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SOCIAL AND BEHAVIORAL BARRIERS TO PATHOGEN TRANSMISSION IN WILD ANIMAL POPULATIONS

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SOCIAL AND BEHAVIORAL BARRIERS
TO PATHOGEN TRANSMISSION
IN WILD ANIMAL POPULATIONS

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ABSTRACT

Disease and pathogens have been studied as regulators of animal populations but not really as selective forces. I propose that pathogens can be major selective forces influencing social behaviors when these are successful at reducing disease transmission. The behaviors whose evolution could have been influenced by pathogen effects include group size, group isolation, mixed species flocking, migration, seasonal sociality, social avoidance, and dominance behaviors. Mate choice, mating system, and sexual selection are put in a new light when examined in terms of disease transmission. It is concluded that pathogen avoidance is a more powerful selective force than has heretofore been recognized.

INTRODUCTION

Pathogens (diseases, internal and external parasites) are a significant source of mortality, particularly in larger organisms not so limited by predation (Freeland, 1976; May, 1983). May (1988) argues that the significance of pathogens is generally underestimated in ecology. When pathogens are considered, they are generally viewed as simple regulators of population size. For overly large, and thus stressed populations it has often been shown that disease is an effective regulatory mechanism. Thus the most common question asked about disease is its contribution to the total mortality or population dynamics of a population (e.g., Anderson and May, 1979a; 1979b; Ball, 1985; May, 1983; Murray, 1987; Murray et al., 1986). Freeland (1983) has proposed that coexistence of taxonomically related species may in fact be limited by pathogens, which may therefore control invasions and the overall structure of communities. While the role of pathogens in regulating animal populations and structuring communities is gaining increasing attention, the evolutionary consequences of pathogen-induced mortality have received inadequate attention. In particular, since pathogen transmission may be affected by the rate of contact between individuals (which is affected by group size, mating, and social behavior), it seems reasonable to wonder whether social behaviors might also be affected by selective pressure to avoid pathogens. However, very little has been done on the possible social implications of disease (as noted by

Terborgh and Jansen, 1986). Typically, it is noted that disease and parasites may be more common in larger social groups and may therefore be a cost of sociality (e.g., Alexander, 1974; Brown and Brown, 1986; Duffy, 1983; Pulliam and Caraco, 1984), but it is usually treated as a simple cost with no more specific consequences for social behavior (i.e., it is not related to dispersal, disease type, mating habits). Not all sources of mortality translate into selective pressures, however, so simple enumerations of costs of disease do not represent a complete analysis of the problem.

I propose that disease (including pests and parasites) may be a significant selective force in the evolution of a variety of social behaviors. The negative consequences of large group size may put an upper limit on group size in some species. Exchanges between groups may be risky enough under some circumstances to favor xenophobic behavior and group isolation. Sexual contact may result in disease and parasite transmission to a sufficient degree that mate choice and mating systems are affected. Dominance contests are affected by the disease status of the competing individuals, a fact that may have several social consequences. Migratory behaviors have disease consequences. These hypotheses and their interactions are elaborated here with the aim of developing a comprehensive picture of the role of pathogens in the evolution of social behavior. An initial exploration of the dynamics of disease, pointing out the key role of transmission mechanisms and effectiveness, leads to a classification of pathogen types and modes of transmission. In this context I consider the available modes of social behavior that could reduce transmission and evaluate them for effectiveness against the various pathogen types. For certain types of pathogens, no social behavior will be selectively advantageous. Whether the results obtained apply to a particular species of course depends on the balance with other selective forces such as predation.

Pathogen Resistance

Mechanisms for predation avoidance include speed, crypsis, early detection, group defense, size, and physical defenses (horns, shells, etc.). In general, none of these defenses are of any use whatsoever in dealing with pathogens. One can neither flee nor turn and fight when confronted with a disease (though one is tempted to try). Active defenses against arthropod pests are discussed below. There are two classes of defenses available: immunologic defenses, and physical barriers. The first is a response, the latter is an attempt to prevent pathogen contact.

Immunologic responses have been extensively studied in both man and animals. There are a bewildering array of defensive measures, including fever, inflammation, antibodies, macrophages and others (see Mettrick, 1987; Hart, 1988; submitted). Digestive chemistry and the intake of certain foods can inhibit gut parasites (Freeland, 1983). Licking of wounds is effective in promoting healing in many species (Hutson et al., 1979; Mandel and Ellison, 1981). Generally speaking, these defenses are more effective in animals that are well-fed and not under stress (physical or social). The immune system is so effective that some diseases are rarely found except in cases of immune suppression such as AIDS (e.g., Kaposi's sarcoma). Nevertheless, disease is still a major cause of mortality, particularly in young animals and during periods of unfavorable weather or poor nutrition (e.g., Borg, 1987). The strength of the immune system is less important if pathogens can be avoided.

Certain physical barriers (e.g., nasal hairs and mucus) are effective in capturing and expelling potentially harmful disease agents. In general, however, external barriers such as shells, fur or horny skin are effective only in discouraging skin diseases and external macroparasites such as mosquitos, flies, leeches, and ticks and any pathogens they may carry. I would argue that behaviors that reduce contact with pathogens have greater importance than physical barriers or even immune responses. When animals are prevented from grooming or from escaping from pests, the consequences are often serious or fatal (e.g., Bell and Clifford, 1964; Edman et al., 1974, Hart, submitted). Thus the means by which animals avoid pathogens and pests deserve serious consideration.

Pathogen Transmission

Many detailed epidemiologic models have been developed to integrate various aspects of pathogen life history (intermediate host, latency period, infective period, host immunity, etc.) so that the net effect of the pathogen can be assessed (Anderson and May 1979a; b; May and Anderson, 1987). A key parameter in such models, but one very difficult to measure, is transmissibility. I will argue later that this is also the key variable upon which evolution can act.

Table 1 summarizes pathogen type in terms of transmission mode. The mode is related to the effectiveness of isolation of social groups or individuals from each other in preventing transmission or attack. Note that in one place mosquitos are listed as a vector, in another as a pest (type of macro-exoparasite). A key result is that isolation is a poor defense against those pathogens that are wide-ranging or have a persistent stage, persistent vector (e.g., ticks), or wide-ranging vector (Lundqvist, 1988). Such pathogens may be labeled ubiquitous. For such pathogens group size or social behaviors may not be significant variables, as shown by Moore et al. (1988) for heteroxenous helminth parasites of bobwhite quail. For these pathogens, only defenses such as general vigor, increased immune system strength, thick fur or skin, avoidance of certain habitats (e.g., swamps) or other non-specific host defenses will be effective. The other general class is contact pathogens. These include diseases and parasites associated with excrement, those transmitted in body fluids (including bites and water droplets in air), sexually transmitted diseases, some skin parasites, and those acquired during food consumption (e.g., trichinosis). For contact pathogens, certain types of behaviors and social barriers could be effective in reducing pathogen-induced mortality.

Pathogen Avoidance

Behavioral mechanisms are important in reducing the incidence of disease. Various movements such as skin twitching are effective in repelling mosquitos (Edman et al., 1974). Feather preening is universally observed in birds, both for flight and for cleanliness. Many mammals lick their fur, with cats being noteworthy in this regard. Scratching and preening are effective for removing skin parasites (Hart, submitted). Other mammals, particularly large ungulates and artiodactyls incapable of preening, utilize mud wallows or dust baths to discourage external parasites. Even birds use dust baths and sometimes are observed letting ants crawl over them, presumably because the ants remove ectoparasites. Some birds also line their nests with aromatic green material to repel nest parasites (Clark and Mason, 1985; Wimberger, 1984). Sunning is an effective sanitary measure for skin infections and ectoparasites and also raises the body temperature which helps immune responses against diseases, just as fever does (Kluger, 1979; Hart, 1988). Higher level behaviors are often observed in animals inhabiting a permanent home site. For example, hymenoptera are quite diligent about removing foreign objects and dead comrades from the nest. Burrow dwelling large mammals (foxes, bears, wolves) generally defecate outside the burrow (Hart, 1988). Cattle avoid grazing directly adjacent to their droppings. Extensive documentation of sanitary behaviors may be found in Hart (1988; submitted).

