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Evolutionary, historical and political economic perspectives on health and disease

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Abstract

The origin and rise of social inequalities that are a feature of the post-Neolithic society play a major role in the pattern of disease in prehistoric and contemporary populations. We use the concept of epidemiological transition to understand changing ecological relationships between humans, pathogens and other disease insults. With the Paleolithic period as a baseline, we begin with ecological and social relationships that minimized the impact of infectious disease. Paleolithic populations would have retained many of the pathogens that they shared with their primate ancestors and would have been exposed to zoonoses that they picked up as they adapted to a foraging existence. The sparse mobile populations would have precluded the existence of endemic infectious disease. About 10,000 years ago, the shift to an agricultural subsistence economy created the first epidemiological transition, marked by the emergence of infections, a pattern that has continued to the present. Beginning about a century ago, some populations have undergone a second epidemiological transition in which public health measures, improved nutrition and medicine resulted in declines in infectious disease and a rise in non-infectious, chronic and degenerative diseases. Human populations are entering the third epidemiological transition in which there is a reemergence of infectious diseases previously thought to be under control, and the emergence of novel diseases. Many of the emerging and reemerging pathogens are antibiotic resistant and some are multi-antibiotic resistant. Inequality continues to widen within and between societies, accelerating the spread of emerging and reemerging diseases.

Keywords: Epidemiological transition; Pathogen; Evolution; Social stratification

Introduction

The United States Surgeon General William T. Stewart, testifying before Congress in 1969, proclaimed that it was now "… time to close the book on infectious disease as a major health threat." Buoyed by the successful development of vaccines, antibiotics and pesticides, Stewart assumed that the eradication of infectious disease was imminent. Two years earlier, in a meeting with state medical officers Stewart claimed smallpox, bubonic plague, and malaria "were things of the past" and predicted (Ryan, 1997:6),

> Typhoid, polio, and diphtheria were heading in the same direction. While syphilis, gonorrhea, and tuberculosis were not quite so readily defeated, it was only a matter of time before every plague that had ever struck fear into the heart of decent Americans would be a distant memory.

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Stewart, as America's chief medical officer, was positioning the government's public health system to respond to the next perceived health challenge of preventing chronic and degenerative diseases.

Stewart's optimism was short lived. There was an institutional myopia that failed to consider the extent of infectious disease in the third world and the consequences of antibiotic and pesticide. Three decades later, The World Health Organization (WHO, 2001, Appendix A) reports that of the 55 million global deaths in the year 2000, 14 million were the result of infectious, parasitic and respiratory diseases. Nearly 4 million deaths due to lower respiratory infections were recorded; approximately 3 million deaths were attributed to HIV/AIDS, over 2 million children succumbed to diarrheal disease, over a million and a half died from tuberculosis and 1 million deaths were from the consequences of malaria. The World Health Organization (WHO, 1995) calculates that 2 billion people in the world are infected with hepatitis B virus, two billion are infected with tuberculosis (Sizemore & Fauci, 2002) and 40 million people have AIDS (UNAIDS/WHO, 2001).

Stewart did not comprehend the degree to which antibiotic-resistant pathogens (Cassell & Mekalanos, 2001; Okeke & Edelman, 2001; van den Bogaard, & Stobberingh, 2000) and pesticide-resistant insects (Roberts & Andre, 1994) would become a problem, making the eradication of some disease vectors an impossibility. The extent of the impacts of ecological disruption (Mayer, 2000; Western, 2001; Woodruff, 2001) and of inequality (Houweling, Kunst, & Mackenbach, 2001; Wagstaff, 2000) on the disease process were also underestimated. *Homo sapiens* continue to create unprecedented ecological disturbances that are having unparalleled evolutionary impact (Palumbi, 2001). These ecological disturbances have accelerated changes in antibiotic and pesticide resistance and cost Americans an estimated 33 billion to 50 billion dollars a year (Palumbi, 2001).

