in the close confines of the ships. Crowded housing also facilitated the spread of infection, while contaminated water supplies and unsanitary living

conditions created a favorable environment for bacteria to flourish. These and other factors combined to facilitate the proliferation and spread of the disease.

*Salmonella typhi* bacilli enter the body through the gastrointestinal tract and gain access to the bloodstream through the lymph system. They are shed in the feces of infected individuals, and may be transmitted through direct contact when unsanitary conditions prevail. Flies may also spread the organism from feces to food, although in endemic areas the organism is transmitted primarily through contaminated water and raw milk. Infection results in inflammation of the ileum[116] and colon. In serious cases, this can be followed by ulceration, hemorrhage, and perforation of the intestine. Onset is gradual, beginning with low-grade fever, headache and abdominal pain. The incubation period is eight to fourteen days and leads to florid bloody diarrhea, prostration, and uncommonly low heart rate. In about 10 percent of infected individuals, a discrete pinkish rash occurs across the chest and abdomen. The "rose spot" lesions resolve within a week. In about 2 percent of affected individuals, severe intestinal bleeding occurs during the third week of infection. Untreated, this complication has a mortality rate of 25 percent. Death usually results from intestinal perforation or severe hemorrhage.[117]

The microorganism responsible for typhoid fever belongs to one of the largest and most widespread families of bacteria on Earth, with over 1,700 serotypes recognized. Salmonellae can colonize the gastrointestinal tracts of a broad range of animal hosts, including mammals, birds, reptiles, amphibians, fish, and insects. Some

types of salmonellae are highly adapted to specific animals, while others have a wide range of hosts. As a consequence of both its versatility and enormous animal reservoir, the eradication of all salmonellosis would be essentially impossible.[118] Salmonellosis is generally a mild disease in humans, however, and is characterized by a few hours or days of vomiting and diarrhea followed by weeks to months of shedding the organism in the feces. While the disease is usually acquired by ingestion of contaminated foods, other means are possible. In the 1970s in the United States, for example, more than 10 percent of salmonellosis was acquired from baby turtles, then a favored children's pet. By contrast, and almost unique among salmonellae, typhoid bacilli are adapted to humans alone. They possess a protective envelope that helps them resist the host's immunologic defenses. The percentage of persons who develop typhoid fever after exposure depends on several factors including the virulence and number of organisms ingested, and the susceptibility of the host. Normally, fecal excretion of the organism persists for some weeks, but about 2 percent of infected persons never clear the bacillus from their stools. As a result, a distinctive feature of the epidemiology of typhoid is the existence of a large number of asymptomatic carriers.[119]

Typhoid was well established in Pennsylvania by the 1760s. Unfortunately, the clinical features of typhoid fever are not sufficiently specific to separate it from the many other febrile or infectious diarrhea conditions that were present in Philadelphia at this time. Its symptoms were similar to those of malaria, but the fever stage was more prolonged and without the regular chills that characterized the

disease. Patients suffering from what was believed to be typhoid fever had fluctuating or recurrent fever accompanied by prostration, abdominal discomfort and sometimes watery diarrhea. Also known as "bilious fever," "the flux," "slow fever," "long fever" and "nervous fever," this water-borne disease increased in incidence as the population grew. Polluted streams and contaminated wells insured its spread during the summer months. Just when typhoid fever first entered Philadelphia is not known, but it seems to have been endemic in the city as bills of mortality refer to deaths from "nervous fever" and "ague" yearly. The disease was apparently familiar to Elizabeth Drinker who, on May 31, 1759, visited a friend "who lay ill of a nervous fever."[120] Benjamin Rush reported that the "slow chronic fever" was very common during the autumnal months in the thickly settled parts of the city from 1760 to 1766.[121] It is reasonable to assume that typhoid fever was spread via contaminated wells and by direct contact, since there was no city-wide water or milk distribution center at this time.[122]

The quality of well water in Philadelphia gradually deteriorated as the century progressed, and was no longer as "sweet-tasting" as it once had been. This condition was not surprising, considering that Philadelphians had been gradually polluting their groundwater for over a century, beginning the day the first privy was dug in the city in the year of its founding in 1682.[123] The water that Philadelphians drank came either from wells, cisterns that collected rainwater, or the Delaware River. Cisterns were usually made water-tight with mortared brick sides and floors and were generally about 8 feet in diameter and less than 10 feet deep. Wells, ranging from 3

to 5 feet or more in diameter, were usually lined with dry-laid bricks and were left open at the bottom. Initially the wells were rather shallow, since the water table was only about 15 feet from the surface in the early years of the century. Wells subsequently became deeper as street paving and cisterns channeled off more and more rainwater. Archaeological investigations have shown that by the end of the century, wells were on average 23 feet deep in order to ensure a flow of water.[124]

The majority of wells were located in the cellars or backyards of private dwellings, where there also was typically a convenient privy. The privy pit was usually constructed in the same fashion as the well, lined with dry-laid bricks and unpaved at the bottom. As of 1769, however, a law was passed requiring privy pits to be shallower than wells. By this time, the seepage between privies and wells had become so widespread and the contamination of drinking water so obvious that a city ordinance forbade the digging of a privy pit to the level of the water.[125] It was also illegal to place a privy above an abandoned well. As a result of this contamination, widespread enteric disorders were common in the city. The connection seems even more evident when, as so often happened, a privy was illegally placed on top of a well that had gone dry, as so many wells did in the wake of street paving and the use of cisterns to collect rainwater. As the population grew, so did the problem of water pollution. As a result, underground streams soon became tainted. Exacerbating the problem were a number of tanneries that spewed offal into Dock Creek, which by the 1760s was an open, foul-smelling sewer.[126]

The tanners were asked to leave in 1739, but in the 1770s several were still in operation. After Palatine fever (typhus) and diphtheria outbreaks swept through the city in the 1740s, taking a huge toll on human life, a petition was placed before the Common Council to fill in a portion of Dock Creek. Although parts were filled and used as grazing land, Dock Creek remained largely malodorous for most of the eighteenth century. While the western branch of the creek was apparently filled as early as 1757 and renamed Dock Street, it was not until 1784 that the Street Commissioners finally finished paving over the northwestern branch.[127] Barbara Liggett, who conducted an archaeological investigation of the area around Front and Dock streets in 1968, has questioned the traditional view of historians that the branches of Dock Creek were natural streambeds. She argues that Dock Creek was originally a swampy area, never used extensively for shipping, and that as its branches became increasingly offensive open sewers during the 1700s, it was subsequently filled in.[128] The area around Dock Creek was a constant source of concern for the people of the city, as this marshy area was a prime breeding ground for mosquitoes - the vector for both malaria and yellow fever.

