

Tropical Environments, Human Activities, and the Transmission of Infectious Diseases

LISA SATTENSPIEL

Department of Anthropology, University of Missouri-Columbia, Columbia, Missouri 65211

KEY WORDS host-pathogen interactions; tropical diseases; cholera; lymphatic filariasis; dracunculiasis

ABSTRACT Throughout recent history, the tropical regions of the world have been affected more severely by infectious diseases than the temperate world. Much of the success of infectious diseases in that region is due to both biological and environmental factors that encourage high levels of biodiversity in hosts, vectors, and pathogens, and social factors that compromise efforts to control diseases. Several of these factors are described. Discussion then shifts to specific types of host-pathogen relationships. The most important of these in the tropics is the relationship between humans, a pathogen, and a vector that carries the pathogen from one human to another. Mosquitoes are the vector responsible for the transmission of many vector-borne human diseases. Characteristics of mosquito-human interactions are described, including cultural behaviors humans have developed that both increase the chances of transmission and help to limit that transmission. The transmission of water-borne diseases, fecal-oral transmission, zoonotic diseases, respiratory illnesses, and sexually transmitted diseases are also discussed. Attention is paid to how diseases with these modes of transmission differ in characteristics and importance in tropical human populations compared to those in temperate regions. Following this general discussion, three case studies are presented in some detail. The diseases chosen for the case studies include cholera, lymphatic filariasis, and dracunculiasis (guinea worm). These three case studies taken together provide examples of the diversity of human host-pathogen interactions as well as ways that human activities have both promoted their spread and helped to control them. The transmission of all three diseases is related to the nature and quality of water sources. The transmission of cholera, a water-borne disease, is related to sanitation practices, physical characteristics of the environment such as temperature and humidity, and modern shipping practices. Lymphatic filariasis, a mosquito-borne disease, has increased in frequency in parts of Africa in recent decades as a consequence of large-scale agricultural development projects that have shifted the nature and quantity of water sources and potential mosquito breeding sites. Dracunculiasis is transmitted by a small crustacean that contaminates sources of drinking water. Because its transmission can be prevented by a simple change in human behavior, filtering all water with a small piece of cloth before using it, dracunculiasis has been the focus of a major eradication effort that is near success. *Yrbk Phys Anthropol* 43:3–31, 2000. © 2000 Wiley-Liss, Inc.

TABLE OF CONTENTS

Introduction	4
The Nature of Equatorial Environments	6
Host-Pathogen Relationships in the Tropics	8
Vector-borne diseases	8
Diseases related to contaminated water	11
Fecal-oral transmission	12
Zoonoses	13
Respiratory illnesses	13
Sexually transmitted diseases	14
Case Studies	16
The recurrent pandemics of cholera	16
Agricultural development, water control, and lymphatic filariasis	20
Human behavioral change and the eradication of dracunculiasis (guinea worm)	24
Conclusions	27
Literature Cited	28

INTRODUCTION

The great medical advances made during the first several decades of the 20th century led many to believe that our war with infectious diseases was largely over, and that attention could be shifted to determining causes, treatments, prevention, and cures for chronic illnesses. This belief had some justification in Western Europe and the United States, but it was never warranted for most parts of the world. Infectious diseases cause around one third of all deaths and remain the leading cause of death in the world today (WHO, 1996b). Malaria, schistosomiasis, hookworm, filarial diseases, and many other diseases have never been controlled and have caused major health problems throughout the tropical world for at least the last several thousand years. Furthermore, new and re-emerging microorganisms, such as HIV, Ebola virus, hantavirus, and drug-resistant tuberculosis, have resulted in serious illness and death in people from many parts of the world who were thought to be shielded from the ravages of infectious diseases (PAHO, 1996; WHO, 1996b). This experience has forced Westerners out of their complacency and led them to recognize that infectious organisms, with their short generation time and potential for rapid evolution, pose a continual threat to all human groups.

The potential for the evolution of new infectious organisms is nowhere more apparent than in the tropical regions of the world, where climatic conditions provide lush environments in which innumerable hosts of infectious organisms thrive. In addition to these ideal environmental conditions, many human activities in these regions, e.g., cutting down rain forest trees for timber or building dams to support intensive agriculture, have disturbed existing environments and provided opportunities for the evolution of new species and strains of infectious diseases.

Studies of host-pathogen interactions have been a focus for ecologists, zoologists, parasitologists, and other biologists for some time, but bioanthropological studies of human infectious diseases have been relatively rare. Recent reviews of the roles biocultural anthropology has played in epidemiology include Barrett et al. (1998), Trostle and Sommerfeld (1996), and Inhorn and Brown (1990). Most bioanthropological studies in epidemiology follow the lead of Frederick Dunn, who was an early advocate of including detailed observations of human behavior in studies of the epidemiology and control of parasitic diseases (Dunn, 1972, 1976, 1979). Jenkins and Howard (1992) asserted that the role of anthropology is to apply *direct* anthropological observations to the study of disease risk factors, as opposed

to limiting studies to retrospective behavioral surveys, and a small number of studies now do this.

Jenkins et al. (1989) focused on specific cultural factors (e.g., local travel, feasting patterns, willingness to contact outsiders, personal hygiene, use of land) that contributed to differential exposure to and mortality from infectious diseases among the Hagahai of Papua New Guinea. They also considered archaeological and ethnohistoric evidence and combined their study of cultural behaviors with serological tests aimed at identifying specific infectious diseases that were recently prevalent in the population (Jenkins et al., 1989). In their epidemiological study of filariasis infection among Hagahai foragers, Desowitz et al. (1993) observed significant sex differences in prevalence of the infection. They suggested anthropological studies that could be done to test hypotheses about reasons for the observed differences, but had not completed the anthropological research at the time of publication.

A number of researchers have studied the role of cultural and social variables in the transmission and control of malaria. Coimbra (1988) looked at the timing of human activities at both daily and seasonal scales in relation to mosquito behavior, availability of breeding sites, forest clearing, road construction, and alterations in fauna as a result of human activities (e.g., selective pressures on mosquitoes and consequent behavioral changes in the mosquitoes as a result of DDT spraying). He also discussed cultural knowledge and attitudes about malaria. Nakazawa et al. (1998) combined the use of medical and entomological studies of malaria with detailed observations of human behaviors relevant to malaria transmission, and with simulations of a mathematical model to test the effectiveness of potential control mechanisms. Brown (1981) focused on the interactions between malaria and human activities in Sardinia. This study will be considered in more detail below.

These kinds of studies, in which standard epidemiological techniques are combined with detailed observations of human activities promoting transmission, are the most

common bioanthropological approaches to studying infectious diseases, but there are also a few studies that link other areas of biological anthropology to infectious diseases. For example, Shell-Duncan and Wood (1997) considered the relationship between the immune system, nutritional status, and gastrointestinal and acute respiratory infection. Williams-Blangero et al. (1995, 1997) studied the genetics of susceptibility to helminth infections in Nepal (Williams-Blangero et al., 1995) and Zimbabwe (Williams-Blangero et al., 1997). The Nepal study also combined traditional epidemiological research to determine rates of helminth infection with ethnographic methods to assess local attitudes about helminth types, cause of infection, symptoms, and treatment (Williams-Blangero et al., 1998).

In general, though, biological anthropologists have had a very limited impact on infectious disease studies. This is surprising, given the fact that infectious diseases of humans, by their very nature, involve both biological issues related to host, pathogen, and vector survival and reproduction, and cultural issues relating to how humans think about and deal with their pathogens. Because biological anthropology generally involves substantial training in the biological sciences as well as in anthropology, biological anthropologists are in a unique position to contribute original insights and observations to studies of disease ecology.

The goal of this review is to elaborate on the nature of studies of disease ecology, with special attention paid to the roles anthropology either has had or could have in this work. The first section will set the stage by describing the nature of equatorial environments and the types of diseases commonly found in equatorial regions. The second section focuses on different mechanisms that infectious diseases have evolved to ensure transmission between hosts. The transmission of mosquito-borne, water-borne, respiratory, fecal-oral, zoonotic, and sexually transmitted diseases is discussed, especially with reference to the specific environmental and cultural characteristics of tropical regions.

The final section of the paper presents in some detail case studies of three specific

diseases, cholera, lymphatic filariasis, and dracunculiasis (guinea worm). The transmission of each of these diseases is closely tied to the use of water by human hosts and disease vectors (if any). Cholera has been chosen both because of its historical importance in the development of sanitation systems and personal hygiene practices worldwide, and because it is an ongoing problem that appears to be tied to increasing worldwide economic activities and environmental shifts resulting from human activities. Lymphatic filariasis is included because it has increased in prevalence as a consequence of agricultural development and associated environmental disruption. The disease is especially common in parts of Ghana, Burkina Faso, and Nigeria, where there have been large-scale water development projects. Dracunculiasis has also increased in these countries. However, unlike lymphatic filariasis, which has been maintaining a high rate of transmission and continues to be a major public health problem in the region, dracunculiasis is projected to follow the path of smallpox and become the next eradicated infectious disease. These three case studies taken together provide examples of the diversity of human host-pathogen interactions as well as ways that human activities have both promoted their spread and helped to control them.

THE NATURE OF EQUATORIAL ENVIRONMENTS

Tropical forests account for just over half of the world's total forest area (World Resources Institute, 1996). These forests form a continuum from moist evergreen forests to forests consisting largely of deciduous trees, with forest type depending primarily on the amount and distribution of annual precipitation. The essential environmental conditions for evergreen moist tropical forests, more commonly known as rain forests, are a hot moist tropical climate with humidity that varies from 80–95%, an average annual temperature (20–28°C) that remains relatively stable from day to day, and annual precipitation that exceeds 1,400 mm and is relatively evenly distributed throughout the year (Borota, 1991). Most tropical rainfall, however, is a consequence of air

convection and tends to be of short duration, high intensity, and patchy distribution. During tropical cloudbursts, water may be released at a rate 40 times greater than in temperate regions. Consequently, in many areas flooding occurs relatively frequently (Kellman and Tackaberry, 1997; Meggers, 1996).

Satellite imagery and terrestrial surveys indicate that about 90% of the soils in the tropics are lacking in one or more major nutrients, and that nutrient-rich soils occur in small isolated patches (Meggers, 1996). The absence of a dormant winter period limits soil rejuvenation, which is further affected by intense chemical weathering and persistent deep percolation of water within soils (Kellman and Tackaberry, 1997). Because of the poor quality of the soils and high rates of leaching and erosion, there is strong selection in tropical environments for organisms that can rapidly degrade organic materials and for plants that maximize use of available nutrients, often at the expense of the rapid growth and productivity that characterize weeds and many common domesticated plants.

Meggers (1996) describes several studies documenting the extent of rain forest diversity. In one study, 23 genera of fungi were identified in a sample of 100 Brazil nuts. In a second study, researchers observed during the rainy season that more than 50% of leaf litter decomposed within 30 days. Nutrients in the leaves were transferred so efficiently to plant roots by mycorrhizal fungi that up to 99.9% of the calcium and phosphorus was recovered.

The diverse plant habitats in tropical rain forests provide ecological niches for an equally diverse insect fauna that, together with flowers and fruits, allows for the survival of numerous species of birds, amphibians, reptiles, and mammals. In fact, a single tree may house 43 different species of ants (Meggers, 1996). This diversity of plant and animal species is common to nearly all microenvironments in tropical regions, but it is not uniform across time and space. Rather, there is significant variation in environments and species composition, even at local scales, that makes it difficult to generalize about the nature of tropical environments over large areas. Nonetheless, two

generalizations that seem to hold in nearly all areas are that soils are relatively poor and biological diversity is great. This biological diversity includes not only plants and animals, but also microorganisms causing infectious diseases of humans. The numerous microhabitats promote speciation, which can result in many closely related species of hosts, vectors, and pathogens inhabiting a fairly small area. Because pathogens and vectors generally have very short generation times, especially in comparison with larger mammalian hosts like humans, there is great potential for the evolution of new pathogens and vectors. Even if these species are not originally adapted to human populations, their short generation times guarantee continued opportunities for the evolution of new forms that may be able to cross species boundaries and lead to human infections.

For a variety of reasons, outbreaks of human disease in the tropics generally are more common during the wet season than in the dry season. Disease vectors, like mosquitoes and flies, are more abundant during the wet season. Many intestinal parasites are transmitted by means of contaminated water or soil, and exposure to these may be more likely during the wet season. People may spend more time inside during the wet season, facilitating the transmission of respiratory infections. Nutritional status can vary seasonally, and low nutritional levels often increase susceptibility to or severity of infectious diseases (Scrimshaw and SanGiovanni, 1997).

However, because of the high temperature, humidity, and rainfall that characterize the tropics, most vectors are able to reproduce effectively at all times of the year. This is one of the main reasons that vector-borne diseases are a much more serious problem in the tropics: in temperate regions, the vectors often die out during the winter. Continued perseverance of vector-borne microorganisms in temperate regions usually involves the development of mechanisms to survive through the winter, such as overwintering in dormant mosquitoes or developing spores that survive in the ground and are impervious to harsh environmental conditions. Although pathogens

may survive the winter, transmission is slowed or interrupted seasonally, which reduces the overall impact of a disease.

Opportunities for the evolution and survival of infectious microorganisms in the tropics are further enhanced by the characteristics of tropical human populations. Around 60 countries, with a total population of over 2 billion persons, are situated entirely or partly within the humid tropics. These populations are growing rapidly, with estimated natural population increases of 1.1–3.8% per year, and many of them are also poor (the average 1991 per capita GNP was slightly over \$1,200 per year) (National Research Council, 1993). A lack of economic resources and increasing population make it difficult to maintain standards of hygiene and healthcare, and help to guarantee that population densities are sufficient to support infectious diseases at endemic levels. Thus, both the natural and the human-made environments of the tropics increase the opportunities for the evolution and maintenance of infectious diseases.

