Parasitism: A Cryptic Determinant of Animal Community Structure

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Traditionally, the major biotic determinants of animal community structure were assumed to be competition and predation. Recent theoretical, experimental and field studies indicate that parasitism is also important. The spectrum of effects that parasites have on host individuals is as broad as the range of parasite–host interactions. This review considers evidence for direct effects of parasites on fecundity and survival that influence host demography, and for indirect effects on host physiology and behaviour that interface with competition and predation.

Parasites are a ubiquitous, yet usually invisible, component of animal communities. They move through the food web; they can form a major component of the biomass of individual organisms; they place energetic demands on their hosts; they alter host behaviour; they frequently increase host death rates and decrease host birth rates; they affect host nutritional status and host growth; they have been shown to alter the outcome of intraspecific and interspecific competition; they can increase host susceptibility to predation; they can influence mate choice; they can alter the sex ratio of the host population. In other words, there are a myriad of ways in which they influence the population dynamics of the host species. By direct extrapolation, therefore, they should influence the abundance and diversity of organisms in the environment. Nevertheless, parasitism has been largely ignored in population and community ecology. This oversight must be corrected if researchers hope fully to understand the factors that influence animal communities.

Effects of parasitism on host demography

Pathology due to parasitic infection can lead directly to reduced host survival[1]. This effect is usually intensity dependent: the rate of host mortality is dependent on the intensity of the parasitic infection (number of parasites in the host). The resultant direct reduction in population size has been demonstrated in laboratory studies[2] and has been reported in many field situations (see review in Ref. 1). There are obvious community consequences that result from reduced numbers of one species; for instance, Ayling[3] has shown that the mortality induced by disease in sponges created patches that allowed colonization by a variety of invertebrates, thus increasing species diversity.

Other important changes are often more subtle. For example, if the variation in intensity of infection and resulting risk of mortality depends on host age or sex, the infection may not only cause changes in the size of the host population, but also alter host demography[2]. As animals age, shifts in diet or behaviour may alter rates of exposure to infective stages of parasites. Infection history and physiological changes during maturation and reproduction also influence the innate or acquired ability to resist infection[4]. These factors lead to differences in the intensity of infection among age or size classes of host[5]. In addition, the pathogenicity of a given infection intensity can vary depending on host age and sex. Three examples illustrate these points.

(1) Gammarid crustaceans (*Gammarus pulex*) are infected with larval stages of an acanthocephalan parasite that significantly reduces the lifespan of the infected host. Brown and Pascoe[6] have shown that the intensity of infection is low in small gammarids, peaks in medium-sized gammarids, then decreases in larger gammarids, presumably in part because heavily infected medium-sized individuals die. The mortality induced by the parasite is even more pronounced when gammarids are exposed to low levels of cadmium. Smaller individuals are most susceptible to the effects of cadmium, but generally have low-intensity infections. Thus, the net effect of infection and cadmium on small individuals is lower than it is on medium-sized gammarids, which are moderately affected by cadmium and heavily infected.

(2) A dipteran parasite of harlequin frogs (*Atelopus varius*) infects female frogs at a higher rate, and causes higher mortality in females than in males. Crump and Pounds[7] suggest that this differential effect may be, in part, responsible for the strongly male-biased sex ratios seen in this species.

(3) Feline parvo virus was introduced into Marion Island, Indian Ocean, in an effort to control feral cat populations. This virus is much more pathogenic in kittens than in adult cats, and its introduction onto the island has resulted in pronounced changes in the age structure of the cat population[8].

It is difficult, in the field, to quantify the contribution of parasitism to host mortality, but it is even more difficult to quantify the effects of parasitism on host reproduction (Box 1). Documented effects that have direct implications for host population abundance and age structure include delayed growth[14] – leading to reduced fecundity[11] – delayed age at sexual maturity[15], increased rates of abortion[16], direct castration[17] and reduced ability to care for young – leading to increased mortality of young[18,19].

