

## PARASITES IN A GLOBAL VILLAGE: FOOD, EMERGING DISEASES, AND EVOLUTION

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**ABSTRACT:** International trade and travel has resulted in increasing movement of people, livestock or other animals, and food products in recent decades, and it has also indirectly resulted in the global spread of parasites and diseases. The potential impact of parasite introduction on human health or food production is a cause for concern. There is evidence that some factors (host specificity, complex life cycles, saturated communities) long believed to prevent the spread and successful establishment of parasites in new areas are unlikely to be efficient barriers. From an epidemiological perspective, the fate of a parasite after introduction will depend on its basic reproductive number,  $R_0$ , and how the parameters that influence it are modified by the introduction. When introductions bring immunologically naive hosts in contact with parasites, the effective host population size or density is increased, as is  $R_0$ , and an epidemic outbreak is likely. In addition, an increase in effective host population size can relax constraints on parasite-induced host mortality, i.e., virulence. Therefore, two of the most important consequences of large-scale and frequent movement of animals or parasites are that disease outbreaks are facilitated and that the new conditions may promote the evolution of more virulent pathogens. These are challenges that need to be addressed quickly to ensure a sufficient food supply and to minimize health risks for our growing population.

### THE GLOBAL BIOLOGICAL VILLAGE

From a biological perspective, our world is rapidly becoming a single planet-wide community. Increase in international trade and travel has led directly to mass movement of people, livestock or other domestic animals, and food products, and indirectly to the global spread of disease vectors and parasites. Anywhere you go these days, you see the same species of crops, trees, flowers, and domestic animals; you often even hear the same birds singing in the trees. The increasing homogeneity of the biological community is not only seen on a global scale, but monocultures are also the norm in local agricultural areas worldwide. Human activities are therefore transforming the earth into a giant ecological community by increasing movement and exchange of organisms between geographical areas that are themselves losing their ecological heterogeneity.

At the same time, there is an increasing incidence of emerging infectious diseases in animal populations worldwide (see Daszak et al., 2000; Cleaveland et al., 2001; Friend et al., 2001). Although part of this increase may be simply due to the fact that interest in (and monitoring of) diseases is greater than before, the underlying trend is most likely real. Emerging infectious diseases can be broadly defined as infectious diseases whose geographical range, host range, or prevalence (or all) are currently increasing. They are frequently associated with changing climatic conditions or environmental degradation, which are often induced by anthropogenic factors (Harvell et al., 1999, 2002; Patz et al., 2000; Marcogliese, 2001). However, they also result from large-scale animal movement and the introduction of pathogens or their vectors (or both) to new areas despite quarantine and surveillance procedures (Dobson and Foufopoulos, 2001; Lounibos, 2002). Because of the mass movement of people and livestock today, we are potentially facing huge problems of disease outbreaks, whose impact on food production and quality (and therefore human health) can be disastrous. The recent epidemic of foot-and-mouth disease in cattle across Britain is a good illustration of the potential consequences of such outbreaks for food production and agro-economy (Keeling et al., 2001; Paarlberg et al., 2002).

Here, I will briefly explore the relationship between parasitic diseases and the mass movement of people and animals in our global village. When possible, I will focus mainly on parasites that affect food production or on food-borne human parasites

because they pose the most immediate threat to our expanding population. I will first discuss why some fundamental features of parasites and parasite communities, often accepted in the literature as barriers against the spread of parasites to new areas or new hosts, are not necessarily going to be effective. Then, I will present an overview of how the current conditions, in which large-scale movement of hosts and parasites have increased, may affect the epidemiology and evolution of parasitic diseases.

