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Infectious Diseases in Ancient Populations

by T. Aidan Cockburn

Infectious diseases result from the interplay of three main factors: the host, the parasite, and the environment. The matter is highly complex, since each of these factors can vary in many ways and many differing diseases can result. In this review, attention will be concentrated on two of these factors: the primate and human hosts and their environments during the periods of man's evolution from the earliest days to the present. A number of distinct eras can be discerned: those of the primate precursors of man, early man, agricultural man, industrial man, and the man of one world. The first three of these eras will be discussed here.

THE PRECURSORS OF MAN

The Primates are assumed to have descended from an insectivorous mammal somewhat resembling the modern tree shrew. This animal is presumed to have had certain parasites and infections, and it is further presumed that some of these parasites and infections still exist, perhaps in somewhat modified forms, in its descendants of today. However, its descendants are now scattered over the world and live in many varying ecological niches (Cockburn 1963, 1967; Cameron 1956; Ruch 1959; Dunn 1966; Fiennes 1967). Some of the ancestral parasites and infections probably failed to survive in certain host genera or species, owing to the differing conditions under which their hosts lived. On the other hand, some host lines would acquire new parasites and infections after branching off from the main phylogenetic tree, and these would continue to exist only in their descendants. This process is depicted in Figure 1.

![Fig. 1. Evolution of Primates and their parasites (from Cockburn 1967).](image)

In a previous work (Cockburn 1963), I have suggested that this is the explanation of the distribution of many parasites and infections common to man and other primates. For example, no fewer than 13 of the intestinal protozoa of man are found also in apes and monkeys, according to the findings of Dobell (1926), Kessel (1928), and Hegner and Chu (1928, 1930). This is difficult to explain on any grounds except that these intestinal
protozoa were passed down to all these primates from a common ancestor.

Similarly, of 34 genera of parasitic helminths in the Hominioidea, man is known to be host to 20, Pan to 26, Pongo to 13, and Hylobates to 14. Seven genera, Trichuris, Strongyloides, Oesophagostomum, Ascaris, Dipetalonema, Dirofilaria, and Beritella, have been reported from all four primate genera (Dunn 1966). The distribution of helminths is apparently related to the phylogenetic relationships of the primate hosts.

Other parasites include lice of the genus Pediculus, malaria parasites, the scabies mite, Acuris, herpes virus, infectious hepatitis virus, and numerous other viruses now being discovered. The monkey equivalent (Herpes simiae) of the human herpes virus is now recognized as a common infection (Wood and Shimada 1954, Keble et al. 1958, Hull and Nash 1960). Infectious hepatitis virus has not yet been isolated with certainty, but the numerous instances of transmission of the disease from chimpanzees to their keepers leave little doubt that the infection is a natural one among chimpanzees (Hillis 1961, Communicable Disease Center 1965). Reviews of the literature on parasites of primates have been made by Ruch (1959), Dunn (1966), and Fiennes (1967). A summary of the viral infections is given by Andrewes (1964). Reviews of malaria parasites in monkeys and apes have been given recently by Coatney (1968), Eyles (1963), Bruce-Chwatt (1965), Bray (1963), and Garnham (1963).

Syphilis and other treponematoses (cf. Hudson 1963, 1965)—among them yaws, pinta, bejel, irkinga, etc.—may also have come down from a nonhuman primate ancestor (see Cockburn 1959, 1961a, 1963, 1967). The possibility that apes and monkeys share these infections has been argued for more than 50 years. Trappers have reported that apes in nature have a yaws-like disease (Raven 1950). A yaws-type lesion called groundou is not uncommon in monkeys (Marchoux and Mesnil 1911, Mouquet 1929, 1930, Bouffard 1909, Secques 1929), although some authorities, such as Ruch (1959), have denied that the condition has anything to do with yaws. Fribourg-Blanc and his colleagues (1965; also personal communication, 1967) tested large samples of sera from many species of ape and monkey and found many of the African, but not the Asian, ones to be serologically positive. This finding was confirmed by the U.S. Public Health Service Communicable Disease Center (personal communication), which tested 220 chimpanzees and found 10% of them positive. The French workers went on to recover treponemes from the popliteal glands of baboons. This is strong evidence in support of the theory that the treponemal infections have existed in man and his ancestors for many millions of years, possibly as far back as the Miocene. Although syphilis itself was “discovered” only when Columbus reached the New World, other diseases of the treponemal group would have existed before then on both sides of the Atlantic.

It seems generally accepted that our original ancestors were tree-living creatures. The primates of South America maintained a strictly arboreal life. On the other hand, many Old World primates, including the ancestors of man, learned to live either partially or entirely on the ground.

This basic difference in ecology must have had a marked effect on the infections to which the primates were exposed. A primate whose living is basically arboreal is exposed to arthropod vectors different from those on the ground. The most obvious of these is the type of mosquito which feeds and breeds in a canopy, unlike those feeding and breeding at ground level. Many more bloodsucking arthropods are found on the ground than in the treetops, the ticks and mites being notable examples. Furthermore, a ground-living primate is very much more exposed to parasitic infections from the feces of other animals. The water drunk by the monkey in a hole halfway up a tree is much more likely to be uncontaminated than that of the streams and lakes used by terrestrial primates. Wherever there are water snails in tropical Africa, there is always the hazard of Schistosoma infections; tree-living primates would not be faced with this hazard to anything approaching the same extent (Paoli 1965, Nelson 1960, Strong et al. 1961). Incidentally, the classic studies of Jane Goodall among the chimpanzees of East Africa have shown that these primates prefer to drink running water and avoid standing pools. This behavior could be important in reducing exposure to schistosomes. Mobility or lack of it can have substantial effects on the parasites and infections maintained in a “herd.” Washburn (1965:89) has said that if we look at the behavior of all the nonhuman primates, we find that these creatures are incredibly restricted in the area that they occupy. Only the gorilla and the baboon have ranges as great as fifteen square miles; while in the majority of the nonhuman primates, an animal spends virtually its entire life within two or three square miles—a tiny area.

Just when early man or his immediate ancestors adopted the roving way of life we do not know. However, it seems that by the time early man appeared in the Olduvai Gorge some 2,000,000 years ago, he was already mobile.

A static group of animals becomes infected over and over again with the same lines of parasites. Because the ground or trees on which they live are soon contaminated with their feces, there is an almost direct route from one intestine to another. There is little contact with other groups of animals, so that parasites are apt to develop high degrees of adaptation to their hosts. The mosquitoes and other arthropods that feed on these animals can breed nearby in the certainty of finding food with very little effort. This tends to produce strains of arthropods with preference for the particular host’s blood and, in turn, infection and reinfection of the host by the same strains of parasites.

In a mobile animal like man, these factors disappear, and highly specific parasites have very little chance of surviving unless they can travel along with the man. On the other hand, a mobile band of primates would be exposed to numerous new infections in areas into which it migrated, and some of these might establish themselves as permanent infections. Perhaps the schistosomal infections of the Far East arose in this fashion. Primates leaving Africa would free themselves automatically from the schistosomes of that continent, since the snails needed for continued transmission would be missing in their new homes. On arriving in the Far East, however, they would encounter a new form already established in many kinds of animals and would come to be included in its ecology. When man first evolved, he must have been a
comparatively rare creature, living in small bands of not more than 200–300 persons. His total population in the world was probably no greater than that of the chimpanzees today. It is obvious, therefore, that those specifically human infections which can live only by rapid transmission from one host to another and which do not form “carrier” associations could not have survived. This matter is discussed in more detail later; it will suffice to state here that it is improbable that infections like measles, smallpox, and mumps were present in those early days. However, one school of thought is of the opinion that very few viruses specific to man could have existed. This idea has been put forward most strongly by Burnet (1946:30–31), who states:

It is generally considered that in the early stages of human evolution primitive man and his subhuman progenitors existed in small wandering groups of at most a few families, and that these groups only rarely came into contact one with the other. Under such circumstances it would be virtually impossible for a pathogen to evolve as a specifically human parasite unless, as is the case with herpes simplex, the period over which a person remained capable of transferring infection was of the order of a generation.

To return to the question of the specifically human virus disease: we have given reasons for believing that in the early phase of human existence, from the beginning of the Pleistocene up to about 10,000 years ago, infectious disease due to microorganisms specifically adapted to the human species was almost nonexistent. The herpes virus could have persisted with very much its present type of activity, but the viruses producing brief infection with subsequent immunity—measles, mumps, and the like—could obviously not have survived in anything like their present form.

Much has happened in the field of primate virology since Burnet expressed his views. The adoption of tissue culture for the laboratory growth of viruses led in the 1950’s to the development of vaccines against diseases like poliomyelitis. This in turn resulted in large-scale commercial production of these vaccines, often using substantial amounts of monkey and ape tissues. These procedures activated latent viruses in the tissues. It soon became apparent that monkeys and apes are hosts to a great many viruses, the existence of which had previously gone unsuspected.

For many types of naturally occurring virus infections in humans, there are equivalent ones to be found in nonhuman primates. Except for those transmitted by arthropods, the differences between monkey viruses and human ones are slight; indeed, when a human infection like polio virus is found in a primate colony, the first conclusion is that it was acquired from some human carrier. Our knowledge of infections in the wild is very incomplete; reports of isolations from animals in captivity must be handled with the greatest of caution (Andrewes 1964, Guilloud 1963, Hahon 1961, Hull et al. 1958, Strode 1951, Arbovirus studies in Bush-Bush Forest, Trinidad 1968, Bhatt et al. 1966).

Since monkeys and apes have many apparently specific viral infections today in spite of their comparatively small numbers, the arguments of Burnet and his fellow thinkers become invalid. Therefore, emerging and early man may also have had many specific viral infections, perhaps as many as the monkeys and apes of today.

