Mammalian Ecology and Epidemiology of Zoonoses

Various aspects of the ecology of mammals predispose their involvement in disease cycles.

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The United Nations' joint FAO/WHO Expert Committee on Zoonoses reports more than 150 zoonotic diseases (*1*). More than half of them constitute major health problems, particularly in countries near the equator. Zoonoses may be defined as "those diseases and infections which are naturally transmitted between vertebrate animals and man" (*2*). (They do not include human diseases, such as tuberculosis, that other vertebrates acquire from man.) Many zoonoses involve small mammals, especially rodents. Yet, with the exception of studies concerned mainly with domestic animals, most studies of zoonoses emphasize the vectors (or intermediate hosts) in disease cycles. Very little information is gathered about the normal hosts when these are nondomesticated animals.

Many zoonoses involve man and domestic animals only as accidental or incidental hosts in terms of the normal life cycles of the pathogens. Audy calls this situation a "cul-de-sac" for the pathogen (*3*). In an accidental host, infections sometimes take a bizarre form and morbidity and mortality may be much greater than in the normal host. This seems to be the case in scrub typhus, Japanese encephalitis, tick-borne encephalitis, Rocky Mountain spotted fever, tularemia, and some other diseases in man. Because of the cryptic nature of these diseases in their normal hosts it is important to determine the characteristics of their endemic life cycles with regard to vector and host biology in order to evaluate the potential for a given zoonosis becoming epidemic. Yet seldom in studies of zoonoses are the normal hosts dealt with beyond being mentioned as the sources of the pathogens or parasites. Little or no information is recorded regarding their ecology, and often the specimens are discarded after examination, so that it is impossible to confirm identifications.

Audy stressed the ecological approach in the study of epidemiology, especially the use of parasite patterns as "ecological labels" in studies of host biology (*3*). But isn't the other side of this coin the use of various aspects of the biology of hosts as "ecological labels" in studies of the life cycles of zoonotic pathogens? Failure to make adequate use of ecological data pertaining to wild host species in epidemiological studies of zoonotic diseases can be a significant oversight. Such information may be valuable for an understanding of the life cycle of the pathogen and may provide significant clues for the control or eradication of the disease. But one must not expect quick answers, since this research area has been largely neglected for a long time. Most research institutions dealing with epidemiological problems have long-established departments concerned with vectors, intermediate hosts, and pathogens; these concerns are traditional. Few have departments devoted to the study of nondomesticated hosts.

This article delineates some of the potentials of increased emphasis on studies of the ecology of hosts in relation to epidemiological studies of zoonoses. My comments on the current widespread approach to the study of zoonotic diseases are not meant to be derogatory. I simply advance them in the hope that an interchange of ideas will bring about a more equitable emphasis on the disciplines of ecology and epidemiology.

The inadequacy of the emphasis on host biology appears to have resulted from differences in the interests of the medically trained professionals and of researchers trained in the basic natural sciences. The former often appear to be more interested in finding treatments and cures for diseases than in attempting to understand the life cycles of the pathogens and in ultimately controlling or preventing diseases. Moreover, as each field has become increasingly specialized, the no-man's-land between medicine and the basic natural sciences has widened. The failure of each of these groups to understand and appreciate the goals and the problems of the other has in some instances even bred a kind of mutual contempt and distrust.

This gap between the medical sciences and the basic natural sciences has been most often filled by epidemiologists. After all, epidemiology is, in a sense, the study of the "ecology" of disease, or at least it should be. The background and training of such researchers, however, is usually medical. There are a few notable exceptions—researchers who have undertaken the background work necessary to develop proficiency in the ecological approach. Those who depend on clinical training alone in attempting to deal with ecological problems are not likely to make any more progress than could be expected of an ecologist entering the field of clinical medicine. Yet, if progress in this research area is impeded partly because of insufficient ecological training and experience on the part of the researchers involved, human suffering is unnecessarily prolonged.

