Mapping the Miasma: Air, Health, and Place in Early Medical Mapping

Medical mapping is broadly assumed to have been a nineteenth century reaction both to the appearance of cholera and the social consciousness of principally British reformers. It is however older, more embedded in the scientific enterprise than the social critique, and in the end, more central to both than researchers typically recognize. This paper argues that medical mapping was from its start in the late 1600s principally a tool for the self-conscious testing of spatial propositions, arguing a relationship between health and place, and between the locus of specific disease incidence and suspected sites of local infectious generation. Through the nineteenth century the resulting work—social and medical—typically advanced a miasmatic theory that argued that infectious diseases were generated spontaneously and diffused naturally through the air. This paper reviews the history of medical cartography as a scientific enterprise in the age of miasma, and the importance of this work to social reformers as an outcome rather than a principal impetus to mapping as a critical tool.

Keywords: Edmond Chadwick, history science, history medicine, medical mapping, miasma.

INTRODUCTION

Medical mapping, and the disease topography it promotes, developed neither as an innovative response to cholera’s invasion of Europe in the 1830s nor the social reformer’s desire to document “the appalling living conditions of the poor” (Robinson, 1982, 156). Rather, medical mapping began in the seventeenth century as a means of administering health protection policies in the face of epidemic disease (Arrieta, 1694), and, in the eighteenth century, became a tool of disease topography (Seaman, 1796) and taxonomy (Finke, 1792). Without losing its administrative utility, medical mapping was from the start a tool for the self-conscious testing of spatial propositions, arguing a relationship between health and place, and between the locus of specific disease incidence and suspected sites of local infectious generation. This work typically advanced a miasmatic theory that argued that infectious diseases were generated spontaneously and diffused naturally through the air.

From at least the seventeenth century onwards, therefore, medical maps served not simply to illustrate a paper or picture a situation but instead to graphically prove an argument. Some sought to prove the general thesis that bad airs caused disease, or alternately, that disease was water rather than airborne. Some argued that a specific disease was autochthonous while others insisted the same disease was, in fact, exogenous. In every case, however, maps carried the burden of proof of spatial arguments about either disease in general or a specific disease itself.
While new to medical mapping, and to medical history (Koch, 2005), this argument is common across the broader discourse of mapping. It therefore seeks to introduce to medicine an understanding of mapping as a critical tool of thinking that others have brought to geology (Rudwick, 1976), biology (Camerini (1993), and the broader analysis of spatial phenomena in general (Wood, 1995). The result serves distinct but related functions. First, it adds depth to a limited, largely descriptive literature on historical medical mapping and its relation to medical science. It does this by simultaneously drawing upon and reconsidering the work of earlier researchers like Jarcho (1970), Stevenson (1965), and Robinson (1982), whose individual reviews of historical medical maps were written without attention to the disease theories that earlier researchers argued in their mapping. Secondly, this review emphasizes the degree to which early disease investigators were also social theorists arguing in their maps for practical, sanitary responses to epidemic outbreaks in the evolving, mercantile and industrial city.

The argument also serves to remind contemporary researchers, medical and cartographic, of the indivisible relations among disease theory, the methods by which disease incidence are studied, and the technologies of research and reportage that are crucial to theoretical discussion and practical application. Medical mapping stands in this telling as one critical element in a complex mangle of practice that is political, social, scientific, and technological at once. It is, in Pickering’s phrase, “the emergently intertwined delineation and reconfiguration of mechanic captures and human intentions, practices, and so on” (Pickering, 1995, 23). In this “mangle” early epidemiology, medical mapping, medical geography, and public health are located within the greater context of the history of science, its technologies of thought and production. What results is a disease ecologic perspective in which viral and bacterial incidence are considered from the start within social and physical contexts.

The consideration of a collection of chronologically ordered medical maps in the context of the evolution of the disease theories in whose service the maps were made results in a general paradigm shift. Maps are transformed from ephemeral additions, illustrations grafted to scientific arguments, into integral instruments of the science that is argued. The necessity for this shift is evident in the manner in which all health disciplines typically date their origins from the work of John Snow (Snow, 1849b, 1854, 1855) whose maps and writings on cholera are seen as the event after which the individual disciplines entered a modern analytic period (Richardson, 1936; Vinten-Johansen et al., 2003). That perspective, this paper argues, begins the tale after it was well begun.

Few examples of seventeenth century medical mapping have survived into the modern era. While mapping for specialized purposes like navigation was rigorously pursued in the 1600s, mapping was an expensive, specialized discipline whose application to less productive, scientific enterprise was necessarily limited. Nor were the technologies of the day, relatively crude in the presentation of locational attributes, easily adapted to medical studies. When produced, the number of private and public libraries capable of archiving the maps were insufficient to secure their survival. Most of the medical maps that were made in this period have therefore been lost, surviving only in notes about their making.

In 1792, for example, Leonhard Ludwig Finke drew a “Nosological Map of the Word” for his three-volume Versuch einer allgemeinen medicinisch-praktischen Geographie. An abridged translation of the full title is Notes on General Practical Medical Geography Dealing with the History of Medical
An ambitious work, it embodied Finke’s attempt to describe a broad topography relating taxonomy of observed diseases and the pharmacology of their treatment to the distribution of peoples affected by those diseases (Barrett, 2000b). This may have been the first use of the phrase “medical geography,” one advanced by Finke in this way: “When one brings together all which is worth knowing with regard to the medical status of any country, then no one can deny that such a work describes the name of a ‘medical geography’” (Howe, 1961, 9).

Unfortunately, the map, now lost, was not included with the printed text, Finke wrote, “because I was afraid that the work would become too expensive I do have it ready but have not sent it to be printed and I think it will not be printed soon” (Barrett, 2000, 917). The technology and expense of printed production was beyond his means, and presumably, that of other writers who excluded working from their own publications.