EARLY MAN

Two million years ago, man was a terrestrial creature probably no more numerous than the chimpanzees of today. He lived in small bands and ate whatever he could gather, kill, or find as carrion. Some of his infections were those handed down from his nonhuman primate ancestor and were probably much the same as those of the apes of his time living in the same environment. These infections have been outlined above. Among the other infections to which he was exposed would be parasites acquired by eating raw many kinds of insects, fish, birds, and mammals. He would also have been vulnerable to what are today called the zoonoses, infections of other animals transmitted to man by ticks, mites, mosquitoes, and other biting arthropods. Two zoonoses that probably occurred are anthrax and botulism.

Anthrax might have occurred in early man under conditions similar to those I witnessed when, in 1943, I was asked by the Colonial Government of the Gold Coast (Ghana) to help in an outbreak of disease in a village in the Northern Territories. On arrival, I found the village quarantined by Africans from the neighboring villages armed with spears. No one was being allowed to leave. The disease was anthrax. The villagers had killed and eaten a cow with a sore on its leg, having decided that the lesion was a snake bite and the cow therefore fit to eat. Thirty-seven people were very ill, and all who had eaten the flesh died from the disastrous intestinal form of the disease.

Endemic Type E botulism is not uncommon among Eskimos and Indians today and presumably occurred in similar fashion in times past. Dolman (1964) lists 18 outbreaks, evenly divided between Alaska and Labrador, between the years 1945 and 1962, in which 52 cases and 28 deaths were caused by eating marine animals.

The chief vehicle in the Alaskan outbreaks was muktuk, an Eskimo delicacy prepared by cutting the skin and underlying blubber or flippers of a beluga into chunks or strips and hanging them on a rack or over a pole outdoors for some days to dry. These pieces are then cured several weeks, or even months, in the comparative warmth of a hut. Mouthful-sized pieces of the matured muktuk are sliced off as needed. Two or three such pieces have been known to kill a hardy Eskimo male. Deaths from botulism have also been attributed to the utjak of the Eskimos of Labrador, prepared by letting seal flippers stand in oil, usually near a stove, for several days, until the skin falls loose and they are ripe for consumption.

Dolman also lists numerous outbreaks from another kind of botulogenic vehicle, variously called “salmon-egg cheese” and “stink eggs,” traditionally popular among the salmon-eating Indians of the Northwest Pacific Coast. This concoction, usually eaten raw, has been responsible since 1940 for 14 authenticated outbreaks of botulism involving 34 persons, of whom 19 died. During
the 23 years since the condition was first recognized there have no doubt been many other unreported outbreaks. Previously an indeterminate number of botulinic deaths and serious illnesses were presumably attributed to accidental or homicidal poisoning.

Tuberculosis in the early days of man was most probably a zoonotic infection. Man may have had a specific mycobacterial infection of his own, but this most likely was leprosy and was associated with his ancestors over vast periods of time. (If this reasoning is correct, then the apes of today probably also have a form of leprosy.) There are several forms of tuberculosis. The human variety of *Mycobacterium* is associated chiefly with pulmonary disease in urban populations long exposed to it and with an acute progressive glandular type in nonurban peoples such as Africans and Eskimos. The earliest evidence of human pulmonary tuberculosis comes from the writings of Hippocrates. The disease probably arose after the invention of agriculture (Cockburn 1963). The bovine *Mycobacterium* is responsible for most of the bone lesions in man. The skeletons, pictures, and clay pots giving evidences of spine disease and hunchbacks in ancient times must reflect infections acquired from cattle via infected milk or flesh. Tuberculosis of avian origin can cause human tuberculosis much like that of the specific human type, but does not spread rapidly from person to person. It is not uncommon in the southern states of the U.S.A. today. Fish tuberculosis can cause skin lesions in man. Several outbreaks associated with swimming pools have been reported.

Modern theory states that *Homo erectus* wandered from his place of origin in Africa to most parts of the Eurasian land mass suitable for occupation. Climatic and geographical conditions in his times were much different from those of today; for much of the time, large sections of the northern parts of the territory were covered with sheets of ice. On leaving Africa, man must have taken with him all those parasites that were transmitted directly person to person, leaving behind those that required vectors found only in Africa. These vectors would include those of filaria, schistosomiasis, trypanosomiasis, many arboviruses, mite- and tick-borne rickettsias and spirochaetes, and malaria. (Of course, we cannot say how the distribution of these vectors and intermediate hosts differed in those times from today.) Temperature would have been an important factor here. All vector-borne infections require certain temperatures for the extrinsic reproduction of the parasite. In the northern limits of the newly occupied territories it would have been too cold to permit the establishment of many infections.

On the other hand, *H. erectus* would have encountered many new infections in his new locations. The animals already established there would have had numerous forms of viruses, intestinal pathogens, and helminths that would be readily transmitted to him in a variety of ways.

The first men to arrive in Australia would have encountered a completely new fauna, the marsupials. The infections of these marsupials would have differed considerably from those of the animals of the Asian area, although unfortunately there is little data on this point. As a result, the zoonotic infections acquired through contact with the marsupials would have been kinds man had not previously experienced.

It would be the same story in the Americas, with the first migrants moving down from the north into a strange land filled with unusual animals. At that time, prehistoric forms of elephants and bison still roamed the prairies, and increasing evidence is being found to show that man hunted them. As man moved south, conditions changed. In the southern continent of the Americas he found an extremely wide range of habitats, with conditions ranging from the almost impassable rain forest of the Amazon to the high mountains of the Andes, the dry coastal plains of Chile, the wide pampas of Argentina and the cold and bleak coasts of Patagonia. In each of these, the infections of the animals would differ, and so of course would those acquired by man. The biting insects of the rain forest would transmit new forms of arboviruses, protozoa, and filaria; in drier regions man would acquire new kinds of leishmaniasis and, perhaps most serious of all, the American form of trypanosomiasis carried by *Triatomata*.

**AGRICULTURAL MAN**

Agriculture probably had a more significant effect on man than any other factor from his first appearance to the present time of scientific revolution. For the first time, not only was there plenty of food for all, but it was of a kind that could be stored for periods of shortage. Man lost his mobility and became tied to his land, and many animals moved into his ecological niche to be supported by him, willingly or otherwise. Population increased and spread, bringing long-separated human groups into contact. All of these changes, as well as the agricultural practices themselves, tended to increase certain infectious hazards.

**LOSS OF MOBILITY**

Man’s loss of mobility meant that various parasites could now establish themselves under conditions which permitted constant reinfection of the host. The hookworms and ascarids could maintain themselves in situations in which the host excreted the eggs and through reinfection became the unwilling host of the larvae that hatched from them. The stage was now set for the massive infections seen only too commonly in children in underdeveloped countries today. The feces of the family are either dropped indiscriminately around the home or in fixed places in the fields to which the members go daily. Either practice makes the continued transmission of the worms inevitable.

**ADDITION OF ANIMALS TO ECOLOGICAL NICHE**

The anchoring of man to his fields led many animals to move in beside him—not only those which he domesticated, such as the cow, pig, sheep, cat, dog, and goat, but many that were unwanted, such as the rat and mouse, English sparrow, tick, flea, and mosquito. So long as man was a wanderer, it would be rare for a mosquito to develop a preference for human blood; but now it was possible for mosquitoes to breed near man’s home or even inside it and be close at hand for a meal when this was required. In this fashion special strains of *Aedes*
Aegypti and Anopheles gambiae would be selected, and they would be excellent transmitters of infections such as yellow fever, dengue, and malaria. Later, man tamed a number of birds, such as the pigeon, jungle fowl, and duck and goose, and they also joined the community centered on man's habitation.

Each of these animals would be infected with its own collection of pathogens. The community living in which they participated would ensure that these pathogens would be spread readily among all its members, and that man would receive samples of them all. Among them would be all the intestinal bacteria, protozoa, helminths, viruses, and, as well, most of the parasites from other tracts of the body. Many of these would be unable to invade him successfully, but occasionally one would succeed. If this one could be transmitted from man to man, then the stage would be set for the evolution of a new specifically human pathogen.

Agricultural Practices

Agricultural practices by themselves do not create new infections, but some can accentuate those already present or convert what was previously an occasional event into a major hazard to health. The subject is a vast one that cannot be covered in this review. It includes the influences of draining swamps on malaria, of the slave trade in transmitting parasites like those of schistosomiasis and filaria from one region to another, of the slash-and-burn type of farming on malaria, of the cutting of undergrowth on trypanosomiasis (which it helps to suppress), and of many other interesting practices. Here, only two main agricultural practices will be discussed: the use of feces and urine as fertilizer, and various techniques of irrigation.

Spreading feces among crops can be a hazardous business. The possibilities of contamination with pathogenic organisms by those handling the material is obvious, but even more serious are the chances of transmission by the food plants fertilized in this way. There are many instances of typhoid epidemics originating in this fashion, and probably many helminths and cyst-forming bacteria are similarly spread. The eggs of Ascaris (roundworm) and the cysts of Entamoeba histolytica (amoebic dysentery) are especially suited for this form of transmission. People walking barefooted on ground so fertilized are particularly vulnerable to massive infection with hookworm. (The South Korean Government has just announced that it is taking action on the use of feces for fertilizer. About 75% of all feces in the country is used for this purpose; 90% of the population have intestinal parasites, the commonest being roundworms and whipworms in that order.)