To understand how disease worked in Philadelphia, one has to think ecologically, seeing human health as an outcome of multiple, reciprocal, and continuing interactions among pathogens, hosts and the surrounding environment. The initial portrayal of the city as a health utopia, often used to promote immigration, slowly gave way to acknowledgement of a harsher reality. Periodic yellow fever and smallpox epidemics tended to overshadow the diseases that played a far more

significant role in shaping the public health of the city. Dysentery, tuberculosis, malaria and typhoid fever were among the more significant of these, as they severely weakened their hosts, making them particularly vulnerable to other infections. Moreover, the urban environment of Philadelphia contained the epidemiological factors necessary for the growth and propagation of a wide variety of disease agents. Massive immigration provided a susceptible population group, while international trade, densely packed streets, unsanitary living conditions, a contaminated water supply, and a hot and humid summer climate combined to create ideal circumstances for the proliferation of both pathogen and vector. All things considered, it is not surprising that an environmental health crisis of such magnitude existed in the city at this time.

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[1] Wilson, 2001, p. 283-324.
[2] Typhus is transmitted by the feces of human body lice, but it can also be transmitted by the rat flea. The microbe enters the body through skin lesions and mucous membranes. The disease was a particular problem among new arrivals to the city.
[3] Rush, 1815, *An Account of the Bilious Remitting and Intermitting Yellow Fever, as it Appeared in Philadelphia in the Year 1794*, Vol. 3, p. 197.
[4] Gehlbach, 2005, p. 69-70.
[5] Ultimately, the emergence of a microbial threat derives from the convergence of (1) genetic and biological factors; (2) physical environmental factors; (3) ecological factors; and (4) social, political, and economic factors. See Smolinski, Hamburg and Lederberg, 2003, "Executive Summary," p. 4.
[6] Many infectious diseases either are strongly influenced by short-term weather conditions or display a seasonality indicating the possible influence of longer-term climatic changes. Climate can directly impact disease transmission through its effects on the replication and movement (perhaps evolution) of pathogens and vectors; climate can also operate indirectly through its impacts on ecology and/or human behavior. See Smolinski, Hamburg and Lederberg, 2003, "Executive Summary," p. 4.
[7] Changes in the environment tend to have the greatest influence on the transmission of microbial agents that are waterborne, airborne, foodborne, or vector-borne, or that have an animal reservoir. See Smolinski, Hamburg and Lederberg, 2003, "Executive Summary," p. 4.
[8] The Christ Church Bills of Mortality annually listed numerous deaths from "consumption," "decay," and "purging and vomiting." There were, for example, 26 deaths from consumption, 19 deaths from decay, and 18 deaths from purging and vomiting listed for the year 1769. There were also 4 deaths recorded for nervous fever (typhoid or typhus) that year. See Klepp, 1991, p. 73.
[9] As used in malaria epidemiology, a defined and circumscribed locality containing the epidemiologic factors needed for transmission: a susceptible human community, a source of infection, a vector population, and appropriate environmental conditions. The term can be applied to other infectious diseases. See Last, 2001, p. 72.
[10] The Christ Church Bills of Mortality annually list excessive deaths from "consumption" (tuberculosis), "decay" (tuberculosis), and "fever, fits and flux." When epidemics of smallpox,

measles and yellow fever struck, epidemic mortality patterns were superimposed upon these already high base-line rates, driving up mortality levels even further.

[11] The condition has several causal agents including the ameba, *Entamoeba histolytica*, and several species of bacteria, especially in the genus *Shigella*. Direct infection can take place in circumstances of extreme crowding. During the eighteenth century, such places typically included jails, hospitals and ships, but houses were also quite crowded, and their role in transmission cannot be overlooked. Indirect spread by fecal contamination of food and water, however, was probably the more common route. See Patterson, 2003, "Dysentery," p. 105; Poirier and Feder, 2001, p. 89.

[12] Grob, 2002, p. 85-6; Duffy, 1953, p. 214-22.

[13] Crane, 1991, p. 191. Excerpt from the *Diary of Elizabeth Drinker*, June 17, 1773.

[14] The Christ Church Bills of Mortality repeatedly show high infant mortality rates. In 1746, for example, there were 74 deaths of children "under two years" of age. This age group showed consistently high death rates year after year. There were 17 deaths from "purging and vomiting," 7 deaths from "teeth" and 3 deaths from "worms" during the same year. See Klepp, 1991, p. 62.

[15] Currie, 1792, p. 68.

[16] Klepp, 1989*, Philadelphia in Transition*, p. 245.

[17] Although it is most likely that bacterial agents were responsible for much of the dysentery that plagued Philadelphia at this time, amebic dysentery may have been present as well. Amebiasis (amebic dysentery) is an infection of the colon caused by a parasitic protozoan, the ameba *Entamoeba histolytica*. Several species of ameba inhabit the large intestine. Most are harmless commensals or minor parasites, usually causing little or no clinical damage. Amebas cause disease when they invade the mucosal and submucosal layers of the large intestine, producing characteristic flask-shaped lesions. In severe cases, the lesions become large and confluent, resulting in substantial tissue destruction, bleeding, loss of fluids, and sloughing patches of mucosa. Damage to the intestinal wall reduces water absorption, and loose stools with blood and mucous are passed. Clinical symptoms range from mild diarrhea and abdominal discomfort to frequent loose stools with blood, mucous, severe pain, emaciation, and prostration. Amebic dysentery tends to be a chronic disease with a gradual onset and little or no fever. The stools tend to be more abundant but less frequent and not bright red with blood, as is common in bacillary dysentery. Amebic dysentery has a longer incubation period, 20-90 days or more, compared to 7 days or less for the bacillary form. The shorter incubation period and greater probability of water transmission of bacillary dysentery makes this form of the disease more likely to occur in dramatic epidemics. Patterson, "Amebic Dysentery," 2003, p. 20-1.

[18] Bacillary dysentery begins when bacteria (*Campylobacter*, *Salmonella*, and *Yersinia*, as well as some strains of the common intestinal bacillus *Escherichia coli*, can invade mucosa of the large intestine and cause dysentery, but members of the genus *Shigella* are by far the most important agents) invade the mucosa of the large intestine, where they can cause mucous secretion, edema, and superficial ulceration and bleeding. The watery diarrhea is probably caused by a toxin that increases the secretions of cells of the intestinal wall. The incubation period is short; often 1 to 4 days. Onset is sudden in children, with fever, drowsiness or irritability, anorexia, nausea, abdominal pain, tenesmus (a spasm of the rectum accompanied by a desire to defecate without the production of significant amounts of feces) and diarrhea. Blood, pus, and mucous appear in diarrheal stools within 3 days. Increasingly frequent watery stools cause dehydration, and death can occur in as little as twelve days. If the patient survives, recovery usually begins after about 2 weeks. In adults, there is usually no fever, and the disease resolves itself in anywhere from 1 to 6 weeks. Symptoms in both children and adults may vary from simple, transient diarrhea to acute dysentery and death. Patterson, 2003, "Bacillary Dysentery," p. 43-4.

