

## FORAGING ON PREY THAT ARE MODIFIED BY PARASITES

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**Abstract.**—A model that weighs the energetic cost of parasitism for a predator against the energetic value of prey items that transmit the parasite to the predator suggests that there is often no selective pressure to avoid parasitized prey. This offers an explanation for why parasites so frequently exploit predators and prey as definitive and intermediate hosts, respectively. Furthermore, predators may actually benefit from their parasites if energetic costs of parasitism are moderate and prey capture is facilitated by parasites. Parasite species that benefit predators through modification of prey are not mutualistic, however.

Although they are often ignored, parasites can affect predator-prey interactions. Many parasites exploit trophic transmission (whereby infective stages are ingested by the host). Frequently, the definitive host is a predator that preys on the intermediate host. Some protozoans, a few nematodes, many trematodes, most cestodes, and all acanthocephalans are transmitted this way. Although these parasites extract a cost from their definitive hosts, and many other costs of foraging have been suggested (Stephens and Krebs 1986), the risk of acquiring parasites is not often considered for foragers (Moore 1983). Why should predators continue to support trophic transmission? Avoiding parasitized prey would appear to be a convenient solution. Perhaps it is difficult for predators to recognize parasitized prey; alternatively, there may be no fitness advantages for predators that avoid parasitized prey. In this article, I present a foraging model that considers trophically transmitted parasites. The model compares the rate of energy gained for a predator if some prey are parasitized and the predator avoids parasitized prey, if some prey are parasitized and the predator ingests parasitized prey, and if no prey are parasitized. This model suggests that predators should not avoid parasitized prey and that they may actually benefit from the presence of parasites.

Predators often take odd or unusual prey individuals (Temple 1987). Parasitized prey can be odd and are often found more frequently than expected in the diet of definitive host predators (Dobson and Keymer 1985). In fact, there is increasing evidence that larval parasites modify the behavior or appearance of intermediate hosts (see reviews in Holmes and Bethel 1972, Moore 1984, and Dobson and Keymer 1985). For example, parasitized prey may be more conspicuous, disoriented, less able to flee, or less likely to show an escape response (Holmes and Bethel 1972).

Sometimes, it may be possible to quantify the effect of a parasite on its interme-

diate host. Dobson and Keymer (1985) define the **degree of parasite-induced behavior modification,  $\alpha$** , as the increased rate at which prey are eaten if parasitized. For parasitized prey with no behavior modification,  $\alpha = 1$ . With  $\alpha = 2$ , parasitized prey are captured twice as often as unparasitized prey. I have interpreted  $\alpha$  as the forage ratio (Ivlev 1961) for parasitized prey divided by the forage ratio for unparasitized prey. Assuming that each parasitized prey carries one parasite (Dobson and Keymer [1985] allow multiple infections), define  $\alpha$  as

$$\alpha = \frac{h_i/H_i}{h_o/H_o}, \quad (1)$$

where  $h_i$  and  $h_o$  represent parasitized (infected) and unparasitized prey eaten by **predators**, and  $H_i$  and  $H_o$  represent parasitized and unparasitized prey in the environment. For example, Feare (1971) found that 13% of the dogwhelks consumed by oystercatchers were parasitized by larval trematodes, compared with a prevalence of 5% in the dogwhelk population as a whole (prevalence is the proportion of hosts that are parasitized [Margolis et al. 1982]). In this case,  $\alpha = (13/5)/(87/95) = 2.8$ . Selectivity for parasitized prey can reach impressive levels. In Argentine alpine lakes, for example, amphipods in the guts of fish are commonly parasitized by acanthocephalan larvae, but living, parasitized amphipods have not been observed despite persistent effort (A. M. Kuris, personal communication).

The modification of parasitized prey potentially hurts predators by increasing their exposure to parasites. Since hosts can learn to avoid parasitized food that they associate with a certain taste (Keymer et al. 1983), it may be possible for some predators to avoid parasitized prey that they associate with a modified behavior or appearance (Lozano 1991). Avoiding parasitized prey, however, carries a cost because it reduces the number of prey items accepted by a predator. In fact, because the modification of prey by parasites may lead to an increase in predation rate, Holmes (Holmes and Bethel 1972; Holmes and Price 1986) suggests there is a trade-off between the cost of parasite acquisition and easier predation. It is plausible, sometimes, that the benefit is greater than the cost (Holmes and Bethel 1972) and that the predator will obtain more energy with parasitism than without parasitism.

#### FORAGING MODEL

The following model compares the costs and benefits of avoiding or ingesting parasitized prey and examines whether modification of prey by parasites can benefit a predator. The net rate of energy gain,  $E$ , acquired by a predator consists of unparasitized prey and parasitized prey, less the cost of parasitism, such that

$$E/t = k(rH_o + \alpha rH_i) - \text{energetic cost of parasites}/t, \quad (2)$$

where  $t$  is time,  $k$  is the energy assimilated from a single prey item, and  **$r$  is the rate of predation on unparasitized prey**. This assumes that a predator's foraging rate for unparasitized prey is independent of whether the predator is parasitized,

parasitized prey have the same energetic value as unparasitized prey, and modification results in an increase in prey availability (or “catchability”).