The Chinese have carried the use of excreta to the extreme, employing it not only on the ground but also to support the growth of plants and fish in artificial ponds or canals. Since numerous snails are present in such places, this practice has caused the population to be heavily infected with a number of flukes. These flukes were present in the areas before the immigration of man, the hosts being various carnivorous and herbivorous animals, and the intermediate transmitters snails and fish, crustaceans, or plants. On arrival in the Orient, man became involved incidentally by eating raw the infected fish or crab or, in the case of the intestinal fluke Fasciolopsis buski, certain bulbs or fruits of edible water plants. The development of fish and water-plant cultivation, using feces as nutrient, converted what was an incidental infection into a major one. In all instances, the eggs of the flukes are excreted in the feces; when these are added to the water, miracidia hatch out and invade the snails present; after an interval, cercariae leave the snails and then encyst in various ways according to the species of fluke. Fasciolopsis buski encyst on almost any aquatic plant, although cattails, water hyacinths, water chestnuts, and bamboos usually serve as "transfer hosts." As many as 1,000 metacercariae have been found on a single water chestnut; if kept moist, most will live for a year. Since it is common practice to peel off the outer skin of water chestnuts with the teeth, infection takes place readily. The mature flukes develop in the intestine within a month.

Clonorchis sinensis, the liver fluke, has much the same cycle except that the cercariae penetrate into fish and the cysts they form are swallowed when the fish is eaten raw or undercooked. Drying, salting, or pickling the fish does not necessarily kill the parasites. Paragonimus, the lung fluke, differs from the others merely by using various freshwater crustaceans for the third stage in its life cycle. Infection occurs when these are eaten raw or partially cooked. The fluke develops in the human lung and the eggs are passed out in the sputum, which is often swallowed so that the eggs appear in the feces.

The numbers of persons in the Orient with these flukes are enormous (some populations are 50% infected); much of this must be blamed on using feces in the water farming of fish and water plants.

Irrigation must have been invented very early in the history of agriculture. As far back as recorded history goes, the civilizations of the Chinese, Egyptians, Sumerians, and Incas were based on the production of crops supported by irrigation. Unfortunately, the artificial use of water in this fashion tends to accentuate infections in the same way as does the use of feces, and here again, a fluke, as well as certain insects, is involved.

The liver fluke Schistosoma has a cycle of transmission involving snails, just as have the others mentioned above, but because the cercariae are capable of penetrating the unbroken human skin man is infected merely by wading or swimming in water containing them. The eggs are excreted in either the feces or the urine. The snails can live in vast numbers in irrigation canals and rivers; one of the world's greatest health problems today is the spread of these snails to new areas being opened up by the vast dam-building and irrigation projects of Asia and Africa. After malaria and tuberculosis, schistosomiasis is probably the greatest cause of morbidity in much of Asia and Africa and will become even more prominent after malaria disappears and as irrigation extends.

Insects readily take advantage of certain aspects of irrigation practices, especially where leaky canals and uneven fields form small ponds of water, or where large lakes are formed behind dams. Anopheles mosquitoes, which transmit malaria, can breed in either the small pools or the lakes, according to the species. Because of this, malaria became a major problem in the artificial
lakes of the Tennessee Valley Authority, and may have been a factor in the breakdown of the old civilization in Ceylon. Culex mosquitoes transmit filaria and viruses; they are often directly linked with irrigation. For example, Culex tarsalis in irrigated areas of the Missouri River Valley Authority has caused several epidemics of encephalitis.

**INCREASE IN POPULATION**

In previous works (Cockburn 1963, 1967) I have theorized that many infections require minimum host populations for permanent maintenance; if the sizes fall below the threshold levels, the infections die out. There are many diseases in which the infectious stages are brief and which exist entirely by rapid transmission of the agents from one host to another. Obviously, this can only happen if large numbers of susceptibles are present to support such a chain of transmission. These have been referred to as the acute community infections; they include rubella, cholera, smallpox, mumps, measles, and chickenpox. Such infections are specific to man and have no animal host other than man.

The effect of the introduction of an acute infection, measles, into a small isolated community was first studied in 1846 by Panum, who collected much valuable data about disease in a small “herd” on the Faroe Islands off the northern coast of Scotland (Panum 1940). In 1846, when the population was 7,782, measles was imported and attacked nearly everyone. A previous epidemic in 1781 had also infected almost the whole population, but the disease had disappeared completely afterward, presumably owing to a lack of susceptible persons. This also happened in 1846, for, after some 95% of the population had been attacked, the disease disappeared again.

Even pathogens that can live in their hosts like commensals for months find it difficult to survive if the population is too small. Bodian (1955) has said that poliomyelitis dies out in small communities after a certain time, even though carriers can excrete the virus for many months.

In a very small population with few susceptible persons, the survival of a pathogen depends on its ability to exist until new hosts appear. Natural selection will, therefore, favor those pathogens that can live in a kind of commensal relationship with their hosts and those that can continue to live away from their hosts. In a small population there would be no infections like measles, which spreads rapidly and immunizes a majority of the population in one epidemic, but many like typhoid, amoebic dysentery, pinta, trachoma, or leprosy, in which the host remains infective for long periods of time, and many like malaria, filaria and schistosomiasis, where the infection not only persists in the host for a long time but also has an outside vector or intermediate host to serve as an additional reservoir.

There is little in the way of precise and well-documented data on the infections of small groups of people living in isolation under the conditions of a hunting economy. It is difficult now to find such groups that have not already been infected through contact with larger civilizations. About three decades ago, what was known about the infections of the Australian Aborigines was written up by Cleland (1928) and Basedow (1932); more recently, Mann (1957) has published work on the eye infections of these people. As would be expected, most of the infections reported are of a chronic nature, such as trachoma, malaria, irkinja (a form of yaws), and roundworms. However, the influence of colonists and traders is obvious. An outbreak of smallpox, for instance, was reported within a year of the first British settlement. It was said to have crossed the continent from the north and possibly was started by Malay fishermen. Even trachoma may have been introduced by the white traders, as maintained by Mann (1957), although others think that Dampier, the first British explorer to reach Australia, was indeed describing the disease when he said that the natives had to throw their heads back to see straight ahead. Tuberculosis is apparently found predominantly only among those natives living in close association with white men.

In Africa, Jelliffe and his colleagues (1962) have studied the infections of the Hadza, a hunting people of northern Tanganyika, about 800 in number, who live an isolated life in the tsetse area of the savannah. The Hadza are very mobile, especially in wet weather. They eat almost anything they can get, including baboon, vulture, and hyena, but not tortoises. The food is usually barbecued. An examination of 62 children showed them to be well nourished and with good teeth. Malaria parasites were present in 27%. In the stools, four children had Taenia, probably from the wart hogs they ate, and three had Giardia. Conjunctivitis was found in 30%; many had ringworm. There was no roundworm or hookworm, presumably because constant moving prevented transmission. In other words, the only infection found was that which could survive in a small population always on the move. Other infections, such as measles, rubella, and chickenpox, come to the Hadza only as introduced infections from populations large enough to support them on a permanent basis.

Neel and his associates (1964, 1968) have reported on the infections of the Xavante Indians of Brazil. Tests for various zoonoses were positive. No evidence was found of tuberculosis or treponemal infections, but there were high percentages of positives for measles, poliomyelitis, whooping cough, and malaria. Unfortunately, these tribes are not completely isolated from the outside world. They had trade articles; these articles could reach them, so could infectious diseases. The only form of trade that cuts transmission to a minimum is that of “silent barter,” where the persons exchanging goods do not come into personal contact with each other. Even in that instance, smallpox can be passed on through infected blankets, as some tribes of North American Indians found to their sorrow.

The matter of population size leads us to the interesting question of the threshold sizes needed to support the acute community infections. If some 7,000 people on the Faroes were not enough to support measles indefinitely, would it have taken ten times, 100 times, or even 1,000 times that number of people?

In Greenland today, the population is about 30,000 people; until recently it was very isolated. Measles was unknown until 1951, when a sailor brought the infection from Denmark. As a result, practically every susceptible person in the area contracted the disease, and then it
disappeared. The same pattern was repeated in 1955, in 1959, and in 1962. The spread of the infection was greatly speeded by the introduction of air travel (Bech 1962, 1965). Presumably the population was too small to support the disease indefinitely.

It is obvious that in large urban populations, such as Greater New York City and Greater London, with their many millions of people, measles can continue to exist throughout the year without breaking the chain of transmission. The threshold size for measles must, therefore, be somewhere between the populations of the Faroe Islands and Greenland and those of Greater New York City and London.

The population of the city of Cincinnati is 503,000 people. If the suburbs and neighboring towns are included, there are a little over a million people living in Greater Cincinnati. In 1961, the City Health Department was invited to take part in a program to test measles vaccine organized by the United States Public Health Service Communicable Disease Center, and for the next two years a careful watch was kept on the measles situation in the city (Guinee et al. 1963). More than 1,000 children took part in an experiment in which one-half were given vaccine and one-half were given placebos. The surveillance programs showed that measles could not be detected for two approximately four-month periods in the latter parts of the years 1962 and 1964. During those periods, cases reported to the Health Department by physicians proved either not to be measles or to have been acquired outside the city. As far as could be determined, the findings support the concept that the chain of transmission during the four-month periods of 1962 and 1964 had, in fact, failed. It may be that in an American city like Cincinnati a population of about 1,000,000 is near the threshold required to support measles as an endemic infection (Cockburn 1967).

Populations of the size needed to support the acute community infections did not exist on earth until the agricultural revolution had progressed to a substantial degree. These infections could not have evolved earlier than 10,000 years ago. Even for some time after this, no single community had a population of a million people. In Iran, India, and China, however, there were civilizations with cities of 100,000–200,000 people, and if a half-dozen of these cities were linked closely enough by trade to permit the interchange of infections, the stage would be set for the acute community infections. That these infections did originate in the Old World seems quite clear. When intercontinental travel opened up the world to the interchange of infections, the natives of the newly discovered areas reacted very severely to the acute community infections of the newcomers, thus demonstrating that they had not experienced them before (Hirsch 1883, Cummins 1939, Stearn and Stearn 1945, Ashburn 1947).

