Medical Anthropology

Contemporary Theory and Method

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CHAPTER 11

DISEASE, ECOLOGY, AND HUMAN BEHAVIOR

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Disease is an inevitable part of life, and coping with disease is a universal aspect of the human experience. During the course of their lives, all humans harbor infections by disease organisms and suffer the consequences of those infections. The experience of disease, by individuals or whole populations, is as inescapable as death itself. Yet the particular diseases that afflict people, as well as the way in which symptoms are interpreted and acted upon, vary greatly among cultures. Understanding the nature of interactions between disease and culture can be a productive way of understanding humanity and is therefore an important topic in medical anthropology. From an anthropological perspective, diseases cannot be explained as purely things in themselves; they must be analyzed and understood within a human context—that is, in relation to ecology and culture.

The distribution of disease in a population is neither constant nor random. Both the type and severity of diseases that characteristically afflict members of a population vary significantly among societies as a result of differences in culture, ecological setting, and historical period. More important, within a single society, there may be striking variations in terms of the nature and severity of diseases that afflict individuals of different ages, sexes, social classes, and ethnic groups. Understanding these social epidemiological patterns presents a challenge to medical anthropologists because disease distributions often reflect culturally coded behaviors.

Culture plays a major role in determining the patterns of disease and death in a population, for two reasons. First, culture may shape important behaviors (with respect to diet, activity patterns, sexual practices, and so forth) that predispose individuals to certain diseases. Second, through culture, people actively change the nature of their environment, often in ways that affect their health. The archeological and historical record clearly demonstrates that the environmental changes caused by humans can have profound effects, positive and negative, on

disease rates. Although humans have a dual system of inheritance through both genes and culture, it is culture that is the the primary mechanism for survival. Culture is a mechanism of adaptation to environmental threats, such as diseases, that act as agents of natural selection in the evolution of both human biology and culture.

Ecology is the study of the relationship of organisms in an environment. Human societies coinhabit their environment with many other organisms, including those producing disease. An ecological approach to human health and illness emphasizes the fact that the environment and its health risks are, to an extent, created by the culture. In many cultures, people think of themselves as masters of their environment because within the food chain they exploit so many plants and animals as sources of energy and nutrients. Yet at the same time humans are being exploited by microorganisms, including those that cause disease, as a source of food and shelter.

The study of disease and human behavior in an ecological setting is a fundamental task for medical anthropology. The approach contributes to basic and applied research in the field by providing a strategy for answering some of the major questions raised by general anthropology and epidemiology. For example, it can be applied to anthropological questions concerning the interaction of biology and culture in human evolution or to questions of why particular cultural behaviors may make sense and be retained in an ecological setting. This type of research strategy is truly biocultural and can help to bridge the gap between biological and cultural anthropology. In epidemiology, the contribution of an anthropological focus on human behavioral patterns can aid in unraveling fundamental questions of disease causality. The study of disease, ecology, and behavior also has important implications for public health programs. Through the study of behavioral patterns related to the social epidemiology of disease, it is possible to design health programs that are both effective and culturally acceptable. Moreover, the ecological approach can help to anticipate the health implications of technological change and political-economic policies.

This chapter discusses basic concepts and methods in the study of disease, ecology, and behavior. Although it covers both basic and applied research issues, it is not an exhaustive review of the literature. It summarizes many illustrative examples of this kind of research, focusing on particular diseases in particular cultural settings. These examples emphasize infectious diseases for several reasons: first, the etiology (causation) of these diseases is the best understood; second, the ecological approach described here is most applicable to this category of disease (although it is also applicable to the study of chronic diseases with complex etiologies, which are characteristic of industrialized societies); and, finally, infectious diseases still represent the major cause of morbidity and mortality in nonindustrial societies.

This chapter is divided into three general sections. First, we introduce some of the most important theoretical concepts. Second, from a diachronic perspective, we discuss the role of disease in biocultural evolution. And finally, using contemporary examples, we develop a typology of diseases and analyze examples

of the interaction of cultural behaviors and disease prevalence rates, from both a micro- and macrosociological perspective. The study of disease, ecology, and behavior remains relatively undeveloped in medical anthropology; thus, there is much opportunity for important research to be done. The anthropological study of disease, we believe, can contribute to both the understanding of the human experience and to the solution of basic human problems.

THE THEORETICAL ORIENTATION OF DISEASE ECOLOGY

What Is Disease?

In the enormous literature of biomedicine, there is no universally accepted definition of disease. Like many theoretically important concepts, "disease" is essentially left undefined and is used in ambiguous ways. For example, it is often defined by what it is not. It is generally seen as a failure of normal physiological activities and a departure from a state of health. But such a definition is uninformative because within it is hidden the problematic concept of normal. Yet it is clear that normality must be considered as culturally constructed, and hence variable. For example, conditions that have been considered as normal in particular populations include persistent diarrhea (Desowitz 1981), malaria (Ackerknecht 1945), the bloody urine of schistosomiasis (Heyneman 1979), and the skin discolorations of pinta (Ackerknecht 1943). "Health," of course, is so notoriously difficult to define that the World Health Organization's (WHO) utopian phrase, "a state of complete physical, mental, and social well-being," has little use for those who wish to measure health.

Medical sociologists have often made the distinction between disease and illness. Disease refers to a set of objective, clinically identifiable symptoms, while illness refers to an individual's perception of those symptoms. This perception motivates the individual to seek medical care or to assume the sick role (Mechanic 1978). A persistent paradox in modern medical systems is the fact that many patients seeking medical care (those who have an illness) do not have any identifiable disease, while at the same time, many people with disease do not define themselves as ill and thus do not seek medical help (Zola 1972). Although the distinction between disease and illness is useful, it is based on a questionable assumption that the biomedical definition of disease is objective and culture free.

When defining disease, it is useful to compare the conceptions of the layperson, the biomedical specialist, and the disease ecologist. Most people, even in complex societies, conceive of diseases as invisible entities "out there," which attack victims and cause discomfort, loss of vitality, and even death. Although diseases can be named, they cannot usually be controlled by ordinary individuals. From this emic perspective, there is little difference between a disease caused by a "germ" and one caused by supernatural agents. In either case, the sick person may be a completely innocent victim of the disease (as in most pediatric cases) or may have partly encouraged it to attack by way of irresponsible behavior

(such as breaking postpartum taboos or smoking cigarettes). For most people, the large number of unknown diseases "out there" makes the world a dangerous place. In this regard, disease can function as a symbolic metaphor for social and cultural issues (Sontag 1978).

For the practitioner of biomedicine, disease is the expression of pathology alone. Diseases can be identified by discrete sets of signs and symptoms or by diagnostic tests. Diseases can be categorized, within the taxonomy of biomedicine, primarily in terms of the biological characteristics of the etiological agents. Both the taxonomic and diagnostic systems of biomedicine, however, are based on certain cultural assumptions about causality and the nature of reality. For example, in the clinical setting, the disease often takes on an existence (from the viewpoint of the practitioner) quite apart from the patient; the disease is treated rather than the person. The idea that biomedicine is itself a cultural construction of reality is a basic insight of "critical medical anthropology" (Baer et al. 1986).

In contrast, from an ecological perspective, disease does not exist as a thing in and of itself. Disease is a process triggered by an interaction between a host and an environmental insult, most often a pathogenic organism or "germ." Disease is one possible outcome of the relationship between the host and the potential pathogen. Since the advent of bacteriology and germ theory, it has been recognized that infection is a necessary but not sufficient condition for disease to occur. Normal, healthy individuals typically harbor many different colonies of viruses and bacteria that are not pathogenic (disease producing) primarily because these agents are held in check by the human immune system. Indeed, individuals are constantly being challenged by microorganisms in their environment (Burnet and White 1978); disease occurs only when the host's immunological system is unable to keep pace with the reproduction of the pathogen, a process that can be accelerated through malnutrition or immunosuppression (Scrimshaw et al. 1968).

According to Jacques May in his classic volume, *The Ecology of Human Disease*, disease is "very simply that alteration of living tissues that jeopardizes their survival in their environment" (1958:1). This means that disease is the temporary expression of maladjustment of an individual trying to cope with the challenges of his or her environment. In this model the eventual outcome of this maladjustment is, on the level of the population, a mutual accommodation between host and pathogen.

What Is Ecology?

