CHAPTER 5

TRANSPORTATION, POPULATION, AND CHOLERA DIFFUSION

Introduction

The methods by which Asiatic cholera diffuses from one region to the next are defined by human population density and transportation features. Due to population and transportation differences, certain regions tend to be impacted by cholera more than others. The means by which *Vibrio cholerae* is spread and the cholera is diffused relates to geographic boundaries defined by human population, an area or point concept. The paths which *Vibrio cholerae* and cholera take are defined by the travels routes, a series of linear features. Areas impacted by these features as epidemic regions are defined by their physical geographic features in relation to human population geography and transportation. Areas which develop into endemic regions are defined by their physical geographic features independent of human and transportation geography.
Transportation Geography.

The simplest chorographic theories of disease distribution paid attention to longitude, latitude, elevation and land form features. By relating each of these features to transportation, theories for disease spread relative to human migration could be argued for specific transportation routes. This view of disease in regard to the cholera epidemic of 1832 in the Americas not only helped support this geographic interpretation of disease diffusion, it also supported the contagion theory for based on the transportation of this human-borne contagion.

With the growing evidence supporting a contagion theory for disease, early localists continued to search for evidence that the miasma they felt to be responsible was somehow brought on board the ships bearing disease victims. Supporters to the atmospheric, soil, geologic and miasma theories for disease tried to explain its passage as the transportation of air, stones, decaying vegetable or animal debris, dirt, and effluvium on board infected ships. One of the more popular arguments claimed that victims on board the ship were infected by “a stratum of atmosphere loaded with some particular influence” such as electricity or one of its gases following passage through a particular region (Bell, 1851, 283). Another argument used by the localists was that the ballast carried in the form of blocks of stone could be responsible for the disease, as stated by Logan in Sacramento in 1851 (Logan, 1851). The possible transfer of cholera into the Carribean by ship cargo was noted in an article published about Havana: “[s]ome said that the person first seized, took the disease from pine timber which was being carried there from Mobile several weeks or months previously to his being attacked” (Anonymous, 1833). Most recently, the possible spillage of water ballast most accepted explanation for the return of *Vibrio cholerae* to the Gulf of Mexico region (Anderson, 1991).
The Cholera Routes: 1832. Cholera first made its way to the North America by numerous vessels bringing emigrants from England to North America in 1832. Circumstantial evidence suggests that cholera was first transported to the Americas by emigration ships landing in New York harbor as early as April 1832, the records of which were noted to missing or removed from the quarantine hospital log books by a former physician in 1837. In June 1832, the first well-documented victims of cholera landed in the immigration ports established along the Saint Lawrence River. In short time, other cholera victims made their way into North America by New York and Quebec.

Ships next brought these cholera victims into the land-locked communities enabling cholera to disperse into the heart of North America in very short time. Cholera next made its way into the Interior Valley by way of popular shipping routes along the Saint Lawrence, Hudson, Ohio, and Mississippi Rivers and across the Great Lakes. The primary route for cholera between 1832 and 1833 was along the Saint Lawrence River into the Midwest, and then southward along the Mississippi River, where it met up with other cases making their way northward along the Mississippi River. The water and land transportation routes which then ensued for cholera demonstrated a behavior typical of early nineteenth century transportation related diffusion patterns based on economic geographic features.

Once vibrio reached the Interior Valley in winter of 1832, it paused before re-erupting in Spring of 1833. Following its re-emergence, cholera next penetrated the Interior Valley by way of land routes. Although much slower, these land routes diffused cholera throughout the remaining parts of the Interior, impacting for the first time a number of rural hinterland communities in the United States. A repeated resurgence of cholera in this region between 1834 and 1836 enabled *Vibrio cholerae* to first establish itself for a short while within certain parts of the Interior Valley, and then make its way for the first time into the “Northwest” in 1837, ceasing its spread just east of the Rocky Mountains.
1848. The second North American cholera epidemic beginning in 1848 travelled similar routes as the 1832 epidemic for several years. This time its initial entry was by way of New Orleans, after which it headed northward along the Mississippi River where it intercepted with vibrio making its way westward from New York and Quebec along the Great Lakes and Hudson, Saint Lawrence and Ohio Rivers. Important to this second cholera epidemic were the three new forms of transportation routes then under development. Canals, railroads, and two major wagon roads, the Cumberland and National Roads, brought emigrants and farming families westward, greatly increasing the population of the Interior Valley and greatly increasing the diffusion of people, culture, economics, and cholera throughout the rest of North America.