### THE GLOBAL SPREAD OF PARASITES

When a group of hosts is introduced into a new geographical area, they may be uninfected by certain parasites for a variety of reasons. If all host movements were limited to 1-off introductions, founder effects would restrict the range of parasites accompanying them. For example, when a few hundred brush-tail possums, *Trichosurus vulpecula*, were introduced from Australia to New Zealand in the late 19th century, they left behind most of their parasite species (Stankiewicz et al., 1997). However, with the volume of animal and human movement today, parasites are bound to follow. For instance, each year, over 500 million people cross international borders aboard commercial airplanes alone (Cetron et al., 1998) and about 15 million refugees are displaced, usually permanently, to other countries (Cookson et al., 1998). Numbers like these guarantee that parasites will follow their human hosts. Movements of animals, whether domestic or wild, do not match these figures but are still important enough to cause the spread of parasites, as seen in the many documented parasite introductions mediated by human activities and involving hosts ranging from marine invertebrates (Torchin et al., 2002) to cattle (Angus, 1996).

The introduction of parasites to new geographic areas will not necessarily result in a disease problem. Some features of parasites or their communities are often presented in textbooks or in the general parasitology literature as fixed traits that would not allow a parasite to thrive under altered transmission conditions. I will consider 3 such features here. First, parasitological wisdom suggests that it may be difficult for a parasite species to persist in a new area if its original host is also not present in sufficient numbers or if there is no alternative host to which it is adapted. Parasites are generally thought to be highly host specific, i.e., only capable of reproducing and persisting locally in a narrow range of host species. Second, helminth parasites

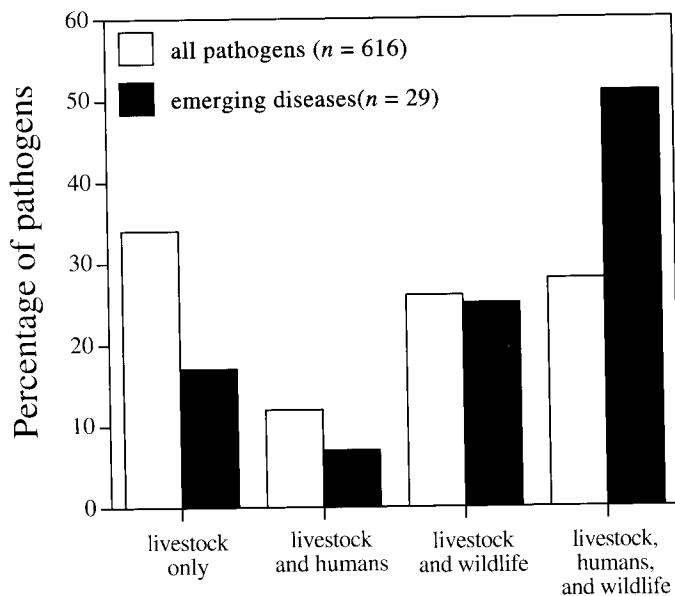


FIGURE 1. Range of known host species of pathogens of livestock; only 1 pathogen species out of 3 is specific to livestock, and only 1 out of 6 emerging diseases of livestock is caused by a pathogen specific to livestock (data from Cleaveland et al., 2001).

with complex life cycles are often thought to be constrained by the need to develop in a strict sequence of host species; in the absence of just 1 of these required hosts, the parasites become locally extinct. Third, there have been many suggestions that undisturbed or equilibrium parasite communities are often saturated with species. This has been proposed for endoparasitic helminth communities, so that even if a new exotic species joins the pool of locally available parasite species, it may not succeed in establishing itself in the host population. These 3 features are often accepted as biological paradigms in the parasitology literature (e.g., Roberts and Janovy, 1996; Bush et al., 2001), together suggesting that parasite introductions are rarely successful. These features are overemphasized, however, and parasites of all kinds display much more plasticity in their use of hosts and transmission pathways than was traditionally believed.

Let us first take a look at host specificity. In a survey of 616 parasites of livestock (35% of which were helminths, and the rest included fungi, protozoans, bacteria, and viruses), Cleaveland et al. (2001) found that about two thirds are also known to infect other host species (Fig. 1), many of which belong to different mammalian orders. The potential for transmission across species is clear, as is that for large taxonomic jumps between hosts. Furthermore, in many of these pathogens of domestic animals, wildlife hosts act as reservoirs from which livestock are infected. Increasing encroachment of farms on wildlife habitats and the global distribution of livestock and their movement across vast geographical distances have resulted in a greater overlap and contact between livestock and a range of wild animals. Far from being prevented by strict host specificity, transfer of parasites from wildlife to livestock, or vice versa, has taken place on a grand scale. In addition, Cleaveland et al. (2001) in their statistical analyses have identified low host specificity and, in particular, the ability to infect more than 1 order

of mammals as key features of parasites that are implicated in emerging diseases (Fig. 1). Thus, host specificity is not as strict as emphasized in many parasitology texts; taxonomic jumps from 1 host to another over short time scales are a common consequence of the movement of hosts into new geographical areas and are the source of most emerging diseases of livestock.

