

On the Capacity of Macroparasites to Control Insect Populations

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ABSTRACT: A graphical model of the population dynamics of macroparasites and their hosts is developed. Three principal means by which the parasites can be regulated are considered: reduction in host density as a result of parasite-induced host mortality, reduction in host density as a result of parasite-induced host sterility, and competition among parasites within multiply-infected hosts. The means by which parasites are regulated has a major effect on the degree to which they can depress host population densities. In particular, a parasite that sterilizes its host is expected to reduce host density more than one that causes an equivalent decline in host fitness through increased mortality. A special case of the model is developed for herbivorous insects that, in the absence of parasites, are limited by larval food resources. Parasites that are regulated via parasite-induced host sterility will control the insect populations below the level set by larval resources if the threshold host density for the parasites (N_T) is less than the ratio of carrying capacity to net reproductive rate of the insects (K/R). Data are presented showing that all three means of parasite regulation, but especially parasite-induced host sterility, can operate in *Howardula aoronymphium*, a nematode parasite of mycophagous *Drosophila* flies. Data from a field cage experiment show that, if these nematodes are regulated primarily via reductions in host density due to this sterility, the parameters N_T , K , and R are such that *Howardula* is likely to play an important role in controlling *Drosophila* populations. However, this conclusion must be tempered by the fact that these nematodes also cause increased host mortality and experience within-host competition, making the conditions for parasite control of the flies more stringent.

Keywords: biological control, *Drosophila*, host-parasite interactions, *Howardula*, insects, nematodes.

Although parasites often adversely affect the survival and fertility of infected host individuals, it is still not clear whether and under what circumstances parasites play an important role in regulating populations of their hosts (Scott and Dobson 1989; Gulland 1995; Hudson and Dobson 1995). Holmes (1982) has argued that in many

vertebrate species most of the individuals succumbing to parasites would fail to reproduce in any case because they are socially subordinate. Parasites may affect a substantial fraction of the host population in such cases without significantly influencing host density. Although examples of successful biological control show that parasites can have a major effect on their host populations (Myers et al. 1954), there are numerous cases of failed biological control programs (Strong et al. 1984; Stiling 1993). It is far from clear what conditions are required for parasite regulation of host populations. A reductionist approach, based on understanding the basic biology of host-parasite interactions, could provide an a priori indication of the likelihood of control (Waage and Mills 1992).

This article aims to identify some general features of host-parasite associations that determine the degree to which parasites can depress the density of their host populations. I first present a simple graphical method for exploring the population dynamics of interactions between hosts and macroparasites. This shows that the mechanism by which the parasites are regulated can have a major effect on their host populations. I then develop a more specific model of directly transmitted macroparasites of insects and determine under what conditions the parasites will depress host densities below carrying capacity, which is determined by larval food resources. Finally, I consider a specific host-parasite association involving mushroom-feeding *Drosophila* flies, and the nematode parasite *Howardula aoronymphium* and ask whether these nematodes play an important role in controlling populations of their hosts.

A Graphical Model of Host and Macroparasite Population Dynamics

In this section, I start with a basic model of the population dynamics of macroparasites and their hosts and then explore how various types of parasite population regulation influence the capacity of parasites to reduce host densities. The model examines host and parasite isoclines of zero net population growth; such a graphical analysis

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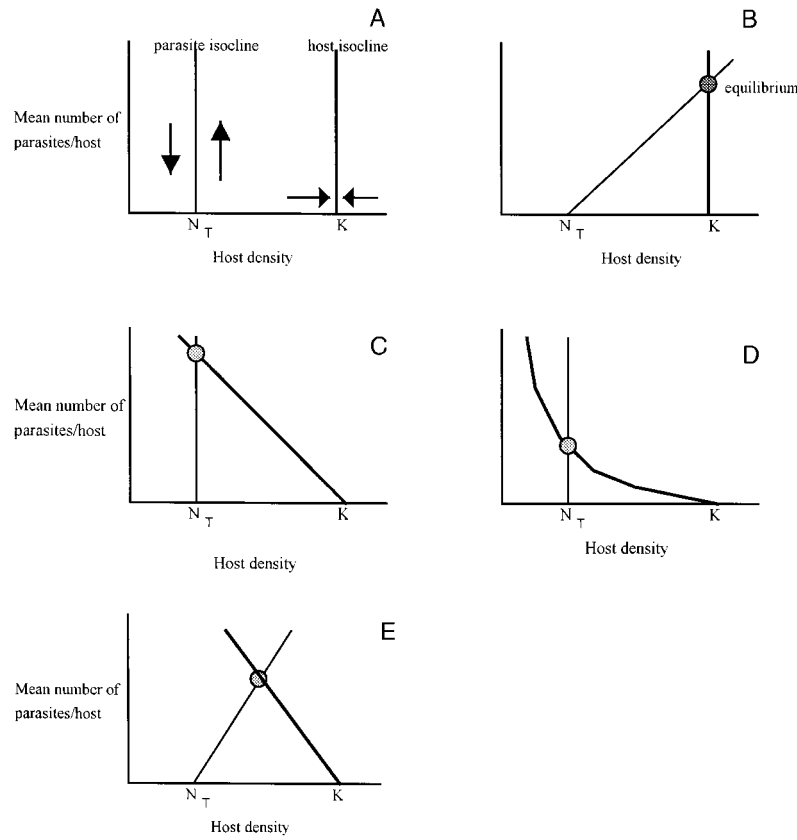


Figure 1: Graphical model of interactions between host and macroparasite populations. The arrows on either side of the host and parasite isoclines indicate whether populations will increase or decrease in that sector of the state space. In all cases, the equilibrium density of hosts in the absence of parasites is indicated by K , the carrying capacity. *A*, Parasite population not regulated. *B*, Parasite population regulated by competition among parasites within multiply-parasitized hosts. Parasites have no deleterious effects on their hosts. *C–E*, Parasites experience no adverse effects of competition within multiply-parasitized hosts. *C*, Host fertility declines as a function of the number of parasites per host. *D*, Hosts rendered completely sterile regardless of intensity of infection. *E*, Host (and therefore parasite) survival declines as a function of the number of parasites per host.

facilitates qualitative investigation of how changes in a model affect the outcome of an interaction between species (MacArthur 1970). The state-space approach to understanding host-macroparasite interactions was introduced by Anderson (1980), who considered the population dynamic consequences of parasite-induced host mortality and how variation in pathogenicity affected the equilibrium host population size (see also McCallum 1994). I will start with a simple but unrealistic model of host-parasite dynamics and then add various types of biological realism. The model uses the same conceptual framework as that introduced by Anderson (1980) but focuses on how the mechanism of parasite population regulation affects the degree to which the host population is depressed below carrying capacity. Particular attention is directed toward parasites of insects whose populations, in the absence of parasites, are limited by larval food. The focus throughout is on how para-

sites affect mean host densities at equilibrium, rather than the stability properties of this equilibrium.