[19] Crane, 1991, p. 188, 192. Excerpts from the *Diary of Elizabeth Drinker*, March 8, 1773 and July 6, 1773.

[20] Paper is entitled *Experiments and Observations on the Mineral Waters of Philadelphia, Abington and Bristol, PA*. See Drinker, 1937, p. 25.

[21] In the course of history, the term "cholera" has been variously applied. "Cholera infantum" was another name for dysentery, and it is not to be confused with cholera, the disease caused by aerobic rod-shaped *Vibrio cholerae* bacilli. Cholera did not reach North America until 1832. The first outbreak was in New York on June 23rd. It was followed by an outbreak in Philadelphia on July 5th of that same year. See Speck, 2003, p. 76.

[22] Rush, 1815, "An Inquiry into the Cause and Cure of the Cholera Infantum," Vol. 2, p. 217.
[23] Wolman, 1974, p. 239-40.
[24] Rush, 1815, "An Inquiry into the Comparative State of Medicine in Philadelphia, between the years 1760 and 1766, and the year 1809," Vol. 4, p. 229.
[25] DuPont, 2003, p. 93.
[26] *Shigella* is spread much more easily than other bacteria. Its infectivity dose (ID) is extremely low. As few as ten *S. dysenteriae* bacteria can cause clinical disease, whereas for many other diseases, hundreds or thousands of cells are required for an infectivity dose (ID).
[27] Patterson, 2003, "Bacillary Dysentery," p. 44.
[28] Grob, 2002, p. 118.
[29] John Jerman's *The American Almanac*, Philadelphia, 1741 contains an essay by John Bartram on the "True Indian Physic" or ipecacuanha as useful in the treatment of bloody flux.
[30] Wolman, 1974, p. 241.
[31] See Estes, 1990, p. 24 for a discussion of *Bateman's Pectoral Drops*; p. 90-1 for a discussion of *Godfrey's Cordial*.
[32]*Pennsylvania Gazette*, March 22, 1764 and April 20, 1769.
[33] August Shubert, German Apothecary, *Pennsylvania Gazette*, September 5, 1765.
[34] Longworth, 2001, p. 276-7.
[35] Last, 2001, p. 158.
[36] Longworth, 2001, p. 37.
[37] Patterson, 2003, "Ascariasis," p. 42.
[38] Carolina pink root was effective in treating worms in children. See Estes, 1990, p. 181 for a discussion of Carolina pinkroot – also known as *Spigelia marilandica*.
[39] Crane, 1991, p. 178, 226-7. Excerpts from the *Diary of Elizabeth Drinker*, July 29, 1772 and August 20-21, 1777.
[40] Klepp, 1989, *Philadelphia in Transition*, p. 230.
[41] The Christ Church Bills of Mortality continued to list significant numbers of deaths from enteric disorders. In 1796, for example, there were 11 deaths from "flux," 18 from "purging and vomiting," 14 from "fits," and 9 from "teeth and worms." See Klepp, 1991, p. 82.
[42] William Currie, 1792, p. 100.
[43] Letter from Elizabeth Drinker to Henry Drinker dated September 9, 1797. Quoted in Drinker, 1937, p. 130.
[44] Cinchona was widely used as a tonic, as an astringent, and as an antiseptic, especially for patients with intermittent fevers (malaria), for dyspepsia (impaired digestion), and for spasmodic symptoms of chest disease, such as cough. It was also prescribed for most patients who had been debilitated by continued fevers, and, for venereal disease patients, in a gargle made with *Conserve of Roses* (flowers of the rose beaten into a uniform mass with sugar), to counteract the side effects of therapeutic mercury in the oral cavity. Cinchona's side effects included vomiting, diarrhea, tachycardia, tinnitus, and partial deafness. Cinchona bark contains about 8% quinine and up to 3% quinidine. See Estes, 1990, p. 48-9 for a discussion of the medicinal qualities of cinchona; p. 53 for a definition of conserve.
[45] The powder of the bark was another variety of cinchona, which was used in the treatment of intermittent fevers. Malaria was an intermittent fever, and always present in the summer at North Bank on the Delaware, where the family often spent the summer months. They required a large supply of bark simply to keep pace with the disease. See Drinker, 1937, p. 130.
[46] Crane, 1991, p. 144-5. Excerpt from the *Diary of Elizabeth Drinker*, September 16, 1768.
[47] Intermittent fevers occur at short, regular periods, remittent fevers at longer, irregular periods. A fever with a three-day cycle was called tertian; one with a four-day cycle was called quartan. Though the doctors labeled Sally's fever a double tertian, Elizabeth Drinker seems to describe a two-day double cycle. Sometimes called agues, most of these recurring fevers were probably various forms of malaria. Medical writers of the era such as William Buchan recognized the connection between the prevalence of these fevers and low marshy areas, stagnant water, and tropical climates. Discussion taken from Crane, 1991, p. 145.
[48] Rush, 1786, "An Inquiry into the Causes and Increase of Bilious and Intermitting Fevers in Pennsylvania, with Hints for Preventing Them," paper read before the American Philosophical Society on December 16, 1785.