In several instances the sources of the organisms causing these new diseases probably were the animals living in close relationship with man. For example, smallpox virus is very similar to a range of viruses found in domestic animals, the closest being cowpox virus (Hahon 1961); measles virus belongs to a group containing dog distemper and the cattle rindeppest viruses; influenza virus is very closely related to viruses found in domestic animals, particularly that of the hog (Andrews 1964, Meenan et al. 1962, Kilbourne 1968).

**Differential Resistance to Infection**

There are three main types of resistance to infection: active immunity, in which the body reacts specifically against the invader; passive immunity, in which antibodies are passed from the mother to the offspring via the placenta, milk, or egg; and genetically inherited nonspecific resistance. The existence of this latter kind of immunity, which will be discussed here, cannot be doubted although its mode of action is usually obscure.

Infection with a pathogen reduces the survival capacity of the host and, all other factors being equal, the host with the most resistance is the one most likely to survive. If this resistance is inherited, then natural selection can be expected to produce a population more and more resistant to the prevalent pathogens, and in time a benign host-pathogen relationship will be established. The earliest expression of this idea I can find is contained in a paper read by W. C. Wells (cited in Darwin 1906) before the Royal Society in 1813, entitled "An Account of a White Female, Part of Whose Skin Resembles That of a Negro." In this paper Wells distinctly recognized the principle of natural selection. He stated that of the accidental varieties of man which occurred among the first few and scattered inhabitants of the middle regions of Africa, one would have been better fitted than the others to withstand the diseases of the country. This race would consequently have multiplied; the others would have decreased, not only because of their inability to sustain the attacks of the disease, but also because of their incapacity to contend with their more vigorous neighbors (cf. Darwin 1858).

The existence of genetically controlled nonspecific resistance has been debated for more than 30 years, chiefly by two teams of workers, one in the United States led by Webster and another in England under Topley and Greenwood. Webster (1932) showed that mice varied in their resistance to infection with *Salmonella* species and that selective breeding would produce, from a strain of mice 40% susceptible to infection, strains that were either 10%, or 90% susceptible. In 1935, Greenwood and his colleagues summarized their results by saying that the available experimental data appeared to have established quite clearly the existence of significant differences in resistance among strains of mice or rats. Even in the most favorable records, however, there was no instance in which animals of the selected strain were uniformly resistant to bacterial infection, even when the test was resistance to infection by the bacteria concerned in a dose that failed to kill 100% of the unselected controls.

This deficiency was made up by Sabin in 1952, when he found a strain of mice 100% resistant to a dosage of yellow fever virus that was 100% lethal to standard test animals. By cross-mating on Mendelian lines, he was able to show that this resistance was inherited according to Mendel's laws.
These reports have been paralleled by similar findings for mouse typhoid (Gowan 1948, 1951), the rat cysticercus disease due to the cat tapeworm, *Taenia taeniaformis* (Curtis et al. 1933), and, at the cellular level, virus infections (Morgan 1960).

The natural selection pressures from infectious diseases can be very intense. Perhaps the best-known instance is the relationship between malaria and the abnormal hemoglobin that causes sickle cell anemia. The latter condition can be lethal, but the abnormal hemoglobin has the big advantage of protecting against the much more serious malaria. Natural selection in malariaous areas has apparently favored persons with the sickle cell trait (Allison 1954, Livingstone 1967). Yellow fever is frequently lethal to Europeans, but the African living in an endemic area is naturally immune (Strode 1951). Conversely, the European is little affected by measles, but populations not previously exposed react disastrously when the virus reaches them. The introduction of measles to America by the Spaniards caused many catastrophes among the Indians.

The intensity of the pressures can be illustrated by the example of smallpox. Until the end of the 18th century, practically everyone in Europe was infected sooner or later. In London about 1750, where half those born were dead before the age of three, there were nearly 20,000 cases of smallpox a year. Haygarth (1793) quotes French estimates that one in ten of all children born died of smallpox and cites Baron Dimsdale, Dr. Percival, and other authorities as stating that the proportion of births to deaths from smallpox in London was 6:1 to one, in Manchester 6:1 to one, in Liverpool 5:1 to one, and in Chester 6:1 to one.

The contrast between the reactions to smallpox of Europeans and American Indians became evident early in the colonization of the New World. Diaz (1956) wrote:

Let us return now to Narvaez and a Negro he brought with him who was full of smallpox, and a very black dose it was for New Spain, for it was because of him that the whole country was stricken, with a great many deaths. According to what the Indians said, they had never had such a disease, and as they did not understand it, they washed themselves very often, and because of that, a great number of them died, so that black as was the luck of Narvaez, still blacker was the death of so many people who were not Christians.

The Spaniards were not affected, for they already had had the disease in childhood. So common was it in Spain to suffer from smallpox as a child that Ruy Diaz de Isla remarks that he knew a man that did not have it until his 20th year.

Perhaps more significant was the experience in Massachusetts. When smallpox broke out in the Plymouth Plantation, many were ill but only 20 persons died, including both young and aged. Yet when the Indians of Connecticut were attacked by the disease, nearly all died. Although the English nursed the sick Indians, none caught the disease (Bradford 1928).

That infectious disease can cause severe damage in a population not previously exposed to it has often been documented. This has led many workers to wonder if some of the ancient civilizations might not have been destroyed in this way. Did malaria lead to the downfall of the Singhalene cultures of 2,000 years ago? Could some infection have been the factor that caused the abandonment of the cities of central Mexico, or the dwellings of Mesa Verde?

Undoubtedly, some local groups or tribes have been brought to near extinction by some pathogens. In addition to the instances involving smallpox just mentioned, one thinks of the reports that whole tribes of Eskimos died in Alaska during the influenza pandemic of 1918-19 and the estimate that worldwide the disease killed 20,000,000 people in that period. Again, sleeping sickness depopulated wide areas of Africa; malaria was so severe in such localities as the Pontine marshes near Rome and parts of northern Greece that only a handful of people could live there; and the Black Death is judged to have killed a quarter of the population of Europe.

One must conclude, nevertheless, that so far there is no good evidence that any single significant culture was ever wiped out by an infection. In theorizing on any particular culture in this fashion, one must ask what specific organism could have produced the disaster. As far as America is concerned, it is my opinion that the acute community infections, such as measles and smallpox, and possibly malaria and yellow fever as well, were introduced after the Spanish invasion. I cannot think of any infection present before that time in the Americas that could have caused morbidity or mortality of such a scale as to result in the breakdown of a civilization. It is a different story in other parts of the world; but even the Black Death or the epidemic in Athens described by Thucydides (MacArthur 1958) did not destroy the civilizations in question. The matter remains an interesting speculation, but nothing more.

**STUDIES OF THE EVIDENCE**

The importance of infectious disease in the control of populations demands that more attention be given to the study of the available evidence of infections in early man. This evidence is found primarily in fossil specimens, but ancient writings (Hoepli 1959, Guerra 1964), paintings, sculptures, poetry, pottery, and folklore are also valuable sources. Analogies can often be made between primitive societies existing today and those of the past; such analogies lead to a better understanding of the societies of early man. Cultural patterns, civilizations, trade routes, occupations, and population sizes are all of great importance. Geographic alterations, climatic fluctuation, and changing distributions of insect vectors must all be considered.

Paleopathology came of age in Egypt in the first decade of the 20th century. At that time, a new dam at Aswan threatened a number of sites of great importance in archaeology, so several investigators attempted to study the materials available before they were lost to science. Over 8,000 mummies were examined and much pathology found (Ruffer 1921, Ruffer and Smith 1910, Ruffer and Ferguson 1911, Smith and Wood Jones 1910, Morse, Brothwell, and Ucko 1964). It was found possible to reconstitute, to a surprising extent, the preserved soft tissue of the mummies and to demonstrate bacteria in sections of skin and various organs, such as the lungs and kidneys. The finds included a mummy with the calcified
eggs of *Schistosoma haematobium* in the kidney, one with a psosas abscess, another with poliomyelitis, and yet another with possible smallpox lesions.

Since then, a great deal of work has been done in the field of the study of ancient human remains for evidence of disease; for reviews of the literature, see Moodie (1923), Pales (1930), Sigerist (1951), Brothwell (1961), Goldstein (1963), Ackerknecht (1965), Jarcho (1966), and Brothwell and Sandison (1967). There are still, however, sharp limitations to investigations of human relics. Usually, only diseases that produce pathologies in bones can be recognized. This limits diagnoses of infectious diseases to those that produce abscesses in bones or deformities such as those resulting from leprosy, tuberculosis, and syphilis.

The soft tissues are of most importance in infectious diseases, and all relics with any tissues still attached demand special attention. The largest groups are still, of course, the Egyptian mummies; these deserve a fresh study using methods that were not available to Ruffer and his colleagues. Their pioneering attempts to reconstitute the soft tissues need reexamining, and better techniques should be devised. The reports that bacteria could be seen in sections should be followed up and attempts made to identify the bacteria. If chickenpox, warts, or molluscum contagiosum existed in those days the scars might be detectable on the skin. Guinea worm was almost certainly present (was it the fiery serpent that afflicted the Israelites in the desert?) and would leave a scar on the ankle. The eggs of lice might still be present on the hairs of the head and pubis. Containers in which intestines were preserved should be examined for parasite eggs or cysts.

A naturally mummified body of 4,000 years ago found in a cave in South America has provided the earliest record of head lice infestation (Ewing 1926); some of the hair was still attached, and on it were found head louse eggs. An even more valuable find was the frozen body of an Inca child found in a small stone building on a mountain near Santiago, Chile at an elevation of 17,658 ft. It was a remarkably well-preserved specimen, estimated as that of a boy eight-to-nine-years old who had died about 450 years ago. Contents of the rectum showed numerous eggs of *Trichurus trichiura* and some unidentified cysts of *Entamoeba* (Pizzi and Schenone 1955).