A second barrier to the spread of parasites into new areas is widely thought to be the need for the availability of a sequence of suitable host species, at least in parasitic helminths with complex life cycles. Again, this may have been overemphasized. There is evidence that many helminths when faced with shortages of certain hosts can quickly adjust their life cycles and transmission strategies. For instance, like all its relatives, the nematode *Camallanus cotti* has a 2-host life cycle; adult worms live in freshwater fish from which they release larvae, which then infect planktonic copepods as intermediate hosts. However, captive fish populations maintained in closed aquaculture systems for several generations remain infected with the nematode despite the complete absence of copepods in the water (Levsen, 2001; Levsen and Jakobsen, 2002). The parasite has rapidly evolved the ability of direct fish-to-fish transmission, switching from a complex to a direct life cycle. Similarly, truncated life cycles are common among parasitic trematodes, with many species displaying a latent ability to abbreviate their life cycle by skipping 1 transmission event, i.e., dropping 1 host from their normal cycle, when conditions demand it (Poulin and Cribb, 2002). This could facilitate the invasion of trematodes into new geographical areas. In a review of environmental and social influences on parasite infections in Thailand, Petney (2001) provided data indicating that about half of the Thai laborers forced to work abroad to earn a better income are infected by the trematode *Opisthorchis viverrini* when they leave Thailand. The parasite is acquired by the ingestion of raw or undercooked fish and is associated with significant pathology of biliary ducts (Petney, 2001). Its eggs, released in the laborers' feces, are lost, and the parasite is not established in new areas unless suitable hosts are found; given the local availability of intermediate snail hosts and the flexibility of trematode transmission cycles, however, anything can happen. The ability of parasites to exploit novel routes of transmission as they become available should never be underestimated. The protozoan *Toxoplasma gondii* is another prime example, with both food- and water-borne transmission interacting with wildlife hosts to create complex and flexible transmission patterns (see Slifko et al., 2000; Tenter et al., 2000).

The third potential barrier to the invasion of exotic parasites in a new area or a new host population is the possibility that most long-standing parasite communities are saturated with species and that there is no room to accommodate new ones. This on its own could limit the risks associated with introduction of parasites after large-scale movement of hosts. There is indeed some evidence for species saturation in helminth communities in a given host species (e.g., Kennedy and Guégan, 1996). However, across species of birds and mammals, the maximum number of helminth species that individual hosts harbor appears proportional to the number of species locally available, i.e., present in the host population, with no trace of an upper limit (Fig. 2). Epidemiological models also suggest that new parasites can successfully join a community and even displace previously established species under a range of conditions (see Roberts et