Fink’s map reflected a general theory of disease generation and diffusion as the geographically specific outcome of uniquely local conditions. The general Hippocratic and Galenic assumption was, as the German physician Hoffman put it in 1746, that diseases were the result of “a fixed and static cause essential to the country, and that they therefore remain in the country without change and variation for many years” (Howe, 1961, 8). Those fixed causes were typically assumed to be airborne, good and bad airs that promoted or inhibited specific infectious diseases in each locality at different times of the year.

An exception to disease as static and endemic were epidemic diseases that periodically swept across regions, nations, and the continent. Influenza was recurrent, and bubonic plague, while broadly endemic, in the seventeenth century was ferociously pandemic, striking across most of Europe in mid-century. The science of the day, steeped in the work of Hippocrates and Galen, considered plague a sickness in the “pestilential atmosphere,” whose precise nature while static was unknown; but practically, the dynamic nature of plague as a contagious disease was broadly recognized if not theoretically addressed. Its containment within effected areas was certainly a practical concern for local administrators even if not a subject of investigation by medical theorists.

For example, a late seventeenth century outbreak of plague in the province of Bari, Italy, was aggressively met with a sophisticated quarantine system administered by Filippo Arrieta, royal auditor for the province under the military governor of Bari and its neighboring provinces, Basilicata and Cappitanata. With his long, official report on containment efforts achieved through the deployment of troops, Arrieta included two detailed maps (Arrieta, 1694, see figures 2 and 3), one of which was described by Jarcho (1970) in his seminal paper on early medical mapping. Jarcho identified two cordons, one forty-five miles in length and composed of 360 barracks enclosing the towns of Monopoli, Conversano, and Castellano where the infection was present. Another, longer cordon separated the province from its neighbors, Cappitanata, Basilica, and Otranto. “Isolation was completed on the costal side by feluccas, two of which are shown on the map” (Jarcho, 1970, 132).

The symbols (churches, hospitals, trees, boats) were drawn in childish profile while the coastline and boundaries were rotated and oblique
Figure 2. Map of the plague in the province of Bari, Naples, 1690-1692, by Filippo Arrieta. The map shows areas most affected and the boundaries of a military quarantine imposed to prevent its spread to neighboring towns and to other provinces. Source: the New York Academy of Medicine.

(Wood, 1992, 174-178). Towns and cities were symbolized by drawings of simplified buildings, churches (with a cross) or hospitals (without) where the ill were typically taken for isolation and, if they did not survive, eventual burial. A “C” distinguished towns where the outbreak had occurred but was past from those “B” towns where the infection was active. Between the towns, trees symbolized, if not rural, then non-urban areas of the province.

Within the map can be seen a complex series of interlocking levels of containment designed to prevent the spread of the plague. The costal patrol symbolized by the feluccas (feluca di guardio) served to prevent shipping and maritime travel. On land, a dark wall with the repeated letter “A” along a “linea di circonvallazione” separated areas of active plague from western neighbors (noted by a “C”) where the plague had been active, and from southerly areas where it had yet to appear. On top of the wall are tents symbolizing the location of troops stationed at quarter-mile intervals to enforce the quarantine.

Within the area of active plague was another containment level ten miles in circumference marked with a “B” and described in the legend but not symbolized in the map. These individual districts separated towns free of plague but susceptible to it (Mola, Polignano, Fasno, etc.) from others in the district where plague was active or had recently been active. These inner cordons were each enforced by the deployment of 250 soldiers from fifty barracks, troops stationed in the towns at risk and charged with their protection. Finally, the map included a general, provincial cordon “D” separating Bari province from its neighbors. Here, too, the quarantine was enforced by troops whose tents are used to symbolize their presence. The military cost of deploying 1,750 troops stationed at 350 barracks was considerable, as Arrieta’s text makes clear.

The map distilled the details of a major military operation designed to halt or at least slow the spread of plague, one that reflected a sophisticated, practical understanding of what since Kendall (1957) has been understood as a pattern of hierarchical, spatial and network diffusion processes (for a discussion see Haggett, 2000, 26-29). Levels of containment separated the province from its neighbors (“D”), districts where plague had been
(“C”) from those where outbreaks were active, and at a third level insulated individual towns still plague free from those where it was evident (“B”). The whole presents a surprisingly modern approach to quarantine as a means of inhibiting the spread of actively epidemic disease.

This is clearer in a second map not discussed by Jarcho in which there are two walled areas, one around the north central area of the province where the plague was active (“D”) and a second (“C”), the legend makes clear, around Bari and nearby towns where the disease had earlier appeared (see figure 3). The broader containment area (“E”) is province wide, separating Bari from its provincial neighbors. Inherent in the map is a theory of plague as a progressive disease whose containment could only be achieved through the restriction of trade and citizen travel. Absent however was any effort to use this data to argue the nature of the disease, its precise agents or vectors. The luxury of scientific consideration was Finke’s, not Arrieta’s. In the summary of his troop deployments Arrieta presented, however, a thoroughly modern and extraordinarily sophisticated quarantine program active at the level of the city and the broad region which modern epidemiologists concerned with disease containment might applaud (Haggett, 2000, 99-103).

Figure 3. Map of plague containment zones in the province of Bari, Naples, 1690-1692. Tents represent troop deployments on provincial borders, zones of active plague and those where plague had already occurred. Source: New York Academy of Medicine.