In this model, the cost of parasitism to the predator is considered to be the combined cost of the parasites within the predator (the parasites within a host are defined as an “infrapopulation” [Margolis et al. 1982]). Parasite fecundity and mortality are assumed to be dependent on the number of parasites within the predator (“intensity” is the number of parasites in a host [Margolis et al. 1982]). The rate of change of a parasite infrapopulation is dependent on the predator’s ingestion of parasites, their successful establishment in the predator, and the parasite mortality rate (R. M. Anderson 1974), such that

$$di/dt = \alpha r q H_i - u i^m, \quad (3a)$$

where  $q$  is the proportion of parasites that establish within the host,  $u$  is the initial, instantaneous, per capita parasite mortality rate,  $i$  is the intensity of the parasite infrapopulation, and  $m$  is a coefficient of an intensity-dependent increase in parasite mortality rate. At equilibrium, therefore, the parasite infrapopulation is

$$\hat{i} = \left( \frac{\alpha r q H_i}{u} \right)^{1/m}, \quad (3b)$$

and the cost of parasitism is the parasite intensity times the per parasite cost (adjusted for crowding), such that

$$E/t = -g \hat{i}^{1-f}, \quad (3c)$$

where  $g$  is the initial rate of energy removed per parasite and  $f$  is a coefficient from zero to one of the intensity-dependent decrease in the energy removed from the host by an individual parasite.

Incorporating the cost of parasitism into equation (2) yields equations for energy gained by a predator under the following three conditions. If the parasite is present and the predator does not avoid parasitized prey,

$$E/t = k(rH_o + \alpha r H_i) - g \hat{i}^{1-f}. \quad (4a)$$

If the parasite is present and the predator avoids parasitized prey,

$$E/t = k r H_o. \quad (4b)$$

Finally, if the parasite is absent from the predator-prey system,

$$E/t = k r H. \quad (4c)$$

The model cannot be interpreted in its present state, because the population equilibria of parasitized and unparasitized predators and prey vary with  $\alpha$  (Dobson and Keymer 1985; Haderler and Freedman 1989). Therefore, the following Lotka-Volterra-style predator-prey model was used to obtain predator-prey population equilibria for substitution into equations (4a)–(4c):

$$dH/dt = bH - dH^2 - rPH \quad (5a)$$

and

$$dP/dt = crPH - jP^2, \quad (5b)$$

where  $b$  is the initial, instantaneous, per capita prey birth rate,  $dH$  is the initial, instantaneous, per capita prey death rate,  $P$  is the number of predators,  $c$  is the conversion of ingested prey into new predators, and  $jP$  is the instantaneous, per capita predator death rate. In addition to the familiar assumptions of Lotka-Volterra models, this model assumes density-dependent mortality for both prey and predator and no handling time (for generating stable equilibria). To incorporate parasites, differential equations were included for parasitized predators ( $P_i$ ) and prey. These equations are outlined schematically in figure 1. It is assumed that all predators are born unparasitized ( $P_o$ ) and there is no immunity to new infections.

Expanding equations (5a) and (5b) yields

$$dH_o/dt = bH - dHH_o - rPH_o - \beta P_i H_o, \quad (6a)$$

$$dH_i/dt = \beta P_i H_o - dHH_i - \alpha rPH_i, \quad (6b)$$

$$dP_o/dt = c(rPH_o + \alpha rPH_i - P_i g_i^{1-f}) - jPP_o - \alpha r qPH_i, \quad (6c)$$

and

$$dP_i/dt = \alpha r P_o H_i - jPP_i, \quad (6d)$$

where  $\beta$  is the transmission rate from predator to prey (parasitized predators excrete parasite eggs that are eaten by prey). Equations (6a)–(6d) are not solvable by analytical techniques, and therefore computer simulation was employed to find various predator-prey equilibria. Predator-prey equilibria, in the absence of parasites, were recorded by setting  $P_i$  and  $H_i$  to zero. These values were then incorporated into equation (4c) to indicate the rate of energy gained by predators in the absence of parasites. This value acts as a point of reference for comparisons with the following situations in which parasites are included.

Predator-prey equilibria in the presence of parasites were recorded according to equations (6a)–(6d) over a range of  $\alpha$ . These values were then incorporated into equation (4a) to indicate the rate of energy gained by predators that ingest parasitized prey and into equation (4b) to show the rate of energy gained by an individual predator that avoids parasitized prey.