As these examples make evident, there are many behavioral adaptations animals use to reduce attacks by ectoparasites and keep their home sites clean to reduce disease. In addition, it is reasonable to consider mechanisms whereby organisms could avoid contacting pathogens carried by conspecifics. In essence, this involves reducing interindividual contact by erection of social barriers, a process in conflict with other evolutionary objectives such as mating, territorial contests, and group living. The nature of an effective barrier depends on the mode of transmission of the pathogen.

Geographic Effects

Certain geographic trends may be expected in the social consequences of pathogens. In general, parasites, skin diseases, and bacterial diseases are more serious in warm-wet environments such as the humid tropics. Disease avoidance should thus be a more potent selective force there. External macroparasites such as biting flies and mosquitos are most serious in swampy habitats at all latitudes, but social behavior has little impact on their effect except that larger groups receive fewer bites per individual (Freeland, 1977; Duncan and Vigne, 1979; Hart, submitted). Respiratory diseases become more common during cold weather and are easily transmitted in groups. Thus the importance of pathogen avoidance behaviors should vary with latitude, season, and wet-dry gradients.

Another type of geographic effect concerns degree of isolation. Islands (habitat islands or real ones) are isolated from diseases, and host populations are low such that number of pathogen species is also likely to be low due to random extinctions of pathogens, as expected from island biogeographic theory and as documented for groups-as-islands by Freeland (1979). Helminth diversity is lower on small oceanic islands (Kennedy, 1978). Freeland (1983) documents reduced number of parasite species in invading species such as starlings when similar host species do not exist in the invaded territory. The birds of the Hawaiian islands had no endemic blood parasites and were consequently very susceptible to those brought by immigrants (Warner, 1968). Parasites of small mammals of the Inner Hebrides have lower host specificities than do the same species in Great Britain or Europe (Thomas, 1953). With both reduced number of disease/pathogen species to start an epidemic, reduced host specificity and low host population size, island populations' social behaviors such as territoriality and flocking are less likely to be the consequence of selection for disease avoidance.

BEHAVIOR OF INDIVIDUALS

I begin by consideration of the manner in which behaviors of individuals may have been influenced by the evolution of pathogen avoidance traits. The traits considered include avoidance behaviors, mate choice, and mate fidelity. The next section considers individual behaviors as they influence group structure and dynamics as well as the behaviors of groups *per se* (e.g., migrations, group size).

Social Avoidance

It would be useful to an individual to avoid contact with those carrying disease if this were possible. During early stages of infection there are usually no external signs of disease, and for many illnesses there never are obvious external signs. But for many diseases there are external signs such as lumps, sores, loss of hair, plumage aberrations (Hamilton and Zuk, 1982), change of skin color, changes in odor, and behavioral changes (Hart, submitted). Do animals use any of these as clues for initiating avoidance behavior?

There is some evidence from human behavior that such clues could exist. There is an innate revulsion in humans to the appearance of gross deformities, open sores, scabs, etc. This is true even in societies not familiar with the germ theory of disease. Movie makers have capitalized on this revulsion in a long series of grade-B movies. I would distinguish this revulsion from xenophobia because it exists even within one's own group. A natural consequence of this revulsion is avoidance of the sick individual or of close contact with them. This results in a reduced probability of transmission of disease. It is not necessary to assume that such revulsion should only be confined to actual contagious diseases because the discrimination of contagious vs noncontagious (e.g., cancer) symptoms may not be possible at the instinctive level. Furthermore, if the cost of avoiding an individual that is apparently, but not in fact, contagious is not very high, there may be little selective pressure for finer discrimination.

There are several lines of evidence indicating that animals could respond to such cues. In primates a period of habituation is needed before a new individual becomes involved in mutual grooming. Such reluctance to contact strangers is consistent with avoidance of disease. Goodall (1986) notes two instances where wild chimps wiped the point of contact of a strange chimp's touch. Goodall also notes several instances where male chimps wiped the diarrhea-stained bottom of a female they wanted to mate with. While chimps are intelligent, they do not know about disease. Thus these observations are very interesting. Several studies have found females to avoid mating with diseased males, in some cases based solely on behavioral clues (e.g., Kennedy et al., 1987).

It may be possible for other clues to be used that are correlated to disease, even though not direct signs of it. As noted above in the section on dominance hierarchies, general low vigor as evidenced by the lack of ability to win dominance contests may be correlated to disease state. Thus dominant individuals can selectively "avoid" sick individuals by forcing them to keep their distance and by subjecting them to increased predator risk.

One source of available information concerns the general health of the individual as perceived by itself. If some physiological state is indicative of enhanced pathogen susceptibility, then when an organism is in that state it should take extra measures to avoid contact with infected individuals. When humans are exhausted, run down, malnourished, or sick, they tend to be irritable and listless and to seek solitude. Similar behaviors are exhibited by sick animals (Hart, 1987; submitted). These behaviors will reduce the rate of contact with other individuals and thus reduce the risk of contracting additional diseases. It is particularly important for a sick individual to avoid catching further diseases because it is often the complications of secondary illnesses that are actually fatal.. Inclusive fitness arguments indicate that a sick individual should avoid giving the illness to other group members if they are related, and thus again should seek solitude. On the other hand, close knit family groups may all be exposed to the same pathogens so that the benefits of continued contact (e.g., grooming) may outweigh the risk of disease transmission. Data on animal behavior in this regard are sketchy, but give the general impression of pointing in the same direction. Sick individuals of livestock do seem to be isolated somewhat from the herd (L. R. Rittenhouse, Dept. Range Science, Colo. State University and Ray Strickland U. Md., pers. comm. 1988), though whether by voluntary isolation or avoidance on the part of others is not clear. Edwards (1988) found a lower degree of touching between infected mice and other group members.

It can be argued that social isolation is merely a coincidental side-effect of debilitation from disease. However, Hart (1987) argues that the behaviors of sick animals form a coherent adaptive response to disease. Depression (lack of interest in surroundings), lack of appetite, and lack of grooming all act to reduce energy expenditures so that disease can be fought better. To this model

I would merely add irritability as a further isolating mechanism and postulate the beneficial consequences of isolation for avoidance of pathogen transmission.

Since it is in the interest of both the sick individual and other group members for the sick to be somewhat isolated, the question of communication of physiological state arises. If a sick animal "looks sick" and other animals can interpret that appearance as "being sick" and act accordingly (by avoidance), then they all benefit. Signals indicative of sickness include skin coloration (e.g., flushed or pale), posture, facial expression, alertness, and behavior. Edwards (1988) found healthy mice to direct increased exploratory behaviors (e.g., sniffing) toward infected individuals, indicating behavioral or perhaps scent clues to disease status. I hypothesize that, because a weakened (malnourished, etc.) individual is also at risk from disease, it should "act sick," even though it is not, as a protective measure. Note that in humans many of the same postural, skin tone, and behavioral cues (e.g., irritability) are present for both sick and exhausted (weakened) individuals.

Since sexual contact provides an opportunity for pathogen transmission of all sorts, I might hypothesize that sexual drive and receptivity will fall when the organism is stressed, starved, or sick. This should not be true for organisms that have a brief mating season or only one brief chance to mate. In support of this hypothesis, I note that in humans sexual-contact disease susceptibility goes up in stressed individuals. In women, yeasts and related disorders are more readily acquired during illness, during menstruation, and during pregnancy. Among promiscuous homosexuals, drug use and previous history of venereal diseases (which reduce immune system strength) are highly predictive of probability of recurrent sexual disease infection. Sexual drive and receptivity in humans are reduced or eliminated by illness. This is most evident in women, who are more affected by sexually transmitted diseases and suffer from diseases that have little effect in men (e.g., chlamidia, yeasts). Thus the evidence favors consideration of pathogen avoidance as a potential selective force affecting sexual drive and receptivity, though other factors also play a role. Such effects will not be evident in animals that only mate once a year or once in a lifetime.