A very exciting find has been made in Russia (Artamonov 1965). In the Altai mountains of that country, many tombs of the Scythians, dating back to 400 B.C., have been excavated, revealing the frozen bodies of the people and their horses. Apparently, the Scythians dug deep holes in the ground for burials and then roofed the tombs with tree trunks. Since the ground was very cold, the water of condensation which fell on the bodies froze, encasing everything in the tomb in ice. Some of the bodies are in an excellent state of preservation and appear as though buried just yesterday. According to Artamonov (personal communication), no medical inspections of the bodies have yet been made, but obviously all kinds of skin lesions should be discernible, and possibly parasites and their eggs may still exist in the parts of alimentary tracts that remain.

The single most promising field at the moment is the study of ancient feces. Very large quantities are available already, and if special attention were given in the field much more would be forthcoming. The few studies of ancient feces already reported make it quite clear that parasite eggs in good condition can survive in recognizable forms for long periods of time.

Biddle (1967:58) gives this account of British studies:

The best work so far comes from Winchester, England. The first report of parasite eggs from archaeological excavations in Britain is that of Taylor (1955) who identified large numbers of eggs of the nematodes *Ascaris lumbricoides* and *Trichuris trichiura* and of the fluke *Dicrocoelium dendriticum* (Rud.) in soil from a medieval timber-lined cess-pit on a site at Middle Brook Street, Winchester. In 1964 Dr. H. H. Williams and Mr. A. W. Pike of the Commonwealth Bureau of Helminthology, alerted by Taylor’s publication, took further samples from a second pit in Lower Brook Street (Biddle 1965, pp. 245-46). Pike has since confirmed the presence of very large numbers of helminth eggs of the nematodes *Ascaris sp.*, *Trichuris sp.* and of the fluke *Dicrocoelium dendriticum* at average concentrations of 450, 2,300, and 216 per gramme respectively (Biddle and Pike 1966). All the eggs were in an excellent state of preservation and were easily recognizable, even the remains of the embryo within the egg being visible.

The possibility of eggs surviving in recognizable forms for perhaps thousands of years is suggested by a study on fecal material of about 3000 B.C. from Peru. The specimens consisted of coprolites that had been rapidly desiccated in the very dry atmosphere and material collected from the abdomen of a skeleton. The specimens were soaked in a 0.5% aqueous solution of sodium tripolyphosphate for 72 hours, after which the mixture was sedimented and the precipitate examined for food particles and parasite eggs. The procedure was so successful that remains of plant and sea foods could be readily recognized and sometimes even the smell was recreated. The eggs of a species of *Diphyllobothrium*, a fish tapeworm common in carnivores in South America today, were identified (Callen and Cameron 1960).

Samuels (1965) examined feces from the Wetherill Mesa cliff dwellings. In one specimen he discovered eggs of the pinworm *Enterobius vermicularis* in which the larvae inside were still clearly visible. Numerous microorganisms were seen but not identified.

The statistical analysis of adequate samples of relics could be a powerful tool in studies of diseases of ancient populations. As long ago as 1910, Smith and Wood Jones showed that the distribution of fractures in Nubian skeletons differed from that of a modern population. More recently, Brothwell (1961) has shown a probable difference in the susceptibility to osteoarthritis in modern and ancient British populations. The time has come when it is no longer enough for a fieldworker to select a few bones with obvious pathologies and ignore the remainder. Fortunately, several workers have undertaken the tedious procedure of examining all the material of a find with significant results. According to Roney (1959), Hooton’s (1930) work on the Indians of Pecos Pueblo is an early example of the statistical study of evidences of disease in ancient populations. Roney also cites the work of Krogman (1940), Angel (1946), Goldstein (1957), Vallois (1937), and Todd (1927). More recent works
along these lines are those of Angel (1967), Roney (1959) on a California archaeological site, Warwick (1964) at a Roman cemetery in York, England, and Anderson (1963) in an Iroquois ossuary. In all of these studies, there is emphasis upon studying a population sample, upon relating disease to age, sex, and race, upon observing temporal trends in disease, and upon relating disease to cultural factors. This approach is, therefore, epidemiological, concerning itself with host and environmental factors in diseases occurring in a population sample.

SUMMARY

1. Most of man’s specific infections are descended from those acquired from his prehuman ancestors. The apes and monkeys of today received infections from the same source.

2. Every change in the environment or culture is reflected in the patterns of the infectious diseases of the population. In particular, the invention of agriculture and the domestication of animals had a profound effect on the evolution of new infections and increased hazards from some previously existing ones.

3. Natural selection results in a population increasingly resistant to a pathogen to which it is exposed; and a population exposed to a pathogen to which it has not developed resistance may be devastated by it.

4. Since infectious disease is one of the most important factors controlling populations, more attention should be given to the study of the available evidence of infections of early man. The soft tissues that are found in a state of preservation are of particular importance, and special care should be taken of them. Feces are also an important and promising source of information. A statistical approach to the evidence of disease in ancient populations can be a powerful research tool.

Comments

by Kenneth A. Bennett

Eugene, Ore., U.S.A. 7 n 70

A close examination of Cockburn’s “References Cited” section indicates that roughly half of the entries were published within the last decade. This could mean a number of different things, one of which undoubtedly reflects the current popularity of paleopathological studies. The lack of material evidence, however, or of any kind of rigorous methodology has not, so far as I have been able to determine, deterred either physical anthropologists or physicians from publishing their conclusions. Often these conclusions are derived by guesswork and as such are neither facts nor even working hypotheses. In this sense, it seems to me of doubtful value to speculate on the existence of anthrax or botulism in early man when we have absolutely no evidence and little hope of ever getting any. As an aside, it may be indicative of some degree of confusion in paleopathological studies to point out that Sterne and Van Heyningen (1958 : 359) begin their discussion of botulism by stating “Botulism is not an infectious disease . . .”

It is difficult to either agree or disagree with the majority of Cockburn’s conclusions, as for the most part they have little basis in fact. It may be, for example, that specific human infections have descended from nonhuman primate ancestors, but the mere observation that we presently share a number of intestinal protozoa with apes and monkeys is hardly the evidential support needed for the first statement in the “Summary.” It may also be that man “adopted the roving way of life” sometime prior to the advent of agriculture, but I suspect that the basis for this argument has all too often rested on the false assumption that mobility is synonymous with adaptive radiation. As long as there is no evidence to influence us one way or the other, it appears to me that one could argue equally as well that this very mobility, even in groups numbering 200 to 300, could have been responsible for the maintenance of some acute community infections.

My main criticism of this paper, if indeed it can be termed a criticism, is that not enough attention has been given to the potential importance of the phenomenon that Pimentel (1961) calls “genetic feedback.” Cockburn does state in the “Summary” that disease is an important controlling factor in human populations, but there is only a vague comment or two on how it functions as one of these factors. To understand disease and its effects on the density of early human populations, we must know as much about the pathogen as we do about the deleterious results it may induce.

One of the better examples of a parasite-host system that has been investigated in depth and illustrates the principles of genetic feedback is the well-known relationship between the myxomatosis virus and the wild European rabbit in Australia. The virus, introduced by man into the wild rabbit population in an attempt to control their numbers, caused in the first epizootic a mortality rate of 97 to 99% (Fenner 1953). Not all cases were fatal, however, and in succeeding epizootics the mortality rate underwent a sharp decline (85 to 95% in the second, and 40 to 60% in the third). Part of the explanation for this decrease may be found in the fact that natural selection favors those rabbits with increased resistance, but an equally important reason involves selection for comparatively nonvirulent strains of the myxomatosis virus. For obvious reasons, the selective premium will not be on the parasite or pathogen that quickly kills his host, but instead on the one that can live within his host without altering appreciably the reproductive potential of the latter. This results in a situation whereby the density of both populations may be controlled by an alternate functioning of the feedback of selection and genetic change (Pimentel 1968).

It has been demonstrated that a number of human diseases (e.g., smallpox and bubonic plague) have, through repeated infections in different generations, lost some of their virulence in certain populations. To claim now that this has been due to genetic feedback would be unfortunately premature, but research in this area promises to shed additional light on the effects of disease in the human ecosystem.

by D. K. Bhattacharyya

Delhi, India. 311 70

Cockburn’s essay was entirely instructive for me, especially because in India studies of this kind have so far not been ventured. The promising results already obtained through coprolite analysis elsewhere make it appear how much significant material has been lost from Indian sites. The suddenness of the decline of the Indus Valley complex has led many to believe that some such disaster as an earthquake, flood, mass massacre, or famine may have been the reason for that decline, but we have no
conclusive proof for any of these possible causes. An analysis of the coprolites from these sites could definitely show whether an epidemic was an added factor, perhaps acting in combination with others. After all, with the amount of trade evidenced for this civilization, the importation of a deadly parasite to which the community was not resistant is not difficult to visualize. The Neolithic Chalcolithic ash mounds in Andhra and other burials found in Adittanalur, T. Narisipur, Maski, Chandoli, Nevassa, Tekkalkota, and Langhraj also had a strong potentiality for yielding paleopathological information for this subcontinent. Unfortunately, nothing towards this end has so far been attempted. The need for such study becomes the more evident if we look into the finds of parasitic helminths and protozoa in contemporary hunting and gathering people (Dunn 1968). That there is an increase in the complexity of the ecosystem and a conscious influence of the number of species of parasites found in a population in the tropical high-rainfall area has already been demonstrated. This discovery makes it seem probable that various kinds of infections will be found among the early Holocene populations of India (Pleistocene man being as yet unknown in this country).