The decline of infectious disease and the rise of chronic disease were thought to be the culmination of a demographic trend that began with the development of public health measures and germ theory-based medicine in the industrial world. The changing pattern of disease was the basis of an epidemiological theory first proposed by Abdul Omran (1971) who on analyzing data from England, Wales and Sweden during the last century, observed a trend in which human populations were experiencing shifts in disease patterns that were of such a fundamental nature that they altered population processes (Omran, 1971:162). Specifically, he modeled the transition of human populations from a state of pathogen-induced infections to a state of chronic, man-made disease (Omran, 1971:163). Omran's (1971, 1983) model was driven by processes that eliminated infectious diseases, allowing populations to age with a subsequent rise in chronic and degenerative diseases and those of "man's" making. Omran's use of the term "man-made" disease anticipated the contemporary roles that pollution and other by-products of the industrial age play in the disease process (Caldwell, 2001).

Our primary objective is to provide an evolutionary perspective on health and disease, using epidemiological transition to examine the role that wealth and poverty play in the process. Specifically, we are interested in the role that disparities in wealth (Brockerhoff & Hewett, 2000; Coburn, 2000; Hawe & Shiell, 2000; Shaw, Orford, Brimblecombe, & Dorling, 2000) play in continuing evolutionary processes that have affected humans for the last 500 generations. Social stratification originally evolved because it brought benefits to emerging elites. In general, these benefits, which often included resources that improved health, came at the expense of the others. We use the concept of macroparasitism (McNeill, 1976) to understand the changing pattern of inequality. When organisms appropriate others as continuing sources of food and energy, we can characterize that relationship as parasitism. Social stratification within societies and between them is an evolutionary strategy that we consider "macroparasitism"[2] (Brown, 1987; McNeill, 1976). We will discuss inequality in the changing relationships of pathogens and people as they experience epidemiological transitions.

Epidemiological transition

Given the failure to eradicate infectious diseases or control emergent diseases, one might be tempted to reject the notion of epidemiological transitions. However, we are applying Omran's model in a broader evolutionary context. Armelagos and colleagues (Armelagos & Barnes, 1999; Barnes, Armelagos, & Morreale, 1999; Barrett, Kuzawa, McDade & Armelagos, 1998) have argued that human populations have gone through an earlier epidemiological transition and are presently experiencing a third.

The shift from foraging to primary food production represents the first epidemiological transition. The domestication of plants and animals in the Neolithic brought about a marked increase in the prevalence of infectious disease. Increase in population size and density, domestication of animals, sedentarism,

[2]It is interesting to note that parasitism originally referred to a human social relationship. According to the *Oxford English Dictionary*, parasitism was defined by the ancient Greeks as a relationship in which a wealthy patron would pay a person to dine with and entertain him. The earliest English recording of the word continues this usage. Beginning from 1727–1741, parasite described biological pathogenesis and then was restricted to botanical relationships.

cultivation and social stratification created a dramatic shift in disease ecology. Controversy surrounds the second epidemiological transition (Omran's original conceptualization). The causes and extents of the decline in infectious disease and the rise of chronic disease remain matters of debate. Finally, we are living in the third epidemiological transition in which many antibiotics are losing their effectiveness (Barrett et al., 1998). This period is characterized by the globalization of reemergent infectious diseases (Hughes, 2001; Morse, 1997) that are often resistant to multiple antibiotics (Farmer, 1997; Jacobs, 1994; Wellems & Plowe, 2001), and the emergence of novel diseases that threaten human populations.

We are well aware of the criticism of the concept of "emerging" disease. Paul Farmer (1996) argues that emerging diseases are only "discovered" when they have an impact on Americans. Lyme disease was an object of research years before wealthy suburbanites built houses in wooded golf course communities that change the ecology enough, creating an environment that brings ticks and rich humans together (Farmer, 1996). Furthermore, he claims that "emerging" diseases are usually presented as a result of human behavior or microbial changes. Even when researchers provide an ecological perspective for emerging disease, they will usually fail to consider them in a broader political–economic context (Farmer, 1996). It is precisely this criticism that we are addressing in the analysis of epidemiological transition presented here. We are interested in the social forces that influence inequality in an increasingly interconnected world, and how these inequalities affect the disease process (Farmer, 1996). Farmer's criticisms echo those made by Meredith Turshen (1977) a quarter of a century ago, in which she claims epidemiological models of that time focused narrowly on relationships between hosts, pathogens and the environment and failed to consider the cultural, political and economic complexity in the disease process.