By the late eighteenth century, administrative and scientific functions were combined in a body of progressively rigorous, self-consciously conceived studies designed to apply miasmatic theories to the study of specific disease outbreaks in urban environments. In this period a series of new diseases primarily effecting coastal ports of mercantile nations (typhoid and yellow fevers, for example) gave rise to the need both to understand their origins and limit their effect. This resulted in the articulation of a sanitarian thesis identifying odiferous areas of untreated urban waste as the source of specific epidemic outbreaks. These sanitarian maps advanced the simple proposition that odiferous areas of urban waste near docklands were proximate to the neighborhoods in which outbreaks occurred. This proximity implied causality. Therefore the remedy was to improve the sanitary facilities of cities, reducing the sources of the foul airs that generated disease. The argument was simultaneously miasmatic and sanitarian.
In the 1790s Valentine Seaman, a surgeon at New York Hospital and a pioneer in nursing education, investigated a yellow fever outbreak in his city as an example of a localized, miasmatic disorder (Stevenson, 1965). In this decade eastern coastal cities were struck repeatedly with a series of ferocious outbreaks that killed thousands of citizens. In 1793 approximately ten percent of Philadelphia’s population perished in a yellow fever outbreak. In 1798 more than 3,000 people died from the disease in a four-month period (Shannon, 1981, 221). Thousands more succumbed in cities like Baltimore and New York City.

Because these outbreaks were concentrated in the docklands of coastal port cities, some assumed the disease somehow was imported on ships. In the language of the day “anticontagionists” believed that these diseases were the manifestation of unseen microscopic “animalculae” that mysteriously traveled in ships, infecting individual travelers who then transmitted the illness to port populations after disembarkation. Disease progression was thus the result of mobile, undetectable poisonous agents specific to each condition and responsible for its spread. To limit the unseen agents’ diffusion it therefore would be necessary to increase ship and traveler quarantine at the expense of rapidly evolving trade.

Opposed to this theory of a peripatetic but undetectable agent was the miasmatic theory. Based on the theory of good and bad airs in the tradition of Hippocrates’s *Airs, Waters, Places*, “contagionists” argued that static and local, odiferous airs occurring were the breeding grounds and vectors of contagious illnesses. The theory included an explanation of disease generation and a therapeutic viewpoint that considered the location of each patient within his or her physical environment (Porter, 1997, 172). Absent microscopes of sufficient power, olfactory and visual senses were the primary tools used to investigate disease generation and diffusion.

Foul humors infected populations at two scales through the processes of “exhalation and contagion” (Shannon, 1981, 222). Exhalation was the product of the stench of rotting vegetables and waste inhaled at distances of 300 to 400 yards. Contagion was a more local effect occurring on a street or in a tenement building. At both scales the localized, static foul airs characterized an environment in which diseases spread spontaneously to nearby residents.

In an attempt to prove the contagionist argument, Valentine Seaman studied the yellow fever outbreak in New York City’s docklands. His results were reported in a fifty-two-page monograph (1796), an article in the then new journal, *Medical Repository* (1798), and another in a book on “bilious fevers” by Noah Webster (Seaman, 1796b). In each text Seaman argued the origin of the outbreak was the smell that arose from the city’s garbage and sewage that accumulated in the harbor area and that “no Yellow Fever can spread, but by the influence of putrid effluvia” (Seaman, 1798, 316). He mapped this argument in the *Medical Repository* article that remains the most frequently cited and perhaps most concise of his statements. Seaman’s maps splendidly distilled the miasmatic theory of disease contagion in his attempt to prove yellow fever was static, not dynamic; a creature of the foul smells generated by urban waste.

In his first map Seaman located ten fatal cases of yellow fever occurring in a 1797 outbreak in an area where “most of the patients infected with dangerous fevers, were either such as resided in the neighborhood of slips (which were or lately had been cleaning out) or whose employment led them to frequent such places” (Seaman, 1798, 317, see figure 4). The map included the index case of the outbreak, a seaman taken sick in East George Street who had recently arrived from South Carolina in the sloop *Polly* on which one crewman had died on the passage northward. Seaman
considered and then rejected the ship as a possible source of the infection. While it was possible, Seaman admitted, that the *Polly* seaman somehow brought the disease with him to New York it was clearly local conditions that gave the fever its deadly affect: “It may be, that a partial principle of death lurked in his [the seaman’s] system, during the whole time after the death of his comrade, and most likely, never would have seriously acted upon him, had he not immersed himself in this or some such like furry-fostering miasmata.”

The real culprit, Seaman concluded, was the urban environment itself, the fetid air in the vicinity of the dockland terminus of the Roosevelt Street drain (not shown on the map) into which city waste poured, “in addition to the other putrid matters that such places are always collecting.” Daily, ebb tide exposed perhaps eight hundred square yards of rotting, perishable materials (everything that a household or small business would throw out) and “putrid matters,” human and animal waste. The smell was foul and the proximity of this odiferous waste area to the yellow fever outbreak was plain to see.

In a second map whose focus was the urban effluvia Seaman continued the proposition that the proximity of the homes of persons with yellow fever and the location of odiferous sites of urban refuse were positively correlated (see figure 5). To do this he included an “S” to symbolize “slips, puddles, filth, and garbage,” and an “x” to locate areas of “convenience,” places he described as “being open and so contiguous to the Market, [that they had become] the common convenience to a multitude of people” (Seaman, 1798, 13). Wastes from these sites washed down the city’s streets to ferment on the tidal flatland of the docklands. What resulted was a map of odor sites that with the map of fever incidence, Seaman believed, proved a cause and effect relationship. Together, the maps demonstrated, Seaman argued, to “every unprejudiced mind that in the city there appears to be an intimate and inseparable connection between the prevalence of Yellow Fever and the existence of putrid effluvia” (Seaman, 1798, 324-25).
There was a relationship between the foul odors and the disease. The odiferous dockside area was also a breeding ground for mosquitoes “never before known, by the oldest inhabitants, to have been so numerous as at this season,” wrote Seaman, who assumed that the fouled, urban air spontaneously generated both yellow fever and a plague of mosquitoes: “The rise of putrid miasmata equally favor the generation of these insects.” The mosquitoes were not perceived as a disease vector carrying the unseen animalcule but as a secondary effect of the miasma that was the cause of the fever.