## RESULTS

Increases in modification of prey by parasites result in a decrease in the prey equilibria and a less dramatic increase in the predator population (fig. 2). In addition, increasing behavior modification causes an asymptotic increase in the prevalence of parasites in predators (fig. 3). This yields an increase in the prevalence of the parasite in the prey population (because the infection rate from predator to prey is increased) followed by an eventual decline in prevalence (because parasitized prey are rapidly removed from the population). These population-level effects are similar to the theoretical results of Dobson and Keymer

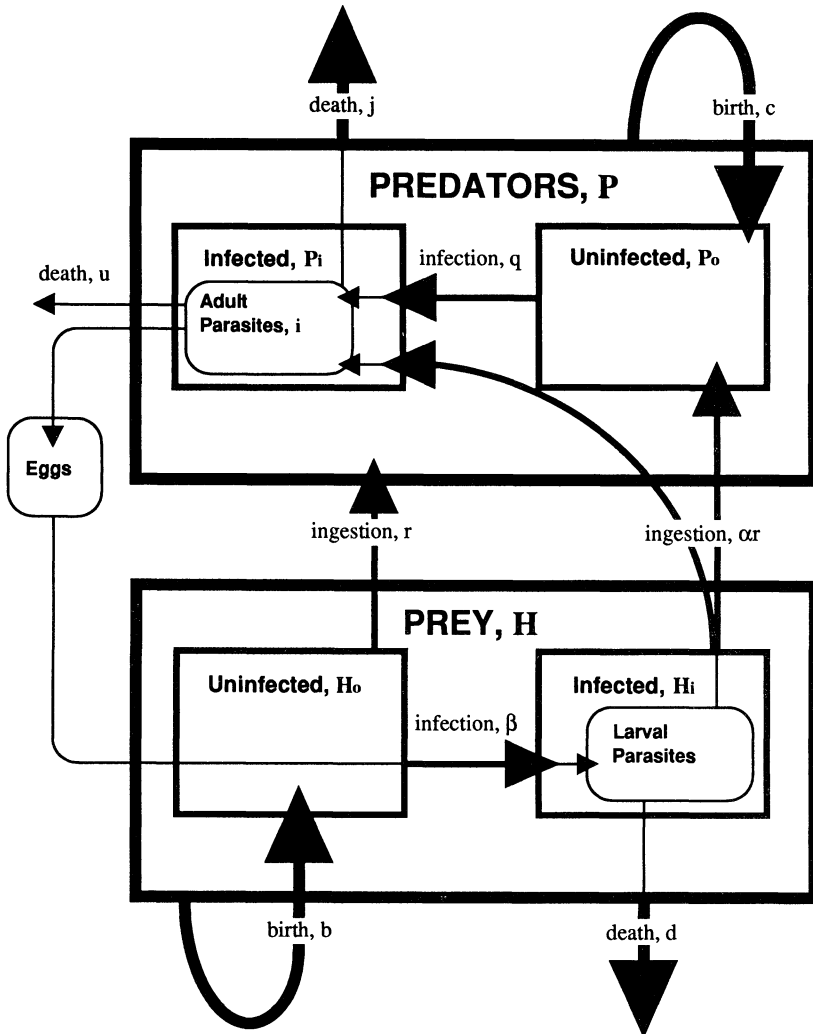


FIG. 1.—Flow chart for predator, prey, and parasite populations

(1985) and Hader and Freedman (1989). Incorporating these population densities into the energy gain functions (eqq. [4a]–[4c]) reveals that an individual predator can benefit from parasites if costs of parasites are moderate and prey are sufficiently modified by parasites (fig. 4). Avoidance of parasitized prey is an appropriate strategy only if parasite cost is high and modification of prey by parasites is low (fig. 4).

#### DISCUSSION OF ASSUMPTIONS

Violations of key assumptions that may have important implications for the outcome of this and other models have not been addressed. The most crucial

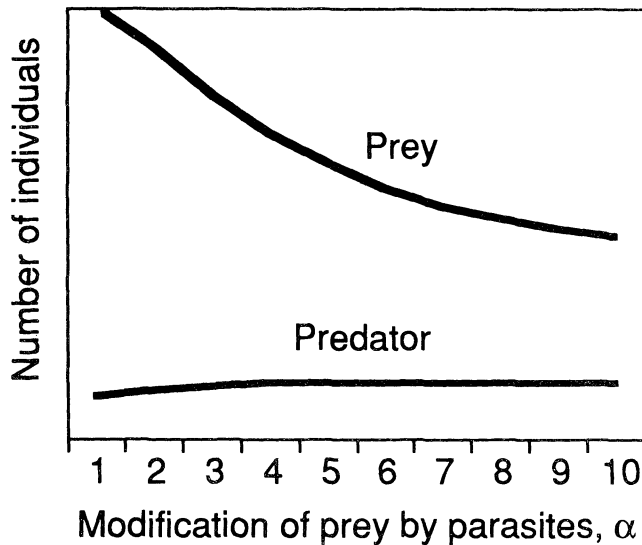


FIG. 2.—The effect of  $\alpha$  on predator and prey equilibrium population size