With economic and technological development, there has been an evolutionary tendency for the disparities between the rich and the poor, the healthy and the sick, to increase. In this century, the widening gap between those on the top and the bottom of the social hierarchy occurs both within and between societies (Houweling et al., 2001; Shaw et al., 2000), and is greater than ever before in human history. This gap has serious health implications. While disease and death are inevitable, a major cause of unnecessary, premature, preventable disease and death is simple; it is extreme poverty.

The Paleolithic as a baseline

Contemporary gatherer-hunters provide the model that we can use to reconstruct disease patterns that would have affected our Paleolithic ancestors. Sprent (1962, 1969) describes "heirloom species" as parasites that we share with early hominids' anthropoid ancestors and that remained with hominids even after speciation. Head and body lice (*Pediculus humanus*), pinworms, protozoa found in modern humans and bacteria such as *salmonella*, *typhi* and *staphylococci* (Cockburn, 1967a, b) are examples of heirloom pathogens.

Zoonoses are souvenir species (Sprent, 1962, 1969) whose primary hosts are non-human animals, and who only incidentally infect humans. Insect bites, processing and eating contaminated meat, and animal bites are sources of zoonotic disease. Avian or ichthyic tuberculosis, leptospirosis, relapsing fever, schistosomiasis, scrub typhus, tetanus, trichinoses, trypanosomiasis and tetanus are among the zoonotic diseases that likely afflicted earlier gatherer-hunters (Cockburn, 1971). However, small population sizes would have precluded infectious diseases having a major evolutionary impact and the daily forays of gatherer-hunters from their base camps and frequent movement of these camps would have decreased their contact with parasites found in human feces. Deadly diseases would soon run their course as the small number of susceptible individuals was infected. Paleolithic populations lacked the common and deadly communicable diseases such as influenza, measles, mumps, and smallpox.

Gatherer-hunters are equal opportunity hosts. Because of their egalitarian nature, they lack the class structure that creates differential exposure to disease among segments of the society. However, as women gather and men hunt (the producing segment of society), they are differentially exposed to disease vectors in the course of their daily subsistence rounds. While men may be exposed to pathogens and parasites as they prepare the animals they kill, women cooking the meat would be similarly exposed. Since the producing segments of the society are differentially infected, diseases may have a more disruptive effect on the survival of the group.

The first epidemiological transition

Agriculture in the Old World is evident from about 10,000 years ago. Five independent areas of cultivation are found in Mesopotamia, Sub-Saharan Africa, Southeast Asia, northern China and southern China. In the Western hemisphere, sites in Mesoamerica and South America represent the sixth and seventh areas of independent centers of domestication. The development of primary food production is the basis of the changing disease pattern. The rapid increase in population size and density, sedentarism, the domestication of animals, extensive ecological disruption from cultivation and the rise of social and economic inequality are all factors that increase infectious disease risk.

By 5000 BCE, large settlements existed in Mesopotamia, and 1000 years later, the rise of centralized polities that controlled vast irrigation systems are evident. In Mexico, by 3500 years ago, well-established settlements arose with evidence of extensive hierarchies rising 1500 years later. The political and economic changes with the development of agriculture created social classes with differential access to resources, a system that continues to this day.

Sedentarism increased parasitic infection because of proximity of the living areas to source of waters and the areas where human waste was deposited. The contiguity of habitation to the space where domesticated animals were kept, created a cluster of disease vectors. Parasites, such as tapeworms associated with domesticated goats, sheep, cattle, pigs, and fowl, would have infected these early farmers. However, recent research on the tapeworm's (*Taenia*) genetic phylogeny (Hoberg, Jones, Rausch, Eom, & Gardner, 2000) suggests that the differentiation among the common parasites occurred during the Paleolithic and that humans were the source of infections in cattle, sheep and goats that occurred after domestication of these species.