Many aspects of the ecology of a host species predispose its involvement in a disease cycle. What follows is a discussion of some ecological factors that appear to be important. In many cases no concrete examples can be given because pertinent, definitive studies have not yet been conducted.

Distribution

The distribution of a zoonotic pathogen is usually unrelated to activities of man, except perhaps indirectly as man alters the environment. The pathogen may exist unknown to man until he happens to become involved in the life cycle (*3*). If the pathogen or parasite is

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dependent on one or a group of vector and host species, then its distribution will be determined by the distribution of the vector and host. I limit my comments to mammalian hosts because these are perhaps the ones that are most commonly involved in zoonoses (and because I know much less about birds and other vertebrates).

Distribution, in the broadest sense, is the geographical distribution of a species, but affinities for specific habitats or ecological communities and distribution, both horizontal and vertical, within the community are also important. Temporal distribution may also determine a species' predisposition to involvement in a disease cycle.

The geographic distribution of most mammals is fairly well known. Much more difficult is the question, Why do they occur where they do and not in other places? Mountain ranges, large bodies of water, or other discontinuities in the environment may be natural barriers for many nonflying mammals, but the geographic boundaries of a species may not coincide with such physical features of the environment. Sometimes the distribution may correlate with the distribution of specific plant communities. In many cases the distribution of a mammalian species includes various habitats that have different plant communities. Thus, the question of what species occur where, and why, is still a very perplexing one. The same question pertaining to pathogens is equally perplexing, and of great consequence to the understanding and control of zoonoses.

For example, Vietnam, Thailand, West Malaysia, and Indonesia have many of the same species of rodents, including rats. Rodents, especially rats, have been implicated in the maintenance of the endemic life cycle of the bubonic plague bacterium (*Pasteurella pestis*). This has been demonstrated by the recovery of the pathogen and the vector flea from the host, but very little other information about this life cycle is available, particularly regarding possible host species that do not live in association with man. Epidemic plague occurs periodically in Vietnam, Thailand, and Indonesia (in Java), but not in Malaysia. It appears to be endemic in Vietnam, but not in Thailand, and it may be endemic in Java (*4*). Why does this peculiar distribution pattern occur? Although some attempts are made to control urban rat species in these countries, very little is done about rats in the rural and forest areas. Yet urban rats (*Rattus rattus*) are not well suited to the role of reservoir host to maintain an endemic life cycle of plague bacterium because a high proportion of the rat population dies, leaving a resistant breeding population after initial infection with the pathogen (*4*, p. 302). Thus far no satisfactory explanation can be offered for the distribution pattern of this disease and the maintenance of its endemic cycle, I think, because of the paucity of information regarding the ecology of the various host and potential host species. The geographic patterns of distribution of other diseases, such as rabies, scrub typhus, tick typhus, and tick-borne encephalitis, are equally perplexing.

Within a given geographic area mammals are not distributed randomly but often display strong affinities for certain types of habitats. The habitats may vary with respect to water supply, soil characteristics, and altitude. One may see a certain association of mammalian species in a lowland forest and a different species association in adjacent forests at higher elevations, especially in countries near the equator (*5*). For example, in West Malaysia, in habitats at altitudes above 1000 meters, some of the flora and fauna (*Dremomys rufigenis, Rattus edwardsi, R. fulvescens*) are more characteristic of areas farther north, such as China and the Himalayan region, than of the adjacent lowland forests (*6*). These communities at high elevations are essentially "ecological islands" in a sea of lowland forest, often quite isolated from other similar communities. In some areas such "ecological islands" have a fairly unique fauna, perhaps as a result of their long-term isolation [examples are species reported only from Mount Kinabalu in East Malaysia (*7*)]. One may see specialized as well as relict species in such communities.

