Part I

Historical aspects
1 Emerging disease in the third epidemiological transition

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In 1969, William T. Stewart, the Surgeon General of the United States, testifying before Congress proposed that it was now ‘time to close the book on infectious disease as a major health threat.’ Stewart and others believed that with the development of antibiotics, vaccines and pesticides, we were on the verge of eradicating infectious disease. Buoyed by this success, Stewart’s testimony before Congress was designed to position the United States public health system to meet its next health challenge: controlling chronic and degenerative diseases.

Stewart’s assessment on the decline of infectious disease and the rise of chronic disease was the fulfillment of an epidemiological theory that was first proposed by Abdul Omran (1971), who argued that human populations were experiencing a shift in health and disease patterns. Omran contended that human disease history could be described as moving through a number of disease stages. Initially, humans passed through ‘the age of pestilence and famine’ to an ‘age of receding pandemics’ and finally into ‘the age of degenerative and man-made diseases’. The basic feature of Omran’s model (1971, 1977, 1983) was the idea that as infectious diseases were eliminated, chronic diseases would increase as the population aged. Finally, epidemiological transition theory had implications for demographic transition theory, which suggested that after the decline in mortality there would be an eventual decline in fertility.

This chapter has three objectives; the first is to interpret and broaden the concept of epidemiological transition into a model that defines a number of dramatic shifts in disease patterns (Armelagos et al. 1996; Armelagos 1998; Armelagos and Barnes 1999; Barnes et al. 1999). Secondly, the evolution of emerging diseases will be discussed from the perspective of three epidemiological transitions (Barrett et al. 1998). While a distinct pattern of disease emerged as our Paleolithic ancestors moved into new ecological niches (Desowitz 1980), their mobility, small population size and low density precluded infectious disease from being a factor in the evolution of these populations. Finally, it will be shown that while emerging diseases have been a characteristic of human adaptation, following the shift to primary food production, there was an acceleration of the trend.

The traditional Hobbesian view of the 4,000,000 years of the Paleolithic was of the gatherer-hunters who foraged for their livelihood. Hobbes describes our ancestors living in ‘continual fear’ with ‘a danger of violent death’ and a life that
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was 'solitary, poor, nasty, brutish, and short' (*Leviathan*, i. xiii. 9). In actuality, Paleolithic populations appear to have been relatively healthy and well nourished. During the Neolithic, the shift to primary food production (agriculture) created the first epidemiological transition associated with the acceleration of emerging diseases. The second epidemiological transition (Omran’s original epidemiological transition) began early in the 20th century with the decline in infectious disease and the rise of chronic diseases. We are entering the third epidemiological transition with the re-emergence of infectious diseases that were thought to be under control (many that are antibiotic resistant) and the rapid emergence of a number of ‘new’ diseases. The existence of antibiotic resistant pathogens (some that are resistant to multiple antibiotics) foretells a possibility that we are living in the eve of the antibiotic era. Finally, the third epidemiological transition is characterized by a transportation system, so vast, so rapid, that the globalization of the disease process (Waters 2001) has produced what has been called the ‘viral superhighway’.

The concept of emerging disease needs to be considered more fully. Emerging infectious diseases are defined by the Institute of Medicine (IOM) as ‘new, re-emerging, or drug-resistant infections whose incidence in humans has increased within the past decades or whose incidence threatens to increase in the near future’ (Hughes 2001). For the public, emerging disease seems to have replaced the anxiety that was produced by the rampant fear of nuclear war following the Cold War of the 1950s. During that era, Hollywood produced a series of popular films such as *The Day the World Ended* (1956) in which radiation following a nuclear holocaust turns survivors into horrible mutants. Now, popular books such as *The Hot Zone* (Preston 1994) and movies such as *Outbreak* have captured the public’s fascination with emerging diseases as threats to human survival. A monkey carrying a deadly new virus from central Africa infects and liquefies the organs of unwitting Californians, creating an epidemic that even the vast biomedical community is incapable of stopping, eventually threatening the annihilation of the human race. Just as we have begun to allay some of the fears of mutant pathogens running amok, there are serious concerns about the reality of bio-terrorism (McDade and Franz 1998; Henderson 1999; Arnon et al. 2001; Dennis 2001). The recent episode of anthrax in the United States attests to the impact that bio-terrorism has had on the western psyche.