To Cockburn's theorization as to the possible descent of many specific infections of man from his prehuman ancestors, there should be, I think, no particular objection. Here only a note of warning needs to be added. The evolution of man definitely does not occur in a simple unilinear succession, as would appear from the author's Figure I. Further, up to this time it has not been conclusively demonstrated that such an evolution took place at one and only one place in the world, though there is a strong suggestion that Africa may have had that honour. A consideration of all the possibilities will lend us in a situation of extreme complexity and diversity of ecosystems. Further, these numerous stages of subhuman types developed different social habits regarding migration and habitat. In other words, the ancestral parasitic type need not give rise to the same parasitic type in both present-day man and apes, as the author himself points out in referring to the development of different strains of arthropods with a preference for the same kind of host. Thus, it would be more apt to assume that parasite gives rise to parasites in present-day apes and in present-day man, where is more similar to a new species. The slight chance in can take care of the multiple significant factors which took part in the evolution of the human form, with fission and fusion at various periods of its developmental past.

by BRUNETTO CHIARELLI:

Torino, Italy. 30 70

Cockburn's paper is the most complete one I know of on this subject. Of particular interest is the author's application of the comparative approach to the problem of infectious diseases in ancient populations, including the infectability of different species of primates. This point of view, occasionally found in recent papers, provides the field with a new and broader perspective. The problems of the infectability of existing small human populations and the relationship between the population and the epidemiological environment are very interesting and clearly exhibited. Therefore, I consider this paper an important stepping stone in the study of the evolution of human populations, and a useful document to be considered in every attempt at prehistoric reconstruction.

I should like to add a few remarks and suggest that some arguments be clarified and further developed.

Among the numerous and proper references related to the parasitism of primates and man, I would suggest the inclusion of the papers of Kuhn (1968).

In the text that refers to Figure 1, I would like to see the differences between Hylobates and Pongo pointed out. Such differences with regard to parasitism seem to be yet another contribution to the recent evidence tending nearly to separate the Hylobatidae from the Hominoidea (Von Koenigswald 1967, 1968; Ankel 1965; Chiarelli 1968).

With reference to hereditary resistance to infections, I think that it would have been useful and important to stress the recent knowledge on the resistance to malaria due not only to the haemoglobinopathies, but also to variations of the red cell enzymes. Such variations are typical not only of man, but also of various species of primates that lived in the same environment as man. Of special interest is the work of Crawford, Morrow, and Motulsky (1967), who have found that the chimpanzee and the gorilla, both parasitized by Plasmodium falciparum riechenowi, also maintain a polymorphism at the G6PD locus, indistinguishable from the sex-linked A and B electrophoretic phenotypes in African populations. Inhabiting similar environments (equatorial Africa) and facing with the same disease vector (Plasmodium falciparum), man and the two anthropoid apes may have solved this high-mortality problem similarly. Although there is no G6PD deficiency in any primate species tested to date (Crawford et al. 1967), this polymorphism may be maintained through selection operating upon the differential enzyme levels, with type A being 15–20% less active than type B (Pik et al. 1960).

For further paleoanthropological and paleoepidemiological data on the ancient Egyptian populations, I suggest that Cockburn get in contact with Dr. Merton Sutinoff (Gibson Laboratories, Radcliffe Infirmary, Oxford, England) and Dr. Emma Rabino Massa (Istituto di Antropologia, via Accademia Albertina 17, Torino, Italy), who are doing research in this field.

by MARIE STRIEGEL CLABEAUX:

Bronx, N.Y., U.S.A. 9 70

Cockburn presents a cogent explanation of the origins and development of infectious diseases in human populations. Their history is reconstructed in a manner most logical and consistent with the available information on both human evolution and human and nonhuman primate disease. Such an undertaking must necessarily be limited, since it is not possible to determine the nature of all those pathologic conditions which have affected man. One could question the utilization of material derived from living populations to extrapolate the presence of the same diseases in the remote past, since pathogens and the manifestations of their activities in the body do change in time. However, since there are diseases common to all of the higher primates (indicative, as the author notes, of transmission from a common ancestor) and since at this time there are virtually no other avenues of approach to the problem, this must be considered a valid methodology.

The field of paleopathology could play a more vital role in the endeavor to trace the history of disease if its practitioners were more population-oriented. The description of a few spectacularly pathological specimens in a collection that may include hundreds of individuals is of little use unless that description is accompanied by complete demographic data. The simple tabulation of deaths by age, sex, and, where possible, relative time of occurrence may provide clues to events such as epidemics, severe childhood diseases, etc. and may also aid in interpreting cultural material. The contribution that epidemiologic paleopathology can make cannot be too strongly emphasized.

Those who work with skeletal populations should pay special heed to Cockburn's concluding remarks. The statistically oriented epidemiologic approach which combines a total health profile
(insofar as it can be reconstructed) with data on the cultural and physical environment should be the aim of the worker in this field. For the anthropological paleopathologist, the relationship between culture and disease is one of the most important aspects of the study of disease in ancient human populations. Much useful data is ignored in the majority of paleopathologic studies. I hope that this situation will soon be corrected. Cockburn’s work in relating cultural practices and diseases in modern populations should serve as an example of how illuminating such a treatment can be.

by W. C. Osman Hill

Dublin, Ireland. 12:70

Cockburn’s title is somewhat misleading. Though ancient populations are comprehensively considered, a large part of the paper, especially that dealing with “agricultural man,” concerns itself with recent practices (e.g., in China). While these may have a distinct bearing upon the synthesis of the evolution of infectious diseases, some indication of this in the title would have been informative.

Epidemiologically, the division into precursors of man, early man, agricultural man, and industrial man is amply justified; these divisions mark the principal cultural phases of man’s progress as a corollary to his environmental relationships (including, more specifically, host–parasite relationships).

“Agicultural man” is but another expression of Child’s Neolithic revolution, which indubitably created a fundamental alteration between man and environment. Its effect is rightly emphasized by Cockburn. Combined with loss of mobility was the problem of increasing concentration of population and consequent greater ease of parasite transmission. Maybe it was at this stage that the virus of the common cold was evolved, though Cockburn makes no allusion to this all too specific human pathogen. No other primate suffers naturally from this disease, though immunologic resemblance among the Hominioidea renders the condition artificially transmissible to the Pongidiae, whose nurses succumb.

Under the heading “Addition of Animals to Ecological Niche” (p. 48), the author is rather vague on the order in which animals were domesticated. No mention is made of tetanus—surely a hazard introduced when man tamed the horse and came to use its manure agriculturally. This was a relatively late event.

Although the author devotes a page and a half to the problem of increase in population, nowhere does he specifically deal with that important phase of cultural history, the establishment of cities and city-states, which began in Mesopotamia in the 7th millennium B.C. The Sumerians, for example, left evidence of their medical knowledge, and so, later, did the Egyptians.

Rivalry between city-states and raids by their uncivilized neighbors inevitably led to mass population movements—hordes of refugees from war zones—with the consequent introduction and dissemination of pathogens hitherto foreign to a given area. In later times, with major local wars, e.g., during and after the Peloponnesian war, agriculture was neglected and deforestation occurred, thereby hastening soil erosion and breakdown of drainage. This opened, during the 1st millennium B.C., the opportunity for the spread of disease, particularly malaria, in the Greek coastal lowlands (Darlington 1690:210). Later growth in size of cities, with subsequent environmental pollution and the evolution of slum conditions, are a logical sequel, leading finally to the conurbations of the 20th century with their built-in reserves of pathogens.

Passing now to the Bronze Age and Early Iron Age, with the emergence of seafaring peoples like the Phoenicians, still greater dissemination must have resulted, especially in the distribution of vectors such as Rattus rattus and its ectoparasites. Cockburn has little or nothing to say of them, yet the Phoenicians travelled very widely—as far as West Africa, for example (Hanno’s voyage), whence tropical diseases were probably introduced to the Mediterranean world, and perhaps even as far as the New World (Boland 1963).

Another serious omission germane to the subject of the title concerns the epidemiological knowledge of the Hebrews gained from the record of their sojourn in Egypt and subsequent experiences during the migration to Palestine. There is a wealth of evidence here on the relation of man and his parasites to the environment. What, in fact, were the “plagues of Egypt”? Some documentation of the hygienic rules elaborated by Moses seems called for, as they include regulations both as regards personal cleanliness and dietetics.

Early Man, a result of a tripartite monograph on disease, is a book of “uncleanliness of issues” in the prophylaxis of venereal disease (Leviticus 15; Darlington 1969) and the prohibition of the flesh of the pig, probably on account of the prevalence of trichinosis infection. Nevertheless, Moses’ zoological knowledge was not always accurate, witness the classification of the leporids as ruminants—or was this an inspired guess at the phenomenon of reflection?

The later sufferings of Job also require explanation. Were the nocturnal aching of his bones the result of a Freund’s phenomenon or due to gonococcal arthritis?

Cockburn’s emphasis on the evidence of paleoecropology is commendable and should be eagerly followed up by archaeologists.

by F. P. Lisowski

Hong Kong. 30:70

The article presented by Cockburn is another contribution to an important aspect of the study of ancient populations that is now being re-examined in the light of new knowledge and better methods of laboratory investigation (cf. Wells 1964, Jarcho 1966, and Brothwell and Sandison 1967, to cite just a few titles). The only “criticism” I have of it is that I wish it were longer and more detailed. What we have is something that stimulates the palate for more. Cockburn’s background is ideal for such a compilation, and one hopes he will furnish us with further studies.

I like the way in which he subdivides his subject into three phases—the precursors of man, early man, and agricultural man—and then argues his various points both by reviewing the literature and from personal observation in the field. Apart from examining ancient remains by modern methods, it is important to study some of the populations in the developing parts of the world that have had minimal contact with the life and medical care that obtains in the developed part. It is by studying these that we may arrive at an approximate answer as to what happened in the past and possibly how ancient man dealt with his ills. Thus I should like to add a few points, by way of comment, as a result of a lengthy stay in Ethiopia a few years ago.

