Evolutionary Biology of Host-Parasite Relationships: Theory Meets Reality
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Parasite-host modeling meets reality: adaptive peaks and their ecological attributes

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1. INTRODUCTION

In this review, we seek to describe the fascinating diversity of feeding strategies exhibited by parasites and other consumers. We begin by discussing the four main types of consumers treated by population models: predators, parasitoids, microparasites and macroparasites. These we view as adaptive peaks. We then explore key differences between these strategies. A primary dichotomy is the number of hosts an individual consumer attacks throughout a particular life-history stage (separating predators from parasites). A secondary dichotomy is how an individual attack affects host fitness (allowing the treatment of parasitoids). We then discuss the rationale for Anderson and May dividing parasites into macroparasites and microparasites according to intensity-dependent pathology (Anderson and May 1979, May and Anderson 1979). Because their dichotomy is one of the most powerful existing paradigms shaping current thinking about parasites, we consider whether it is sufficient to adequately describe all parasite strategies. In addition, we note that its fundamental categorical basis, intensity, is easy to misapply as parasite body size or taxonomy. We recognize how body size is very strongly associated with the different adaptive syndromes. We further argue that relative and absolute sizes of hosts and consumers are the primary selective gradients responsible for the evolution of different consumer strategies. We then introduce a fourth dichotomy by recognizing that (for parasites) it is important to determine whether the parasite needs the host to die to further the parasite's development (allowing the ability to distinguish, categorically, between parasitoids and parasitic castrators). We end by uniting all four dichotomies to define twelve distinct consumer strategies. This creates many previously unrecognized consumer types within a four-dimensional parameter space for which we are able to assign names and examples. The end result is a suite of distinct consumer strategies representing eight types of parasites and four types of predators.

2. AN EVOLUTIONARY DEFINITION OF INTIMATE INTERSPECIFIC INTERACTIONS

Here, we seek an evolutionary basis for the determination of the types of consumer interactions. Sewall Wright's (1931) concept of adaptive peaks influences our approach: among species, selection for similar optima for the interacting cluster of traits that comprise a life history strategy will yield analogous adaptive peaks; an adaptive syndrome across species. The consumer interactions of interest to us are antagonistic in that one of the interactors, the consumer, feeds on or otherwise takes resources from the other, the host/prey. The interactions we consider are specific to a particular life history phase of the consumer. A parasite's relations with its intermediate host when it is a larva may be of a very different nature from its relations with its definitive host. Also, the progeny resulting from asexual reproduction in a host will be considered the equivalent of a single individual since they are genetically identical. Our proposed dichotomies are applicable across all taxa; including animal-plant, plant-pathogen, phage-bacteria and microbial/helminth-animal host relations, the last of which form the bulk of our examples. Also, to avoid neologisms, we take terms for the resulting types of consumers from the literature that sometimes has used them in a taxon-specific manner, but for which the definition of the concept can, in fact, readily apply to other organisms (Kuris 1974, 1997).

Population biology has long explored the interesting dynamics that can occur when one species feeds on another. The most widely known models are derivatives from Lotka-Volterra predator-prey models (Lotka 1925; Volterra 1926). An important component of these models, the functional response (Holling 1966), was a direct attempt to deal with how predators eat many prey. This is a key difference from disease models, where it is clearly implied that a parasite exploits only one host during a particular phase of its ontogeny. Therefore, a primary dichotomy for dividing consumers is the number of hosts or prey attacked during a particular life-history phase. Consumers that attack one host are generally termed parasites. Consumers that attack more than one prey are predators.

Parasitoid insects, being of great importance for biological control, have been extensively modeled (Hassell 1978, Chesson and Murdoch 1986, Hochberg and Holt 1999). While parasitoids are undeniably parasitic, they are also distinctly different from more typical parasites, in that, like a predator, they kill their host. Parasitoid models are derived more from predator-prey theory than from epidemiology. The acceptance of "parasitoid" as a category distinct from other parasites suggests that the effect of an individual parasite on host fitness is an additional dichotomy useful for distinguishing among parasites. More specifically, does infection by a single parasite necessarily take host fitness to zero or not? Some entomologists view the term parasitoid as a property limited to the insects. However, we know that many unrelated lineages independently evolved this life history (Table 1).