Even the biomedical community’s view on ‘emerging’ disease has been questioned. Paul Farmer (1996) suggests that emerging diseases are only ‘discovered’ when they have an impact on our daily existence. For example, Lyme disease (*Borrelia burgdorferi*) was studied long ‘before suburban reforestation and golf courses complicated the equation by creating an environment agreeable to both ticks and affluent humans’ (Farmer 1996). Even when a more holistic ecological perspective is taken, it is often limited to a position that considers ‘emerging’ disease as the result of human behavior or microbial changes that fails to place them in a broader political–economic context. There is a failure to ‘ask how large-scale social forces influence unequally positioned individuals in increasingly interconnected populations’ (Farmer 1996) and how inequality affects the disease process. Social inequalities are an essential element in understanding emerging disease patterns.
The world’s biggest killer and greatest cause of ill health and suffering across the globe is listed almost at the end of the International Classification of Disease. It is given in code Z59.5 – extreme poverty (WHO 1995). The WHO ICD classification does not define extreme poverty. However, the need for basic human resources such as food, water, shelter, access to health care, and adequate social support, might allow us to make a minimal definition (Armelagos and Brown 2002). The World Bank estimates that three billion people in the world live on less than two dollars a day. Each year, in the developing world, 12.2 million children under five die. These deaths could be prevented for just a few cents per child. The World Bank’s Global Burden of Disease Study pioneered the use of Disability Adjusted Life Years (DALYs) in assessing the impact of preventable disease (Hollinghurst et al. 2000) in the world today.

A perspective on the evolution of social inequality

The evolution of social inequality has not been studied extensively. There are only a few studies that analyze the gap that exists between individuals within a society and the gaps between societies. The relationship between inequality and health can be studied from an evolutionary perspective (Goodman et al. 1995). The analysis of changing patterns of health/disease and social organization in the prehistoric past allows us to better understand health inequalities in the contemporary world (Fayynter 1989; McGuire and Fayynter 1991).

Beginning in the Neolithic, inequalities within and between societies have accelerated with advances in technology (Figure 1.1). The gap between classes within society and differences in wealth among societies continues to widen. The disparity within and between nations in the present world order continues and the prospect that the gap will narrow is unlikely.

An evolutionary perspective allows us to examine the relationship between health and wealth, disease and poverty as part of continuing historical processes that

![Figure 1.1 Cultural evolution, health and inequality.](image-url)
have affected thousands of human generations. While stratification may occur without inequality, these quickly become inextricably linked. Social stratification originally evolved because it brought benefits to emerging elites which effected their well being. In most instances, the benefits accrued to the wealthy came at the expense of the poor (Armelagos and Brown 2002).

When evaluating the disease process, we tend to focus on pathogens (the microparasites) that use the host as a source of food and energy. However, just as microparasites are the source of disease, there are factors within societies that exacerbate survival to such an extent that they are as effective as the parasites themselves. For example, social stratification is an evolutionary strategy in which one segment exploits other segments of the social system to such a degree that their resources are limited and their health is at risk. The pattern and impact of exploitation is as parasitic as a pathogen. McNeil (1976) and Brown (1987) describe the process of exploitation as ‘macroparasitism’ and see it as a force in understanding the evolution of disease.

The evolution of technological change since the Neolithic has widened the gap between the rich and the poor, the healthy and the sick within and between societies. The gap between those at the top and the bottom of the social hierarchy in the 21st century is greater than ever before in human history (Armelagos and Brown 2002).

**Paleolithic baseline**

The reconstruction of Paleolithic disease ecology requires the triangulation of methods and data from a number of sources. Archeological analysis of foraging populations provides direct evidence of their patterns of mortality and morbidity. The genomic diversity of pathogens and parasites provides clues to the phylogenetic relationships and patterns of adaptation to their hosts. Applying a molecular clock allows scientists to determine when the pathogen began to parasitize the host. Molecular analysis of the three modern taenid tapeworms that parasitize humans and were assumed to have become a problem during the Neolithic are now thought to have originated as human parasites in the Paleolithic (Hoberg et al. 2000, 2001).