Second only to the river routes for vibrio dispersal were the canal routes, either active or being dug by late 1840s. Since these canals were often constructed between large and adjacent to lakes and important riverways, these shorelines were often vulnerable to contamination with *Vibrio cholerae* between 1849 and 1856, and unlike the much larger routes taken by large vessels, canal routes often placed ships right in the heart of the towns and villages they passed through, making frequent stops than along the way and interconnecting various cities, towns and villages otherwise detached from one another. In this way, cholera rapidly made its way from the highly populated regions where it became a full-blown epidemic to the sub-urban, rural and hinterland communities where its impacts were felt, although not as significantly. An examples of cholera diffusion by canal is the summer 1849 epidemic carried along the Chesapeake and Ohio Canal back and forth between Williamsport and Cumberland until its peak in late August (United States Government, 1875). Similarly, on May 22, 1849, a "dirty canal boat" arriving in Philadelphia brought to the city three cholera victims from *Hudson Valley, New York, after which a major epidemic took place* (United States Government, 1875).
The third inland route for cholera was by way of the newly developed railroads. Like with the canals, sizeable townships developed along these routes and were placed well-placed in terms of time and distance. In addition, railroads often passed close to low-income neighborhoods where the risk for disease was the greatest. In this way cholera was spread to an African American community in Kentucky after the train sewage was dumped onto the rails, leading it to flow into a nearby stream feeding the local village’s local water supply (United States Government, 1875, 588). Importance evidence pointing to the railroad as a cause for cholera dispersal came in Panama in 1855 when, according to Buel, cholera deaths were greatly reduced following the development of the Panama railroads. Buel interpreted this result as a reduction in disease incidence due to reduced fatigue and exposure on behalf of the emigrants (Buel, 1856, 326). Its actual cause was most likely the removal of passengers and cholera victims from the highly contaminated water routes.

The least conspicuous travel routes taken by cholera were the roadways heading inland from coastal townships and between other land-borne townships. The speed at which cholera travelled by these land routes was determined largely by their popularity and degree of contact with lowland and wetland environments. According to medical geographer Dr. Daniel Drake, these routes were “slow and tedious,” but still quite effective. In Lexington, Kentucky, it took thirty-two days for cholera to travel just twelve miles to the nearby town of Covington (United States Government, 1875, 588). The much larger, more heavily travelled land routes, like the Cumberland Gap and National Road facilitated the passage of cholera from the Mid-Atlantic states of Virginia and Pennsylvania into the eastern Interior Valley cities (ibid).

In spite of the numerous canals, railways and crossroads laid during the mid-nineteenth century, the most important transportation routes for cholera between 1849 and 1853 remained the major intercontinental transoceanic routes. International ocean travel was the most important way for vibrio to reach very large populations in
very short periods of time. This allowed for resurgences in cholera repeatedly over long periods of time throughout the nineteenth century. Cholera has its way of finding the quickest and most effective routes to other countries. Therefore, the more rapid the migration route, the more quickly cholera would be dispersed not only into the coastal cities, but also into the right local ecology.

Population Geography

In the past, epidemic regions were defined by boundaries that were politically based and relied heavily on certain forms of culturally-definable living practices. More current boundaries useful for defining cholera geography relate to important social issues such as population density, poverty, urbanization, public health, and health care allocation.