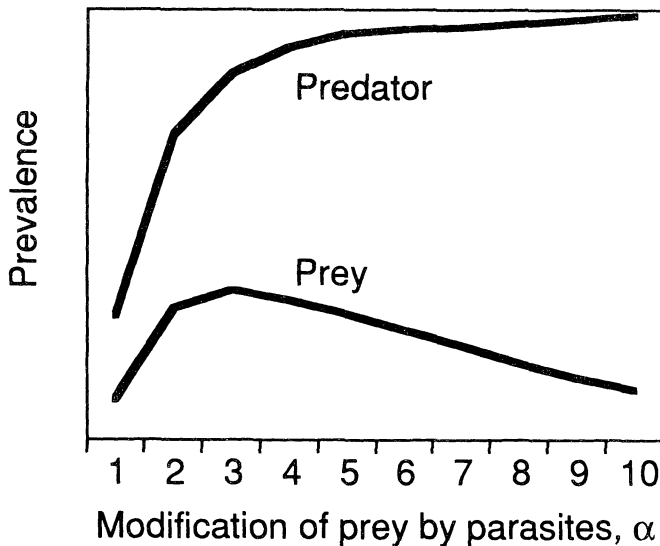


FIG. 3.—The prevalence of parasitized prey and predators as a function of  $\alpha$

assumption is that values of  $\alpha$  greater than one must reflect an increase in the catchability of parasitized prey (Moore and Gotelli 1990). Otherwise the model is irrelevant. For example, selectivity of parasitized prey by predators may occur in the absence of modification by parasites if a parasite has a constant transmission rate to the intermediate host. In this case, parasite prevalence and intensity

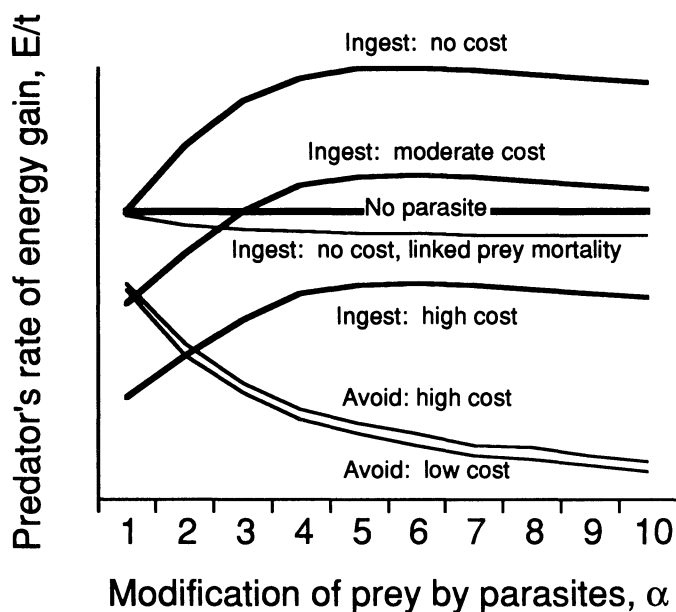


FIG. 4.—The effect of  $\alpha$  and parasite cost on a predator's rate of energy gain for cases in which a predator avoids or ingests parasitized prey or in which no prey are parasitized.

in the intermediate host may be correlated with age, and, if a predator prefers prey on the basis of a variable associated with age, such as size, it will appear that the predator is selecting parasitized prey.

Furthermore, if all sources of nonpredation mortality in the intermediate host increase in direct proportion with  $\alpha$  (prey mortality =  $\alpha dHH_1$ ), no benefit for the predator is possible under any circumstances (fig. 4). This is because prey densities would decrease with  $\alpha$  and the predator would encounter less prey than if the parasite were absent. This would not necessarily be the case if non-definitive-host predators enjoyed increased prey capture of parasitized prey but did not reduce prey population levels substantially.

Another critical assumption is that the rate of predation must be independent of whether a predator feeds on parasitized prey. If a predator becomes satiated because modification has increased prey availability, the actual rate of ingestion will be less than the model predicts. In this case, however, the predator may gain other benefits from reduced foraging time, such as decreased exposure to its own predators (McNamara and Houston 1987). If the predator develops anorexia in response to parasitism, its foraging rate will decline, and the model will no longer be valid. Although anorexia is noted as a response to parasitism (Symons 1989), it is infrequently reported for hosts that become infected by eating prey. Nonetheless, if the predator becomes sick because of parasites and can no longer exploit prey at the same level as a healthy individual, the model is no longer valid. In this case, selectivity on parasitized prey could be a result of a reduced capacity to capture unparasitized prey.

The assumption that there is only one larval parasite per parasitized prey is violated in many, but not all, systems. Intensity-dependent effects on behavior are likely to be seen in "typical parasites" (*sensu* Kuris 1974), whereas the effects of parasitic castrators are likely to be independent of intensity (Kuris 1974). In the former case, the intensity of larval parasites in parasitized intermediate hosts is often distributed as a negative binomial (Crofton 1971). For prey containing more than one larval parasite, Dobson and Keymer (1985) assume the effect on behavior is additive. Under this condition, the conclusions of the model will hold. If the effect is not additive, however, heavily parasitized intermediate hosts will not be vulnerable in proportion to the potential costs of parasitism. In this case,  $\alpha$  should be based on the mean intensity in an intermediate host, and the model can predict only an average rate of energy gain.