The most important assumptions underlying the following models are that, first, in the absence of parasites, the host population is stably regulated at density K , the carrying capacity and, second, for the parasites there is a threshold density of hosts, N_T , below which the parasite cannot invade a community because its population growth rate is negative (Bailey 1975; but see Getz and Pickering 1983). In order to examine the effect of parasites on mean host density, I assume that K , N_T , and the parasite reproductive rate are such that the parasites and hosts can coexist (Anderson 1980).

For the simplest and most unrealistic model, suppose that the parasites—actually commensals in this case—have no deleterious effects on their hosts and that they experience no adverse consequences of crowding within hosts. Figure 1A presents the isoclines of zero net popu-

lation growth for both species as a function of host and parasite density. As can be seen, the host population stabilizes at K , regardless of parasite density. The parasites will invade only if $N_T < K$ (see also Anderson 1980; Anderson and May 1981). However, this condition also implies that the parasite density per host will increase indefinitely unless some density-dependent factor comes into play to limit the parasites.

In general, two sorts of density-dependent mechanisms can affect the parasites. First, parasites may experience density-dependent reductions in survival and reproduction because of either within-host competition in multiply-parasitized hosts or a defensive response by the host that increases with parasite density. Second, through their deleterious effects on host survival and reproduction, the parasites may drive the hosts to a density at which parasite population growth is no longer positive. These possibilities will be considered in turn below.

Anderson and May (1991, sec. 16.2.2) present a simple model of the dynamics of a direct life-cycle macroparasite. Here I further simplify their model by assuming that the parasite is an all-female species, that all transmission stages passed to the external environment survive to become infective, and that all parasites gaining entry to the host survive to sexual maturity. The net reproductive rate of such a macroparasite, R_0 , is equal to the probability that an infective stage parasite does infect a host times the number of offspring produced in successful infections. Using Anderson and May's notation, the probability of infection is the product of the life expectancy of infective stage parasites ($1/[\mu_2 + \beta N]$) and the probability per unit time that an infective stage will infect a host (βN), where μ_2 is the death rate of infective stages in the external environment, β is the transmission rate, and N is host density. The number of offspring produced in successful infections equals the rate of production of transmission stages (λ) times the life expectancy of adult parasites ($1/[\mu + \mu_1]$), where μ is the mortality rate of uninfected hosts and μ_1 is the increase in host mortality caused by parasites. Putting these together yields $R_0 = \lambda\beta N/[(\mu + \mu_1)(\mu_2 + \beta N)]$. Host threshold density (N_T) is defined as the density of hosts at which $R_0 = 1$. The parasite population declines when $N < N_T$ and increases when $N > N_T$. Setting $R_0 = 1$ yields $N_T = \mu_2(\mu + \mu_1)/\{\beta[\lambda - (\mu + \mu_1)]\}$. Because only a small fraction of a macroparasite's offspring infect a host, λ is likely to be much greater than $\mu + \mu_1$; therefore, $N_T \approx \mu_2(\mu + \mu_1)/\beta\lambda$. Finally, because the mortality rate of infective stages in the external environment (μ_2) and the transmission rate (β) are probably less dependent on parasite density than the other parameters, the expression for host threshold density can be further simplified to

$$N_T \propto (\mu + \mu_1)/\lambda. \quad (1)$$

That is, the host threshold density is directly proportional to host mortality rate and inversely proportional to the rate of parasite reproduction. This equation can be used to explore how various density-dependent factors influence the parasite isocline.

First, consider a parasite species that has no adverse effects on its host but that does experience within-host competition. If within-host competition causes the production of parasite transmission stages (λ) to decline with the number of parasites per host, as is often the case (Jaenike 1996a), then N_T increases with the mean number of parasites per host. In other words, the density of hosts required for positive growth of the parasite population increases with mean parasite density. Thus, the parasite isocline will be tilted to the right in the state-space diagram (fig. 1B). As can be seen in the figure, this leads to a stable equilibrium density of parasites, with the host equilibrium density remaining at K . The exact shape of the host and isoclines, whether straight lines or curved, depends on how the host or parasite fitness varies with parasite number per host and the statistical distribution of parasites among hosts (Jaenike 1996a). Whether these isoclines are straight (as presented in fig. 1A–C) or curved does not affect the qualitative conclusions of this analysis.

Next, assume that host fitness varies inversely with the number of parasites per host but that parasite fitness is unaffected. Thus, the parasite isocline is unaffected by mean parasite density in this case. This might occur if parasites complete their reproduction before host survival is affected or if they affect only the fertility of infected hosts. For simplicity, assume that the parasite-induced reduction in host survival or fertility is proportional to number of parasites per host and that these deleterious effects are manifested after the density-dependent phase of host population growth. For insect hosts, this could occur if resource limitation acts on larval stages, with parasites affecting the survival and fertility of adults. The host population growth can thus be modeled as a modified logistic (Anderson 1980):

$$dN/dt = rN(1 - N/K) - \mu_p PN, \quad (2)$$

where $rN(1 - N/K)$ represents growth during the resource-limited phase of the life cycle, P is the mean number of parasites per host, and μ_p is the per-parasite decrease in host fitness, whether via parasite-induced mortality or sterility. As a result of parasite-induced reductions in host fitness, the host isocline, $N_T = K(1 - P\mu_p/r)$, has a negative slope, that is, it is tilted to the left in the state-space diagram (fig. 1C). At equilibrium,

where the host and parasite isoclines cross, the hosts are reduced to density N_T , the threshold for parasite population growth.