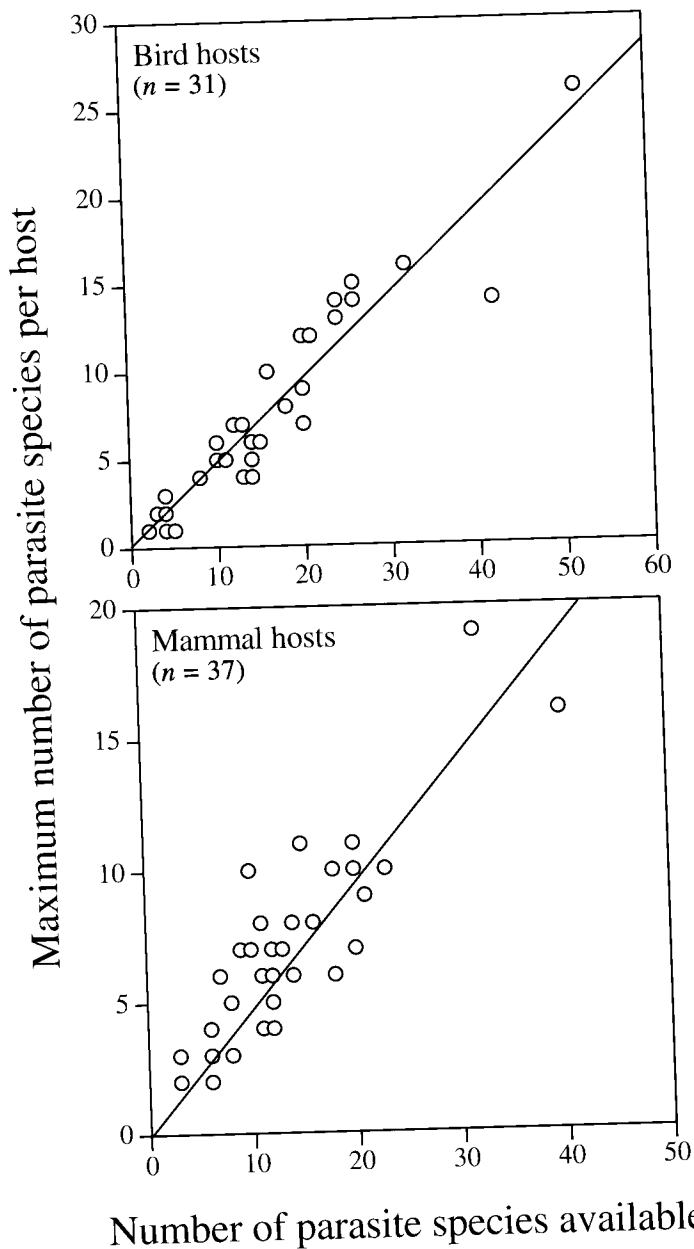


FIGURE 2. Relationship between the maximum number of intestinal helminth species per host individual and the number of species locally available in the host population, among different populations of bird and mammal hosts. Whereas a curvilinear function would suggest that hosts become saturated with parasite species, the linear relation observed suggests that the more the parasite species that are available locally, the more the host can accommodate (data from Poulin, 1998).

al., 2002). It is therefore unlikely that newly introduced parasite species will be routinely squeezed out of the parasite communities they have recently joined.

The biological features of parasites that may, at first glance, appear as barriers to their spread and establishment in new areas or new host species are in fact more plastic than widely believed. The apparent host specificity of certain pathogens and the complex transmission routes followed by others often show much flexibility, and there is often space in established parasite communities for new species. In addition, the conditions faced

by parasites after their introduction to new areas or new host populations (see below) may even favor their transmission and influence their evolution.

### EVOLUTIONARY EPIDEMIOLOGY IN THE GLOBAL VILLAGE

Large-scale movement of people and livestock serves to increase connectivity among populations that would otherwise be isolated if it were not for human technology replacing natural dispersal and migration. For instance, the transportation of livestock between geographically distant areas has the same effect as the creation of corridors between nearby subpopulations, except that it occurs on a larger spatial scale. The associated movement of parasites results in gene flow between populations that would otherwise not be connected and creates a large-scale parasite metapopulation (*sensu* Hanski and Simberloff, 1997) where extinctions in local patches are only likely to be ephemeral.