The assumption that parasitized prey have the same energy content as unparasitized prey can be violated by (1) a positive correlation between size and exposure to parasites, (2) a negative correlation between size and parasite-induced mortality, (3) a reduction in growth associated with parasitism, or (4) an increase in growth associated with parasitism. If parasitized prey have a lower energy content than unparasitized prey (mechanism 2 or 3), predators that forage on parasitized prey will ingest less energy than predicted by the model. Of course, if 1 or 4 is correct, predators that forage on parasitized prey will gain more energy than the model predicts.

If other prey items are included in the predator's diet, the qualitative outcome of foraging on this parasitized species will not be affected. However, the energetic return of the parasitized prey species could be devalued to the extent that switching to another prey species would make a more efficient use of the predator's time and resources.

Finally, the regulation of parasite infrapopulations is not well understood. In support of the model, high parasite intensities can result in increased parasite mortality and reduced parasite fecundity (a logical correlate of per parasite cost) (Read 1951; Jones and Tan 1971; Keymer 1982). If parasite infrapopulation regulation is based on an immune response, however, the cost of parasitism is discontinuous (it stops when the host becomes immune), whereas the benefit of foraging on parasitized prey is continuous (it continues through the predator's lifetime). Therefore, if immunity is permanent, a benefit will occur given enough time. A benefit may also occur if immunity is concomitant (immunity requires continuous antigenic stimulation), depending on the cost of the parasite. If parasite infrapopulations are not regulated, a benefit will be less likely and avoidance more plausible.

## DISCUSSION

### *Avoidance and Benefit*

In figure 4, where the energy rate curve for ingestion crosses above the *No parasite* line, a predator can benefit from parasites. A benefit is always possible, given some modification of the intermediate host by parasites, if there is no cost of parasitism. Even with moderate costs of parasitism, a benefit for predators is



possible if modification is high enough. The rate of energy gain is not a positive linear function of the degree of modification, however, because, at high levels of modification, prey become rare and the number of prey ingested declines.

The effects of parasites on intermediate hosts and the resultant benefit to definitive hosts may play an important role in predator-prey dynamics and foraging strategy. The parasite provides a delivery service for hard-to-get prey. If parasites allow a more efficient exploitation of otherwise difficult-to-capture prey items, they may be an important factor determining diet breadth and the impact of predation as a force in structuring prey populations. This stands in marked contrast to the traditional view of a predator as an agent that weeds out sick individuals and brings about the increased health of the prey population (Slobodkin 1974; Holmes 1982). Although thinning occurs, its eventual impact is to perpetuate parasite transmission and future predator success.

Even for cases in which a predator does not benefit from parasites, there may be no selection for avoiding parasitized prey unless there is a high cost of parasitism and little modification of prey by parasites. For example, oystercatchers reject clams that are heavily parasitized by trematode metacercariae, but it is not evident that rejected clams are easier prey than less parasitized clams (Hulscher 1973). Avoidance, assuming it is a heritable trait, can spread in a predator population only if it allows individuals to increase their fitness (expressed in the model as  $E/t$ ). For the cases in which avoidance is profitable, the spread of avoidance (assuming heritability and perfect recognition of parasitized prey) would eventually lead to the local extinction of the parasite and an increase in the energy gain for all predators. At a moderate degree of modification and a high cost of parasitism, avoidance by all predators would lead to an increase in the energy gain of the population; however, avoidance cannot be selected for because it results, initially, in a decrease in an individual's fitness. Furthermore, if the ability of a predator to recognize parasitized prey is positively correlated with how different the prey appears, the ability to avoid parasitized prey will be least when the net costs are the highest. These results help explain, even without relying on arguments about constraints of recognition or heritability, why the modification of intermediate hosts is such a successful and pervasive strategy for trophically transmitted parasites.

### *Adaptations of Prey*

Intermediate hosts appear to suffer greater fitness costs due to parasites than definitive hosts. Parasitic castration and increased mortality due to modification by parasites can be consequences of parasitism for intermediate hosts. In many cases, the prey intermediate host becomes parasitized by eating eggs or larval parasites in food. With such high costs of parasitism, why don't intermediate hosts avoid food resources that contain parasite eggs?

Moore (1983) found no significant difference in the feeding rate of terrestrial isopods (which serve as the intermediate host for the acanthocephalan *Plagiorhynchus*) when she presented the pill bugs with starling (definitive host) feces with and without parasite eggs. She suggests that the ingestion of food-rich bird

feces might be worth the risk of parasitic castration for isopods. Therefore, for prey, the costs of avoidance may also outweigh the risks of parasitism.