For many species of insect-parasitic nematodes, a single parasite suffices to completely sterilize its host (Welch 1965). For instance, females of *Drosophila putrida* are always rendered completely sterile by the nematode *Howardula aoronymphium*, regardless of worm burden (Jaenike 1992). In such cases, the immediate effect of the parasites on host population growth is proportional to the fraction of hosts that are infected, rather than the number of parasites per host. Thus, the shape of the host isocline reflects the proportion of the host population that is infected, which will depend on the statistical distribution of parasites among hosts. If parasites are randomly distributed among hosts, then

$$dN/dt = rN(1 - N/K) - N(1 - e^{-p}), \quad (3)$$

where $1 - e^{-p}$ is the fraction of the population that is infected. Because the fraction of hosts parasitized increases with the mean number of parasites per host, the per capita effect of the parasites on the host population increases monotonically with parasite density, yielding a curved host isocline that is tilted to the left (fig. 1D). Although parasite density-dependent effects are not manifested at the level of individual parasitized hosts in this case, there is a net density-dependent effect at the level of the host population.

Suppose next that the primary effect of parasites is on host survival, resulting in a curtailed period of parasite reproduction, and that there is no within-host competition among parasites. In general, the rate of parasite-induced host mortality, μ_1 , increases monotonically with parasite load (Jaenike 1996a). From equation (1) above, it is clear that, as the rate of parasite-induced host mortality (μ_1) increases, so does the density of hosts required for parasite population growth. Thus, increased host mortality caused by greater parasite densities will cause the parasite isocline to be tilted to the right. Because the effects on host population growth of host mortality and host sterility are the same in this model, the host isocline will be tilted to the left, as in the preceding case. The host and parasite isoclines will therefore intersect at some point between N_T and K , the exact position depending on the relative strengths of density-dependent reductions in parasite versus host fitness (fig. 1E).

The models discussed to this point have focused on very general host-macroparasite interactions. I now consider more specifically certain types of insect-parasitic nematodes. Suppose that, in the absence of parasites, a population of insects is regulated by the abundance of larval food resources. Furthermore, let there be a simple

relation between the number of breeding adults in one generation and the number of emergent adults in the next, as illustrated in figure 2A. In this figure, K represents the equilibrium density of emergent adult insects, which is determined by the abundance of larval food resources, and R is the net reproductive rate, which equals the number of emergent offspring per breeding adult when resources are not limiting. It is assumed that resources do not become limiting until the density of larvae is above K . With respect to host-parasite interactions, N_T refers to the density of breeding adult insects required to produce a density of host larvae that allow the parasites to invade the community.

Given this simple scenario of insect regulation by larval food resources and/or macroparasites, under what conditions will the parasites reduce host populations below carrying capacity? There are two senses in which this could occur. First, do the parasites reduce the number of breeding insects below K ? Clearly, if there is any parasite-induced mortality or sterility that acts before breeding, the answer is yes. The second, and more fundamental, respect in which parasites can depress host populations is by keeping the number of emergent adults below K . This would indicate that larval resources no longer limit the density of the host population. For purposes of biological control of herbivorous insects whose larvae consume crop plants, this is the more important issue. Under what conditions does this occur?

Separate but functionally coupled isoclines for the number of breeding adults and the number of emergent adult offspring can be incorporated into a state-space diagram of insect host and macroparasite densities. In figure 2B and C, the number of emergent adult offspring is determined solely by the number of breeding adults, using the same functional relationship as that shown in figure 2A. In this figure, functionally paired densities fall along the same horizontal line. For instance, a breeding density of K/R yields a density of K emergent offspring. If the number of breeding adult insects exceeds K/R , then the number of emergent adults in the next generation remains at K , regardless of parasitism. However, if the number of breeding adults falls below K/R , then the number of emergent adult offspring will be directly proportional to the number of breeding adults, which, as shown previously, varies inversely with parasite density. Thus, the number of emergent adults will no longer be limited by larval food. Whether or not the number of breeding adults falls below K/R depends on where the host and parasite isoclines intersect.

Figure 2B illustrates a case in which the parasites are regulated solely by parasite-induced host sterility, so that the parasite isocline remains vertical. Because the host

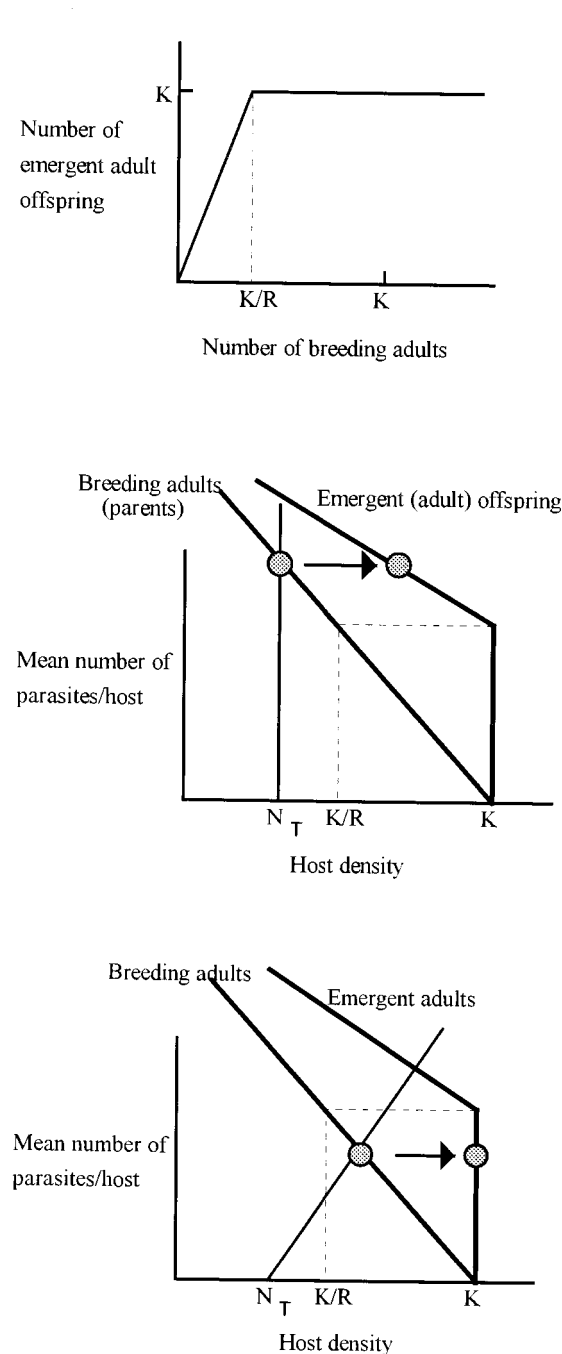


Figure 2: Model of parasite control versus food limitation of an insect host population. *A*, The number of adults produced each generation from a given quantity of resource depends on the number of breeding adults the previous generation. The maximum number of emergent adults (K) is limited by resource availability. The slope of the line at low density is R ; hence, the number of emergent adults plateaus when the density of breeding adults reaches K/R . *B*, *C*, State-space diagrams of an insect host and macroparasite populations. For each density of breeding adult insects there is a corresponding density of emergent adult offspring (as in *A*), which can be determined by moving