Parasitism has also been shown to have major effects on host movement, frequently mediated by effects of infection on host behaviour. Rabid foxes (*Vulpes vulpes*) disperse more widely than healthy individuals[20]; badgers (*Meles meles*) infected with bovine tuberculosis move into man-made shelters more readily[21]; nematode parasites have been shown to increase the spontaneous running tendency of infected mice (*Mus musculus*); *Dicrocoelium*-infected ants move to the top of blades of grass; and gammarids infected with larval acanthocephalans move to the top of the water column[22]. One of the best-documented examples concerns the effect of a nematode, *Onchocerca volvulus*, on movement of human communities in regions of Africa where the parasite is en-

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demic. The parasite is transmitted by blackflies (*Simulium* spp.), and causes major eye disease leading to 'river blindness'. At regular intervals, whole communities move away from the rivers where blackflies breed, and live in less-fertile areas for a generation or two, before they return to the river[23]. It has even been suggested that bird migrations, the regular movement of packs of wolves and the migration of wildebeest across the Serengeti plains can be at least partially explained as an evolutionary adaptation to avoid high rates of parasitism[24].

Indirect effects on predation

Ingestion of infected prey can affect the health of predators. Reduced activity[9] and anorexia[25] are commonly reported in infected animals and both may have direct implications for the ability of predators to obtain food, or for the type of prey that they are able to catch. Correlations of stomach contents and parasite infection might provide indirect information on this hypothesis. However, an experiment could easily be designed in which the feeding habits of a predator were monitored before and after anti-parasitic treatment. Alternatively, feeding behaviour could be examined before and after experimental infection with parasites. These types of experiment would provide a more direct indication of the impact of infection on capture rates and/or prey preference. Shifts in feeding habits have been shown to have dramatic influences on animal and plant communities (see e.g. Refs 26, 27).

Body size is affected by parasitism through effects on nutritional status related to anorexia, vomiting and diarrhoea, reduced absorption due to mucosal damage in the intestine and leakage of fluids and nutrients into the intestine during haemorrhage[25]. Direct competition between parasite and host for specific nutrients has also been documented[25]. This has consequences for growth rate and subsequent adult body size. Body size of both prey and predators is known to be an important determinant of patterns of prey selection and predation risk[28], and thus parasite-induced changes can significantly alter host life history.

Another point of interface with predation is through increased susceptibility of infected hosts (particularly intermediate hosts) to predation. Parasitic infections have been shown to reduce vigor, alter movement patterns and/or debilitate a host, thus increasing its risk of predation[29]. This may facilitate transmission of the parasite in cases where the predator is a potential host, or may prevent transmission (if the predator–prey link is not involved in the parasite life cycle)[22].

Some parasites induce behavioural changes that increase the probability of spatial and temporal overlap with predatory definitive hosts[30]. Such changes may alter the feeding ecology of the predator by increasing the availability of infected individuals within a prey population. This may have ramifications for the prey community and in turn for the wider community. Such effects would apply equally regardless of whether the predator is a required link for the parasite life cycle. However, the predator faces opposing risks in 'choosing' to select the available (parasitized) prey item. This is of primary importance when the prey are the intermediate host and the predator is the definitive (final) host for the parasite. Although energy expenditures for catching infected prey may be reduced, the presence of larval stages of parasites within that prey may result in future energetic costs to the predator. Effects of that infection on predator survival and reproduction, competitive fitness or subsequent ability to obtain prey may all influence community structure.

To our knowledge, the resulting risk–benefit balance has not been studied. There are at least three possible scenarios, depending on whether the predator can also be the final host for the parasite, and on the degree of 'harm' that the parasite does to the final host. In these scenarios, we assume that all potential prey items are equally abundant and of equal nutritional value, but that parasitized prey items can be obtained by the predator at a lower cost. First, if the predator does not become infected following ingestion of infected prey, it might tend to choose debilitated (parasitized) prey. Second, in situations in which the predator does become infected by parasites present in the prey, but where the potential parasites available have minimal effects on the predator, it might also choose debilitated prey, assuming that the benefits of reduced capture costs outweigh the costs of prey discrimination or future parasitism. Third, if the parasites present in prey species are pathogenic to the predator, there may have been selection pressure to avoid choosing the debilitated prey.

