CHAPTER III

ASIATIC CHOLERA AND DYSENTERY ECOLOGY

Introduction

Asiatic cholera recurs frequently in areas where *Vibrio cholerae* (common name vibrio) infects people who are interacting with the local ecology. Known as endemic regions, the result of an initial infection from these regions is often the spread of the disease to other individuals, either through direct contact or indirectly by way of contaminated personal belongings, food and water supplies, and numerous reinfections induced by interacting with the local ecology. In areas where the natural ecology required by vibrio is missing, epidemic regions exist where vibrio maintains itself by infecting one individual after another and/or by surviving in the local environment (i.e. a contaminated water well) for brief periods of time between disease victims.

Modern dysentery commonly occurs due to two predominantly tropical microorganisms--*Shigella dysenteriae* (shigella bacteria) or *Entamoeba histolytica* (amoeba). These organisms cause diarrhea by entering the intestinal tract and inflaming the inner surface of the intestines and/or the secretion of fluids into these intestines. The spread of these two dysentery organisms is typically from one person to the next through direct contact or indirectly by way of infecting waters, foods, clothing and personal belongings. Once the natural ecology of these
organisms characteristic of endemic regions is no longer involved, epidemic regions develop where direct person to person contact becomes necessary for the disease to continue to be spread.

These three bacterial agents responsible for Asiatic cholera and modern dysentery epidemics are similar in how they are spread from person to person. Individuals infected by each of these organisms typically have similar reasons for why they were successfully infected. Intestinal tract infections in general are facilitated by low stomach acidity. Therefore, each of these organisms have similar human physiology and geographic requirements in order for single cases and epidemics to erupt. Since the spread of these diseases is primarily anthropogenic in nature, the prevention of these epidemics comes by preventing its further spread from person to person. Therefore, by changing a given human population setting, such as by killing off disease susceptible individuals or by dispersing the population at risk thereby reducing the chance for spread by contact, an epidemic becomes less likely to continue. Should a second disease with similar diffusion requirements infect the now-modified population, it is less capable of diffusing through the population setting.

Throughout history, a number of ubiquitous bacteria have also been linked to the various diarrhea epidemics often confused with shigellic and amoebic dysentery. These microorganisms are less dependent on human population features to form their endemic or epidemic regions. They cause disease whenever people interact with them as a natural part of the surrounding environment. Many of these microorganisms are opportunistic in nature in that they typically infect people whose immune systems have been weakened by poor nutrition, fatigue, alcoholism,
or severe illness. The diseases these organisms produce are axenotic-zoonotic in nature in that they are spread from animal hosts or reservoirs to people. Due to the littering of the Oregon Trail by animal carcasses, one or more opportunistic bacteria-induced several dysentery epidemics along the Oregon Trail between 1844 and 1852.

To differentiate the Asiatic cholera region of the Trail from opportunistic dysentery regions, the biology and ecology of each have to be considered. Whereas the vibrio responsible for Asiatic cholera epidemic infects people as a direct consequence of the human and natural ecological activities, the bacteria responsible for dysentery along the Oregon Trail tend to be more ubiquitous in nature and less selective in producing epidemic regions. With the organisms responsible for opportunistic dysentery readily available as early as the first trail migrations, the introduction of Vibrio cholerae onto the Great Plains in 1849 caused these dysentery regions to diffuse westward, forming new disease regions further west by 1850.

VIBRIO BIOLOGY

Microbiology

The bacterium responsible for Asiatic cholera, Vibrio cholerae, exists in numerous genetically-identified forms referred to in the medical writings as serogroups and biotypes. The serogroup of Vibrio linked to most modern Asiatic cholera epidemics, Vibrio cholerae O1, presumably identical to the past epidemic strains, is referred to throughout this thesis unless otherwise noted. The biotypes of
*Vibrio cholerae* O1 noted in this study are the nineteenth century *Vibrio cholerae* Classical and its early twentieth century replacement *Vibrio cholerae* El Tor. As of this date, the El Tor biotype is the most common Asiatic cholera pathogen, although several new genetic variations of a potentially more virile nature have surfaced in recent years, including several *Vibrio cholerae* non-O1 strains and *Vibrio cholerae* O139 (Kay, Bopp, and Wells 1994, Morris and the Cholera Laboratory Task Force 1994, Wachsmuth et al. 1994).

In spite of these differences, the various serotypes and biotypes of vibrio have similar behaviors as disease-causing organisms. Each induces significant amounts of diarrhea while residing in the human intestinal tract, leading to death due to dehydration. Both also prefer similar ecosystems outside the human body, and tend to reside in similar nesting places or niduses from where new cases and epidemics erupt due to human-environment interactions. Understanding the ecology of *Vibrio cholerae* is useful for differentiating endemic regions where the bacterium survives naturally in close proximity to human populations, from epidemic regions where its persistence is due to non-ecological reasons related to human populations. For this reason the behavior of the modern *Vibrio cholerae* El Tor provides us with important insight into the *V. cholerae* Classical.

**The Environment**

As detailed by studies of the ecology of *Vibrio cholerae* El Tor, a number of zoocic and geographic features define where the best niduses occur (Table III). Due to its environmental requirements, vibrio survives well in regions which are replenished each spring with nutrient-rich detritus being discharged into deltas and
estuaries due to snow melt and spring rainwater run-off. These water-based environments also provide vibrio with several mechanisms for migration, namely: (1) the flow of oceanic water along coastlines which carry vibrio from one ecosystem to the next, (2) the modification of water-based transportation by way of climatic and weather changes; and (3) periodic changes in water flow related to tidal activity. The ability of vibrio to migrate into and out of estuaries not only causes it to come in contact with nutrient rich discharges produced by urban settings but also in some cases to infect people engaged in outdoor activities within this same environment.

TABLE III

ENVIRONMENTAL PREFERENCES OF *VIBRIO CHOLERAE*
(ADAPTED IN PART FROM KAYSNER AND HILL (1994, 32) AND FROM CONCLUSIONS DRAWN BY THIS STUDY)

<table>
<thead>
<tr>
<th>Environmental Features</th>
<th>Vibrio Preferences</th>
</tr>
</thead>
<tbody>
<tr>
<td>Temperature</td>
<td>Refrigeration increases vibrio lifespan, see growth at Temperatures &gt; 10°C (50°F)</td>
</tr>
<tr>
<td>Humidity</td>
<td>Survival is better with higher humidity</td>
</tr>
<tr>
<td>pH</td>
<td>Range of 6.0 to 10.0, 7.0 to 8.5 is favored</td>
</tr>
<tr>
<td>Salinity</td>
<td>0.25% to 3%; optimum conditions at 2% with adequate sodium ions</td>
</tr>
<tr>
<td>Organic Debris</td>
<td>Serves as nutrient, extending vibrio survival</td>
</tr>
<tr>
<td>Sunlight</td>
<td>Reduces lifespan of vibrio</td>
</tr>
<tr>
<td>Osmotic Pressure</td>
<td>High osmotic pressure reduces vibrio vitality</td>
</tr>
<tr>
<td>Geography</td>
<td>Favors estuarine and deltaic environments</td>
</tr>
<tr>
<td>Ecology</td>
<td>Favors ecosystems with crustacea, some fish, some algae, and some aquatic vascular plants</td>
</tr>
</tbody>
</table>
In a study of United States coastline settings by Seidler and Evans (1984), the most important features for a stable vibrio environment were found to be pH, salinity and dissolved Oxygen stores. Since the ocean water environment is often too saline for vibrio, the estuary creates more stable growing conditions due to a mixing of fresh water river effluence with naturally flowing ocean water cross-currents. This mixing of waters also replenishes its environment with dissolved oxygen stores and helps to stabilize the pH and alkalinity that exists in this environment, making it more supportive of vibrio survival.

