Evolution in Health and Disease

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HUMAN EVOLUTION AND DISEASE: PUTTING THE STONE AGE IN PERSPECTIVE

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The search for single gene mutations is one of the best funded areas of medical research budgets, yet these mutations may account for a small fraction of all diseases. The true culprits are often wild-type genes that were adaptive in past environments. For example, less than 2 per cent of all breast cancers can be attributed to the BRCA mutations and other susceptibility genes (Peto *et al.* 1996); 98 per cent of malignancies are probably caused by normal genes in the face of novel life-history patterns (Short 1976; Eaton *et al.* 1994).

The change in the environment of the genes has been one of the centrepieces of the new evolutionary medicine (Eaton *et al.* 1988; Williams and Nesse 1991; Nesse and Williams 1994). In particular, it has been argued that human biology is adapted to the hunter–gatherer lifestyle of the 'Stone Age'. From a genetic standpoint, modern humans have been viewed as Upper Palaeolithic preagricultural foragers (Eaton *et al.* 1988). To situate these arguments in time, we note that the Old Stone Age or Palaeolithic is the period from about 2.5 million years ago to 10 000 years ago (Lewin 1993). Anatomically modern *Homo sapiens* first appeared in Africa approximately 130 000 years ago (Foley and Lahr 1992). Hunting and gathering predominated over scavenging by 55 000 years ago (Steiner and Kuhn 1992), and the technological revolution of the Upper Palaeolithic began about 40 000 years ago (Lewin 1993; Jurmain *et al.* 1997). Those who view contemporary humans as 'Stone Agers in the fast lane' (Eaton *et al.* 1988) assume that the 'environment of evolutionary adaptedness' ended 10 000 years ago (Symons 1979; Tooby and Cosmides 1989; Barkow *et al.* 1992).

Ten thousand years is long enough for roughly 400 human generations, implying that we are genetically similar, but not identical, to our ancestors of the Upper Palaeolithic. The forces of evolution (natural selection, gene flow, mutation, and drift) continue to act on human populations and have demonstrably altered allele frequencies since the origin of agriculture (Durham 1991). The best documented examples of natural selection in modern human populations are the evolution of malaria resistance (Neel 1947; Livingstone 1958, 1984; Cavalli-Sforza and Bodmer 1971; Durham 1991) and lactose tolerance (Simoons 1978; Durham 1991), both of which are thought to have originated within the past few thousand years. In other species, natural selection has been observed over just a few generations (Grant 1986), reminding us that it is imprudent to underestimate the importance of micro-evolution in contemporary human populations. None the less, it is unlikely that a large percentage of the 100 000 odd genes in the human genome are more recent than the Stone Age (see Sokal *et al.* 1991; Neel 1994).

If our genes have changed relatively little over the past 10 000 years, does it follow that we have mostly Stone Age genes? The answer must surely be that 2.5 million years is a flash in the pan compared with the nearly 4 billion years of evolution behind *Homo sapiens*. The molecular evidence shows that most of our genes are of far greater antiquity than the Pleistocene. For example, we continue to use

the same genetic code as all other species on Earth including bacteria (Ridley 1993). Many of our proteins, such as the histones that package our DNA, are virtually identical to those of other organisms ranging from yeast, to sea urchins, to marigolds, to mice (van Holde 1989). From a genetic standpoint, the Stone Age may have no greater significance than any other period of our evolutionary past (see Foley 1995/1996).

Although there is no genetic reason for emphasizing the Stone Age, perhaps there is an ecological reason. Was the Palaeolithic/Neolithic boundary and the rise of agriculture the most important transition in the aetiology of modern disease? The view that agriculture was the chief novelty that selection has not caught up with is implicit in much of the work on Darwinian medicine (e.g. Eaton *et al.* 1988, 1994). It seems to derive from the broader supposition that human behaviour is rarely adaptive except among populations that pursue a lifestyle similar to ancestral hunter–gatherers (e.g. Barkow *et al.* 1992). Recently settled foragers under the influence of missionaries are sometimes thought to provide better data on human adaptation than traditional agriculturists who do not use contraception and have practised essentially the same mode of subsistence for centuries, if not millennia (e.g. Eaton *et al.* 1994).

In this chapter we will attempt to put the Stone Age in perspective by demonstrating that the neolithic revolution was just one of many transitions relevant to human disease. Different medical problems have their origins in different transitions, and it is helpful to be judicious in deciding which problems can, in fact, be ascribed to agriculture. Thus, rather than search for Stone Age legacies, we will discuss a variety of disease-provoking transitions over prehistoric and historic time.