We can use these dichotomies to create a 2X2 table of consumer interactions, comprised of four cells, in which we can place typical parasites, predators and parasitoids. Doing so creates an empty category for consumers that exploit more than one prey yet do not reduce that prey's fitness to zero. This vacancy is well filled by a common group of consumers, such as the familiar mosquitoes, which take small meals from several hosts/prey. These have been termed micropredators and we describe them below with the other three categories.

1) Typical parasite - an individual consumer attacks only one host and usually causes little or no pathology in that host. To avoid confusion, we will always term these "typical parasites" when we mean this specific category. We will use the general term "parasite' in its vague and vernacular sense to include the other types of antagonistic interspecific interactions when there is no ambiguity about its meaning. As we will discuss later, Anderson and May subdivided this category into microparasites and macroparasites.

2) Parasitoid – an individual consumer attacks one and only one host. As a necessary outcome of that interaction, it always kills that host. Very similar to parasitoids are parasitic castrators (Kuris 1974). These parasites divert the host's reproductive efforts to their own. Although they generally do not affect the viability of the host, they kill the host in an evolutionary sense since they block all further reproduction and drop the residual fitness of the parasitically castrated host to nil. The functional similarity between parasitoids and parasitic castrators is reinforced by several symbionts that. as a regular aspect of their impact, first castrate and then kill their hosts (Kanneworf 1965, Kuris 1974).

3) Predator - an individual consumer attacks and then kills more than one prey. While most predators are not symbiotic, many are; although the term is not often applied to those interactions. Symbiotic predators include the nicothoid copepods and those nemerteans that feed on brooded crab eggs (Kuris 1991,1993), and the many highly prey-specific nudibranchs, sea spiders, etc., that live on host colonies and feed on individual bryozoan zooids, hydroid polyps, etc.

4) Micropredator - an individual consumer attacks more than one individual host and causes little or no pathology, taking a small meal before moving on. Micropredators are often vectors for other infectious agents, that may, of course, have another sort of interaction with their host(s).

Table 1. Exploitative interspecific interaction adaptive peaks recognized by the trophic relationship between an individual consumer and its hosts (or prey). The 2X2 contingency table is based on the number of hosts (prey) attacked by an individual consumer and the effect of that individual consumer on its host (prey) expressed as residual reproductive value (RRV). Some examples across a wide taxonomic range are provided for each type of consumer.

	Number of Hosts Attacked by an Individual Consumer	
Effect on host RRV	One host attacked	>One host attacked
RRV > 0 (Host survives)	Typical Parasite Digene Metacercaria Adult Trematodes *Giardia* Scale Insects Cold Virus Malaria	Micropredator Mosquitos Some Leeches Lampreys Leaf Hoppers Koala
RRV = 0 (Host killed)	Parasitoid Braconid Wasps Tachinid Flies Larval Gordian Worms Many Hyperiid Amphipods Bruchid Beetle Larvae Lytic Phage Parasitic Castrator Digene Rediae & Sporocysts *Schistocephalus* Tapeworm Plerocercoids Rhizocephala Entoniscid Isopods Strepsiptera	Predator Starfish Cats Wolves Tunicates Tiger Beetles Sparrows

Detailed characterization of these types of consumers, and important aspects of their ecology and evolution, have been presented elsewhere (Perez 1931, Doutt 1959, Kuris 1973, 1974, 1997, Kuris and Norton 1985, Baudoin 1975, Janzen 1975, Eggleton and Belshaw 1992, Lafferty 1993a,b, Renaud et al., 1996).

3. MICROPARASITE AND MACROPARASITE MODELS: A BRIEF HISTORY

The microparasite/macroparasite dichotomy is the prevailing manner in which typical parasites are categorized. This dichotomy springs from the history of host-parasite models. The earliest host-parasite equations derive from the mass action models of Bernoulli (1760) that were applied more extensively as variations of the susceptible, exposed, infectious, recovered host (SEIR) models (Ross 1916, Kermack and McKendrick 1927, McKendrick 1940,May 2000). Such models helped explain the epidemiology of human pathogens and received important experimental support from the laboratory studies of Stiven (1964, 1968) in the *Hydra-Hydramoeba* system. In these models, the host is the effective unit, and parasite dynamics within the host are not tracked. Hence, these models use parasite prevalence (the proportion of infected hosts, Bush et al. 1997), not the number of parasites per infected host, to describe the parasite population. These models helped formalize epidemiological concepts such as host threshold density (Bailey 1957), and herd immunity(Anderson and May 1982). They are still of great value today (Earn et al. 2000).