One of the more important human ecological features for vibrio survival is the discharge of human-generated sewage containing well-digested organic materials enriched by a significant amount of nutrients. This human ecological interpretation of vibrio behavior can be used to define several ecosystems where several of the more common Vibrio species survive. The natural reservoirs noted for these Vibrio species are primarily coastal (deltaic) in nature but also include some fresh, brackish and estuarine water environments. When these natural environments are combined with human-generated (physioanthropic) sewage outflow environments, a particular species, Vibrio cholerae, has the ability to survive as a suspended or free-floating organism capable of infecting people exposed to these contaminated waters (Table IV).
## TABLE IV

**PRINCIPLE RESERVOIRS FOR VIBRIO SPECIES ASSOCIATED WITH FOOD-BORNE DISEASES IN THE UNITED STATES (BLAKE 1984, 580)**

<table>
<thead>
<tr>
<th>Species</th>
<th>Reservoir</th>
<th>Sewage</th>
<th>Free-living?</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>V. cholerae</em> O1</td>
<td>fresh and coastal water</td>
<td>sewage</td>
<td>free living</td>
</tr>
<tr>
<td><em>V. cholerae</em> non-O1</td>
<td>unclear</td>
<td>sewage</td>
<td>probably free living</td>
</tr>
<tr>
<td><em>V. hollisae</em></td>
<td>probably coastal water</td>
<td>unknown</td>
<td>unknown</td>
</tr>
<tr>
<td><em>V. mimicus</em></td>
<td>probably coastal water</td>
<td>unknown</td>
<td>unknown</td>
</tr>
<tr>
<td><em>V. parahaemolyticus</em></td>
<td>estuarine and coastal water</td>
<td>possible sewage-borne strains</td>
<td>free-living</td>
</tr>
<tr>
<td><em>V. vulnificus</em></td>
<td>coastal and brackish water</td>
<td>unknown?</td>
<td>free-living</td>
</tr>
</tbody>
</table>

### Vibrio Ecology

Due to the tendency for vibrio to utilize organic debris and other forms of organic waste for nutrition, vibrio often likes to bind to the chitinous exoskeleton of living animals which are detritus feeders. In Colwell’s studies of the natural ecology of vibrio, a close symbiotic-like relationship was show to exist with the copepod, a microscopic animal which periodically migrates into estuaries for feeding and reproductive purposes (Colwell 1984). By adhering next to the oral cavity of the copepod, *Vibrio cholerae* maximizes its exposure to incoming organic debris consumed by the copepod, and is thought to enter the copepod’s gut at times in order to initiate reproductive processes (Oppenheimer 1978, West and Colwell 1984, Colwell and Huq 1994). This vibrio may also be carried into and out of the estuary by this mobile organism, or be moved from nutrient-poor environments into nutrient-rich environments. In a study related to this migration behavior, vibrio has also been shown to attach to egg sacs of copepods, a process which facilitates its
dispersal into new ecosystems by way of egg release into new environments sought out by the copepod during periods of environmental stress (Huq et al. 1984, 521, 529).

Other organisms important to vibrio ecology include prawns (Kundu and How 1938), shrimp (Roberts, Bradford Jr., and Barbay 1984) and oysters (Ronchetti, 1911-1912, Pollitzer 1959, 877, Colwell and Liston, 1960, 1961, Baross and Liston 1970). In freshwater environments, the crayfish is suspected to support vibrio growth (Pollitzer 1959, 860). Like the copepod, a number of these shellfish provide vibrio with a chitinous shell to adhere to for protection and assist it in migrating to new ecosystems during periods of ecological stress (Kane, Culver, and Mathieu 1994). Another advantage to this relationship with marine organisms is that it improves vibrio’s chance of becoming a part of the local human ecology and infecting people to cause Asiatic cholera outbreaks.

**Vibrio Human Ecology**

Since both animal and plant ecology play important roles in vibrio survival, possible axenotic zoonotic associations exist between vibrio, marine animals and people. Marine animals are involved in the passage of vibrio to humans due to its ability to survive on edible fish and shellfish. It may also be passed to human by way of a number of environmental causes, including contact with vibrio-contaminated animal parts, contact with ocean water in which vibrio remains suspended, and/or particular surfaces on which vibrio continues to exist ecologically or as a simple contaminant produced by other cholera victims.
The shells of crabs in particular support vibrio activity in both its natural and human environments. In the natural setting, the chitinous shell protects vibrio from possible environmentally-induced chemical damage, including changes in the pH, alkalinity, and salinity of the water. Likewise, this adherence to crab shells has been shown to prevent the destruction of vibrio during its pass through the normally acidic stomach environment following consumption (Dietrich, Hackney and Grodner 1984).

Due to this link between the consumption of crabs and cholera epidemic onset, a number of studies have been carried out detailing the ability for vibrio to reside on foods and other domestic items. Preliminary studies focused on its ability to spread by way of foreign and food surfaces (Pollitzer 1959) (Appendix B). In more recent studies, Blake (1984) suggested that the various forms of vibrio were spread primarily by undercooked crabs (particularly for *Vibrio cholerae* O1 and *V. parahaemolyticus*), lobsters (*V. mimicus*), crawfish (*V. mimicus*), raw oysters (*V. cholerae* non-O1, *V. hollisae*, and *V. vulnificus*), and possibly turtle (*V. cholerae* O1). In a more detailed study focusing on culturally-defined causes for the disease, Mintz, Popovic and Blake (1994) demonstrated certain food preparation techniques involving various seafood and other tropical food stuffs to be linked to Asiatic cholera incidence in United States citizens returning from international travel (Table V).
TABLE V

FOODS ASSOCIATED WITH *VIBRIO CHOLERAE* CASES IN THE UNITED STATES, 1972 TO PRESENT (MINTZ, POPOVIC AND BLAKE 1994, 350)

<table>
<thead>
<tr>
<th>Country</th>
<th>Year</th>
<th>Food</th>
</tr>
</thead>
<tbody>
<tr>
<td>Australia</td>
<td>1972</td>
<td>Hors' d'oeuvres</td>
</tr>
<tr>
<td>Italy</td>
<td>1973</td>
<td>raw mussels</td>
</tr>
<tr>
<td>Portugal</td>
<td>1974</td>
<td>raw/undercooked mussels</td>
</tr>
<tr>
<td>Guam</td>
<td>1974</td>
<td>home-preserved pada (salted raw fish)</td>
</tr>
<tr>
<td>Gilbert Islands</td>
<td>1977</td>
<td>raw and salt fish and clams</td>
</tr>
<tr>
<td>Singapore</td>
<td>1982</td>
<td>cooked squid</td>
</tr>
<tr>
<td>Truk</td>
<td>1982</td>
<td>ill food handlers infection</td>
</tr>
<tr>
<td>Mali</td>
<td>1984</td>
<td>millet gruel</td>
</tr>
<tr>
<td>Guinea</td>
<td>1986</td>
<td>leftover cooked rice</td>
</tr>
<tr>
<td>Thailand</td>
<td>1987</td>
<td>raw pork</td>
</tr>
<tr>
<td>Guinea-Bissau</td>
<td>1987</td>
<td>cooked crabs</td>
</tr>
<tr>
<td>Malawi</td>
<td>1990</td>
<td>cooked pigeon peas</td>
</tr>
<tr>
<td>Chuuk</td>
<td>1990</td>
<td>raw fish</td>
</tr>
<tr>
<td>Maryland (Thailand)</td>
<td>1991</td>
<td>frozen coconut milk</td>
</tr>
<tr>
<td>Ecuador</td>
<td>1991</td>
<td>seafood</td>
</tr>
<tr>
<td>Peru</td>
<td>1991</td>
<td>cooked rice</td>
</tr>
<tr>
<td>Peru</td>
<td>1991</td>
<td>raw vegetables and fruit</td>
</tr>
<tr>
<td>Peru</td>
<td>1991</td>
<td>street vendor food</td>
</tr>
<tr>
<td>New Jersey (Ecuador)</td>
<td>1991</td>
<td>cooked crabs in cold salad</td>
</tr>
<tr>
<td>New York (Ecuador)</td>
<td>1991</td>
<td>cooked crabs in cold salad</td>
</tr>
<tr>
<td>California (Peru)</td>
<td>1992</td>
<td>shrimp and fish in cold salad</td>
</tr>
<tr>
<td>Louisiana</td>
<td>1978</td>
<td>cooked crabs</td>
</tr>
<tr>
<td>Texas</td>
<td>1981</td>
<td>cooked rice</td>
</tr>
<tr>
<td>Louisiana</td>
<td>1986</td>
<td>cooked crab, cooked or raw shrimp</td>
</tr>
<tr>
<td>Colorado</td>
<td>1988</td>
<td>raw oysters</td>
</tr>
</tbody>
</table>