BIPEDALISM

Disease-provoking transitions both predate and postdate the palaeolithic. We begin with bipedalism, the transition that distinguishes hominids from other primates. Bipedalism evolved in the Pliocene over 4 million years ago (White *et al.* 1994; Wolpoff 1995), but the oldest clear-cut evidence is provided by footprints in 3.5-million-year-old volcanic tuff at Laetoli in northern Tanzania (Leakey and Hay 1979). Although bipedalism is of ancient origin, some features of the human skeleton remain poorly adapted to this locomotory style. For example, the lower back and knee joints are plagued by osteological malfunction, with lower back pain among the foremost causes of lost working days. These instabilities reflect the need for joint surfaces to carry weights for which they were never originally designed: the body's full weight is now borne on two legs rather than four. Physical design constraints have apparently made it difficult to modify the joints to the standards required to maintain full anatomical stability without rendering them so large as to impede locomotion. Vertebral arthritis is found even in prehistoric hunter–gatherers, and is not merely a pathology of modern lifestyles. The lower spine bears the most weight and is usually the most affected (Bridges 1994).

During the shift to bipedalism, the long narrow pelvis characteristic of the great apes was shortened and broadened, although the bowl shape characteristic of modern humans was not achieved until around 2 million years ago (Aiello and Dean 1990). This remodelling provided a stable base for support of the trunk and gut during bipedal locomotion. It also narrowed the birth canal through which the baby has to pass. Another major change in hominid evolution was a dramatic increase in brain size. Among the Australopithecines, the encephalization quotient (ratio of the species' actual brain size to that expected for a primate of that species' body mass) was 2.5, whereas in common chimpanzees (*Pan troglodytes*) the ratio is 2.0. The ratio grew to 3.1 in early *Homo* and to 5.8 in modern humans (Lewin 1993).

The passage of a fetus at birth depends on the size of the infant's head relative to the size of the maternal birth canal. Therefore, brain evolution was only possible in the human lineage through alteration of the typical primate

trajectory of brain:body growth. In other primates, brain mass increases less rapidly in relation to body mass from birth onwards (Martin 1989). In humans, however, the rapid brain growth characteristic of fetuses continues for about 12 months after birth. Specifically, from birth to the completion of brain growth, the brain increases in size by a factor of nearly 3.5 in humans, compared with a factor of only 2.3 in other primates (Martin 1989). Human brains are thus small at birth relative to adult size. None the less, the head size of human infants at birth is scarcely smaller than adult female pelvic diameter (Smith and Tompkins 1995). This is due not only to a narrow pelvis, but also to the fact that human neonates are heavier than other primates when maternal weight is controlled. In contrast with the situation in humans, the pelvic outlet in apes permits comparatively easy passage of the newborn infant. It is not surprising, therefore, that birth in humans is fraught with more obstetric complications than in other primates (Smith and Tompkins 1995). In summary, the shift to bipedalism coupled with large brain size contributed to medical problems ranging from back ache and knee trouble to obstetric and perinatal complications.

The significance of all perinatal complications relative to other health problems was assessed in the Global Burden of Disease Study. The purpose of this study was to derive a measure, called the disability-adjusted life year (DALY), that permits the severity of different conditions to be compared (Murray and Lopez 1996). DALYs are calculated from: (i) the number of years of life lost due to premature mortality, and (ii) the number of years of life lived with disability (adjusted for the severity of the disability). Thus, they reflect the mortality and morbidity associated with each health problem. In 1990, conditions arising during the perinatal period (low birth weight and birth asphyxia or birth trauma) ranked third among the leading causes of DALYs world-wide (Table 8.1).

Table 8.1 The leading causes of morbidity and mortality as measured by disability-adjusted life years (DALYs)

Disease or injury	1990 Rank	Percentage of total DALYs
Lower respiratory infections	1	8.2
Diarrhoeal diseases	2	7.2
Conditions arising during the perinatal period	3	6.7
Unipolar major depression	4	3.7
Ischaemic heart disease	5	3.4
Cerebrovascular disease	6	2.8
Tuberculosis	7	2.8
Measles	8	2.6
Road traffic accidents	9	2.5
Congenital anomalies	10	2.4
Malaria	11	2.3
Chronic obstructive pulmonary disease	12	2.1
Falls	13	1.9
Iron-deficiency anaemia	14	1.8
Protein–energy malnutrition	15	1.5
War	16	1.5
Self-inflicted injuries	17	1.4
Violence	19	1.3
HIV	28	0.8
Trachea, bronchus, and lung cancers	33	0.6

From Murray and Lopez (1996).