The consideration of the disease ecology of contemporary gatherer-hunters provides a model for the types of disease that would have affected Paleolithic foragers. Sprent (1969a, 1969b) distinguishes two classes of parasites that would have afflicted gatherer-hunters. ‘Heirloom species’ are a class of parasites that have had a long-standing relationship with our anthropoid ancestors and that continued to infect them as they evolved to hominids. Head and body lice (*Pediculus humanus*), pinworms, and possibly yaws, malaria are heirloom species. Certain lice have been ectoparasites since the Oligocene (Laird 1989). Most of the internal protozoa found in modern humans and such bacteria as salmonella, typhli, and staphylococci (Cockburn 1967a, 1967b) are also heirloom species. In contrast to heirloom parasites that had longstanding relationships with anthropoids and hominids are ‘souvenir’ species that are ‘picked up’ during daily activity. Souvenir species are zoonoses whose primary hosts are non-human animals and they only incidentally infect humans.
Zoonoses are passed on to humans through insect bites, preparing and consuming contaminated flesh, and from animal bites. Sleeping sickness, tetanus, scrub typhus, relapsing fever, rickettsiosis, tularemia, avian or ichthyic tuberculosis, leptospirosis, and schistosomiasis are among the zoonotic diseases that likely afflicted earlier gatherer-hunters (Cockburn 1971). Small population size would have precluded infections of many bacteria and viruses. However, synanthropic relationships with the vectors served to maintain such human host-specific diseases as yellow fever and louse-borne relapsing fever (Laird 1989) in earlier foragers. Anopheles, the vector, necessary for transmission of malaria had evolved by the Miocene era by adapting to the canopy environment, suggesting that it would be present in the Paleolithic. Livingstone (1958) dismisses the threat of malaria in early hominids because of their small population size and an adaptation to the savanna, an environment that would not have included mosquitoes that carry the malaria plasmodium. If malaria was contracted, it would have been an isolated incidence. Recent analysis of the genetic structure of variants of glucose-6-phosphate dehydrogenase confirms that malaria has only recently had a major impact on human populations (Tishkoff et al. 2001). The independent 'A' and 'Med' mutations in glucose-6-phosphate dehydrogenase suggest that this polymorphism originated at least 10,000 years ago.

The range of the earliest hominids was probably restricted to the tropical grassy woodland savannah, limiting the variety of pathogens that could be potential disease agents. As Dicke (1932) and Lambrecht (1964, 1967, 1980, 1985) note, hominids would have found extensive areas of Africa uninhabitable because of tsetse flies and the trypanosomes. Lambrecht also argues that as human species moved into new ecological niches the pattern of trypanosome infection would have changed. Beginning 200,000 years ago, as populations moved out Africa, there was expansion into temperate and tundra habitats that would have changed the disease ecology for the trypanosomes.

The diseases that are missing from the pantheon of Paleolithic pathogens are very informative. Contagious diseases such as influenza, measles, mumps, and smallpox would not have been present. There would have been few viruses infecting early hominids (Burnet 1962). Countering this claim, Cockburn (1967b) suggests that the viral diseases found in non-human primates would have been easily transmitted to early hominids.

Hominid populations remained stable throughout the Paleolithic. Fertility and mortality rates in populations would have to have been balanced for population size to remain low. Conventional wisdom has argued that Paleolithic populations experienced maximum fertility and high mortality. Armelagos et al. (1975) have offered an alternative scenario in the shift from gathering and hunting to agriculture. The picture that has emerged suggests a much bleaker picture of health. Instead of experiencing improved health, there is evidence of a substantial increase in infectious and nutritional disease following the shift to agriculture (Cohen and Armelagos 1984). The implication is that a population experiencing maximum fertility during the Paleolithic could not have increased fertility that would have led to population growth as their nutrition and health deteriorated. The demographic changes following the Neolithic suggest that during the
Paleolithic a stable population controlled by moderate fertility existed. Following the Neolithic revolution, there was a dramatic growth in population size and an increase in density even with a decline in nutrition and health because of increases in fertility.