Seaman did not draw the maps he made. Rather, he drew or had drawn symbols of disease incidence and effluvial sites onto an existing copper-plate map of the city. The process of etching a copperplate was complex, expensive, and time consuming. Nor was there a need for a new plate to be made; in 1789 and again in the 1790s copperplate city maps were being commercially produced in New York City: “in the late eighteenth century the city of New York was frequently charted for the benefit of its citizens, its visitors, and its government” (Stevenson, 1965, 237).

While the commercial maps made Seaman’s maps possible, the limitations of copperplate frustrated his attempt at a more comprehensive graphic. He lamented, for example, “the want of proper marks to identify it [the disease] where it is slight” (Seaman, 1798, 317). Nor was he able to include on his map all the identified cases without diminishing the legibility of the map. Nevertheless, Seaman’s map admirably advanced his argument that the epicenter of the outbreak was proximate to foul odiferous airs generated whose foul stench the science of the day “knew” to be generative of disease. To prevent future outbreaks a program of urban waste treatment and control was the logical, necessary conclusion.

A generation later the technology of printing had improved sufficiently to permit better maps of an 1819 yellow fever outbreak in New York City. Accompanying a detailed study of that outbreak, Felix Pascalis mapped fatal incidents of yellow fever at the level of city blocks in a manner similar to early urban tax maps of residential location. Each death was numbered to reflect its order of occurrence. The result looks like a land parcel or taxation map. Here, too, mapping showed that mortality was concentrated in an area that was overflowing with “perishing and fermenting materials,” producing “an offensive smell and, no doubt also, deleterious miasmata” (Pascalis, 1819, 17).

Advances in mapping technology and production permitted Pascalis a denser level of case reportage than Seaman. Here, perhaps for the first time, a relatively accurate density of occurrence was used to argue about the locus of an infectious disease in relation to environmental contaminants. “It will be seen, by the annexed diagram, that in the vicinity of Old Slip, out of 57 cases, the enormous proportion of 34 or 35 originated from that single block . . . ten persons, out of the number of 83 sent to Fort Richmond, the greater part from that block, shortly after sickened with the malignant fever, and three of them died in the Marine Hospital” (Pascalis, 1819, 241, see figure 6).

Pascalis described but did not map the effluvial sites detailed in his text. The sheer density of cases, and the general description of nearby odiferous sites encouraged, however, the conclusion that “yellow fever is produced by impure and deleterious exhalations from putrid substances” (Pascalis, 1819, 17).

The relationship between the density of clustered cases and the proximate waste sites not only proved that the disease was “engendered by domestic causes,” foul airs that pervaded certain areas, but disproved, he...
hoped, the contagionist argument that yellow fever was “communicated by human contagion from foreign ports.” As the final clause of his monograph’s long title made clear, his work was self-consciously crafted with both goals in mind. His mapped study was undertaken “with a view of ascertaining, by comparative arguments, whether the distemper was engendered by domestic causes, or communicated by human contagion from foreign ports” (Pascalis, 1819).

Issues of disease theory and generation had very practical consequences. Miasmatists assumed that control of future outbreaks would require sanitarian attention to accumulations of waste washed from the city to the river’s edge, the assumed locus of disease generation and aerial diffusion. Unnecessary, however, would be any effort that might impede growing regional and international ship traffic through stricter quarantine procedures. And, of course, Pascalis was correct: The odiferous waste sites, breeding grounds for mosquitoes, were complicit in the yellow fever outbreak. If the miasmatic theory of disease missed the intervening vector it nonetheless caught a critical aspect of the epidemiological problem.

Epidemic cholera, also called “cholera morbus,” or more popularly, “Asiatic cholera,” became by the 1830s the epidemic disease on which many researchers focused. First identified in Calcutta in 1781-2, it was the cause of death of approximately 20,000 pilgrims at Hurdwar in 1783-4. The first outbreak among British troops occurred in Jessar, India, in August 1817 when 3,000 members of the 10,000-man British army then stationed in India under the Marquis of Hastings died of the disease (Morris, 1976, 23). In 1830, the disease had spread to St. Petersburg, Russia, and in the fall of 1831 arrived in Sunderland, England, where keelman William Sproat was the first of more than 50,000 English to die in the first pandemic (Morris, 1976, 11). Between the first and second pandemics, a number of researchers studied the 1831-33 epidemic, arguing in their work for a correspondence between the “good airs” of higher altitudes and the “bad airs” of the odiferous, densely inhabited riverbanks of British maritime cities.

An example is Thomas Shapter’s frontispiece map of The History of Cholera in 1832. Published on the eve of the second pandemic in 1849, the map “showing the location where the deaths caused by pestilential cholera occurred in 1832-34” sought to describe, in the words of a Lancet reviewer (Lancet, 1849, 317), a “city close, confined, badly drained, and still worse supplied with water” (see, Vinten-Johansen et al. 2003, 324, see figure 7). In its careful attention both to the incidence of the disease and the environment in which it proliferated, the map carried, for Shapter and his contemporaries, a powerful proof of the miasmatic theory of cholera’s generation and diffusion.

Despite its attention to water sources in the city, a focus that gained currency in the 1850s, Shapter’s map served primarily an airborne, miasmatic argument in several ways. Not the least of them was a descriptive function familiarizing late 1840s readers with 1830s Exeter: convalescent homes, burial grounds, soup kitchens, and sites for the disposal of the clothes of the infected, etc. are all marked. During the bubonic plague of the 1600s special burial pits for the victims of epidemics had been created; special locations for the cleansing of the clothes of the afflicted were instituted. In the cholera epidemic of 1831 these protocols returned, the evidence of them embedded in the map (see figure 8).