Interestingly, the third scenario has not been observed. This may be because it is not an option, or because the cost–benefit ratio is unfavourable, or because we haven't looked for it in appropriate animals. Clearly, the extent to which these scenarios would be expected depends on the probability of the host encountering a pathogenic parasite in potential prey (the prevalence and frequency distribution of the

Box 1. Parasitism and host fitness

The parasitology literature is replete with examples of effects of parasites on host physiology, but few studies have also examined the ecological and fitness consequences of these alterations. A notable exception is the long-term study by Joseph Schall and colleagues of the malarial parasite *Plasmodium mexicanum* and its vertebrate host, the western fence lizard (*Sceloporus occidentalis*) in northern California.

Schall *et al.* have described the substantial pathology and numerous physiological changes in naturally infected lizards. These effects include anaemia, reduced haemoglobin concentration and decreased maximal oxygen consumption and aerobic scope[9,10]. Subsequently, the effect of parasitism on host fitness was assessed in natural populations. Although infected lizards suffered increased mortality in captivity, malarial infections did not lead to differential mortality in natural populations. The costs of parasitism on host fitness were manifested through differences in reproduction. Schall demonstrated that one of the physiological changes – reduction in the accumulation and storage of lipids – led to a reduction in clutch size in infected females[11].

More subtle behavioural changes have been linked with malarial infection and several have implications for host reproductive success. Infected lizards are socially less active, possibly due to their reduced haemoglobin levels and aerobic capabilities[12]. The infection hinders male courtship behaviour and disadvantages infected males in male–male competition[13].

Schall's work has demonstrated that the physiological effects of parasitism influence the demography of western fence lizard populations. The extent to which parasite effects on host populations are translated into community-level effects will be one of the questions for the 1990s.

Box 2. Parasites and the distribution of water boatmen

The natural distribution of water boatmen (Corixidae) in lakes in British Columbia, Canada, is correlated with salinity. *Cenocorixa expleta* occurs only in lakes of moderate to high salinity whereas *C. bifida* occurs in freshwater lakes and lakes of low to moderate salinity. Over several years, Scudder and his students[38,39] have compared a range of physiological and ecological parameters in an attempt to understand the mechanisms whereby salinity differentially influences these two species of water boatmen.

Experimental studies on the effect of salinity on adult survival revealed that *C. expleta* was able to survive at higher salinities than *C. bifida*. However, both species survived well at low salinities. Therefore, the exclusion of *C. expleta* from lakes of low salinity was not explained by effects of salinity on survival. Osmotic and ionic balance at low salinities was also similar in both species. The research has shown no difference in availability of predators that could explain the absence of *C. expleta* at low salinities. Egg hatching and phenology are similar for both species. In lakes where they co-occur, there is no spatial or temporal segregation of microhabitat use. Although feeding-preference studies revealed considerable overlap in the fundamental feeding niche (Fig. 1a), suggesting the possibility of interspecific competition, serological studies of the actual feeding habits of both species from both sympatric and allopatric habitats revealed virtually identical patterns (Fig. 1b).

Differences were seen, however, with respect to mite parasitism (Fig. 1c). Four species of water mites were found, but only in lakes of low salinity. Both species of water boatmen are susceptible to mite parasitism, but infection levels in the field and under experimental trials were significantly higher in *C. expleta* than in *C. bifida*. The natural occurrence of *C. expleta* only at salinities greater than the upper tolerance range of the water mites, combined with the high susceptibility of *C. expleta* to infection, and the evidence that water mites delay sexual development and inhibit egg production in water boatmen, has led Scudder to conclude that the exclusion of *C. expleta* from lakes of low salinity results from the presence of water mites in these lakes.

pathogenic parasite in the intermediate host population).