The first epidemiological transition: disease in agricultural populations

The earliest evidence of primary food production in the Old World is from about 10,000 years ago. There are independent areas of cultivation in Mesopotamia (based on barley and wheat), sub-Saharan Africa (based on millets and plantains), Southeast Asia (based on rice), northern China (based on millet) and southern China (based on rice). Centres in the New World originate later based on the domestication of maize (Mesoamerica) and potatoes (South America). Significant settlements are evident in the Tigris–Euphrates area by 7000 years ago, and a thousand years later there are centralized governments controlling vast irrigation systems. This development created social classes with differential access to resources. In the Valley of Mexico, there were well-established settlements by 3500 BP and by 1 CE these settlements show extensive hierarchies.

Ecological changes increase the potential for disease load following the shift to primary food production. Sedentary villages increase parasitic disease infection by increasing contact with human waste. While sedentarism could and did occur prior to the Neolithic period in those areas with abundant resources, the shift to agriculture necessitated sedentary living. In sedentary populations the proximity of habitation areas and their waste deposit sites to the water supply is a source of contamination. In gathering-hunting groups, the frequent movement of the base camp and frequent forays away from the base camp by men and women, would decrease their contact with human wastes.

Animal husbandry also increased the frequency of contact with a steady supply of disease vectors. Zoonotic infections would have been contracted from domesticated animals, such as goats, sheep, cattle, pigs, and fowl, as well as the unwanted domestic animals such as rodents and sparrows, which developed permanent habitats in and around human dwellings. Products of domesticated animals such as milk, hair, and skin, as well as the dust raised by the animals, could transmit anthrax, Q fever, brucellosis, and tuberculosis. Breaking the sod during cultivation exposes workers to insect bites and diseases such as scrub typhus (Audy 1961). Livingstone (1958) showed that slash-and-burn agriculture in West Africa exposed populations to *Anopheles gambiae*, a mosquito that is the vector for *Plasmodium falciparum*, which causes malaria. The combination of disruptive environmental farming practices and the presence of domestic animals also increases human contact with arthropod vectors carrying yellow fever, trypanosomiasis, and filariasis, which now developed a preference for human blood. Some vectors developed dependent relationships with human habitats, the best example of which is *Aedes aegypti* (the vector for yellow fever and dengue), an artificial container breeder. Various agricultural practices increased contact with non-vector parasites, such as...
irrigation (contact with schistosomal cercariae) and the use of faeces as fertilizer (infection with intestinal flukes) (Cockburn 1971).

The shift to agriculture heralded a change in ecology that resulted in diseases not frequently encountered by earlier foraging populations. The shift from a varied, well-balanced diet to one that contained fewer types of food sometimes resulted in dietary deficiencies. Food was stored in large quantities and widely distributed, probably resulting in outbreaks of food poisoning (Audy 1961). In Cohen and Armelagos (1984) there are a number of studies that show a decline in health following the Neolithic. The combination of a complex society, increasing divisions of class, epidemic disease, and dietary insufficiencies, no doubt added mental stress to the list of illnesses.

**Urban development and disease**

The growth of urban centres is a recent development in human history. In the Near East, large cities were established 6700 years ago. In the New World, large urban settlements were in existence by 1400 years ago. Urban centres at Memphis (Egypt) reached 30,000 souls by 3100 BCE, Ur (Babylonia) reached 65,000 inhabitants by 2030 BCE and Babylon had a population of 200,000 by 612 BCE (Chandler 1987). Settlements of this size increased the already difficult problem of removing human wastes and delivering uncontaminated water. Cholera, which is transmitted by contaminated water, was a potential problem. Diseases such as typhus (carried by lice) and the plague bacillus (transmitted by fleas or by the respiratory route), could be spread from person to person. Viral diseases such as measles, mumps, chicken-pox, and smallpox could be spread in a similar fashion. There were for the first time, during the period of urbanization, populations large enough to maintain disease in an endemic form. Cockburn (1967a) estimates that populations of one million would be necessary to maintain measles as an endemic disease. Others (Black et al. 1974) suggest that a population of only 200,000 would be required to maintain measles. Black and colleagues (1974) argue that a population of only a 1000 people is needed to sustain chicken-pox as an endemic disease. What was an endemic disease in one population could be the source of a serious epidemic disease in another group. Cross-continental trade and travel resulted in intense epidemics (McNeill 1976; Zinsser 1935). The Black Death took its toll in Europe in the 1300s. The epidemic eliminated at least a quarter of the European population (approximately 25 million people) (Laird 1989).