At another level the map was a self-conscious topography of the relationship between the city’s geography and the incidence of disease over time, one that linked bad air in lowland, riverside areas near effluvia sites with the most intense areas of disease incidence in the years 1832,
1833, and 1834. Deaths occurring in each year were distinguished in the map by both symbol shape and color. As well, mortality data was aggregated in the legend at the scale of the individual parishes, each parish symbolized in the map by a capital letter. Like Seaman’s and Pascalis’s, Shapter’s map again presented a miasmatic argument based on proximity. It differed in the depth of data, the extent of the mapped detail, and the level of aggregation. The resulting map identified “a few isolated spots in which a remarkable and undue amount of mortality took place” (Shapter, 224) in relatively low-lying areas of dense habitation near the river where effluvial build-up resulted in the odiferous, miasmatic airs Shapter blamed for the epidemic.

Richard Grainger’s 1849 Cholera Map of the Metropolis (Grainger, 1850, Appendix B) presented a similar argument at a different scale. Here altitude was related to location odiferous sites that correlated positively with cholera. The hypothesis was that an inverse correlation existed between increasing altitude and disease incidence. Inked by hand on an existing lithograph of Greater London’s parish jurisdictions, the map boldly displayed occurrence based on data reported by parish districts and the physical domain of the city’s political districts. Its focus was not the neighborhood but the whole metropolis (see figure 9). In this Grainger followed the 1830s initiatives of the “Paris School” (Porter, 1997, 406-8) medico-statisticians whose approach involved numerical and graphical analysis of ever more broadly constituted regional and national datasets (Jarcho, 1970, 1974).

On Grainger’s map three different densities of disease occurrence can be seen. Small numerals designating elevation above sea level are included in the map to support of the theory that generative, miasmatic airs tended to settle around low-lying riverbanks. And there was a correspon-

Figure 9. Grainger’s density map of the 1849 cholera epidemic in London showing intensity by political district and sub-district. Source: the College of Physicians of Philadelphia Library, Philadelphia, PA. (see page 91 for color version)
Incidence of cholera was highest in those areas nearest the Thames riverbank where elevation was lowest. Where cholera did occur at higher elevations, Grainger carefully mapped local circumstances that might have contributed to the anomaly. Thus in Islington (number 8) “Bad ventilation and no drainage” is written near a darkly colored, localized outbreak. And in Westminster, “over-crowding” was noted in the area of Fennings Buildings where a dense outbreak’s epicenter is surrounded by a pattern of moderate occurrence. “Open sewers” are mapped near an outbreak at Barrington Crescent in Lambeth (number 30), and “putrid water” near Lambeth Church sub-district (number 28). These were riverside areas into which the city’s sewage flowed and from which much of the city’s water was drawn. Famously odiferous into the 1860s, it was as much the overpowering stench of the Thames River as much as the health problems that might result from them that argued for the river’s rehabilitation through an expensive embankment (Porter, 1998).

While the general correspondence of altitude and relative health might be explained by “exhalation” it did not explain the typically uneven spatial distribution of disease in areas of equal altitude. Variation in disease rates in neighborhoods was assumed to result from the processes of “contagion,” the close quartered contact with foul airs in a single building or apartment. Social reformers concerned with the living conditions and the health status of lower class, working populations carefully described the correlation between disease incidence and economic status. Living conditions, resulting in fearfully dense housing without sufficient ventilation or sanitation, provided the precise environment for foul airs to generate within tenement apartments and between tenant neighbors, creating a demonstrably increased severity of disease.

Among those whose work advanced this argument was Edmund Chadwick, whose famous Report on the Sanitary Conditions of the Labouring Population (Chadwick: 1842, 160) offered a “forceful indictment of unsanitary living conditions in the industrial slums, as well as a severe criticism of physicians ignorant of the causes of contagion and of the moribund local health boards” (Melosi, 2000, 46). Well researched and well argued, the report included a host of tables (see figure 10) and “A Sanitary Map of the Town of Leeds.” (see figure 11)

On it cholera and other communicable diseases are located at the homes of the deceased. Statistics of healthier and less healthy populations are summarized in the map legend. Across the city the “less cleansed” areas are shaded a darker brown and these, not surprisingly, are areas largely inhabited by working rather than more moneyed families. Blue spots are hand inked onto the map to indicate “localities in which cholera prevailed,” while red spots are used to identify residences from which the victims of “contagious disease have been sent to the House of Recovery from 1834 to 1839.” Using the evolving statistical approaches of the day, the ratio of “good” (healthy) to “bad” streets by parish district was calculated based on deaths and births by area population. The message was clear: irrespective of altitude, increasing density of population correlated negatively with income, positively with mortality, and negatively with rate of birth. In the map, increasing density of population also correlated with the blue and red dots.

The choice of color schemes was unfortunate. Over time the map has faded and with it the distinction between shades of brown and red. On a different but related black and white map, Chadwick mapped the relationship between health and socioeconomic class in Bethal Green. Here the absence of color and the relative simplicity of the symbols promoted
Figure 11. Chadwick's map of Leeds in which increasing incidence of contagious disease is correlated graphically with declining income. Map courtesy of Wellcome Trust. (see page 92 for color version)

clarity (see figure 12). Again, disease incidence is located at the homes of decedents. Intensity of incidence correlates positively with the lower socioeconomic and densely settled neighborhoods. Taken for granted was that such neighborhoods, without adequate sanitation, also had fouler airs in streets and home, promoting disease generation by general exhalation and by inhalation at the household scale.