These relationships between vibrio, shellfish, and fish play an important role in the spread of vibrio to human populations residing near Asiatic cholera endemic and epidemic regions. Once consumed, vibrio can pass through the stomach environment unaffected by stomach acidity if it attaches to the right food
substances, after which it thrives in the human intestinal tract about 3 days, but may last as long as 26 days in a “healthy,” non-convalescent victims, and 42 days in convalescent victims (Greig 1914, Pollitzer 1959, 867).

**Vibrio Behavior and Demographics**

This human ecology of vibrio in relation to demography and human behavior provides important insights into how *Vibrio cholerae* behaves in the human body. Once this organism is placed in close proximity to a population of people, one or more disease-related behaviors may result in the development of disease. In endemic regions, Asiatic cholera recurs regularly causing a temporary immunity to develop in that population over time due to recurring exposure and infection by the bacterium. In epidemic regions, large numbers of cases develop in densely populated settings in very short time due to a lack of immunity and a disease which does not recur periodically or regularly due to a lack of local natural ecological features. Therefore epidemics often have a greater impact on populations in which large numbers of people are susceptible to the disease. This implies an ecological relationship between Asiatic cholera and large human population settings close to the nidus where various human behaviors make it more likely for vibrio to connect with and infect the right people. Therefore, by surviving in regions close to large human populations, vibrio not only benefits from the human waste discharged into the estuary or delta, but also increases its likelihood of surviving as a disease-producing organism (Cash *et al*. 1974, Blake 1984).

To infect people, vibrio has to first avoid exposure to stomach acids and enter the intestinal tract environment where it then begins to engage in its normal
biological activities. Vibrio enters the intestines by either adhering to the chitin surfaces of certain food items, being ingested with alkaline food substances and beverages, or entering individuals with low stomach acidity (Greenberg et al. 1984). This low stomach acidity requirement is important to note since it typically occurs in people who either inherit this condition (achlorhydric people) or are engaged in personal and social behaviors related to poor nutrition, alcoholism and physical stress (hypochlorhydric people). In the case of the hypochlorhydric individual, personal and sociological reasons often exist for this condition, suggesting that large populations are more likely to be infected by the disease due to the greater number of people per unit area potentially predisposed to becoming infected by *Vibrio cholerae*. Since each of these human genetic and personal behavioral causes for Asiatic cholera development is common in certain large population settings, such settings placed close to deltas and estuaries become some of the first to be impacted by the global diffusion process.

**Vibrio and the Human Intestinal Environment**

By passing into the intestines, the threat of stomach acids is greatly reduced by the secretion of highly alkaline bicarbonates by the pancreas. This changes the pH of the intestine tract and makes it more useful as a growing medium by vibrio. To improve this environment vibrio secretes a toxin which causes sodium ions to enter the gut followed by the osmosis of water, in the end creating a more hospitable estuary-like environment similar to the copepod gut in which vibrio is suspected of reproducing (Kaper, Fasano, and Trucksis 1994, 150-151).
According to Huq et al. (1984), the probability interactions between vibrio and people increases with each stage vibrio passes through in the food web. An early introduction into the food web increases the size of its population within the infected organism and improves its chance for infecting people. Vibrio may also be introduced into a population by way of contaminated drinking water or water used in recreational activities.

**Incidence**

For *Vibrio cholerae* Classical, the morbidity and mortality behaviors have been demonstrated quite well statistically by past medical geographers and epidemiologists (Appendix C). The age groups of people most susceptible to the disease are both children and elders, although a greater number of cases and associated fatalities tend to occur in middle age groups (ca. 40-60) due to the day to day living activities they engage in which expose them to either other Asiatic cholera carriers and victims, possessions contaminated by these victims, and the various naturally-formed ecological settings common to outdoor human recreation activities. The result of this pathogenic activity is the production of large amounts of vibrio, followed by its elimination from the body in the form of “rice water stools,” a whitish, cloudy fluid diarrhea discharge resembling the water poured off of rice in which numerous small white particulates are seen (Kaper, Fasano, and Trucksis 1994, 150-151).
Summary

Since both the human gut and the natural estuary provide vibrio with the chemical and physical features needed for its survival, the potential for preventing the transmission of Asiatic cholera to other people seems identical from culture to culture. Proper sanitation practices reduce or prevent human contact with whatever vibrio is surviving in the local environment. The modification of certain living practices common to low socioeconomic status also impacts the likelihood for contacting this disease and becoming fatally dehydrated. Therefore, the best way to avoid Asiatic cholera is to engage in clean and healthy living practices.

DYSENTERY ECOLOGY

Dysentery is a severe form of diarrhea accompanied by bleeding and mucus. It is typically induced by the ingestion of specific microbial organisms. The most common modern forms of dysentery produced by *Entamoeba histolytica* (amoebic dysentery) and *Shigella dysenteriae* (bacillary dysentery) rely heavily upon human population features to be transmitted from one person to the next. Both were probably uncommon to the Oregon Trail, except perhaps during its first few weeks of travel. In addition, changes in trail population brought on by a constantly dividing trail and the impact of the Asiatic cholera epidemic made the more western parts of the migration through the Great Plains less populated and therefore less likely to support these two primarily human-dependent anthropogenic forms of dysentery (for reviews of each, and additional geographic reasons for their exclusion, see Appendix D). The more common form of dysentery to the Trail environment was induced by opportunistic infections like *Salmonella* and
*Campylobacter.* Several other possible bacterial causes like *Escherichia* and *Yersinia* are reviewed in the appendix as well.

**Salmonella**

Salmonellosis is produced by various members of the genus *Salmonella.* A strain of *Salmonella enterica* was the first to be identified as a cause for diarrhea following the ingestion of raw meat from an infected cow during the late 1800s. Then known as Gaertner’s bacillus, this organism has since proved capable of producing numerous forms of food-generated non-typhoidal *Salmonella* infections.

The most famous bacteria of this genus, *Salmonella enterica* Typhi, is responsible for Typhoid fever (Tauxe and Pavia 1994, 613). *Salmonella paratyphi* and the *S. schottmulleri* have been associated with paratyphoid fevers. Two other strains of *Salmonella*, sometimes referred to as distinct species, *S. enterica* Cholerae-suis and *S. enterica* Enteritidis, resemble Asiatic cholera in their epidemic behavior.

The zoonotic behavior of salmonella most often takes the form of an infection of living animal tissues, most commonly involving wild animals and livestock (May 1958; Waterman, Juarez and Carr 1990; Tauxe and Pavia 1994; Miller, Hohmann, and Pegues 1995). Dairy cattle are known to carry *S. dublin* and *S. typhimurium* (Tauxe and Pavia 1994). *Salmonella enterica* Bovis-morbificans relies on cattle for its reservoir (*ibid*). In each of these zoonotic disease cases, the various forms of *Salmonella* may be spread through human contact with decaying animal parts and carcasses (Clarenburg 1964, 150; Banatula et al. 1999).
The geographic distribution of salmonella is global, with epidemics known to occur in sub-arctic regions like Saskatchewan as well as the tropical climates of Southeast Asia. Soil chemistry is important to determining whether or not salmonella will remain in a region. Ground water activity can spread it from one place to the next. Salmonella tends to survive longer in the outdoors during cold temperatures, becoming more infectious as the natural thaw-water flow reach its peak (May 1958 177).