In the lowland forests one may also see differences in species associations in adjacent (but different) habitats. For example, in West Malaysia certain mammals, such as *Ratufa bicolor* and *Pteromyscus pulverulentus*, may be common in the climax forests but rare or nonexistent in secondary or regenerating forests. Other species, such as *Rattus tiomanicus* and *Rattus annandalei*, are much more common in regenerating forests but rare or nonexistent in climax forests. Some species, such as *Rattus rattus* and *Rattus exulans*, seem to occur mostly in association with man and are common around villages, plantations, and other disturbed areas but rare in forests (*6*, *8*).

Some evidence indicates that pathogens also may be more prevalent in some habitats than in others. In Malaysia, for example, parasitic protozoans, related to malaria parasites, occur more often in arboreal rodents in the primary forests than in rodents living in secondary forests (*9*). These differences may, of course, also be influenced by vector distribution and other factors.

Not all species of mammals are necessarily restricted to single habitat or elevation. For example, *Rattus sabanus* lives in various types of habitats, and *R. bowersii*, at various elevations (*6*, *8*). It is perhaps particularly important to recognize such species because of their potential for transmitting pathogens or parasites characteristic of one habitat into another. Thus, pathogens that are normally endemic to forests may be introduced into conspecific populations that associate with man, and man may ultimately become involved. In the United States, for example, animals that are normally forest dwellers, such as rabbits (*Sylvilagus floridanus*) and squirrels (*Sciurus niger*), are becoming increasingly associated with suburban areas, roadside areas, and urban parks. Of course, this does not mean that each rabbit is a public enemy, but one should realize that the potential routes of introduction of such zoonoses as tularemia, plague, and encephalomyelitis are present. Bats, the only flying mammals, are capable of transporting pathogens over great distances, even across great expanses of water. Yet, astonishingly little is known about the biology of these and other mammals, especially in countries near the equator—even about the biology of species that have already been implicated in various zoonoses.

To complicate matters more, even within a habitat, such as a mature equatorial rain forest, mammals are not distributed randomly. Among the nonflying mammals in West Malaysia approximately two-thirds of the species are arboreal, living in the canopy or the subcanopy and seldom descending to the ground (*10*). Moreover, many of the species considered terrestrial are actually semiarboreal. Little is known about the arboreal species in particular, because it is difficult to place traps in the canopy (which is often 50 or 60 meters high) and because the lower strata of the canopy shield the upper canopy from observation.

Correlated with the vertical distribution of host species in West Malaysia is a vertical distribution of the prevalence of parasites, particularly of blood parasites, including plasmodia. Arboreal rodents, such as squirrels and flying squirrels, were found to be parasitized far more often than species, such as rats, that are active primarily on the ground (*11*). Apparently the risk of infection differs in the various vertical zones in the forest. These differences in rates of parasitizations may be correlated with the vertical distribution of vectors, which in turn may depend on specific microclimates and microhabitats that occur in the various vertical zones, or with differences in resistance of hosts to the parasites.

Distribution through time is another complicating dimension. Species differ with respect to periodicity of activity. Broadly speaking, one may divide them into groups that are primarily diurnal, nocturnal, or crepuscular. Within these general categories, species may confine their activity to certain shorter periods; this restricts their use of the environment both temporally and spatially. This has important bearing on their involvement in disease cycles that are maintained in conjunction with vector species that are also periodic. Transmission of a pathogen or parasite would occur only between those vectors and hosts that are predisposed to come in contact as a result of their characteristic activity periods. Perhaps a host may be accessible to the vector while the host is active, or it may be more susceptible while it is inactive in the nest. Perhaps it is accessible only when it visits specific parts of its habitat. Perhaps these visits occur only at certain times of day. Perhaps they are seasonal. If the activity of the vector is periodic and its time of greatest abundance seasonal, then the host's risk of infection is also periodic or seasonal. However, in most zoonoses only the host and the vector are known; the place, time, and other circumstances of transmission are not known.