Mate Choice

Avoidance of sick individuals could also occur during mate choice. It has been proposed that sexual selection for secondary sexual traits such as bright plumage could be effective if the secondary traits were indicative of genetic resistance to diseases or parasites (Hamilton and Zuk, 1982). For example, it has been shown that in birds such as peacocks, those families with increased problems with external parasites have more species with bright plumage, that within a family species with more parasites tend to have more gaudy plumage, and that within a species the individuals with the brightest plumage and biggest tails tend to have the least parasites (Hamilton and Zuk, 1982). This same effect has been found in freshwater fish (Ward, 1988) where sexual dichromatism is highest in families with higher parasite loads. Guppy females have been shown to prefer unparasitized males, which also display more (Kennedy et al., 1987). Thus, in such species where parasites are a greater selective pressure, mate choice biased toward brightly colored males enhances selection of parasite resistance genes for the offspring. An overlooked consideration here is that mating offers an excellent opportunity for disease or parasite transmission (including both regular and sexually transmitted diseases, skin parasites, and yeast and fungal infections), including transmission to the offspring (Service, 1986). Simian HIV is an example of a sexually transmitted disease. Male or female sterility may result from infection by brucellosis (Witter, 1981) or myxomatosis (Yuill, 1981). The fitness value of a sterile individual is zero. The risk of disease transmission during mating is probably grossly underestimated. It has been shown in rats that prevention of male grooming after copulation leads to a high incidence of transmission of genital infections (Hart and Haugen, 1971; Hart et al. 1987). Hart (submitted) argues that post copulatory autogrooming in the genital area evolved for prevention of disease and notes that species that exhibit such behavior exhibit fewer sexually transmitted diseases than those that do not. This risk of transmission is particularly marked in species that normally are not in close physical contact except during matings, which is the case for many brightly feathered birds, which do not flock. It has been noted that even a 24-hour association is sufficient for transfer of a full complement of lice to an uninfected mouse (Bell and Clifford, 1964). It may thus be in the female's best interest to attract or choose a mate who is not diseased to avoid getting contaminated. Secondary sex characters as well as size, singing intensity, fur luster, feather length and brightness, and aggressiveness may be indicative of general health and could thus be disease avoidance criteria during mate selection (e.g., Kennedy et al., 1987). Avoidance of disease could be a much stronger selective pressure than choosing a mate with good genes. Thus the mate choice issue may be a dual one - selection of the genetically best mate and avoidance of contamination. Interestingly, mate choice based on arbitrary secondary characteristics indicative of either genetic disease resistance or current pathogen-free status could lead to the same result. If a female chooses a male based on any arbitrary trait that is affected by overall vigor (e.g., antler size, vocal volume, tail length, bright colors), then that choice will be adaptive for disease avoidance and will simultaneously select for disease resistance genes. In this context it is not hard to see how the "sexual selection" process can become initiated, whereas it is harder to see how traditional sexual selection, based on purely sexual, nonadaptive characters, can become initiated (the difficulties are described by Kirkpatrick, 1986; Read, 1988).

If mate choice is affected by pathogen avoidance, then male choosiness should be exhibited in many cases and not just female choosiness. This could lead to reversed sexual displays (females brighter). Some such cases are observed in birds. It is even possible for both sexes to be choosy. In the context of female choice, it is possible that males might not remain entirely passive in their responses. Most studies that have been done have not been aimed at determining whether male mating is indiscriminate. G. Hepp in detailed studies of mate choice in black ducks (Hepp, 1989), observed (pers. comm.) that both sexes were choosey, even though males might have more than one mate. Edwards and Barnard (1987) found that male mice mated less often with females infected by Trichinella pathogens. Disease avoidance issues suggest that a more careful look at these behaviors is necessary. Some cases that are currently not considered may turn out to be related to this issue. For example, mate choice arguments generally are based on sexual dichromatism data, but if both sexes are choosy then both sexes may exhibit elaborate sexual displays. Examples could include the elaborate mating rituals of some birds such as cranes. Such displays would not be much affected by the increased predation pressure placed on brightly colored individuals. Vocal facility (singing, calling) is another such trait. These arguments suggest that the whole issue of choosiness needs reconsideration and that mutual choosiness could be a common phenomenon.

A difficulty with testing between sexual selection for disease resistance genes versus disease transmission prevention is that both could be operative and both lead to similar behavioral and secondary sex trait results. Certain evidence is available. Hamilton and Zuk (1982) observed that bisexual brightness and sexual dimorphism tend to decline on islands. They interpret this in terms of loss of parasite species upon colonization with consequent reduction in selective pressure for parasite resistance genes. This could, however, also reduce the necessity for avoiding contact with diseased individuals. Read (1988) cites data on interspecific correlation between male brightness in birds and species blood parasite load, a result consistent with both theories. Heavy parasite loads have also been shown to reduce mating success (Read, 1988). Read (1988) also notes that polygynous bird species are more brightly colored and also might have higher rates of sexually transmitted diseases. This link between mating system and coloration would tend to support the hypothesis that the animals are avoiding contamination rather than selecting for better genes for their offspring.

The comparison between fish and birds provides a possible test between the two hypotheses. Ward (1988) found greater relative male brightness (sexual dichromatism) in freshwater fish species that have more parasite species. For species that do not touch when mating, as in many oceanic fish, mate choice correlated with parasite load (if it exists) must be associated with selection for disease-resistance genes and not with disease avoidance. Many freshwater dichromatic species, however, do engage in extensive physical contact as part of their courtship behaviors (John Aho, U. of Ga. Ecology Laboratory, Pers. comm.). Such external contact could be even more dangerous for fish than sexual contact is for birds because skin diseases are major killers among fish. Data on extent of touching during courtship need to be compiled to test these two hypotheses. Another case that clearly supports the Hamilton-Zuk hypothesis is when animals exhibit bright coloration but only mate once, as in some salmon. In such cases there can be no disease avoidance because the fish die right after mating. Thus fish should provide key evidence for testing disease as a component of sexual selection theories.