The milk, hair, and skin of domesticates, as well as animal dust, transmitted anthrax, Q fever, brucellosis, and tuberculosis. Peridomestic animals such as rodents and sparrows, which are drawn to human habitats, are also a source of disease. Cultivation often exposes workers to insect bites, and diseases such as scrub typhus becomes common (Audy, 1961). Livingstone (1958) and Wiesenfeld (1967) show that slash-and-burn agriculture in West Africa exposed populations to the mosquito that is the vector for malaria. Environmental disturbances during the clearing and cultivating of land increase human contact with arthropod vectors that prefer human habitats and that carry yellow fever and filariasis. For example, *Aedes aegypti* (the vector for yellow fever and dengue) is an artificial container breeder. Irrigation agriculture (Sattenspiel, 2000; Watts, Khallaayoune, Bensefia, Laamrani, & Gryseels, 1998) and fertilizing with human waste increases contact with non-vector parasites (Cockburn, 1971).

Surprisingly, agriculture subsistence increased dietary deficiencies that had health implication for agriculturalists after the Neolithic (Cohen & Armelagos, 1984). Agricultural subsistence invariably reduces the variety of foods that are available to people (Armelagos, 1987) and many increase a reliance on a single-grain crop such a millet, rice, wheat or maize. The reduction of the dietary niche (Katz, 1987) resulted in dietary deficiencies that can increase the impact of infectious disease (Guerrant, Lima, & Davidson, 2000; Rice, Sacco, Hyder, & Black, 2000; Stephenson, Latham, & Ottesen, 2000) while food storage increased the potential for food poisoning (Brothwell & Brothwell, 1998). The combination of a complex society, increasing divisions of class, epidemic disease, and dietary insufficiencies increases the stress levels in the population.

Paynter (1989) suggests that social stratification may not have initially reflected inequality. Even though the distinctions between rulers and ruled may have been great, there may be equality within the ruled group. Nevertheless, research on Middle Mississippian populations (Goodman, Lallo, Armelagos, & Rose, 1984)—well-defined chiefdoms—suggests that those populations were suffering from significant nutritional and infectious disease stress.

Urban development and disease during the Neolithic

The impact of contemporary urbanization on health has been studied extensively (Lawrence, 1999; McMichael, 2000). Large settlements increased the already difficult problem of removing human wastes and delivering uncontaminated water. Cholera, a waterborne disease, became a potential problem and lice that carried typhus and the fleas infested with the plague bacillus spread disease from person to person. High population densities enhanced the respiratory transmission of the plague and the transmission of viral diseases such as measles, mumps, chicken pox, and smallpox.

The rapid urbanization of human populations and expansion into new ecological zones represents one of the most important forces in the evolution of infectious disease. From their earliest beginnings, urban centers established in the Near East and in the western hemisphere grew rapidly. Urban centers at Memphis (Egypt) reached 30,000 individuals by 3100 BCE, Ur in Babylonia reached 65,000 inhabitants by 2030 BCE and Babylon had a population of 200,000 by 612 BCE (Chandler, 1987). Populations of this size could maintain some diseases in an endemic form. For example, estimates of the population size necessary to maintain measles vary from 200,000 (Black et al., 1974) to 1,000,000 people (Cockburn, 1967a).

The processes of urbanization and globalization (Robertson, 1992), and the era of exploration in the fifteenth and sixteenth centuries were linked. McNeill (1976) describes the confluence of civilizations in Eurasia (500 BCE–1200 CE) and the development of the Mongol Empire (1200–1500 CE), which created a unique disease pool where pathogens are shared over a large area. Earlier trade in the Old World and exploration in the fifteenth and sixteenth centuries made inevitable transcontinental and transoceanic disease exchange that increased the potential for an endemic disease to be transmitted as epidemic disease in areas of contact.