For the typhoid and paratyphoid strains of Salmonella, pollution of water is often a precursor for many epidemics. The flow of human waste from sewers into local waterways following a period of heavy rain is a common cause for epidemics. Camp settings make for highly contagious disease settings.

The most common vehicle linked to the spread of *Salmonella* is contaminated food, especially poorly processed fish, shellfish, meats and dairy products. The meats of wild fowl are known to carry several species of this organism. Oysters may carry Salmonella when they are grown in beds in sewage contaminated water.

Most of the current salmonella-induced epidemics occur in industrialized agriculture settings and are often linked to improper meat processing techniques. For this reason, many salmonella epidemics are considered anthropogenic in nature, their incidence increasing in direct relationship with the region's population density and economic development. Food handlers who deal with raw milk, fruit, poultry, unprocessed raw meats, and wild animal meats are often linked to these epidemics. Historically, the salmonella epidemics have been linked to well-developed portions of the Eastern United States, more so than the Pacific
Northwestern States. Within a single human population setting, the infection of cattle and beef products is the most common cause for the rapid passage of this disease to large numbers of people (Tauxe and Pavia 1994, 620).

*Salmonella* outbreaks are characterized by the rapid development of numerous cases from a common source of exposure. This disease impacts mostly children under 15 years of age, followed by rapid decline in fatality experienced by middle age groups until 65 years of age or greater is reached. As with Asiatic cholera and shigellic dysentery, those most susceptible to *Salmonella* infections suffer from hypochlorhydria (ibid 621).

Symptoms of this diarrhea include mucus- and blood-streaked stools, prolonged diarrhea, vomiting and fever (ibid 624). The fever onset may differentiate this disease from the more severe bloody diarrhea or dysentery due to shigella. In many cases, the fever resolves itself in 48 to 72 hours, making cases that last more than ten days unusual and suggestive of another bacterial cause (Miller, Hohmann and Pegues 1995, 2020). When deaths do occur, they often result in fatalities due to the septic shock and an arrest of heart functions associated with the development of a massive blood-based infection (Tauxe and Pavia 1994, 614).

*Campylobacter*

Campylobacter is a zoonotic disease associated with cattle and sheep. *Campylobacter jejuni, C. fetus, C. coli, C. laridis*, and *C. pylori* are the most noted species in this genus, of which, the first two are considered opportunist organisms which are capable of infecting people with compromised immune systems.
Common causes for this susceptibility include chronic alcoholism, cancer, diabetes, heart disease, and old age. A number of farm animals and domestic pets often serve as common hosts for these bacteria. Given the right living conditions, these organisms may survive for outside of the human body for several weeks as contaminants of a number of food products, milk, fecal material and water (Allos and Taylor 1994, Blaser 1995).

Broadly dispersed throughout the world, campylobacter is capable of infecting both developed and developing countries and has its greatest impact on children under five years of age, and adults who are 20 to 30 years of age or 60 to 80 years of age, who often demonstrate a high incidence of mortality due to diarrhea.

Seasonally, campylobacter is most active during the warm summer months. Within an environment bearing a neutral to alkaline pH, it may survive as long as three months, even at temperatures approaching 100 degrees Fahrenheit. Recent reviews suggest it favors a warm climate to initiate its resurgence in the form of dysentery in the United States setting (Taylor, McDermott and Little 1983, Allos and Taylor 1994).

Hyperendemic regions are noted to exist for campylobacter, places where new-comers become easily infected whereas regular residents experience infrequent dysentery outbreaks due to a temporary immunity they have developed (Bolton et al. 1999). During the mid-nineteenth century the rapidly developing United States could have experienced this hyperepidemic behavior permanent settlement like to early fort settlements where stable local populations existed, a
behavior which enables new epidemics to develop each time migrating populations pass through.

Another feature favoring campylobacter-induced dysentery in pioneer regions is the very low cell count needed for the bacterium to successfully infect individuals. As few as 500 cells have been shown to initiate an epidemic. Undercooked foods can harbor the bacteria and spread the infection to unsuspecting victims. People who come in direct contact with cattle, sheep, beef, horses, swine, and chickens may pick up and carry this disease to other animals and people. As with salmonella-induced dysentery, unpasteurized milk is a common cause for the campylobacter cases where bovine udder mastitis is common in dairy cows. Campylobacter has also been associated with diarrhea epidemics in the Rocky Mountains due to the contamination of drinking water (ibid).

According to Allos and Taylor (1994) campylobacter-induced dysentery is twice as likely to occur in humans as salmonella and 4.5 times more likely than shigella. Some species are capable of eliciting a severe, dehydrating form of diarrhea. *Campylobacter jejuni* in particular, like *Vibrio cholerae* and *Shigella dysenterica*, produces a toxin with diarrhea-inducing effects. In current epidemic strains, this diarrhea lasts from two to three weeks and is accompanied by abdominal pain and fever.

**Summary**

In cases of dysentery when the continuation of the epidemic is dependent primarily upon human interactions, *Shigella*-induced dysentery may have occurred but is not included in this study of the dysentery noted along the higher altitude
regions of the Oregon Trail. Of the most likely opportunistic bacteria associated with dysentery, *Salmonella* and *Campylobacter* are the most likely causes along the Oregon Trail due to their association with both live animals and decaying carcasses and their possibility of spreading by way of various fomites and vehicles (for further coverage on other causes for diarrhea on the Oregon Trail, see Appendix D).
CHAPTER IV

ASIATIC CHOLERA AND DYSENTERY GEOGRAPHY

The geographic features underlying the Asiatic cholera and dysentery epidemic diffusion processes are useful in differentiating between the two. For Asiatic cholera, once the ecosystem of its pathogen, *Vibrio cholerae*, is departed, the resulting inland migration has attached to it specific biogeographic and physical geographic features responsible for how the pathogen may or may not continue to behave in epidemic form. Furthermore, as these biogeographic and physical geographic requirements for vibrio survival are reduced due to the inland migration, the behavior of its attached disease becomes defined more so by the human population features, making human geography the most important determinant as to whether or not Asiatic cholera will continue to diffuse inland away from its natural ecological setting and the attached tropical climatic setting.

Unlike Asiatic cholera, opportunistic dysentery has numerous bacterial causes. In the case of the epidemics which infect the Oregon Trail, one or more species of opportunistic bacteria are responsible. For this reason, whereas Asiatic cholera has a well-defined history with both endemic and epidemic regions defined by the ecology and geography of vibrio, the numerous bacterial causes for dysentery are often endemic and ubiquitous in nature, and behave according to a number of physical geographic, zoogeographic, and human geographic causes.
Aside from these ecological interpretations of the disease patterns, human geographic requirements play important roles in differentiating Oregon Trail dysentery from Asiatic cholera regions once the disease has diffused inland, which for vibrio is away from its natural ecological setting. Since Asiatic cholera is spread primarily through person-to-person contact once it leaves its original nidus, a method of disease transmission greatly reduced by the human fatalities it causes. With respect to a typical migration route like the Oregon Trail, numerous forks and cut-offs further diminishes the impact of this diffusion process might have by further reducing population density.

The organisms responsible for dysentery, however, are not solely reliant on human geographic features for their continued survival. As opportunistic bacteria, they are dependent on the behavior of animals and are of axenotic, zoonotic origin. They rely primarily on the location of decaying carcasses along the Oregon Trail and the ability of these carcasses to infect people without need for a direct transmission process. The geography of these organisms and animal hosts not only helps to explain why Asiatic cholera ceased its northwestward migration along the Trail whereas dysentery continued this diffusion westward.