Ecology is the study of the relationship between a species and its total environment. Most often considered a subfield of biology, ecology deals with the interactions between organisms and their environment on the population, community, and ecosystem levels of organization (Ehrlich et al. 1973; Orlove 1980). Integral to most ecological studies is the idea that the complex set of interactions between organisms in an ecological niche (territory) makes up a system (Odum 1971). This "ecosystem" includes not only natural resources (water and minerals, for example) but also plants, animals, and humans. Two of the assumptions of this model are that the ecosystem is maintained through mutually dependent interactions between members of the system and that the common goal of the various species in the system is homeostasis. The primary benefit of homeostatic balance is the prevention of environmental degradation.

In this view, human activities such as agriculture create imbalances in natural ecosystems. Humans are capable not only of ecological change but, potentially, of ecological destruction. There is no doubt that humans have often been responsible for radical changes in their environment and that such ecological changes have had negative effects on health. But this observation does not require the assumption of a cooperative, mutually dependent "system" in nature, which would maintain itself if not for human disruption. Modern evolutionary theorists, in fact, question whether community ecosystems are a biological reality at all (Ayala 1983). With few exceptions, the apparent "system" may be nothing more than the sum total of individual behaviors aimed at the maximization of reproductive success.

What Is Disease Ecology?

Disease ecology focuses on the interactions between two organisms: the pathogen and the host. Unlike the more general ecological approach, however, the emphasis is not on the harmonic cooperation between humans and agents of disease (Armelagos et al. 1978). This is because diseases are most often viewed as serious threats to human health. Humans are, from the viewpoint of a disease organism, the environment in which the disease organism lives and reproduces and to which it must adapt. The disease ecologist tries, metaphorically, to understand the disease organism's "worldview" and its adaptive strategies for survival and reproduction. The ecological model is much more easily applied to diseases caused by infectious agents than the diseases of life-style that characterize modern affluent populations.

The notion of adaptation is a fundamental principle of disease ecology—adaptation from both the perspective of the disease agent and the host. As Richard Lieban states, "health and disease are measures of the effectiveness with which human groups, combining cultural and biological resources, adapt to their environments" (1973:1031).

Disease ecology is one of the foundations of medical anthropology and is, by definition, a biocultural enterprise, as are the closely related disciplines of medical geography and epidemiology. The study of disease ecology both allows and requires a bridging of the biological and cultural paradigms in anthropology. This approach in medical anthropology owes much to the pioneering work of A. Alland, whose book, *Adaptation in Cultural Evolution* (1970), used evolutionary theory to examine how cultural behaviors enhance hygiene, health and reproductive fitness. While an emphasis on disease ecology may be widespread in the current teaching of medical anthropology (McElroy and Townsend 1989), we agree with David Landy (1983) that the ecological approach has been more an unquestioned paradigmatic orientation than the basis of actual research.

DISEASE AND BIOCULTURAL EVOLUTION

Evolution refers to the process of change over time. Human evolution includes changes in biology, through modification of gene frequencies, and cultural forms. It is the latter, cultural change, that accounts for the tremendous success of our species. An important but often misunderstood point is that evolutionary change does not imply progress; evolution, whether biological or cultural, does not necessarily mean that things get better. Furthermore, evolutionary change occurs only in relation to a particular environment. The classic example of this fact is the evolution of the gene for the sickle-cell trait that was context dependent on an environment characterized by *Plasmodium falciparum* malaria.

Natural selection is the primary driving force for evolutionary change in biological and cultural systems. This means that, in general, traits that improve the chance of survival and reproduction in an environment will be maintained or increase in frequency. Conversely traits that result in premature death or decreased fertility will become very rare or disappear in the long run. These generalizations do not imply that biological or cultural traits are always able to solve environmental problems. It is important to remember that selection occurs only upon preexisting variations of genetic or cultural forms; in evolution, necessity is not the mother of invention. In both biological and cultural evolution, traits are selected that enhance reproductive fitness. However, there are important differences between these two processes because biological and cultural evolution differ in terms of units of variation, sources of variation, and measures of adaptive value (for an extended discussion of these differences, see Brown 1986).

The actual agents of natural selection are seldom specified in studies of biocultural evolution, but there appears to be tacit agreement that the most important factors are ones that cause differential mortality. Five major categories of agents of natural selection should be considered: diseases, food shortages, trauma and accidents, predation and competition with conspecifics (same, similar or related species); and thermoregulation. The geneticist J. B. S. Haldane (1949) was one of the first theoreticians to emphasize the importance of the first source, disease, in evolution. Disease is important in human biocultural evolution for the simple fact that it causes death—or, to put it in Darwinian terms, it results in differential rates of mortality and fertility.

Disease and Evolution: Three Mechanisms

There are three main mechanisms through which disease affects human biological and cultural evolution: large-scale mortality from epidemics, excess mortality from endemic diseases, and parasitism (Brown 1987).

The primary way in which disease affects the process of natural selection has been through the massive mortality caused by epidemics. Evolution can occur very rapidly in the context of the enormously strong selective pressure of an epidemic because it carries the possibility of extinction (Haldane 1949). A well-studied example of this phenomenon involves myxomatosis, a viral disease of rabbits that was introduced in 1950 to the wild rabbit population of Australia as

a means of controlling overpopulation (Fenner and Ratcliffe 1965). In the first year after introduction, the die-off of the rabbit population was 99.8 percent; in the second year, it was 90 percent; and, by the seventh year, it was only 25 percent. Fifteen years following the introduction of the disease, the rabbit population was only one-fifth its original size, but the mortality due to myxomatosis was nearly zero. This change was the result of powerful selection of both the rabbit population and the virus. If rabbits had been eradicated in Australia, then the virus would have become extinct there too; thus, mutual adaptation was to the advantage of both species. The myxomatosis example illustrates an important process in which virulent epidemic diseases eventually become benign endemic diseases in a population through the process of mutual accommodation.

The massive mortality associated with epidemic diseases has also had an important effect on human cultural history. The most comprehensive treatment of this theme is in W. H. McNeill's landmark volume, *Plagues and Peoples* (1976), in which he demonstrates the active role that epidemics have played in the expansion of empires throughout history. Such expansion was facilitated, McNeill argues, by the "confluence of disease pools"; that is, infectious diseases were unwittingly spread from state-level societies with a complex repertoire of endemic childhood diseases to smaller and simpler societies, for which the introduction of these new diseases brought massive population losses and socioeconomic disorganization.

McNeill argues that disease played a crucial role in accelerating the conquest, subjugation, and acculturation of tribes and chiefdoms. This historical process depended on the biological transition of introduced epidemic diseases into local endemic diseases characteristic of childhood. In addition to diseases of obvious historical importance, like plague, smallpox, and syphilis, McNeill suggests that less dramatic agents such as measles, chicken pox, diphtheria, and unnamed respiratory and gastrointestinal disorders (which he generally calls "microparasites") followed this pattern. The well-known example of infectious epidemics and the depopulation of North American Indian groups (sometimes before actual face-to-face contact with Europeans) is a case in point: disease played an important role in the saga of how the West was won (see Krech 1978).

The second mechanism by which disease affects the processes of natural and cultural selection is through gradual population losses from endemic diseases. Endemic diseases can have important demographic effects that are often not recognized by the population itself. High infant mortality rates, for example, may be considered an uncontrollable fact of life and may be compensated for through high birthrates and associated cultural beliefs regarding child spacing and ideal family size. The negative demographic and socioeconomic effects of endemic childhood diseases can be significant. For example, endemic malaria in a tropical environment usually has a low case-fatality ratio (approximately 1 death per 100 cases), especially for adults (Bruce-Chwatt 1980). However, because malarial infections are so widespread, debilitating victims who eventually succumb to other diseases, the demographic impact of endemic malaria can be remarkably strong. This can be seen in "natural experiments" of malaria erad-

ication in which health improvements have resulted in sudden and unprecedented increases in population growth rates. Such effects have been seen following malaria control programs in Sri Lanka (where malaria control appears to have accounted for 26 percent of the increase in population growth rates [Gray 1974]) and in Sardinia (Brown 1986). In a different type of study, conducted in communities with endemic malaria along the northern shore of Lake Victoria, Kenya, the single health intervention of insecticide spraying resulted in a 50 percent overall reduction in child and infant mortality rates in four years (Payne et al. 1976).