A and parasite isoclines intersect at a host density below K/R in the case shown, the number of emergent adults is controlled by parasites rather than resource availability. Within-host competition among parasites (or a parasite-density-dependent defensive response by the host) will tilt the parasite isocline to the right. For the example shown in figure 2*C*, the host and parasite isoclines intersect at a host density greater than K/R . Therefore, the number of emergent adults at equilibrium is not affected by parasitism; rather, they are limited by larval resources. Thus, within-host competition among parasites makes more stringent the conditions under which parasites will limit host populations. Similarly, because parasite-induced host mortality tilts the parasite isocline to the right, a parasite that affects host survival is less likely to limit host densities than one that causes an equivalent decline in host fitness through reductions in host fertility, all else being equal.

In summary, the models presented above show that the degree to which parasites can depress host densities depends on how the parasites themselves are regulated, whether by competition among parasites within multiply-infected hosts (fig. 1*B*), parasite-induced host sterility (fig. 1*C*), or parasite-induced host mortality (fig. 1*E*). In the next section, I evaluate the role these three mechanisms may play in regulating populations of the *Drosophila*-parasitic nematode *H. aoronymphium*. I also present data from a field cage experiment on host carrying capacity (K), net reproductive rate (R), and threshold density (N_T) for *H. aoronymphium* to determine if these parameter values support the possibility that *Howardula* plays a role in controlling *Drosophila* populations.

Howardula aoronymphium: A Nematode Parasite of Mycophagous *Drosophila*

Howardula aoronymphium (Allantonematidae: Tylenchida) infects mycophagous *Drosophila* belonging to the *quinaria* and *testacea* species groups. Nematodes classi-

horizontally from the breeding adult isocline to emergent adult isocline. The arrows show the functional relationship between the density of breeding adults and the density of their emergent offspring when the system is at equilibrium. *B*, The parasites are regulated via parasite-induced host sterility, causing the parasite isocline to be vertical and the host isocline to be tilted to the left. Because the isoclines intersect at N_T , the parasites will control the number of emergent hosts below K if $N_T < K/R$. *C*, The parasites are regulated via parasite-induced host mortality, causing both the host and parasite isoclines to be tilted. In the case shown, the isoclines intersect at a host density greater than K/R , showing that food availability, rather than parasites, limits the number of emergent adults each generation.

Table 1: Effect of parasite density on host survival

Trial	Host species ^{genetic marker}	1 parasite/fly		>1 parasite/fly		n	χ^2	Pr*
		Released	Recaptured	Released	Recaptured			
1	<i>D. putrida</i> ^{brown}	57	85	30	39	211	.098	.75
	<i>D. putrida</i> ^{red}	34	36	16	15	101	.004	.95
	<i>D. neotestacea</i> ^{red}	26	21	12	5	64	.657	.42
	<i>D. neotestacea</i> ^{apricot}	33	3	9	0	45	.022	.88
2	<i>D. putrida</i> ^{brown}	16	10	81	74	181	.443	.51
	<i>D. neotestacea</i> ^{red}	47	9	26	2	84	.641	.42
3	<i>D. putrida</i> ^{brown}	34	63	50	45	192	5.334	.021
	<i>D. neotestacea</i> ^{red}	29	24	71	23	147	5.828	.016
4	<i>D. putrida</i> ^{brown}	14	34	1	0	49	.181	.67
	<i>D. neotestacea</i> ^{red}	14	0	5	0	19
5	<i>D. putrida</i> ^{brown}	22	13	30	5	70	3.665	.056
	<i>D. neotestacea</i> ^{red}	17	9	28	2	56	5.236	.022

Note: Data for numbers released were obtained from a sample of 100 flies collected just prior to release; not all of these flies were parasitized. Chi-square values test the hypothesis that flies parasitized with a single worm were as likely to be recaptured as flies with more than one motherworm. A significant value indicates that the multiply-parasitized flies experienced significantly higher rates of parasite-induced mortality. Data are from an earlier study (Jaenike et al. 1995).

* Combined probability (Fisher 1954): $\chi^2 = -2\sum \ln Pr = 36.0$ (22 df); $P = .03$.

fied as *H. aoronymphium* have been identified in Europe (Welch 1959), North America (Montague and Jaenike 1985), and Japan (Kimura and Toda 1990), although it is possible that these different geographical populations represent distinct biological species. Analysis of mtDNA sequence variation has revealed that there are at least two species of *Howardula* that parasitize mycophagous *Drosophila* in the eastern United States (Jaenike 1996b). The results reported here deal only with the more common species, which, at the molecular level, is nearly identical to European *H. aoronymphium* (D. D. Shoemaker, unpublished data). Because *H. aoronymphium* was originally described on the basis of European specimens, the nematodes considered in this study are referred to as *H. aoronymphium*. Molecular analysis of mtDNA revealed that all strains and selected populations used in the studies described below had identical haplotypes (D. D. Shoemaker, unpublished data). In eastern North America, the principal hosts of *H. aoronymphium* are *D. falleni* and *D. recens* of the *quinaria* group and *D. neotestacea* and *D. putrida* of the *testacea* group (Jaenike 1992).

Infection occurs when an inseminated female nematode pierces and invades through the cuticle of a fly larva. After emergence of the adult fly, the motherworm grows rapidly and begins to produce larval offspring, which are then expelled from the host via the anus or ovipositor when the fly visits a mushroom. The larval worms then mate within the mushroom and begin the cycle anew. *Howardula aoronymphium* therefore has a direct life cycle with no intermediate hosts.