The importance of the temporal factor in the distribution of hosts may be illustrated by the occurrence of red blood cell protozoans in the arboreal rodents of Malaysia. Undoubtedly other factors are also involved, but the correlations are very suggestive. Among the arboreal rodents are several giant squirrels. The diurnal species (*Ratufa bicolor* and *R. affinis*) are commonly infected with *Hepatocystis* (a malaria-like parasite), but so far no diurnal species has been found to be parasitized with *Plasmodium* (*9, 11, 12*). Two of the nocturnal species (*Petaurista petaurista* and *P. elegans*), on the other hand, are frequently parasitized with *Plasmodium* but not with *Hepatocystis* (*12, 13*). These two groups of squirrels occur in the same habitats and even in the same trees, at similar heights, but they differ most strikingly in that *Ratufa* is active during the day and *Petaurista* at night.

Feeding Habits

Food is an important element of the environment of mammals. The presence of certain foods may limit a species' distribution. The abundance of food may account for seasonal fluctuation in the physiological state of the individual, and thus for its resistance to disease and perhaps for its reproductive potential. The diets of some mammals are generalized while those of others are restricted. It may follow from this that some species have general, whereas others have restricted, distributions.

Parasites, in the form of eggs, larvae, or adults, may be transmitted not only with food but sometimes also in other ways during feeding. Thus, both a mammal's diet and its feeding habits may determine the likelihood of its acquiring an infection. A carnivore may be bitten by vector ectoparasites of the prey. The diet of prey animals may determine what parasites the predator may acquire. In West Malaysia it has already been shown, in a general way, that diets of mammals that include a large proportion of insects or meat predispose the mammal to infection by endoparasites, particularly helminths (*11*). Moreover, some mammals may be accessible to vectors, or directly to the parasites, only in places where they feed; the parasites need not be in the food itself. Fruit bats, for instance, are most accessible to nocturnal mosquitoes when the host comes to rest or during feeding.

Since man is largely carnivorous he can readily become involved in life cycles of disease-producing organisms that depend on predator-prey interrelationships. Examples of this are plentiful: trichinosis, taeniasis, trematode infections, and so on. Parasites may also be acquired from contaminated food. In West Malaysia the rat lung worm, *Angiostrongylus cantonensis*, is a common endemic parasite of snails and of rats that eat snails—for example, *Rattus tiomanicus* (*14*). This worm is possibly one of the infective agents responsible for eosinophilic meningoencephalitis in man (*15*). The parasite may be acquired by man when he eats infected snails or even lettuce contaminated by certain mollusks (*14*). Similarly, *Paragonimus westermanni*, a lung worm, may be acquired from eating infected land crabs (*16*).

Reproduction

Sometimes the enzootic phase (the phase characterized by low-level, continuing infection) of a disease cycle may become epizootic (characterized by high-level, sporadic infection) if a large number of susceptible hosts become available. Epidemics in man and domestic animals may follow. Thus, the recruitment rate and the overall population turnover of potential host species are important considerations in epidemiological studies of zoonoses.

In the tropics some mammals breed throughout the year while others have seasonal peaks of reproduction. Generally speaking, adult animals, when they become infected, either die or become immune. The transmission cycle of the pathogen requires susceptible individuals. Thus, it is important to know when young animals, which are susceptible, appear on the scene. Does this occur in a particular season or throughout the year? Litter size is also important, because the rate at which young are produced depends on this, as well as on infant survival. The females of some species have successive pregnancies while others breed infrequently. Reproductive age is also important. If their longevity is the same, species in which females breed at an early age would have larger breeding populations, and a higher reproductive capacity, than species in which breeding is delayed.

The species that have a high reproductive capacity are probably more important in disease cycles than those that have a slow population turnover. However, their relative importance also depends on the nature of the life cycle of the pathogen or parasite. The different types of disease-producing organisms may use different tactics. Perhaps those that cause the most severe morbidity and mortality are the least well adapted to their hosts in terms of long-

term infection. They practice a sort of "slash-and-burn" economy. The endemic pathogens often appear to do little damage to their normal hosts, and the infections persist "silently" in a given locality. However, if an abnormal host, such as man, enters the scene and perhaps alters the normal habitat and thus perhaps also the normal transmission cycle, the endemic phase may shift to an epidemic phase, and the nature of the infection in the abnormal host may differ markedly from that in the normal host. A good example of this is the shift from the endemic cycle of jungle yellow fever in primates in the jungle canopy to an epidemic phase in man when jungles are cut and vector mosquitoes from the canopy are compelled to seek host species, including man, nearer the ground.