[49] Rush, 1815, "An Account of the Climate of Pennsylvania, and its Influence on the Human Body," Vol. 2, p. 25.
[50] Climate clearly has a bearing on the density of mosquito breeding and hence the likelihood of malaria transmission. Long hot summers both encourage multiple cycles of mosquito reproduction and speed the development of the parasite within the vector. Humphreys, 2001, p. 11.
[51] A list of transmission factors can be found in Manson-Bahr and Bell, 1982, *Manson's Tropical Diseases*, p. 43.
[52] Gehlbach, 2005, p. 86.
[53] For measurements of endemicity and immunity, see Manson-Bahr and Bell, 1982, *Manson's Tropical Diseases*, p. 12-15, 44; Miller, 1984, "Malaria," p. 226-27, 233; De Bevoise, 1995, p. 145. Natural genetic resistance and passive transmission of maternal resistance to infants both exist to some degree. The natural passive protection in infants lasts four to six months but is lost thereafter. With regard to acquired resistance, it should be noted that the immune system, when not otherwise compromised, responds with reasonable effectiveness to any malarial invasion. In a primary attack, lymphoid macrophages and humoral antibodies can destroy most of the invaders, but their counterattack is only mounted against the parasites during their development in the red blood cells. Ironically, incomplete victory is the best result, the more so for those who will continue to live in regions of high endemicity. So long as some blood cells remain parasitized, the immune reaction is stimulated, and resistance is maintained – and even builds - against succeeding attacks. If all parasites are destroyed, however, acquired resistance is rapidly and easily lost. Modern antimalarial drugs also produce that result, as does prolonged absences from an endemic area. See De Bevoise, 1995, p. 233.
[54] John Bartram in a Letter to Peter Collinson dated November 2, 1746 in *Philadelphia Medical and Physical Journal*, 1804, Vol. 1, p. 146-151.
[55] Wolman, 1974, p. 225; Klepp, 1991, p. 61-88.
[56] Infective mosquitoes need to live close to human dwellings in large numbers for the cycle of malaria to be maintained in the human population. The mosquitoes must be able to ingest a large number of infective forms of the parasite, and the must be willing and able to bite humans repeatedly. The cycle requires sufficient numbers of human and animal hosts to maintain the presence of the parasites. Climate and environment also have to be favorable for long-term mosquito survival and breeding. See Beck and Davies, 1981; Horsfall, 1972; Barnes, 2005, p. 70.
[57] Taylor, 1945, p. 24.
[58] *P. vivax* is synonymous with tertian, benign tertian, and simple intermittent fever, all of which describe an infection characterized by a high fever that recurs every forty-eight hours, or a little less, and rarely results in death. *P. falciparum* has been variously known as malignant tertian, subtertian, aestivo-autumnal, and pernicious. The fever cycle is less regular, as the name subtertian suggests. If untreated, primary attacks are often fatal. For a discussion, see Manson-Bahr and Bell, 1982, *Manson's Tropical Diseases*, p. 3-6; Miller, 1984, "Malaria," p. 223-39.
[59] A parasite is an organism that lives in or on another organism (the host), obtaining nourishment from it. See Rothenberg et al, 2000, p. 422.
[60] As in all fever spikes, the body undergoes a change in the "thermostat" that regulates internal temperature. Sensing (falsely) that the body's temperature is too low, the hypothalamus (a part of the brain that controls many functions including body temperature) orders shivering in order to increase the temperature, creating the sensation of severe chills that accompanies the rise of fever. That rise in temperature is so rapid that the victim's teeth chatter and the bed vibrates. Once the new level has been reached, the patient feels terribly hot, and then as the temperature falls, profuse sweating follows, leaving the sufferer washed out and exhausted. See Humphreys, 2001, p. 8-9.
[61] See Humphreys, 2001, p. 8-9.
[62] Drake and Levine, 1964, p. 710.
[63] Benjamin Rush observed that "families enjoy good health, for many years, in the swamps of Delaware and North Carolina, while they are in their natural state, but that sickness always follows the action of the rays of the sun upon the moist surface of the earth, after they are cleared. For this reason, the cultivation of a country should always follow the cutting down of its timber, in order to prevent the new ground becoming, by its exhalations, a source of disease." See Rush, 1815, "An Inquiry into the Various Sources of the Usual Forms of Summer and Autumnal Disease, in the United States, and the Means of Preventing Them," Vol. 4, p. 136.

[64] Gehlbach, 2005, p. 86-7.
[65] *P. vivax* (causing benign tertian malaria) and *P. falciparum* (causing malignant tertian malaria) are responsible for the great majority of malaria cases throughout the world. Within each species, however, there are different strains of the pathogen, and each causes a unique reaction in the host. The pattern of fever spikes may vary slightly, but more important with reference to species and strain is the fact that acquired immunities tend to be species- and strain-specific. A person who is infected with *P. vivax*, St. Elizabeth Strain, for example, tends to develop resistance only to that strain. See Rutman and Rutman, 1976, p. 36.
[66] Historian Margaret Humphreys argues that toward the end of the eighteenth century, medical parlance moved toward greater exactitude, as the profession understood fevers with greater specificity. Although the label *intermittent fever* had been used at least as early as 1609, in the eighteenth and nineteenth centuries it came to characterize the disease now called malaria. As with the term *ague*, the mapping was not exact. Many viral diseases, as well as tuberculosis and some malignancies, may feature waxing and waning fevers, and these would have offered confusion. "Intermitting" fevers were often contrasted to "continued" ones, which included typhoid and typhus. Other adjectives might be applied, such as *putrid, malignant*, (often used to describe yellow fever) or *remittent*, in order to bring greater precision and prognostic skill to the bedside assessment. Furthermore, the classic case of intermittent fever, with a smooth pattern of forty-eight hour cycles, usually occurred only in the malaria "virgin" who was entering a malarious country for the first time. Seasoned residents would have bouts of illness, spiking chills and fever, and other manifestations, but since they combined steady exposure to the parasite with some degree of acquired immunity, they rarely displayed the clear pattern that textbooks described. Therefore, true intermittents were seasoning illnesses; locals never got them – because they already had them. Still, the use of the term *intermittent* began to distinguish medical language from that of the common folk, who continued to complain of agues. See Humphreys, 2001, p. 28.
[67] Crane, 1991, p. 144-5. Excerpts from the *Diary of Elizabeth Drinker*, September 11, 1768, September 12, 1768, September 13, 1768 and September 23, 1768.
[68] For a discussion of the various stages of the disease, see Gehlbach, 2005, p. 75.
[69] Malnutrition has long been known to play a role in susceptibility to death from diarrhea, respiratory infection and malaria. Not as well understood are the roles of famine, war, crowding, urbanization and population growth. See Smolinski, Hamburg and Lederberg, 2003, p. 220.
[70] Livingstone, 1986, p. 17-28.
[71] Barnes, 2005, p. 86.
[72] Malaria was not hyperendemic in Philadelphia because the cold winter climate proved fatal to the mosquito.
[73] Ryan and Ray, 2004, p. 715.
[74] Fragmentary evidence indicates that tuberculosis was present in the early days of settlement. Contemporary accounts refer to deaths from "consumption," "scrofula," "pleurisies," and "phthisis." The symptoms, which included coughs, fever and bloody sputum, are consistent with the modern understanding of tuberculosis, but eighteenth-century bills of mortality were often unable to distinguish between tuberculosis and other pulmonary disorders.
[75] Numerous deaths from "consumption" and "decay" are listed yearly in the Christ Church Bills of Mortality. Tuberculosis and dysentery were responsible for more deaths than any other disease in eighteenth-century Philadelphia.
[76] Cofactors are factors that must join with one another to produce a given result. See Rothenberg et al, 2000, p. 127.
[77] Elizabeth's fourth child, William, developed pulmonary tuberculosis in 1788, when he was twenty-one years old. While he lived for fifty-four years, he was an invalid for much of that time. Drinker, 1937, p. 15.
[78] Grob, 2002, p. 110-2.
[79] Even these are not entirely certain. Syphilis was usually reported as consumption. Typhus and typhoid fever were often confused. Bilious fever could also be typhus or malaria. For a discussion, see Klepp, 1989, *Philadelphia in Transition*, p 305.
[80] Klepp, 1989, *Philadelphia in Transition*, p. 279-80.
[81] Gehlback, 2005, p. 124.