During that time there occurred quite a serious outbreak of yellow fever in the southern part of the country, an area that succumbs from time to time to these attacks. It is considered that the reservoir for this particular virus is the nonhuman primate population in that region (P. Neri, personal communication, 1968). This is very much in line with what Cockburn says in his section about the precursors of man and the transmission of certain infectious diseases.

Similar to early historic sedentary man, the Ethiopian population of today is subject to a large variety of infectious diseases. This fact was quite clear from my examination of 12 human cadavers at the Institute of Medical Sciences in Addis Ababa, as well as from some 14 postmortem examinations in the field; in all these specimens, most of the lymph nodes were enlarged, and particularly so in the abdomen. Furthermore, in all of the adult specimens of 25 to 50 years of age the thymus gland was not only present but enlarged (on the average
populations is multifaceted and could be discussed from many viewpoints. Cockburn has chosen well in the aspects covered and has exercised care in his conclusions, though not everyone will agree with all of his interpretations.

How far one can generalize from the primates to early man is certainly debatable. With the differences in mobility and in dietary habits, it may be that food-related or food-carried infections of early man were more closely related to certain of the carnivores than to the primates (Schaller and Lowther 1969).

The effects of irrigation and formation of artificial bodies of water on human disease cannot be overemphasized. Van der Schalie (1960) states that schistosomiasis has now replaced malaria as a chief hindrance to progress in many underdeveloped regions of the world.

Because of the physical debility this disease produces in its victims all the heavy labor of Egypt is supplied from Upper Egypt, where the incidence of schistosomiasis is low. The Aswan Dam will open Upper Egypt to schistosomiasis and may well cancel out the benefits from the dam construction. Prior to the relocation of Tonga villages in connection with the Kariba Dam project in Central Africa, village mosquito populations could be controlled through spraying. This is no longer possible for villages and fish camps sited close to a lake of 2,000 sq. mi. surface area. With this large, permanent breeding area, the villages are now heavily infested with mosquitoes (Thayer Scudder, personal communication, 1970). Though no study for malaria has been done under the new conditions, an increased incidence is likely. The water projects are also likely to alter disease patterns by increasing local population density.

Cockburn discusses minimum population size for maintaining infections. This should be open to statistical study. Theoretical conclusions based on studies of duration of infectability could be checked both historically, from compilations of previous epidemics, and by using partially immunized groups as natural laboratories.

The possible findings from ancient feces draw attention to a chronic problem. It is difficult to communicate to fieldworkers (and others) the potentialities for information in materials handled by new techniques. The importance, when excavating, of saving samples of materials outside the worker’s own interest must be more widely disseminated. There is also delayed transmission of methods from other fields which might be applicable. For example, Courville’s excellent detailed studies of cranial injuries in early populations in Oceania and America are little known in anthropology, having been published in relatively obscure medical journals (Courville 1948, 1951, 1952, 1956; Courville and Abbott 1942).

by CALVIN WELLS

Norfolk, England. 26 j 70

What diseases infected ancient human and subhuman populations? Cockburn’s latest attempt to infer a few answers attracts by its persuasive shrewdness, no less than it wows by clear presentation. He reviews, most usefully, current reasonable assumptions. I concur with his general opinions and wish to comment on only a few details.

It is almost certainly true that some of the ancestral parasites and infections ... failed to survive in certain host genera and species.

Unfortunately palaeopathological remains, whilst offering some suggestive hints, seem nowhere to give conclusive proof that this happened. We probably need further developments in palaeo-scopy before final proof is forthcoming.

Cockburn suggests that, on quitting Africa, man took with him those parasites which were transmitted from person to person, leaving behind those which needed vectors found only in Africa. Although probably true, this principle was perhaps modified by the ability of parasites to adapt to other vectors when their hosts infiltrated a new environment. No doubt most ancient migrations proceeded slowly, during which time many generations of parasites churned through the mill of natural selection. The more tolerant species or strains probably changed vectors in midstream with ease, if not with eagerness.

Parasites evolve in conjunction with their hosts and vectors, but some will be more versatile than others. Would anyone care to predict what extension of behaviour might be shown, under evolutionary pressures, by the organisms of Well’s disease, brucellosis, warts, or botulism? What is the truth about sudanoglossus?

Cockburn refers to goundou in primates. He is right to be noncommittal about this interesting lesion: it is probably premature to ascribe it to yaws or any treponemal infection.

Reading Cockburn’s paper, I found myself asking, “When is a disease not a disease?” Is poliomielitis always pathological in humans? Or is it rather a blessed symbiosis which only at times
flares beyond the bounds of normality? Is it or is it not normal to accommodate Bacillus coli in our tripe?

Even diseases which are clinically well defined—tuberculosis, leprosy, actinomycosis—are enigmatic enough in ancient skeletons, despite obtrusive bone lesions. How much more elusive are soft-tissue, nonspecific bacterial invasions, to say nothing of viral infections! Until palaeoepidemiology takes a long haul forward we shall probably continue to fumble amid diagnostic uncertainty with most such lesions.

Cockburn rightly emphasizes the significance of helminth eggs, Entamoeba cysts, and the like. If these are objectively identified, presumption of infestation may be made, independently of the querulous ambiguity of surviving shreds of tissue. Natural sciences pass through various stages, more or less fruitful, which, for historical convenience, may be cliché-labelled by post-ster: “the humoral period,” “the phlogiston era,” “the age of Newtonian physics,” “the age of antiseptic surgery.” Perhaps palaeopathology will reap its richest harvest during the next quarter-century if it aims to enter “the facies phase.” It is easy to see this as a neglected path of enquiry, with abundant rewards for a modest outlay of effort. If so, Cockburn will have helped to sign-post this elementary track.

by SRBOLJUB ŽIVANOVIĆ

London, England. 22 : 70

The study of infectious diseases in ancient populations is a very interesting and promising subject, but sufficient data are still not available to provide a basis for a theory that would explain the origins of particular diseases. The evidence given in this article does not sufficiently support the contention that “most of man’s specific infections are descended from those acquired from his praehuman ancestors” nor that different species of primates acquired the infections from the same source. Not much is known about the evolution of different parasites, but we do know that it is not very difficult to induce mutations in primitive organisms. Similalities between certain parasites in different species may merely mean that they have adapted to similar environments or similar conditions. Thus the fact that the same intestinal protozoa are found in various recent apes and monkeys does not imply that all these protozoa were passed down to all these primates from a common ancestor. It may mean, instead, that the protozoa found similar conditions of life in these primates and so survived in all of them. That is, it is neither proof that the protozoa found in various recent primate species have a common ancestor, nor that the various primates have a common ancestor, but that a similar symbiosis between the protozoa and various primates exists, or existed in the past. Different primates may be infected in different ways by the same parasite, and not necessarily in the same stage of development. Similar serological reactions in various species of monkeys are simply due to infection by similar, or the same, microorganisms. Serological differences between Asian and African monkeys or recovery of treponemias in baboons cannot be a strong evidence in support of the theory that some treponemal infections have existed in man and his ancestors for many millions of years.

I agree with the author that there are numerous possibilities for studying infectious diseases and epidemiology in ancient populations, but I feel that it is too early yet to draw any conclusions. Much more study is needed before the whole problem becomes clear. The author is to be commended for drawing attention to it and for making an attempt to explain it.

Reply

by T. AIDEN COCKBURN

The remarks of the distinguished commentators are much appreciated, as are the references they give to additional papers not listed in my article.1 Almost all ask for more information on matters of special interest to them. The article is already lengthy, however, and if all were to be satisfied, the result would be another book. Much additional information, including some on the questions, raised by Bennett, of genetic feedback and myxomatosis virus in rabbits, is given in my two existing books (1963, 1967).

Some unkind critic once defined prehistory as

the study of the unverifiable to prove the unwarrantable about what never happened anyway.

This appears to be in line with the stance taken by Bennett in his comments. It is such a widespread viewpoint that the first chapter of my first book was devoted to this subject and titled “Speculation in Research.” A paragraph from this chapter (Cockburn 1963:8) dealing with the propriety of using analogy to theorize on past happenings is as follows:

The most reliable data come from our knowledge of infectious diseases today: by analogy, we can attempt to portray the diseases and infections of animals of past eras. There is much difference of opinion on the reliability of this method. One of its leading proponents was Wood Jones (1929) who thought that it was at least as good as the evidence produced by the geologic record. He received harsh criticism from his colleagues who described the evolution of the horse, using fossils to illustrate the various steps, did it correctly, but by error used fossil horses that were not in the correct line of descent. The task could have been done just as well by using analogies with existing animals. Le Gros Clark (1949) supports this, saying, “It is particularly noteworthy how closely some of the fossil types conform to intermediate stages that have been postulated on the evidence of comparative anatomy. Discoveries of such fossil relics, indeed, provide a remarkable vindication of the well-established methods of morphology . . . .” On the other hand, Hooton (1946) replies that this is not true. For example, early man walked upright but had a small brain, and since no such creature exists at the present time, no amount of reasoning by analogy could have inferred his existence. In the study of infections, such reasonings are largely academic, since all we have is the method of analogy, although some day it may be possible to demonstrate ancient infections with some certainty.

To Bhattacharya, I would state that my four years’ experience in India (see Cockburn 1960, 1961b), including studies in the Indus Valley, impressed me with the importance and antiquity of smallpox in that subcontinent. Smallpox probably evolved either in India or China and must have been devastating when it first appeared. Its effects on the Indus Valley complex could well have been severe.

To Chiarelli, I would reply that I deliberately played down the subject of malaria, haemoglobin, and red cell enzymes because it is so well known. Almost every author gives it prominence, so I chose to use other illustrations.

Claveaux stresses the importance of being population-oriented, and in this I am in full agreement with her. Incidentally, any study of an ossuary or cemetery shows that a large fraction of the skeletons are of small children. What were the main reasons for the deaths of these children? Hunger, violence, or infectious disease? I would suggest that the last was just as important as the first and much more so than the second.