Those species of mammals that acquire low-level infections and have a less severe immune response are perhaps the most suitable as reservoir hosts, particularly if successive litters are produced throughout the year, resulting in a steady and continuous recruitment of nonimmune or susceptible individuals into the population. The pathogen is able to persist, although at a low level.

On the other hand, mammals that acquire heavy infections, followed by a strong immune response or death, generally are not suitable as reservoir hosts. If their population turnover is rapid—that is, if they have large and frequent litters and reproduce at an early age—they serve well as amplifying hosts. Seasonal breeders are significant in the life cycles of pathogens only during the host's breeding season, when susceptible individuals are recruited into the population. An example of this is the house rat, *Rattus rattus*, which is very susceptible to bubonic plague. Infections are heavy, and the rats often die. Local populations may be entirely exterminated by the disease, or only resistant individuals may remain (*4*). Thus, it is not possible for such a species to be important in the long-term maintenance of endemic plague. Other species, perhaps those not usually in close association with man, may be much more important.

At present we know very little about the reproductive cycles of mammals in equatorial countries, especially of those species that do not live in close association with man. Yet this information is important in evaluating the potential involvement of various species in zoonoses.

Behavioral Factors

Some species may appear so similar morphologically that even professional taxonomists may confuse them. They may live in the same habitat or in similar habitats but, because of differences in their behavior, function as discrete ecological entities. To illustrate this, one may take a striking example from among the invertebrates, "the field cricket" *Gryllus*. Leading taxonomists for a long time accepted this as a single species. However, on the basis of sounds and other behavioral characteristics, specialists were able to distinguish 17 species (*17*). The taxonomy of various mammals, especially the genus *Rattus*, is in a similar state of flux. However, this is a problem only for taxonomists—the species have no trouble telling one another apart. In their habitats each species is a separate ecologically functioning unit. Thus, the degree of their involvement in zoonotic disease cycles may also differ, depending on ecological factors such as specific habitat selection, periodicity of activity, dietary preferences, and reproductive activity.

Involvement in a zoonosis may also depend (i) on whether the nesting site is in the ground or in a tree, (ii) on the height of the nest, and (iii) on whether the nest is loosely constructed in the open or is in a tree cavity with abundant nesting material acting as a barrier to some vectors or as a home for others. Such manifestations of behavior may determine the types of vectors to which a host species is accessible while it is inactive. Yet few or no data are available for ascertaining the specific roles of these factors.

In some species behavior patterns appear to limit the size of the breeding population. The population may be highly organized in a given area, with reproduction limited to individuals in possession of territories. If this organization is disturbed by man, even by man's attempts at control, the population may increase rather than decrease (*18*). Yet, because of the paucity of information about the life histories of potential host species, it is very difficult to evaluate the effects of control measures and the resultant effects on zoonoses, especially in equatorial ecosystems.

One of the standard activities in epidemiological studies of zoonoses is to survey potential host species and their pathogens or their antibodies against pathogens. Most commonly a single approach is used, such as trapping with standard baits. The results are tabulated, and estimates are made of the involvement of various species in the life cycle of the zoonotic pathogen and the relative abundances of the various host and potential host species in the area. But in doing this one must take into account behavioral differences, both in species and in individuals. If the traps are placed on the ground, the probability of capturing arboreal species is greatly reduced and their relative abundances will be greatly underestimated. Some species, such as *Callosciurus nigrovittatus* in West Malaysia, are behaviorally predisposed to enter traps readily, while others, such as *Ratufa affinis*, tend to avoid them. These squirrels are observed in the same habitats, but their habits differ. The relative abundances of species that enter traps readily would be greatly overestimated in a trapping survey. Moreover, if a standard, single bait is used, some species may not be attracted to the traps.