[82] Crane, 1991, p. 537-9. Excerpt from the *Diary of Elizabeth Drinker*, December 31, 1793.
[83] Klepp, 1989, *Philadelphia in Transition*, p. 285. See Table 4.11. The Christ Church Bills of Mortality for 1751, for example, list 26 deaths from "consumption" and 9 deaths from "pleurisy." See Klepp, 1991, p. 63.
[84] Klepp, 1994, p. 478.
[85] Tennent, 1742, p. 3. Quoted in Duffy, 1953, *Epidemics*, p. 200.
[86] Kalm, 1772; reprint, 1987, p. 32; Holmberg, 1990, p. 1229.
[87] The entries in Elizabeth Drinker's diary for 1786 were irregular and the volumes for 1787 and 1788 were burned. As a result, there is no systematic record of the first years of William's illness.
[88] Patterson, 1986, p. 11-28; Caulfield, 1950, p. 21-52; Rush, 1815, "An Account of the Influenza, as it Appeared in Philadelphia in the Autumn of 1789, in the Spring of 1790, and the Winter of 1791", Vol. 2, p. 265-273; Grob, 2002, p. 88.
[89] Whenever tuberculosis has become endemic, most children are exposed to the disease. Some of those individuals escaping the disease during childhood will develop reactivated infection later, usually during adolescence or early adulthood. Most adult cases of tuberculosis develop from reactivated infections. Sometimes reactivated infection does not develop until old age. Once reactivated, the disease has the potential to develop into active, contagious pulmonary tuberculosis. See Garay, 1996, p. 373-412.
[90] Klepp, 1989, *Philadelphia in Transitions*, p. 269.
[91] Rush, 1815, "Thoughts upon the Cause and Cure of Pulmonary Consumption", Vol. 2, p. 37-8.
[92] Over thirty species of genus the *Mycobacterium* have been identified, more than fifteen of which can cause disorders similar, but not identical, to tuberculosis. Human disease typically is caused by members of the species *M. tuberculosis*. In addition, mycobacteria can cause disease in a wide variety of animals, including birds, fish, rodents, elephants, and cattle. Of its animal forms, only bovine tuberculosis can infect people. For a discussion, see Johnston, 2003, "Tuberculosis," p. 337.
[93] Drinker, 1937, p. 76.
[94] Holmberg, 1990, p. 1228-32; Webster, 1799, Vol. 2, p. 209.
[95] Johnston, 2003, "Tuberculosis," p. 337.
[96] A communicable disease is any disease transmitted from one person or animal to another, either directly through body discharges or indirectly through substances or objects. Communicable diseases include those caused by viruses, bacteria, fungi and parasites. An infectious disease is a disease caused by a pathogenic agent, such as a bacterium or virus. The disease may or may not be contagious. See Rothenberg et al, 2000, p. 130 for a definition of communicable disease; p. 287 for a definition of infectious disease.
[97] Gehlback, 2005, p. 136.
[98] Johnston, 2003, "Tuberculosis," p. 338.
[99] Drinker, 1937, p. 67.
[100] Crane, 1991, p. 449. Excerpt from the *Diary of Elizabeth Drinker*, July 21, 1789.
[101] Crane, 1991, p. 537-8. Excerpt from the *Diary of Elizabeth Drinker*, December 31, 1793.
[102] Drinker, 1937, p. 84.
[103] Klepp, 1989, *Philadelphia in Transition*, p. 286-7.
[104] Drinker, 1937, p. 84-9.
[105] Klepp, 1989, *Philadelphia in Transition*, p. 287.
[106] Barnes, 2005, p. 163.
[107] Klepp, 1989, *Philadelphia in Transition*, p. 287-8.
[108] The incidence of tuberculosis among humans increased in proportion to the density of the population. Furthermore, the sharing of living quarters between humans and animals was an especially important facet of the evolution of human tuberculosis. See Torrey and Yolken, 2005, p. 43.
[109] *Mycobacterium tuberculosis* can survive for a time outside of the host, protected by its complex fatty (lipid) capsule. Depending on the temperature, mycobacteria in sputum can survive for several months in shaded areas. Direct sunlight destroys the microbes quickly, but they can survive within fine droplets and dust particles for several days away from sunlight, and they have been known to survive in sewage for up to two hundred days. The ideal setting outside of the host for these microbes lies within dark, manmade structures with poor sanitation and poor ventilation for both human beings

and cattle. The greater the concentration of humans and animals within these settings, the greater chance for maintaining the disease within both groups. See Barnes, 2005, p. 160-1.

[110] We now know that although tuberculosis represents a contagious disease, the course of infection within the individual depends on genetics. Studies with mice indicate that a specific gene regulates the responses to infection by the mycobacteria. Without this gene, the macrophages (cells that engulf and ingest invading microorganisms and cell debris) fail to signal a certain type of T-cell. Yet, back-up inflammatory responses by the immune system can still contain the infection unless it is impaired by other disease or poor nutrition. Immune systems with impaired T-cell function, such as those with HIV infection, cannot respond to signals from infected macrophages, and the disease takes over. How the immune system reacts, or fails to react, will determine the course of the disease. See Schurr and Skamene, 1996, p. 247-58.

[111] Aufderheide and Rodríguez-Martin, 1998, p. 125.

[112] Klepp, 1989, *Philadelphia in Transition*, p. 287.

[113] Johnston, 1993, "Tuberculosis," p. 1060.

[114] Aufderheide and Rodríguez-Martin, 1998, p. 126.

[115] Grob, 2002, p. 53.

[116] The ileum is the distal portion of the small intestine. See Rothenberg et al, 2000, p. 279.

[117] Poirier and Feder, 2001, p. 95.

[118] LeBaron and Taylor, 2003, p. 345.

[119] Many examples can be cited to demonstrate that, in appropriate circumstances, a single human carrier may be responsible for large typhoid epidemics. Of these carriers, perhaps the most famous was "Typhoid Mary." She was a professional cook who ultimately was confirmed to be a typhoid carrier. Over the course of a decade, inexplicable typhoid outbreaks had occurred in seven of the eight families for whom Mary Mallon had worked. Her stool samples persistently revealed typhoid bacilli, and she was warned not to pursue her cooking career. When a subsequent epidemic was attributed to her continuing occupation, she was forcibly isolated for the remainder of her life. See Aufderheide and Rodríguez-Martin, 1998, p. 191.

[120] Crane, 1991, p. 21. Excerpt from the *Diary of Elizabeth Drinker*, May 31, 1759.