Altruistic host suicide was first suggested as an explanation for the altered behavior of parasitized hosts by Shapiro (1976). In this case, altered behaviors are assumed to be host adaptations against parasitism such that a host's intentional death reduces the risk of parasitism for its kin. Holmes (1982) has suggested that if prey are parasitically castrated, increased predation, due to behavior modification, might benefit the prey population by removing unproductive, resource-consuming individuals. Although the presence of castrated individuals can negatively affect uninfected individuals through competition (Lafferty 1991), incorporating parasitic castration into equation (6a) (changing  $bH$  to  $bH_0$ ) does not support the prediction that suicide is adaptive. This is because the prey population equilibrium continues to decrease as modification by parasites is increased since predation on castrated prey eventually feeds back, via transmission, to a higher proportion of parasitized prey.

### *Adaptations of Parasites*

Trophically transmitted parasites should evolve to increase  $\alpha$  and limit pathology for definitive hosts. Natural selection will favor parasites with traits that increase the probability that the death of the intermediate host will result in transmission (Wright 1966) and/or shorten their generation time by increasing the rate at which transmission occurs. Not all parasite traits that change prey behavior are necessarily adaptive, however, especially if they are constrained by phylogeny (Moore and Gotelli 1990). In addition, by reducing its impact on the definitive host, a parasite might reduce the likelihood that the predator will choose to avoid parasitized prey in the future (this requires group selection, however). The parasite might also gain the immediate benefit of not inducing a hostile immune response (Sprent 1969). In fact, parasites that exploit trophic transmission generally cause little pathology for their definitive hosts (Bailey 1975; Kennedy 1975; Geraci and St. Aubin 1987), especially when compared with the major effects that intermediate hosts suffer. For example, over a wide range of infection intensities with the tapeworm *Hymenolepis citelli*, the energy budget of white-footed mice was reduced by only 2% (Munger and Karasov 1989). These adaptations do not mean that parasites gain a fitness advantage because they benefit their definitive hosts. Instead, host benefit is an incidental result of natural selection for parasite traits that increase transmission and survival.

### *Mutualism*

Mutualism can be categorized by whether a third party (outside host and parasite) is required for the host to benefit (Abrams 1987). A direct benefit may occur if the parasite provides some kind of nutritive supplement for the host. An indirect benefit occurs when the parasite mediates interactions with a third party (usually host enemies) to the advantage of the host (Boucher et al. [1982] describe direct and indirect interactions as symbiotic and nonsymbiotic mutualisms). Through indirect benefit, a parasite may have the net impact of a mutualist.

Although mutualism has been described as an interaction that increases the rate of growth of populations of two interacting species (discussion in Boucher et al. 1982 and Freedman et al. 1987 for three-species interactions), from an evolutionary perspective, mutualism must be based at the level of the individual (Kuris 1980; Janzen 1985). In other words, individuals that provide a benefit must also obtain a benefit. This may or may not be consistent with interactions at the population or species level (Abrams 1987).

The only evidence of a direct benefit for hosts from parasites (excluding digestive symbionts) is from Lincicome (1971) who found increased weight gains in rats infected with the protozoan *Trypanosoma lewisi* or the nematode *Trichinella spiralis* under special circumstances. Under normal circumstances, *T. lewisi* can cause arthritis, abortion, and, in young rats, death (Duca 1939; Shaw and Dusanic 1973), and the fitness of individuals infected with *T. spiralis* is apparently reduced, since female mice show reductions in fecundity proportional to the intensity of infection (Weatherly 1971). Therefore, in nature, *T. lewisi* and *T. spiralis* should not be considered beneficial for their rodent hosts.

Examples of parasites that indirectly provide a benefit for their hosts are more clearly substantiated. A host, because of parasites, may enjoy freedom from competitors. For example, the nematode parasite *Parelaphostrongylus tenuis* has little effect on white-tailed deer but causes severe morbidity in moose, which frees deer from competition (Barbehenn 1969; R. C. Anderson 1972; but see Nudds 1990). In Africa, native grazers are protected from competition with livestock because the latter develop wasting disease (nagana) after infection with sylvatic trypanosomes. This disease has historically determined patterns of human settlement and is responsible for preserving vast areas of unspoiled wilderness (Ford 1971). These examples do not indicate a mutualistic relationship between host and parasite individuals. Although host species A individuals may benefit from a parasite species that reduces the level of competition with host species B, the individual parasites that provide the benefit for host species A individuals do not receive a benefit from the same host species A individuals; these parasite individuals are inside host species B individuals.