AGRICULTURE

We now take a long step from the origin of bipedalism to the advent of agriculture early in the Holocene. The health implications of this transition were examined at a symposium on palaeopathology at the origins of agriculture (Cohen and Armelagos 1984*a*). We briefly summarize the conclusions of this symposium, then discuss recent criticisms of these conclusions (Wood *et al.* 1992). Skeletal evidence suggested that Palaeolithic foragers experienced seasonal and periodic physiological stress, but not the severe or chronic stress found in Neolithic farming populations (Roosevelt 1984). During the Mesolithic, a transitional period of intensive hunting–gathering and incipient agriculture, the proportion of starch in the diet rose, producing various dental diseases, but dietary quality remained high (Cohen and Armelagos 1984*b*). With the intensification of agriculture in the neolithic, skeletal evidence suggested that malnutrition and disease had become widespread. Compared with the skulls of Palaeolithic foragers, those of Neolithic farmers had a high frequency of tooth enamel hypoplasias indicative of nutritional deficiency or childhood illness (e.g. measles) (Roberts and Manchester 1995). The Neolithic material also displayed a high incidence of porosities of the skull (porotic hyperostosis) and orbits (cribra orbitalia) associated with anaemia. Infectious disease lesions were reported to be much more numerous among farmers than among their hunting–gathering predecessors at the same locales (Cohen and Armelagos 1984*b*). Ten of 13 palaeodemographic sequences implied a decline in mean age at death with the adoption of agriculture and, in some cases, a downward trend in life expectancy for adults (Cohen and Armelagos 1984; Goodman *et al.* 1984*b*).

In their critique, Wood *et al.* (1992) argue that it is difficult to tell, for any locale, whether health got better or worse with the transition to agriculture. They point out that skeletal lesions (e.g. enamel hypoplasias, porotic hyperostosis, cribra orbitalis) occur only in individuals who were sufficiently healthy to survive the illness that caused the lesions. When the illness is fatal the skeleton is unaffected. Therefore, the higher frequencies of skeletal lesions in neolithic compared with Palaeolithic samples could reflect an enhanced ability to survive episodes of illness and stress. They might also be due to a decline in other, competing causes of death that leave no mark on the skeleton. For these reasons, Wood *et al.* suggest that improved health may lead to worse skeletons, not better ones as previously supposed. (To explore this idea, it would be helpful to compare the skeletons of elites and commoners in stratified societies.) The decline in mean age at death is also hard to interpret because it may reflect an increase in fertility as well as mortality (Milner *et al.* 1989; Wood *et al.* 1992). Until the relationship between the health of a living community and that of the skeletons it buries is better understood, it will be difficult to draw firm conclusions about the epidemiology of the Neolithic.

None the less, if we suppose that the spread of agriculture produced waves of disease and mortality (Cohen and Armelagos 1984), this might seem to support the caricature that there was a qualitative leap from adaptation to maladaptation from the Palaeolithic to the Neolithic (see Barkow *et al.* 1992). The alternative hypothesis, however, is that agriculture became an increasingly viable mode of subsistence as the returns from foraging diminished (Cohen 1977; Smith *et al.* 1984). The Pleistocene big game of Europe had vanished or was on the wane, and Palaeolithic population growth had spurred competition for wild food resources. In areas where wild grains could be efficiently cultivated, the shift to agriculture made more calories of food available (Flannery 1973).

In the Near East, agriculture originated about 9000 years ago. By 5000 years before present it had spread to almost all suitable areas of Europe (Ammerman and Cavalli-Sforza 1984; Sokal *et al.* 1991). The radial rate of advance averaged about 1 km per year (Menozzi *et al.* 1978). Using genetic evidence, it is possible to determine whether the mechanism of transmission was cultural diffusion,

with few genetic consequences, or demic diffusion, defined as the geographical expansion of a population whose size is increasing. The evidence supports the demic diffusion hypothesis. Agriculture spread because the farmers outreproduced the hunter–gatherers (Menozzi *et al.* 1978; Sokal *et al.* 1991). This view is reinforced by palaeodemographic data. Population growth had begun among the foragers, but was greatly accelerated among the farmers (Cohen and Armelagos 1984*b*).

The higher fitness of the agriculturists is at odds with the hypothesis that the transition from foraging to farming was such a dramatic cultural change that it rendered human behaviour maladaptive (e.g. Barkow *et al.* 1992). The mere fact that humans passed more generations as hunter–gatherers than as farmers does not establish the significance of the transition. The advent of agriculture was not the first change in mode of subsistence over human evolution, nor was it even the first change in which culture played an important role. It would be just as persuasive to argue that the behaviour of Upper Palaeolithic hunter–gatherers was out of joint with its selective background because most of the ancestors of these foragers were vegetarians. Technological change during the Upper Palaeolithic, as documented by the explosion in artefact types, was already monumental (e.g. Butzer 1977; Jurmain *et al.* 1997).