[121] Rush, 1815, "An Inquiry into the Comparative State of Medicine in Philadelphia between the Years 1760 and 1766, and the Year 1809," Vol. 4, p. 230.

[122] Wolman, 1974, p. 238.

[123] McCarthy, 1987, p. 5.

[124] Cosans, 1975, "Franklin Court Report," p. 1. Report prepared for Independence National Historic Park, Philadelphia. See McCarthy, Cosans-Zebooker and Henry, 1985, "Philadelphia Privies and Their Fills: A Consideration of Their Interpretive Value." Paper presented at the Middle Atlantic Archaeology Conference, Rehoboth Beach, Delaware.

[125] Cosans, 1975, "Franklin Court Report," p. 1. Report prepared for Independence National Historic Park, Philadelphia. See McCarthy, Cosans-Zebooker and Henry, 1985, "Philadelphia Privies and Their Fills: A Consideration of Their Interpretive Value." Paper presented at the Middle Atlantic Archaeology Conference, Rehoboth Beach, Delaware.

[126] Cotter, Roberts and Parrington, 1993, p. 46.

[127] Cotter, Roberts and Parrington, 1993, p. 164. See Figure 4.12.

[128] Liggett, 1971, "The Archaeology of Philadelphia: The Dock and Budd's Row." Paper presented at the Annual Meeting of the Society of Architectural Historians, Chicago.

# CHAPTER 7

# CONCLUSION

The emergence and spread of microbial threats are driven by a complex set of factors, the convergence of which can lead to consequences of disease much greater than any single factor might suggest.[1] The urban environment of Philadelphia contained the epidemiological factors necessary for the growth and propagation of a wide variety of disease agents, while the social, demographic and behavioral characteristics of the people of the city provided the opportunity for "new" diseases to appear. This dissertation examines the multiple factors that influenced the pattern and distribution of infectious disease in Philadelphia between the years 1690 and 1807. What emerges from this study is a complex picture of a city undergoing rapid cultural and epidemiological changes. Large-scale immigration supplied a susceptible population group, as international trade, densely packed streets, unsanitary living conditions, a contaminated water supply, and a hot and humid summer climate combined to create ideal circumstances for the proliferation of both pathogen and vector. These factors combined to set the stage for the many public health crises that plagued Philadelphia for more than one hundred years.

René Dubos argued that in order for disease to affect many persons in a community at the same time there must be an imbalance in the factors that form the *total environment* – one's physical, biological, psychological, cultural, political, socioeconomic and historical world.[2] Absent this imbalance, these factors would otherwise tend to ecological equilibrium. Philadelphia was undergoing considerable transformations through much of the eighteenth century. Within eighty years of its founding, a small village on the banks of the Delaware River became the largest city in the American colonies, and capital of a new nation. Its population grew from 4,000 in 1700 to over 40,000 in 1775. Much of this growth can be attributed to a constant flow of immigrants from Europe, but a rural to urban demographic shift was also taking place in and around Philadelphia, dramatically increasing the numbers of susceptible people living in the city. The destabilized environment that resulted from the massive influx of infectious and susceptible people to the region, in combination with standing water, poor hygiene, malnutrition,[3] crowded houses, and the urbanization of the physical landscape of the city favored the transmission of many diseases.

Historical inquiry brings a valuable perspective to the understanding of disease emergence by focusing on "the consequences of human actions, and the conditions that cause or permit certain developments."[4] It can, however, focus too narrowly on the purely human round of existence – who decided what for whom, how, why, and what then. Biological inquiry, on the other hand, often views causation as driven by natural processes alone, neglecting the critical role of human

agency. Consider, for example, the manner in which tuberculosis is spread in a given population. Because it is a disease acquired almost exclusively by the respiratory route, infection requires exposure to someone actively transmitting bacteria. The disease is not highly communicable, however, so transmission is most likely when there is prolonged, close contact between individuals – most typically in households. By focusing too closely on the biological processes of transmission, the cultural factors are obscured, and our understanding of the disease becomes muddled. Conversely, the human role in causal processes often is inflated when the relationship of human decision and action to its surrounding environment is blurred or omitted altogether.

Even when the relationship is addressed as one of agency and structure, conceptualization may still be limited by an anthropocentric bias if humans are thought to *act within* various frameworks. Still, where human health is concerned, the boundaries are not so easily delineated. It is unclear, for instance, if the mosquito is simply part of the environment – a structural component of human existence – or if it has the ability to act and to produce effect that we associate with agency.[5] Consequently, it becomes difficult to know who or what were the "agents of disease" in eighteenth-century Philadelphia. *Mycobacterium tuberculosis* certainly was a causal factor in the existence of tuberculosis in the city, but so too were crowded homes, poverty, and cultural attitudes that encouraged sharing a bed with a sick family member. In order to understand the complexities of these relationships, we need to move away from tradition and think about historical processes ecologically,

using the broadest possible formulation of causation. This type of thinking allows us to understand how humans *interact with* rather than *acting within* an ever-changing environment – one that is fully capable of responding to insult in like measure. William McNeill has put the matter bluntly: "We will never escape the ecosystem and the limits of the ecosystem. Whether we like it or not, we are caught in the food chain, eating and being eaten. It is one of the conditions of life."[6]

The concept of causation becomes increasingly complex as we learn more about disease. Consider, for example, malaria and its transmission in Philadelphia. Often multiple factors operated simultaneously and at different levels in order to produce this disease in humans. While some were more directly related than others, several of the elements were absolutely necessary for transmission to occur. There would be no illness, for example, without the parasite *Plasmodium* in the bloodstream. At the same time, without *Anopheles* mosquitoes, there would be no *Plasmodium* in the bloodstream, and without standing water for breeding and temperatures warm enough to support procreation, there would be no *Anopheles* mosquito. In turn, without human reservoirs of parasites, there would be no source for a large supply of the *Plasmodium*. And so the cycle continued, with extensive interlocking components operating throughout the entire process.[7]

Certain characteristics of the environment – though less obviously causal, were also important in the transmission of malaria in Philadelphia. Several connect the occurrence of fever with environmental changes that accompanied the city's

growth and expansion. It was known, for example, that clearing the forests for firewood resulted in the creation of more marshy ground around the city.[8] It was only after clearing began, the trees felled, houses built, immigrants arriving, and the trappings of urbanization begun that life became unhealthy. It was at this point in the city's development when "the ague" appeared.[9] Once Philadelphia passed through this phase and into a more stable one, the disease leveled off and became endemic in the population. A nineteenth-century observer noted that "the first breaking up of the soil appears, from a variety of observation, scattered through our topographical descriptions, to be frequently followed by autumnal fever; and, on the other hand, long-continued cultivation is accompanied by diminution of that disease."[10] The pattern was so commonly observed that people accepted the predicted fluctuations in health as part of the cost of settlement.