Cross-continental trade and travel resulted in intense epidemics (McNeill, 1976; Zinsser, 1935). The plague, which was an endemic disease in China, spread by trade to Europe and became epidemic during the 1300s, killing

a quarter of the 100 million inhabitants (Gottfried, 1983). The introduction of novel diseases such as measles to groups that did not have the experience to deal with them (McNeill, 1976) had overwhelming effects. In line with his metaphor of macroparasitism, McNeill (1978) describes civilizations as "digesting" encountered populations, as in the swift decimation of Native Americans by disease vectors that cleared the path ahead of the explorers. Explorers would frequently find villages abandoned in aftermath of ravaging diseases (Dobyns, 1983; Ramenofsky, 1987).

The transmission of disease is usually a two-way street. Since the Americas never had the collection of domesticated animals characteristic of the Old World Neolithic, they did not have a large variety of endemic zoonoses that could easily be transmitted to the Europeans. However, it is likely that the New World was the source of the treponemal infection transmitted to Europe. The Native Americans had an endemic non-venereal treponemal infection (Rothschild, Calderon, Coppa, & Rothschild, 2000) that was transmitted to the Old World (Baker & Armelagos, 1988). When introduced into the Old World, the sexual transmission of the treponeme resulted in a more severe and acute infection. Counterclaims that pre-Columbian syphilis existed in Europe have been made (Dutour, Pálfi, Bérato, & Brun, 1994), but the resolution of this debate may await the recovery of material that can be identified as a treponemal pathogen from Old World, pre-Columbian archaeological bone.

Urbanization in a industrial world

The process of industrialization, which began a little over 200 years ago, led to an even greater environmental and social transformation. In 1800, London was the only city in the world with a million inhabitants. City dwellers would have been 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 such 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 centers, with their extremely high mortality, were not able to maintain their population base by reproduction until late in the 1800s. Mortality outstripped fertility, requiring the in-migration of rural populations to the city in order to maintain their numbers. Economic factors drew migrants to the squalor of the cities that were "population sinks".

In 1800, when London was the only city of a million people, just 3% of the world's population lived in an urban setting. By 1900, urban dwellers had increased to 14%, with 12 cities having a million or more inhabitants. Fifty years later (1950) this proportion had increased to 30%. Now, about 47% of the population lives in urban areas, and the number of cities with over a million inhabitants has risen to 411 (United Nations, 2000). In the developed nations, the percent of urbanites has reached 76%—nearly twice the proportion of developing nations. The projected increase for the year 2030 is that 60% of the world's population will be living in urban centers and that this increase will occur in the less developed nations (United Nations, 2002) which lack the infrastructure to meet the health needs of urban population (Garrett, 1994, 2001).

In 1950, New York City with a population 12 million was the largest megacity (populations over 5 million) in the world, and at that time, only two of the largest top 10 urban agglomerates were in developing countries. At the beginning of this century, six of the largest urban areas (all with over 12 million inhabitants) were in developing countries, and it is projected that by 2015, there will be eight centers, all with over 17 million inhabitants, in the same category, four of which will be on the Indian subcontinent.

The detrimental effects of industrialization have continued globally as pollution from the industrial production of commodities has created health concerns (The American Lung Association, 2001). The implications of contaminated water, pesticide use and depleted ozone on human health and food production are significant, for at no other periods in human history have the changes in the environment been so rapid and so extreme. Consequently, WHO has made the health problems of cities one of its major initiatives (Goldstein, 2000; Harpham, Burton, & Blue, 2001; Kenzer, 2000; McMichael, 2000; Tsouros, 2000).

The second epidemiological transition: the rise of chronic and degenerative disease

The increasing prevalence of chronic diseases is related to increases in lifespan longevity that have occurred over the past few centuries. Cultural advances have resulted in a larger percentage of individuals reaching the oldest age segment of the population, while the technological advances that characterize the second epidemiological transition often result in an increase in environmental degradation.