A number of common human diseases that are now largely limited to the tropics were once widely distributed throughout the world. For example, from the late eighteenth through the middle of the nineteenth century virtually all residents of the Canadian province of Ontario suffered from malaria (Roland, 1985). Charles Dickens (1844), in his novel *Martin Chuzzlewit*, vividly describes a disease that it almost certainly malaria:

They found Martin in the house, lying wrapped up in his blanket on the ground. He was, to all appearance, very ill indeed, and shook and shivered horribly: not as people do from cold, but in a frightful kind of spasm or convulsion, that racked his whole body. Mark's friend pronounced his disease an aggravated kind of fever, accompanied with ague; which was very common in those parts, and which he predicted would be worse to-morrow, and for many more to-morrows. He had had it himself off and on, he said, for a couple of years or so; but he was thankful that, while so many he had known had died about him, he had escaped with his life (Chapter 33).

and the environment in which it was found, appropriately named "Eden":

At last they stopped. At Eden too. The waters of the Deluge might have left it but a week before: so choked with slime and matted growth was the hideous swamp which bore that name (Chapter 23).

The causes of the extinction of malaria and other "tropical" diseases in temperate regions are not fully understood, but changes in standard of living are at least as important as environmental and ecological factors. There is little evidence of significant regional cooling in Ontario since 1840, and three species of anopheline mosquitoes capable of transmitting malaria continue to be endemic in the region (Dowlatabadi, 1997). What seems to have been of greatest importance in stemming the transmission of malaria in North America is human activities, such as draining Martin Chuzzlewit's swamps, managing other sources of surface water, and active mosquito surveillance. For this reason, an understanding of the transmission and distribution of infectious diseases in tropical regions must necessarily involve an intertwining of both ecological and cultural factors. In addition, many of the characteristics of disease transmission systems are similar no matter the environment, so the discussions below, while centered on diseases now found primarily in the tropics, will address issues that are also of relevance to disease transmission in temperate regions.

HOST-PATHOGEN RELATIONSHIPS IN THE TROPICS

Human infectious diseases by their very nature involve an interaction between at least two organisms, humans and the pathogen, and in many cases other organisms are involved as well. Pathogenic organisms use many different strategies, called modes of transmission, to spread from one host to another. All organisms involved in disease transmission cycles have evolved mechanisms to promote or inhibit transmission. The wide variety of pathogens, vectors, and transmission modes involved and the creative intelligence of humans have all contributed to the incredible diversity of strategies humans and their pathogens have evolved in order to survive.

Modes of transmission of human infectious diseases can be divided into two basic types: direct modes, whereby transmission occurs directly from one primary host to another, and indirect modes, whereby an intermediate host or agent is needed to effect

TABLE 1. Modes of transmission of human infectious diseases

Mode	Examples
<i>Direct transmission</i>	
Respiratory	Measles, influenza, common cold
Fecal-oral	Hepatitis A, rotavirus, giardia
Sexual	Gonorrhea, syphilis, genital herpes, HIV
Direct physical contact	Chicken pox, herpes simplex, yaws
Vertical (congenital)	Syphilis, rubella, HIV
<i>Indirect transmission</i>	
Vector-borne	
Mosquitoes	Malaria, lymphatic filariasis, yellow fever, dengue fever
Flies	Onchocerciasis, trypanosomiasis, leishmaniasis
Ticks	Lyme disease, Rocky Mountain spotted fever
Fleas	Bubonic plague, murine typhus fever
Lice	Louse-borne typhus fever, trench fever
Other	Chagas' disease
Complex cycles	Dracunculiasis, schistosomiasis, hydatid disease
Vehicle-borne	
Water-borne	Cholera, hepatitis B, dysentery
Food-borne	Salmonellosis, tapeworm
Soil-borne	Hookworm, histoplasmosis, tetanus
Needle sharing ¹	Hepatitis B, HIV

¹ Can occur as a consequence of both vaccinations and intravenous drug use.

transmission from one primary host to another primary host (Table 1). Direct modes of transmission include respiratory, fecal-oral, sexual, and congenital transmission, as well as transmission by direct physical contact. Major modes of indirect transmission include water-borne, food-borne, soil-borne, and vector-borne transmission, needle-sharing, and more complex life cycles that involve several different hosts and vectors. The most important modes of transmission in the tropics are probably vector-borne, respiratory, fecal-oral, water-borne, and sexual, although diseases with all modes of transmission commonly occur.

Vector-borne diseases

In vector-borne transmission, a nonvertebrate host (most often an arthropod) either mechanically carries a pathogen from one

vertebrate host to another, or serves as a secondary host in which the pathogen completes part of its life cycle. Mosquitoes, ticks, fleas, lice, sandflies, midges, and gnats are the most common vectors (Chin, 2000). Many serious tropical diseases, including malaria, yellow fever, dengue fever, filariasis, onchocerciasis, and Chagas' disease are transmitted by means of nonvertebrate vectors.

Mosquitoes are a more common vector than other arthropods, and although there are several other important disease-carrying vectors, mosquitoes have received more attention than other vectors throughout human history. The clear relationship between the prevalence of hemoglobin S and high rates of malaria attests to the importance and long history of at least one mosquito-borne disease during human evolution. As might be expected of such a long-standing problem, humans have a complex interaction with mosquitoes that includes both modifications in daily activities and more long-term cultural adaptations, such as innovations in housing. In conjunction with these human strategies, different mosquito species have developed a variety of strategies to ensure that they get their next meal and consequently are able to reproduce.

A blood meal is usually necessary for a female mosquito to produce and lay eggs. This biological imperative, combined with the sheer abundance of mosquitoes in the environment, guarantees that mosquito bites will be common in any environment where mosquitoes and humans coexist. Once the eggs are produced, the female mosquito must find a suitable location for laying the eggs. This location is most often in or near a fresh water source, although some species prefer brackish water (Beaver and Jung, 1985). The eggs hatch and release larvae into the water, where they complete their life cycle and emerge as adult mosquitoes.

A number of factors influence the relative importance of a particular mosquito species as a potential vector of human disease. These include 1) host preference and whether the mosquitoes readily feed on humans (*anthropophilic*) or on other animals (*zoophilic*), 2) whether they spend a signifi-

cant amount of time in association with humans (such mosquitoes are called *domestic*), 3) whether they feed inside dwellings (*endophilic*) or outside (*exophilic*), and 4) whether they are nocturnal, diurnal, or active only at dawn or dusk (*crepuscular*) (Beaver and Jung, 1985). Anthropophilic species are responsible for many of the most severe human mosquito-borne diseases, including malaria, urban yellow fever, dengue fever, and lymphatic filariasis, while zoophilic species are usually involved in zoonoses of humans, i.e., diseases which are native to other animals but which occasionally cause outbreaks of human illnesses.

The distribution and transmission of yellow fever illustrates how a single disease can take different forms depending on local characteristics of either humans or their vectors. Yellow fever has two distinct modes of transmission: an urban mode that is made possible by the bites of highly domestic populations of *Aedes aegypti* mosquitoes, and a sylvatic mode that is facilitated by the bite of several other species of mosquitoes. The mosquitoes transmitting sylvan yellow fever tend to be zoophilic and exophilic, and are at most semidomesticated. Thus, this form of the disease usually occurs either when humans have encroached on the environment of the mosquito and the natural animal host (usually a monkey species), or when bands of monkeys take advantage of a ready food source on human agricultural lands. Either situation results in increased opportunities for human-mosquito interactions, leading to disease transmission. On the other hand, *Aedes aegypti* mosquitoes, which transmit the urban form of yellow fever, prefer to breed in and around human settlements in discarded containers that collect small amounts of rainwater. They are very much at home in human communities and are the main means of dengue fever transmission as well as urban yellow fever (Beaver and Jung, 1985).

Humans have devised many ways to deal with infectious diseases, and much of what is known about how culture influences the distribution and control of infectious diseases comes from studies of malaria. Brown (1981) centered his studies on human-mosquito-malaria interactions on the island of

Sardinia. Malaria in Sardinia, although now extinct (Brown, 1998), was carried by a sylvatic species of mosquito that preferred to breed in fresh water rather than brackish water. The mosquitoes were found in all ecological zones of the island, including highland regions, but predominated in low elevations with fresh water suitable for breeding, and were most active at dawn and dusk. Because Sardinia is temperate, not tropical, malaria transmission was seasonal. Furthermore, the geographic distribution of malaria on the island was highly heterogeneous. Brown (1998) found that the geographic distribution was explained in large part by two variables, elevation and settlement size. Because the mosquito was a sylvatic species, the disease was more common in smaller, agricultural communities and more common at lower elevations. The effect of elevation was related to temperature differences and the amount of standing water available to breeding mosquitoes.

Sardinian settlement patterns consisted of highly nucleated communities situated at "high ground" locations. Furthermore, the agro-pastoralist Sardinians engaged in inverse transhumance, where permanent settlements were situated in the highlands and flocks were taken to the lowlands for the winter. (In regular transhumance, permanent settlements are in the lowlands and flocks are taken to the highlands for the summer.) Although the ultimate cause of these patterns was traced to other factors, these practices allowed the Sardinians to take advantage of the lowland resources at times when malaria was not a problem. The Sardinians also had cultural rules that limited the geographical mobility of particular social groups and class-related behaviors that limited malaria exposure for the landed elite. In particular, women had lower malaria prevalence as a consequence of cultural rules restricting their mobility outside of the settlements, and in some areas the elite shifted their residences during peak malaria times. In addition, local health-related behaviors were prescribed by a belief system (folk-medical theories of fever causation and preventive medicine, especially in relation to the concept of *buona*

aria, or good air) that may have reduced the probability of malaria relapses.

A number of other human activities have helped to promote or prevent malaria transmission. For example, people may engage in work, social, or sexual activities outside of houses during peak mosquito biting times (Wood, 1979; Coimbra, 1988). Brazilian gold miners typically live in dwellings that have only plastic tarpaulin or palm frond roofs, with no walls that control officers can spray to keep mosquitoes out. Consequently, the miners are at risk for bites from malaria-carrying mosquitoes throughout the night (Coimbra, 1988). Prothero (1965) mentioned that Sudanese traveled illegally across dry or shallow river beds into Ethiopia to buy an alcoholic liquor that was forbidden in their country. These trips were taken at times when mosquito activities were at a maximum. Prothero (1965) also pointed out that in some areas there was unequal attention to eradication procedures across political boundaries, e.g., water tanks on one side of a political border were treated with insecticides, but less than 100 yards away on the other side, tanks obviously available to the same mosquitoes were not treated.

Agricultural activities have had an ongoing influence on the distribution of malaria, primarily because they often result in new breeding opportunities for mosquitoes and bring susceptible humans into closer contact with those mosquitoes. Sometimes a shift to different crops, such as rice, which requires extensive irrigation, provides increased breeding sites and increased human population densities. Severe epidemics began in Holland when land reclamation sufficiently reduced water salinity to invite mass mosquito production (Wood, 1979).

It may seem like the majority of human activities increase malaria transmission, but in fact many human practices reduce malaria transmission (Wood, 1979). For example, in many mosquito-infected parts of the world the use of heavy clothing that covers most of the body is routine. In some places, domesticated animals are kept relatively near human habitations to draw mosquitoes to them rather than to humans (although other diseases may become more of a problem because of that). Some practices,

such as clearing jungles and draining water sites, actually reduce the number of potential mosquito breeding sites. In some regions people build temporary living quarters away from mosquitoes during peak seasons or build houses on stilts to limit exposure to mosquito species that live closer to the ground (Laderman, 1975). Many people have discovered folk insecticides such as pyrethrum, which is derived from the chrysanthemum, or eucalyptus (Shiple, 1908). In addition, one of the more widely promoted practices is the use of bed nets and screens on windows and doors to reduce the amount of human-mosquito contact.

Diseases related to contaminated water

Water-borne transmission is a much greater problem in tropical regions than in temperate regions. The risk of transmission itself is increased in large part because of the widespread occurrence of inadequate water sanitation systems. The neglect of these systems has led in some places to the emergence of chlorine-resistant strains of cryptosporidiosis and giardiasis (Bradley, 1998). The tropical pattern of rainfall, with its frequent heavy cloudbursts, exacerbates the problem of inadequate sanitation systems, because it can result in high volumes of sewage-laden water flowing near dwellings and playing children. In addition, nutritional deficiencies commonly interact with water-borne diarrheal diseases, leading to more severe disease outcomes (Chen and Scrimshaw, 1983). Population pressure in some parts of Africa has resulted in a shift to a semirural way of life, where people live at high population densities while maintaining small garden plots for subsistence. Because of the population density, the plots may be so small that placing a well the recommended 30 m from a pit latrine may place the latrine very close to a neighbor's well. So even if a household is protecting itself from contaminated water, its well is likely to be contaminated by a neighboring household's waste (Bradley, 1998).

In parts of the world with no indoor plumbing, multiple sources of water are often available, and the use of a particular source may depend on the purpose for the water as well as the availability at different

sources. As Watts et al. (1998) pointed out, it may be more tempting to use water from a nearby standing pipe that is possibly contaminated than to use a more protected source that is further away. Latrines may be present in a community, but they may not be used (Jenkins and Howard, 1992); rather, defecation and urination may take place near ponds used as a source of drinking water. Because of differences in activities in and around water, water-related diseases may show differential prevalence rates in males and females or adults and children. Other behaviors that may affect the transmission of water-borne illnesses include bathing for hygienic reasons and laundering; swimming; ritual bathing; behaviors to reduce contact with contaminated water, such as crossing streams on bridges or promoting the use of covered wells; fishing; and washing and watering domesticated animals (Dunn, 1979; Mata, 1982).