The third mechanism through which disease can affect the process of natural selection is parasitism, a concept generally neglected by medical anthropologists (Brown 1987). Parasitism refers to an evolutionary strategy in the struggle for life in which the underlying problems are eating and being eaten. The relationship of hosts and parasites is usually one of mutual adaptation through interactions that produce a state of equilibrium. It is disadvantageous for a parasite to kill its host, although most parasites cause some degree of real damage to their hosts, manifested through diseases that may affect the growth rate of the host population (Anderson and May 1978).

It has often been assumed, particularly in the literature of public health, that parasitic diseases sap the energy of individuals and therefore limit the possibility of cultural advancement. This vicious circle argument—that "people are sick because they are poor and they get poorer because they are sick" (Winslow 1951)—is an underlying tenet of international health policy. There is much research yet to be done by medical anthropologists to understand better the effects of parasitism on human behavior.

Disease and Cultural Evolution

Cultural systems have evolved from the original human life-style of food foraging to modern industrialized states. Anthropologists have long recognized a general pattern of cultural evolution from simple to complex societies and from low-energy- to high-energy-harnessing economies (Sahlins and Service 1960). This is simply a pattern of general historical change (evolution) from prehistory to the present that has been characterized by four processes: increased population size, expansion of technology, increased social inequality, and increased transformation of the environment.

Disease ecology and epidemiological patterns are correlated with stages in cultural evolution (Armelagos and Dewey 1970; McElroy and Townsend 1989). In general, food-foraging populations throughout history had relatively low rates of infectious diseases due to their small population size and mobility, although the total morbidity and mortality from disease varied with ecological setting (Dunn 1968). Diseases that require larger contiguous populations in order to be transmitted (such as measles, mumps, smallpox, and influenza) were probably nonexistent until the introduction of agriculture and preindustrial cities. Paleopathological studies of the health implications surrounding the introduction of agriculture have demonstrated that in virtually every society on record, the new

economic form was associated with increases in malnutrition and infectious disease (Brothwell and Sandison 1967; Cohen and Armelagos 1984). The high prevalence of infectious diseases in the preindustrial cities of ancient civilizations resulted in consistent labor shortages and population decline (McNeill 1976; Knauft 1987). Today, despite advances of biomedical science since the eighteenth century, modern complex societies are characterized by a new epidemiological pattern—the Western diseases of obesity, hypertension, cardiovascular disease, and so forth (Trowell and Burkitt 1981; Brown and Konner 1987). In short, throughout history, new cultural patterns brought new disease problems.

Biological and Cultural Adaptations to Disease

The concept of adaptation refers to a fundamental process of evolution in which particular traits are selected in a given environment because they increase an organism's chances for survival and reproduction. Adaptation implies that the environment poses certain "problems," which organisms in the environment must "solve." Natural selection is the mechanism by which such solutions are found (Lewontin 1978, 1984). The concept does not imply that the resulting biological or cultural traits are the only or optimal solutions to environmental problems. Most important, it does not mean that adaptations exist for every environmental problem (or disease). Indeed, the fact that cultural behaviors play a direct role in disease transmission and can hinder disease control programs is an important theme in the third section of this chapter.

Although primarily used in evolutionary biology, the concept of adaptation has been central to discussions in both medical anthropology and cultural ecology (Alland 1966, 1970; Alland and McCay 1973; Brown 1986; Ellen 1982; Landy 1983a; Netting 1965; Rappaport 1976, 1979). Anthropologists have been concerned with describing examples of the successful outcome of adaptations on a genetic or cultural level. In terms of genetic adaptation to disease, the most comprehensive work focuses on polymorphisms of the hemoglobin system, such as sickle-cell trait and other hereditary disorders of the blood, which are most likely the result of natural selection by malaria.

Similarly, the human immune system can be viewed as the product of genetic adaptation to disease pressures. A primary biological characteristic of the immune system is its adaptability; in other words, it is a generalized mechanism capable of providing protection against potential (yet-to-evolve) pathogens (Baker 1984). The evolution of the immune system is the product of human adaptation to disease; at the same time, the immune system has required that disease organisms adapt to their host-victims. This pattern of mutual adaptation is an important feature of the relationship between humans and disease (Dubos 1965). From this perspective, agents of acute, lethal infectious diseases are less well adapted to their human environment than the agents of endemic or chronic infections. Thus, more lethal forms of a disease are probably younger and have had a shorter history of adaptation to the host.

Cultural adaptations to diseases include behaviors and beliefs that function to limit morbidity and mortality in two general ways. First, some behaviors and

beliefs have preventive functions by reducing exposure to disease organisms for certain segments of society. Second, others involve appropriate therapy for diseases, generally termed ethnomedicine.

Particular patterns of social organization and behavior may have latent functions in preventing the spread of disease, even though their conscious purpose may be unrelated to health. Examples of such preventive adaptations include settlement patterns in elevated locations removed from malaria-endemic lowlands (Brown 1981); storage of night soil before its use as fertilizer (Alland 1970); and traditional laundry soaps with molluscicidal properties in schistosomiasis-endemic areas (Kloos and McCullough 1982). Another way of looking at this is to consider the ways in which the presence of disease in various ecological settings has limited economic or productive possibilities. For example, the presence of endemic malaria may make lowland areas unsuitable for human habitation, thereby restricting subsistence strategies that are too costly in terms of health.

In contrast, the cultural behaviors related to curative medicine are usually the result of conscious attempts to control sickness and death. Yet there is little evidence to suggest that either traditional curative medicine or modern scientific medicine has had any significant impact on general health or fecundity. T. McKeown (1976a, 1976b) has conclusively demonstrated that changes in life-style (such as better sanitation, nutrition, and birth control), and not the advancement of medicine, best account for improvements in health over the last two centuries.

Examples of Cultural Adaptations to Disease: Malaria

Anthropologists interested in cultural adaptations to disease have paid particular attention to the problem of malaria. This may be due to the fact that malaria has reportedly killed more people than any other single disease (Livingstone 1971) and that genetic adaptations to this disease have been well studied. The importance of human behavioral factors in malaria control has long been recognized by malariologists (see bibliography by Sotiroff-Junker 1978). The identification of culturally adaptive behaviors to malaria requires knowledge of the biological etiology of the disease, the social distribution of the disease, and local variation in the ecology of insect vectors of the disease.

The medical anthropological literature includes a number of examples of cultural adaptations to malaria. J. M. May (1960), for example, has suggested that the traditional house type of the hill tribes of Vietnam, where cooking and sleeping platforms are elevated on stilts, reduced exposure of the population to the mosquito vector *A. minimus*, which has a flight ceiling of about 10 feet.

P. J. Brown, in his analysis of traditional Sardinian culture (1981), argues that the nucleated settlement pattern, particularly the pastoral pattern of inverse transhumance (flock movement to high elevations in summer), reduces exposure to malaria. In the ecological context of a nondomestic vector (*A. labranchiae*), social groups that are expected to stay within the confines of the nucleated settlement have the lowest rates of the disease. In addition, traditional behaviors based on the folk etiology of miasma also have a preventive effect (Brown 1986).

C. P. MacCormack (1984, 1985) has studied cultural traditions and behavioral factors related to malaria control in Tanzania. This work has led to further explorations of preventive adaptations, which reduce exposure to the vector. In Sierra Leone, for example, individuals envelope themselves at night, the prime mosquito feeding period, in a thick cotton cloth, which is inpenetrable by the local malaria vector (*A. albimanus*). Similarly, in many other parts of Africa, people traditionally sleep under locally woven bed nets, which can be impregnated with mosquito repellent.

In a different vein, S. V. Katz and J. Schall (1979) have examined the practice of fava bean consumption and its relationship to malaria in the circum-Mediterranean region (where populations have high gene frequencies of glucose–6-phosphate dehydrogenase ([G6Pd]) deficiency). This dietary staple appears to have antimalarial qualities. However, for males with the G6Pd deficiency trait, fava bean consumption can trigger a potentially fatal hemolytic crisis. Through an analysis of the biochemistry of the gene-bean interaction, Katz and Schall argue that the combination of nonexpressed gene and fava bean consumption provides significant protection from malaria death in females.