In terms of the graphical models presented above, the *Drosophila* (host) isocline could be affected by parasite-induced host sterility and mortality, and the *Howardula* (parasite) isocline could be affected by parasite-induced host mortality and within-host competition among parasites. The following sections examine whether these three processes are operative in this particular host-parasite association and whether the parameters K , N_T , and R are such that parasite control of these *Drosophila* populations is possible.

Parasite-Induced Host Mortality

In a series of field experiments, Jaenike et al. (1995) showed that infection of *D. putrida* and *D. neotestacea* with *H. aoronymphium* significantly increases the rate of adult mortality in the field, both for singly- and multiply-infected hosts. Here I reanalyzed these data to determine whether the rate of parasite-induced host mortality depends on worm burden. In field trials 3 and 5, multiply-infected individuals of both *D. putrida* and *D. neotestacea* were significantly less likely to be captured (and hence suffered greater mortality) than flies infected with just one motherworm (table 1). In the other three trials, the effects were not individually significant. However, the combined probability from the separate trials indicates that multiply-parasitized flies suffer significantly greater mortality than singly-infected flies ($P = .03$).

For the *Drosophila-Howardula* association, the per capita rate of parasite-induced host mortality increases with the mean number of parasites per host because, first, a

greater proportion of the flies are parasitized—flies infected with just one motherworm suffer substantially greater mortality than uninfected flies (Jaenike et al. 1995)—and, second, the rate of parasite-induced host mortality is greater for multiply-infected flies. As noted above, such parasite-induced host mortality will affect both the host and parasite isoclines, causing them to intersect somewhere between N_T and K .

Parasite-Induced Host Sterility and Within-Host Competition among Parasites: Methods

The following experiments were designed to examine how the density of parasites within hosts affected motherworm growth and fly fertility, as well as to assay genetic variation for host specificity within *H. aoronymphium*. Additional details on these experiments will be presented elsewhere.

Experiment 1. Parasitized individuals of *D. falleni*, *D. recens*, *D. putrida*, and *D. neotestacea* were collected from two areas in western New York in September 1992. The flies were placed together in a large population cage, and their parasitized offspring used to establish 15 populations of *H. aoronymphium* that were maintained in the laboratory using *D. falleni*, *D. putrida*, or both species as hosts. Experimental infections were obtained by grinding parasitized flies in *Drosophila* Ringer’s solution, applying the resulting larval nematode slurry to a piece of *Agaricus bisporus* mushroom, and adding 20 eggs of *D. falleni*, *D. putrida*, *D. neotestacea*, or *D. recens* to serve as host. Emergent adults were kept on Instant *Drosophila* Medium at 21°C for 1 wk and then frozen at –20°C. Numbers of motherworms, the size of all motherworms (longitudinal section area), and the number of mature fly eggs (stage 10 or later) were determined in all flies. Size was determined by drawing the worm’s outline using a camera lucida and measuring this area with a planimeter.

Experiment 2. In September 1994, males and females of *D. falleni*, *D. neotestacea*, and *D. putrida* were collected in Monroe County, New York, and placed individually in vials with unparasitized lab-reared females of the same species. All cultures established with parasitized flies from the wild were kept, yielding 35 independently derived strains of *H. aoronymphium*. Experimental infections with each strain were conducted as in experiment 1, except that the host species tested—*D. putrida*, *D. neotestacea*, and *D. falleni*—were reared together. Details of these procedures will be presented elsewhere.

As a measure of motherworm fecundity, the density of larval nematodes (the motherworm’s offspring) within hosts was estimated visually in both experiments. Parasit-

Table 2: Correlation between worm burden and *Drosophila* female fertility

<i>Host species and experiment</i>	N	r_s	P	<i>Intercept</i>	<i>Slope</i>
<i>D. falleni:</i>					
1	233	–.25	.0001	9.23	–1.31
2	140	–.17	.04	19.76	–2.02
<i>D. neotestacea:</i>					
1	152	–.24	.003	.90	–.07
2	163	–.35	.0001	17.60	–6.84
<i>D. putrida:</i>					
1	248	.01	.82	.009	–.0002
2	204	–.18	.008	16.16	–6.56
<i>D. recens:</i>					
1	207	–.05	.45	5.79	–.33

Note: Female fertility was measured as number of mature eggs (stage 10 or later) carried by 1-wk-old flies.

ized flies were categorized into four classes: class 0 flies had no larval worms, class 1 had 1–20, class 3 had 21–500, and class 4 had 501–2,000 larval worms. To eliminate the effect of motherworm number on larval density, the density of larval nematodes was regressed against motherworm size for all flies infected with just a single motherworm. This tests whether motherworm size is an appropriate, albeit indirect, measure of fecundity.

Statistical Analysis. For each host species, the number of mature fly eggs per female was correlated with the number of motherworms using PROC CORR; motherworm size was regressed against worm number per host with PROC REG; and larval density class was correlated with motherworm size with PROC CORR (SAS Institute 1994). Data were pooled across the 15 nematode populations and 35 nematode strains for experiments 1 and 2, respectively.

Parasite-Induced Host Sterility: Results

Experiments 1 and 2 both yielded a range of worm burdens similar to those seen in nature, that is, between one and 10 motherworms per fly (Jaenike and Anderson 1992; Jaenike 1994). The fertility of parasitized female *D. falleni* and *D. neotestacea* declined significantly with the number of motherworms per fly in both experiments (table 2). For *D. putrida*, the correlation was insignificant in experiment 1 (because almost every fly was completely sterile, regardless of worm burden), but it was significantly negative in the second experiment. For *D. recens*, a minor host studied only in experiment 1, there was a negative but nonsignificant correlation between worm

Table 3: Correlation between motherworm size and the density class of larval nematodes in flies infected by a single motherworm

Experiment	Host species	N	r_s	P
1	<i>D. falleni</i>	114	.16	.096
	<i>D. recens</i>	9	.57	.14
	<i>D. neotestacea</i>	23	.58	.003
	<i>D. putrida</i>	141	.21	.014
2	<i>D. falleni</i>	102	.25	.010
	<i>D. neotestacea</i>	171	.15	.044
	<i>D. putrida</i>	216	.26	.0001

burden and female fertility. The fertility of all flies, whether parasitized or not, was greater in experiment 2, probably because they were provided with fresh mushrooms to feed on during the 1-wk aging period. The two experiments therefore show that the deleterious effect of *Howardula* on their *Drosophila* hosts can occur under different nutritional conditions.