The period of urban development can also be characterized by the exploration and expansion of populations into new areas, which resulted in the introduction of novel diseases to groups that had little resistance to them (McNeill 1976). McNeill (1978) describes the process in which civilizations ‘digest’ the encountered populations as their disease vectors clear their path allowing easy access to expansion. For example, European-carried smallpox and measles destroyed millions of Native Americans following contact (Dobyns 1983; Ramenofsky 1987, 1993).

The exchange of disease can be a two-way street. For example, the exploration of the New World may have been the source of the treponemal infection that was
transmitted to the Old World (Baker and Armelagos 1988). The treponemal infection in the New World was endemic and not sexually transmitted (Rothschild et al. 2000). When introduced into the Old World there was a different mode of disease transmission. The sexual transmission of the treponeme created a different environment for the pathogen, and it resulted in a more severe and acute infection. Furthermore, crowding in the urban centres created changes in sexual practices, such as prostitution, and an increase in sexual promiscuity may have been a factor in the new venereal transmission of the pathogen (Hudson 1965). Claims that pre-Columbian syphilis existed in Europe have been made in response to the claims of New World origin of the disease (Pálfy et al. 1992; Dutour et al. 1994). The resolution of this debate may await the recovery of material that can be identified as treponemal pathogen from Old World, pre-Columbian archaeological bone.

The process of industrialization, which began a little over 200 years ago, led to an even greater environmental and social transformation. London in 1800 was the only city in the world with a million inhabitants. City dwellers would be forced to contend with industrial wastes and polluted water and air. Slums that rose in industrial cities would become the focal point for poverty and the spread of disease. Epidemics of smallpox, typhus, typhoid, diphtheria, measles, and yellow fever in urban settings are well documented (Polgar 1964). Tuberculosis and respiratory diseases such as pneumonia and bronchitis are associated with harsh working situations and crowded living conditions. Urban population centres, with their extremely high mortality, were not able to maintain their population base through the reproductive capacity of those living in the city. Mortality outstripped fertility, requiring in-migration of rural populations to the city in order to maintain its numbers.

Recently much attention has been focused on the detrimental effects of industrialization on the international environment, including water, land, and atmosphere. Massive industrial production of commodities has caused pollution. Increasingly there is concern over the health implications of contaminated water supplies, over-use of pesticides in commercialized agriculture, atmospheric chemicals, and the future effects of depleted ozone on human health and food production. At no other time in human history have the changes in the environment been more rapid and so extreme. Increasing incidence of cancer among young people and the increase in respiratory disease has been implicated in these environmental changes.

The United Nations Population Fund (2001) reports that in 2000, 47 per cent (2.9 billion people) of the world population are living in an urban setting. In 30 years, that number will increase to 60 per cent. WHO has addressed the issue of health in urban settings with their healthy cities initiative (Goldstein 2000; Kenzer 2000; Tsouros 2000). The WHO programme attempts to systematically address poverty, the vulnerability of segments of the populations, and the lack of access that these populations have to health care.
The second epidemiological transition: the rise of chronic and degenerative disease

Traditionally, the term ‘epidemiological transition’ refers to the shift from acute infectious diseases to chronic non-infectious, degenerative diseases. The increasing prevalence of these chronic diseases is related to an increase in longevity. Cultural advances result in a larger percentage of individuals reaching the oldest age segment of the population. In addition, the technological advances that characterize the second epidemiological transition result in an increase in environmental degradation. An interesting characteristic of many of the chronic diseases is that they are particularly prevalent and ‘epidemic-like’ in transitional societies, or in those populations undergoing the shift from developing to developed modes of production. In developing countries, many of the chronic diseases associated with the epidemiological transition appear first in members of the upper socioeconomic strata (Burkitt 1973), because of their access to Western products and practices.