Increased population density directly impacts the likelihood of cholera onset in a given region. Attached to the problem of increased population density is the predisposition to cholera due to poor sanitation and uncontrolled or unmonitored human movements. In addition, highly populated regions tend to have areas where low income people reside in close quarters within and around the urban setting where unsanitary conditions persist. A number of nineteenth century geographers, paid close attention to this urban and suburban dilemma, defining it as a principle cause for many of the local diseases which took place. Those favoring the theory of cholera propagation by increased population density used this reasoning to explain its dissemination through miasma or zymoses (fermented waste) throughout these unsanitary low income communities (Johnston, 1856, 58).

In Hennen’s initial interpretation of disease as a geographical phenomenon, he discussed the importance of population assessment but failed to mention the value of relating population number or density to disease and epidemic behaviors (Hennen, 1821).
Similarly, Marshall (1832) focussed mostly on physical geographic features in relation to
disease diffusion, failing to mention population density, but paying heed to
politically-defined population features including race and place of stay. One of the first
interpretations of cholera and other diseases as a population phenomena in America was
made by California medical geographer Stillman (1851), who noted an increase in
diarrhea (in Stillman’s account, including cholera) and dysentery problems in relation to
the rapidly growing population of Sacramento Valley. In 1856, British geographer
Johnston also noted an association to exist between large populations and disease due to
sanitation problems (Johnston, 1856). During the migration of argonauts to California by
way of the Panama isthmus, population movement was felt to be responsible for the
yellow fever common to the region and its spread as an epidemic (Buel, 1856, 1859).

One reason population density was not considered an important feature in
describing disease diffusion during the early nineteenth century related to the findings of
a number of the cholera epidemics in which disease patterns did not match population
sizes. In Havana, Cuba, for example, cholera struck two equally populated regions
differently (Anonymous, 1833, 672), which some geographers interpreted as proof that
position (i.e. relative to sin and windflow) and topography rather than population density
were the most important factors controlling disease diffusion. It wasn’t until the second
cholera epidemic began to take hold of California that a local medical climatologist
publicly blamed the increase in these diseases on population density (Logan 1851).
Several years later, a French medical geographer discussing California’s population
dynamics drew similar conclusions (Boudin, 1857a; Anonymous, 1864).

Other evidence suggesting that population density not only impacted cholera
behavior but also increased the likelihood for its onset took place on board the emigration
vessels and within the rapidly growing emigrant communities. Carroll felt cholera was
first brought to the Americas by ships due to crowded conditions on board and within
boarding houses due to “the want of sufficient house room” (Carroll, 1854, 322).
Stillman noted the high incidence of “diarrhea” (a term he used when referring to cholera cases as well) in “the thickly settled part of San Francisco” known as “Happy Valley” (Stillman, 1851, 290).

Likewise, a local review of the crowding of steamers making their way to San Francisco following the gold rush shed some light on this issue. Steamers were reporting a greater number of deaths on board than the much smaller sailing vessels. In an article published in the Weekly Times, it was noted that the Panama Route between March and September 1852 carried 9828 passengers, of whom 43 percent died due to cholera, whereas sailing vessels taking the same route brought 3720 passengers to California, dying at a rate of only 5.1 percent (Anonymous. 1852, Mortality of Ocean Steamers).

Defining the Geography of Cholera

The initial definitions of cholera in space and time provided a three-dimensional spatial definition based on the miasma theory. According to the miasma theory, the geography of disease dispersal takes place due to the substance diffusing through the atmosphere, transmitted the disease from one individual to the next. This “malaria” or “miasma” migrated according to features dependent on local topography and weather and independent of human population size. By interpreting the miasmic interpretation of disease relative to human populations, a way to document the disease over space and time developed enabling a more accurate definition of its ultimate cause to be developed.