Anderson and May realized that SEIR models were not sufficient to describe the population dynamics of all parasites (Anderson 1974, May1977). In particular, they recognized the importance of intensity, the number of parasites in an infected host (Bush et al. 1997), for helminth population dynamics. Because the frequency distribution of helminths among hosts can often be described by a negative binomial (Northam and Rocha 1958, Crofton 1971a), mean intensity is not an adequate descriptor of the parasite population. In particular, mean intensity does a poor job of describing the effect of parasites on their hosts (a goal of primary importance to medical and veterinary studies). For example, in human schistosomiasis, hosts with average worm burden suffer relatively little morbidity. Most of the worms occur in a disproportionately long tail of the heavily infected portion of the host population, where such high intensity infections can be lethal to the host (Cheever 1968, Hairston 1973, Smith et al. 1974).

To better model helminths, May and Anderson (1979) developed methods to account for the number of parasites per host. Although they did not keep track of the actual number of parasites in each host, they used sophisticated mathematics to account for an aggregated distribution of parasites among infected hosts. They also specifically kept track of the number of individual parasites in the parasite population, the number of hosts and the number of parasite free-living stages.

A key outcome of these models was the realization that aggregation of pathogenic parasites can have a powerful effect on the regulation of a parasite population (Crofton 1971a,b, Anderson 1976). Mortality will be higher for heavily infected hosts which, when they die, claim the lives of a disproportionately large number of parasite individuals.

These intensity-dependent models were better able to accommodate the biology of many adult parasitic worms. In their vertebrate hosts, these are typically parasites with a long generation time that do not usually reproduce within the host, (but direct reproduction could "occur at a low rate"), immunity is often dependent on worm intensity and would be of short duration, and hosts would tend to be continually reinfected (Anderson and May 1979). Because most parasites with this life-history are helminths, and because helminths are much larger in body size than protozoans, bacteria and viruses, the intensity dependent models were termed "macroparasite" models (Anderson and May 1979, May and Anderson 1979).

Anderson and May noted that SEIR models still worked under the assumptions of a short generation time, "*extremely* high rates of direct reproduction within the host", " a *tendency* to induce immunity to reinfection in those hosts that survive the initial onslaught",

and "the duration of infection is *typically* short in relation to the expected life-span of the host" (italics ours). To emphasize the vagueness of this characterization, they further remarked "there are, of course, many exceptions" (Anderson and May 1979). Because most parasites and pathogens that fit these assumptions (many protozoans, bacteria and viruses) are relatively tiny, Anderson and May called the SEIR models "microparasite" models.

It should be clear from this discussion that the key attribute needed to decide which set of models to use is not the taxonomic status, nor the size of the parasite, but the likelihood of intensity dependence (McCallum and Scott 1994). Modelers often note that the decision to use a macro or microparasite model is associated with parasite size, but to the extent that they are familiar with parasite life-history, emphasize the focus on prevalence (microparasite) or intensity (macroparasite) to determine the family of models to apply to a given host-parasite interaction (Anderson 1993, McCallum and Scott 1994).

Thus, twenty years ago, Anderson and May (1979, May and Anderson 1979) provided two general sets of analytical mathematical models for exploring host-parasite population dynamics. They were elegant, relatively simple mathematically, and they captured some important elements of the population interactions of hosts and parasites. These valuable features led to their rapid adoption and widespread use by mathematical modelers, and they have been applied to many host-parasite interactions, particularly those of medical (e. g., Anderson and May 1985, Earn et al 2000), veterinary (Grenfell et al., 1987) and wildlife management importance (Dobson and Hudson 1994). The conceptual innovation of the Anderson and May models was their classification of parasites into two types. Later we will incorporate this dichotomy into our approach.