Wells raises the question of reaction of certain pathogens to unusual evolutionary pressures. Of course, many
organisms can spread readily by means other than those usually utilized. Flea-transmitted plague bacilli and mosquito-borne Venezuelan encephalitis virus are notorious for their abilities to spread by air: tularemia organisms normally carried by biting insects to mammals can cause great water-borne epidemics if the mammals happen to be muskrats.

A change in environment might well lead to utilization of such potentialities and produce unusual forms of infectious diseases. On the other hand, some pathogens seem to have disappeared or become relatively harmless. Wells mentions the *sudor anglicus* or English sweat of the 13th and 14th centuries. Was this influenza, or some other disease that has since disappeared? In the 19th century, scarlet fever was the leading cause of death in children; now it is of only minor importance. No one knows why.

Hill has misunderstood the purpose of my giving data about current practices. The sole reason for this was to draw comparisons among past infections by analogies with present ones.

Lisowski's desire for more is appreciated. Perhaps some day a monograph on this subject will be attempted and his assistance requested. So much of the information is scattered in the journals of so many differing disciplines, however, that it would be a monumental task, one requiring the collaboration of scores of scientists. Here is a challenge for some university to organize the project.

Roffenburgh comments rightly on the growing threat from schistosomiasis. I agree that a statistical study on minimum population sizes for infections is urgently required. Perhaps he can stimulate some department to undertake the task.

Živanović is skeptical of my view that the intestinal protozoa of man are derived from those of man's ancestors and suggests that the similarities may be the result of convergent evolution. There are some difficulties with his hypothesis. For example, what were the differing organisms that evolved simultaneously in many primates' intestines until they all came to resemble one another? Where did they come from? Did the ancestral parasite have any intestinal protozoa, and if so, what happened to them? Did the evolving primates start off without protozoa and pick up their parasite loads at later dates? Are the intestines of all primates so much alike—regardless of whether the food consists of flowers, leaves, seeds, or flesh—that diverse protozoa must converge into common forms? I find these questions too difficult to answer and prefer the simpler solution that all intestinal protozoa were passed down from the ancestor common to all the primates.

This review was written in early 1968. Since that time, much significant work has been reported. Fribourg-Blanc and Mollaret (1968) have succeeded in isolating in hamsters a strain of pathogenic treponeme from a West African baboon. The appearance of the lesions in the hamster is consistent with the concept that the treponeme is the same as that causing yaws in humans. These workers have now examined 2,000 sera from primates of different species in different parts of the world. Positive sera have been found in animals in parts of Central Africa. The areas of human yaws and nonhuman primate treponematosis coincide.

Kuhn, Brown, and Falcone (1968) have tested 250 chimpanzees serologically and found 19% positive to one or another of three tests for syphilis. A wide range of other primate species was studied and antibodies found in a significant percentage of humans. These results suggest the possibility of widespread resistance or susceptibility. My conversations with scientists in the past two years give me the impression that this is a majority opinion. Several have challenged me to produce evidence of differential resistance of a genetic nature in human populations. I shall therefore discuss the matter at some length.

Obviously, in any particular epidemic a number of factors are involved, and it is impossible to disentangle them in order to determine their relative importance. However, the phenomenon of high mortality applies with such regularity to so many infections in so many diverse cultures that it is my opinion there must be a common factor. I admit readily to a bias in favor of differential susceptibility that has been with me since my earliest days as a physician. This bias arose from my experience with measles.

In 1935, I was in private practice in the coal-mining town of Bedlington, England when the triennial measles epidemic struck. Walking or cycling, I would visit the homes of sick children, and in one day would see 20–30 new measles cases. Those were the Depression years, and the children's diets were decidedly subnormal. It was also the days before antibiotics, so that treatment was mostly symptomatic and ineffectual. Yet out of more than 500 sick children under my care, not a single one died. There were plenty of complications, such as pneumonias and running ears, but no deaths.

In 1944, I was in the British Army in West Africa helping form the 81st and 82nd West African Divisions. About 50 cases of measles occurred among the families of the African soldiers, but this
was quite a different story. They received the same care as the children in Bed-lington, and their food was not too widely different. Of course, they had lots of parasites. However, they were far sicker and more prostrate than the English children, and several died. The only way I could account for the difference was in the body's reactions to the virus. (For contrary opinions on the subject of measles in Africans, see Seminar on the epidemiology and prevention of measles and rubella 1965.) Today measles in the unvaccinated is a prime killer in West Africa.

Some of the factors in genetically controlled susceptibility are given in an editorial in the British Medical Journal (see Genetics and infection 1969). Listed there are the following: agammaglobulinemia in children; selective IgA deficiency in children; an inherited deficiency of DPNH oxidase activity that is possibly sex-linked in some families but autosomal in others; a defective granulopoiesis with intramedullary destruction of granulocytes which is inherited as an autosomal recessive (the Chediak-Higashi syndrome), the basic defect being possibly in the structure of the membranes surrounding cytoplasmic organelles; an absence of the enzyme myeloperoxidase in polymorphonuclear leucocytes and monocytes, but not in eosinophils. This last type has been described in only one patient with disseminated candidiasis. His leucocytes showed deficient capacity to kill ingested Candida albicans and some bacteria.

The question of the relationship of the blood groups to susceptibility to infection is hotly debated at the present. Having no special knowledge of the subject, I shall not refer further to it. Apart from the well-known story of malaria and sickle cell anemia, the best example seems to me to be the resistance of Africans, and the great susceptibility of Europeans, to yellow fever (see earlier in this review).

Many groups of people practice discrimination in breeding, restricting marriage to limited populations for religious or social reasons. Obvious examples of this are the Catholics, Jews, and Parsees. Selection on the basis of infectious disease only is much rarer, but where it occurs it is of interest in the study of inherited resistance to infection. I shall give two examples here:

1. Leprosy, among the people of Gojjam in the Ethiopian Highlands, is believed to be hereditary rather than infectious. Therefore, strict genetic isolation, but no physical isolation, is practiced. Full genealogies are known for seven generations back, and marriage is forbidden between anyone who has leprosy in his genealogy and anyone who has not. In contrast, persons known to have leprosy may act as domestic servants of non-leprosy families and mix socially with them in other ways, with no other discrimination being practiced or fear of infection being felt.

2. Tinea imbricata (Tokelau ringworm) affects about 150,000 persons in the lowlands of eastern New Guinea and the neighboring islands. Sufferers experience continual itching and a severe social disability. People with the chronic disease are called pukpuk, pidgin for "crocodile"; the uninfectected are called "clean skins". Children are usually infected with Trichophyton concentricum by their mothers or nurses within the first few months of life. Appearance of the disease after the second year is uncommon. The disease restricts the choice of a marriage partner and is an important contributing cause of bachelorhood among men. According to Schofield, Parkinson, and Jeffrey 1963: 225):

Unequivocal evidence for or against a genetic factor has not been found. However the findings, that the great majority of children have become infected in the first 2 years of life and that children of infected mothers become significantly less often infected if their fathers are "clean skins" than if their fathers, too, have the disease, are suggestive. Children up to 2 years of age in such intimate contact with their mothers that this contact risk for the children in both the Groups I and II must have been maximal. It is difficult to envisage any relative environmental advantage which "clean skin" fathers could confer upon these children at this age; therefore it is quite possible that this advantage may be a genetic one. Comparison of reinfection rates with primary infection rates among adults in the same environment indicates that some people are permanently more prone to the disease than others.

Kuru is an infectious disease that probably has a genetic component. This fatal organic disease of the central nervous system occurs in a relatively small area of the Central Highlands of New Guinea. It affects people of the Fore linguistic group, of whom there are some 10,000 to 15,000 individuals. Between 100 and 200 of these people die of the disease each year. To the Fore people, kuru is the most important disease that occurs, and it is a dominant factor in their culture (Zigas and Gajdusek 1957). Recently the agent of kuru has been identified as a virus. There is an asymptomatic incubation period of 14 to 38 months (Gibbs and Gajdusek 1970). The most likely mechanism of transmission is cannibalism. Women and small children eat the brain, and this is known to be heavily infected. Only women and small children acquire the disease. Since cannibalism was stopped in the late '50's, the disease has decreased sharply. Alpers writes (1970: 137):

The argument for genetic susceptibility rests not on the familial incidence (for this could equally point to an environmental factor), nor on the genetic analysis of pedigrees (so we are not necessarily postulating a single gene effect), but on the fact that the disease remains confined to the Fore-speaking people and those who intermarry with them. The surrounding people have essentially the same culture as the Fore, including, until administrative contact, the practice of cannibalism, and the same recent history. The one thing that distinguishes kuru-affected from kuru-free clans and hamlets is intermarriage with the Fore.

The field of infectious diseases is so vast that it cannot be covered in toto in a review of this nature. Some of the major areas to which only passing references have been made include the nature and evolution of the pathogens themselves, the roles of the intermediate hosts and arthropod vectors, the mortalities produced by infections in the varying peoples, and the influences of these mortalities on the destinies of the populations. One thing is quite clear, that the history of mankind has been shaped to a considerable degree by infectious disease. It is hoped that this paper will interest anthropologists into taking a closer look at this important but neglected aspect of the study of man.

References Cited

ACKERNCKERT, ERWIN H. 1965. History and geography of the most important diseases. New York: Hafer.


ANGELE, J. L. 1946. Skeletal changes in current anthropology


Courville, Cyril B. 1948. Injuries to the skull and brain in Oceania, with reference to the mechanism and nature of such injuries, the measures used in protection against them, and their treatment particularly among the Melanesians. Bulletin of the Los Angeles Neurological Society 16:14–70.


Haygarth, J. 1793. A sketch of a plan to exterminate the canid maladies from Great Britain and to introduce general inoculation. London.


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