As a spatial and temporal phenomenon, advocates of the miasma theory could compar and contrast the epidemic findings with the findings of their counterparts, the localists. The localists theory of disease attributes disease to a given location, defined by such attributes as latitude and longitude, elevation above sea level or local water body, placement relative to nidus, source or point of origin, or placement relative to the
causative such as a type of topographic feature, soil, water body, plant, animal or weather region. If this cause is considered to be independent of malaria, miasma, or similar atmospheric influences like electricity and wind patterns, then the diffusion of this substance is greatly minimized by the stability of the feature(s) causing that disease at a given location. A pathogen like detritus or animal waste for example is limited first by its placement within the environment, and secondly by the local topographic features impacting not only its diffusion away from the place of origin or nidus, but also the likelihood that it will impact human populations residing in or passing through the region. This early nineteenth century geographic interpretation of disease is directly responsible for many of the current analytical methods in use for epidemic and disease analysis and supported the germ theory for disease once the first pathogenic bacteria were discovered.

The geographic definition of disease patterns and routes employs point, linear, spatial and temporal features with which qualitative and quantitative methods of interpretation and analyses can be made. Qualitative theories of analyzing disease behavior existed throughout the nineteenth century. Quantitative methods were pretty much lacking in the professional journals, although several geographers eluded to it (Bell, 1851).

Muhry’s work on the disease patterns began with the notion that disease is due either to a zymotic or meteorologic misbehavior or dyscrasia. Muhry relied on latitude, temperature and moisture to define four classes of disease based on their progression and distribution: 1) ubiquitous diseases which relate to a specific cause or set of causes found in any part of the globe, 2) diseases which are defined as existing within specific zones definable using specific geographic features like topography and weather, 3) endemic diseases which are natural to an area but do not influence those residing in the region due
to natural selection features, and 4) very localized diseases known to but a few specific parts of the earth (Muhry, 1856; Anonymous, 1857, 313).

Muhry’s work helped to further define those disease with known causes. At the time of Muhry’s writing, cholera had just transformed from being a very localized disease inherent only to the Bay of Bengal and parts of India defined by local geographic features to a disease more ubiquitous in its behavior, impacting a large number of cultures previously unexposed to it as a natural selection feature. Muhry’s four classes at first fit the scenarios proposed by localists, but quickly lost these associations to the regional view of cholera due to its rapidly changing behavior.

In 1863, another medical geographer Milroy noted the importance of human behavior in relation to cholera, which dealt more with the ability of the disease to be pestilential and migrate first by increasing and multiplying within the body of its victim and then diffusing into healthy victims around the victim as a miasmatic-like entity such as a gas or effluvium. Milroy felt this atmospheric agency spread by diffusing through the atmosphere but was greatly influenced by environmental features including storms, landscape, ecology, etc. (Milroy, 1863, 480-481). Both Muhry’s and Milroy’s theorems fit the initial map of cholera provided by Paul (1851) and suggested that population density had little effect on the spread of cholera (Paul, 1851).

Once the causative agent for cholera was identified as a bacterium, the means for the spread of the disease became quite apparent. Koch’s identification of Vibrio cholerae in 188(4) (Koch, 1884) enabled late nineteenth century medical geographers to begin linking cholera to population density and diffusion relative to water-related geographic features (Haviland, 1892).

During the twentieth century, three types of descriptions for epidemic spatial diffusion patterns were popularized. Hierarchical and non-hierarchical descriptions of disease diffusion were produced as extensions of Christaller’s work on the diffusion of innovation and central place theory (Christaller, 1935). Pyle took a spatial and temporal
look at disease behavior, using the 1832, 1849 and 1866 epidemics to define three behavioral patterns: frontier, mixed, and developed. Cliff et al. (1981) provided a categorization of disease type based on population size relative to diffusion patterns and cyclicity. Types I, II, and III. Other methods of spatial interpretation of possible value to disease interpretation include those put to use by Von Thunen (1826), Whittlesey (1929) and Meinig (1965).