We do not dispute the lag periods that inevitably separate environmental change from adaptation, nor do we dispute the argument that when environmental change is particularly rapid, maladaptation will tend to be more pronounced. We do, however, question the tendency to view the Palaeolithic/Neolithic boundary as a definitive cut-off that separates the human lineage into: (i) inclusive fitness-maximizing hunter–gatherers, and (ii) maladapted cultural beings. A growing body of field and historical studies suggests that reproductive striving continues to translate into reproductive success in traditional, kin-based societies that have not undergone the demographic transition of the past century. The mode of subsistence is variable across these societies and does not appear to be a crucial parameter with respect to the adaptiveness of behaviour (see Alexander 1979, 1987; Irons 1979; Dickemann 1981; Hartung 1982; Borgerhoff-Mulder 1987, 1991; Chagnon 1988; Cronk 1991; Dunbar 1991; Smith 1992*a*, *b*; Dunbar *et al.* 1995; Betzig 1997).

Although the Palaeolithic/Neolithic boundary was probably not a watershed between adaptation and maladaptation, it did change the kinds of diseases affecting human population. Palaeolithic hunter–gatherers fought a limited range of infectious diseases because their small population sizes afforded limited opportunities for infection to pass from host to host (Cohen 1989). Among the most common afflictions of hunter–gatherer bands were zoonoses and chronic infections that survive for a long time in a single host and can therefore be reliably transmitted. Examples include bacterial infections (e.g. staphylococcus, streptococcus), various intestinal protozoans such as amoebas, and possibly the herpes virus (Cohen 1989). Many of the epidemics of recent history are caused by pathogens that survive only in large populations in which new hosts are continuously produced through birth and immigration. Measles, which may have originated as a canine or bovine virus, is a good example (Cohen 1989). The measles virus will die out at any one locale unless a fresh supply of new victims arrives as quickly as the old hosts are used up. Black (1975, 1980) has documented the disappearance of the measles virus from isolated island populations and makes a convincing case that it could not persist before the emergence of large population centres.

Protection from infection in small, isolated groups is also well illustrated by the plague epidemic in France in 1720–1722. Eighty-eight per cent of villages with fewer than 100 people were spared, but all cities with more than 10000 inhabitants were severely afflicted (Biraben 1968). The fates of towns of intermediate size were closely proportional to population size.

Although the agricultural revolution brought an upsurge in population, the increase in numbers was small compared with the acceleration

in population growth after the Industrial Revolution (Roberts and Manchester 1995). Limited population growth occurred even among prehistoric hunter–gatherers and may have been one impetus for the adoption of agriculture (Cohen and Armelagos 1984*b*; Roosevelt 1984). Thus, although population growth has been one of the most important factors impinging on human health, it has occurred at many times and places and was not unique to the Neolithic.

In addition to spurring population growth, the neolithic revolution caused people to settle down in villages. This change probably resulted in local accumulation of human wastes and the proliferation of diseases transmitted through the faecal–oral route. Although plumbing and sewerage systems now help substantially where available, diarrhoeal diseases continue to be a major public health problem. The Global Burden of Disease Study ranked diarrhoeal disease as the second leading cause of morbidity and mortality in 1990 (Murray and Lopez 1996).

Compared with foragers, sedentary populations tend to build substantial houses and spend more hours indoors in enclosed air spaces. The result is increased exposure to the many pathogens that do not survive in sunlight, such as the influenza virus. Permanent dwellings are prone to infestation by vermin such as rats and fleas (Cohen 1989; Dyer 1989). Urban yellow fever is hosted by a mosquito that breeds almost exclusively in water stored in human habitations (Johnson 1975).

Sedentism also increases the transmission of schistosomiasis, malaria, and other diseases that must somehow be passed from host to host despite dilution in water or air. After being excreted into fresh water by a human host, the fluke that causes schistosomiasis passes a developmental stage in a freshwater snail before it can again be transmitted. If the human population has moved away from the area, transmission is blocked. But if the population has stayed put, transmission is more likely (Cohen 1989). Malaria was favoured by sedentism because an infectious mosquito must bite two or more people within a short time-span for transmission to occur (Livingstone 1958, 1984).