The sequential changes in the landscape had an effect on the transmission of malaria as well, by directly influencing the *Anopheles* mosquito. Before the land was cleared, the deeply shaded woodlands were inhospitable for breeding. *Anopheles* prefers to lay its eggs in water that receives direct sunlight. Once the trees were gone, existing ponds and pools of water became exposed. In addition, as the land became more urbanized, new ponds were formed, and water collected in depressions in the landscape created by clearing rocks and stumps. This activity created new habitat for both humans and insects. This pattern continued until swampy land was drained in order to provide additional space for housing, diminishing the mosquito-breeding habitat.[11] Also, mosquitoes may have actually preferred pigs and cattle to humans as

a source of blood meals, and increasing numbers of livestock in the city may have helped to lessen the occurrence of the disease.[12] Human blood contains less of the amino acid *isoleucine* than does the blood of other animals. This amino acid is necessary for protein synthesis in egg production within the female mosquito. Smaller amounts of *isoleucine* lead to smaller numbers of eggs.[13] Steady supplies of human blood would have to be available on a regular basis to make up for fewer eggs produced with each blood meal, and even then, most mosquitoes would still prefer animal blood if it were available.[14]

Changing characteristics of the host also played a significant role in the transmission of malaria. During active periods of human migration, for example, the disease tended to spread. Individuals with chronic plasmodial infections carried their parasites with them when they moved to new communities, and susceptible newcomers became fresh hosts. The greater the movement of people from place to place, the more malaria flourished. As communities stabilized, and the population became immune, the frequency of disease declined. With the establishment of a settled community came better housing and improved nutrition. Tighter houses meant less access for mosquitoes, and a better nourished populace was better equipped to develop resistance to the disease.[15]

Miasmatists like Benjamin Rush, and those who followed him, intimated an understanding of the complexities of causal relationships. Rush attributed the growing incidence of malaria in Pennsylvania to "the establishment and increase of

mill-ponds" and to "the clearing of woodlands without draining and cultivating them."[16] He also noted that the removal of trees between the Schuylkill and Delaware Rivers resulted in an increase in fevers, and that as needed grist mills spread throughout the colony, fever invariably accompanied them. Fevers that were once largely confined to the banks of the Delaware River now ventured eight to ten miles inland. Although Rush correctly surmised the association between fever and stagnant water, he did not understand the role the mosquito played in the transmission of malaria. He advocated a program of filth removal, and believed that this would eliminate miasma, commonly assumed to be the cause of these fevers.[17]

These many "secondary" elements in the transmission of malaria demonstrate a critical property of a causal relationship; the elimination of any one of them may interrupt the transmission of disease. The elimination of the parasite, the mosquito, the mosquito's breeding habitat, or infected hosts upon whom mosquitoes feed will break the transmission of the disease. Understanding the intricate pattern of malaria transmission, and specifically identifying characteristics that are critical in the causal path, do more than simply satisfy an etiologic[18] curiosity. They suggest opportunities for intervention. Thoughts about preventing *mal-aria* existed long before Benjamin Rush advocated street cleaning and the elimination of stagnant ponds of water.[19] Hippocrates' Greeks appreciated that avoiding marshy areas and avoiding exposure to the evening air lessened one's chances of contracting fever.[20]

Opportunities to break the cycle of malaria transmission came with understanding of the interrelationships between the human host, the insect vector, the parasite, and the environment. The decline of autumnal fever, however, occurred well in advance of any systematic efforts to eradicate it. Long before twentieth-century initiatives that aggressively attacked the *Anopheles* by draining marshes, pouring suffocating oil on ponds, and lavishing DDT, rates of malaria were falling. While reasons for the decline are not certain, it appears they relate to changes in the environment. Malaria was part of a changing ecology. Conditions no longer favored the *Anopheles* or her*Plasmodia* parasites. The stabilization of population migration into the city, mature farming practices that drained land for cultivation and brought livestock close to human habitation to provide an alternative food source for mosquitoes, and a rising prosperity that brought improved housing and better nourishment all contributed to the eradication of malaria both from Philadelphia and the United States.[21]

This kind of multicausal thinking is particularly useful in historical inquiry. It sheds light on historical meanings more readily than it helps to prevent disease, however. For epidemiologists, explanatory models carry a predictive as well as an analytical function, and those models have always been mathematical. Interlocking ecological systems, however, are mediated by social factors and so hinder attempts at explanation by ecologists.[22] The models inspire confidence to the extent that all the relevant variables are equally quantifiable, but the web of causation necessarily includes variables that by their very nature cannot be expressed in numbers since they

are *relationships* and not *things*.[23] Historical inquiry, on the other hand, need not be mathematically exact in order to draw important insights, and causal factors can be considered without reducing each to numerical values. As was seen in the discussion of malaria transmission, when the distinction between cause and effect blurs, variables begin to lose their independent or dependent aspects, and the interactive nature of forces becomes clearer. As such, the tools of the health sciences, unassisted by the historical imagination, fall short in their efforts to explain the dynamics of health. On the other hand, historical inquiry needs these tools to interpret the significance of health in the history of populations.[24] The complementary nature of these two processes is quite evident in this analysis of Philadelphia's urban health crises.

The complexity of cause and consequence dominates the history of public health in eighteenth-century Philadelphia. The fundamental role of culture, social, economic and political conditions in modulating the ecological opportunities for infectious diseases is a dominant theme running through this story of the interplay between human ecology and the microbial world. A debilitated population, rampant population growth, massive immigration, a densely packed urban environment, and a severe lack of public sanitation combined to create an almost constant public health threat in this city throughout much of the eighteenth century.

No issue could be a more fundamental measure of sustainability than public health, and the increasing emergence and reemergence of infectious diseases globally

is possibly the world's most challenging public health problem today. Yet this problem is incomprehensible without a vastly broadened research perspective, if not an entirely new paradigm.[25] The study of public health and epidemiology has been tracing outbreaks of infectious disease back to their point of origin for a long time, but the scale of the approach is widening, and a series of new strategies to study complex disease dynamics are being adopted. For example, there is a strong zoonotic skew to emerging infectious diseases in humans. HIV/AIDS and influenza H5N1, for example, have wildlife reservoir hosts, while others cause outbreaks with high case fatality rates and have neither vaccine nor cure. In classical epidemiology, outbreaks of these diseases would be traced back to their origin in wildlife, and studies of human contact with wildlife undertaken. A socio-ecological approach would enhance this analysis, however. SARS, for example, is a disease which has recently been identified as originating in *Rhinolophus* spp. bats and emerging via the wildlife trade in China.[26] Understanding the process by which SARS emerged may ultimately involve studying the expansion of wildlife trade in China to determine the threshold levels that allowed sufficient contact between bats, civets, and humans to cause pathogen spill-over. It may also involve studying the anthropogenic pressures on these bats: if bats are over-collected and populations thinned, how does this affect transmission dynamics within the wildlife host, pathogen prevalence and, therefore, risk to people? The challenge to researchers here is to break down disciplinary divides between, for example, medicine and ecology; virology and wildlife biology, and sociology and epidemiology to better understand the combined ecological and social dynamics at play.[27]