Another type of indirect benefit occurs when parasites protect hosts from other parasites. For example, it has been suggested that the presence of a cowbird brood parasite may generate a net benefit for host nestlings by eating botflies, which, under some circumstances, may be the major source of nestling mortality (Smith 1968). This phenomenon is even more likely to occur between parasites in similar host niches in which competition between parasites is strong. Parasites will increase both their own and their host's fitness by helping to prevent subsequent parasitism (Schad 1966; Holmes 1983; Freeland 1986). Heterologous immunity, in particular (see review in Christensen et al. 1987), is a plausible benefit of parasitism. Under conditions of concomitant immunity, for example, a host may be better off retaining established parasites if this affords protection against new, more pathogenic, infections. These interactions can appropriately be viewed as indirectly mutualistic, because parasites benefit the host individual that they establish in.

The benefit received by predators, due to the modification of prey by parasites,

is also indirect because it requires the involvement of a third party (the intermediate host prey item). Freedman (1990) considers that the predator and parasite are obligate mutualists if the persistence of the predator population is dependent on the presence of the parasite. In the present model, although the predator benefits, the parasite is not necessarily mutualistic in the evolutionary sense. The benefit that the host receives is based on the ingestion of parasitized prey, not the establishment of parasites. Individual parasites that become established may have a net negative impact on the predator, whereas parasites that are ingested, but fail to establish, clearly benefit the predator. This is clearly a case in which one's definition of mutualism has an impact on how the relationship is classified.

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## LITERATURE CITED

- Abrams, P. A. 1987. On classifying interactions between populations. *Oecologia* (Berlin) 73:272–281.
- Anderson, R. C. 1972. The ecological relationships of meningeal worm and native cervids in North America. *Journal of Wildlife Diseases* 8:304–310.
- Anderson, R. M. 1974. Mathematical models of host–helminth parasite interactions. Pages 43–69 in M. B. Usher and M. H. Williamson, eds. *Ecological stability*. Chapman & Hall, New York.
- Bailey, G. N. A. 1975. Energetics of a host–parasite system: a preliminary report. *International Journal for Parasitology* 5:609–613.
- Barbehenn, K. R. 1969. Host–parasite relationships and species diversity in mammals: an hypothesis. *Biotropica* 1:29–35.
- Boucher, D. H., S. James, and K. H. Keeler. 1982. The ecology of mutualism. *Annual Review of Ecology and Systematics* 13:315–347.
- Christensen, N. O., P. Nansen, B. O. Fagbemi, and J. Monrad. 1987. Heterologous antagonistic and synergistic interactions between helminths and between helminths and protozoans in concurrent experimental infection of mammalian hosts. *Parasitology Research* 73:387–410.
- Crofton, H. D. 1971. A quantitative approach to parasitism. *Parasitology* 63:179–194.
- Dobson, A. P., and A. E. Keymer. 1985. Life history models. Pages 347–384 in D. W. T. Crompton and B. B. Nickol, eds. *Acanthocephalan biology*. Cambridge University Press, Cambridge.
- Duca, C. J. 1939. Studies on the age resistance against trypanosome infections. II. The resistance of rats of different age groups to *Trypanosoma lewisi*, and the blood response of rats infected with this parasite. *American Journal of Hygiene* 29:25–32.
- Feare, C. J. 1971. Predation of limpets and dogwhelks by oystercatchers. *Bird Study* 18:121–129.
- Ford, J. 1971. The role of trypanosomiasis in African ecology: a study of the tsetse-fly problem. Clarendon, Oxford.
- Freedman, H. I. 1990. A model of predator–prey dynamics as modified by the action of a parasite. *Mathematical Biosciences* 99:143–155.