The development of the germ theory of disease has been considered as the major force behind the decline of some infectious diseases. However, others have noted infectious diseases were declining before the initiation of many immunization programs and therapeutic practices (McKeown, 1979). Critics of McKeown have focused on his use of evidence for improved nutrition (Johansson,

1992; Schofield & Reher, 1991) and failure to consider improvements in public health practices (Johansson, 1992; Kunitz, 1991; Schofield & Reher, 1991). The development of immunization resulted in the control of many infections and was the primary factor in the recent eradication of smallpox; as well, in 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 expectancies at birth; the resulting increase in elderly individuals has yielded an increase in chronic and degenerative diseases.

At another level, critics argue that many countries have never experienced the second epidemiological transition and in others, the transformation varied (Barrett et al., 1998). Since the 1960s, in some countries the rate of the decline in infectious disease has decelerated (Gwatkin, 1980), never reaching the levels in the developing world (Gobalet, 1989). Rapid urbanization, marked social inequalities and a lack of public health infrastructure have resulted in the pattern where the poorest segments of the population are exposed to infectious diseases, while chronic degenerative diseases have increased among the affluent and emerging middle classes (Muktatkar, 1995). In Mexico and Brazil and other middle-income countries, socioeconomic status is inversely related to risk of chronic diseases (Popkin, 1994). This pattern is similar to what we see in affluent nations such as the United States and the United Kingdom (Kaplan & Keil, 1993). This differential impact within and between nations reflects the influences of economic factors on the disease process. We have never considered epidemiological theory as a unilinear evolutionary model, and the variation others see as problematic, we see as an object of further study.

The third epidemiological transition

Human populations are in the midst of the third epidemiological transition, in which there is a reemergence of infectious diseases, many of which are multiple antibiotic resistant, and have a great potential for global impact. In this 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) and degenerative disease of the second.

The reemergence of infectious diseases has been one of the most interesting evolutionary stories of the last decade, capturing the interest of scientists and the public (Drexler, 2002). A list of the 29 most recent emerging diseases has been reported in the last three decades (Lederberg, 1998; Lederberg, Shope, & Oaks, 1992), citing these as the result of an interaction of social, demographic and environmental changes in global ecology and in the adaptation and genetics of microbes. Morse (1995) sees emerging disease as a result of demographic changes, international commerce and travel, technological change, breakdown of public health measures (Garrett, 1994, 2001) and microbial adaptation.

Morse describes ecological disruptions that result from agricultural development projects, dams, deforestation, floods, droughts and climatic changes, all of which have resulted in the emergence of diseases such as Argentine hemorrhagic fever, Korean hemorrhagic fever (Hantaan) and Hantavirus pulmonary syndrome. Human demographic behavior has been a factor in the spread of dengue (which is now becoming an urban disease), as well as the source for the introduction and spread of HIV and other sexually transmitted diseases.

The forces that generate the reemergence of many of these diseases are the ecological change that brings humans into contact with pathogens. Except for the Brazilian pururic fever, which may represent a new strain of *Haemophilus influenzae, Biotype aegyptius*, most of the emerging diseases are anthropogenic. The role of humans in the development of antibiotic resistance by way of medical and agricultural practices is a deadly example of human impact on pathogenic evolution.

Conclusion: current inequality and health

We have provided an evolutionary perspective for understanding the emergence of disease since the Neolithic. Furthermore, inequalities between and within societies, which increase the risk for contracting infectious diseases that began in the Neolithic, have widened. In the United States from 1947 to 1979, all economic levels experienced three decades of improved prosperity (Table 1). The bottom 80% of American families experienced improvements of income of at least 100%. The top 20% showed 99% growth and the top 5% with the highest family income realized an 86% increase. But from 1979 to 1998, there occurred a remarkable economic decline for the families in the lower economic range. The poorest 20% of families actually suffered a 5% decline in family income. The second quintile improved 3%, the middle quintile gained 8%, and the fourth quintile realized a 15% increase. The richest 20% of families showed a 38% increase and the top 5% showed an improvement of 64%. The disparity is even greater after income taxes are calculated (Table 1).