The transmission of water-borne pathogens has also been affected significantly by the construction of large-scale water control systems as a consequence of agricultural development. The creation of Lake Volta in Ghana increased the rate of transmission of schistosomiasis, although because of differences in the secondary snail host, there was an unexpected shift from the intestinal to the urinary form of the disease (Bradley, 1998). The Richard Toll irrigation project near the mouth of the Senegal River brought irrigation to new areas and expanded it in others. This project resulted in an explosive outbreak of schistosomiasis, with an infection rate in one village of almost 100% in people over 5 years of age (Stelma et al., 1993; Watts et al., 1998).

Environmental change may also affect the frequency of water-borne illnesses. Many scientists believe that temperatures and humidity levels are rising worldwide, and that severe weather is becoming more frequent and intense (Epstein, 1999). Heavy rates of rainfall associated with these changes lead to increased flooding, which increases risk of transmission of disease organisms carried in the water. Such flooding can also overwhelm not only the inadequate sanitation systems of the tropics, but also systems

in the developed world. Global warming and related environmental changes may also promote the growth of algae and associated plankton blooms, which may significantly affect the distribution of one of the most common and feared water-borne diseases, cholera (Colwell, 1996; Colwell and Huq, 1994; Mata, 1994). The epidemiology of cholera will be discussed in more detail below.

Fecal-oral transmission

Several diseases of public health importance, especially gastrointestinal illnesses and some macroparasites, are spread by fecal-oral transmission. In these diseases, infectious agents are excreted in the feces of humans or other animals. Water-borne transmission sometimes involves infectious agents that originate in human feces, but in fecal-oral transmission, susceptible humans come into *direct* contact with feces and (usually) inadvertently introduce the infectious organisms into their mouths, by which means they enter the body and cause illness. Diarrheal diseases, the vast majority of which are spread through fecal-oral transmission, are the second leading cause of mortality among children under age 5 worldwide, killing around 2 million each year (WHO, 1998b).

Because of the nature of most sanitation systems, the transmission of many fecal-oral diseases is affected by the same kinds of factors as water-borne diseases. For example, the location of latrines or defecating near water sources may provide ready opportunities for fecal-oral transmission. In the latter case, the water-borne transmission would be facilitated by using contaminated water, while fecal-oral transmission may occur by coming into physical contact with feces near the water and may not be directly related to the water itself.

Several factors influencing fecal-oral transmission are more important in tropical regions than in temperate regions. For example, a lack of adequate indoor plumbing may lead people to defecate near agricultural fields. In addition to direct contact with the feces themselves, contaminated soil may cling to agricultural products. In most parts of the world, regular outdoor

vegetable markets are held where this produce is brought from the (contaminated) fields and set out on cloths or directly on the ground. Unless all produce is carefully washed before using it, which may be difficult in areas without running water, the stage is set for disease spread by fecal-oral transmission. Open sewers, which are still common in many cities of the developing world, also promote the transmission of such diseases, especially because they are often not deep enough to carry the rainwater resulting from frequent tropical cloudbursts. Consequently, contaminated water often runs in the streets alongside people going about their daily business.

Deforestation and other environmental changes may also influence the transmission of fecal-oral diseases or similar diseases. Lilley et al. (1997) described factors that may have led to an increase in hookworm transmission on the island of Jamaica. Hookworm is not a fecal-oral disease, because transmission occurs through direct skin contact rather than oral ingestion, but it is a consequence of contact with fecally contaminated soils, so that its transmission is affected by most of the same factors as fecal-oral diseases. The Jamaica study suggested that increased hookworm prevalence may have been favored by deforestation of the river's watershed that caused the river channel to accumulate silt rapidly, so that it had to be constantly dredged. After major political upheavals in 1990, maintenance of the river channel was no longer possible, so that beginning in 1992 the river frequently flooded the town they studied and surrounding areas. This altered river drainage patterns and turned much of the community into a delta. It may also have changed the soil type, especially surface deposits, and increased the chances for the soil to remain moist, which increased the opportunities for hookworm transmission. Although deforestation may not have been the only cause, Lilley et al. (1997) indicated that there were no other dramatic environmental changes. They also had reason to believe that the changes they observed were not due to changes in the age structure of the population.

Zoonoses

Zoonoses, diseases affecting humans but for which the primary host is an animal other than humans, are a much larger proportion of the infectious disease experience in the tropics than in temperate regions. Reasons for this include a relative lack of living structures that are climate-controlled and protected from the natural environment, more time spent in outside activities that can bring people into contact with animal host populations, and a greater diversity of potential animal host populations. Zoonoses are an important source of emerging human diseases, and the continued discovery of new zoonoses indicates that the pool of zoonotic diseases is far from being exhausted (Morse, 1995). Recently discovered zoonotic infections include Argentine and Bolivian hemorrhagic fevers, bovine spongiform encephalopathy (mad cow disease), Ebola and Marburg viruses, hantaviruses, Lassa fever, and Rift Valley fever.

It is nearly impossible to predict when or where the next zoonotic disease of humans will emerge, or what its importance will be to humans both in the local region of emergence and on a global scale (Murphy, 1998). One of the main factors influencing the emergence of new zoonotic diseases is the short generation time and consequent potential for evolutionary change in microorganisms. This allows them to adapt rapidly to changing and diverse environments. Many of the common animal host populations, especially rodents, also have a very short generation time relative to humans, which provides abundant, ever-changing, and ready host populations in which new diseases can survive. When these host populations adapt to a life near human dwellings or when human activities, such as hunting or mining, put people into contact with the host populations, the stage is set for new pathogens to evolve ways to infect humans.

Recent studies have associated a number of human behaviors directly with the emergence of new zoonotic diseases. For example, changes in agricultural practices have been associated with the emergence of Argentine and Bolivian fevers; a natural envi-

ronmental cycle leading to an unusually mild and wet winter and spring has been implicated in the recent hantavirus outbreak in the American Southwest; urbanization favoring the rodent host and increasing exposure to the virus in homes has been linked to outbreaks of Lassa fever; and the development of water control systems has increased the transmission of Rift Valley fever (Morse, 1995).

A recent upsurge of monkeypox cases in the Democratic Republic of Congo has been related to a number of human activities. It is thought that the elimination of smallpox vaccinations increased the pool of people who are susceptible to monkeypox, because the smallpox vaccinations provided cross-immunity to the related monkeypox virus (Cohen, 1997). Civil war in the Congo may also have contributed to the problem, because a growing threat of starvation may have increased native hunting of squirrels, monkeys, and rats that are reservoirs for monkeypox. It is also possible that monkeypox has evolved into a more virulent or more transmissible form (Cohen, 1997).

Because human-human transmission of HIV is common and regular, this disease is not considered a zoonosis. However, it is thought to have originated as a zoonosis. This idea dates from the earliest days of the epidemic, and recent new evidence provides more substantial support for the hypothesis. Hahn et al. (2000) derived molecular phylogenies for the primate lentiviruses, including HIV-1 and HIV-2. Their evidence indicates that HIV-1 most likely arose as a consequence of simian immunodeficiency (SIV) transmission from chimpanzees to humans, with the most likely chimpanzee source being the West African *Pan troglodytes troglodytes*. HIV-2 appears to have resulted from cross-transmission of SIV between sooty mangabeys and humans in West Africa.

Respiratory illnesses

Most people associate diseases like malaria, yellow fever, or Ebola fever with the tropics, but tend to think of colds, influenza, and pneumonia as diseases primarily of temperate regions. However, relative to other infectious diseases, these diseases and

others spread through respiratory transmission are nearly as common in the tropics as they are in temperate regions. In fact, acute respiratory infections are the leading cause of death worldwide among children under age 5 and result in over 2 million deaths annually (WHO, 1998b).

Respiratory transmission can occur if infectious organisms are spread through the air when a person coughs, sneezes, or even breathes. However, it occurs only if a susceptible person is in close enough contact with an infectious person to breathe in the infectious droplets released by the infectious person. This happens much more easily when large numbers of humans live in close association with one another on a regular basis; hence, droplet transmission increased significantly in importance after humans settled down and began living in larger, more sedentary communities. In addition to colds, influenza, and pneumonia, many of the familiar childhood diseases, such as measles, mumps, and influenza, are spread by this means.

Some characteristics of tropical environments that are less common in the temperate regions tend to promote respiratory transmission. For example, because residents of most tropical countries tend to be poorer than their counterparts in many temperate countries, there is much more extensive use of public transportation, especially busses and trains. These vehicles are often overcrowded and are used on long journeys from the home village to a market center. The chances of escaping infection by the cold virus being shed by one person in the van is remote, indeed.

On the other hand, some characteristics of tropical environments may discourage respiratory transmission relative to opportunities in temperate regions. Residents of tropical regions may spend more time outside, where infectious droplets are more likely to diffuse into a larger volume of air. Also, housing for the majority of tropical residents is less likely to consist of a closed, climate-controlled air conditioning system and more likely to consist of open windows that will draw infected droplets out into the environment and away from susceptible humans. Because of these counteracting influ-

ences, the tropical and temperate experiences with respiratory diseases are not as different as the experiences with diseases that are transmitted by other mechanisms.

Sexually transmitted diseases

Many of the most feared human diseases are spread through sexual contact, including HIV, gonorrhea, syphilis, and genital herpes. Sexually transmitted disease (STD) organisms usually require a warm, moist environment for survival and do not live long outside the human body. STDs, especially gonorrhea, have long been a cause of significant levels of infertility and reduced fecundity in tropical regions of Africa (McFalls and McFalls, 1984), and tropical regions throughout the world have also been especially hard-hit by the HIV epidemic.

The reasons for the severity of HIV and other STD epidemics in tropical regions are complex. Because of the nature of sexual transmission, social factors are of greatest significance. Primary social factors include, for example, high rates of poverty, low status of women, recent social upheavals, increasing rural-urban migration, and inadequate medical services (Quinn, 1995; Wasserheit, 1995). Increased levels of rural-urban migration in many parts of the world have led to higher rates of unemployment and social disruption, which have, in turn, resulted in an increasing number of young women entering the commercial sex trade as a means of survival. In northern Thailand, agricultural modernization occurring in the last three decades has favored the employment of males over females. This process, and a culture that emphasizes the economic responsibility of females to earn household income, are thought to be the main factors responsible for the extensive migration of young women to become commercial sex workers in the cities and resort areas of Thailand (Wawer et al., 1996). Similar processes have been an influence on rates of prostitution in parts of Africa and elsewhere (Quinn, 1995). For a number of reasons, commercial sex workers have experienced high rates of many STDs, which are passed on to their male customers, who then take them back to their wives at home in

rural areas, who would otherwise be at low risk.

Biological factors have also influenced the spread of STDs. Some of the most important include the presence of other circulating pathogens, which can reduce resistance and increase susceptibility to STDs (Clemetson et al., 1993; Holmberg et al., 1989; Holmes, 1995; Kreiss et al., 1989; Plummer et al., 1990; Quinn, 1996; Quinn and Fauci, 1998); the development of antibiotic-resistant strains as a consequence of inadequate healthcare or reluctance to continue with long-term costly treatments; use of sexual practices, such as douching or genital mutilation, that may increase the probability of transmission (Kingsley et al., 1987; Sweet et al., 1986; Wasserheit, 1995); or widespread malnutrition, which may lower immune system function and increase susceptibility. Genetic variability in the pathogens themselves and their reproductive strategies may also account for some of the variation in observed rates and patterns of transmission of STDs worldwide (Ewald, 1994).

It is clear from the discussion above that microorganisms have developed a variety of strategies to maintain transmission from one host to another and guarantee their survival through time. The success of these strategies is influenced both by biological and environmental factors and by cultural and social factors (see Table 2). Some of these strategies, like respiratory and sexual transmission, depend upon direct contact between susceptible and infectious humans. Because of this, these modes of transmission tend to be most strongly affected by social factors that bring humans together, e.g., increased urbanization, use of mass transportation systems, or economic activities. Other strategies, such as vector-borne and zoonotic transmission, depend upon another animal species to guarantee that the transmission cycle will be maintained. The biological constraints of these other species are usually such that both environmental and cultural factors heavily influence the probability of disease transmission and maintenance. Water-borne and fecal-oral transmission do not usually require a secondary animal host or vector, but they can

TABLE 2. Some features of tropical environments that influence infectious disease transmission¹

<i>Environmental or biological factors</i>	<i>Mode(s) of transmission most strongly affected²</i>
High ambient temperature	Vector-borne, zoonotic
High humidity	Vector-borne, zoonotic
Abundant rainfall with frequent cloudbursts	Vector-borne, water-borne, fecal-oral, zoonotic
Biodiversity in hosts and pathogens	Vector-borne, zoonotic
Large number of cocirculating pathogens	STD
Little seasonality	Vector-borne, zoonotic
Global warming	Vector-borne, water-borne, fecal-oral, zoonotic
<i>Cultural or social factors</i>	<i>Mode(s) of transmission most strongly affected</i>
Household structure and facilities	Vector-borne, water-borne, fecal-oral, zoonotic
Location of houses	Vector-borne, water-borne, fecal-oral, zoonotic
Agricultural activities	Vector-borne, water-borne, fecal-oral, zoonotic
Deforestation	Vector-borne, water-borne, fecal-oral, zoonotic
Water control projects	Vector-borne, water-borne, fecal-oral, zoonotic
Inadequate sanitation	Water-borne, fecal-oral, zoonotic
Inadequate healthcare	All
Malnutrition	All
Increased human population density	Vector-borne, water-borne, zoonotic, respiratory
Antibiotic or pesticide resistance	Vector-borne, respiratory, STD
Use of clothing	Vector-borne, zoonotic
Timing of outside activities	Vector-borne, zoonotic
Social disruption	Zoonotic, STD

¹ This is not an exhaustive list. In addition, many of these characteristics are also important in temperate environments.