While we will not detail the subsequent use of the microparasite/macroparasite (m/m) models, it is important to recognize that this classification often shifted from a mathematical convenience associated with size, to a categorical classification scheme of parasites based on size. This shift is a good example of how language shapes thought according to the linguistic relativism hypothesis (Whorf 1956). The prefixes macro- and micro- focus attention on the body sizes of the parasites and away from the issue of intensity. Perhaps this was due, in part, to a tendency for Anderson and May to leave out the mathematical underpinnings of their definitions; often focusing on biological attributes for convenience. Anderson (1993), for example, simply defined microparasites as "small/and possess the ability to multiply directly and rapidly within the host population".

Beyond the modeling literature, macroparasite and microparasite have become widely adopted descriptors to characterize parasites. Here, the most common attribute has been size, and the terms have devolved to a sort of crude taxonomy. Protozoa and smaller microbes are "microparasites" and helminths and arthopods are "macroparasites" even though some small parasites may be better modeled as macroparasites (e.g., *Ichthyophthirius multifilis* ciliates, *Eimeria tenella* coccidians, Mackinnon and Hawes 1961) and many large parasites may be better modeled as microparasites (e.,g., larval digenes in molluscs and rhizocephalan barnacles). Perhaps this could have been avoided had the models been named intensity dependent/independent.

4. SIZE AND ADAPTIVE PEAKS

To what extent is size responsible for determining the type of life history strategy a certain animal employs? While there is no direct linkage between parasite size and m/m models, we stress that size is a key corollary of an evolutionary categorization of host-parasite adaptive peaks, much as Anderson and May argued that it was generally associated with their dichotomy. While, it is clear that typical parasites can be much smaller than their hosts, and that predators can be much larger than their prey (e.g., filter feeders), the maximum relative size of the former and the minimum relative size of the latter are less obvious.

It is important to first acknowledge the extent to which consumer body size can be a plastic trait. The size of the host can strongly affect the size of an individual parasitoid or

castrator within that host. This size relationship may often approach the high correlation found between two body parts in a logarithmic allometric relationship. The relationship between size of adult entoniscid isopod parasitic castrators (*Portunion conformis*) in adult shore crabs (*Hemigrapsus oregonensis*) is shown in Figure 1. While typical parasites do not show this *intraspecific* size correlation, they do show a host-parasite size correlation for interspecific comparisons (Morand et al. 1996, Sorci et al. 1997). Predator growth can affect the size range of prey that can be attacked. The importance of relative size to predators is seen in the developmental response (Murdoch 1971) when larger (older) predators consume more prey of a broader size range (e. g., Kuris and Roberts 1990). For these types of relationships, size/age/stage structured models are highly informative because they subsume the developmental response.

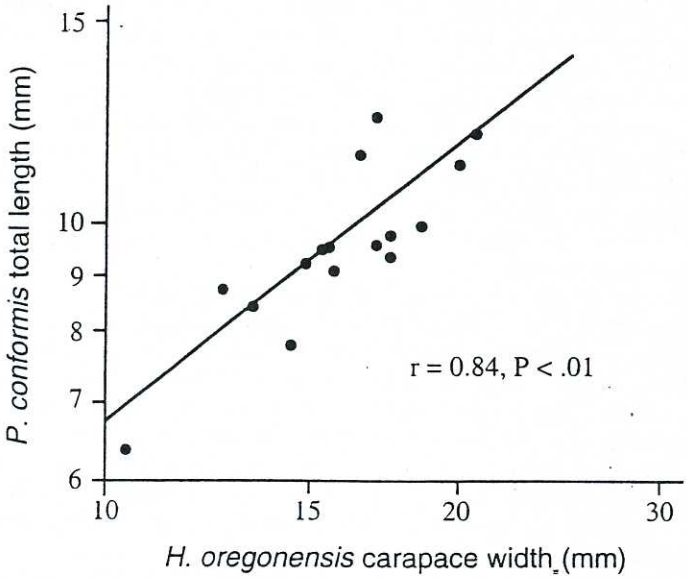

Figure 1. Allometric relationship between a host, the crab *Hemigrapsus oregonensis*, and a parasitic castrator, the isopod *Portunion conformis*, logY = 0.11 + 0.78logX (Data from Kuris1971).