The trapping experience of the Division of Medical Ecology at the Institute for Medical Research, Kuala Lumpur, Malaysia, makes it appear that some species, particularly arboreal species, are very rare. However, such species were found to be common when other collecting methods were used, such as searching for nests and capturing the inhabitants.

Conclusions

The ecological factors pertaining to hosts which I have briefly discussed above are by no means the only ones. So little research has been done in this area that it is not possible to anticipate all of the ramifications of host biology that may have bearing on involvement in the life cycles of zoonotic pathogens. It has been suggested by Audy (*3*) and others that epidemiological problems should be approached in a multidisciplinary manner. We have a long way to go before attaining this goal. Unless these problems are attacked by team effort, with each aspect of many different biomedical interests represented on the teams, we may expect to see increasing numbers of papers that list parasites and their hosts, or hosts and their parasites, but little progress in understanding the ecology of the zoonotic diseases and in finding measures for their ultimate control.

Summary

Insufficient use has been made of ecological data concerning potential hosts in studies to determine the life cycles of zoonotic parasites and pathogens. Factors such as the geographical distribution of hosts, the altitudes at which they live, their affinities for specific habitats, their vertical distribution within the habitat, and the periodicity of their activities have bearing on the hosts' predisposition to involvement in disease cycles. Diets and feeding habits may determine the likelihood of acquiring infection. Reproductive characteristics determine whether a species is suitable as a reservoir or as an amplifying host. Behavioral factors, such as selection of a particular kind of nest site, may also predispose the involvement of the host with parasites and pathogens. Behavior patterns may determine the maximum population densities of hosts. Estimates of population sizes, of relative abundances of species, and of the involvement of species in disease cycles may be strongly influenced by the collecting and sampling methods that are used and also by the behavioral response of the mammals toward collecting devices, such as traps.

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Prospects for Genetic Intervention in Man

Control of polygenic behavioral traits is much less likely than cure of monogenic diseases.

Bernard D. Davis

Extrapolating from the spectacular successes of molecular genetics, a number of essays and symposia (*1*) have considered the feasibility of various forms of genetic intervention (*2*) in man. Some of these statements, and many articles in the popular press, have tended toward exuberant, Promethean predictions of unlimited control and have led the public to expect the blueprinting of human personalities. Most geneticists, however, have had more restrained second thoughts.

Nevertheless, recent alarms about this problem have caused wide public concern, and understandably so. With nuclear energy threatening global catastrophe and with so many other technological advances visibly damaging the quality of life, who would wish to have scientists tampering with man's inner nature? Indeed, fear of such manipulation may arouse even more anxiety than fear of death. The mass media have accordingly welcomed sensational pronouncements about the dangers.

While such dangers clearly exist, it also seems clear that some scientists have dramatized them (*3*) in order to help persuade the public of the need for radical changes in our form of government (*4*). But however laudable the desire to improve our social structure, and however urgent the need to improve our protection against harmful uses of science and technology, exaggeration of the dangers from genetics will inevitably contribute to an already distorted public view, which increasingly blames science for our problems and ignores its contributions to our welfare. Indeed, irresponsible hyperbole on the genetic issue has already influenced the funding of research (*5*). It therefore seems important to try to assess objectively the prospects for modifying the pattern of genes of a human being by various means. But let us first note two genetic principles that must be taken into account.

Relevant Genetic Principles

Polygenic traits and behavioral genetics. The recognition of a gene, in classical genetics, depends on following the distribution of two alternative forms (alleles) from parents to progeny. In the early years of genetics, after the rediscovery of Mendel's laws in 1900, this analysis was possible only for those genes that exerted an all-or-none control over a corresponding monogenic trait—for example, flower color, eye color, or a hereditary disease such as hemophilia. The study of such genes has continued to dominate genetics. However, monogenic traits constitute a small, special class. Most traits are

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