² Most of these factors have some influence on all types of diseases.

survive in the environment in the absence of direct human-human contact. Their transmission tends to be less influenced by natural variation in the physical environment (e.g., temperature and humidity) than is the transmission of vector-borne and zoonotic diseases, but their spread tends to be strongly influenced by human activities that alter the environment or that bring humans into closer contact with the natural environment.

The diversity of interactions among human hosts, their pathogens, and vectors or

alternate hosts of those pathogens guarantees that numerous factors influence the transmission of human infectious diseases. The long human generation time compared to that of other species in the transmission cycle means that the most important of these factors are culturally driven. Nonetheless, the essential human host-pathogen interaction is biological. Consequently, the study of infectious disease ecology in human populations is fundamentally a biocultural endeavor, which is ideally suited to the interests and expertise of biological anthropologists.

The most effective way to understand biological, cultural, and social factors important to the transmission of human infectious diseases is to discuss in depth the complex relationships between particular pathogens, their human host, other species involved in the disease cycle, and the social and environmental milieu within which disease transmission occurs. For this reason, the remainder of this review presents detailed case studies of cholera, lymphatic filariasis, and dracunculiasis.

CASE STUDIES

Cholera, lymphatic filariasis, and dracunculiasis have been chosen as illustrations of disease ecology for a number of reasons. First, they are all major problems in tropical regions of the world, but have received little attention in the anthropological literature, and are likely to be unfamiliar to many readers. Second, the transmission of all three of these diseases is strongly influenced by the nature of water use in an area, but their modes of transmission are different. Cholera is a water-borne infection that has a strong environmental component apparently linked to global warming, predominant ocean current patterns, and related factors. Lymphatic filariasis is a mosquito-borne disease whose transmission in western Africa has been linked directly to the development and maintenance of water control systems. Dracunculiasis (or guinea worm) is contracted by humans when they drink water contaminated with a small copepod that carries the worm.

A third reason to consider these diseases is that they can be used to illustrate human

practices that both aid in the spread of infectious diseases and help to control their transmission, both in the past and in the present. Cholera was one of the primary stimuli for the development of adequate sanitation systems in 19th century Europe and North America, systems that are still slowly making their way into the developing regions of the world. Most people are aware of the ravages of cholera 100 years or more ago, but may not realize that it is an ongoing problem in most parts of the world today. Increases in the prevalence of lymphatic filariasis are largely due to environmental disruption resulting from agricultural and water control activities that stemmed originally from European colonialism and economic expansion. Dracunculiasis has been the target of a major world health initiative that has centered on its eradication. Description of the approach taken in this campaign, which is nearing success, provides a window into the ways local and global communities can work together to find ways to improve the health and well-being of humans worldwide.

The recurrent pandemics of cholera

Recent years have seen numerous articles in popular books, magazines, and newspapers about "new" and re-emerging infectious diseases that are rapidly spreading throughout the world (e.g., Garrett, 1994; Preston, 1994). While it may seem like this is a late 20th century phenomenon, new diseases have, in fact, been sources of terror throughout human history. Cholera was perhaps the most feared disease of all.

The term "cholera" was first noted in the works of Hippocrates, and the disease itself is almost certainly an ancient human disease (Barua, 1992). It is a consequence of infection with a bacillus, *Vibrio cholerae*, and causes acute intestinal illness with watery, profuse diarrhea and rapid dehydration, followed over half the time by death, sometimes within a few hours of the first symptoms (Chin, 2000). One of the distinctive features of cholera is that it appears in both a regular seasonal pattern in endemic regions and in explosive outbreaks that may start simultaneously in several distant foci. This temporal patterning suggests that en-

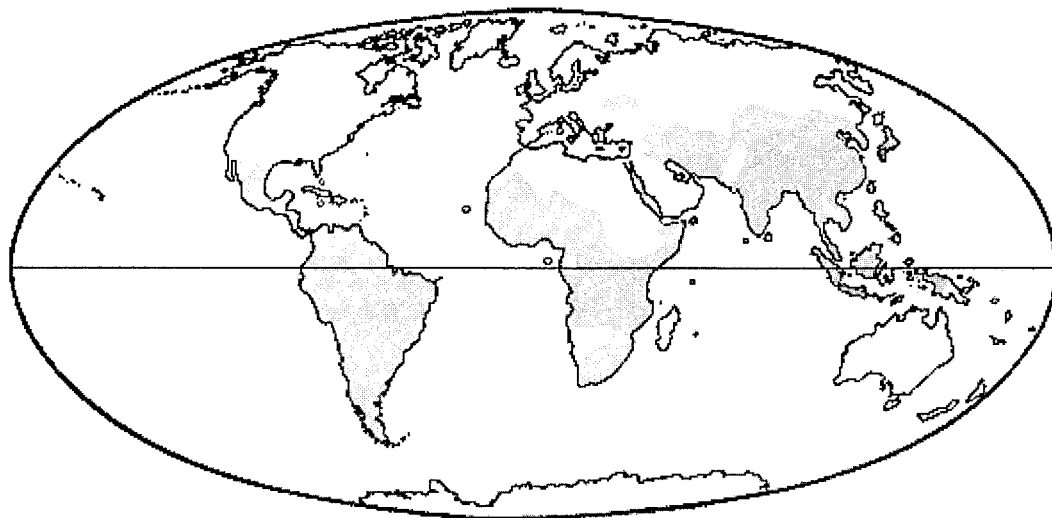


Fig. 1. Regions reporting cholera epidemics between January 1, 1995 and December 31, 1998. Sporadic cases were also reported from nearly all regions of the world (data sources: WHO, 1996a, 1997, 1998a, 1999).

vironmental factors may trigger the epidemic process (Glass et al., 1982; Kaper et al., 1995). In endemic regions, children under age 10 experience the disease most frequently, with a secondary peak in women of childbearing age; in populations without prior exposure to the disease, all age groups are affected equally (Glass and Black, 1982; Glass et al., 1982; Holmberg et al., 1984; Kaper et al., 1995). Antibiotic resistance has been observed and fluctuates from year to year, perhaps due to clonal diversity among strains (Faruque et al., 1998).

Some researchers believe cholera was sporadically present in Europe during ancient times, but there is no general agreement that it existed in Western Europe until the early 1830s. Beginning in 1817, there have been seven great pandemics of the disease. The first pandemic was largely limited to southern and western Asia and eastern Africa; pandemics 2–6 were found worldwide; the most recent pandemic has been limited to developing regions of the world, especially southern Asia, Africa, and South America (Barua, 1992). The geographic distribution of this disease during the last 5 years is shown in Figure 1.

Characteristics of the major pandemics were reviewed by Barua (1992), Speck (1993), Lacey (1995), and Colwell (1996).

The first cholera pandemic, which occurred between 1817 and 1823, had a more limited geographic distribution than other epidemics. It began in India and affected people in the Near East, southern Asia, and Japan, and spread as a consequence of the Oman War and the war between Persia and Turkey.

The second pandemic (1829–1851) probably also originated in India, but may have begun in Russia. It spread throughout Asia, the Near East, the Arabian Peninsula, Europe, and North America. Moscow was especially hard hit, and John Snow's famous Broad Street pump incident occurred during London's experience with the second pandemic.¹ Intravenous fluid therapy, which is used to replace the substantial amount of fluids lost in the severe diarrhea of the disease, was also recognized as an effective

¹John Snow was the first person to clearly demonstrate the role of contaminated water in the transmission of cholera. He conducted a landmark field study where he plotted all deaths from cholera in a large part of London, along with the primary water sources for the region. Two different companies supplied water, with distributions that overlapped in an area that included the Broad Street pump. Snow was able to conclusively trace the cholera to contaminated water coming from one company and, in particular, to the Broad Street pump itself. He then convinced the authorities to remove the handle of the pump (Barua, 1992; Longmate, 1966; Snow, 1965). The epidemic ended soon after, but since the epidemic was waning anyway by that time, the removal of the pump handle probably did not have a major influence (Brody et al., 1999).

treatment during this pandemic (Lacey, 1995).

The third pandemic (1852–1859) peaked between 1853 and 1854 and was rampant in Persia, Mesopotamia, Northern Europe, North America, the West Indies, and Colombia. Global spread was aided by the movement of French soldiers during the Crimean War, and the third pandemic ultimately spread throughout Asia and Africa as well. It was during this pandemic that the causative organism, *Vibrio cholerae*, was identified.

An outbreak of cholera during the yearly pilgrimage to Mecca in 1865 was a major cause of the fourth pandemic (1863–1879). About 30% of the estimated 90,000 pilgrims died from the disease, and the remaining survivors efficiently and rapidly carried it back to their homes in Europe and Africa. Wars in Germany, Austria, and Italy further aided in the spread of the disease. The epidemic also reached the United States in the fall of 1865, but the widespread epidemic was delayed until the spring of 1866.

Mecca was also the site of the beginning of the fifth pandemic (1881–1896) and the sixth pandemic (1899–1923). Both of these epidemics spread throughout most of the rest of the world, but the Western Hemisphere was affected much less than in previous epidemics.

Between 1926 and 1960 the world was largely free of epidemic cholera, although endemic foci remained. During this time, the ability to treat cases of the disease improved dramatically, with the result that mortality rates declined significantly. Then, beginning in 1961, the seventh pandemic arrived on the scene. Most researchers believe that the seventh pandemic is still occurring, and in addition, in 1992 a new strain of cholera, *V. cholerae* O139 Bengal, was identified, leading some researchers to declare the beginning of an eighth pandemic. However, as of August 1999, cases of the new cholera strain were still confined to Southeast Asia and had not reached pandemic status (WHO, 1999).

The seventh pandemic originated in Asia in 1961 and initially spread to the USSR and Iraq. In 1970 an explosive outbreak occurred in the Middle East and in West Af-

rica, which had been cholera-free for a century. Significant numbers of new cases have continued to occur in Africa since that time, but there is little indication that the West African cholera crossed the Atlantic. In 1991 cholera returned to South America, Central America, and Mexico after an absence of over 100 years. The disease entered the continent along the Pacific coast rather than the Atlantic coast, which would have been expected given recent disease foci in Africa (Kaper et al., 1995).

Notifications of cholera cases continue to come from all parts of the developing world, with most recent cases caused by the El Tor subtype associated with the seventh pandemic (Fig. 1).²

Because the spread of the disease is linked to transoceanic shipping and the poor sanitation levels found in much of the developing world, authorities fully expected the seventh pandemic to spread quickly to South America. However, as this did not occur until 1991, some 20 years after the end of the second phase of the epidemic, the South American epidemic is presented as a textbook example of a re-emerging disease. In fact, according to Gangarosa and Tauxe (1992), the Latin American epidemic “resembles the great urban epidemics of 19th century Europe and the United States, and is spreading with late 20th century velocity” because of the advent of modern means of transportation such as intercontinental air travel.

The major mode of transmission of cholera is through contaminated water supplies, but it can be spread several other ways, the most important of which is through contaminated foods (Glass and Black, 1992). As Colwell (1996) aptly expressed, the “history of cholera reveals a remarkably strong association with the sea,” with worldwide patterns of disease spread always following

²Notifications and interesting comments on a myriad of infectious diseases, not only in humans, but in plants and animals as well, are posted to an e-mail digest on a regular basis. The e-mail digest is called ProMED-mail and is sponsored by the Program for Monitoring Emerging Diseases of the International Society of Infectious Diseases. For more information, visit their web site at www.promedmail.org. Other sources for recent information on infectious disease outbreaks worldwide include the U.S. Centers for Disease Control (www.cdc.gov) and the World Health Organization (www.who.int).

patterns of human mobility, especially along ocean and river shipping routes, but also in association with pilgrimages, wars, and other activities (Barua, 1992; Colwell, 1996; Glass and Black, 1992). Europeans realized very early in their experience with the disease that it was associated with water from public waterworks and possibly also with contaminated waterways (Pollitzer, 1959). This recognition influenced sanitary reforms in 19th century Europe, although many researchers believe that the impact of cholera itself was actually less than that of other diseases, such as tuberculosis or typhus (Evans, 1992; Morris, 1976; Pelling, 1978; Rosenberg, 1965).

The adoption of indoor plumbing and sewage control systems originated in these 19th century reforms and has proved an effective means of controlling not only cholera, but other water-borne diseases in the developed world. Cholera disappeared from Europe after 1925, except for an epidemic in Spain and Portugal in the 1960s and sporadically occurring cases. However, the developing (largely tropical) world lacks the socioeconomic base of the developed (largely temperate) world, and sanitation facilities are often still inadequate in those regions. Because of this, cholera is now primarily a tropical disease that is usually found elsewhere only in association with natural disasters that destroy or disrupt local sanitation facilities.

Ballast discharge from a ship in Lima, Peru has been blamed for seeding the South American continent with cholera bacteria. However, more recent research on the biology of the bacteria has shown that it can attach to plankton or algae and enter a state of dormancy until climatic conditions allow it to revert to an infective form. There is a growing opinion among scientists that cholera epidemics most often begin when plankton blooms triggered by a climatic event lead to the release of cholera into the environment. El Niño is thought to be the trigger event for the South American epidemic (Colwell, 1996; Colwell and Huq, 1994; Mata, 1994). Aside from such environmental events, other factors thought to be important in the timing of cholera outbreaks include water pollution, which can upset the

balance of marine ecosystems; overharvesting of fish and shellfish, which can reinforce algal growth; and the loss of wetlands and ocean habitats (Epstein et al., 1993).