London physician and anesthesiologist John Snow argued a very different theory of cholera as a water rather than airborne disease. Snow believed that the disease was passed interpersonally through contact with the waste products of those who were already ill and through drinking contaminated water drawn from the polluted riverbanks of the industrializing city. As Snow put it: “The water works that supply the south of London take water from the Thames mostly at places near which the chief sewers run into it. Moreover, the wells in this part of London are very liable to be contaminated by the contents of cesspools . . . these are the chief sources of the high mortality on the south of the Thames, and where they are not in operation there has been comparative immunity from the disease” (Snow, 1849a, 749).

In the 1850s Snow advanced his argument through two famous studies. The larger and more ambitious South London study (Snow, 1855) was detailed and complex. Its mapping suffered from the limitations of a printing technology that muddied and made near unintelligible its colors (see figure 13). In a separate but related neighborhood-scale study Snow considered a cholera outbreak in the Broad Street, Soho, area near his home (Snow, 1855, 1855a). These most famous of nineteenth century medical maps failed, however, to convince Snow’s contemporaries of his waterborne thesis. “Is this evidence scientific?” the *Lancet* asked in an 1855 editorial (Vinten-Johansen et al, 2003, 344). “Is it in accordance with the experience of men who have studied the question without being blinded by theories?” The answer was no. Snow was respected as the leading authority on clinical anesthesiology but not for his work in this area. “The truth is, that the well whence Dr. Snow draws all sanitary truth is the main sewer. His *specus*, or den, is a drain. In riding his hobby very hard, he has
Chadwick’s map of Bethel Green correlating class and neighborhood in the incidence of disease. The map was part of a greater attempt to locate disease within specific physical and socioeconomic environments in which disease incidence was promoted or inhibited. Source: British Library.

Figure 13. Detail of John Snow’s map of Cholera in the Broad Street outbreak in 1854. Each bar represents one death in a topography that attempted to relate the water source ("Pump") to pattern of cases in the neighborhood outbreak. Source: College of Physicians of Philadelphia.

fallen down a gully-hole and has never been able to get out again" (Lancet, 1855). Far from being lauded as an exponent of modern research methods, many believed, in the words of the Lancet, that “Snow had deviated from his usual scientific practice. He had presented conclusions without experimental evidence or statistics to back them up.”

The problem was not that Snow advocated a new general theory of disease but that he argued a limited theory of a single disease—cholera—that questioned prevailing disease theory. In effect Snow, argued for a specific exception to the miasmatic theory without a theoretical justification that would explain that exception. As importantly, at least to skeptics, Snow’s data was ambiguous. His maps easily could be read as supporting a theory correlating a greater incidence of cholera in riverside parishes with the bad airs of lowland riverbanks where noxious waste collected. Edmund Parks, for example, argued that the centralized pattern of the outbreak centered on the Broad Street pump was exactly what one might expect if a noxious miasma was in fact the cause of the disease (exhalation) that then spread by contagion among the area’s habitations. Furthermore, Parks
pointed out, there were so many pumps in the area that no matter where the epidemic had its center one would surely be close by (Parks, 1855).

It is worth pausing to note parenthetically the importance of Snow’s mapped work to modern epidemiology (Rothman, 2002), medical cartography, medical geography, and public health (Vinten-Johansen et al. 2003, 392-399). While rejected in Snow’s day, his studies of both the Broad Street outbreak and the South London epidemic remain first cases in the teaching of each discipline’s approach to disease. In medical cartography this is especially true of the iconic Broad Street map (McLeod, 2000) whose presentation is graphically clear and whose focused database even today is easy to manipulate.

As importantly, it presents a clear example of a type of graphic “map thinking” (Brody et al. 2000) characterized by a topographic approach to a local area study of disease (Koch and Denike, 2004). Its simplicity makes the conclusion, to us, sufficiently obvious that it is virtually an advertisement for medical cartography advanced reflexively by a slew of twentieth century writers (Vinten-Johansen et al. 2003, 396-399; McLeod, 2000). Over the last 160 years, however, its appearance has been changed with the context of its presentation. It’s database has been truncated—fewer wells and fewer deaths presented—to advance graphic simplicity. The symbolization has been changed. Today it stands not simply as testimony to Snow’s work and thinking but to the manner in which datasets are manipulated—cartographically and statistically—in service of an author’s focus, theories, and personal attentions (Koch, 2004).

Over the next twenty years Snow’s special theory of cholera transmission was accommodated within a generally miasmatic theory of disease generation. In effect, the argument became a thesis in which at one scale, cholera was a miasmatic condition that, at another, was transported on ships between countries. At a third scale the argument became that the disease could also be transmitted interpersonally. All three scales were discussed and described at the end of the third pandemic, which began in the 1860s, at an 1874 an international congress on cholera held in Vienna.

At the end of the Congress the researchers agreed on a general set of “facts.” First, it was “unanimous affirmed ‘that the Asiatic cholera, susceptible of epidemic extension, is not developed spontaneously, except in India, and when it appears in other countries it is invariably by introduction from without” (Woodworth, 1874, 54). The assumption remained, however, that “the surrounding air is the principal vehicle of the generative agent of cholera; but the transmission of the malady by the atmosphere, in the immense majority of cases, is restricted to the close vicinity of the focus of emission. As to facts asserted of transportation to a distance of one or many miles, they are not conclusive” (Christie, 1876, 476). Finally, researchers also agreed “cholera can be transmitted by personal effects coming from an infected place, especially such as have served for the sick from cholera; and certain facts show that the disease can be carried to a distance by these effects ” (Woodworth, 1875, 47).

A number of studies presented at the Vienna conference considered evidence mapped at various scales based on data collected during the first pandemics and the one that began in the 1860s. Perhaps the greatest study of the third pandemic, one owing much to the conference itself, was a thousand page report, The Cholera Epidemic of 1873 in the United States, by army captain Dr. Ely McClellan. McClellan served under the U.S. Surgeon General, Dr. John M. Woodworth, who had been ordered by the U.S. Congress to investigate and then report on the U.S. epidemic. Woodworth assigned McClellan to investigate the progress of cholera as it marched
from New Orleans up the Mississippi through more than 10 states. To gather data McClellan, with the assistance of Dr. John C. Peters relied on local health officials in effected cities.