There are potential epidemiological as well as evolutionary consequences associated with these greater movements. From an epidemiological perspective, when a pathogen first appears in a population of naive hosts, either because it or the hosts have been introduced to a new area, its dynamics can follow 1 of 2 extreme trajectories: the pathogen either becomes extinct locally or it initiates an epidemic outbreak. The fate of the pathogen depends on its basic reproductive number,  $R_0$  (see Anderson and May, 1991; Hudson et al., 2002). For microparasites (bacteria, viruses, protozoans, and fungi capable of rapid multiplication in a host),  $R_0$  is formally defined as the number of secondary infections produced by the first individual pathogen to enter a population of fully susceptible hosts. For macroparasites (helminths and arthropods, where reproduction occurs by transmission of free-living infective stages that pass from 1 host to the next), it is the average number of female offspring produced throughout the lifetime of a female parasite that would themselves achieve reproductive maturity in the absence of density-dependent constraints. When  $R_0$  is less than 1, the pathogen cannot maintain itself and declines to extinction. When  $R_0$  is greater than 1, however, an epidemic outbreak may occur. Mathematical expressions of  $R_0$  vary, but they are all formulated as a ratio. In this ratio, host population size and some measure of transmission efficiency (either a rate of contact with susceptible hosts for microparasites or a birth rate of transmission stages for macroparasites) are in the numerator and natural host mortality plus parasite-induced host mortality are in the denominator.

In light of this fundamental parameter of disease dynamics, we can see a number of epidemiological consequences of host movement. First, high levels of host movement over large geographical distances should lead to more frequent disease epidemics. When a novel microparasite is introduced into a naive host population (or when naive hosts are brought within the geographical range of a pathogen they have not faced before), the effective host population size, *i.e.*, the pool of susceptible hosts, is suddenly higher from the parasite's perspective. This increases the current value of  $R_0$  beyond what it would be normally and allows the pathogen to spread. Indeed, Dobson and Fofopoulos (2001) have found that most current epidemics in wildlife hosts involve pathogens introduced from a different

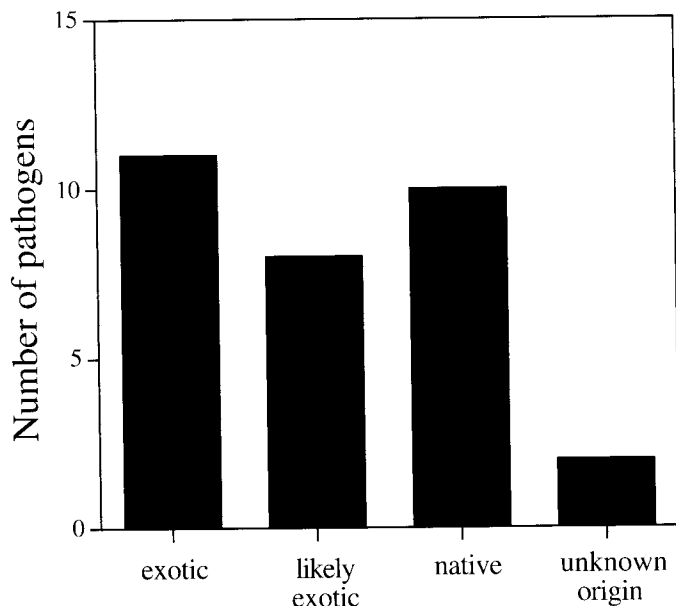


FIGURE 3. Probable geographical origin of pathogens responsible for 31 emerging disease outbreaks in wildlife hosts reported in North America between June 1998 and June 2000: at least one third, and possibly more than half, are of exotic origin (data from Dobson and Foutopoulos, 2001).

geographical area (Fig. 3); the same situation probably also applies to livestock diseases.

Second, the ability of a pathogen to cause an epidemic outbreak will be related to its ability to use other host species in the area where it is introduced. Again, this would increase effective host population size and raise the value of  $R_0$ , all other things being equal. Thus, generalist parasites are more likely to trigger emerging disease problems. If 1 of the host species used by the pathogen is an abundant wildlife reservoir host, the disease problem can persist and prove difficult to control. For instance, bovine tuberculosis, caused by *Mycobacterium bovis* and introduced to New Zealand along with cattle, is proving difficult to eradicate primarily because of its use of wild mammal species as reservoir hosts (Roberts, 1996). Ironically, the main reservoir host is the possum *T. vulpecula*, which was also introduced to New Zealand. A similar situation exists in Britain, where badgers, *Meles meles*, act as reservoirs and maintain bovine tuberculosis in southwestern England, where densities of badgers are highest (Clifton-Hadley et al., 1995; Krebs et al., 1998). The bottom line is that the ability to use many hosts favors the spread and maintenance of infectious diseases, especially when a new pathogen is introduced into an area inhabited by immunologically naive hosts.