In order to determine more definitively how *V. cholerae* survives and is maintained in the environment, between 1987–1990 Colwell (1996) conducted an extensive environmental study in Bangladesh. Results showed that the abundance of cholera was related to the abundance of copepods in seawater. Studies of survival of the cholera bacterium in seawater also indicated that it was capable of surviving in a culturable state for a relatively long period of time, which would allow it to be carried by ocean currents over very long distances (Munro and Colwell, 1996). In poorer conditions it appears the bacterium can enter a viable but nonculturable state that can allow it to be transported in nutrient-poor seawater and in association with plankton for several months and over thousands of kilometers (Shiba et al., 1995). Similarly, within a particular geographic location the organism can persist for many years, which may explain the reappearance of the disease after a long period of quiescence or apparent absence (Colwell, 1996).

In addition to environmental characteristics that can stimulate the reappearance of cholera in an area, a number of human behavioral risk factors have been shown to be of importance in the epidemiology of cholera. A study in Burundi indicated that bathing in and drinking water from Lake Tanganyika was significantly associated with illness. Furthermore, an observed increase in the risk of becoming ill with increasing time spent in the lake indicated that there was at least a moderate dose-response relation. The type of drinking water storage device was also associated with increased cholera risk (Birmingham et al., 1997). In Ecuador, drinking unboiled water, drinking a beverage from a street vendor, eating raw seafood, and eating cooked crab were associated with illness from cholera, while always boiling water at home, and the presence of soap in either the kitchen or bathroom at home, were protective against cholera (Weber et al., 1994). A study of cholera transmission in Matlab, Bangladesh

suggested that sharing a latrine with other households and living in flood-controlled areas increased the risk of hospitalization due to cholera, while use of tubewell water, defecation in places other than latrines, shellfish consumption, breastfeeding, and malnutrition were not associated with increased risk of cholera (Emch, 1999). Koo et al. (1996) found that food and beverages sold by street vendors, leftover rice, and unwashed fruits and vegetables were important sources of cholera transmission.

The seventh cholera pandemic has resulted in a huge number of cases. In 1991 alone there were over 570,000 cases and nearly 17,000 deaths worldwide, with 391,171 (68%) of the cases and 3,871 (23%) of the deaths occurring in Latin America (Gangarosa and Tauxe, 1992). About 222,000 cases were reported to the World Health Organization in 1999, with 193,000 reported from Africa, 22,000 cases reported from Asia, 7,000 reported from the Americas, and fewer than 20 reported from the rest of the world (WHO, 2000a). However, in comparison to the 50% mortality commonly observed in pre-20th century epidemics, the case-fatality rates have been relatively low: less than 1% in South America to around 20% in parts of Africa, with a worldwide mean of around 4.5% (WHO, 1998a). The reason for this has much to do with the influence of government policies on the spread of infectious diseases. Because so much was already known about the epidemiology of cholera, the countries of South America had 20 years to prepare for its arrival. With the aid of local governments, the Pan American Health Organization (PAHO), the International Children's Fund (UNICEF), and the World Health Organization (WHO), efforts were made to guarantee adequate levels of surveillance, training of healthcare workers, laboratory capacity for confirmation of cases, methods for disinfecting water and foods, and proper disposal of human waste (Gangarosa and Tauxe, 1992; Mata, 1994; PAHO, 1994).

Authorities have worked with local residents to find culturally appropriate preventive measures. For example, during yearly floods in Bangladesh, it can be impossible to build a fire to boil water to prevent the spread of cholera. However, people have

been taught to use finely woven sari material to filter the water, which has been shown to reduce the number of bacteria in the water and may, as a result, reduce the number of cholera cases (Colwell, 1996). Blake et al. (1977) found that a common Amazonian practice of adding citrus juice to water to improve its taste protected from cholera because the acid in the fruit killed the cholera bacteria. Other protective practices include treating water at home by boiling it or by adding chlorine to it, using small-mouthed vessels to store water, pouring water out of the storage container rather than scooping it out with a cup, and using hand soap (Tauxe et al., 1999).

In addition, health authorities now have an effective means of treatment, oral rehydration therapy (ORT), available and accessible in all but the most remote territories (Tauxe and Blake, 1992). As a consequence of these government actions and policies, mortality from cholera is largely a thing of the past, although as Glass and Black (1992) point out, scientists do not yet have enough epidemiological understanding to contain the disease and prevent its emergence and spread.

Agricultural development, water control, and lymphatic filariasis

Agriculture has long been a common cause of human environmental change and has had far-reaching consequences for human-pathogen interactions. During the latter half of the 20th century, small-scale agriculture gave way to large-scale agricultural development projects in many parts of the tropics. The goal of many of these projects was and is to increase agricultural productivity and the development of cash crops, a process which often involves environmental degradation and alteration of preexisting ecosystems. These changes in the ecosystem can have a significant influence on the types of infectious diseases present, as well as on their spread and maintenance. The remainder of this section will illustrate how the development of large-scale water control facilities in Ghana, West Africa has influenced the transmission of a parasitic infection, lymphatic filariasis.

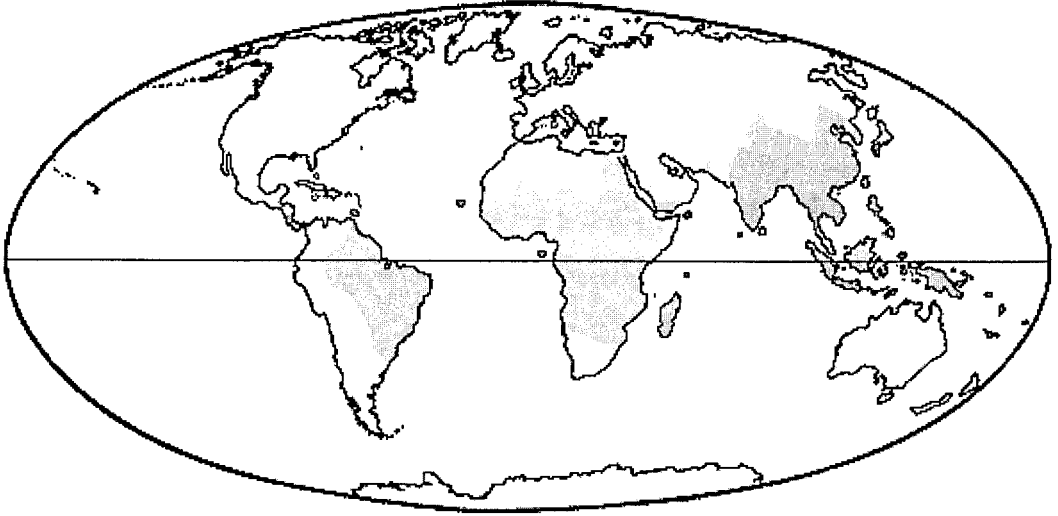


Fig. 2. Global distribution of lymphatic filariasis in 1999. Darker regions are endemic areas; endemicity in lighter regions is uncertain (data source: WHO, 2000b).

Lymphatic filariasis occurs as a consequence of infection with a specific group of parasitic nematodes, the filarial worms. As of 1984, the World Health Organization estimated a prevalence of 90.2 million cases worldwide and 905 million people at risk for infection in the endemic zones of Asia, Africa, and Central and South America (WHO, 1984) (Fig. 2). The disease tends to be overlooked in Africa, even though the continent has about 25 million cases (Hunter, 1992).

Filarial worms are transmitted by mosquitoes, which serve as the intermediate host for the microfilarial form of the parasite. Microfilariae enter a female mosquito when she takes a blood meal from an infected human. Depending especially on the ambient temperature, over the next 10–14 days, the parasite progresses through three larval stages until it reaches an infectious stage and migrates to the proboscis of a female mosquito. The infectious larvae enter a human's skin when bitten by the infected mosquito and move into the lymphatic system, where they migrate into the vicinity of draining lymph nodes. Over the next 9 months the larvae go through one more larval stage and finally mature into thread-like adult worms that are 5–10 cm long. Both male and female adult worms live in the lymph channels for 5–10 years.

Females periodically release microfilariae into the bloodstream, where they can live for up to 12 months if not ingested by a mosquito (Grove, 1983; WHO, 1987).

Three helminth species can cause lymphatic filariasis: *Wucheria bancrofti*, *Brugia malayi*, and *Brugia timor* (Routh and Bhowmik, 1994). Bancroftian filariasis, caused by *Wucheria bancrofti*, is the most widespread human lymphatic filarial infection. It is widespread throughout the tropical regions of Asia, especially in India, and is also common in local areas in Africa, Madagascar, northern South America, parts of Central America, some of the Caribbean islands, and many Pacific islands. The other two filarial species have a more restricted distribution that is limited to parts of Asia (WHO, 1987) (Fig. 2).

W. bancrofti exists in both a rural and an urban type, and both of these forms are usually nocturnally periodic. This helminth species shows marked peaks of microfilarial density in human blood during the night hours, with peak density occurring around midnight. Patients infected with *W. bancrofti* in the South Pacific often show diurnal subperiodicity, where the circulating levels of microfilariae are greatest during the day. Other parasite species are nocturnally subperiodic, with circulating levels of

microfilariae greatest at night (Grove, 1983; WHO, 1987). The main distinction between periodic and subperiodic forms is that periodic forms are barely present in the blood at nonpeak times, while subperiodic forms are present at all times but occur at higher densities during peak times.

Urban *W. bancrofti* is transmitted mainly by the mosquito species, *Culex quinquefasciatus*, in tropical regions and by *Culex pipiens pallens* and *Culex pipiens molestus* in subtropical regions. Rural *W. bancrofti* is transmitted by several species of *Anopheles* mosquito, occasionally by *Aedes* species, and rarely by *Mansonia uniformis*. *C. quinquefasciatus* mosquitoes breed in polluted waters, drains, pit latrines, and similar water sources (WHO, 1987). They tend to bite at night and live in or near houses (Grove, 1983). *Anopheles gambiae*, another common filariasis vector, breeds in a wide variety of water bodies, mostly rests indoors, and bites either indoors or outdoors at night (WHO, 1987). The time of peak biting activity of these mosquitoes is closely linked to the time of peak levels of circulating microfilariae in human blood (Grove, 1983; WHO, 1987), which indicates how closely host, vector, and pathogen biologies can be related.

The clinical course of lymphatic filariasis is divided into asymptomatic, acute, and chronic stages, but there is some variability in disease manifestations within a region. During the asymptomatic stage, microfilariae are present in the blood but produce no clinical symptoms. This stage varies in length from a short time to several years. The acute stage, which lasts from several days to 4–6 weeks, consists of recurrent fevers associated with inflamed lymph nodes and lymph vessels. Each attack lasts for several days and then subsides spontaneously. Occasionally the acute stage can lead to continued inflammation of the lymph vessels and formation of abscesses that can ulcerate and lead to scarring. The chronic stage usually develops only in individuals over age 15 and only occurs in a small proportion of infections. The most obvious manifestations of the chronic stage of the disease are hydrocele and swelling of the testes and marked swellings of the entire lower limb, the scrotum, the entire arm, the vulva,

or the breast. These swellings are usually not painful and may be combined with thickening of the skin, which has earned the chronic stage of lymphatic filariasis the colorful name of elephantiasis (WHO, 1987).

Because lymphatic filariasis is a mosquito-borne disease, its distribution has been strongly affected by water control projects that have influenced the availability of breeding sites for mosquitoes. Several species of mosquitoes are involved in transmission of the disease in different parts of Africa, but although mosquitoes are ubiquitous in the tropics, the disease generally is found only in scattered endemic foci that are subject to local influences (Hunter, 1992).

In their study of filariasis in Ghana, Hunter et al. (1982) found that improvements in the water control system stemming from agricultural development projects occurred at three levels: the construction of large-scale dams to provide hydroelectric power, the construction of smaller impoundments used for more localized agricultural and pastoral activities, and the development of irrigation systems. All of these projects can result in several types of ecological disturbance. For example, forests are destroyed when lakes are formed behind dams and the speed of the water in the dammed river is changed. This leads to changes in the rate of erosion and promotes the development of pools of standing water, which provide new breeding grounds for infectious disease-carrying vectors, such as mosquitoes or flies (Brinkmann, 1994; Hunter et al., 1982; Service, 1989). The establishment of rice plantations in Africa has led to increasing numbers of anopheline mosquitoes (Grove, 1983; WHO, 1974).

In addition to ecological disturbances, water control projects can also lead to economic and social disruption. Residents of areas to be flooded following construction of a dam must be resettled. This resettlement may bring together people from different social, religious, or ethnic backgrounds, leading to less social cohesion than in the original settlements (Service, 1989). Residents of such communities may be unable or unwilling to maintain communal latrines and other sanitary facilities, which may promote the transmission of filariasis and other water-influenced infectious diseases (Derban,

1975; Service, 1989). An influx of migrants coming to work on the development project itself may aggravate local conditions, causing housing shortages, overcrowding, or rising costs of living (Hunter et al., 1982), which may create additional difficulties controlling infectious diseases. Furthermore, migrants may not have been exposed previously to filariasis or other indigenous infectious diseases and may bring new diseases into the region, further increasing the chances of disease spread within communities.