This ambitious goal will not be reached easily and will require science and education initiatives that cross disciplinary as well as institutional, societal, and cultural boundaries.[28] Historians can contribute to this endeavor by studying disease in past populations where the "global" scope is smaller, the rhythm of life is slower and the variables influencing the emergence of disease are fewer. Epidemic and endemic disease on the scale experienced by Philadelphia are metaphors for a human society out of harmony with itself and its enveloping environment. Clearly, by appreciating the complex dynamic between social, cultural and ecological processes in the emergence of disease in this eighteenth-century city, we can potentially gain insights into the underlying causes of the recent upsurge in emerging infectious diseases today.

Although it can be argued that all human action is in some sense an intervention into ongoing processes, the intricacy of multiple and shared interaction is such that while the results are not random, neither are they easily predictable or controllable. Consequences are often unexpected, and as this history has shown, they are usually unintended as well.[29] If a practical lesson emerges from the Philadelphia experience for policy makers today, it is that disease will result from actions and initiatives that disturb equilibrium in the total environment. Changes in human culture, technology and environmental incursions nearly always have consequences for health and disease. The continuing interplay between human culture and the microbial world may be an ancient narrative, but today this plot is growing increasingly complex. Antibiotic over-use, increased human mobility, long-distance

trade, urbanization, expanding numbers of refugees, and the exacerbation of poverty in inner-city communities all have great consequences for infectious diseases. While those who wanted to clear the forests to expand Philadelphia can be excused for their unawareness of ecological principles and probable results, we have no such excuse today. A keen awareness and understanding of the processes by which interventions yield epidemiological consequences should guide our policy makers to proceed with increased caution, insight and wisdom.

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[1] Genetic and biological factors allow microbes to adapt and change, and can make people more or less susceptible to infections. Changes in the physical environment can impact on the ecology of vectors and animal reservoirs, the transmissibility of microbes, and the activities of humans that expose them to certain threats. Human behavior, both individual and collective, is perhaps the most complex factor in the emergence of disease. Emergence is especially complicated by social, political, and economic factors, including the development of cities, the disruption of global ecosystems, the expansion of international trade and commerce, and poverty. See Smolinski, Hamburg and Lederberg, 2003, "Executive Summary," p. 2.

[2] Dubos, 1959, p. 110-1.

[3] Malnutrition has long been known to play a role in susceptibility to death from diarrhea, respiratory infection and malaria. Not as well understood are the roles of famine, war, crowding, urbanization and population growth. See Smolinski, Hamburg and Lederberg, 2003, p. 220.

[4] Morse, 1992, p. 38.

[5] This discussion is taken from De Bevoise, 1995, p. 186.

[6] McNeill, 1993, p. 36.

[7] Gehlbach, 2005, p. 86.

[8] Klepp, 1989, *Philadelphia in Transition*, p. 226.

[9] Benjamin Rush observed that "families enjoy good health, for many years, in the swamps of Delaware and North Carolina, while they are in their natural state, but that sickness always follows the action of the rays of the sun upon the moist surface of the earth, after they are cleared. For this reason, the cultivation of a country should always follow the cutting down of its timber, in order to prevent the new ground becoming, by its exhalations, a source of disease." See Rush, 1815, "An Inquiry into the Various Sources of the Usual Forms of Summer and Autumnal Disease, in the United States, and the Means of Preventing Them," Vol. 4, p. 136.

[10] Drake and Levine, 1964, p. 710.

[11] Gehlbach, 2005, p. 87.

[12] Malaria has been viewed in the context of stable or unstable transmission, reflecting in part attributes of *Anopheles* species that affect their *vectorial capacity*. These include density, longevity, tendency to feed on humans, and duration of the extrinsic incubation period of the parasite in the vector. *Stable malaria* is most often associated with *P. falciparum* infection in highly endemic settings. It is characterized by low fluctuations in parasite incidence in human and vector populations, high prevalence, and high seroprevalence for antibodies. Epidemics are unlikely under these conditions, even though transmission continues at high rates. In such settings, vectors tend to be highly anthropophagic, exhibit greater longevity, and have relatively low, stable densities but still exhibit considerable seasonal variation. *Unstable malaria* tends to be associated with *P. vivax* infections in endemic settings of high fluctuations in disease incidence. Vectors tend to be zoophagic, have seasonally profound variation in population densities, have low or no detectable field infection rates, and may have shorter longevity than do those in stable malaria settings. Epidemics can occur in

conditions of unstable malaria if environmental changes favor increased vector-human contact, e.g., during civil strife, following water projects such as dams or irrigation schemes, or when a new vector is introduced into an area. See Mullen and Durden, 2002, p. 244.
[13] Edman, 1991, p. 8-9.
[14] Barnes, 2005, p. 68.
[15] Gehlbach, 2005, p. 89.
[16] Rush, 1786, "An Inquiry into the Causes and Increase of Bilious and Intermitting Fevers in Pennsylvania, with Hints for Preventing Them," paper read before the American Philosophical Society on December 16, 1785.
[17] Rush, 1815, "An Account of the Climate of Pennsylvania, and its Influence on the Human Body," Vol. 2, p. 25.
[18] Etiology is the study of the causes of disease. See Rothenberg et al, 2000, p. 201.
[19] Rush, 1815, "An Inquiry into the Various Sources of the Usual Forms of Summer and Autumnal Disease, in the United States, and the Means of Preventing Them," Vol., 4, p. 136.
[20] Gehlbach, 2005, p. 88.
[21] Gehlbach, 2005, p. 88-9.
[22] This point raises the basic question of whether current epidemiological models are capable of translation into truly effective preventative action. The science of epidemiology encounters serious theoretical problems somewhere between the web of causation and the mathematical model, not to mention the practical problems of implementation between model and disease prevention. See Enzenberger. 1974, p. 3-31.
[23] De Bevoise, 1995, p. 189; Enzenberger, 1974, p. 3-31.
[24] De Bevoise, 1995, p. 188-9; Enzenberger, 1974, p. 3-31.
[25] Wilcox and Colwell, 2005, p. 254.
[26] Li et al, 2005, p. 676-9.
[27] Daszak, 2005, p. 239.
[28] See Kaneshiro et al, 2005, p. 349-60.
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New York Historical Society

Cadwallader Colden Letter Collection

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