One way to understand how various life-history strategies evolved is to compare the relative fitness of different strategies under different conditions while holding constant many other factors such as phylogeny, morphology and mode of reproduction. Recent work has indicated that a macro-ecological approach can be useful for predicting and evaluating the functional relationships between consumers and the types of prey they eat (Carbone et al. 1999). Lafferty (1997a) used an analogue of R_0, the basic population growth rate, or the number of successful offspring per individual in the absence of crowding, to predict the size relationships for prey and food for different consumer strategies. We summarize this approach in some detail here. Our R_0 analogue, R_0', is an individual's reproductive power times its life-span divided by its mass. Several components of R_0' scale with consumer body mass, and host body mass, such that they equal a constant times body mass raised to a positive or negative exponent (CM^c). For example, for some animals, life span scales as $CM^{1/4}$ so that life span is, on average, 1.8 times longer for every ten-fold increase in body mass (Peters 1983). Reproductive power (expressed as $K_0K_1/(K_0 + K_1)$ where $K_1 = CM^{-1/4}$ and K_0 is the rate of food consumption) essentially describes the rate at which ingested calories are converted into

reproduction and is limited either by metabolism or consumption (or both), each of which scale with consumer body mass (Brown et al. 1993). R_0' is, therefore, similar to the proportion of an individual's body mass that is converted into successful offspring over the course of an individual's life. For both R_0 and R_0', a value less than one indicates negative population growth and simple models can reveal maximum values of R_0' for a particular life-history strategy, or compare values of R_0' among different life-history strategies placed under otherwise identical conditions. We have investigated the influence of consumer and host body mass on R_0' for the life-history strategies in Table 1.

For a predator, reproductive power is a function of the prey encounter rate (increasing with predator mass and decreasing with prey mass) times the probability of capture success (increasing with predator mass and decreasing with prey mass) times the consumable mass of each prey item. Micropredators are similar except that their probability of success (based on an undetected attack) increases with host mass and decreases with micropredator mass. Another difference is that it is the size of the micropredator, not the host, that constrains the amount consumed per attack. Comparing micropredators with predators indicates that, when the consumer is smaller than its prey, micropredation is more profitable and when the consumer is larger than the prey, predation is more profitable.

A parasite differs in that if it finds a host, its survival is tied to its host's survival (on average, this provides an advantage to parasitizing larger, longer-lived, hosts). Typical parasites may negatively affect their host's life-span, particularly if they are present at high intensities, or are large relative to the host (providing another advantage to parasitizing large, relatively long-lived hosts). Parasitic castrators do not appreciably reduce their host's life span but are constrained in the amount of food they can consume by the amount of energy the host puts into reproduction (a function that tends to peak at intermediate body sizes (Brown et al. 1993)). Unlike other parasites, a parasitoid can consume the entire host without compromising its own survival. Two advantages of parasitism over free-living life-histories are a higher food conversion efficiency and fewer constraints on food availability (Bailey 1975).

Our models suggest that variation in consumer and host body masses can favor the evolution of different consumer life-history strategies. The optimal life histories for a particular consumer-host body mass combination fall into discrete adaptive peaks that depend on both relative and absolute body masses (Figure 2a). Both the models and empirical observations indicate that predators, micropredators, and typical parasites exploit the entire spectrum of absolute host body sizes except that in our model typical parasites cannot persist in the smallest hosts and only typical parasites can exploit the largest hosts. Predation is an optimal adaptive peak when prey are relatively small, and typical parasitism (and micropredation) are optimal when hosts are relatively large. The model also indicates that consumer size scales positively but at a ratio of less than one with host/prey size.

For a consumer to feed on a host of similar size, parasitoid and parasitic castration life histories are favored in the model. Parasitoids can survive in small hosts, but are absent from medium sized hosts, where parasitic castration tends to be more profitable. In nature, parasitoids are found in larval insects and other relatively small hosts. Even bacteria experience significant mortality from parasitoid natural enemies (lytic phage). This may be a consequence of the longer life spans of larger hosts which make the parasitoid strategy relatively inefficient compared to other life histories, such as parasitism, when integrated over the life span of a host. Hosts with large body sizes, though long lived, tend to invest relatively little in reproductive tissue (Brown et al. 1993), making parasitic castration a less efficient consumer strategy. Parasitic castrators are found in a wider range of host body sizes than parasitoids (from very small hosts up to large crabs and fishes) but they are clearly absent from the largest hosts. In general, one advantage of being a host with a large body size is that you are no longer suitable for parasitoids and parasitic castrators.