ASIATIC CHOLERA GEOGRAPHY

The nidus or nest for Asiatic cholera is where endemics erupt repeatedly and where global epidemics or pandemics are born. The original nidus for *Vibrio cholerae* is suspected to be the Bay of Bengal situated off the coast of India and Bangladesh, where detritus discharged of the Ganges river provides it with valuable nutrients supplemented by human waste (May 1951a, 1951b) (Figure 8).
Figure 8. The Asiatic Cholera Niduses in India and Southeast Asia (May 1951b)
This placement of the nidus allows vibrio to survive within an active ecosystem close to a sizable human population, two features responsible as well for the more recent development of niduses within the delta and estuary environments of Chesapeake Bay, the Gulf of Mexico, and the coastlines of parts of Africa and Latin America (Blake 1994a, 1994b, Swerdlow and Isaacson 1994, Tauxe et al. 1994). According to Haggett, the original nidus for *Vibrio cholerae* in the Ganges River Delta (about 20 degrees north, 70 degrees west) developed as a long term consequence of the local ecology and human geography of this region. Haggett suggests endemic cholera first developed in this nidus about 5000 years ago due to the human population growth. As human population density increased, the ability of vibrio to successfully infect new victims quite regularly allowed for the development and maintenance of infections chains followed by the formation of an endemic nidus (Haggett 1994). Therefore, both ecologically and demographically, the spatial requirements for a vibrio nidus and the development of an Asiatic cholera endemic region can be more accurately defined.

By understanding the biogeography, physical geography and human geography of *Vibrio cholerae* as a potential pathogen, we are provided with insights into why the disease develops elsewhere in the world and well-distanced from the original nidus. This interpretation of *Vibrio cholerae* Classical, the evidence for which is provided by nineteenth century medical journals, helps to define how and why certain endemic and epidemic regions for Asiatic cholera exist as well as how vibrio tends to behave before and during the infection process within particular population settings. Such nodal and ecological environments for vibrio may in turn be related to the Oregon Trail.
Biogeography

Biogeography plays a key role in defining the different ecosystems, niduses and potential endemic and epidemic regions for Asiatic cholera (Figure 9). By residing naturally within an ecosystem formed by warm salt water mixed with detritus-rich fresh water river discharges, *Vibrio cholerae* remains biologically active and stable within this environment regardless of its irregular and often unpredictable contact with nearby human populations. Between human infection periods, vibrio develops important ecological connections with various animals in the regions. Colwell (1984) suggests that these commensal relationships between vibrio and the various animal populations are due to their shared need for decaying organic matter or other organisms closely associated with that organic matter.

Studies supporting Colwell’s conclusion include several classical ecological studies in which bottom feeding fish were found to be capable of carrying vibrio inland along estuaries and large rivers, for example Hilsa (*Hilsa ilisha*), Climbing Perch (*Anabas testudineus*) and Murrel (*Opiocephalus punctatus*) of India and Bangladesh (Pandit and Hora 1951, 861-862). By residing mostly in the bottoms of murky waters of estuarine and deltaic environments, these fish are capable of carrying vibrio to new ecosystems, swimming well below the surface and therefore protecting vibrio from excess exposure to fatal solar radiation.

Vibrio also has been shown to have numerous associations with algae and plants. As early as the mid-nineteenth century, an association of Asiatic cholera to aquatic river plants was made by several medical geographers who related Asiatic cholera incidence to stagnant waterways due to ongoing plant decomposition (Hall 1855, Buel 1856, Stillman 1851, Estrauzlas 1873). More recent studies on this
Asiatic Cholera

HUMAN CAUSES:
CONTACT WITH VIBRIO-CONTAMINATED WATERS
CONTACT WITH VIBRIO-BEARING ORGANISMS
REDUCED STOMACH ACIDITY
LOWERED IMMUNITY?

V = Vibrio cholerae
C = Copepod
d = detritus

Algae
Oyster
C
 Mixed Salt and Fresh Waters
 d
 Shrimp

DELTA

Gulf of Mexico Ecology

ESTUARY

RIVER

Great Plains Human Ecology

Figure 9. *Vibrio cholerae* in the Natural and Human Ecological Settings
particular aspect of vibrio ecology behavior demonstrate similar relationships between disease incidence and local vegetation patterns involving duckweed (*Lemna minor*), water lilies (*Nuphar* and *Nymphaea* spp.), sedges (*Cyperus* and *Carex* spp.), reeds (*Arundinaria gigantea*), rush (*Juncus* and *Scirpus* spp.) and water hyacinth (*Eichhornia* spp.) (Islam, Draser and Bradley 1990, Islam, Alam and Neogi 1992, Spira et al. 1981). Within brackish water and fresh water environments, vascular plants are suspected of being supportive of vibrio due to the nutrients they provide as well as a growing surface to attach to and another means of protection from harmful solar radiation (Huq et al. 1984, 521).

The most recent methods used to study disease behavior focus on vibrio ecology. Since *Vibrio cholerae* has also been associated with rapidly dispersing *Rhizoclonium fonatanum* blue-green algal populations, a food source for copepods (Islam, Draser and Bradley 1989, Islam 1990), remote sensing has become an important tool for monitoring algal blooms and copepod activity related to vibrio activity, a disease surveillance technique potentially applicable Asiatic cholera endemic prediction (Epstein 1998). Similarly, a number of early twentieth century studies focusing on Asiatic cholera prediction by way of tidal and weather monitoring processes (Rogers 1928, Russell and Sundararajan 1928), have more recently been matched by similar studies about disease behavior based on El Nino activity and how these long term and short term changes in climate, lunar (tidal) activities, and solar-generated seasonal activities might impact the copepod which vibrio survives on (Arbona and Crum 1996, Dold 1999; CHAART 2000).
Physical Geography

In the United States, a number of physical geographic features define cholera-prone regions outside of the typical nidal or endemic region for *Vibrio cholerae*. Local fluvial features, the elevation of land above local water levels, and topography or landscape provide the means for a transitory nidal-like region to develop under the right climatic conditions. These nidal-like regions in turn enable vibrio to remain viable outside the human body between victims, increasing its likelihood of finding new victims well after the last infected person has passed away. In warmer regions, for example, as vibrio migrates inland along estuarine rivers and makes contact with inland populated regions, new cases often erupt due to highly susceptible people residing within a town or city. As this vibrio migrates further inland and further away from its estuary environment, it becomes more detached from its natural ecological setting and begins to depend more upon features present in the local physical and human geographic settings to remain alive. The most important of the physical geographic determinants for vibrio behavior inland are local hydrology and flood plain geography.

During its residency in the deltas and estuaries just off the coast, *Vibrio cholerae* was provided with ample amounts of dissolved sodium ion stores in water and proper alkalinity typical of this natural setting (Tison and Kelly 1984). By diffusing inland, changes in water chemistry reduce its ability to maintain itself ecologically outside the human body for long periods of time, thereby increasing its dependence on the process of repeatedly infecting human victims. For this reason, vibrio tends to follow heavily traveled rivers, an observation which led Benjamin to coin the term “Riverine cholera” for this form of diffusion (Pollitzer 1959, 876).
Well-distanced from the estuarine environment, hydrochemical features become more important to the survival of vibrio in regions devoid of adequate human populations (Jusatz 1977). As certain river reaches or channel segments are passed, some may suddenly become capable of serving as important transitory niduses for vibrio during its more stressful periods of inland activity due to their salinity and large amounts of natural and human generated detritus. One of the best examples of these rivers in North America is perhaps the Platte River of the Midwest, a braided river surrounded by broad flood plains through which fairly alkaline, moderately warm saline waters flow.