Field studies of the three primary hosts of *H. aoronymphium* have shown that parasitized females of *D. putrida* and *D. neotestacea* are usually rendered completely sterile by these parasites, while parasitized females of *D. falleni* are on average about 50% as fertile as unparasitized flies (Jaenike 1992). Both the field studies reported previously and the controlled laboratory experiments presented here show that *Howardula* have a major effect on the host isocline through parasite-induced host sterility.

Within-Host Competition among Parasites: Results

Among the flies that were infected with just one motherworm, there was a positive correlation between motherworm size and larval nematode density for all host species in both experiments (table 3). These correlations were statistically significant ($P < .05$) in five out of seven cases. In one of the nonsignificant cases (*D. neotestacea* in experiment 1), the sample size was very small ($n = 9$), resulting in a test with low power. These results indicate that a motherworm's size is a suitable indicator of her fecundity.

In both experiments and for all host species, there were highly significant negative correlations between the size to which motherworms had grown in 1-wk-old flies and the number of motherworms per fly ($P < .0001$ for all host species in both experiments). Data for experiment 1 are shown in figure 3; experiment 2 yielded very similar results. These results indicate that, when the prevalence of parasitism is high, which results in substantial numbers of multiply-parasitized flies, within-host competition among parasites will affect the parasite isocline.

Can *Howardula* Control *Drosophila* Populations? The Parameters K , R , and N_T

The simple model outlined above indicates that parasite control of an insect population below a level set by the insect's larval resources requires that the threshold density for the parasites (N_T) be less than the host's carrying capacity (K) divided by its net reproductive rate (R). Data obtained from a field cage experiment on *Drosophila-Howardula* population dynamics can be used to address this question. In the experiment, the details of which have been published (Jaenike and Anderson 1992), a fixed number of wild-caught *D. falleni* (21 males and 30 females) and *D. neotestacea* (eight males and 22 females) adults were introduced into each of 15 field cages, and the number of mushrooms was varied among cages (either one, 5, or 15). Based on the prevalence of parasitism in the populations from which these flies were obtained, the number of motherworms introduced per cage was approximately 10. One can therefore examine the input and output of *Drosophila* adults and *Howardula* motherworms on a per mushroom basis (table 4). For simplicity, I consider the combined densities of the two *Drosophila* species in determining R , K , and N_T .

The lowest density of flies per mushroom occurred in the 15-mushroom cages, and in these about 5.4 adult *Drosophila* females emerged per female introduced. Thus, the net reproductive rate, R , was about 5 in this experiment.

To determine K , the carrying capacity per mushroom, I took the inverse of the input and output of females flies per mushroom for each cage and obtained a Lineweaver-Burk plot of these transformed data (Lehninger 1982). (Data from one of the one-mushroom cages was omitted because the number of flies emerging from that mushroom was about 1/3 of the others in that category.) The Y-intercept in such a plot yields the inverse of the carrying capacity, by analogy with a simple model of enzyme kinetics in which the parents can be viewed as the substrate and offspring as product. A linear regression of output⁻¹ against input⁻¹ is highly significant ($r^2 = 0.91$, $P < .0001$), yielding a carrying capacity of about 66 female flies per mushroom (95% confidence limits = 57–78; PROC REG; SAS Institute 1994). Thus, the quantity K/R is approximately 12 in this experiment. The model presented above indicates that if the host and parasite isoclines intersect at a host density less than this value, then the parasites can control their insect hosts below carrying capacity.

If the *Howardula* population is regulated solely by changes in *Drosophila* density brought about by parasite-induced sterility, then the host and parasite isoclines are expected to intersect at a host density of N_T . Data from