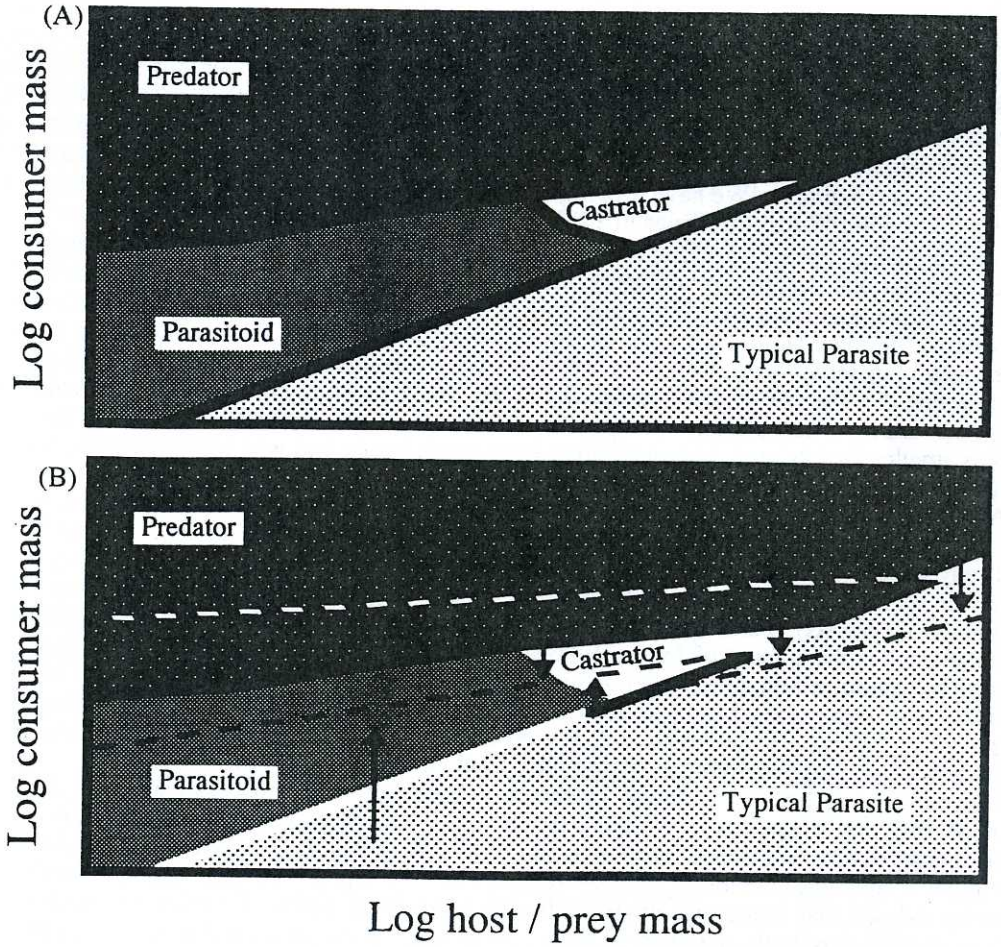

Figure 2a. Life-history strategies suited to different consumer and host/prey body masses. Isoclines of R_0' (fitness analogue) for pair-wise comparisons of predators, parasitic castrators, parasitoids and typical parasites divide the space into four polygons, each of which we have labeled with the name of the consumer which has the highest R_0' in that space. 2b. Superimposed (as dashed lines) on a copy of Figure 2a are the R_0' maxima for each polygon. In addition, segments of isoclines that are invadable by evolving consumer body mass are indicated by white lines and arrows. For example, the bottom left arrow represents a parasite that would increase its fitness by increasing size (a trajectory that heads for the parasite maxima of which the left side is hidden by the Parasitoid polygon). However, before the parasite reaches its optimal parasite body size, it enters a region of parameter space where it could increase its fitness more by acting as a parasitoid. One could also imagine horizontal arrows that would allow a shift in strategy associated with a shift to a host with a different body size. Another possible mode of evolution would be for a host to increase body size so as to be less suitable for some types of natural enemies (e.g., parasitoids and castrators).