**Latitude.** Asiatic cholera often changes its latitude and thereby experiences a climate change as it migrates inland along rivers. Such regional climate patterns in relation to latitude and disease often played an important role in understanding the disease diffusion process during the mid-nineteenth century, an important part of medical geography first described in detail by two British doctors, Johnston (1856) and Little (1874). Johnston considered certain diseases to be endemic to tropical or “Torrid disease realms” because of the warmer climates, which he defined as having a mean annual temperature of 72 degrees Fahrenheit or more (Johnston 1856). Johnston termed the cooler regions “Inflammatory disease realms” due to the nature of the epidemics they produced, and “Catarrh disease realms” for the more colder portions then traveled. During the later part of the nineteenth century, these regions were renamed “Tropical,” “Temperate,” and “Arctic” disease realms (Little 1874).

Since Asiatic cholera was born in and behaved more aggressively within the tropical environment, Johnston and Little expected it to become less fatal once it
left this latitude-defined climatic region. The further away from the tropical setting progressed, the less active it became until it ceased its diffusion process due to significant climatic changes. This climate-regulated behavior of Asiatic cholera has historically been matched by two other forms of “tropical dysentery” brought on by shigella and amoeba, each of which, according to Johnston’s and Little’s arguments demonstrated reduced activity in developing countries located in the much cooler climates of the northern and southern temperate zones. This dysentery could in turn be differentiated from the more opportunistic forms of dysentery already present in the much cooler climates experienced by still other developing countries, like the United States.

**Topography.** This climate-generated model for disease behavior also has its matching elevation-related component, particularly for regions where mountain climates prevail. The higher up a mountain one ascends in these regions, the less virulent the epidemic is expected to become and the more it dependent it is on direct person-to-person transfer to continue its diffusion process. This topographical view of Asiatic cholera behavior is most applicable to any flood plain setting in which epidemics erupt. Whereas along coastlines elevation is typically measured as a vertical distance above local sea level, along inland rivers and large water bodies, this elevation may be measured as a vertical distance above the local water level. On the oceanic shorelines, Asiatic cholera occurred most prevalently due to its close ecological association with the marine environment. In riparian flood plain environments, this behavior of cholera relative to elevation above local water surfaces was best demonstrated by William Farr of England, who in his 1852 study of cholera in London showed that residents living close to a river
had the greatest risk of becoming infected (Farr 1852 in Susser 1973, 53, Stolley and Lasky 1998) (Figure 10).

![Graph showing relationship between elevation and cholera deaths.](image)

**Figure 10.** Cholera Deaths in Trinity, England in 1848, Relative to Proximity to Stream., Number of People per Acre, and Average Mortality per Year of Areas Defined by Elevation above Stream (Farr 1852, in Susser 1973, 53)

This relationship between elevation and Asiatic cholera incidence existed in large part due to the way this impacts the survivability of vibrio within alluvial plain settings and aquifers between disease victims. Proximity to a local river setting implies not only proximity to a preferred environmental setting, but also proximity to disease hosts interacting with or migrating along that same environmental setting. Farr’s study also demonstrated that this higher incidence of Asiatic cholera deaths did not necessarily ensue due to high population density, for
following ascent from the stream, cholera deaths decreased considerably in spite of greater population densities at 40 to 60 and 100 feet above the local river. Similar studies by Snow and other physicians have also demonstrated proximity to river to be linked to Asiatic cholera onset due to the tendency for drinking waters to be contaminated by nearby sewage overflow and water main leaks (Snow 1849, 1936).

This association between Asiatic cholera deaths and elevation or distance above local water level formed the core of many nineteenth century medical topographers’ arguments for the development of cholera in epidemic form within shoreline urban settings. Several mid-nineteenth century American physicians used this reason to explain why communities residing at low elevations in Ohio and Illinois were more heavily impacted during the 1832 and 1849 epidemics (Hall 1855, Knapp 1855a, 1855b). In 1889, British medical geographer Felkin concluded that the people most prone to cholera resided near the mouth of the Ganges in houses close to the river, adding “should the hill men descend from their mountains, or have any communication with the inhabitants of the plain, they suffer severely” (Felkin 1889, 21). Likewise, India cholera specialist Swaroop related elevation to Asiatic cholera by defining the typical birthplace for a cholera epidemic in India as a densely populated region where humid climate prevailed and where low-lying lands were placed adjacent to rivers less than 500 feet above sea level (Pollitzer 1959, 824; Swaroop, 1951).

The above topographic observations about Asiatic cholera activity by medical geographers suggest that rapid changes in elevation, such as by migrating through mountain ranges, is one of the most important topographic limitations to Asiatic cholera diffusion. Elevation not only reduces the likelihood for vibrio
survival due to changes in landscape features and available water supplies, but also because of how these changes impact local temperature conditions. For these reasons, mountains formed an important barrier to cholera diffusion. During one of the cholera epidemics of the late nineteenth century in India, for example, cholera diffusion along the east coast was checked by the sparsely inhabited hills which stretched down to the coastline (Rogers 1928; Pollitzer 1959, 56).

**Soil and Geology.** Soil-based theories for Asiatic cholera began to surface during the early 1800s, but it wasn’t until the late 1840s, when Bavarian scientist and hygienist Max von Pettenkofer speculated that air within the soil layers could be a cause, that several pedological theories for Asiatic cholera became popular (Hume 1927). A major component of Pettenkofer’s theory claimed that aerated soil which bore oxygen could facilitate disease production due to its tendency to emit effluvium from its substratum (a variation of the miasma theory). Pettenkofer believed regions prone to this effluvium lacked a solid rock foundation and contained a porous soil rich in organic detritus which produced toxic substances capable of “ripening” the causative agents for the disease. Referred to as the “telluric” and “terrene” theory by other medical geographers, Pettenkofer’s supporters proposed that cholera ensued whenever soils became impregnated by its contagion and bore the environmental conditions required for its causative agent to proliferate and disperse in epidemic form (Garrison 1929, 658).

By 1850, this soil theory for disease led California physician Stillman to blame the local diarrhea epidemics on the alluvial fans situated at the base of the mountains near San Francisco, which bore gravelly soil and a nearby roaring stream (Stillman, 1851). Still other interpretations of the same two decades later
by medical hydrologists helped in the development of another variation of the miasmatic theory of disease--the zymotic or fermentation theory (for examples, see Viele 1874/5 and Gardner 1977; for a historically important pedological interpretation of a “zymotic disease”--namely tuberculosis or consumption--see Bowditch 1866).

Another disease theory similar to the pedological theory was posed by a medical geologist who stated that tertiary and quaternary beds located close to riverways were the most susceptible landforms for disease development (Bell, 1851). In particular, the “calcareo-magnesian water” produced by these geologic formations was a theorized cause for many devastating epidemics within river and stream shore communities (Lea 1851, Bell 1851 335-337). An important extension of this geologic view of disease during the 1870s related rock type to subterranean water flow and resulting water-borne disease distribution (Latham 1877). Latham deduced that disease was due to the lack of permeability of certain underground strata and surmised that the impact of local geology on water flow (especially underground waterflow) could be used to explain why certain epidemics were well-localized.

In 1884 (the year Koch’s bacterial theory for disease was published), Bellew used similar reasoning to define the key physiographic feature for cholera epidemic regions as “a low-lying alluvial soil, which is more or less supersaturated with ground water in a state of stagnation or but comparatively very slight motion, and which is subject to periodic inundations or water-logging by the seasonal flooding of the great rivers by which those areas are traversed in deltaic formation.” (Pollitzer 1959 820).
Climate. According to Hirsch (1883-1886) the nineteenth century cholera epidemics occurred mostly in the summer. Supporting this association between temperature and the development of a nidus are several studies detailing endemicity relative to climate in India (Rogers 1928, Russell and Sundararajan 1928). Recent observations of climate behaviors in relation to Asiatic cholera suggest that a reduction in temperature to about 50 degrees Fahrenheit impeded the diffusion of Asiatic cholera across large regions, whereas 40 degrees Fahrenheit halts this diffusion process completely (Pollitzer 1959 827-828).