A final example of cultural adaptations to malaria is the herbal medicines of the Hausa of Nigeria. N. L. Etkin and P. J. Ross (1982a, 1982b) have identified thirty-one "antimalarial plant medicines" used by either herbal specialists or the general population in response to the symptoms of malaria. Some of these medicinal plants have been shown to change the oxidation-reduction status of red blood cells, a physiological condition known to impede the development of the malaria parasite (Eaton et al. 1976). Empirical tests of the traditional medicines, using an animal model of malaria, also demonstrate that three of these substances were highly effective cures.

These are five examples of disease-limiting cultural behaviors illustrating the general principles suggested by the current theory of biocultural evolution. However, this discussion should not imply that cultural behaviors always or regularly enhance health. There are many examples, from both the historical and ethnographic record, in which cultural behaviors function to increase the prevalence of diseases. Such cases represent a challenge to both theoretical and applied medical anthropology.

DISEASE AND HUMAN BEHAVIORAL PATTERNS

As the field of epidemiology has made clear since its inception in the late 1800s, diseases are not distributed randomly in human populations. Some individuals—and some groups of individuals—are at increased risk from various diseases, for reasons that are often unclear. Epidemiologists describe patterns of disease occurrence through space and through time and attempt to elucidate disease etiology through the search for risk factors that appear to be significantly associated with disease outcome.

Disease risk factors are of two major types. Endogenous risk factors are those that are biologically intrinsic to the human host. For example, genetic diseases,

such as sickle-cell anemia or hemophilia, have, by definition, an endogenous etiology. More commonly, however, genetic inheritance implies a predisposition to a disease that requires other variables or cofactors for expression to occur. Exogenous risk factors are those that are extrinsic to the body of the human host. Some of these may be biotic, such as microorganisms that cause infectious diseases, and others are nonbiotic substances present in the environment, such as toxic chemicals in the workplace. In most cases of disease, both endogenous and exogenous factors are involved—hence, the notion of multiple causation or multifactorial etiology (Dunn and Janes 1986).

Humans may unwittingly increase the likelihood of disease by exposing themselves or others to risk factors of both the exogenous and endogenous variety. In many cases, this enhanced exposure potential occurs through disruption of existing ecological relationships among the host, the agent(s) of disease, and the environment. In this way, human behavior itself may be said to be a risk factor for disease in that human activity may be a necessary component in the chain of events leading to a disease outcome.

Anthropologists, as professional observers and interpreters of human behavior, have an obvious and crucial role to play in the understanding of disease etiology: they can facilitate risk factor identification by describing distinctive patterns of human behavior related to the social distribution of disease. In this capacity, anthropologists may contribute directly to the generation of causal hypotheses, as they did in the case of kuru and cannibalism in New Guinea (Hunt 1978). In addition, anthropological descriptions of risk factor exposure based on long-term ethnographic observation may be more valid than those normally obtained through the standard epidemiological technique of questionnaire surveys.

Perhaps most important, anthropologists are especially equipped to understand disease-promoting human behaviors in sociocultural context. This includes the distribution of these behaviors through space and time, as well as the ideological and political-economic factors that serve to legitimate these behaviors. It is in this latter capacity—as interpreters of human behavior who elucidate how and why people act the way they do—that anthropologists may contribute directly to medical anthropological theory building and indirectly to disease prevention and control.

DISEASE ETIOLOGY: CATEGORIES AND CASE EXAMPLES

Human behavioral factors play a role in every major category of disease causation, although their role is sometimes subtle or indirect. Six major etiological categories are described in this section, and examples of these disease types are provided. Where applicable, examples of anthropological interest are presented to highlight the behavioral components in the etiological causal web and the ways in which anthropologists have contributed to their understanding.

Genetic

Genetic abnormalities that are heritable or occur as a result of mutation may be responsible for disease if they interfere with the normal functioning of the

affected individual. So-called genetic diseases must be distinguished from congenital diseases, which, although appearing at birth, may be due to factors in the intrauterine environment acting upon the fetus (Sheldon 1984).[1]

Among the most thoroughly understood of the genetic diseases is a group of conditions called hemoglobinopathies, including the sickle-cell trait (Hb^S), G6PD deficiency, thalassemia, and hemoglobins Hb^C and Hb^F (Livingstone 1985). These hemoglobin defects have received the most attention from anthropologists, who have been interested in their potentially protective effects against *P. falciparum* malaria.

In the 1950s, researchers began to suspect that various heritable human biochemical polymorphisms conferred protection to affected individuals against specific infectious diseases. Through descriptive epidemiology, A. C. Allison (1954) was the first to hypothesize that the heterozygous condition known as sickle-cell trait appeared with greater frequency in areas of Africa in which potentially lethal *P. falciparum* malaria was present. This association led Allison to hypothesize that hemoglobin S, when present in the heterozygous condition, conferred protection from death by malaria; this association has only recently been systematically confirmed (Durham 1983).

In a now-classic anthropological work that followed, F. B. Livingstone (1958) related the widespread distribution of the sickle-cell trait in West Africa to the history of human behavior, technological transfer, and ecological disruption in that region. He suggested that falciparum malaria did not spread widely in West Africa until the introduction of iron tools and, subsequently, swidden agriculture. The diffusion of the new technology, leading to changes in production capacity and the alteration of the forest habitat, effectively increased the available breeding grounds for *Anopheles gambiae*, the major mosquito vector of *P. falciparum*, as well as the density of sedentary human populations. This, in turn, allowed falciparum malaria to become established as an endemic disease among agricultural groups in West Africa and as a significant selective agent for the sickle-cell allele. In short, human behavior (swidden agriculture), through its effect on the environment (destruction of forest habitats and creation of *A. gambiae* breeding sites), affected the distribution and incidence of not only one but two endemic diseases in West Africa (falciparum malaria and sickle-cell anemia), as well as the structure of the gene pool in this region. (See Livingstone 1976 for a historical reconstruction.)

In a refinement of Livingstone's work, S. L. Wiesenfeld (1967) demonstrated that the particular type of agricultural system utilized significantly affected the rates of both sickle-cell trait and falciparum malaria. Specifically, societies heavily reliant on root and tree crops (the Malaysian agricultural complex) created a more malarious environment, leading to a selective advantage for individuals with the heterozygous condition in those societies.

Nutritional

Disease may result from malnutrition—either from dietary deficiency or excessive (or otherwise harmful) consumption patterns. The most common world-

wide cause of disease attributable to nutrition is malnutrition due to inadequate caloric intake (protein-energy malnutrition) (Sheldon 1984). However, protein-energy malnutrition must be understood not only as a biomedical "disease" but as a reflection of social inequality and consequent hunger (Cassidy 1982).

In addition, nutrition plays a major role in most of the diseases of civilization, including diabetes mellitus, coronary heart disease, hypertension, and even some forms of cancer. Yet because of the etiological complexity of these conditions, the magnitude of the contribution of nutritional risk factors has yet to be fully delineated. Furthermore, the nutritional component in, for example, coronary heart disease may vary from one population to the next and even between individuals.

Despite the current uncertainty surrounding nutritional factors in these First World diseases, it is clear that a number of specific vitamin- and mineral-deficiency diseases, largely eliminated in the industrialized world, continue to plague populations in poorer nations. These include the five major vitamin-deficiency diseases: beriberi (lack of thiamin); pellagra (lack of niacin); scurvy (lack of vitamin C); rickets (lack of vitamin D); and keratomalacia (lack of vitamin A). In addition, two of the mineral-deficiency diseases, anemia and goiter (from inadequate intake of iron and iodine, respectively), are found widely throughout the Third World.

In a study of nutritional deficiency and its effects on social organization in the Andean region of Ecuador, Greene (1973, 1977, 1980) has shown how the neurobiological consequences of nutritional-deficiency diseases are related to the development and continuation of a highly stratified social system. In this context, adequately nourished landowners exploit the malnourished rural populace (*indigenas* and *mestizos*) for cheap labor. Indigenous diets low in iodine and protein have led to high rates of goiter and protein-energy malnutrition, the latter exacerbated in this case by the early weaning of children to low-protein diets. The problem of endemic goiter is serious because of its association with cretinism and deaf-mutism. As Greene explains, the large number of mentally deficient individuals in this population has led to a redefinition of normalcy to markedly lower levels of cognitive functioning and an attempt by society to integrate behaviorally impaired individuals into the community (see also Buchbinder 1977).