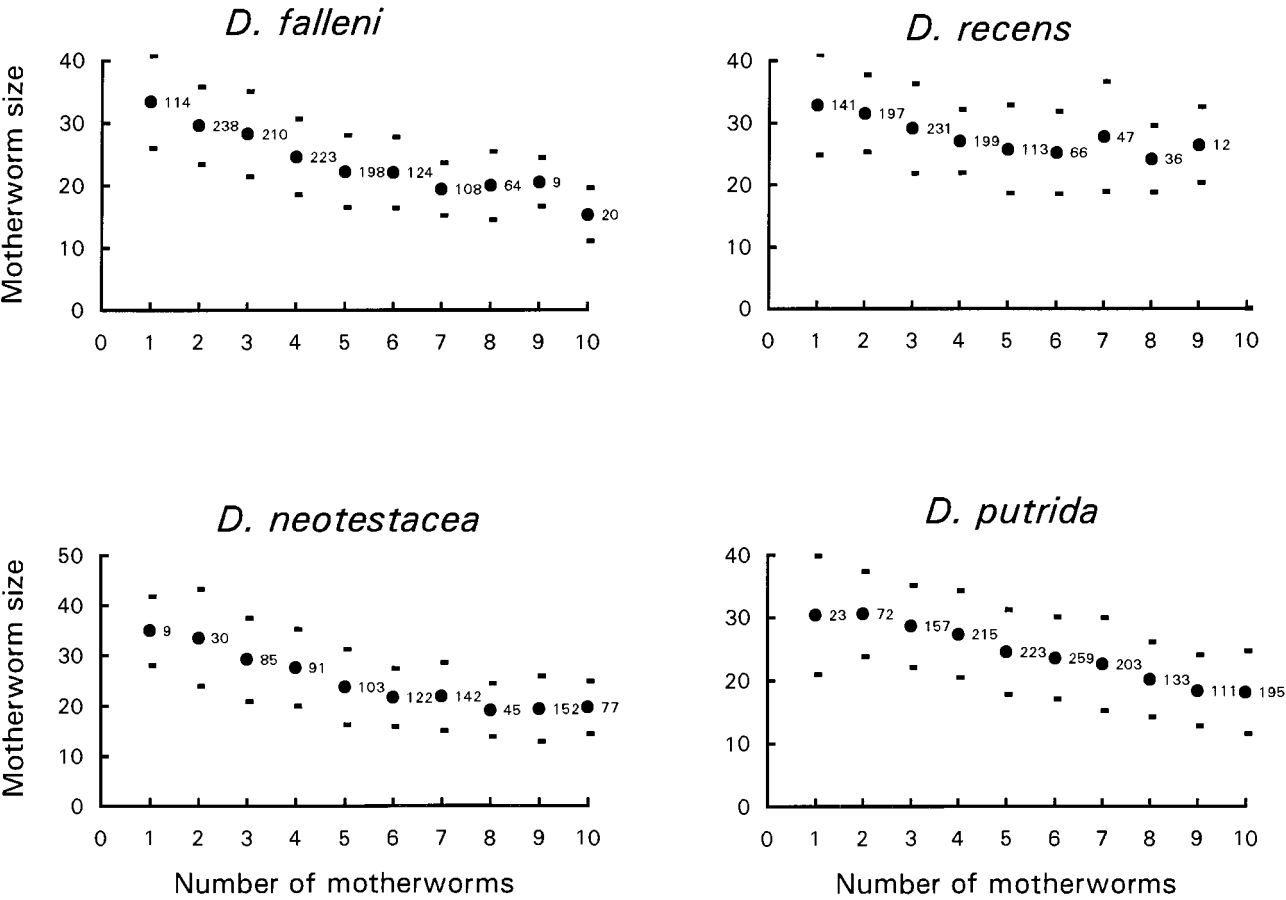


Figure 3: Motherworm size (longitudinal section area in 1-wk-old adult flies) as a function of the number of motherworms per fly in experiment 1. Mean \pm 1 SD (mm²) is shown for each intensity of parasitism. Numbers beside each point indicate sample sizes.

the field cage experiment can be used to determine the approximate magnitude of N_T . It can be seen in table 4 that, at the lowest density of *Drosophila* tested (3.5 female flies per mushroom), the *Howardula* experienced nearly a 30-fold increase in numbers between the parental and

Table 4: Input and output of female *Drosophila* and *Howardula* motherworms per mushroom in a field cage experiment

Number of mushrooms per cage	Input per mushroom		Output per mushroom	
	Female <i>Drosophila</i>	<i>Howardula</i> motherworms	Female <i>Drosophila</i>	<i>Howardula</i> motherworms
1	52	9.8	58	90.5
5	10.4	2	34	33.6
15	3.5	.7	19	19.7

Note: *Drosophila* species included *D. falleni* and *D. neotestacea*. Data are from an earlier study (Jaenike and Anderson 1992).

offspring generations. Thus, in environments like these field cages, N_T must be considerably lower than 3.5 flies per mushroom, which is substantially less than K/R (≈ 12). Thus, if *Howardula* populations are regulated primarily through their effects on host fertility, then it appears likely that they can control populations of their *Drosophila* hosts to levels below those set by larval resources. This conclusion must be tempered by the data presented previously that *Howardula* populations may also be regulated by parasite-induced host mortality and within-host competition among motherworms, both of which make the conditions for parasite control of *Drosophila* populations more stringent.

Discussion

This article presents a graphical model for investigating population-level interactions between macroparasites and their hosts, focusing particularly on insects whose popu-

lations are limited by larval food resources when parasites are absent. The analysis shows that the degree to which parasites depress host densities depends on how the parasite populations are regulated, whether by parasite-induced host sterility, parasite-induced host mortality, or competition among parasites within multiply-infected hosts.

In the context of the graphical model, parasite-induced host sterility acts by tilting the host isocline so that it intersects the parasite isocline at N_T , the threshold host density for invasion of the parasite. In this case, the parasites can have a major effect on equilibrium host density. Within-host competition among parasites tilts the parasite isocline to the right, leaving the host isocline and equilibrium density unaffected, so that the host and parasite isoclines intersect at host density K , the carrying capacity. Finally, parasite-induced host mortality causes the host isocline to tilt left and the parasite isocline to tilt right, causing them to intersect between N_T and K . Thus, regulation of the parasites by parasite-induced host sterility has the greatest effect on host density, regulation by competition among parasites in multiply-infected hosts has the least effect, and regulation via parasite-induced host mortality is intermediate. Anderson (1980) and McCallum (1994) examine how variation in the rate of parasite-induced host mortality (μ_1) affects the equilibrium abundance of the host and conclude that intermediate values of μ_1 have the greatest effect.

The model presented above assumes that parasites are randomly distributed among hosts. However, macroparasites generally exhibit aggregated distributions among hosts (Anderson and Gordon 1982), and this can affect the degree to which parasites depress host densities at equilibrium. If host fitness declines exponentially to zero as within-host parasite density increases, then, for a given mean number of parasites per host, aggregation of parasites results in greater mean host fitness but lower mean parasite fitness. For such an exponential fitness function, parasite aggregation reduces the leftward tilt of the host isocline, while increasing the rightward tilt of the parasite isocline. As a result, the host and parasite isoclines will intersect closer to K , thus lessening the degree to which the parasites depress equilibrium host density (see also Anderson 1980). Sigmoid fitness functions, however, can result in greater parasite depression of host density. A detailed discussion of the effect of parasite aggregation on host and parasite populations is presented elsewhere (Jaenike 1996a). In any case, the complications arising from parasite aggregation do not qualitatively affect the general way in which the different modes of parasite population regulation affect the host and parasite isoclines.

It has been argued that parasites could maximize their individual fitness by utilization of host resources com-

mitted to reproduction (causing host castration) because loss to parasites of somatic tissue or resources directed to those tissues would cause increased host, and therefore parasite, mortality (Kuris 1974; Baudoin 1975; Obreski 1975). The model presented in this article shows that, for comparable reductions in host fitness, parasites that cause host castration are likely to reduce host densities more than parasites that increase the rate of host mortality. Thus, selection acting at the level of individual parasites may amplify the effects of parasites on host population size.