Although sedentism promoted some diseases, such as malaria, studies of modern populations during the transition to sedentism usually report a reduction in mortality. For example, among the Ache of Paraguay, mortality rates on reservations over the past 15 years are demonstrably much lower than those among nomadic forest Ache prior to contact (Hill and Hurtado 1996). Roth (1985) reported lower mortality in sedentary than in nomadic groups in six populations, with no difference in two other populations. The health advantages of sedentism include enhanced ability to care for the diseased and infirm, and opportunities to stockpile surplus food in granaries. Sedentism also enables people to develop some immunity to the parasites of a given region, at least until new parasites are introduced from outside. Even today, traveller's diarrhoea is the unpleasant result of encounters with new pathogens for which prior immunity has not been established.

Subsequent to the agricultural revolution, novel means of transportation (shipping, railroads, and air travel) have been major forces in the spread of infectious diseases (Cohen 1989). Although the waves of disease have been numerous, three major transitions stand out: (i) the linking of China, India, and the Mediterranean by land and sea; (ii) the spread of the Mongol empire in the thirteenth century; and (iii) the advent of European exploration in the fifteenth century (McNeil 1976, 1980). The Old World epidemics of measles, malaria, and smallpox were probably introduced into the Americas around the time of Columbus (Merbs 1992). Bubonic plague had a limited geographical distribution in Asia until it was spread by human transportation (Davis *et al.* 1975; McNeil 1980). In the twentieth century, the rapid intercontinental spread of AIDS can be attributed to air travel.

MODERN AFFLUENT DIETS

Population growth, sedentism, and mobility have challenged human health through the

spread of infectious disease. Other changes have led to epidemics of chronic disease, including ischaemic heart disease, the killer ranked fifth in the Global Burden of Disease Study (Table 8.1). The chronic diseases are often called 'diseases of affluence' because they were uncommon among both hunter–gatherers and peasants. Diseases of affluence owe their origin and frequency to lack of exercise as well as to rich and unbalanced diets, especially diets that are low in fibre and high in fats, sweets, and red meat (Widdowson 1991). For example, studies of Australian Aboriginals have shown that populations following ancestral lifestyles are significantly less prone to chronic conditions such as obesity, hypertension, diabetes mellitus, coronary heart disease, and insulin resistance (O'Dea 1991). In contrast, westernized Aboriginals living settled urban lives often exhibit these lifestyle diseases at elevated frequencies compared with their European counterparts. Even a temporary reversion to a traditional lifestyle can dramatically reverse these effects (O'Dea 1984).

O'Dea (1991) has commented on the 'feast-and-famine' dietary patterns characteristic of hunter–gatherer Aboriginals, especially with respect to fatty foods. It is not unheard of for an Aboriginal to consume 2 kg of meat at a sitting, and such meat is likely to be taken from the fattest parts of the carcass. However, most of the wild-caught animals eaten by Aboriginals are leaner than domestic stock (especially those from intensive farming systems), and their fat contains significantly more polyunsaturated fats (principally those of the *n*-3 series) than that of modern farm breeds. When body fat reserves have been depleted by seasonal food stress, selection of fat-rich animals is actually healthier than a diet of lean meat, in part because lean meat requires more energy to digest (Speth 1987). Well-fed urbanized individuals who pursue a high fat diet are prone to diabetes and other chronic diseases (O'Dea 1991). The attractiveness of sweets and fats is particularly dangerous when combined with an equally natural inclination to minimize energy expenditure (an adaptive strategy when food is scarce). These two factors together are largely responsible for the rapidity with which Aboriginals succumb to diseases of affluence when they settle in urban environments.

An additional feature of the diets of hunter–gatherers is their sheer breadth (Flannery 1973). Hunter–gatherers commonly eat in excess of 100 different plant species, whereas people in most traditional agricultural societies typically only eat about 10 to 15. This in itself has been responsible for a wide range of dietary deficiencies and their associated medical conditions, including kwashiorkor, xerophthalmia, and conditions associated with low body mass (Widdowson 1991). Eaton *et al.* (1996) (see also Chapter 22) demonstrate numerous differences between putative Pleistocene hunter–gatherer diets (based on the diets of modern foragers) and modern affluent diets. For example, the ancestral diet was probably significantly lower in the fatty acids that raise serum cholesterol, but higher in protein from lean meat animals. It was also lower in sodium and higher in fibre. In particular, modern Americans may consume only one-fifth as much fibre as foraging peoples, and perhaps only one-tenth as much as free-living chimpanzees (Eaton *et al.* 1996).