Supporting the view that cholera behavior is greatly influenced by climate and local weather was the observation made during the early 1800s that vibrio tends to be non-pathogenic during the cold months, during which time it may even “hibernate” within its natural environment. Examples of this overwintering followed the introduction of vibrio to the cold estuaries of Canada and New York just prior to the 1832 and 1849 epidemics. Whereas the initial fatalities due to Asiatic cholera during the mid-winters of 1832 and 1849 in Quebec, Toronto, New York, Cincinnati and Charleston were few, a resurgence of cholera ensued with the warming of the estuarine water environments resulting in a significant number of fatalities. These observations suggest that past epidemics which have been historically interpreted as re-introductions of vibrio into a region, might have occurred due to the ability of vibrio to adapt to the new ecological setting. By developing a new nidus, a new Asiatic cholera endemic region was developed, a possibility greatly supported by studies carried out in recent years in and around the Gulf of Mexico and Chesapeake Bay (Colwell 1981, 1984), along with studies
linking the behavioral tendencies of vibrio to seasonal changes in temperature and salinity (Singleton et al. 1982).

**Rainfall.** Due to the sensitivity of vibrio to water-based growing mediums, the impact of rainfall on cholera incidence has historically led to mixed opinions about the impacts of rainfall on cholera activity. McPheeters's (1850) study of cholera incidence relative to rainfall in St. Louis demonstrated that an inactivation or cessation of cholera took place soon after the initiation of a period of heavy rain. This finding, however, differed from Hall's review of cholera introduction into Illinois from 1851 to 1853 (Hall 1855), which stated that cholera activity ensued following a period of incessant rainfall, suggesting precipitation increases vibrio activity in a region due to either the introduction of new nutrient sources to the growing medium and/or the diffusion of vibrio from latrines into new water sources such as wells.

Even more perplexing is the likelihood that both an increase and decrease in cholera fatalities following heavy rainfall could occur for different hydrological reasons. An initial period of rain can result in the passage of vibrio from contaminated latrines and water wells to nearby uncontaminated water wells enabling it to infect new people. During periods of incessant rain, however, this vibrio may instead be effectively diluted so as to reduce its vitality and cell count in the local waters, followed by its discharge into local rivers and/or a reduction in its viability initiated by changes in salinity, alkalinity and nutrient suspension. This reasoning provides possible reasons for why vibrio is less viable with increased distance from flood plain settings. Such an effect in the freshwater environment contrasts greatly with effect of similar changes involving an estuarine ecosystem,
where periods of heavy rain improve vibrio growing conditions by increasing detritus suspension in the water and changing the migration of copepods and other chitin bearing animals (Oppenheimer et al. 1978, Huq et al. 1984).

**Review.** Asiatic cholera has been referred to as "the archetype of geographically-related water-borne diseases" (Clemow, 1903, 25). According to Felkin, it "follows the course of rivers . . . due to the fact that the riparian areas possess soil saturated with water and decaying organic matter" (Felkin, 1889, 23). Recent studies suggest the importance of the saline environment in the development of new vibrio niduses historically may be lessening, as indicated by its discovery in brackish fresh water creeks in Australia (Bashford et al. 1979) and even some of the more common ditch environments (Desmarshelier and Reichelt 1981, West and Lee, 1984).

With the exception of these more recently evolved cholera nidal requirements, certain waterways and springs were most likely linked to the behavior of *Vibrio cholerae* along the Platte River portion of the Overland Trail to Oregon. The alkaline, saline waters typical of certain parts of this region have the potential of successfully harboring vibrio between human cases. Should such an endemic region develop, the physical geography of vibrio behavior suggests that whatever cholera epidemics were produced would be most aggressive in low-lying, water-soaked regions where highly susceptible groups of people resided in non-hygienic, highly popular camp grounds. The survival of *Vibrio cholerae* is highly dependent upon the local physiography and topographic land features and how these prevent or encourage contacts to occur between any potential disease victims and the infected local water supplies. Since many of these associations
between vibrio, water and people are the consequences of human ecology and are often population related, human geography plays one of the more important roles in determining the diffusion behavior of this disease.

**Human Geography and Hierarchical Diffusion**

Human geography defines cholera diffusion by the way people behave socially as well as in their environment setting. As *Vibrio cholerae* diffused globally, certain transportation routes were more heavily utilized enabling it to impact important trade routes. This diffusion pattern ultimately brought Asiatic cholera to North America by 1832. Repeating this pattern from 1846 to 1856, vibrio made its way to southern New Orleans by late 1848, and from there to the interior valley in Spring 1849 (Figure 11).

Although less-popular than the immigration route by way of New York City, the port of New Orleans was the most important route for vibrio diffusion into the United States during the mid-nineteenth century. The primary reason for this preference on behalf of the vibrio was the warmer climate the Gulf of Mexico provided and the proximity of New Orleans to the Mississippi delta, a place where the ecological requirements for vibrio could be met. Once it left its ecological setting near New Orleans, a number of human geographic features either supported, hampered or prevented its diffusion process into North America.
Figure 11. The Cholera Pandemic of 1842 to 1862 (pandemic region based on May 1951b; North American cholera routes from Peters and McClellan 1875; trade routes from daily ship registries in the following newspapers: Sandwich Island News (1848-1849), Alta California (San Francisco 1849-1850), Oregon Statesman (Oregon City 1849-1850) and Oregonian (Portland 1850); all newspapers on microfilm at Oregon Historical Society, Portland, OR)
Due to its reliance on human transportation for introduction to heavily populated urban settings, Asiatic cholera is typically spread in hierarchical fashion along well established human migration routes. Whereas locally, migrating animal carriers or the flow of vibrio-containing estuarine waters inland during periods of climatic and weather change may be primarily responsible for vibrio migration areally, the pandemic diffusion of cholera across long distances between continents in very short time is due mostly to human migration along waterways.

Several methods for the transportation of vibrio by uninfected human victims are worthy to note. The contamination of stored personal belongings, such as coats and jackets locked away in trunks, or by way of a food or liquid previously infected by a deceased cholera victim has been linked to the spread of disease between continents and across oceans throughout the nineteenth century (Peters and McClellan 1875). A third method for cholera spread proposed most recently by epidemiologists is from port to port by way of ballast water tanks infected with various vibrio-ridden microorganisms, fauna, and algal products (Chen et al. 1991), a method similar to those proposed by a Havana physician (contaminated logs on board) (Anonymous 1833) and a Californian medical climatologist (contaminated ballast soil dug in Sacramento and shipped to San Francisco) (Logan 1851).

Canals and railroads have been implicated as methods of cholera spread due to their proximity to water and/or the establishment of numerous towns equally spaced from each other along the more popular canal routes. The canal routes which were more susceptible to disease tended to form important links between undeveloped rural communities and much larger port cities. During the mid-nineteenth century, the limited development of trains and canals west of Ohio
made rivers the primary routes of migration into the Interior Valley (Peters and McClellan 1875).

An unusual case of cholera diffusion through high elevation regions is important to note since it demonstrates the ability of human behaviors to overcome this limitation. The transport of Asiatic cholera through the 6000 feet Ural mountains escarpment was accomplished by Russian soldiers in 1831 due to the rapid migration of people interacting in such a way so as to produce new disease victims in very short time (Scoutteten 1832). This suggests that in order for the Asiatic cholera to migrate through impassable mountain regions, human population features have to dominate throughout the epidemic. This reasoning is also related to the sequent occupance related behavior of disease diffusion since simple technological improvements in developing countries can make them more susceptible to the disease, such as simple improvements in transportation systems and human population density features.