The negative consequences of agricultural development tend to receive the most notice, but positive consequences of the projects are also observed. As Brinkmann (1994) noted, economic growth is often necessary to control, prevent, and cure infectious diseases. Funds must be made available for treatment of the ill and maintenance of sanitation systems, and massive education campaigns must often be mounted to ensure success in control efforts. The development projects themselves may provide the local economic prosperity necessary to provide the funds. Also, development projects generally require the establishment of an adequate road and transportation system, which increases the ease with which social services can be provided.

Hunter (1992) reviewed the impact of water control projects on the spread of lymphatic filariasis in northeast Ghana. He recognized four levels of disease within the study region: 1) nine chiefdoms with no reported cases, 2) 24 chiefdoms where infection was reported, but was not perceived to be a public health problem, 3) eight chiefdoms in which there was elevated disease transmission, and 4) two localized hot spots. All level 3 chiefdoms were recipients of foreign assistance in the form of agricultural development projects, which usually consisted of the construction of small village dams. The two hot spots were associated with two irrigation schemes involving 16 villages.

The village dams, built between 1958–1962 and generally of multipurpose clay-core construction, were used to provide a community water supply, livestock watering, fishing, dry season vegetable garden-

ing, and soil conservation. The purpose of the overall development project was to provide year-round food production to help prevent periodic periods of food shortage, to improve nutrition, to increase the health of livestock, and to create dry season cash incomes through crop sales. They led to increased availability of food, improvement of the physical environment because of easier access to water, a regular flow of income from cash crops, and other positive outcomes. However, there were also significant and serious negative consequences of the development activities.

The dams were constructed without any measures for the protection of human health, and after 25–30 years many of them were in moderate or serious disrepair. Even when they were nonfunctional, they still provided habitats for snails (which carry schistosomiasis, another prevalent disease) and mosquitoes (Hunter, 1992).

The health consequences of the development projects were significant. Hunter (1992) was able to show that the closer a house was to the main canals, the greater the frequency of filarial disease. This increase in disease was due to the greater intensity of mosquito biting near the water. Many residents complained that the number of mosquitoes was so high that it was impossible to sleep outside, even during the dry season (Hunter, 1992). The projects themselves were not necessarily the source of filariasis to the area; in some areas the disease was present long before irrigation was available, but problems with the maintenance of dams and irrigation systems exacerbated existing problems.

The development projects were an important factor involved in the increased problems with lymphatic filariasis, but there were other important players as well. These included the Ministry of Health, who tended to ignore the disease, as well as the victims themselves, who tended to passively accept their lot. This passivity was due to both a perceived lack of effective treatments, either from Western medical clinics or from traditional healers, and a general acceptance of people with the disease by the community (Gyapong et al., 1996; Hunter, 1992). Local political leaders, who had some

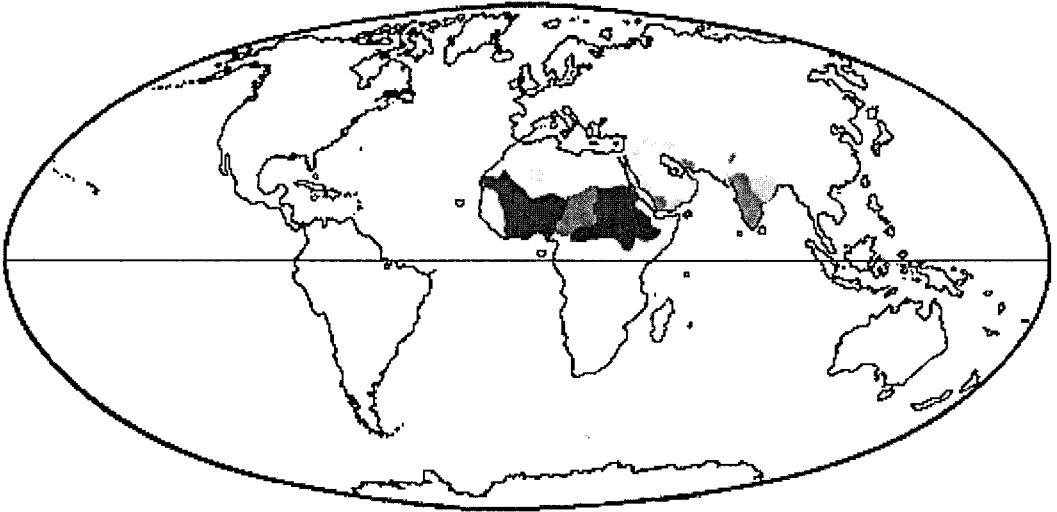


Fig. 3. Progress toward eradication of dracunculiasis. Darkest areas remain endemic at present; medium areas were endemic in the 1980s but have now been certified free of the disease; lightest areas became disease-free in the 1970s (data sources: Muller, 1971; Watts, 1998b).

knowledge of the political process and some hope of being heard but did nothing, and international funding organizations, who should have been addressing health issues during the development process, also shared part of the responsibility (Stock, 1992).

Human behavioral change and the eradication of dracunculiasis (guinea worm)

The life cycles and host-parasite interactions of most parasitic diseases, including lymphatic filariasis, are so complex that public health authorities must focus primarily on control of the diseases, thus reducing the incidence to a point where the disease is no longer a public health problem. This strategy differs fundamentally from strategies used for many bacterial and viral diseases such as smallpox or measles, where the goal is eradication of the disease, i.e., eliminating the organism that causes the disease (Hopkins and Ruiz-Tiben, 1991).

According to the World Health Organization (1996b), only one parasitic disease stands a reasonable chance of being eradicated in the near future, i.e., dracunculiasis

(also called guinea worm).³ The global campaign to eradicate this disease began with a declaration by the United States Centers for Disease Control that the 1980s would be the International Drinking Water Supply and Sanitation Decade. In 1981, the steering committee for this program included dracunculiasis eradication as part of a broader effort to bring safe drinking water to all people. In 1986, the World Health Assembly adopted a resolution to make dracunculiasis the first disease after smallpox to be targeted for eradication (Hopkins et al., 1993). The original target date for eradication was 1995, but the eradication campaign has not yet been successful, although there have been marked reductions in the prevalence of the disease.

The shrinking distribution of dracunculiasis is illustrated in Figure 3. Prior to 1920 the disease was endemic in large parts of equatorial Africa, the Arabian Peninsula, the Middle East, and southern Asia, and it was also found in local foci in the Caribbean, South America, the East Indies, and Russia.

³Some bacterial and viral diseases have also been targeted for elimination in the near future, including measles and polio (WHO, 1996b).

By the 1970s, the disease had died out in all regions with local foci and became more patchily distributed in eastern Africa, the Arabian Peninsula, the Middle East, and Asia outside of India and Pakistan (Muller, 1971). In 1982 there were 5–10 million cases of dracunculiasis distributed throughout sub-Saharan Africa, in the Arabian Peninsula, and in India. By 1995 there were only around 115,000 cases recorded, and all but 142 of these were in Africa (Watts, 1998b). In Africa the distribution covers a band, sometimes called the “Guinea worm belt,” that stretches across the continent from Mauritania to Ethiopia (Hunter, 1996; Richards and Hopkins, 1989; Watts, 1987a, 1998b; WHO, 1996b). The disease is endemic in many of the same areas as lymphatic filariasis, and, since its intermediate copepod host, a small crustacean, lives in small ponds and other slow-moving water sources, its distribution and prevalence have also been significantly affected by agricultural and water development activities.

Dracunculiasis is an ancient human infection that may have been the “fiery serpent” said by Moses to have attacked the Israelites on the shores of the Red Sea. A calcified worm has been detected in at least one Egyptian mummy, and the infection has been described in writings dating to at least Greek and Roman times. A major method used to treat the parasite, winding an emerging worm slowly around a stick to remove it from the body, was known in ancient times and is thought by some to be the origin of the medical symbol, the Staff of Aesculapius (Hopkins, 1993).

Female *Dracunculus medinensis* worms, the parasite responsible for dracunculiasis, are among the largest nematodes known and may be over 1 m long (Hunter, 1996; Watts, 1998a). They have a complex life cycle, which involves not only humans, but an intermediate host, the cyclops, a free-living freshwater crustacean. Larvae of the dracunculus worm are discharged into cyclops-containing water and eaten by the cyclops, where they undergo part of their development. Humans then drink cyclops-infested water and ingest the crustaceans along with the worm larvae they contain. The worm larvae travel through the body, eventually

ending up in various abdominal tissues, where they reproduce sexually. Male worms die and remain in the host, sometimes becoming calcified and preserved, while the fertilized female continues to grow and may contain one million embryos per 30 cm of her body. As the embryos begin to mature, the female travels through the body and usually ends up in the lower legs or feet. Once in position, the female worm secretes a powerful irritant, pierces the human’s skin, and raises a painful blister, which erupts upon immersion into water, expelling some of the embryos into the water. A small ulcer then forms, which allows continued periodic expulsions until all embryos are discharged. If the embryos are discharged into a cyclops-containing pond, some of them will be eaten by the cyclops and the cycle will begin again. The entire life cycle takes about 1 year (Bierlich, 1995; Hopkins et al., 1993; Hunter, 1996).

The throbbing pain associated with the blister and other more serious complications often incapacitate a person with a worm that is discharging embryos. Furthermore, the life cycle of the worm is highly seasonal, causing many people to be incapacitated simultaneously within a single community. This period of incapacitation commonly lasts for 90–100 days and can cause serious disruption to agricultural, economic, educational, and recreational activities (Edungbola et al., 1988; Hunter, 1996; Ilegbodu et al., 1991; WHO, 1996b).

The global campaign for the eradication of smallpox successfully relied on a single measure, immunization, to control and eradicate the disease, but this is not possible with dracunculiasis. There is no natural immunity to the disease, so as with most other parasitic diseases, it has not been possible to develop a successful vaccine. Also, no medicines are available to cure cases of the disease (Hunter, 1996; WHO, 1996b). Many regions of the world where dracunculiasis is still a public health problem are in remote areas with a very poor health infrastructure and little access to preventive care (Audibert et al., 1993). Furthermore, local beliefs about the disease can differ significantly from views of the Western health authorities. Dracunculiasis is considered a

natural and inescapable feature of life, and in some areas the worm itself is considered to be a part of normal human anatomy (Bierlich, 1995; Brieger and Kendall, 1992).

However, the biology and ecology of dracunculiasis are such that authorities are optimistic about its eventual eradication. Humans are the only mammalian host, so that it is necessary only to break the chain of transmission within human groups (Hopkins, 1983; Watts, 1998a,b). Furthermore, the disease is transmitted only by contaminated water, and multiple strategies of prevention are feasible even in remote locations. These methods include providing safe sources of drinking water, educating people about how to avoid contaminated water by using easily affordable cloth filters, encouraging infected persons to avoid wading in drinking water sources when the worm is emerging, and chemically treating cyclops-containing ponds (Audibert et al., 1993; Brieger et al., 1991; Diamenu and Nyaku, 1998; Hopkins and Ruiz-Tiben, 1991; Hunter, 1996; WHO, 1996b).

Several studies have shown that eradication of dracunculiasis will not be possible without active community involvement in surveillance and control activities (Akpovi et al., 1981; Brieger and Kendall, 1992, 1996; Edungbola and Watts, 1990; Hunter, 1996; Richards and Hopkins, 1989). Community involvement is crucial to the success of the control campaign for two reasons: the transmission of the parasite is closely tied to human activities, and the disease cannot be cured, but can only be prevented by altering human behaviors. In fact, human behaviors help to explain much of the geographic variability in prevalence of the disease and success in controlling its transmission.

Watts (1986a,b, 1987b) presented an excellent illustration of the impact of human behaviors on dracunculiasis transmission. Because water collection is the activity most frequently associated with contamination of water sources, she focused on studying daily water use patterns in India and Nigeria. Dracunculiasis is more prevalent in Africa than in India, and in addition, Indian males and females differ more in incidence of the disease than do African males and females. Watts (1986a,b, 1987b) showed that these

differences were due both to the nature of the water source (shallow pond vs. different types of well water) and to differences in the daily activities of males and females in the two regions. Indian males were exposed to untreated water when farming or attending festivals, but Indian females were relatively protected because of the isolation of *purdah*. In Africa, women and children were exposed when they collected the daily water rations, but men were infected at least as often by using the same ponds while they were out in their fields and during other times. Rates of infection were also influenced by patterns of daily mobility, which differed between the two regions.

The knowledge resulting from this and other work (e.g., Bierlich, 1995; Ilegbodu et al., 1991) has emphasized the importance of using local knowledge and customs in surveillance activities. Brieger and Kendall (1996) provide a practical example of how to do this. They conducted a detailed study of the market arrangements in a region in southwest Nigeria and used information gathered in the study to recruit native people to conduct surveillance in their home communities and report illnesses to health authorities. This method proved an effective way for the government to keep track of the disease in spite of a limited number of health workers.

However, there can also be hidden pitfalls. Diamenu and Nyaka (1998) observed that residents of two villages in Ghana were well aware that the use of nylon cloth filters could prevent transmission of the guinea worms, but they did not realize that they should actively seek to replenish their supplies when they ran out. Instead, they waited for health volunteers to deliver a new supply to them and if they became infected before the new filters were delivered, then they blamed this on the delays of the volunteers. They also thought that the "official" filters were specially treated with pesticides, so that they felt that the use of alternative filtering materials would not prevent disease transmission.