Traditional agricultural populations lack the dietary breadth of hunter–gatherers but continue to eat a high fibre diet. They suffer a concomitantly lower incidence of appendicitis, diverticulosis coli, gallbladder disease, and tumours of the colon (Burkitt *et al.* 1972), all of which were uncommon in England until the end of the nineteenth century or later and may be associated with improved milling techniques. Central African populations eat a high fibre diet and remain relatively free of these digestive system diseases; stool weight in these populations is up to four times that of the British. Fibre acts to increase the rate of transit in the large intestine, and Burkitt *et al.* (1972) suggested that this helps to reduce the intestinal muscosa's exposure to passing oncogens. The highly processed diets of contemporary industrial societies allow more complete extraction of nutrients, thereby exacerbating problems of obesity, and reduce the rate of faecal passage, thereby increasing exposure to oncogens.

THE DEMOGRAPHIC TRANSITION

Eaton *et al.* (1994) have shown that the reproductive experiences of women in affluent Western nations differ greatly from those of contemporary forager women, whom they refer to as the best available surrogates for discerning reproductive patterns prior to the origin of agriculture. Compared with women in eight forager populations, they show that American women experience early menarche (12.8 versus 16.1 years), late first birth (26.0 versus 19.5 years), nursing of short intensity and duration (3 months versus 30 months), and low final parity (1.8 children versus 5.0). Epidemiological evidence has linked each of these changes to increased risk for reproductive cancers (Apter and Vihko 1982; Brinton *et al.* 1988; Layde *et al.* 1989). The primary mechanism underlying these changes is an increase in the proportion of the lifespan spent in menstrual cycling (Short 1976; Henderson *et al.* 1985, 1993). During menstrual cycling, the ovarian hormones stimulate cell division in breast and uterine epithelium, enhancing the risk for the DNA-copying errors responsible for neoplastic transformation (Henderson *et al.* 1993; Spicer and Pike 1993).

Eaton *et al.* conclude: '... the reproductive risk factors for women's cancers suggested by epidemiological investigations are largely, though not exclusively, a recapitulation of reproductive differences between women in affluent 20th century nations and women living before the advent of agriculture'. Thus, Eaton *et al.* imply that cultural changes dating all the way back to the origin of agriculture are relevant to reproductive cancers.

However, the reproductive patterns that Eaton *et al.* report for foragers continue to be widespread among traditional, agricultural populations in the Third World (Campbell and Wood 1989). For example, among the Dogon, who are millet farmers in Mali, West Africa, the median age at menarche is 16 years, first birth occurs at about age 19, postpartum amenorrhoea lasts a median of 20 months, and the total fertility rate is 8.6 live births (Strassmann 1992, 1997). Menstruation was monitored among Dogon women via a census of women's visits to menstrual huts and the results were corroborated by hormonal data from urine collections (Strassmann 1996*a*). In the study population, the median number of menses per lifetime was estimated at 109 (Strassmann 1997). This value is much lower than the approximately 400 menses experienced by Western women, but is not strikingly different from the estimate of 160 ovulations per lifetime among foragers (Eaton *et al.* 1994). The apparent difference may reflect the fact that the estimate for foragers was primarily based on information on nursing and interbirth interval length, rather than on data on menstruation or ovulation.

Both traditional agriculturists and contemporary foragers lack the reproductive parameters (early menarche, late first birth, bottle-feeding, and low parity) that are risk factors for reproductive cancers. They practise natural, rather than target, fertility, which means that reproduction is not controlled in a parity-dependent fashion (Henry 1961). The implication is that the demographic transition, not the origin of agriculture, played a major role in the aetiology of women's reproductive cancers. In industrialized nations, this transition occurred as recently as the past century. Adiposity may be another risk factor (Ziegler *et al.* 1996), but the evidence is less conclusive: for adiposity, we can thank the modern affluent diet.

AIR POLLUTION

Indoor air pollution may be as ancient as the control of fire by *Homo erectus* but continues to be a major cause of lower respiratory tract disease in developing countries. In the Global Burden of Disease Study, lower respiratory infections were the number one cause of morbidity and mortality (Table 8.1). Young infants who inhale wood smoke from cooking fires while being carried on their mothers' backs are at particularly high risk (Pio *et al.* 1985; Collings *et al.* 1990; Morris *et al.* 1990; Brauer *et al.* 1996). Outdoor air pollution is an environ

mental novelty that dates at least as far back as the prohibition on burning soft coal in England in 1273. Other early evidence includes a treatise on the problem of smoke published in 1684 (Shprentz 1996). At least as far back as the thirteenth century, English royalty escaped polluted air by extended visits to the countryside. The problem of low air quality is worth examining in some detail because it has become so pervasive.