Indirect effects on competition

Parasitism influences both intraspecific and interspecific competition. Host behaviour may be modified by parasitism (usually in an intensity-dependent fashion), resulting in significant fitness implications for that individual host. For example, infected adult sticklebacks (*Gasterosteus aculeatus*) alter their foraging tactics to compensate for reduced competitive abilities[31] and infected juveniles alter their risk-avoidance behaviour[32]. It has been suggested that parasitism exerts a negative influence on male dominance, stamina and social activities that are important in mate competition[33]. The proposed association between genetically based resistance to parasitism and male secondary sexual traits implicates parasitism in female mate choice[34].

The effect of parasitism on interspecific competition is likely to be important through its impact on species richness. A host species whose fitness is impaired by parasitism would be at a selective disadvantage in competition with a closely related but relatively unaffected species. In Park's[35] classic work with *Tribolium* beetles, a sporozoan parasite (*Adelina tribolii*) had a greater negative impact on the competitively superior beetle species, and its presence shifted the outcome of competitive interactions between the species.

Pathology associated with infection may directly exclude host species, thereby reducing species richness. Parasites help to create the patterns of spatial distribution of animals not only through the extirpation of competing species from particular areas (e.g. moose, *Alces alces*, are paralysed due to *Pneumostrongylus tenuis* transmitted from white-tailed deer, *Odocoileus virginianus*), but also by limiting the invasion of new species[36]. Freeland[37] suggested that species richness is limited by the high susceptibilities of potential invaders to the present parasite fauna. Conversely, parasitism can increase species richness and evenness through differential negative effects on the abundance of competitively superior hosts. A nonspecific parasite utilizing a group of related hosts in a frequency-dependent manner (in proportion to host abundance), or a selective parasite preferentially infecting a competitively dominant host species, should lead to increased species diversity. In this context, such a parasite would be considered as a 'keystone parasite'.

Case studies

Perhaps the best example of the impact of parasitism on host community structure is the ramifications of the introduction of the myxoma virus into England. This parasite caused a rapid decline in the rabbit (*Oryctolagus cuniculus*) population, which in turn changed the vegetation patterns and influenced both invertebrate and vertebrate populations[27]. A second example concerns a long-term study on the determinants of community structure of water boatmen in saline lakes in British Columbia, Canada (Box 2).

Another well-documented example of the effect of disease on community structure comes from the plant-ecology literature. Osborne[40] reported the variety of consequences for animal and plant communities that resulted from the arrival of Dutch Elm disease in England. The increased number of dead trees decreased the availability of habitat for a number of birds, but also resulted in an increase in the number of beetle larvae in the dead trees. This increased the availability of food for other bird species. When the dead trees were felled, the vegetation changed due to increased light reaching the ground. With more herbs and shrubs, other species of birds were able to establish.

Future studies

Are communities haunted by the ghost of parasitism past? To what extent does parasitism play a role in the organization of communities? Are the indirect effects of parasitism significant components of competition and predation? These questions cannot fully be answered by making inferences from studies of individuals and populations. Instead, community-level studies must be conducted that include parasitism as a potential structuring force and as an additional or alternative explanation for observed patterns.

The inclusion of parasites in experimental community-level studies must begin with a survey of potential parasites to assess their prevalence and relative impact among different host species. It is clear that there will be tremendous variation among parasite species in their role within the host community. Scott and Dobson[41] suggested that effects may be more easily attributed to parasites with direct life cycles as there will be fewer time delays in the interaction between the parasite and its host population compared with life cycles involving both