Monogamy

It is not difficult to account for monogamy within a season because in most such cases both parents are involved in rearing the young. An altruism argument (based on the advantage of all current offspring being full-sibs) has been raised for the evolution of (female) monogamy even when the male does not help raise the young (Peck and Feldman, 1988) but their model does not apply to between-season monogamy unless the young stay around (as in primates). It is more difficult to account for long-term or lifelong monogamy, however, as documented for many bird species (Freed, 1987) and canids (Moehlman, 1987). There is also some evidence for long-term monogamy in small rodents (Foltz, 1981). Diamond (1987) discusses a hypothesis that animals will "divorce" to increase their reproductive success. Cited studies, all on birds, indicate that long-lived species tend to stay together and that the probability of "divorce" increases for pairs that fail to rear a brood the previous year. Reproductive failure makes it likely that a new mate will provide a better chance of reproducing. As for long-term monogamy, it is argued that considerable effort goes into achieving successful pairing. In this view, monogamous pairs benefit from better teamwork which takes time to build. For example, newly paired birds fledge fewer young than those paired longer (although no effect was found in Freed, 1987). I propose as a hypothesis that an additional benefit of monogamy could be the avoidance of sexually transmitted diseases (catalogued above). As is perfectly clear from AIDS, a guaranteed prevention mechanism for a sexually transmitted disease is lifetime monogamy. Consider a cohort of 20,000 of a species that breeds annually, lives for 15 years, mates once at random with another cohort member every year, and spreads (with 100% efficiency) a sterilizing sexual disease initially harbored by two individuals. By the end of the 15th breeding season, almost every individual will be sterile. For individuals breeding once a year but only breeding for five years, the initial two infected would only grow to 32 (from .1% of the total to .16%). Thus the potential importance of monogamy for disease prevention increases exponentially with lifespan. This is especially true with respect to diseases with a long latent period. A prediction from this result is that long-term monogamy should increase in frequency for long-lived species. It is also likely that sexually transmitted diseases will have more adverse consequences in species in which the mature stage is widely dispersing (group ≈ population) and mates over a period of years. This describes birds in particular, yielding the hypothesis that long-lived birds should exhibit greater monogamy than short-lived birds, and birds in general should be more monogamous than sedentary animals. Birds are in fact substantially more monogamous and exhibit greater long-term monogamy than other animals (Freed, 1987). In particular, almost all long-lived birds appear to largely forge long-term pair bonds. Canids are also very wide-ranging and notably monogamous. For many mammals, effective population sizes, and thus rates of spread for a sexual disease, are low (Chepko-Sade et al., 1987) compared to wide-ranging birds and canids. Comparison of incidence of sexually transmitted disease in birds and canids with other groups could help test these ideas.

Another phenomena that could be related to disease prevention is cohort fidelity. In some duck species, for example, when given a choice, individuals will preferentially mate with others from the same age cohort (Hepp 1989). This type of mating behavior, even in the absence of monogamy, could very effectively retard the spread of sexually transmitted diseases. Perfect cohort fidelity would be like a species with non-overlapping generations. No exclusively sexually transmitted disease is possible in such a case. Since cohort fidelity is not perfect, some spread to younger animals always occurs, but the role of disease suppression in the origin of cohort fidelity nevertheless deserves examination.

Polygamous associations need not always lead to higher risks from disease. Wild horses have a harem mating system. In a study by Berger (1987), young unmated females left their natal group and quickly joined a new group where they remained. Young males left to become solitary or join all-male groups. Young males who succeeded in taking over a harem were almost always sexually naive. There are few chances for sexual transmission of diseases in such a system. Thus in comparative studies care should be taken when classifying species. The horses studied were polygamous but not promiscuous. On the other hand, in some monkey troupes mating may be promiscuous within a group but only virgin males or females move between groups. Again, disease transmission in such a system is low. It is worth asking how much sexual disease transmission has been a factor governing whether those emigrating are virgins or not.

GROUP SIZE AND GROUP BEHAVIORS

An interesting long-standing question in behavioral ecology is what determines group size and interaction between groups in social species. The usual question asked is "why are species social?" since thought has focused on competition which clearly goes up in large groups. But given the ubiquity of social groups (even reptiles such as turtles and alligators bask in groups) it is perhaps also appropriate to ask "why is this species not social?" and "why do groups of this species maintain isolation from other groups?" Perhaps solitary living or rigid separation of groups from one another are not the "normal" behavior in all cases, but may reflect adaptations for disease avoidance. The framework for studying group behavior that is usually used is evaluation of costs and benefits at the individual level (e.g., Pulliam and Caraco, 1984). If the benefits of staying in a group are greater than the benefits of solitary life or if the risk due to inter-group movement is high, then the individual should remain in the group. Advantages of group living include detection of predators, defense against predators, increased hunting efficiency in predators, increased detection of patchy resources, and defense of resource patches from other groups of the same or other species. Costs of group living include inbreeding suppression of fitness for small groups, decreased per capita food availability or reproduction rates for large groups, and time spent in agonistic encounters, particularly in larger groups. Group living also has consequences for disease. In general, larger groupings will both harbor more pathogen types and be at greater risk of epidemics. Turning the question around, it is very difficult to think of cases where larger groups are more protected from pathogens than smaller groups. Some animals could alter their environment such that pathogens are less favored, but it is more likely that the opposite will prevail, particularly with respect to the huge amount of waste produced by a large group. One of the few ways a larger group could reduce pathogen effects is if food is shared. If an individual is sick, it will be less able to hunt and may starve. Food sharing may enable an individual to recover. Only in predators (e.g., wolves, lions) is such behavior observed (except for parents feeding their young). Information centers are an additional, though less effective form of food sharing. Specific allo-grooming also occurs, which could reduce disease risk. Primates groom each other's fur frequently (Freeland, 1981a; Goodall, 1986) as do many other species. Adults of many species lick the fur of their young often to keep them clean. Horses practice mutual tail flicking, in which two horses stand head to tail and use their tails to keep biting flies away from each other (Hughes et al., 1981). Maximal benefits of such mutual grooming are achieved at a small group size, however.

Transmission Paths and Consequences

Disease transmission may be an important factor in social behavior in those species that form relatively permanent groups. Complete lack of exchange of members to prevent disease transmission conflicts with exchanges for avoidance of inbreeding and movement of individuals to create groups that are more optimal for the individual with respect to food supply, mating, and predator avoidance. Two approaches are taken here for exploring this problem: 1) tracing the path of disease-related consequences for an individual who chooses to move to a new group or a group that accepts a new member, and 2) using an epidemiologic approach to study the population consequences of different rates of exchange (contact) between groups.

Disease transmission due to intergroup contact is a consequence of the dynamics of exchanges of members or contact between groups. In the limit, entire groups merge and separate, but my concern here is with relatively distinct groups because groups that combine and break up frequently behave almost like a continuous larger population. It is necessary to consider group exchanges from the point of view of the individual. The decision to stay or leave is determined by the balance between costs and benefits. There are several benefits to leaving the natal group. It may be overcrowded. Young males may have a better chance of achieving dominance in another group than in their natal group, as has been observed for some baboons. For small groups, inbreeding suppression of fitness and lack of mates due to incest taboos and unbalanced age and sex ratios may favor emmigration.

The costs of emmigrating may also be high, with some of the costs due to disease (Fig. 1, Tab. 2). During solitary travel between groups, some types of animals may be at increased risk of predation. Once it finds a new group, it must be accepted by it. Rejection leads to further solitary travel and continued predation risk, or a return to the original group. Rejection by the new group results from social barriers to immigrants, mainly aggression, that may arise when the receiving group is too overcrowded or because of selection to reduce disease by limiting contacts between groups. As noted above, isolated small groups will be relatively protected from epidemics. If aggressive reactions to immigrants preferentially discourage the weak, who may be differentially loaded with or susceptible to pathogens, then the group also protects itself directly from disease. Once the immigrant is accepted, it may catch a disease or get a parasite from members of the new group, to which it is not adapted or to which it has no antibodies, thereby killing it or reducing its fitness. Conversely, the immigrant may bring pathogens with it. This could have several consequences. The new group may be destroyed, forcing the immigrant to move again. Short of this, an epidemic can reduce vigor of the group such that predation risk on all group members (including the immigrant) is increased due to decreased alertness of group members. Introduced diseases can also kill potential mates or offspring of the immigrant, thereby reducing its fitness. Finally, in an epidemic situation, secondary diseases may gain a foothold and spread to the original immigrant. If none of these consequences occurs, the individual may be said to have successfully changed groups.

Disease transmission between groups can also result from other types of contacts besides exchanges of members. A prominent mechanism is territory defense. If individuals or groups defend a territory with physical contests between neighbors then disease transmission may be enhanced, particularly if biting occurs. Rabies is a prominent example of a disease transmitted by bites, in particular by foxes who defend their territories by direct contact. Many animals have territorial defenses that emphasize visual or vocal displays rather than physical contact, e.g. wolves, song birds. Such non-contact defenses not only prevent injury, but may also reduce the risk of disease spread.