Health authorities have been successful in limiting the spread of dracunculiasis in many areas, with enormous health, social, and economic benefits for the populations

involved. For example, some areas have seen 40% increases in food production, increases of 25% in the amount of land under cultivation, and decreases from 60% school absenteeism rates to 13% absenteeism (WHO, 1996b). However, dracunculiasis has not yet been eradicated and the target date keeps getting pushed further into the future. Reasons for this include civil wars and other disturbances that interfere with surveillance and prevention activities, apathetic officials, inadequate funding, inertia accompanying requests to change longstanding customs and traditions, traditional views that the disease is not really a problem, and difficulties with promoting and maintaining water purification systems (Bierlich, 1995; Brieger and Kendall, 1996; Hopkins et al., 1993; Hunter, 1996). Even so, most authorities feel that the end is in sight, and that shortly after the arrival of the 21st century, dracunculiasis will share the fate of smallpox. Furthermore, the combination of attention to the biological nature of infectious disease organisms, the characteristics of the environment in which humans live, and the cultural behaviors that influence transmission of those organisms illustrated by the dracunculiasis eradication program points the way to success in dealing with other illnesses of modern society.

CONCLUSIONS

Several international associations have long been actively involved in improving the health and well-being of peoples of the tropics, but the outcome of public health campaigns in tropical regions of the world has not often been successful. Most infectious diseases appear to become much more serious problems as a consequence of development and modernization schemes than they were prior to the economic improvements, although few studies compare disease experiences before and after development (Hunter et al., 1982).

A number of reasons for the minimal success of these campaigns can be identified. First, most development projects, if they address health issues at all, focus on only a single disease. Yet, numerous diseases circulate simultaneously within a tropical

community. What works to limit the spread of one disease may increase the spread of a different disease, so that control measures may need to focus on community ecology rather than simple host-parasite interactions. For example, the Tono rice irrigation project in Ghana provided an abundance of good drinking water and essentially eliminated dracunculiasis in adjoining villages. But at the same time, provision of that same good drinking water resulted in the development of numerous new surface water sources, which significantly increased the number of mosquito breeding spots. This resulted in a significant increase in the prevalence of mosquito-borne filariasis. As Hunter (1997, p. 77) cogently stated, "the regional 'hot spot' of guinea worm disease was transformed into the regional 'hot spot' of elephantiasis." A similar event occurred in the Upper Region of Ghana, when extensive water control projects ameliorated the effects of guinea worm, but led to a tripling of the incidence of schistosomiasis (Hunter, 1981, 1997).

A second factor that may be partly responsible for the lack of success of public health efforts in the tropics is that, from a pathogen's point of view, when transmission becomes more difficult there may be selection for less virulent strains, so that hosts survive long enough for the pathogen to reproduce itself (Ewald, 1994; Wills, 1996). The newly evolved strains may well be able to better withstand attempts to control their reproduction, which may lessen the chances of success of control strategies.

The ecological community in a tropical environment does not just consist of pathogens, their vectors, and their nonhuman mammalian hosts; it includes humans as an essential component, both because humans can serve as hosts of a pathogen species and aid in its survival, and because in their activities humans both unintentionally and intentionally alter the environments of all species within the community. Yet most public health efforts have shortchanged the contributions of anthropologists and other human behavioral scientists and have focused much more on the ecological adaptations of the disease organism or its vectors (Dunn, 1979; Yacoub and Whiteford, 1994).

There are notable exceptions to this general pattern, including numerous studies of AIDS transmission, Susan Watts' work on the transmission of dracunculiasis, and the efforts spearheaded by Rita Colwell and colleagues to try to find culturally acceptable, affordable, and appropriate behaviors to stem the transmission of cholera. What unites many of these studies is that the researchers are knowledgeable not only about the underlying biology of humans, their pathogens, and vectors or alternate hosts, but also about characteristics of the human cultures of the regions and how elements of those cultures can be drawn upon to aid in controlling the diseases affecting the people.

Given the central role of humans in the ecology of most tropical communities, it is surprising that anthropologists have not been more intricately involved in the study of that ecology. And because of the close interface between biology and culture in the spread and maintenance of infectious diseases in human populations worldwide, biological anthropologists have the ideal training to study effectively not only how, when, and why diseases spread from person to person and how those diseases influence the survival and reproduction of the human host, but also the strategies that culturally diverse human populations have developed or could be convinced to adopt to deal with those diseases. Perhaps the 21st century will bring more biological anthropologists to the task of uniting biology and culture in the study of infectious disease ecology in human populations.

LITERATURE CITED

- Akpovi SU, Johnson DC, Brieger WR. 1981. Guinea worm control: testing the efficacy of health education in primary care. *Int J Health Educ* 24:229–237.
- Audibert M, Coulibaly D, Doumbo O, Kodio B, Soula G, Traore S. 1993. Social and epidemiological aspects of guinea worm control. *Soc Sci Med* 36:463–474.
- Barrett R, Kuzawa CW, McDade T, Armelagos GJ. 1998. Emerging and re-emerging infectious diseases: the third epidemiological transition. *Annu Rev Anthropol* 27:247–271.
- Barua D. 1992. History of cholera. In: Barua D, Greenough WB III, editors. *Cholera*. New York: Plenum. p 1–36.
- Beaver PC, Jung RC. 1985. *Animal agents and vectors of human disease*, 5th ed. Philadelphia: Lea & Febiger.
- Bierlich B. 1995. Notions and treatment of guinea worm in northern Ghana. *Soc Sci Med* 41:501–509.
- Birmingham ME, Lee LA, Ndayimirije N, Nkurikiye S, Hersh BS, Wells JG, Deming MS. 1997. Epidemic cholera in Burundi: patterns of transmission in the Great Rift Valley Lake Region. *Lancet* 349:981–985.
- Blake PA, Rosenberg ML, Florenca J, Costa JB, Quintino LP, Gangarosa EJ. 1977. Cholera in Portugal, 1974. II. Modes of transmission. *Am J Epidemiol* 105:344–348.
- Borota J. 1991. *Tropical forests: some African and Asian case studies of composition and structure*. Amsterdam: Elsevier.
- Bradley DJ. 1998. The influence of local changes in the rise of infectious disease. In: Greenwood B, De Cock K, editors. *New and resurgent infections: prediction, detection and management of tomorrow's epidemics*. Chichester: John Wiley & Sons, Ltd. p 1–15.
- Brieger WR, Kendall C. 1992. Learning from local knowledge to improve disease surveillance: perceptions of the guinea worm illness experience. *Health Educ Res* 7:471–485.
- Brieger WR, Kendall C. 1996. The Yoruba farm market as a communication channel in guinea worm disease surveillance. *Soc Sci Med* 42:233–243.
- Brieger WR, Ramakrishna J, Adeniyi JD, Sridhar MKC, Kale OO. 1991. Guinea worm control case study: planning a multi-strategy approach. *Soc Sci Med* 32:1319–1326.
- Brinkmann UK. 1994. Economic development and tropical disease. *Ann NY Acad Sci* 740:303–311.
- Brody H, Vinten-Johansen P, Paneth N, Rip MR. 1999. John Snow revisited: getting a handle on the Broad Street pump. *Pharos* 62:2–8.
- Brown PJ. 1981. Cultural adaptations to endemic malaria in Sardinia. *Med Anthropol* 5:313–339.
- Brown PJ. 1998. Introduction to "Cultural adaptations to endemic malaria in Sardinia". In: Brown PJ, editor. *Understanding and applying medical anthropology*. Mountain View, CA: Mayfield. p 79.
- Chen LC, Scrimshaw NS. 1983. *Diarrhea and malnutrition: interactions, mechanisms, and interventions*. New York: Plenum Press.
- Chin J, editor. 2000. *Control of communicable diseases manual*, 17th ed. Washington, DC: American Public Health Association.
- Clemetson DA, Moss GB, Willerford DM, Hensel M, Emonyi W, Holmes KK, Plummer F, Ndinya-Achola JO, Roberts PL, Hillier S, Kreiss J. 1993. Detection of HIV DNA in cervical and vaginal secretions. *JAMA* 269:2860–2854.
- Cohen J. 1997. Is an old virus up to new tricks? *Science* 277:312–313.
- Coimbra CEA Jr. 1988. Human factors in the epidemiology of malaria in the Brazilian Amazon. *Hum Org* 47:254–260.
- Colwell RR. 1996. Global climate and infectious disease: the cholera paradigm. *Science* 274:2025–2031.
- Colwell RR, Huq A. 1994. Environmental reservoir of *Vibrio cholerae*: the causative agent of cholera. *Ann NY Acad Sci* 740:44–54.
- Derban LKA. 1975. Some environmental health problems associated with industrial development in Ghana. *Ciba Found Symp* 32:49–71.
- Desowitz RS, Jenkins C, Anian G. 1993. Bancroftian filariasis in an isolated hunter-gatherer shifting horticulturalist group in Papua New Guinea. *Bull WHO* 71:55–58.
- Diamenu SK, Nyaku AA. 1998. Guinea worm disease—a chance for successful eradication in the Volta region, Ghana. *Soc Sci Med* 47:405–410.

- Dickens C. 1844. Martin Chuzzlewit. London: Chapman and Hall.
- Dowlatabadi H. 1997. Assessing the health impacts of climate change. *Climatic Change* 35:137-144.
- Dunn FL. 1972. Intestinal parasitism in Malayan aborigines (Orang Asli). *Bull WHO* 46:99-113.
- Dunn FL. 1976. Human behavioural factors in the epidemiology and control of *Wuchereria* and *Brugia* infections. *Bull Public Health Soc Malaysia* 10:34-44.
- Dunn FL. 1979. Behavioural aspects of the control of parasitic diseases. *Bull WHO* 57:499-512.
- Edungbola LD, Watts SJ. 1990. The elimination of dracunculiasis in Igbon, Oyo State, Nigeria: the success of self-help activities. *J Trop Med Hyg* 93:1-6
- Edungbola LD, Watts SJ, Alabi TO, Bello AB. 1988. The impact of a UNICEF-assisted rural water project on the prevalence of guinea worm disease in Asa, Kwara State, Nigeria. *Am J Trop Med Hyg* 39:79-85.
- Emch M. 1999. Diarrheal disease risk in Matlab, Bangladesh. *Soc Sci Med* 49:519-530.
- Epstein PR. 1999. Climate and health. *Science* 25:347-348.
- Epstein PR, Ford TE, Colwell RR. 1993. Marine ecosystems. *Lancet* 342:1216-1219.
- Evans RJ. 1992. Epidemics and revolutions: cholera in nineteenth century Europe. In: Ranger T, Slack P, editors. *Epidemics and ideas*. Cambridge: Cambridge University Press. p 149-173.
- Ewald PW. 1994. *Evolution of infectious disease*. Oxford: Oxford University Press.
- Faruque SM, Albert MJ, Mekalanos JJ. 1998. Epidemiology, genetics, and ecology of toxigenic *Vibrio cholerae*. *Microbiol Mol Biol Rev* 62:1301-1314.
- Gangarosa EJ, Tauxe RV. 1992. Epilogue: the Latin American cholera epidemic. In: Barua D, Greenough WB III, editors. *Cholera*. New York: Plenum. p 351-358.
- Garrett L. 1994. *The coming plague: newly emerging diseases in a world out of balance*. New York: Penguin.
- Glass RI, Black RE. 1992. The epidemiology of cholera. In: Barua D, Greenough WB III, editors. *Cholera*. New York: Plenum. p 129-154.
- Glass RI, Becker S, Huq MI, Stoll BJ, Khan MU, Merson MH, Lee JV, Black RE. 1982. Endemic cholera in rural Bangladesh, 1966-1980. *Am J Epidemiol* 116: 959-970.
- Grove DI. 1983. Selective primary health care: strategies for the control of disease in the developing world. VII. Filariasis. *Rev Infect Dis* 5:933-944.
- Gyapong M, Gyapong JO, Adjei S, Vlassoff C, Weiss M. 1996. Filariasis in northern Ghana: some cultural beliefs and practices and their implications for disease control. *Soc Sci Med* 43:235-242.
- Hahn BH, Shaw GM, De Cock KM, Sharp PM. 2000. AIDS as a zoonosis: scientific and public health implications. *Science* 287:607-614.
- Holmberg SD, Harris JR, Kay DE, Hargrett NT, Parker RDR, Kansou N, Rao NU, Blake PA. 1984. Foodborne transmission of cholera in Micronesian households. *Lancet* i(8372):325-328.
- Holmberg SD, Horsburgh CR, Ward JW, Jaffe HW. 1989. Biologic factors in the sexual transmission of human immunodeficiency viruses. *J Infect Dis* 160: 116-125.
- Holmes KK. 1995. Human ecology and behavior and sexually transmitted bacterial infections. In: Roizman B, editor. *Infectious diseases in an age of change: the impact of human ecology and behavior on disease transmission*. Washington, DC: National Academy Press. p 189-211.
- Hopkins DR. 1983. Dracunculiasis: an eradicable scourge. *Epidemiol Rev* 5:208-219.
- Hopkins DR. 1993. Dracunculiasis. In: Kiple KF, editor. *The Cambridge world history of human disease*. Cambridge: Cambridge University Press. p 687-689.
- Hopkins DR, Ruiz-Tiben E. 1991. Strategies for dracunculiasis eradication. *Bull WHO* 69:533-540.
- Hopkins DR, Ruiz-Tiben E, Kaiser RL, Agle AN, Withers PC Jr. 1993. Dracunculiasis eradication: beginning of the end. *Am J Trop Med Hyg* 49:281-289.
- Hunter JM. 1981. Past explosion and future threat: exacerbation of red water disease (*Schistosomiasis hematobium*) in the Upper Region of Ghana. *Geojournal* 5:305-313.
- Hunter JM. 1992. Elephantiasis: a disease of development in North East Ghana. *Soc Sci Med* 35:627-649.
- Hunter JM. 1996. An introduction to guinea worm on the eve of its departure: dracunculiasis transmission, health effects, ecology and control. *Soc Sci Med* 43: 1399-1425.
- Hunter JM. 1997. Bore holes and the vanishing of Guinea worm disease in Ghana's Upper Region. *Soc Sci Med* 45:71-89.
- Hunter JM, Rey L, Scott D. 1982. Man-made lakes and man-made diseases: towards a policy resolution. *Soc Sci Med* 16:1127-1145.
- Ilegbodu VA, Ilegbodu AE, Wise RA, Christensen BL, Kale OO. 1991. Clinical manifestations, disability and use of folk medicine in dracunculus infection in Nigeria. *J Trop Med Hyg* 94:35-41.
- Inhorn MC, Brown PJ. 1990. The anthropology of infectious disease. *Annu Rev Anthropol* 19:89-117.
- Jenkins C, Howard P. 1992. The use of ethnography and structured observations in the study of risk factors for the transmission of diarrhea in highland Papua New Guinea. *Med Anthropol* 15:1-16.
- Jenkins C, Dimitrakakis M, Cook I, Sanders R, Stallman N. 1989. Culture change and epidemiological patterns among the Hagahai, Papua New Guinea. *Hum Ecol* 17:27-57.
- Kaper JB, Morris JG Jr, Levine MM. 1995. Cholera. *Clin Microbiol Rev* 8:48-86.
- Kellman M, Tackaberry R. 1997. Tropical environments: the functioning and management of tropical ecosystems. London: Routledge.
- Kingsley LA, Detels R, Kaslow R, Polk BF, Rinaldo CR, Chmiel J, Detre K, Kelsey SF, Odaka N, Ostrow D, VanRaden M, Visscher B. 1987. Risk factors for seroconversion to human immunodeficiency virus among male homosexuals. Results from the Multicenter AIDS Cohort Study. *Lancet* i:345-349.
- Koo D, Aragon A, Moscoso V, Gudiel M, Bietti L, Carrillo N, Chojoj J, Gordillo B, Cano F, Cameron DN, Wells JG, Bean NH, Tauxe RV. 1996. Epidemic cholera in Guatemala, 1993: transmission of a newly introduced epidemic strain by street vendors. *Epidemiol Infect* 116:121-126.
- Kreiss JK, Coombs R, Plummer F, Holmes KK, Nikora B, Cameron W, Nguni E, Ndinya-Achola JO, Corey L. 1989. Isolation of human immunodeficiency virus from genital ulcers in Nairobi prostitutes. *J Infect Dis* 160:380-384.
- Lacey SW. 1995. Cholera: calamitous past, ominous future. *Clin Infect Dis* 20:1409-1419.
- Laderman C. 1975. Malaria and progress: some historical and ecological considerations. *Soc Sci Med* 9:587-594.
- Lilley B, Lammie P, Dickerson J, Eberhard M. 1997. An increase in hookworm infection temporarily associated with ecological change. *Emerg Infect Dis* 3:391-393. Also available at: <http://www.cdc.gov/ncidod/eid/vol3no3/lilley.htm>