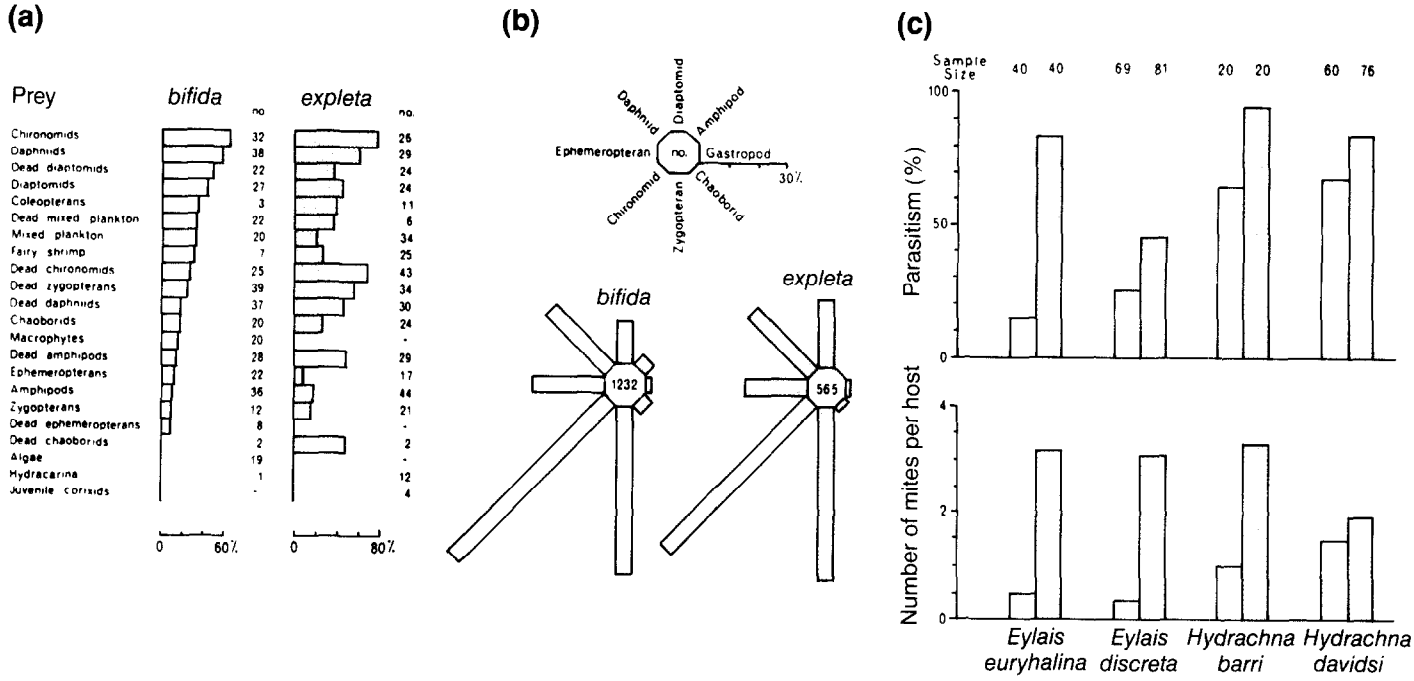

Fig. 1. (a) Comparison of fundamental feeding niches of two species of water boatmen (Corixidae). Percentage acceptance of food items offered to adult *Cenocorixa bifida* (left) and *C. expleta* (right). Results arranged in order of acceptance by adult *C. bifida*. (no. = number of individual predators tested.) (b) Comparison of realized feeding niches. Diagram shows feeding pattern of *C. bifida* (left) and *C. expleta* (right) expressed as percentage of guts showing immunological reactions with each antiserum tested. Results from adults and larvae are combined. The upper diagram shows the scale and the groups of organisms tested. (no. = number of guts tested.) (c) Comparison of prevalence of parasitism (upper diagram) and mean number of mites per host (lower diagram) when *C. bifida* (unshaded) and *C. expleta* (shaded) were exposed experimentally to mites of four species. *From Ref. 38; the specific identifications of the four mite species have been corrected since publication of the original paper.*

intermediate and definitive host species.

Ideally, manipulative experiments should be undertaken to involve treatments with parasitism alone and in combination with other potential abiotic and biotic forces. Such factorial experiments will facilitate assessment of the relative importance of each determinant, and more importantly, of interactions among them. As always, the conditions will ideally mimic natural conditions. Having defined the gold standard, it is also important to realize that such studies will be extremely difficult. Therefore, it is equally important to include the measurement of parasite parameters in large long-term ecological studies and to record carefully changes that occur due to natural or human perturbations to the environment. The response of the host community to such perturbations, although unreplicated, is often the best experiment that ecologists can have.