The hierarchical model for disease diffusion states that once a disease initiates in a region, its dispersion follows a route first to the uppermost tier (level) in the diffusion model followed by descension from upper to lower tiers throughout the diffusion model, thereby impacting neighboring sectors and tiers [Figure]. Cascade diffusion follows the route only in descending order, disabling the disease from reaching upper levels (Cliff et al, 1981, 9). These hierarchical and non-hierarchical manners of disease diffusion can in turn be applied to other geographic methods of epidemic analysis.

These epidemic diffusion routes were defined separately by geographers Pyle's classification of disease diffusion models consists of temporal and spatial features. He defined the first stage of disease behavior following its initiation in a particular area as "frontier," which follows a direct linear pattern of spread along a given route. The second stage of disease spread defined by Pyle, "mixed", results in the formation of secondary niduses or foci from which the disease disseminates into new regions. The third pattern defined by Pyle, "developed", relies on urban regions for epidemic eruptions to occur. The most important differences between Pyle's subclasses is in the temporal patterns. Frontier disease patterns tend to occur in sequence, with the largest cities hit before the smaller, much younger cities. The developed region disease pattern has an epidemic erupting about the same time at each of its different cities, suggesting either efficient transportation and pathogenesis through various forms of human contact or eruptions in each of these regions at about the same time as separate disease foci.
Cliff et al.'s descriptions define a recurring disease pattern for a given area. In Type I areas, the disease recurs in regular cycles and rarely misses a cycle. Type II patterns tend to recur cyclically, but may miss a cycle occasionally. Type III patterns cycle but recur irregularly.

As an active epidemic, cholera behavior matches the traits posed by Christaller, Pyle and Cliff et al. The degree of resolve used in differentiating it diffusion pattern change however following a closer interpretation of the afflicted region. At first glance, the cholera diffusion through the United States appears to follow Christaller's hierarchical model of diffusion, during which time it first strikes major cities of international economic importance, followed by other major cities of national and international importance before diffusing into the suburban and rural lands of the deep interior or hinterlands.

Cholera follows Christaller's hierarchical model to some extent once the epidemic begins. However, the initiation of the epidemic in a given locality, be it urban or rural, takes place due to a separate set of processes. A cholera epidemic typically begins by impacting an individual from a low income community. Behaving according to the hierarchical diffusion model, cholera then makes its way to the urban center before being passed on to other hierarchically-defined locations.

The cholera epidemic which entered Canada via Quebec for example made its way to Toronto and then large cities along the Great Lakes including Buffalo, Detroit and Chicago by first impacting the poor communities and boarding houses resided in by low income Irish. Its diffusion into the United States took palce due to the impoverished French families residing along the Saint Lawrence. From each of these regions, smaller epidemics erupted in the various townships proximal to the larger cities. Similar, as the 1832 epidemic made its way through New York in 1832, it ascended the Hudson River and merged with the Canadian cholera spread, thereafter making its way westward.
towards Ohio, and then along the Ohio River into the Midwest to large cities in Indiana and Illinois.

Once cholera made its way to the midwest, it followed a more cascadian like diffusion model, passing from one fort to the next along the Missouri River before reaching its physiographic, climatic and human geographic barrier in the Rocky Mountains. This non-hierarchical or direct diffusion model demonstrates how cholera makes its way town by town along transportation routes in developing regions.

When viewed as the transfer of cholera from ports to cities, and then on to towns forts and finally rural villages and homesteads in the hinterlands, this method of cholera diffusion exemplifies the hierarchical model of epidemic diffusion. A closer review of its behavior as a single disease type recurring over time portrays cholera as a non-hierarchical diffusion type. The areas most cholera-prone in an economically developed region are sociologically and economically defined as low-income regions in and around developed urban regions. Once it has taken hold of its first victims, this cholera then makes its way back to the core urban region where a full-fledged epidemic then erupts. This method of cholera propagation counters the hierarchically-defined central point theory of spatially-defined diffusion patterns, providing instead a central point theory of disease diffusion which relies more upon low-income status.