Epidemiology

The effect of a disease will depend on its virulence, duration of host immunity, transmissibility, host population growth rate, and length of infective period (May and Anderson, 1979a; b). The longer the infective period, the more individual contacts will be made and the more chances for transmission. Higher virulence increases transmission up to a point, but can also reduce the transmission rate by killing animals faster which reduces the time available for transmission. Below a certain population density, there are not enough inter-individual contacts to yield a sufficiently high transmission rate for an epidemic to occur (May and Anderson, 1979a; b). This threshold level can be designated N_T. At first thought, it might seem logical to propose that group sizes should evolve so as not to exceed N_T for any serious diseases that might be acting on the population.

Consideration of the distribution of social groups throws some light on whether groups have evolved for sizes below N_T. A local population partitioned into small groups does not have a uniform transmission rate, but two: within a group and between groups. Many animals within social groups live physically very close to one another, such as lion prides and den-dwelling family groups, such that they sleep atop one another, nip at each other, wrestle, and lick each other's fur. This physical proximity means that disease transmission within the group is extremely efficient. Studies of human measles have found high rates of spread within the family relative to between families, though the family size is well below N_T. Fox rabies exhibits a similar pattern. For such diseases social living will decrease effective N_T. On the other hand, many social groups that are not as physically close (no fur licking, occupation of separate burrows as in a prairie dog colony) may not have such a high transmission rate. For example, for measles, May and Anderson (1979a) estimate, for a poorly fed population with case-mortality of 30% and per annum disease-free growth rate of 3%, N_T = 380,000 people. This is far above the group size for most social species if there is no exchange of members between groups or local populations. For herding ungulates and some birds, however, group size may be well over N_T, such that selective pressures due to disease mortality could exist.

Since epidemics are largely eliminated if a population is broken into small isolated subpopulations, the question of group isolation arises. There is a complete continuum from species whose groups never exchange members to those in which groups form and disperse at will, such as foraging flocks of some bird species. There are also species that come together in huge flocks for breeding and are solitary or in small groups the rest of the year. Is disease transmission a factor in determining group isolation and/or group size?

To study potential barriers to disease spread, epidemiologic models can be constructed. At one extreme are populations that are fully mixed with no barriers. At the other extreme, the population may consist of small groups that are completely isolated. As groups get larger, (which increases intragroup transmission) how much must intergroup transmission go down to prevent an epidemic? Or: For a constant group size, as virulence or transmissibility go up, how much must intergroup transmission go down to prevent an epidemic? Finally, at what group size does disease become a serious cost? Such questions can be asked using epidemiologic models with a spatial component. A fully general model is not easy to develop because so much depends on the particulars of the disease, spatial pattern of groups, and social behavior, leading to a multivariable analysis. N_T will not be simply the group size, but rather a function of the frequency of inter-group contact and extent of contact between each group. Such an analysis will not be performed here.

An interesting aspect of many diseases is that a little contact may lead to immunity whereas no contact leaves the individual susceptible to a severe case during an epidemic. During the chronic phase of many diseases, an individual may come into contact with doses of the pathogen sufficient to generate an immune response but insufficient to cause the disease. This is particularly so in a healthy population. An individual is most likely to encounter these vaccinating conditions if it is in a large enough group for most diseases of the population to be endemic in the group. Animals in such a group will be more protected during an epidemic or an unfavorable season than those in groups so small that most diseases are novel. This is a case where larger groups may provide protection instead of increased risk.

Dominance Hierarchies

Dominance hierarchies are established in most social groups. Subordinate animals are typically forced to the periphery of the group or to more exposed sites (e.g., Ekman, 1987), where they experience increased predation since predators typically attack from the periphery (Hamilton, 1971; Pulliam and Caraco, 1984). The advantage to the dominants is both increased access to food via location and decreased time spent in predator surveillance, and decreased predation risk. These benefits presumably compensate for the costs of maintaining dominance. For the subordinate, the benefit of staying in the group (e.g., increased foraging success relative to solitary foraging, Baker et al., 1981) has traditionally been assumed to outweigh the increased risk of predation on the periphery, otherwise it would leave (Pulliam and Caraco, 1984). However, a subordinate bird will not necessarily improve its lot by seeking another flock because it is likely to be subordinate in the new flock as well. Solitary travel in search of a new group may be particularly risky (though in wild horses solitary travel is not hazardous (Berger, 1987), at least under current conditions). If the choice is between subordinate status and solitary living, then the individual also benefits from staying. Such individuals may also benefit from group membership by having a future probability of less subordinate status, as well as by learning foraging skills.

A complicating factor in dominance hierarchies is the role of pathogens. In competitive contests, dominance usually goes to the more energetic, aggressive, and larger individual. While not all weak individuals are sick, it is certainly true that most sick individuals are weak and may also be smaller due to weight loss or growth suppression. Thus individuals with diseases or heavy parasite loads should differentially lose dominance contests, as demonstrated for mice by Freeland (1981b), Rau (1984) and others, and be forced to the edge of the group. Subordinate individuals also emmigrate more. The peripheral area, where predators concentrate, will thus contain a higher proportion of diseased individuals. Of those under attack, diseased individuals are easier prey because they are less alert, slower, and put up less resistance. Thus that component of the peripheral population made up of diseased individuals will suffer a disproportionate share of mortality. Any reduction in the number of diseased individuals will also reduce the risks of infection for the remaining group members. If the activities of a dominant individual force diseased individuals to the edge of the group where they suffer greater mortality or disperse, then the dominant individual has increased its own fitness by reducing its risk of contracting disease, as well as by diverting predation pressure to others. In addition, it has created the maximum possible physical distance between itself and diseased individuals, thereby further reducing its own risk of contamination even if the infected animals are not killed. Thus fitness benefits resulting from reduction in pathogen transmission could favor the evolution of social structures such as dominance hierarchies. Conversely, dominance hierarchies evolved for other reasons could have beneficial disease consequences.

Another effect of dominance hierarchies relates to intergroup member exchanges. If dominant individuals tend to drive off the sick, then the pool of unattached individuals attempting to join a new group may be disproportionately diseased, increasing selection pressure for xenophobic behavior by the receiving group. Aggressive interactions with immigrants could drive away those less vigorous individuals who are more likely to be diseased, whereas healthy individuals would be more likely to successfully immigrate by overcoming initial aggressive reactions. This effect has been observed often in primates when unattached males attempt to join a group. In species with harems, only the most vigorous, and probably disease free, males are able to take over from the resident male.

Movement Patterns

Animal movement can have significant disease consequences. Animals in some cases may relocate to avoid pests. Colonial birds sometimes relocate their nests or even the rookery when nest parasites become too common (Brown and Brown, 1986). Baboon troops have been observed to relocate when their sleeping area becomes fouled and to return at intervals sufficient for nematode parasites to return to low levels (Hausfater and Meade, 1982). Freeland (1980) observed that mangabeys (Cercocebus albigena) in Uganda contaminate their immediate surroundings with feces, including walking and sleeping surfaces and vegetation that might be eaten. Although they did not specifically avoid feces, they were observed to remain in one location longer during and after rains, when feces were washed away, than during dry periods. Inactivity during rain was ruled out as an explanation.