The Harvard Six City Study (Dockery *et al.* 1993) is one of the most comprehensive recent studies of the health effects of air pollution. The researchers established air quality monitoring stations in six American cities and followed the health of 8000 adults over a 16-year period. Confounding factors such as age, sex, smoking, education, occupational exposures, and body-mass index were statistically controlled. After adjusting for these variables, the risk of mortality associated with exposure to fine particles was approximately 26 per cent higher for the residents of the most polluted city—Steubenville, Ohio—compared with the cleanest city—Portage, Wisconsin (odds ratio = 1.26; 95% confidence interval = 1.08–1.47). The researchers concluded that this increase in relative risk shortens life expectancy by 1 to 2 years for the Ohio residents compared with the Wisconsin residents. Many of the premature deaths due to air pollution appear to be caused by aggravation of cardiopulmonary disease (e.g. hypertension, ischaemic heart disease, atherosclerosis, bronchitis, and pneumonia). The risk of death from cardiopulmonary disease was approximately 37 per cent higher (odds ratio = 1.37; 95% confidence interval = 1.11–1.68) for the residents of the most polluted city compared with the least polluted city.

None the less, the risk of death from air pollution was not as high as the risk from smoking. After adjustment for confounding factors, the relative risk of mortality per annum among smokers was 2.0 compared with non-smokers. The risk of death from lung cancer was 8.4 times greater among smokers than non-smokers; the risk from cardiopulmonary disease was 2.3 times greater among smokers.

A study by the American Cancer Society (ACS) reached similar conclusions (Pope *et al.* 1995). This study linked monitoring data on fine particles in 50 American cities with the health of nearly 300 000 people who were prospectively studied from 1982 to 1989. Residents of the most polluted city had an approximately 17 per cent greater risk of mortality (odds ratio = 1.17; 95% confidence interval = 1.09–1.26) than residents of the cleanest city. The ACS study, like the Harvard Six Cities Study, concluded that modest air pollution exposures are shortening the lives of Americans by 1 to 2 years in the most polluted areas. The Natural Resources Defense Council (NRDC) (Shprentz 1996) extrapolated the more conservative findings of the ACS study to 239 American cities for which data on air quality were available. Assuming a linear relationship between particle concentration levels and risk of premature mortality, the NRDC estimates that 64 000 (range: 38 000–88 000) Americans die prematurely each year on account of particulate air pollution in the 239 urban areas.

The magnitude of the air pollution problem becomes evident when deaths due to air pollution are compared with deaths from other causes. In the 239 urban areas in 1989, a total of 29 000 deaths were caused by automobile accidents, less than half the mortality associated with air pollution (Shprentz 1996); 19 000 deaths were attributed to homicide. Deaths from breast cancer and AIDS for the 239 cities are not available, but nation-wide there were 16 000 deaths from AIDS and 43 000 deaths from breast cancer. Thus, if the NRDC is correct, more deaths are caused by particulate air pollution than breast cancer and AIDS combined. Mortality attributable to air pollution pales only in comparison with mortality from smoking: 419 000 American deaths in 1990 (Shprentz 1996).

LOSS OF KIN SUPPORT

In the Global Burden of Disease Study, unipolar major depression ranked fourth among health problems in 1990 (Table 8.1) and was pro-

jected to rise to second place by 2020. The projected increase does not reflect age-specific changes in depression, but rather an increase in the proportion of young adults in the population (Murray and Lopez 1996). Few studies have tackled the causes of this global epidemic of depression, but a contributing factor may be the breakdown of kin support networks and the attendant loss of psychological and material security. In traditional subsistence economies, the introduction of cash crops has been the major cause of the dissolution of extended families. Examples include coconut trees in Fiji and onion gardens in West Africa, both of which provoked competition among relatives and reduced the number of kin who work together in collaborative units. In the West, the breakdown of kin support networks can probably be traced to urbanization, occupational specialization, and mobility subsequent to the Industrial Revolution.

Evidence that kin support protects health and survivorship derives from several studies. One of the most powerful is a recent study of helping behaviour in the Israeli city of Haifa (Shavit *et al.* 1994). The study took place during the 1991 Gulf War, when Iraq launched 40 Scud missiles against coastal Israeli cities. The investigators were interested in how Israelis used their social networks during this mortal threat. The study design included two surveys of a stratified random sample of residents of Haifa, the first during the Gulf War and the other a year later. Respondents to both surveys indicated that they were more solicitous of kin than non-kin. For example, they were more likely to provide wartime shelter to kin than to friends. During and after missile attacks, they preferentially made phone calls to check up on kin to find out if assistance was needed. Less costly forms of aid, such as advice (e.g. how to seal a room against gas), were more likely to be exchanged between friends than kin (Shavit *et al.* 1994). Because the researchers were unaware of inclusive fitness theory (Hamilton 1964), their findings were not prejudiced by theoretical expectations.