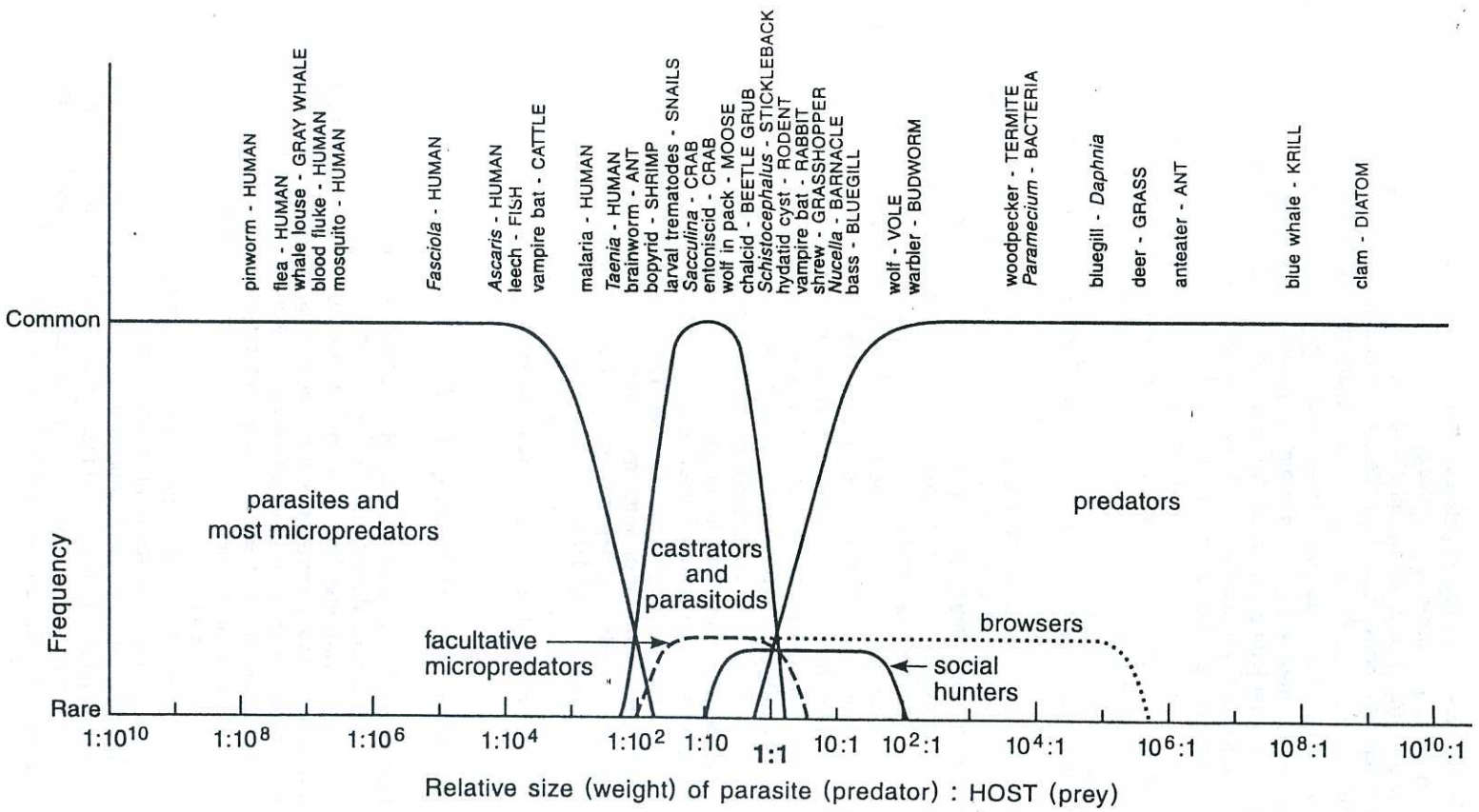

Figure 3. The frequency of relative body sizes of consumers and hosts.