The data on income equality for the world shows the same pattern of rising inequality (Table 2). The relative income of the richest 20% of the global population has increased from 70% to 89% during the last decades. During that same period, the lowest quintile's income declined from 2.3% to 1.2%. The 60% of the population in the middle experienced a nearly 65% decline in

Table 1
Rising together and drifting apart. Changes in family income from 1947 to 1979 and 1979 to 1998 by quintile and top 5%. Parentheses in the drifting apart column represents after taxes income

	Rising together (%)	Drifting apart (%)
	1947–1979	1979–1998
Top 5%	86	64 (115)
Top 20%	99	38 (43)
Fourth 20%	114	15 (14)
Middle 20%	111	8 (8)
Second 20%	100	3 (1)
Bottom 20%	116	−5 (−9)

Source: 1947–1979: Analysis of US Census Bureau data in Economic Policy Institute, The State of Working America 1994–1995, p. 37. 1979-1998: US Census Bureau, Historical Income Tables, Table F-3. The after taxes data is from Center on Budget and Policy Priorities, The Widening Income Gulf, September 4, 1999.

Table 2
Growing global disparity

	1960 (%)	1970 (%)	1980 (%)	1989 (%)	1998 (%)
Highest 20%	70.2	73.9	76.3	82.7	89.0
Middle 60%	27.5	23.6	22.0	15.9	9.8
Lowest 20%	2.3	2.3	1.7	1.4	1.2
Gini	.54	.57	.60	.65	.70

Source: United Nations Development Program 1996. Extracted by poorcity.richcity.com/entundp.htm.

wealth. This disparity is reflected in The World Bank's Gini coefficient. A coefficient of zero would indicate equal distribution of income and a coefficient of one would indicate that all the income is in the hands of one individual or group. The Gini index has increased globally from .54 to .70 in the last 40 years.

To put this inequality in another perspective, the 200 richest people in the world have a combined wealth equivalent to the wealth of 2.5 billion of the poorest people. Three Americans (Bill Gates, Paul Allen and Warren Buffett) have personal wealth greater that the GDP (Gross Domestic Products) of the world's 41 poorest nations and their 550 million citizens (Gates, 2000:6). In terms of health, 968 million people in developing nations are without access to safe water, 2.4 billion lack basic sanitation, 163 million children under 5 years of age are underweight and 11 million children die each year from preventable diseases (UNDP, 2001).

However, we have to address an ongoing debate in the social sciences centered on the relationship, if any, between economic inequalities, characterized chiefly by differential incomes, and health outcomes. R.G. Wilkinson (1998) is well known as an outspoken proponent of the notion that income inequality is a driving force in the creation of class discrimination and both external and internal value judgments based on one's position in an economically based social hierarchy (Wilkinson, 1999). Describing statistically robust correlations between the level of income inequality in and between societies and differential health outcomes, Wilkinson has shown that wealthier individuals tend to be healthier than those who are poorer. Such a hypothesis is supported when relative income differences are linked to levels of social solidarity and relative egalitarianism (Egolf, Lasker, Wolf, & Potvin, 1992), and when placed in the larger context of globalization (Coburn, 2000; McMichael, 2000). Recently, critics such as Deaton (2001) have rejected Wilkinson's hypothesis that relative income inequality impacts health, emphasizing impoverishment and social discrimination as more important variables than income. Yet Deaton's critique holds only if one looks at income as a static and exclusive category, a theoretical perspective that does not seem particularly useful. To say that political inequality and income inequality are separate things (Deaton, 2001, 24), especially in a capitalist system such as Britain or the United States, ignores their interconnectivity and the effects that racism and sexism can have on job opportunities and thus income. Montague (1996) corroborates Wilkinson's statements that morbidity and mortality are higher amongst the poor than the rich, and agrees that it is not the average income of a society that is the most important indicator, but the size of the gap between the richest and the poorest in a society. This conclusion has been observed at international levels (Syme, 2000) and is supported within the historical and evolutionary frameworks of this paper.

Global capitalism as an economic strategy (macroparasitism) has allowed members of the so-called 'First World' to accumulate vast quantities of material and social capital, often through the large-scale exploitation of resources and people in the 'Third World'. The social power gained by elites can insulate them from food shortages and unhygienic living conditions endured by the poor, which are in part due to the actions of elites (Gardner & Halweil, 2000).