In a somewhat different vein, anthropologists and clinicians have suggested that dietary habits in southern China may be responsible for the high rates of an otherwise rare cancer, nasopharyngeal carcinoma (NPC). H.C. Ho (1972) first proposed that consumption of salted marine fish may be responsible for the high rates of NPC in Guangdong province, where salted fish is commonly used to supplement rice among the lower social classes. Furthermore, in this region, infants are fed a mixture of salted fish with mushy rice during the weaning and postweaning period, which may account for the relatively young age at onset of this tumor (Anderson et al. 1978). According to D. P. Huang and colleagues (1978), the process of salting and drying fish leads to the production of volatile nitrosamines, which are known carcinogens in animals. In four major epidemiological studies of the association between Cantonese salted fish and NPC in humans, researchers have found increased risks of the disease ranging from 2.6- to 40-fold, depending on the timing and intensity of salted fish consumption (Henderson et al. 1976; Geser et al. 1978; Armstrong et al. 1983; Yu et al. 1986).

Environmental

Agents occurring naturally or as a result of human intervention in the external environment may cause disease. Physical agents, including unusual temperatures, electrical hazards, and irradiation, as well as trauma, may produce pathology (Sheldon 1984).

Of great interest to epidemiologists in the past 20 years has been the effect of exposure to various substances, especially toxic chemicals, in the workplace. For example, occupational epidemiologists have shown that exposure to the dust of asbestos, a substance once commonly used in construction, is a primary causal factor in the development of mesothelioma, an otherwise rare tumor (Selikoff et al. 1968). Moreover, exposure to asbestos appears to exacerbate the carcinogenic effects of cigarette smoking in the development of lung cancer (Hammond et al. 1979). In another major occupational study of Pennsylvania steelworkers, investigators have shown that men who work on the coke (liquified coal) ovens and are exposed to coke oven fumes over an extended period of time suffer significantly higher rates of mortality from respiratory cancers (Lloyd et al. 1970; Lloyd 1971). As with the previous example, coke oven workers who also smoke appear to be at increased risk.

Numerous other occupational groups have been shown to be at higher risk of various diseases because of workplace exposures, including miners, agricultural laborers exposed to various pesticides, and workers in cotton mills, dry cleaners, and the reinforced plastics industry, to name only a few. In addition, contaminants in the air and water, especially in urban, industrial areas, may place the general public at increased risk of disease, although the long-term health effects of environmental pollution remain speculative.

Psychogenic

It is now recognized that "psychogenic" factors may cause organic disease. "Psychosomatic illness" is the broad rubric under which somatic complaints of unknown etiology with a presumed psychological component are often placed. Unfortunately, etiological explanations for these conditions have tended to be reductionistic, involving either mental models or biological models but rarely synthetic models.

Anthropologists have perpetuated this dualism through an ongoing debate about the nature and etiology of voodoo death. Some anthropologists have argued that voodoo death occurs when the psychosocially traumatized victim gives up the will to live, thereby experiencing a form of "social" death (Thompson 1939; Warner 1958; Lewis 1977), while others have concluded that voodoo death occurs as the result of demonstrable biological mechanisms, such as dysfunction of the

automatic nervous system (Cannon 1942), surgical shock from terror (Yap 1974, 1977), difficulty in swallowing (Lex 1974), or dehydration (Eastwell 1982). Although the cause of voodoo death probably involves some combination of biological, psychological, and culturally determined behavioral factors, such a synthetic model has yet to be fully developed.

Iatrogenic

With the expansion of medicine, iatrogenic factors, or the deleterious effects of medical interventions, have been recognized as a growing cause of disease (Illich 1975). Perhaps the most common type of clinical iatrogenesis involves the negative effects of medications (such as stroke following the administration of oral contraceptives, congenital limb defects following the administration of the tranquilizer thalidomide to pregnant women, blindness following the administration of antiparasitic medications, involuntary facial and other body movements following the administration of antipsychotic drugs). However, nondrug therapies and even diagnostic procedures may be iatrogenic. For example, the common therapeutic practice during the first half of this century of irradiating the head and neck region for the treatment of, among other things, adolescent acne was later found to be a cause of thyroid cancer in individuals who had undergone this procedure 10 to 35 years earlier (Jackson 1984).

Criticism of the iatrogenic nature of medical practice has been directed most vociferously at Western biomedicine (Illich 1975). Yet evidence from the ethnographic and clinical literature suggests that iatrogenesis is not an exclusively Western phenomenon. For example, on the Guinea Coast of West Africa, where infection with the subcutaneous tissue-dwelling guinea worm (*Dracunculus medinensis*) is endemic, traditional healers' practices, which include piercing the guinea worm ulcer with a red-hot metal rod, are partly responsible for the high rates of secondary infection and considerable morbidity accompanying this helminthic parasitic disease (Edungbola and Watts 1985). Similarly, in Egypt, where the chlamydial eye disease trachoma is endemic and leads to visual impairment and blindness in rural populations, traditional healers' practices may lead to further ocular injury (Lane and Millar 1987; Millar and Lane 1988). These "ethno-ophthalmological" practices include, among other things, scraping the inner surface of the eyelid with an unsterilized shaving blade or slicing open an infant's eyes with the blood-drenched tip of a goose or pigeon feather in order to ensure that the child's eyes are "big and beautiful." Robert Trotter (1987) has shown that the Mexican-American remedies for the folk illness *empacho* (caused by food sticking to the stomach lining), *azaron* and *greta* (red lead oxite paint and lead protoxite, respectively) contain about 90 percent lead tetroxide and are a cause of lead poisoning. Anthropologists have recently been involved in alerting the local and medical communities to the dangers of these two folk remedies.

Infectious

Biologic agents, ranging in complexity from microscopic, obligate intracellular viruses to large and structurally complex helminthic parasites, are the cause of

Table 11.1
Infectious Diseases: Classification and Definitions

Class	Types	Examples of Diseases Caused
Viruses	RNA viruses	Poliomyelitis
	DNA viruses	Smallpox
	Unclassified viruses	Hepatitis B
	Slow viruses	Kuru
Bacteria	Gram-negative cocci	Gonorrhea
	Gram-positivecocci	Streptococcal, staphylococcal infections
	Gram-negative bacilli	Cholera
	Gram-positive bacilli	Diphtheria
	Spirochetes	Syphilis
	Anaerobic bacteria	Tetanus
	Mycobacteria	Leprosy
	Miscellaneous forms	Donovanosis
Agents intermediate between viruses and bacteria	Chlamydial agents	Trachoma
	Mycoplasmal agents	Mycoplasmal pneumonia
	Rickettsial agents	Typhus
Fungi	No major subclassification	Candidiasis
Parasites	Helminths	Schistosomiasis
	Protozoa	Malaria
	Ectoparasites	Lice

Adapted from Mandell et al. (1985)

infectious diseases in humans. Disease occurs when the interaction between the human host and the infectious agent, or the host-parasite relationship, is no longer symbiotic, shifting in favor of the agent. The most successful agents are not those that overcome and kill the host quickly, thus preventing their own reproduction. Rather, all the infectious agents, including viruses, bacteria, fungi, parasites, and several classes of intermediate forms, are more successful as either symbionts or commensals—that is, as agents infecting the human host without causing disease (Sheldon 1984).

The variety and complexity of the infectious agents, in terms of their biological characteristics, their reproductive strategies, and their modes of transmission, are impressive (Burnet and White 1978). Table 11.1 provides a basic taxonomy of the five major classes of infectious agents, with an example of each subtype. Table 11.2 summarizes the major routes of transmission of the infectious agents, providing examples of diseases spread through each pathway.