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Genetic Control of Migratory Behaviour in Birds

Peter Berthold

Although it has long been suspected that biannual migration in birds has a direct genetic basis, only in the last decade have details of the inheritance of behavioural traits such as migratory activity and directional preferences been demonstrated. A *model has now been developed to estimate how inexperienced first-time migrants manage to reach their unknown winter quarters on the basis of inherited spatio-temporal programs. Furthermore, in obligate partial migrants the decision to migrate or not has been shown to have a strong genetic base. Migratoriness and sedentariness in partial migrants have been shown to have a high potential for rapid evolution.* A *recent set of results has suggested that novel migratory habits can evolve in less than* 25 *years.* A *possible consequence is that environmental changes, including 'greenhouse' effects, might considerably alter avian migration systems by acting on genetic variation for migratory tendencies.*

Before genetic control mechanisms for migration in birds had been demonstrated, 'endogenous' factors affecting long-distance intercontinental migration had been postulated. These factors had been documented in the 1970s and were termed 'circannual rhythms' or 'internal calendars'[1,2].

About ten years ago it became possible to trace these endogenous factors back to their genetic base. One prerequisite for these investigations was the establishment of a system for breeding free-living migratory birds on a large scale in captivity. This was accomplished for a European warbler, the blackcap (*Sylvia atricapilla*)[3]. This species has an extraordinarily wide range of migratory habits (Fig. 1) and is thus especially suitable for long-term programs aimed at elucidating the genotype–environment interactions that occur during migration. Migratory behaviour in nocturnal migrants such as the blackcap can easily be measured quantitatively as nocturnal migratory restlessness or *zugunruhe*[2].

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Heritability of basic behavioural traits

A migratory species has to know when to leave the breeding grounds, where to go, when to end the journey and how to organize the trip. Although all these essential features should be regulated and affected by both external and internal factors, a direct genetic control has now been demonstrated for all of them.

The urge to migrate

With the exception of young cuckoos (*Cuculus canorus*), which never meet their parents, the first departure of all obligate migrants could theoretically be triggered by experienced conspecifics. In singly migrating species, however, it has generally been hypothesized that endogenous factors are crucial and that the urge to migrate is innate. However, the actual genetic basis and heritability of the urge to migrate have only recently been demonstrated experimentally. When blackcaps from a fully migratory population (from Central Europe) and a resident population (from the Cape Verde Islands) were cross-bred, 40% of the F_1 hybrids were found to be migratory[4]. Thus, the urge to migrate can be bred into a nonmigratory bird population. Similarly, the urge to migrate can be enhanced or reduced by selective-breeding experiments in partially migratory populations. There are, however, limits to this inheritance. The fact that in the cross-breeding experiment not all hybrids became migratory indicates that it is not a single locus that determines the urge to migrate. It is more likely to be a multilocus system with a reaction threshold[4].

Endogenous time programs and migratory distance

Many migrants are thought to migrate singly to distant wintering areas, even though in such species winter quarters are known to be well defined (and hence effectively predetermined). Thus, inexperienced individuals of such species must somehow have 'knowledge' of these areas.

Several hypotheses have been proposed to account for the means of reaching unknown winter quarters. Innate knowledge of specific local factors, such as star patterns, endogenous distance and energy, have been suggested, as have endogenous distance and time programs[2]. A substantial amount of evidence has supported the idea that endogenous time programs play a major role[5]. The most important finding was that the amount of migratory activity displayed by caged individuals of various species and populations was correlated with the distance needed to be covered. Again, cross-breeding experiments with birds of populations with different migratory habits have shown that the amount of migratory activity can be inherited. When blackcaps from Central Europe (long-distance migrants) were cross-bred with conspecifics from the Canary Islands (short-distance migrants) or with