A more subtle effect results from migration. Consider a species that migrates seasonally some large distance. An area that becomes fouled and unsanitary during one season will become largely clean again by the following year. In between, nest and skin parasites such as ticks and fleas will have nothing to eat and will be greatly reduced in number. For example, wide spacing in rabbits results in reduced flea numbers (Mohr, 1963) for these same reasons. Excrement piles will decompose between seasons and wash away. Significant benefits could thus accrue to migratory species, even though long distance migratory behavior is not likely to arise from this cause alone (it may result from predator avoidance (Fryxell et al., 1988), or the usual weather and food supply factors). Abandonment of rookeries during the non-mating season could, however, result from this effect. Another consequence of migratory behavior is the effect on internal parasites. Flukes, tapeworms, and similar parasites often require an intermediate host. Such arrangements are often very specific. The more widely a host species ranges, particularly seasonally, the more difficult it will be for parasites to locate appropriate intermediate hosts or to persist in the different parts of the range. External parasites must then produce more young or persist longer (Lundqvist, 1988) to find a suitable host. These effects will be greatest if the host rarely returns to the same exact location in successive years. Transovarian pathogen transmission in the vector as in ticks and mites (Service, 1986) is a mechanism by which the pathogen can persist for several generations without a host. Consideration of epidemiology thus leads to some unexpected benefits of migratory behavior. On the other hand, wide-ranging species will encounter more individuals of and species of pests and parasites (Mohr, 1963; Mohr and Stumpf, 1964), as will those with low habitat and diet specificity (Kennedy et al., 1986).

Mixed-Species Groupings

A curious phenomenon is the occurrence of mixed-species groups. It has been argued that mixed-species herds in, e.g., the Serengetti favor predator protection by combining species with different predator detection modalities (e.g., sight, hearing, smell) together. While this has logical appeal and may be true, mixed groups also occur where the species are very similar. A disease avoidance model may have some applicability. While some diseases are pandemic (e.g., rinderpest and avian influenza) most are species specific or can attack only closely related species (Freeland, 1983; Stock and Holmes, 1987). For such pathogens, an animal in a mixed-species group will have reduced contact with conspecifics, and thus lower disease contagion risk, compared to an animal in a conspecific group of the same size, while still obtaining the same predator detection benefits of a larger group. The fact that mixed species bird flocks and animal herds are most common in the tropics is consistent with this hypothesis. Mixed species flocks are also found in deciduous forests in winter (Grubb, 1975; 1978) when disease is a more serious threat. Note that no high degree of coevolution is required because any combination of 2 or more different species will provide the same protection. This is consistent with the observation that the species mix in these types of groups is in constant flux. The greater the differences in size, phylogeny, diet, etc. among the species the less transmissible diseases and parasites are likely to be (Freeland 1983) and thus the greater the benefit of mixed species groups with respect to disease.

A detailed case study by Freeland (1977) of monkeys in Uganda adds support to this model. He correlated multispecies group formation with times of day when flies and mosquitos are more prevalent, namely dawn and dusk. The timing of association was unrelated to eagle attacks or to foraging activity, nor was there a shortage of sleeping sites. Biting insects in these forests carry yellow fever, monkey malaria (Hepatocystis kochi), and other diseases that cause significant mortality in these monkeys. Freeland (1977) noted experiments showing that monkeys in groups get fewer bites than single monkeys (as also demonstrated for horses, Duncan and Vigne, 1979), as expected if insects are distributed randomly in space, and hypothesized that troops of different species come together to reduce bites and therefore to decrease disease risk. A permanent increase in troop size would achieve the same thing but might be disadvantageous for other reasons such as being inflexible with respect to food supply. An advantage of mixed species groups not noted by Freeland is the benefit of being bitten by an insect that has previously bitten an individual of a different species carrying a disease to which the victim is not susceptible. Transmission of many diseases by biting insects is most efficient when two or more individuals are bitten in a short period of time (Service, 1986). In Freeland's study several of the diseases affect all monkey species (e.g., yellow fever) but some of them are species specific. Thus both reduced numbers of bites and reduced transmission due to incompatible host diseases could contribute to these multispecies groupings.

Seasonal Effects

Many species are migratory, particularly birds. It is notable that degree of sociality varies by species. Many are solitary or form small groups except during the breeding season, while for others the converse is true. Not all variations in degree of sociality are associated with migration. While it is evident for many species that aggregation during the breeding season is a direct consequence of the scarcity of appropriate sites (e.g., colonial seabirds nesting where predators are scarce), this may not be the complete story. It is reasonable to postulate seasonal effects on degree of sociality that result from disease. In temperate regions winter is a particularly high risk period for disease. Animals are stressed by cold and inadequate nutrition. In addition, many highly contagious diseases (influenzas and other respiratory diseases) are particularly common in cold weather. It might, then, be advantageous for animals to avoid large flocks or herds during the winter to decrease the risk of epidemics. Even if disease risk is constant over the year, a species might benefit from solitary or small group life during the entire year except the breeding season. Evidence for disease as a factor in the evolution of seasonal sociality can be obtained in several ways. Disease-induced mortality can be compared between solitary and gregarious times of year. Seasonal variation in disease in species that are gregarious all year can be determined. Finally, artificially large groupings at normally solitary seasons can be compared for disease rate. For example, efforts by wildlife managers to supplement food supplies of species such as ducks, geese, deer, and elk may result in unnaturally large groupings at certain seasons which could be studied for epidemics.

CONCLUSIONS

Past treatments of the role of disease and pathogens have suffered from viewing them as agents with a passive effect: animals get sick and then they die. My argument is that the disease cycle has a weak link at the point of transmission. Certain social behaviors and systems lead to reduced pathogen transmission and thus may be selected for. Such selective pressures will not exist for ubiquitous pathogens. Not all behaviors that reduce pathogen transmission have necessarily evolved for that reason, but the possibility of such an origin for many traits deserves further exploration.

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LITERATURE CITED

Alexander, R. D. 1974. The evolution of social behavior. Ann. Rev. Ecol. Syst. 5:325-383.

Anderson, R. M. and R. M. May. 1979a. Population biology of infectious diseases: Part I. Nature 280:361-367.

Anderson, R. M. and R. M. May. 1979b. Population biology of infectious diseases: Part II. Nature 280:455-461.

Baker, M. C., C. S. Belcher, L. C. Deutsch, G. L. Sherman and D. B. Sherman. 1981. Foraging success in junco flocks and the effects of social hierarchy. Anim. Behav. 29:137-142.

Ball, F. G. 1985. Spatial models for the spread and control of rabies incorporating group size. pp. 197-222. *In*: P. J. Bacon (ed.) Population Dynamics of Rabies in Wildlife. Academic Press, NY.

Barton, R. 1985. Grooming site preferences in primates and their functional implications. Int. J. Primatol. 6: 519-532.

Bell, J. F. and C. M. Clifford. 1964. Effect of limb disability on lousiness in mice. **II.** Intersex grooming relationships. Exp. Parasitol. 15: 340-349.

Berger, J. 1987. Reproductive fates of dispersers in a harem-dwelling ungulate: the wild horse. p. 41-54 in: B. Chepko-Sade and Z. T. Halpin, eds., Mammalian Dispersal Patterns. U. of Chicago Press, Chicago.

Borg, K. 1987. A review of wildlife diseases from Scandanavia. J. Wildl. Disease 23: 527-533.

Brown, C. R. and M. B. Brown, 1986. Ectoparasitism as a cost of coloniality in cliff swallows (Hirundo pyrrhonota). Ecology 67:1206-1218.

Chepko-Sade, B. D., W. M. Shields, J. Berger, Z. T. Halpin, W. T. Jones, L. L. Rogers, J. P. Rood, and A. T. Smith. 1987. The effects of dispersal and social structure on effective population size. p. 287-321 In: Chepko-Sade and Z. T. Halpin (eds.) Mammalian Dispersal Patterns. U. of Chicago Press, Chicago.

Clark, L. and J. R. Mason. 1985. Use of nest material as insecticidal and anti-pathogenic agents by the European starling. Oecologia 67: 169-176.

Diamond, J. M. 1987. A darwinian theory of divorce. Nature 329:765-766.

Duffy, D. C. 1983. The ecology of tick parasitism on densely nesting Peruvian seabirds. Ecology 64:110-119.

Duncan, P. and N. Vigne. 1979. The effect of group size in horses on the rate of attacks by blood-sucking flies. An. Behav. 27:623-625.