An additional aspect of the macroparasitic strategy is ideological in its attribution of causality regarding global inequity and health. Blame is often assigned to outside entities such as genes, miasma or germs, or else to the moral inadequacy of individuals in failing to raise themselves up out of their situation. Diseases such as AIDS and leprosy are often attributed to the unsavory behavior or lack of moral fiber of the victims (Farmer,

1996), rather than to the statistical vulnerability and chronic misery of being poor. This allows elite classes to present themselves as superior in both biological and ideological spheres, obscuring the role that they play in causing, exacerbating and perpetuating the problem of global inequality and widespread poverty.

The fundamental similarity in communities with undernourished, sickly individuals and inadequate measures for dealing with these issues is poverty, and it is poverty that perpetuates unhealthy conditions. World Health Organization statistics claim that in 1998, those in the poorest countries, who had access to only 10% of the global resources allocated for health care and treatment, shouldered 90% of the global disease burden. Also, over 50% of those who live in the 46 poorest countries in the world have little access to sanitation facilities or safe drinking water. In these countries, factors such as resource scarcity, inadequate education and pollution must be addressed before any permanent solutions can be reached (United Nations, 2000).

Max Weber's definition of social class hinged on the notion of differential "life chances". Health indicators of morbidity, mortality and life expectancy are therefore appropriate measures of "life chances" that vary inversely with wealth. The concept of "life chances" supports the notion that poverty is, at its core, a reduction in or elimination of choices, as well as access to strategic resources such as food, clean water, medicine, land, money, education and social mobility. Gardner and Halweil (2000) attribute much of the plight of the poor not to shortages, but to the unequal distribution of wealth and the concentration of resources in a privileged global minority. They see the origins of this asymmetry in the political and economic decisions of the late 20th century such as the support of agribusiness and "trickle-down" economics, and the failure to support the efforts of independent farms and businesses in both the First and "developing" Third Worlds. Echoing these statements is a quote from the British Medical Journal (1996), which reads, "What matters in determining mortality and health in a society is less the overall wealth of that society and more how evenly wealth is distributed. The more equally wealth is distributed, the better the health of society."

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 (ICD). It is given in code Z59.5—extreme poverty. Of course, with this WHO ICD classification there is the question: what is *extreme* poverty? Extreme—compared to what? Certainly basic human needs of food, water, adequate shelter, access to health care, and adequate social support might allow us to make a minimal definition. The World Bank estimates that 3 billion people in the world live on less than $2/day. But is it appropriate or advantageous in considering health indicators to think of relative deprivation?

The downward spiral of disease and poverty ("the poor are sicker, and they become sicker because they are poor") has been used as the rationale for "investments in public health" for the past 60 years. The implication is that a public health intervention would reverse this into an upward spiral of health and wealth; an example is the "malaria blocks development model" (Brown, 1997). Recently, Sachs and colleagues (Gallup & Sachs, 2001, Sachs & Malaney, 2002, Sachs, 2001) have demonstrated again *on the level of cross-national data*, that diseases like malaria carry a heavy economic burden that limit the possibility of economic growth in malaria-prone nations.

These issues are at the core of medical anthropology in its most ancient beginnings, and it is impossible to understand current issues of inequality without examining their historical roots. It is also necessary to examine the rise of chronic and epidemic diseases, nutritional stress and social inequalities as inextricably linked as we have done in this paper. It is hoped that based on conclusions here, others will take this multifaceted and evolutionary approach, addressing epidemiology, nutrition, poverty and globalization, in order to gain the level of understanding necessary to begin to address these problems. Rudolf Virchow in 1849 (Die Einheitsbestrebungen) argued, "Once medicine is established as anthropology, and once the interests of the privileged no longer determine the course of public events, the physiologist and the practitioner will be counted among the elder statesmen who support the social structure. Medicine is a social science in its very bone and marrow." (Virchow 1958)

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