Whether infection with a specific microorganism results in disease depends upon a number of intervening variables, the most important of which are the pathogenicity of the agent (its inherent ability to cause disease), the route of

Table 11.2
Infectious Diseases: Routes of Transmission

Exogenous routes	Definition	Examples
Contact		
Direct (person-to-person)	Source of infection and host come in physical contact, allowing for direct transfer of infectious agents	Sexually transmitted diseases (e.g., gonorrhea, syphilis)
Indirect	Agents transmitted from source to host via passive transfer, usually on inanimate object	Diphtheria
Droplet	Relatively large microorganisms (> 5 um) spread over very short distances (< 1 meter) on droplets produced by talking, coughing, or sneezing	Measles
Common vehicle	Single inanimate vehicle, usually food or water, acts as medium of transfer of infectious agent to multiple hosts	
Active vehicle	Medium in which agent multiples	Clostridium Perfringen in contaiminated gravy
Passive vehicle	Medium is agent of transmission only	Hepatitis A
Airborne	Small (< 5 um) infectious agent is transmitted through air from disseminating source (human, animal, or inanimate object) to host over distances > 1 meter in dust or droplet	Tuberculosis
nuclei Vector-borne	Vector is intermediate host, usually an insect, which "carries" infectious agent from one host to another	
External	Infectious agent is carried on body of vector to host, without undergoing any physiological changes during transmission	Trachoma (fly carries Chlamydia trachomatis)
Internal	Infectious agent is carried within body of vector, where it is either harbored without undergoing physiological changes or where it undergoes biological trans mission phase (reproduction accompanied by physiological changes	Plague (Yersinia pestis within flea); Malaria Plasmodium spp. in female mosquito

Adapted from Brachman (1985b) and Evans (1982a)

Table 11.3
Patterns of Acquired Immunity to Infectious Agents

Type of Immunity	Definition	Examples
Natural immunity	Follows the natural occurrence of an infectious disease and involves one or both of host's two basicdefense mechanisms, antibody production (both humoral and local) and cell-mediated immunity	Rubella
Artificial immunity		
Active	Results from vaccination using killed vaccines, attenuated vaccines, Poliomyelitis toxoids, Tetanus	Typhoid fever
Passive	Acquired through artificial transfer of antibodies from one person (or animal) to another or by natural transfer of maternal antibodies to fetus via placenta	Hepatitis A (gammaglobulin)

Adapted from Evans (1982a) and Brachman (1985b)

transmission of the agent to the host, and the nature and strength of host defense mechanisms (Brachman 1985b). All of these factors are affected by the environment. Environmental factors, including such natural factors as temperature, moisture, altitude, and indigenous plants and animals, as well as such artificial factors as dams and irrigation schemes, human dwellings, and domesticated animals, may serve to promote the transmission of an infectious disease or, conversely, to limit or prevent its occurrence. Typically the infectious diseases are categorized into two major types, acute and chronic, according to the ways in which they affect susceptible populations through space and through time.

Acute infectious diseases, like measles and influenza, are generally characterized by sudden onset, marked symptomatology, and, most important, rapid resolution, either through death of affected individuals or the self-limiting nature of the illness. In many cases, natural immunity to subsequent infection is acquired following recovery (Table 11.3). When this occurs on a community-wide level, it is known as herd immunity.

In so-called virgin-soil populations (those without herd immunity), acute infectious diseases tend to occur in epidemics, which are said to exist when an unusual number of cases of the disease occurs in a given time period and geographic area as compared with the previous experience with the disease in the same area (Evans 1982a). The classic diagnostic features of an epidemic are listed in Table 11.4.

Table 11.4
Diagnostic Features of an Epidemic

Feature	Definition
1. Index Case	the primary case of an illness that may serve as a source of infection to others
2. Incubation Period	the definable interval between exposure and the appearance of the first detectable sign or symptom of the illness
3. Attack or Case Ratio	the incidence rate in the affected population during the outbreak
4. Epidemic Curve	the temporal pattern of the epidemic as illustrated by a histogram plotting number of cases against time interval

(Adapted from Evans 1986).

For diseases already present at some identifiable level in the community, it is necessary to know the total number of existing cases (prevalence), as well as the total number of new cases in the population still at risk (incidence), in order to determine whether an increase over normal levels of disease (an epidemic) has occurred. When such increases occur over a widespread area (such as a region, a continent, or globally), the term *pandemic* is used to designate the widespread geographic distribution of the epidemic.

Chronic infectious diseases, on the other hand, pose more difficult problems in definition because their course of occurrence and diffusion in susceptible populations must be viewed over years rather than days, weeks, or months (Evans 1982b). Chronic infectious diseases, such as schistosomiasis and tuberculosis, not only lack the short course of the acute infections, but they typically, although not invariably, lack the classic diagnostic features of an epidemic.[2] In general, chronic infectious diseases are endemic, a term denoting the constant or usual presence of an infection or a disease in a community (Evans 1982a).

From the standpoint of disease and human behavioral studies, the chronic infectious diseases are of greatest inherent interest. Although acute, epidemic infectious diseases are potentially devastating, they tend to burn themselves out quickly in populations, before behavioral and ideological responses on the part of the affected population are typically called into play. Chronic infectious diseases, on the other hand, are often associated with high morbidity, which may result in the incapacitation of members of affected populations. Because of their morbidity and their continual presence in the community, chronic infectious

diseases may trigger adaptive responses, including culturally conditioned behavioral changes that may reduce, intentionally or unintentionally, disease transmission.

Such behavioral change is most likely to occur when affected populations are aware of the nature of the infectious agent, its route of transmission, and human behavioral factors involved in this transmission cycle (Alland 1970). Information of this sort, usually the domain of Western biomedicine, is not regularly or effectively communicated to those most in need of understanding. Moreover, health education programs designed to prevent infectious diseases through behavioral change have had a limited impact because of a variety of complex problems, ranging from lack of voluntary community participation in prevention efforts (Philips 1955; Barnes and Jenkins 1972) to health educators' lack of understanding of local channels of communication and authority (Hanks and Hanks 1955).

The parasitic infection echinococcosis (hydatid disease) provides an instructive example of the complex behavioral dimensions of a chronic infectious disease. Echinococcosis is a zoonosis, an infectious disease transmitted from animals to humans. The disease occurs in areas of the world where humans, dogs, and domesticated livestock (primarily sheep) live in close association with one another. Areas of high prevalence include the Middle East, the Mediterranean region (especially Cyprus and Sardinia), East Africa, New Zealand, Argentina, and among scattered indigenous tribal and Basque populations living in the United States and Canada (Matossian et al. 1977; Shantz 1983).

Both humans and livestock contract the life-threatening larval parasitic infection through accidental ingestion of *Echinococcus granulosus* eggs. These eggs are excreted in dog feces and may be present on an infected dog's fur or in contaminated food and water resources (Katz et al. 1982). Dogs, the usual final host of the tapeworm parasite, contract the infection primarily through consumption of the offal of infected livestock. Although they harbor the adult tapeworms in their intestinal tracts, dogs do not normally suffer apparent disease. Humans and livestock, on the other hand, serve as the host of the immature larval forms of the parasite. When a human or a sheep accidentally ingests an *E. granulosus* egg, the larval parasite is liberated in the small intestine, where it penetrates the intestinal wall, enters the portal (and rarely the lymphatic) circulation, and then passes to the lungs or some other organ (including the brain). In the organs, echinococcal larvae grow into hydatid cysts, or large fluid-filled lesions, enclosed in a relatively delicate membrane and virtually brimming with daughter generations of infectious larvae (hydatid sand). If ruptured, these cysts release their metastatic contents, resulting in the "seeding" of new sites in the body. Patients often die when this occurs, usually as a result of shock.

Unwittingly, humans create the conditions by which they contract the infection. The crucial behavioral variables involved in transmission are as follows: (1) using dogs to tend livestock; (2) permitting these dogs to defecate in livestock grazing areas, thereby allowing livestock to become infected; (3) slaughtering infected livestock and feeding the viscera to working dogs, thereby

allowing dogs to become infected; and (4) treating infected working dogs as pets and companions, thereby promoting the risk of human infection through accidental ingestion of *E. granulosus* eggs present on dog fur.

This last point is crucial, for numerous epidemiological studies conducted in echinococcosis-endemic areas have shown that prevalence rates are highest among groups whose working dogs also function as pets. Nowhere is this more apparent than among the highly infected Turkana of Kenya and the neighboring tribes of southwestern Ethiopia. In richly detailed reports by medical geographers (French et al. 1982; French and Nelson 1982) and anthropologists (Fuller and Fuller 1981), cultural practices thought to promote echinococcosis in this region have been described. For example, dogs are the constant companions of men (who use them for sheepherding and protection), women (who use them as babysitters and nursemaids), and children (who use them as play companions). Of particular interest from the standpoint of echinococcosis transmission are the women's "nurse dogs," which are specially trained to care for children. These animals not only lick up the vomit and feces deposited by infants in the living quarters but also lick the face and anus of infants who soil themselves, thereby transmitting *E. granulosus* eggs from dog to human via the contaminated fur and muzzle. Moreover, these dogs sleep and sometimes defecate in the house, thereby disseminating infective material throughout the domestic environment.