Summary

A combination of human and physical geographic features suggests that rivers attached to estuaries and deltas are the most likely diffusion routes inland for Asiatic cholera. They not only provide Vibrio cholerae with supporting natural and human ecological settings but also bear a number of additional riparian features capable of supporting vibrio between disease cases, such as alluvial fans and certain topographic, alluvial, hydrologic, and plant and animal related biogeographic features. Since these rivers often serve as the most important routes of travel from seaports to higher elevation regions further inland, the climate along
these routes of travel can either support an epidemic or impede its diffusion further. In the United States, these important deltaic or estuarine transportation routes have historically been the Saint Lawrence, Hudson, and Mississippi Rivers, with the large Ohio and Missouri serving as important branches off of these important international trade routes to further inland urban regions. As the rivers get even smaller, one geographer noted that the vibrio is more likely to travel through well-formed deep valleys or small narrow-channeled streams where numerous town and city are interlinked along a well-delineated pathway (Dunham 1904).

Relating these disease diffusion features to Oregon Trail history, the various towns and forts in the Midwest served as important way-points for the disease by providing it with new victims to infect and in turn diffuse further into the interior of the country. In addition, a number of local climatic and physiographic features assisted Asiatic cholera in this diffusion inland. Latitude and elevation changes introduces vibrio to cooler environments where, according to some geographers, it became less likely to continue inducing disastrous epidemic results. The most important physiographic features for Asiatic cholera diffusion into the Interior Valley relate to hydrology and topography. The alkaline chemistry of Platte River water and its alluvial soil and broad flood plains are features which might have improved the viability of the organism vibrio outside the human body between disease victims by residing in well and aquifers. The braided shape of Platte River made for even more accommodating physiographic changes, changes which were lost as travel progressed further westward along this the river.

With the loss of these physical geographic features along the western half of the Trail, the likelihood that Asiatic cholera would continue traveling westward
depended greatly on the human population features. By the time Fort Laramie was reached, the diffusion of Asiatic cholera became primarily anthroponotic in nature and depended solely on person-to-person contact to continue this migration westward. As the Trail diaries show (next chapter), this diffusion was no longer possible due to the significant demographic changes taking place during the Platte river cholera epidemics.

DYSENTERY GEOGRAPHY

A dysentery epidemic may be caused by a number of bacterial and non-bacterial organisms. When interpreted by its primary symptom—bloody diarrhea—spatial interpretations often lack the specificity associated with tropical borne Asiatic cholera. Since the opportunistic bacteria responsible for the dysentery are often ubiquitous in nature, they lack a physiographically definable regional specificity related to their ecology, but may often be linked to animal carcasses.

As noted in the previous chapter, the agents responsible for dysentery were more capable of surviving the colder mountain environments than the bacterium responsible for Asiatic cholera. Therefore, their epidemic activity was unimpeded by these geographic and climate features that prevented the continued diffusion of Asiatic cholera along the Oregon Trail. Instead, their diffusion of opportunistic dysentery depended primarily on the physiographic features responsible for animal deaths and increased human susceptibility (Figure 12).
Opportunistic Dysentery

HUMAN CAUSES:
LOWERED IMMUNITY
FATIGUE
POOR NUTRITION

Person-to-person contact

Insect vehicles

Animal Deaths induced by fatigue, alkaline water, lack of forage.

FROM LATRINE TO WATERSHED

CONTAMINATED WELL WATER

Great Plains Human Ecology

Figure 12. Opportunistic Dysentery Ecology in the Human-Environment Setting
THE TWO DISEASE REGIONS

Based solely on the physiographic and climatic spatial behaviors defined in this chapter, two disease regions can be defined in the United States relative to the Oregon Trail (Figure 13). The Asiatic cholera region existed from the Gulf of Mexico and extended through much of the Platte River flood plain wherever human populations existed. The vibrio preferred places where dense population clusters supported continual epidemic activity; most likely it thrived in places where the right salinity and alkalinity also existed, such as along the Platte River aquifers. In such places, vibrio depended less on human population features, at least during the summer season. Therefore, the Asiatic cholera region of the Oregon Trail may be interpreted as an extension of a much larger epidemic region formed in the well-populated states east of the Mississippi River (Figure 14). Diffusing westward along the Oregon trail, it made its way as far west as Fort Laramie, where the last true cases were recorded. With the ascent of the Rocky Mountains, many of the living requirements for vibrio were eliminated, resulting in a rapid reduction in Asiatic cholera deaths and any subsequent “cholera” cases were actually a form of dysentery. The conditions which ceased the migration of Vibrio cholerae to Oregon west of Fort Laramie led in turn to the development of dysentery epidemics for years to come. For this reason, two distinct “cholera” regions formed along the Trail, each with its own geographic requirements (Table VI). The best evidence supporting this conclusion is the diary evidence reviewed in the next two chapters.
Figure 13. Cholera and Dysentery Trail Deaths Relative to Forts, Trading Posts and Missions, ca. 1850-1853
Figure 14. Possible Cholera Routes to the West Coast, ca. 1850-1853

KEY
Possible Cholera Routes

- Forts (Ghent)
- California Trail
- Oregon Trail
- Santa Fe/Old Spanish Trail

Developing (<2 per sq. mi.)
6-17
18-44
45-89
90 or more

Population data is from Lord and Lord 1953, p. 69.
### TABLE VI

**THE HUMAN ECOLOGY AND GEOGRAPHY OF "CHOLERA"**

<table>
<thead>
<tr>
<th>DISEASE FEATURES</th>
<th>OREGON TRAIL &quot;CHOLERA&quot; TYPE</th>
<th>OPPORTUNISTIC DYSENTERY</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bacterial cause</strong></td>
<td>Vibrio cholerae Classical</td>
<td>various, but esp. Salmonella and Campylobacter</td>
</tr>
<tr>
<td><strong>Diarrhea Symptom</strong></td>
<td>rice water stools</td>
<td>bloody, mucousy stools</td>
</tr>
<tr>
<td><strong>Fatality</strong></td>
<td>usually within 36-72 hours; due to dehydration</td>
<td>not as common; may take days to weeks; due to dehydration, immune changes</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>HUMAN ECOLOGY FEATURES</th>
<th>OREGON TRAIL &quot;CHOLERA&quot; TYPE</th>
<th>OPPORTUNISTIC DYSENTERY</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Population</strong></td>
<td>Urban areas (population size, density, and socioeconomic complexity)</td>
<td>Population density less important; stage of population development significant due to poor sanitation</td>
</tr>
<tr>
<td><strong>DiseaseTransmission Process</strong></td>
<td>Further inland, changes from ecological to person-to-person transmission</td>
<td>Carcass is source; less dependent on person-to-person; animal-to-person?; insect vectors?</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>GEOGRAPHIC FEATURES</th>
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<th>OPPORTUNISTIC DYSENTERY</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ecological Setting</strong></td>
<td>Deltas; Estuaries; maybe salty alkaline waters</td>
<td>Ubiquitous, but prefers decaying organic material (animal carcasses)</td>
</tr>
<tr>
<td><strong>Animal</strong></td>
<td>Copepods; shellfish; crustacea (ecological relationship)</td>
<td>Bison carcasses (1844), oxen and cattle carcasses (1850-3)</td>
</tr>
<tr>
<td><strong>Climate</strong></td>
<td>Tropical and warm temperate</td>
<td>No preference</td>
</tr>
<tr>
<td><strong>Topography</strong></td>
<td>Near deltas and estuaries</td>
<td>No preference</td>
</tr>
<tr>
<td><strong>Elevation</strong></td>
<td>Prefers low elevations</td>
<td>Rapid elevation changes (fatal to oxen and cattle)</td>
</tr>
</tbody>
</table>