- Longmate N. 1966. King Cholera: the biography of a disease. London: Hamish Hamilton.
- Mata L. 1982. Sociocultural factors in the control and prevention of parasitic diseases. *Rev Infect Dis* 4:871-879.
- Mata L. 1994. Cholera El Tor in Latin America, 1991-1993. *Ann NY Acad Sci* 740:55-68.
- McFalls JA Jr, McFalls MH 1984. Disease and fertility. New York: Academic Press.
- Meggors BJ. 1996. Amazonia: man and culture in a counterfeit paradise. Washington, DC: Smithsonian Institution Press.
- Morris RJ. 1976. Cholera 1832. New York: Holmes & Meier.
- Morse SS. 1995. Factors in the emergence of infectious diseases. *Emerg Infect Dis* 1:7-15. Also available at: <http://www.cdc.gov/ncidod/eid/vol1no1/morse.htm>
- Muller R. 1971. Dracunculosis and dracunculiasis. *Adv Parasitol* 9:73-151.
- Munro PM, Colwell RR. 1996. Fate of *Vibrio cholerae* O1 in seawater microcosms. *Water Res* 30:47-50.
- Murphy FA. 1998. Emerging zoonoses. *Emerg Infect Dis* 4:429-435. Also available at: <http://www.cdc.gov/ncidod/eid/vol4no3/murphy.htm>
- Nakazawa M, Ohmae H, Ishii A, Leafasia J. 1998. Malaria infection and human behavioral factors: a stochastic model analysis for direct observation data in the Solomon Islands. *Am J Hum Ecol* 10:781-789.
- National Research Council. 1993. Sustainable agriculture and environment in the humid tropics. Washington, DC: National Academy Press.
- Pan American Health Organization (PAHO). 1994. Cholera situation in the Americas. *Epidemiol Bull* 15:13-16.
- Pan American Health Organization (PAHO). 1996. New, emerging, and re-emerging infectious diseases. *Bull Pan Am Health Organ* 30:176-181.
- Pelling M. 1978. Cholera, fever, and English medicine, 1825-1865. Oxford: Oxford University Press.
- Plummer FA, Wainsberg MA, Plourde P, Jessamine P, D'Costa LT, Wamola IA, Ronald AR. 1990. Detection of human immunodeficiency virus type 1 (HIV-1) in genital ulcer exudate of HIV-1-infected men by culture and gene amplification. *J Infect Dis* 161:810-811.
- Pollitzer R. 1959. Cholera. Monograph no. 43. Geneva: World Health Organization.
- Preston R. 1994. The hot zone. New York: Anchor Books, Doubleday.
- Prothero PM. 1965. Migrants and malaria. London: Longmans Green and Co.
- Quinn TC. 1995. Population migration and the spread of types 1 and 2 human immunodeficiency viruses. In: Roizman B, editor. Infectious diseases in an age of change: the impact of human ecology and behavior on disease transmission. Washington, DC: National Academy Press. p 77-97.
- Quinn TC. 1996. Association of sexually transmitted diseases and infection with the human immunodeficiency virus: biological cofactors and markers of behavioral interventions. *Int J STD AIDS [Suppl]* 7:17-24.
- Quinn TC, Fauci AS. 1998. The AIDS epidemic: demographic aspects, population biology, and virus evolution. In: Krause RM, editor. Emerging infections: biomedical research reports. San Diego: Academic Press. p 327-363.
- Richards F, Hopkins D. 1989. Surveillance: the foundation for control and elimination of dracunculiasis in Africa. *Int J Epidemiol* 18:934-943.
- Roland C. 1985. Malaria. In: Marsh JH, editor. Canadian encyclopedia. Edmonton, Alberta: Hurtig. p. 1287.
- Rosenberg C. 1965. Cholera in nineteenth-century Europe: a tool for social and economic analysis. *Comp Stud Soc Hist* 8:45-463.
- Routh HB, Bhowmik KR. 1994. Filariasis. *Dermatol Clin* 12:719-727.
- Scrimshaw NS, SanGiovanni JP. 1997. Synergism of nutrition, infection, and immunity: an overview. *Am J Clin Nutr* 66:464-477.
- Service MW. 1989. Irrigation: boon or bane? In: Service MW, editor. Demography and vector-borne disease. Boca Raton, FL: CRC Press. p 237-254.
- Shell-Duncan B, Wood JW. 1997. The evaluation of delayed-type hypersensitivity responsiveness and nutritional status as predictors of gastro-intestinal and acute respiratory infection: a prospective field study among traditional nomadic Kenyan children. *J Trop Pediatr* 43:25-32.
- Shiba T, Hill RT, Straube W, Colwell RR. 1995. Decrease in culturability of *Vibrio cholerae* caused by glucose. *Appl Environ Microbiol* 61:2583-2588.
- Shiple AE. 1908. Pearls and parasites. London: John Murry.
- Snow J. 1965. Snow on cholera. Being a reprint of two papers. New York: Hafner.
- Speck RS. 1993. Cholera. In: Kiple KF, editor. The Cambridge world history of human disease. Cambridge: Cambridge University Press. p 642-649.
- Stelma FF, Talla I, Polman K, Niang M, Sturrock RF, Deedler AM, Gryseels B. 1993. Epidemiology of *Schistosoma mansoni* infection in a recently exposed community in northern Senegal. *Am J Trop Med Hyg* 49:701-706.
- Stock R. 1992. Who to blame? What to do? *Soc Sci Med* 35:645-648.
- Sweet RL, Blankfort-Doyle M, Robbie MO, Schachter J. 1986. The occurrence of chlamydial and gonococcal salpingitis during the menstrual cycle. *JAMA* 255:2062-2064.
- Tauxe RV, Blake PA. 1992. Epidemic cholera in Latin America. *JAMA* 267:1388-1390.
- Tauxe RV, Mintz ED, Quick RE. 1999. Epidemic cholera in the New World: translating field epidemiology into new prevention strategies. *Emerg Infect Dis* 1:141-146. Also available at: <http://www.cdc.gov/ncidod/eid/vol1no4/tauxe.htm>
- Trostle JA, Sommerfeld J. 1996. Medical anthropology and epidemiology. *Annu Rev Anthropol* 25:253-274.
- Wasserheit JN. 1995. Effect of human ecology and behavior on sexually transmitted diseases, including HIV infection. In: Roizman B, editor. Infectious diseases in an age of change: the impact of human ecology and behavior on disease transmission. Washington, DC: National Academy Press. p 141-156.
- Watts SJ. 1986a. Human behaviour and the transmission of dracunculiasis: a case study from the Ilorin area of Nigeria. *Int J Epidemiol* 15:252-256.
- Watts SJ. 1986b. The comparative study of patterns of guinea worm prevalence as a guide to control strategies. *Soc Sci Med* 23:975-982.
- Watts SJ. 1987a. Dracunculiasis in Africa in 1986: its geographic extent, incidence, and at-risk population. *Am J Trop Med Hyg* 37:119-125.
- Watts SJ. 1987b. Population mobility and disease transmission: the example of guinea worm. *Soc Sci Med* 25:1073-1081.
- Watts SJ. 1998a. An ancient scourge: the end of dracunculiasis in Egypt. *Soc Sci Med* 46:811-819.

- Watts SJ. 1998b. Perceptions and priorities in disease eradication: dracunculiasis eradication in Africa. *Soc Sci Med* 46:799–810.
- Watts S, Khallaayoune K, Bensefia R, Laamrani H, Gryseels B. 1998. The study of human behavior and schistosomiasis transmission in an irrigated area in Morocco. *Soc Sci Med* 46:755–765.
- Wawer MJ, Podhisita C, Kanungsukkasem U, Pramualratana A, McNamara R. 1996. Origins and working conditions of female sex workers in urban Thailand: consequences of social context for HIV transmission. *Soc Sci Med* 42:453–462.
- Weber JT, Mintz ED, Cañizares R, Semiglia A, Gomez I, Sempértegui R, Dávila A, Greene KD, Pühr ND, Cameron DN, Tenover FC, Barrett TJ, Bean NH, Ivey C, Tauxe RV, Blake PA. 1994. Epidemic cholera in Ecuador: multidrug resistance and transmission by water and seafood. *Epidemiol Infect* 112:1–11.
- Williams-Blangero S, Blangero J, Upreti RP, Adhikari BN, Upadhyaya RP, Jha B, Rai SK, Subedi J, Robinson ES. 1995. Genetic analysis of roundworm burden in the Jirels of Nepal. *Am J Trop Med Hyg* 53:110–111.
- Williams-Blangero S, Blangero J, Bradley M. 1997. Quantitative genetic analysis of susceptibility to hookworm infection in a population from rural Zimbabwe. *Hum Biol* 69:201–208.
- Williams-Blangero S, Subedi J, Upadhyaya RP, Manral DB, Khadka K, Jirel S, Robinson ES, Blangero J. 1998. Attitudes towards helminthic infection in the Jirel population of eastern Nepal. *Soc Sci Med* 47:371–379.
- Wills C. 1996. *Yellow fever, black goddess: the coevolution of people and plagues*. Reading, MA: Addison-Wesley.
- Wood CS. 1979. *Human sickness and health*. Mountain View, CA: Mayfield.
- World Health Organization (WHO). 1974. Third report of the Expert Committee on Filariasis. WHO Tech Rep Ser 542.
- World Health Organization (WHO). 1984. Lymphatic filariasis. Fourth report of the WHO Expert Committee on Filariasis. WHO Tech Rep Series 702.
- World Health Organization (WHO). 1987. Control of lymphatic filariasis: a manual for health personnel. Geneva: WHO.
- World Health Organization (WHO). 1996a. Cholera in 1995. *Wkly Epidemiol Rec* 71:157–163.
- World Health Organization (WHO). 1996b. The world health report 1996: fighting disease, fighting development. Report of the Director-General. Geneva: WHO.
- World Health Organization (WHO). 1997. Cholera in 1996. *Wkly Epidemiol Rec* 72:229–235.
- World Health Organization (WHO). 1998a. Cholera in 1997. *Wkly Epidemiol Rec* 73:201–207.
- World Health Organization (WHO). 1998b. Reducing mortality from major killers of children. WHO fact sheet no. 178. Geneva: WHO.
- World Health Organization (WHO). 1999. Cholera in 1998. *Wkly Epidemiol Rec* 74:257–263.
- World Health Organization (WHO). 2000a. Global cholera update. <http://www.who.int/emc/diseases/cholera/choltbl999.html>
- World Health Organization (WHO). 2000b. Lymphatic filariasis: situation in 2000. *Wkly Epidemiol Rec* 75:206–208.
- World Resources Institute. 1996. *World resources 1996–1997*. Oxford: Oxford University Press.
- Yacoob M, Whiteford LM. 1994. Behavior in water supply and sanitation. *Hum Org* 53:330–335.