These differences between cholera behavior in the urban, borderland, suburban and rural setting relate to Whittlesey’s sequent areal occupancy writings. The transition of a region from wilderness or hinterland to rural setting, followed by the growth of that rural setting into a larger town and then city, matches descriptions of sequent occupancy interpretations provided by Whittlesey’s following. If disease is viewed as the result of a given lifestyle including occupation status, then Whittelsey’s proposals meld together well with the epidemic patterns defined by Pyle and Cliff.

The behavior of cholera in newly-developing regions in which population patterns exist in aggregates which are diffusely scattered follows the Type I means for disease
diffusion as noted by Pyle. Once an area becomes better settled and possesses either increasing numbers of people, an increasing economic stability, or an increasing number of immigrants from developed countries, the segregation of rural, sub-urban and urban communities takes place based on socio-economic status. This change in the population then lends to changes in disease patterns as type II patterns for disease diffusion take hold due to the separation of highly-susceptible populations from those least-susceptible to cholera due to their distance from contaminated regions and reduced likelihood of becoming cholera-prone due a combined malnourishment and alcoholism problem.

This reversal of the socio-economic-based hierarchical model of cholera diffusion through space is exemplified by the introduction of cholera into the rural, suburban and central urban low income communities in Cincinnati, Ohio in 1849, Kingston, Jamaica in 1832, and central New York City, New York in 1832. In each of these highly populated cities, cholera had to first make its way into the densely populated urban region by making its way into low-income family communities, which due to their place of stay (near water or in the lowlands) and history of malnutrition and alcoholism became the first to develop cholera, facilitating its spread into communities resided by well-nourished middle and upper class residents within the same city.

This role played by socio-economic status on cholera diffusion represents an important issue in both historical and modern medicine. Cholera remains one of the most frequently recurring causes for disease, epidemics and endemics in developing countries and low-income regions situated close to ecologically-defined *Vibrio cholerae* regions, such as along the United States shorelines of the Gulf of Mexico where low-income cajun, creole, Native American, Hispanic and caucasian fishing communities reside.

The diffusion of populations into low income residencies makes for easy introduction and diffusion of deadly diseases like cholera into neighborhoods often bordered by higher income establishments. The the prevention and elimination of cholera as a recurring threat to a given population may not onvolve the elimination of the
physical causative factors for the disease, Vibrio cholerae, but rather the elimination of these sociological-based disease causes. With the presence of low-income, malnourished, and often alcohol-consuming communities, nearby communities, in which one or more of these predisposing factors are missing, become more susceptible to catching the disease due to its neighboring poor communities.

Chapter Conclusion

By studying the tranportation of cholera and its changes in place over time enabled valid arguments to be posed in favor of the contagion theory and against the localist theory based on the atmospheric, geologic, miasmic, and soil (Pettenkofer's telluric) theories for the origin of cholera. The 1873 review of cholera pointed out the following evidence: 1) Once cholera became epidemic in foreign country, with which North America as in communication with, those residing in North American developed cholera, 2) such an epidemic took place only after vessels arrived from that country, and their infected passengers discharged from it, and 3) once an epidemic began in the port cities, it took significant time for the disease to reach the interior of the continent, in which time large numbers of carriers developed leading to a much broader spread of the disease (United States Government, 1875, 593) This geographic interpretation of cholera based on transportation geography inferred one of the first accurate depictions of its cause just prior to the discovery of its pathogen Vibrio cholerae during the late nineteenth century.

Cholera-dispersion in the developing urban setting is affected by transportation, local military activities, living conditions, occupation, personal living habits and genetics, and socio-economic status. Areas with high population density have additional factors to consider related to urbanization, including public health and health care issues related to sanitation and sewage disposal practices. Local economic development and
industrialization can impact the likelihood of cholera introduction into a given urban setting. Finally, changes in local disease ecology brought on by environmental changes in urban regions can increase and decrease the likelihood of cholera initiation taking place.