Among the Turkana, potentially infective dog feces are highly valued as a traditional medicinal and cosmetic substance. Wounds are dressed with a baked mixture of dog feces and charcoal and a mixture of colored earth and dog feces, smeared over the face and body, is used to ward off evil spirits, as well as to protect women from the dermatologically damaging effects of their heavy layered necklaces, which are rarely removed (French, et al. 1982). Interestingly, the Turkana make no ethnomedical association between the symptoms of human echinococcosis, hydatid cysts in livestock, and dogs but rather believe the illness to be transmitted by supernatural curses resulting from social tensions (Fuller and Fuller 1981). This situation makes cultural adaptations to the disease less likely.

TWO PERSPECTIVES ON DISEASE AND HUMAN BEHAVIOR: THE SCHISTOSOMIASIS EXAMPLE

Human behavioral factors in disease causation have been viewed largely from a microsociological perspective. That is, the individual manifestations of culturally prescribed behavioral patterns are seen as risk factors for individual contraction of disease.

Certainly understanding human behavior is extremely important for a thorough description of disease etiology. However, the danger of viewing disease and human behavior on a microsociological level is that individuals may be incorrectly considered responsible, even culpable, for their own diseases. Even worse, entire societies may be blamed for maintaining unhealthful practices in their cultural repertoires.

To avoid such victim blaming and to understand disease causation, adoption of a macrosociological perspective is necessary. From this standpoint, disease is viewed on the level of the population, and disease rates are seen as the result of political and socioeconomic forces, operating through time and in some cases on a worldwide level. The macrosociological perspective emphasizes larger social forces and not the cumulative effects of individual behaviors as the ultimate causes of poor health.

Any medical anthropological study that hopes to shed light on the disease-behavior connection must ultimately adopt both perspectives. Unfortunately, the social scientific literature contains many examples of studies undertaken from one perspective or the other, but synthetic studies that attempt to evaluate behavioral patterns and to place these patterns in macrostructural context are rare. Furthermore, the current tendency within medical anthropology is to blame the overarching social-political-economic system for the health problems experienced at the local level without first describing in detail what those local health problems are and how behavioral risk factors may be involved.

The social scientific literature on the parasitic disease schistosomiasis is a particularly useful illustration of this problematic dualism. Furthermore, the rapid spread of schistosomiasis on the African continent today is largely due to the interaction of human behavioral and ecological factors, which must be viewed within a larger political-economic context.

Schistosomiasis (bilharzia) is a life-threatening blood fluke infection of humans. Like malaria and a number of other parasitic diseases, it is water based in that the three major species of schistosomes (*Schistosoma haematobium, S. mansoni*, and *S. japonicum*) share a developmental life cycle in which water plays a major role (Katz et al. 1982; Jordan 1985). Briefly, infected humans pass the eggs of the parasite, which are contained in their urine (*S. haematobium*) or feces (*S. mansoni* and *S. japonicum*), into the water, particularly in areas lacking modern sanitation. The eggs develop in the water, hatch, and release larval forms of the parasite. If the appropriate form of snail is present, these larvae penetrate the snail tissue, where they continue development. After several weeks, infective larvae (cercariae) are released from the snail into the water, where they live independently for up to 48 hours. These motile larvae seek out and penetrate human skin; once inside the human circulatory system, they mature into adult worms, mate, and pass to the veins of the bladder (*S. haematobium*) or mesenteric venules (*S. mansoni* and *S. japonicum*). Attached by their suckers to the walls of the veins, the adult worms, coupled for life, mate continuously during their five-to-ten-year life span and produce hundreds to thousands of eggs each day. These eggs cause morbidity in humans, adhering to the vessel walls and causing damage to the bladder or intestine. After they are eliminated in human waste, these eggs allow the parasitic life cycle to continue.

Because of the obvious human role in the perpetuation of the schistosomal life cycle, numerous studies of human water-contact behavior and schistosomiasis transmission have been undertaken within the past 30 years. These studies, advocated and supported by WHO (1979), can be characterized as microsocio-

logical in nature because of their primary focus on human behavioral factors in schistosomiasis transmission.

The first studies of this type were undertaken in the 1960s in locations ranging from Surinam (Van der Kuyp 1961) and Puerto Rico (Jobin and Ruiz-Tiben 1968) to Rhodesia (Husting 1970, 1983) and Egypt (Farooq 1966; Farooq et al. 1966; Farooq and Mallah 1966; Farooq and Samaan 1967). The most extensive investigations were carried out in Egypt, where M. Farooq and his colleagues performed elaborate observational studies of the daily social, occupational, and religious uses of water in a Nile Delta village. Their most striking finding was that Muslims had higher schistosomiasis prevalence rates than Christians due to the frequent practice of *wudu*, or ritual ablution before prayer, among the Muslims. Furthermore, the researchers concluded that swimming, a popular summertime activity for children, was responsible for the high rates of infection in the younger age groups.

Following a decade-long gap in research activity, a new generation of schistosomiasis investigators began to undertake water-contact studies in Africa (Kloos et al. 1977, 1980–1981, 1983; Dalton and Pole 1978; Polderman 1979; Edungbola 1980; Fenwick et al. 1982). As with the earlier studies, most of these more recent works examined the ways in which individuals became infected through water contact rather than the ways in which individuals infected water through urination and defecation in waterways. A notable exception was provided by an anthropologist, Ann Cheesmond, who with a colleague studied human excretory behavior in a schistosomiasis-endemic area of the Gezira, Sudan (Cheesmond and Fenwick 1981). From the standpoint of schistosomiasis transmission and control, Cheesmond's findings were heartening: 70 percent of the urination episodes and 93 percent of the defecation episodes observed occurred in sites far removed from any body of water, privacy being a more important consideration than proximity to water for the purposes of ablution. In fact, only 31 percent of those observed washed themselves after excretion, despite Islamic prescriptions to do so.

Despite the large number of water-contact studies undertaken and the recent major impetus for future water-contact studies from WHO, such studies are limited by their reliance on observation alone. As anthropologist Frederick Dunn aptly noted in an essay, "Behavioural Aspects of the Control of Parasitic Diseases":

> Let us consider human water contact, as one important element in the epidemiology of schistosomiasis. Any study of water contact must take into account at least the following: consumption of water (drinking, cooking, etc.); excretion and postexcretory ablutions in the water; bathing for hygienic reasons and laundering; swimming and other play in the water; ritual bathing; health education efforts to minimize water contact through changes in behaviour; technical efforts to minimize water contact by providing alternatives, e.g., bridges, safe laundry sites, and latrines; fishing; agricultural practices involving water use and contact; washing and watering of domestic animals; and travel practices, especially stream-crossing and boating, that require contact with water. . . . In so far as the programme may require change in human behaviour it will not suffice to have only this detailed description. A further series of studies, *essentially anthropological and psychological*, will be needed in each situation to specify why people behave as they do, where and when. . . . Any effort to change human behavior must rest on such studies. (1979:503, emphasis added)

Unfortunately, few of the behavioral studies surrounding schistosomiasis have assessed the underlying cultural logic of water-contact patterns or, for that matter, whether groups affected by schistosomiasis associate this condition with water and water-related activities. In three studies in which community members were questioned about their knowledge of schistosomiasis and its transmission, investigators found high levels of awareness of the disease and its symptoms but varying levels of knowledge about transmission or ways in which individuals could protect themselves from infection (Kloos et al. 1980–1981; Tiglao 1982; Zumstein 1983). Furthermore, as H. Kloos and colleagues noted, villagers in rural Ethiopia perceived schistosomiasis, with its vague symptoms, to be a relatively minor health problem, considering their struggle with more readily apparent helminthic infections, such as *Ascaris* (giant roundworm).

Most of these schistosomiasis studies have attempted to quantify behavior and correlate disease-promoting behavior and disease prevalence. However, few are truly anthropological because they fail to place the behavioral patterns observed in sociocultural context. Moreover, few of the studies successfully bridge the micro-to-macro gap by contextualizing water-contact patterns in terms of political, economic, or ecological origins of unsafe water itself.