With increasing developments in technology, medicine, and science, the germ theory of disease causation developed. While there is some controversy as to the role that medicine played in the decline of some of the infectious diseases (McKeown 1979), there was a better understanding of the source of infectious disease and this admittedly resulted in increasing control over many infectious diseases. The development of immunization resulted in the control of many infections and recently was the primary factor in the eradication of smallpox. In the developed nations, a number of other communicable diseases have diminished in importance. The decrease in infectious diseases and the subsequent reduction in infant mortality have resulted in greater life expectancy at birth. The increase in longevity for adults has resulted in an increase in chronic and degenerative diseases.

Many diseases of the second epidemiological transition share common, etiological factors related to human adaptation, including diet, activity level, mental stress, behavioural practices, and environmental pollution. For example, the industrialization and commercialization of food often results in malnutrition, especially for those societies in ‘transition’ from subsistence forms of food provision to agri-business. Many do not have the economic capacity to purchase food that meets their nutritional requirements (Fleuret and Fleuret 1980). Obesity and high intakes of refined carbohydrates are related to the increasing incidence of heart disease and diabetes. Obesity is considered to be a common form of malnutrition in developed countries and is a direct result of an increasingly sedentary life-style in conjunction with steady or increasing caloric intakes. A unique characteristic of the chronic diseases is their relatively recent appearance in human history as a major cause of morbidity. According to Corrucini and Samvit (1983), this is indicative of a strong environmental factor in disease etiology. While biological factors such as genetics are no doubt important in determining who is most likely to succumb to which disease, genetics alone cannot explain the rapid increase in chronic disease. Critics of McKeown have focused on his use of evidence for improved nutrition (Johansson 1991, 1992; Schofield and Reher 1991) and failure

The third epidemiological transition

Human populations are in the midst of the third epidemiological transition. There is a re-emergence of infectious diseases that have multiple antibiotic resistance. Furthermore, the emergence of diseases has a potential for having a global impact. In a sense, the contemporary transition does not eliminate the possible co-existence of infectious diseases typical of the first epidemiological transition (some 10,000 years ago) in our own time; the World Health Organization reports that of the 50,000,000 deaths each year, 17,500,000 are the result of infectious and parasitic disease. WHO states that two billion people in the world are infected with hepatitis B virus (WHO 1995). Two billion of the world’s population have tuberculosis (8 million cases contracted every year and 3 million die in that period). In the last thirty years, 40 million people have become infected with HIV and 3 million people have died during that period.

Humans have lived in urban centres for only 0.125 per cent of our history. This may explain the paucity of evidence for the genetic response to specific disease. Svanborg-Eden and Levin (1990) challenge the proposition that infectious disease is a major force in the selection and evolution of genetic variability in human populations. They argue that there are three constraints for infectious disease to act as an effective agent of natural selection. First, most variation in the frequency of infectious disease is the result of environmental factors. Second, the array of host defenses is general in their actions and overlapping in their functions. Third, the specific immune defenses are adaptive at the somatic level and therefore there is less of a need for selection leading to germ-line evolution. Four decades ago, Lederberg (1963) suggested that diseases that had animal reservoirs could lead to the development of disease resistance in human populations. He argued that the persistence of ‘small differentials’ could lead to genetic immunity.


The Institute of Medicine (IOM) (Lederberg et al. 1992) reports that the emergence of disease is the result of an interaction of social, demographic, and
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environmental changes in a global ecology and in the adaptation and genetics of
the microbe. Similarly, Morse (1995) sees emerging disease as a result of
demographic changes, international commerce and travel, technological change,
breakdown of public health measures, and microbial adaptation.

Among the ecological changes Morse describes are agricultural development
projects, dams, deforestation, floods, droughts and climatic changes that resulted
in the emergence of diseases such as Argentine hemorrhagic fever, Korean
hemorrhagic fever (Hantaan) and Hantavirus pulmonary syndrome. Human
demographic behaviour has been a factor in the spread of dengue, the source for
the introduction and spread of HIV and other sexually transmitted disease.