Edman, J. D., L. A. Weber, and A. A. Schmid. 1974. Effect of host defenses on the feeding pattern of *Culex nigripalpus* when offered a choice of blood sources. J. Parasitol. 60: 874-883.

Edwards, J. C., and C. J. Barnard. 1987. The effects of *Trichinella* infection on intersexual interactions between mice. Anim. Behav. 35: 533-540.

Edwards, J. C. 1988. The effects of *Trichinella spiralis* infection on social interactions in mixed groups of infected and uninfected male mice. Anim. Behav. 36: 529-540.

Ekman, J. 1987. Exposure and time use in willow tit flocks: the cost of subordination. Anim. Behav. 35:445-452.

Foltz, D. W. 1981. Genetic evidence for long-term monogamy in a small rodent, *Peromyscus polionotus*. Am. Nat. 117: 665-675.

Foltz, D. W. and J. L. Hoogland. 1983. Genetic evidence of outbreeding in the black-tailed prairie dog (*Cynomys ludovicianus*). Evolution 37:273-281.

Freed, L. A. 1987. The long-term pair bond of tropical house wrens: Advantage or constraint? Am. Nat. 130:507-525.

Freeland, W. J. 1976. Pathogens and the evolution of primate sociality. Biotropica 8:12-24.

Freeland, W. J. 1977. Blood-sucking flies and primate polyspecific associations. Nature 269:801-802.

Freeland, W. J. 1979. Primate social groups as biological islands. Ecology 60:719-728.

Freeland, W. J. 1980. Mangebey (*Cercocebus albigena*) movement patterns in relation to food availability and fecal contamination. Ecology 61:1297-1303.

Freeland, W. J. 1981a. Functional aspects of primate grooming. Ohio Acad. Sci. 81:173-177.

Freeland, W. J. 1981b. Parasitism and behavioral dominance among male mice. Science 213:461-462.

Freeland, W. J. 1983. Parasites and the coexistence of animal host species. Am. Nat. 121:223-236.

Fryxell, J. M., J. Greever, and A. R. E. Sinclair. 1988. Why are migratory ungulates so abundant? Am. Nat. 131: 781-798.

Goodall, J. 1986. The Chimpanzees of Gombe. Harvard University Press, Cambridge.

Hamilton, W. D. 1971. Geometry for the selfish herd. J. Theor. Biol. 31: 295-311.

Hamilton, W. D. and M. Zuk. 1982. Heritable true fitness and bright birds: A role for parasites? Science 218:384-387.

Hart, B. L. and C. M. Haugen. 1971. Prevention of genital grooming in mating behavior of male rats (*Rattus norvegicus*). Anim. Behav. 19: 230-232.

Hart, B. L. 1985. The behavior of domestic animals. New York: Freeman.

Hart, B. L. 1987. Behavior of sick animals. Veterinary clinics of North America: Food Animal Practice 2:383-391.

Hart, B. L., E. Korinek, and P. Brennan. 1987. Postcopulatory genital grooming in male rats: Prevention of sexually transmitted infections. Physiol. Behav. 41: 321-325.

Hart, B. L. 1988. Biological bases of the behavior of sick animals. Neurosci. Biobehav. Rev. 12: 123-137.

Hart, B. L. Submitted. Animal behavior and disease management: Five strategies. Neurosci. and Biobehav. Rev.

Hausfater, G. and B. J. Meade. 1982. Alternation of sleeping groves by yellow baboons (*Papio cynocephalus*) as a strategy for parasite avoidance. Primates 23: 287-297.

Hepp, G. R. 1989. Benefits, costs, and determinants of dominance in American black ducks. Behavior In Press.

Hoogland, J. L. 1979. Aggression, ectoparasitism, and other possible costs of prairie dog (Sciuridae, *Cynomys* spp.) coloniality. Behavior 69:1-35.

Hoogland, J. L. 1981. The evolution of coloniality in white-tailed and black-tailed prairie dogs (Sciuridae: *Cynomys leucurus* and *C. ludovicianus*). Ecology 62:252-272.

Hughes, R. D., P. Duncan, and J. Dawson. 1981. Interactions between Camargue horses and horseflies (*Diptera: Tabanidae*). Bull. Ent. Res. 71:227-242.

Hutson, J. M., M. Nial, D. Evan, and R. Fowler. 1979. Effect of salivary glands on wound contraction in mice. Nature 279: 793-795.

Keiper, R. R. and J. Berger. 1982. Refuge-seeking and pest avoidance by feral horses in desert and island environments. Appl. Anim. Ethol. 9:111-120.

Kennedy, C. R. 1978. The parasite fauna of resident char *Salvelinus alpinus* from Artic islands, with special reference to Bear island. J. Fish Biol. 13:457-466.

Kennedy, C. R., A. O. Bush, and J. M. Aho. 1986. Patterns in helminth communities: why are birds and fish different ? Parasitology 93:205-215.

Kennedy, C. E. J., J. A. Endler, S. L. Poynton, and H. McMinn. 1987. Parasite load predicts mate choice in guppies. Behav. Ecol. Sociobiol. 21: 291-295.

Kirkpatrick, M. 1986. Sexual selection and cycling parasites: A simulation study of Hamilton's hypothesis. J. theor. Biol. 119: 263-271.

Kirkpatrick, M. 1989. Sexual Selection: Is bigger always better? Nature 337: 116-117.

Kluger, M. J. 1979. Fever. its biology, evolution and function. Princeton: Princeton University Press.

Lundqvist L. 1988. Reproductive strategies of ectoparasites on small mammals. Can. J. Zool. 66:774-781.

Mandel, I. D. and S. A. Ellison. 1981. Naturally occurring defense mechanisms in saliva. In Tanzer, J. M., ed. Special Suppl. Microbiology Abstractrs: 367-379.

May, R. M. 1983. Parasitic infections as regulators of animal populations. Am. Sci. 71:36-45.

May, R. M. and R. M. Anderson. 1987. Transmission dynamics of HIV infection. Nature 326:137-142.

May, R. M. 1988. Conservation and disease. Conserv. Biol. 2: 28-30.

Mettrick, D. F. 1987. Parasitology: today and tomorrow. Can. J. Zool. 65:812-822.

Moehlman, P. D. 1987. Social organization in Jackals. Am. Sci. 75:366-375.

Mohr, C. O. 1963. Relation of flea infestations to spacing between cottontail rabbits. J. Wildl. Manage. 27: 71-76.

Mohr, C. O. and W. A. Stumpf. 1964. Relation of tick and chigger infestations to home areas of California meadow mice. J. Med. Ent. 1: 73-77.

Moore, J., D. Simberloff, and M. Freehling. 1988. Relationships between bobwhite quail social-group size and intestinal helminth parasitism. Am. Nat. 131:22-32.

Murray, J. D. 1987. Modeling the spread of rabies. Am. Sci. 75:280-284.

Murray, J. D., E. A. Stanley, and D. L. Brown. 1986. On the spatial spread of rabies among foxes. Proc. R. Soc. Lond. B. 229:111-150.

Peck, J. R. and M. W. Feldman. 1988. Kin selection and the evolution of monogamy. Science 240:1672-1674.

Pulliam, H. R. and T. Caraco. 1984. Living in groups: Is there an optimal group size? p. 122-147 In: J. R. Krebs and N. B. Davies (eds), Behavioral Ecology: An Evolutionary Approach, 2nd Edition. Sinauer Assoc. Inc., Sunderland, MASS.

Rau, M. E. 1984. Loss of behavioral dominance in male mice infected with *Trichinella spiralis*. Parasitology 88: 371-373.

Read, A. F. 1988. Sexual selection and the role of parasites. TREE 3:97-102.

Schwabe, C., H. P. Rieman, and C. E. Franti. 1977. Epidemiology in Veterinary Practice. Pub ____________.