McClellan’s goal was to prove that cholera was introduced at the port of New Orleans and then spread up the Mississippi by riverboat and train. He favored the idea that cholera was transmitted interpersonally by the wastes of contaminated humans but remained open to a theory of contagion that assumed the close, dank air of overcrowded riverboat quarters was also complicit in the spread of the disease. With other members of the Vienna conference he believed the disease was endemic only in India and differed fundamentally from the “summer cholera,” or food poisoning, that often caused diarrheic disease in nineteenth century cities.

The report he prepared for Dr. Woodworth was extraordinarily map rich. It included maps from the Vienna conference drawn by British and French researchers who had mapped the progress of cholera internationally and nationally in its two earlier pandemics. With earlier U.S. maps of previous epidemics these maps provided the context in which McClellan located his study of the third pandemic’s epidemic progress. For each of the cities whose health experts submitted a report, McClellan either modified maps of disease incidence his respondents had submitted or personally drew (or had drawn) maps that together would argue a general pattern of diffusion. Together these maps presented a pattern of disease spread based on riverboat and train networks in the then western U.S. that resulted in localized outbreaks resulting from interpersonal contact with persons infected by exposure to river or secondarily rail traffic and commerce.

McClellan built a powerful argument based on the reports of local respondents. For example, the New Orleans report McClellan received from Dr. C. R. White, president of the Louisiana Board of Health, insisted that most Louisiana physicians “warrant the belief that it was not Asiatic cholera” that struck their state but only an unusually severe occurrence of endemic diarrhea, also called cholera. “The prevalence of cholera at the same period of 1873 may be viewed as the natural tendency of that portion of the year, exaggerated into serious, and deadly, and somewhat general disease, by the presence of local poison, engendered by filth and magnified by unusual meteorological conditions” (Woodworth, 1875, 101). In short, summer heat and rains combined with local filth to generate foul airs causing endemic summer diarrhea that in 1874 was unusual in its intensity but normal in its occurrence. White’s was an argument both Seaman and Pascalis would have understood and applauded.

McClellan first complimented the “admirable and exhaustive report of Dr. White,” and then rejected conclusively “the theory that the cholera epidemic of 1873 originated de novo at New Orleans.” He made his case on the back of White’s own data with a two-step mapped analysis that remains today a useful model of mapped, epidemiological thinking. He first located on a map of the city the cases that White identified, numbering them sequentially on the basis of chronology. After plotting the location of the homes of these first deaths, McClellan drew lines connecting the homes of the deceased to the steamboat levee where they worked, numbering the vectors based on the date of diagnosis. They may live here, and here, and here, McClellan’s map said, but all the cholera victims worked on the levee where the disease was introduced by ship. Where cholera victims lived elsewhere, places where either another line would be illegible or a blackened city square would be hard to read, he marked the house with an “x”. That most of these homes were on or near the river, or clearly vectored from it, added weight to the map’s argument.
McClellan then drew an incomplete circle centered on the steamship levee to define a “cholera area” of greatest incidence (see figure 14). The circle served several functions. First, it defined an area of greatest intensity to refute Dr. White’s insistence upon a diffuse urban outbreak of miasmatic origin. The uniform pattern of occurrence expected of an airborne disease was replaced here by a pattern of disease clusters whose individual cases all were decedents with intimate connection to the local docklands. Secondly, the circle localized disease occurrence within an area centered on the steamship levee, advancing a geographic proposition in which relation to the docklands correlated with incidence of disease. In McClellan’s treatment, the steamships and riverboats were the assumed carriers of the disease. Their docks were the center of the circle around which the homes of the decedents swirled. Third, McClellan’s circle served as a signature technique McClellan used to link the maps of cholera, one to another, in all the affected cities. In this way he created a consistent graphic that implicitly argued his case for all cities rather than for any one city uniquely.

“Upon the accompanying map a circle has been described, the center of which rests upon the river-front of Canal streets. The diameter of this circle is long enough to include the locality at which case No. 15 died. It will be observed that the circle embraces but the heart of the city of New Orleans, and that a large portion of the city is without its limits...it will be found that the vast majority of the cholera-deaths in 1873 occurred within

Figure 14. McClellan’s map of the New Orleans cholera outbreak in 1873. Source: Rare Books and Special Collections: University of British Columbia.
the area of this circle” (Woodworth, 1875, 106). Like John Snow with his map of cholera cases clustered near the Broad Street pump, McClellan argued that were the epidemic local and miasmatic, another less irregular pattern of disease occurrence would have appeared.

The conclusion was clear: “Dr. White’s doctrine of ‘non-importation’ will not stand.” Up and down the river, “the unfortunate individuals who contracted cholera upon or near the steamboat levee came in contact with the poison which had been imported in the effects of emigrants from the cholera-infected districts of Europe” (Woodworth, 1875, 111). McClellan was of course aware of the important arguments for urban hygiene and cleanliness as disease prevention polices which were founded on the theory of exhalation. He quoted extensively from correspondents in those Mississippi cities who accepted the thesis of cholera’s general importation without abandoning a belief in exhalation and contagion as generative forces in the propagation of cholera in the densely habited quarters of the poor. Whether the disease was air or waterborne, the unsanitary city was everywhere complicit.