The engine that is driving the re-emergence of many of these diseases is the
ecological change that brings humans into contact with pathogens. Except for
Brazilian pururic fever which may represent a new strain of *Haemophilus influenzae*,
biotype *aegyptius* most of the emerging diseases are of cultural origin. The role of
humans in the development of antibiotic resistance by medical and agricultural
practices is well established. Humans are clearly ‘the world’s greatest evolutionary
force’ (Palumbi 2001). Palumbi argues:

Human ecological impact has enormous evolutionary consequences as well
and can greatly accelerate evolutionary change in the species around us,
especially disease organisms, agricultural pests, commensals, and species
hunted commercially. For example, some forms of bacterial infection are
insensitive to all but the most powerful antibiotics, yet these infections are
increasingly common in hospitals. Some insects are tolerant of so many
different insecticides that chemical control is useless. Such examples illustrate
the pervasive intersection of biological evolution with human life, effects
that generate substantial daily impacts and produce increasing economic
burden.

(Palumbi 2001)

The acceleration of evolution, according to Palumbi, costs the United States at
least $33 billion and as much as $50 billion a year in costs related to the antibiotic
and pesticide resistance in organisms.

Conclusion: ‘getting ahead of the curve’

The CDC (1994), following the IOM recommendation, proposed a plan, which
was later modified (CDC 1998), suggesting a four-pronged attack on emerging
disease. First, they emphasize a need to strengthen infectious surveillance and
response that can detect and contain infectious agents. Second, they address the
need to research issues raised by these challenges. Third, there is a need to repair
the public health infrastructures and training. Finally, there is a need to strengthen
prevention and control programs ‘locally, nationally, and globally’. The CDC and
IOM plans focus on detection and response that enhances the capacity of
infrastructure to detect and respond to disease threats.
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David Satcher (1995), Director of the CDC, in the inaugural issue of *Emerging Infectious Disease* is forceful in his assessment of what may be the key in responding to infectious diseases:

We cannot overstate the role of behavioral science in our effort to get ahead of the curve with emerging infections. Having the science or laboratory technology to control infectious diseases is not enough, unless we can influence people to behave in ways that minimize the transmission of infections and maximize the efforts of medical interventions. For example, even though HIV/AIDS does not have a vaccine or cure, it is almost entirely preventable. For many people, however, reducing the risk for HIV infection and AIDS requires important changes in lifestyle or behavior. We must use our knowledge of human behavior to help people make lifestyle changes and prevent disease. (Satcher 1995)

We understand the proximate and ultimate causes of disease. We understand the cultural practices that allow the pathogen to ‘jump’ their species barrier and escape their geographic boundaries. The observation that the widening economic and political gap has been the pattern of our post-Neolithic history does not mean that it is inevitable. The issue of inequality must be addressed. The question remains as to our will to deal with inequality. To date, the issue seems to have been avoided and accepted as an inevitable aspect of evolution. It is time to reconsider the issue.

These are the words with which Edward B. Tylor (1881) choose to close his book *Anthropology*, written over a century ago:

Readers who have come thus far need not be told in so many words of what the facts must already brought to their minds – that the study of man and civilization is not only a matter of scientific interest, but at once passes into the practical business of life. We have in it the means of understanding our lives in and our place in the world, vaguely and imperfectly it is true, but at any rate more clearly than any former generation. The knowledge of man’s course of life, from remote past to the present, will not only help us forecast the future, but may guide us in our duty of leaving the world better than we found it.

(Tylor 1881: 439–440)

The words echo advice that we can live with today.2

Notes

1 Demographic transition theory is a generalized model of population structure that is the basis for understanding fertility and mortality processes in contemporary populations. In the first stage, which was thought to represent most of human evolutionary history, populations were at their highest natural fertility and high mortality, resulting in little increase in natural population. In the second stage, there is a decrease in mortality and natural fertility remains high, resulting in a rapid increase in population. The third stage, mortality rates are low and birthrates begin to decline, resulting in a slow population growth. In the last stage, there is low fertility and low
mortality with no natural increase in population. Given the evidence that Paleolithic populations were controlling fertility, demographic transition theory needs to be re-evaluated.

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References

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