Service, M. W. 1986. Blood-Sucking Insects: Vectors of Disease. Edward Arnold, London.

Stock, T. M. and J. C. Holmes. 1987. Host specificity and exchange of intestinal helminths among four species of grebes (Podicipedidae). Can. J. Zool. 65:669-676.

Taylor, E. L. 1954. Grazing behavior and helminitic disease. Brit. J. Anim. Behav. 2: 61-62.

Terborgh, J. and C. H. Janson. 1986. The socioecology of primate groups. Ann. Rev. Ecol. Syst. 17:111-135.

Thomas, R. J. 1953. On the nematode and trematode parasites of some small mammals from the Inner Hebrides. J. Helminthol. 28:153-168.

Ward, P. I. 1988. Sexual dichromatism and parasitism in British and Irish freshwater fish. Anim. Behav. 36: 1210-1215.

Warner, R. E. 1968. The role of introduced diseases in the extinction of the endemic Hawaiian avifauna. Condor 70:101-120.

Wimberg [illegible] 1984. The use of green plant material in bird nests to avoid ectoparasites. Auk 101: 615-618.

Witter, J. F. 1981. Brucellosis. pp. 280-287, in: J. W. Davis, L. H. Karstad, and D. O. Trainer (eds). Infectious diseases of wild animals. Iowa State U. Press, Ames.

Yuill, T. M. 1981. Myxomatosis and fibromatosis. pp. 154-177, in: J. W. Davis, L. H. Karstad, and D. O. Trainer (eds). Infectious diseases of wild animals. Iowa State U. Press, Ames.

Zeh, D. W. and J. A. Zeh. 1988. Condition-dependent sex ornaments and field tests of sexual-selection theory. Am. Nat. 132: 454-459.

Table 1. Pathogen and pest types as related to group social behavior.

Pathogen Type	Effectiveness of Group Isolation
Pathogens with airborne vectors (e.g., mosquitos)	Effective only with large distances, but fewer bites/individual in larger groups.
Pathogens with limited dispersal vectors (e.g., fleas)	Effective
Pathogens with persistent vectors (e.g., ticks) or persistent dispersal stages (e.g., inside prey)	Ineffective
Contact pathogens (by bite, in body fluids)	Effective, unless pathogen carried by many host species (e.g., rabies, rinderpest).
Sexually transmited diseases	Long-term isolation required, cohort mating fidelity effective.
Pathogens of filth (excrement associated diseases and macroparasites)	Isolation effective and larger groups have more pathogen species. Sanitary behaviors helpful.
External macroparasites and pests - wide ranging or persistent (e.g., flies, mosquitos, ticks)	Ineffective, but reduced per capita damage in larger groups (Freeland 1977; Hughes et al. 1981) and habitat selection can be effective (Hughes et al. 1981; Keiper and Berger 1982).
External macroparasites and pests - contact distributed (e.g., fleas, lice)	Effective, but increase in larger groups up to certain size (?)
Monoxenous parasites	Effective, increased intensity in larger groups (Moore et al. 1988).
Heteroxenous parasites	Ineffective, little relationship to group size (Moore et al. 1988).

See Schwabe et al. (1977) and Service (1986) for more detail on vectors and transmission mechanisms.

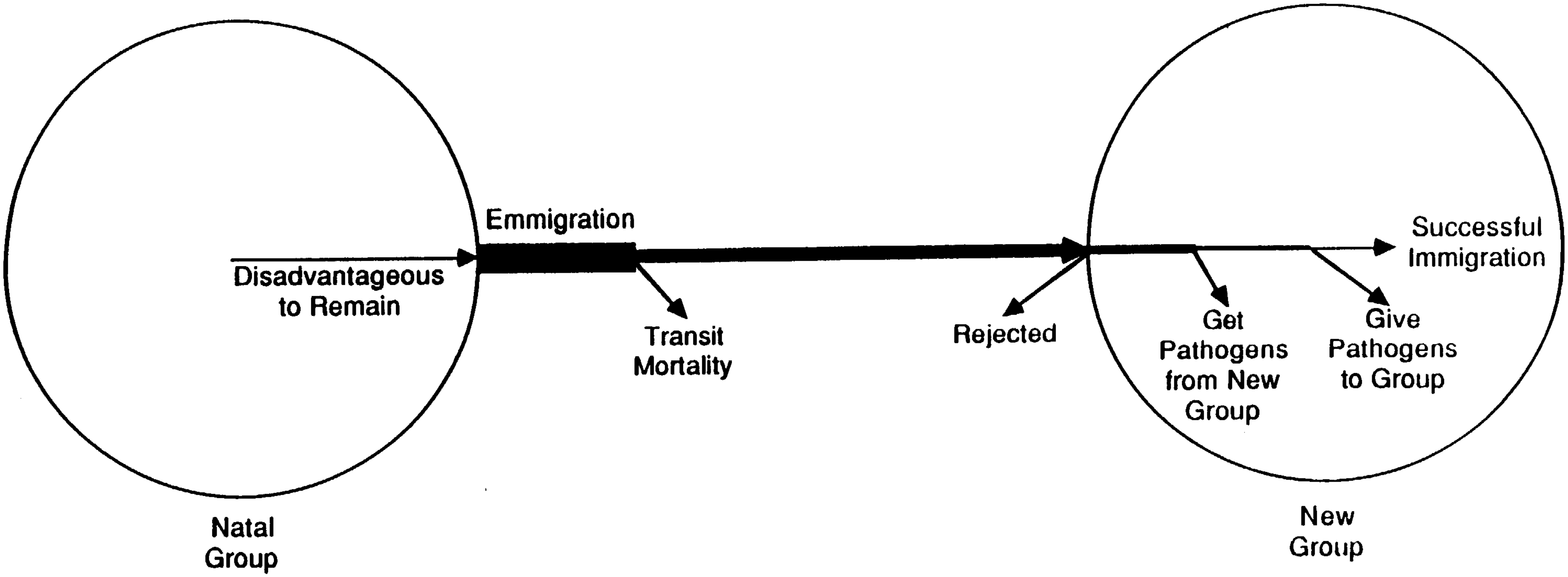
Table 2. Costs and benefits of emigration from natal group as influenced by disease.

Costs of staying	1. Inbreeding suppression of fitness.
	2. Lack of mates due to incest taboo and unbalanced age and sex ratios.
	3. Overcrowding.
	4. Difficulty of dominance (for males) in natal group.
Costs of leaving:	1. Increased mortality of emigrants during migration due to lack of social supports.
	2. Possible rejection by new group (increased if selection for reduced inter-group exchange to reduce disease).
	3. Chance of getting pathogens from new group.
	4. Chance of carrying disease to new group. Consequences can be:
	A. New group wiped out, immigrant again must seek new group and solitary travel risks.
	B. Potential mates killed. Fitness now zero.
	C. Imported pathogens reduce survival or vigor of offspring. Fitness of parent thereby reduced.
	D. Pathogen spread weakens group members, yielding:
	a. Increased predation risk due to decreased alertness or smaller group size.
	b. Spread of other diseases (secondary epidemic), to which immigrant may be susceptible.

Table 3. Potential effects of disease transmission on social groups.

1. Limit group size to below effective epidemic size. Group size may therefore be below that at which predator defense or resource exploitation is optimal.
2. Reduce frequency of groups coalescing into larger groups.
3. Reduce emigration.
4. Increase group aggression against immigrants.
5. Increase inter-group distance.
6. Increase long term (inter-year) monogamy, particularly for sexually transmitted diseases for which there are long term carriers (e.g., males asymptomatic) or where disease has long, slow action (e.g., herpes, AIDS, syphilis).

Figure 1. Emmigration and disease. In addition to the usual mortality risk of emmigration, an emmigrant may be rejeted, catch a disease from the new group, or transmit a disease to the new group with various consequences (see Table 2).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