In Memphis, Tenn., for example, Drs. H.H. Erskin and J.C. Peters, the latter from New York City, argued the city’s foul airs and its unsanitary ways made it a prime candidate for an epidemic outbreak (see figure 15). “The place was rife with the elements of a great plague, and only needed the specific germ to diffuse it widely and fatal,” they reported. The city’s “sanitary condition was shameful and a disgrace When cholera was announced the streets were unclean, the alleys reeking with filth, the back yards even in the case of our prominent citizens, who blushed to be made the subjects of public exposure, were full of slops and garbage privies had remained unemptied for years and were in many places running over with the foul accumulations” (Woodworth, 1875, 139).

It was McClellan’s genius to build a series of case studies showing the disease’s diffusion while permitting, through local respondents, different theories of the environmental determinants of cholera to have full exposition. While the maps did not include the locus of wastes described in the text, or in earlier maps, it did not need them. The text carried that theme and nineteenth century readers would have assumed the working class nature of docklands and the socioeconomic conditions that prevailed there.

GERM THEORY: LATE 1800s

The cholera question was definitively settled when improvements in microscopy permitted the identification of Vibrio cholerae by Robert Koch in 1883. The discovery was enfolded in the exposition of Pasteur’s germ theory of disease, one that paid less attention to the medium of disease agency, water or air, to focus on the agent itself. This did not end the concern with the condition of urban air or its relationship to disease, however. Instead it transposed that concern from one that was causal to one that was contextual. Odiferous areas of urban waste continued to be constructed as potential sites of disease generation and transmission. In the then emerging field of public health, odor was symptomatic of unimproved urban infrastructure—specifically the handling water and waste—that was not simply unaesthetic but a hazard to health as well.

In a number of cities it became common practice for public health officials to map the odiferous sewer outlets and areas of stagnant water as a way of identifying unhealthy locations whose airs, while no longer perceived as generative, were assumed to be symptomatic. An 1878 map of offensive odors in Boston serves here as an example of this class of maps whose purpose was primarily sanitary and health-related (see figure 16). As the city expanded, landfill extended the urban base across shallow
marshes and statuaries. Sewer and water piping were then laid to provide basic sanitation and water services.

At another scale the belief that good air promoted health, and bad air disease, became part of a broader climatic determinism. Systematized in the late 1880s, the promise was of a “historical-geographical pathology,” eventually shortened to “medical geography.” The focus was, as Hirsch explained in his 1881 Handbuch der Historisch-Geographischen Pathologie, translated into English as a Handbook of Geographical and Historical Pathology (Cliff and Haggett, 2003, 11-12), “the geographically dependent factors (such as race, nationality, soil conditions, climate, social factors, etc.) that have to be considered essential for the occurrence and distribution of individual diseases” (Hirsch, 1883, Vol.1, 2).

The result was a period of geographic determinism in which illness was assumed to be largely the effect of the air, climate, soils, wind, temperature, and other physical characteristics of an environment inhabited by specific, at risk populations. These factors explained both different patterns of disease incidence and, some believed, the basic nature of disease itself. “We must no longer be contented with the mere statement that certain geographical facts in the distribution of disease are coincident with certain other facts connected with the soil and atmosphere,” Alfred Haviland insisted in the 1892 preface to the second edition of his Geographical Distribution of Disease in Great Britain. “The time has arrived when the cause of the disease itself must be thoroughly investigated, and its relation to the soil and the atmosphere ascertained” (Haviland, 1892, viii). This determinism was fundamentally different from the environmental argu-
Haviland and his contemporaries mapped a positive correlation between the incidence of disease and local geographies in which local airs were assumed to be generative of disease (see figure 17). Like others before them, the data was right but the explanation was wrong, missing intervening vectors that tied specific diseases to local environments. For example, respiratory diseases were more common in mining districts but not because of the chill, valley airs but because of the living and working conditions of low paid and ill-housed coal miners. Tuberculosis could be found in dense settlements in industrial cities where the transmission of the bacillus was favored not by “dank airs” in overcrowded tenements but by the ease with which the disease was transferred interpersonally in those dwellings.

CONCLUSION

The arguments made in the maps reviewed in this paper emphasize the relationship perceived by generations of health researchers over the last three hundred years. Together they assert the ecological perspective in which disease incidence is necessarily considered within a broad context of social and physical variables. To these are added issues of the technolo-
gies of production and distribution that generally affect not simply the use of maps but more generally scientific research and publication.

The primary focus of this work has been the period in which epidemic disease was considered within the context of two theories of disease, both existing in the period before modern bacteriology and virology permitted their decisive development and eventual conjunction. Air and water are recognized today as important media for a range of infectious agents. In the end it was not either or. It was both and. That said, the broader perspective offered in this historical review has contemporary as well as antiquarian significance. The entwined importance of health, place and air remains a critical concern of contemporary investigators. Modern researchers, like their predecessors, regularly consider the relation between air quality and disease incidence (bronchitis, influenza, etc.). Of at least equal importance is the potential for airborne transmission of diseases like multi-drug resistant tuberculosis in communities that are at once poor and overcrowded. These interests coexists with intense concern over waterborne viruses and issues of urban water supply in developed as well as developing regions. Understanding the history of these concerns historically is relevant to our perception of them in the modern age.

In considering relationships between infectious agents and their modern environments mapping remains a critical tool. Today that mapping typically is carried out on a GIS platform using electronic data in a medium facilitating digital data collection and electronic as well as print dissemination of graphic and statistical results. While these technological changes have been transforming, the essential idea of a spatial proposition that equates patterns of incidence with environmental influences remains fundamentally unchanged. The modern perspective of disease ecology, mapped and statistical, is the direct inheritor of the tradition here traced to Arrieta, Seaman, and Pascal. Perhaps the greatest lesson this history teaches, therefore, is the importance of “map thinking” (Brody et al., 2000).
of framing a spatial proposition to consider rigorously possible cause and real relations within complex and rich environments. That has not changed. It remains the way we do business, the basic argument of map thinking itself.

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1“endnote reference”

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