A third potential consequence of the interaction between the large-scale movement of hosts and parasites and the parameters shaping  $R_0$  has an evolutionary as well as an epidemiological flavor. Not only can the mass movement of animals across wide distances and the geographical spread of pathogens lead to more frequent emerging diseases, but the severity of the diseases could also increase under these new conditions. The virulence of the pathogen is part of the formulation of  $R_0$ , appearing in the denominator as the parasite-induced host mortality, and it is possible to model the evolution of pathogen virulence using

the expression for  $R_0$  as a starting point (see Frank, 1996). All else being equal, an increase in virulence will reduce  $R_0$ ; high virulence can cause a decrease in host population size below the threshold necessary for the maintenance of the pathogen. Hosts of highly virulent pathogens die before they can transmit the pathogen, even if high virulence allows a pathogen to produce infective stages at a high rate. There is thus a trade-off between virulence and transmissibility, favoring high virulence only when opportunities for transmission are frequent. The spread of virulent strains of pathogens is therefore constrained by the limited number of available hosts or the low rate of contacts among them, i.e., by the opportunities for transmission. This constraint, however, is relaxed at least temporarily when host population size or density is very large or when alternative hosts are available. These are the conditions that often accompany the introduction of a new pathogen to a naive population of susceptible hosts. Under these conditions, a more virulent strain could achieve greater transmission rates than a benign one without compromising its persistence and driving its host and itself to rapid local extinction. The mass movement of people, animals, and food between geographical regions could therefore promote the evolution of more virulent pathogens and facilitate their maintenance.

The above suggestion may seem far-fetched, but there are precedents. Other large-scale anthropogenic changes made to the living and transmission conditions experienced by parasites have driven their rapid evolution. The extensive and worldwide use of anthelmintics to combat nematode parasites of sheep and cattle has altered the conditions under which natural selection acts on these parasites. The most publicized outcome has been the development and spread of drug resistance in many different species of nematodes (e.g., Jackson and Coop, 2000). However, because rates of within-host adult parasite mortality have been increased by drug administration over several parasite generations, we might also expect other evolutionary changes. Several authors have argued that the selective pressure imposed on parasites by anthelmintics should favor changes in age at maturity and possibly in virulence (Medley, 1994; Poulin, 1996; Skorpington and Read, 1998). Recently, these predictions received empirical support from a demonstration that showed that adult size and thus fecundity in the nematode *Teladorsagia circumcincta* have evolved differently in drug-resistant and susceptible isolates subjected to different anthelmintic treatments (Leignel and Cabaret, 2001). It is therefore not unlikely that other changes in the parasites' world, such as increased introductions into naive host populations, could also select for changes in parasite life history traits, or virulence, or both.

## THE FUTURE

Recent reviews suggest that the rise in emerging diseases in humans, domestic animals, and wildlife is just starting and generally paint a bleak picture of the current situation and of our immediate future (Harvell et al., 1999; Daszak et al., 2000; Cleaveland et al., 2001; Dobson and Foutopoulos, 2001; Taylor et al., 2001). It is unlikely that we will be able to completely prevent the movement and introduction of diseases using quarantine procedures, given the sheer volume of movement involving people, animals, and food products. Our health, our food supply, and natural ecosystems will always be at risk. Two

of the most important consequences of the large-scale and frequent movement of animals or parasites may be that disease outbreaks will be facilitated and that the new conditions may promote the evolution of more virulent pathogens. In the short term, our best hope is to be able to control emerging diseases by reducing their spatial and temporal scale. To do this, we now possess an arsenal of modern tools from molecular biology and epidemiology that needs to be deployed rapidly to counteract the potentially devastating effects of emerging infectious diseases (Thompson, 2001). These include the use of sensitive and accurate DNA-based diagnostic methods for surveillance and early detection of diseases and powerful mathematical models to predict the likely dynamics of epidemics. We know what to expect, and we know the causes and have the solutions to many infectious diseases; we now must commit enough resources to this ongoing battle.

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