Many macroparasites of invertebrates commonly sterilize their hosts (Welch 1965; Kuris 1974; Baudoin 1975). For instance, allantonematid nematodes, which parasitize various Thysanoptera, Coleoptera, Lepidoptera, Diptera, and Hymenoptera, generally reduce the fertility of their hosts and do not kill them outright (Welch 1965; Poinar 1975). This suggests that nematode parasites could play an important role in controlling their insect hosts. For example, the nematode *Deladenus siricidicola*, which sterilizes females of the wood wasp *Sirex noctilio*, has proven to be a highly effective control agent of this forest pest in Australia (Bedding 1993). Other nematodes are known to sterilize such insect pests as blueberry thrips, bark beetles, and face flies (Nickle 1963, 1967; Nickle and Wood 1964) and could, therefore, play an important role in controlling populations of these species.

The graphical models show that if parasites are regulated via parasite-induced host sterility or mortality, then the degree to which host density is depressed by the parasites will depend on the difference between K and N_T . For insect host species that are subject to potential food limitation of larvae, parasites will control the hosts below carrying capacity (K) if the host and parasite isoclines intersect at a value less than K/R , where R is the net reproductive rate of the hosts. If the parasites are regulated solely by parasite-induced host sterility, this simplifies to the requirement $N_T < K/R$. This conclusion emphasizes the usefulness of estimating these parameters for making a priori assessments of the prospects for biological control. Although N_T is central to understanding the dynamics of host-parasite interactions (Anderson and May 1978), this parameter has rarely if ever been measured in natural populations (but see Jaffee et al. 1992 for a realistic soil microcosm example).

The findings presented in this article show that all three of the mechanisms identified above may contribute to regulation of the *Drosophila*-parasitic nematode *Howardula aoronymphium*. First, these nematodes sterilize females of *D. putrida* and *D. neotestacea* and substantially reduce the fertility of female *D. falleni* (Jaenike 1992, 1996c). In the wild, the parasite-induced sterility of the former two species is independent of worm burden since

a single nematode almost always renders these flies completely sterile. Because the distribution of the number of motherworms per fly is approximately random in natural populations (Jaenike 1994), the fraction of *D. putrida* and *D. neotestacea* capable of reproduction will drop asymptotically to zero as the density of parasites increases. The host isocline will therefore be curved, as shown in figure 1D. The laboratory results presented here show that the fertility of parasitized females of *D. falleni* declines with worm burden (table 2), and this will contribute to the leftward tilt of the *D. falleni* isocline. In the laboratory, females of *D. putrida* and *D. neotestacea* show a decline in fertility with worm burden (table 2), but this is probably seldom relevant under natural conditions, where almost all parasitized flies are completely sterile (Jaenike 1992). The greater fertility of parasitized flies maintained in the laboratory is probably due to greater food availability under these conditions.

Second, the mean mortality rate in a *Drosophila* population will increase with parasite density for two reasons. The primary effect is that parasitized flies, even those infected with just one motherworm, experience significantly elevated mortality (Jaenike et al. 1995). As mean parasite density increases, so does the prevalence of parasitism in the host population, thus increasing the overall mortality rate of the flies. In addition, under some environmental conditions, the rate of parasite-induced mortality increases with the number of parasites per host (table 1). Because parasitized females of *D. putrida* and *D. neotestacea* are completely sterile regardless of worm burden, the elevated mortality of parasitized flies will not directly affect the population dynamics of these two species. However, parasite-induced host mortality will reduce the reproductive rate of the nematodes. Because the rate of host mortality can increase with worm burden, this will cause the parasite isocline to be tilted the right.

Finally, competition among parasites within multiply-infected hosts is also operative (fig. 3). Such competition causes the parasite isocline to be tilted to the right. Thus, empirical studies on mycophagous *Drosophila* and the nematode *Howardula aoronymphium* demonstrate that the host and parasite isoclines will be tilted to the left and right, respectively. Consequently, the number of breeding flies will be held somewhere between N_T and K by these parasites.

There remains the question of whether *Howardula* can control populations of *Drosophila* to levels below those set by larval resources. In the simple model presented above, it was shown that this will occur if the host and parasite isoclines intersect at a host density less than K/R . Data from the field cage experiment indicated that, for *Agaricus bisporus* mushrooms, $K/R \approx 12$ and that N_T is substantially less than 3.5 female flies per mushroom be-

cause the nematodes exhibited a net reproductive rate of about 30 even at this lowest host density tested. Thus, in small field cages with *A. bisporus* breeding sites, N_T is considerably less than K/R .

If *Howardula* populations were regulated solely by parasite-induced host sterility, the field cage results would suggest that *Howardula* can control *Drosophila* populations below the carrying capacity set by larval resources. However, both within-host competition among motherworms and, more important, parasite-induced host mortality are probably also important in regulating these parasites, and these factors will cause the parasite isocline to be tilted to the right, perhaps causing it to intersect the host isocline at a host density greater than K/R . Thus, it remains to be determined whether *Howardula* plays an important role in controlling populations of their *Drosophila* hosts. Nevertheless, the types of models and experiments described here might be used in other situations to assess the prospects for macroparasite control of insect pests, especially for parasites whose primary effect is reduced host fertility.

Waage and Mills (1992) have stressed that, whereas much of classical biological control is largely an empirical discipline, it would be desirable to incorporate basic ecological theory into the search for effective control agents. They identify two approaches—holistic and reductionist—by which such theory can be applied to biological control programs. The holistic approach emphasizes population-level observations of a pest species and various enemies under natural conditions. A natural enemy that coexists with the pest at low density is more likely to be an effective control agent than one that coexists with the pest at high density. A drawback to this holistic approach is that, without control over other variables, one cannot be certain that a particular natural enemy is responsible for control in the low-density situation. The reductionist approach focuses instead on parameters that affect individual-level interactions between the two species, such as searching efficiency and handling time. Waage and Mills (1992) note, however, that it is often difficult to measure all of the relevant parameters and that, in any case, it is unrealistic to break these interactions down into specific component parts.

The approach I have advocated represents a middle ground between the holistic and reductionist. Rather than focusing on a large number of parameters that affect interactions between host and parasite individuals, I stress that three demographic parameters— N_T , K , and R —and the means by which parasite populations are regulated are of central importance in determining the degree to which macroparasites can depress host densities. The theoretical analysis indicates that parasites that sterilize rather than kill their hosts are more likely to be

effective control agents. More generally, such parasites, though rarely studied, could play an important role in determining the abundance of various insects in natural communities.

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