- Freedman, H. I., J. F. Addicot, and B. Rai. 1987. Obligate mutualism with a predator: stability and persistence of three-species models. *Theoretical Population Biology* 32:157–175.
- Freeland, W. J. 1986. Arms races and covenants: the evolution of parasite communities. Pages 289–309 in J. Kikkawa and D. Anderson, eds. *Community ecology, pattern and process*. Blackwell Scientific, Boston.
- Geraci, J. R., and D. J. St. Aubin. 1987. Effects of parasites on marine mammals. *International Journal for Parasitology* 5:407–414.
- Hadeler, K. P., and H. I. Freedman. 1989. Predator-prey populations with parasitic infection. *Journal of Mathematical Biology* 27:609–631.
- Holmes, J. C. 1982. Impact of infectious disease agents on the population growth and geographical distribution of animals. Pages 37–51 in R. M. Anderson and R. M. May, eds. *Dahlem workshop on population biology of infectious disease agents*. Springer, Berlin.
- . 1983. Evolutionary relationships between parasitic helminths and their hosts. Pages 161–185 in D. J. Futuyma and M. Slatkin, eds. *Coevolution*. Sinauer, Sunderland, Mass.
- Holmes, J. C., and W. M. Bethel. 1972. Modification of intermediate host behaviour by parasites. Pages 123–149 in E. U. Canning and C. A. Wright, eds. *Behavioural aspects of parasite transmission*. Academic Press, London.
- Holmes, J. C., and P. W. Price. 1986. Communities of parasites. Pages 187–213 in J. Kikkawa and D. J. Anderson, eds. *Community ecology: pattern and process*. Blackwell Scientific, Boston.
- Hulscher, J. B. 1973. Burying-depth and trematode infection in *Macoma balthica*. *Netherlands Journal of Sea Research* 6:141–156.
- Ivlev, V. S. 1961. *Experimental ecology of the feeding of fishes*. Yale University Press, New Haven, Conn.
- Janzen, D. H. 1985. The natural history of mutualisms. Pages 40–99 in D. H. Boucher, ed. *The biology of mutualism: ecology and evolution*. Oxford University Press, New York.
- Jones, A. W., and B. D. Tan. 1971. Effect of crowding upon growth and fecundity in the mouse bile duct tapeworm *Hymenolepis microstoma*. *Journal of Parasitology* 57:88–93.
- Kennedy, C. R. 1975. *Ecological animal parasitology*. Blackwell Scientific, Oxford.
- Keymer, A. D., D. W. T. Crompton, and B. J. Sahakian. 1983. Parasite-induced learned taste aversion involving *Nippostrongylus* in rats. *Parasitology* 86:455–460.
- Keymer, A. E. 1982. Density-dependent mechanisms in the regulation of intestinal helminth populations. *Parasitology* 84:573–587.
- Kuris, A. M. 1974. Trophic interactions: similarity of parasitic castrators to parasitoids. *Quarterly Review of Biology* 49:129–148.
- . 1980. An ecological classification of symbiotic associations. Paper presented at the 146th annual meeting of the American Association for the Advancement of Science, San Francisco.
- Lafferty, K. D. 1991. Effects of parasitic castration on the salt marsh snail, *Cerithidea californica*. Ph.D. thesis. University of California, Santa Barbara.
- Lincicome, D. R. 1971. The goodness of parasitism: a new hypothesis. Pages 139–227 in T. C. Cheng, ed. *Aspects of the biology of symbiosis*. University Park, Baltimore.
- Lozano, G. A. 1991. Optimal foraging theory: a possible role for parasites. *Oikos* 60:391–395.
- Margolis, L., G. W. Esch, J. C. Holmes, A. M. Kuris, and G. M. Schad. 1982. The use of ecological terms in parasitology (report of an ad hoc committee of the American Society of Parasitologists). *Journal of Parasitology* 68:131–133.
- McNamara, J. M., and A. I. Houston. 1987. Starvation and predation as factors limiting population size. *Ecology* 68:1515–1519.
- Moore, J. 1983. Responses of an avian predator and its isopod prey to an acanthocephalan parasite. *Ecology* 64:1000–1015.
- . 1984. Altered behavioral responses in intermediate hosts—an acanthocephalan parasite strategy. *American Naturalist* 123:572–577.
- Moore, J., and N. J. Gotelli. 1990. A phylogenetic perspective on the evolution of altered host behaviors: a critical look at the manipulation hypothesis. Pages 193–233 in C. J. Barnard and J. M. Behnke, eds. *Parasitism and host behavior*. Taylor & Francis, London.
- Munger, J. C., and W. H. Karasov. 1989. Sublethal parasites and host energy budgets: tapeworm infection in white-footed mice. *Ecology* 70:904–921.

- Nudds, T. D. 1990. Retroductive logic in retrospect—the ecological effects of meningeal worms. *Journal of Wildlife Management* 54:396–402.
- Read, C. P. 1951. The “crowding effect” in tapeworm infections. *Journal of Parasitology* 37:174–178.
- Schad, G. A. 1966. Immunity, competition, and natural regulation of helminth populations. *American Naturalist* 100:359–364.
- Shapiro, A. 1976. Beau geste? *American Naturalist* 110:900–902.
- Shaw, G. L., and D. G. Dusanic. 1973. *Trypanosoma lewisi*: termination of pregnancy in the infected rat. *Experimental Parasitology* 33:46–55.
- Slobodkin, L. B. 1974. Prudent predation does not require group selection. *American Naturalist* 108:665–678.
- Smith, N. G. 1968. The advantage to being parasitized. *Nature (London)* 219:690–694.
- Sprent, J. F. A. 1969. Evolutionary aspects of immunity in zooparasitic infections. Pages 3–62 in G. J. Jackson, R. Herman, and I. Singer, eds. *Immunity to parasitic animals*. Appleton-Century-Crofts, New York.
- Stephens, D. W., and J. R. Krebs. 1986. *Foraging theory*. Princeton University Press, Princeton, N.J.
- Symons, L. E. A. 1989. Pathophysiology of endoparasitic infection: compared with endoparasitic infestation and microbial infection. Academic Press, Sydney.
- Temple, S. A. 1987. Do predators always capture substandard individuals disproportionately from prey populations? *Ecology* 68:669–674.
- Weatherly, N. 1971. Effects on litter size and litter survival in Swiss mice infected with *Trichinella spiralis* during gestation. *Journal of Parasitology* 57:298–301.
- Wright, C. A. 1966. The pathogenesis of helminths in the Mollusca. *Helminthological Abstracts* 35:201–224.

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