The results of the Israeli study echo the finding that Americans tend to rely on non-kin for companionship but borrow money from kin (Fischer 1982). They also corroborate a growing body of evidence that help is given more freely to kin without corresponding demands, whereas with non-kin strict reciprocation is the rule (e.g. Dunbar *et al.* 1995). Although the Israeli study revealed the potential importance of kin support, a historical study of Plymouth Colony provided tentative evidence that kin support may actually translate into higher survivorship (McCullough and York Barton 1990). After the arrival of the Mayflower at Plymouth Rock in December, 1620, 52 per cent of the population died in the first winter from disease, malnutrition, and lack of preparedness. However, individuals with more relatives in the population had lower mortality. Similarly, an epidemiological study in Newcastle, England, demonstrated that kin support can reduce morbidity and mortality under chronic poverty (Spence 1954). On the Caribbean island of Dominica, children raised in household environments in which kin support was lacking were at risk for abnormal cortisol levels and a high frequency of illness (Flinn and England 1995).

These observations have important implications for the health and well-being of individuals living in modern large-scale polities. Although many individuals work hard to maintain their extended kinship networks (Dunbar and Spoor 1995), the physical distances that often separate family members make it difficult to do so. Where kin networks are not readily available to provide support, we should expect morbidity to be higher. Even the social support offered by a pet has been reported to improve survivorship in nursing homes. Measures that help to re-establish kinship networks, and their surrogates, may contribute positively to public health.

SUMMARY

In evolutionary medicine, environmental novelty has often been linked to the end of the Pleistocene 10 000 years ago. Modern health problems have been viewed as the outcome of

differences between current lifestyles and the behaviour of ancestral hunter–gatherers. The implicit assumption is that unhealthy lifestyles originated with agriculture. This view is vulnerable in two respects. First, the palaeodemographic data do not show, unequivocally, that health and survivorship actually deteriorated from the Palaeolithic to the Neolithic (Wood *et al.* 1992). Second, the transition to agriculture is just one of many candidates for disease-inducing transitions in the human past. In this chapter we emphasized both the shift to bipedalism, which goes all the way back to the Pliocene, and more recent changes, such as the demographic transition, air pollution, and the loss of kin networks.

Changes that promoted infectious disease include population growth and the expansion of transportation and trade networks. Although both occurred during the neolithic, the salient episodes of growth and expansion are considerably more recent. Epidemics of most chronic degenerative diseases (e.g. osteoporosis, diabetes, coronary heart disease, reproductive and digestive cancers) are associated with affluence and continue to be relatively uncommon in contemporary agrarian societies. Short life expectancy is the main difference: people die of other causes before the diseases of senescence set in. The lower incidence of degenerative disease in traditional agrarian societies is also due to lifestyle differences such as ample exercise, diets low in fat and high in fibre, and infrequent ovarian cycling (see Burkitt *et al.* 1972; Gray *et al.* 1979; Panterbrick 1993; Strassmann 1997). The problem of dental caries is found in both affluent and agrarian societies and is one of the few chronic health conditions that is firmly rooted in the transition to agriculture (Roosevelt 1984).

The Global Burden of Disease Study (Murray and Lopez 1996) permits comparison of the relative importance of different health problems. The five leading causes of death and disability in 1990, as estimated in this study, were as follows: (i) infection of the lower respiratory tract, (ii) diarrhoeal disease, (iii) perinatal problems, (iv) unipolar major depression, and (v) ischaemic heart disease. These afflictions have multiple causes that are considered in other chapters of this volume. Here we focused specifically on the role of environmental novelty. Rather than equate environmental novelty to the Holocene (the past 10 000 years), we tried to clarify some of the novel circumstances relevant to each health problem. This approach draws attention to immediate and correctable concerns such as smoking, air pollution, and traffic accidents.

The alternative view is that human biology is adapted to a specific period of our evolutionary past, the so-called 'environment of evolutionary adaptedness'. This period is usually matched to the upper palaeolithic (40 000 to 10 000 years ago) or to the entire Pleistocene (1.8 million years ago to 10 000 years ago). The major weakness of this concept is that it ignores the fact that human evolution has been mosaic in form: different components of our biology evolved at different stages. Moreover, it discards human evolution before and after two arbitrary cut-off points, the second of which corresponds to the rise of agriculture. Our analysis of the transition to agriculture uncovered no empirical evidence that it was a watershed between adaptation and maladaptation. If we were forced to make a simple dichotomy of this sort, the demographic transition of the past century would be a stronger candidate.

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