This last issue can be raised in terms of water-resource development projects and their effect on the spread of schistosomiasis. Research on this issue can be characterized as macrosociological because it focuses on the ecological disruption and health hazards engendered by politically and economically motivated development schemes. As Charles Hughes and John Hunter (1970) note in their review of disease and development in Africa, few of the economic development projects initiated on that continent over the past two centuries have been undertaken within a preconceived, ecological framework. This lack of ecological foresight has resulted in the escalation of "developo-genic" diseases, including schistosomiasis, onchocerciasis, trypanosomiasis, and malaria.

Of these diseases, schistosomiasis is the most rapidly spreading (Heyneman 1983a), a spread attributable almost entirely to the construction of high dams for hydroelectric power, artificial lakes for fish breeding, reservoirs for water storage, and irrigation systems for agriculture (Heyneman 1971, 1979, 1983a; Scudder 1973; Kloos and Thompson 1979). The expansion of old waterways and the creation of new ones has provided an ecological free zone for snails, the intermediate hosts. As the snail population has spread into new aquatic environments, so have schistosomal parasites and human infections.

The spread of schistosomiasis has been the most severe in Africa, and particularly in Egypt. This is largely due to the construction over the past century of the Aswan Dam–Lake Nasser complex (designed to provide hydroelectric power and perennial irrigation to the country). In a cross-sectional survey carried out in the 1950s in four selected sites in Egypt, schistosomiasis prevalence rates

increased an average of 51 percent in three years (Lanoix 1958). Although the Egyptian government has made efforts over the past two decades to control the schistosomiasis problem among the rural population through mass treatment campaigns and molluscicide (chemical extermination of the snail population), a recent report by Egyptian scholars has suggested that few, if any, real gains in schistosomiasis control have been made (Abdel-Salam et al. 1986).

Egypt is not alone in its predicament. The schistosomal upsurge witnessed there has been repeated over and over again in Africa following the construction of virtually every major dam and reservoir complex, irrigation system, and artificial lake (Desowitz 1981). In studies undertaken in the Awash Valley of Ethiopia, H. Kloos and his colleagues have described the expanding distribution of schistosome-transmitting snail populations and escalating rates of human infection following government-sponsored creation of large, irrigated farming estates (Kloos 1977, 1985; Kloos and Lemma 1977; Kloos et al. 1978; Kloos and Thompson 1979). In Sudan, the disease cycle was established within a few years of the start of the Gezira scheme, a large-scale, irrigated cotton project south of Khartoum (Kloos and Thompson 1979; Fenwick et al. 1981; Gruenbaum 1983). In this case, the change in irrigation methods from seasonal flooding to the use of pump irrigation created more extensive and stable snail habitats and intensified human water contact during periods of crop irrigation. In Nigeria, *S. haematobium* prevalence rates soared following construction of a low earth dam and perennial access to a large body of infective water (Pugh and Gilles 1978). This increase was likely to continue, researchers predicted, given government plans to build more dams in the area.

CONCLUSION: DIRECTIONS FOR FUTURE RESEARCH

The study of disease and human behavior from an ecological perspective has contributed, and should continue to contribute, to the solution of theoretical questions in general and medical anthropology and practical problems in public health. The research strategy described in this chapter has two complementary dimensions: (1) analysis of the social and ecological distribution of disease as it affects human culture and biology and (2) analysis of human behavior, and its sociocultural determinants, as it affects the changing distribution of disease. Both approaches require the crossing of the subdisciplinary boundaries that currently divide anthropology, for they examine the interaction of cultural and biological phenomena from both a diachronic and synchronic perspective. We believe that this "biocultural" orientation, stemming from an earlier, holistic tradition in anthropology, continues to be theoretically attractive and is directly applicable to the improvement of health, particularly in less-"developed" countries. Furthermore, because this biocultural approach to problems of disease and human behavior concerns all of the subdisciplines of anthropology (as well as epidemiology and medical geography), it has the potential to provide a synthesizing theoretical framework and, in so doing, to unify the now fragmented discipline itself.

This chapter on the study of disease and human behavior from an ecological perspective has emphasized five major themes:

1. Diseases occur within ecological settings and thus are context dependent.
2. Cultural practices can directly alter ecological relationships between hosts and agents of disease and can thereby influence, either positively or negatively, human health.
3. Biological and cultural traits with adaptive value against disease will generally be selected for and be maintained in a population, according to evolutionary theory, because they enhance reproductive fitness.
4. Human behavior plays a significant role in the etiology of every major category of disease and particularly the infectious diseases.
5. The understanding of the influence of human behavior on disease rates requires a synthesis of micro- and macrosociological perspectives.

Although a significant amount of exemplary research on the interaction of disease and human behavior in ecological context has already been conducted, the opportunities for future medical anthropological research in this field are great. Of the numerous diseases that now plague populations and individuals around the world, an inordinately small number of them have been studied by anthropologists, despite the fact that many of these diseases are significant causes of morbidity and mortality and are recognized as such by those afflicted, who may view the disease with great alarm. Moreover, many of the anthropological studies of disease that have been conducted to date have not been undertaken for their own sake; rather, they have been part and parcel of biomedical initiatives to elucidate the causes of diseases or to eliminate them in a "culturally appropriate" manner. Finally, both diseases themselves and the culturally determined behaviors influencing them are constantly changing; new diseases, such as acquired immune deficiency syndrome (AIDS), continue to appear. The introduction of new diseases and the appearance of new twists on old ones present major challenges to anthropologists interested in biological and cultural adaptations to disease threats. Nowhere is this more apparent today than with AIDS, which provides a graphic and tragic example of the complex interactions between a disease agent and human behavior within varying ecological contexts.

Given this scenario, three research priorities stand out as particularly important. First, bioculturally oriented medical anthropologists must attempt to refine the definition of disease so that it is no longer defined by what it is not (the absence of "health") or by juxtaposing it as the objective counterpart to the more subjective concept of "illness." If bioculturally oriented medical anthropologists are to study diseases without being labeled handmaidens of the biomedical establishment by so-called critical medical anthropologists, then disease models that acknowledge and incorporate the fundamental differences in culturally constructed notions of disease (including, necessarily, the Western biomedical construction) must be formulated.

Second, studies of cultural adaptations to disease threats must progress beyond the level of description to quantitatively rigorous analyses of the effects of

particular behaviors on disease morbidity and mortality. Such work has recently been forthcoming in the study of AIDS with respect to the various practices involved in the spread of the human immunodeficiency virus (HIV) (Gorman 1986; Feldman and Johnson 1986).

Finally, future studies of the disease-culture interaction must begin to combine the micro-and macrosociological levels of analysis. There is a paucity of, and hence a pressing need for, synthetic models to describe disease problems. Most research today tends to focus either on individual behavioral risk factors for various diseases (often based on observational or questionnaire survey data alone) or on the overarching political-economic system that allows such diseases to be maintained (even in the absence of supportive ethnographic data). Unfortunately, such either-or research may lead to victim blaming—if not by the researchers themselves (who may never have intended to assign culpability for the disease problems under study), then by those who use the research to justify their own political and economic biases and objectives. Synthetic models, which examine in detail the interaction of disease and culture on a local level and frame this interaction in terms of the regional, national, and global forces impinging upon it, are needed if disease problems are to be fully understood.

NOTES

1. Research on genetic diseases perhaps overemphasizes endogenous risk factors, and differences in disease prevalence between racial groups are often misinterpreted as being purely genetic rather than reflecting socially generated behavioral differences (Krieger and Basset 1986). For example, sickle-cell anemia accounts for a very small fraction of mortality among American blacks and is a comparatively minor public health problem, whereas hypertension and stroke, both major causes of morbidity and mortality among blacks, are largely related to such exogenous factors as diet, smoking, exercise, and stress.

Furtheremore, genetic diseases are based, in part, on cultural assumptions of normality and a failure of the biomedical community to recognize the range of physiological variation among humans. For example, the finding of S. Garn et al. (1975) that normal levels of iron in circulating blood are lower among American blacks necessitated a change in clinical definitions of anemia. This definitional broadening made an "epidemic" of anemia among American black women disappear.

2. Epidemic outbreaks of chronic infectious diseases may also occur following population movements, changes in vector or animal reservoir populations, environmental disruption, or any event that increases exposure of human populations to a given infectious agent.