The models also suggest pathways for exploiters to evolve from one adaptive peak to another. A particular life-history strategy can drive the evolution of consumer body mass toward an optimum. For example, all else being equal, there is an optimal parasite body size for a particular host body size. In some circumstances, it may be possible to encounter the opportunity to increase R_0' even more by changing life history strategies (such as from parasitoidism to parasitic castration or visa versa, Figure 2b). Such options for a life-history shift from one adaptive peak to another may be particularly available if the consumer has broad host specificity, including hosts of different body masses. Selection for host specificity includes an opportunity to alter the trophic strategy of a consumer.

The largest typical parasite relative to its host size reaches somewhat more than 1% of the weight of its host (*e. g.*, adult *Hymenolepis diminuta* in the rat or an hydatid cyst in an intermediate host). The smallest solitary predators relative to prey size are usually highly specialized killing machines (*e. g.*, weasels, vipers, wind scorpions). These can be as small as perhaps a half-order of magnitude smaller than the mass of their prey. Figure 3 scales the relative sizes of parasites (predators) versus host (prey) sizes and spans at least 18 orders of magnitude.

From a parasite:host size ratio of about 1:100, to a predator:prey size ratio of about 1:1, it appears to be energetically or behaviorally impossible to effectively consume hosts or prey using the life history strategies of parasitism, micropredation or predation (excepting the relatively few examples of some micropredators and social hunters). This ecological gap is rather precisely filled by the numerous examples of parasitoids and parasitic castrators (Fig. 3). The smallest known parasitoids and parasitic castrators are about 1% the size of their hosts. The largest approach about half the mass of their individual hosts (Kuris 1974).

Considering how many examples of all these trophic relationships exist in the world, the rather sharp boundaries to the relative size limits for all these types of relationships argue for very strong ecological constraints on these trophic adaptive syndromes.

Analysis of the size constraints of these adaptive syndromes suggests the need to acknowledge social hunting and facultative micropredation (~browsing) as likely additional adaptive syndromes. These are relatively plastic trophic strategies. Predators that use social hunting to exploit large prey items can also function as typical predators when attacking relatively small prey items. For some micropredators, the impact on an individual host/prey (micropredation or predation) depends on the size of that individual. For example, some leeches are micropredators of large fish but predators on small invertebrates. Transitioning between micropredation and predation is a relatively common strategy for vampire bats, lampreys, cookie-cutter sharks, and many browsers (*e. g.*, deer).

5. WHEN HOST DEATH IS REQUIRED

As we have hinted, the dichotomies of "number of hosts/prey attacked", and fitness consequences to that host, do not fully distinguish between parasitoids and parasitic castrators. These two adaptive syndromes are sufficiently distinct to merit distinguishing among them in a classification scheme. Consideration of the parasitic castrator/parasitoid dichotomy reveals that the fate of the parasites upon the death of the host is dramatically different. For a parasitoid to complete its life cycle, death of the host is a necessary consequence of the interaction. When requisite death of the host is entered as a third dichotomy, castrators are readily distinguished from parasitoids (Figure 4).

An interesting additional adaptive syndrome emerges when this division is applied to typical parasites. Some parasites in intermediate hosts are transmitted when a predatory host feeds on an infected prey host and, though they do not kill their host, require its death. An extensive literature has documented that these parasites often modify the behavior of their hosts so as to greatly increase the likelihood that the infected host is eaten by an appropriate next host (Holmes and Bethel 1972, Dobson 1988, Lafferty 1992, 1997b, Lafferty and Morris 1996,

Poulin 1994, Kuris 1997, Lafferty et al. this volume). A key difference between trophically transmitted parasites and other parasites can be seen when revisiting the key findings of macroparasite models. Parasite-increased trophic transmission (PITT) crops hosts with high intensity parasite loads (much as the intensity-pathology relationship increases the mortality rates of heavily infected hosts in a macroparasite model). But, with PITT, the parasite is not lost (dies with its host), as in a macroparasite model. The parasite has *succeeded* and maximized its fitness with the (fortunate) death of its host. Behavior modification intensifies this effect (Lafferty 1992, Lafferty and Morris 1996, Kuris 1997) and probably permits PITT to operate at lower parasite intensities. Behavior modification also adds a parasite into predator/prey population dynamics models. Because the host-death-required dichotomy, when applied to predators, does not reveal any additional insight, we limit its application to parasites.

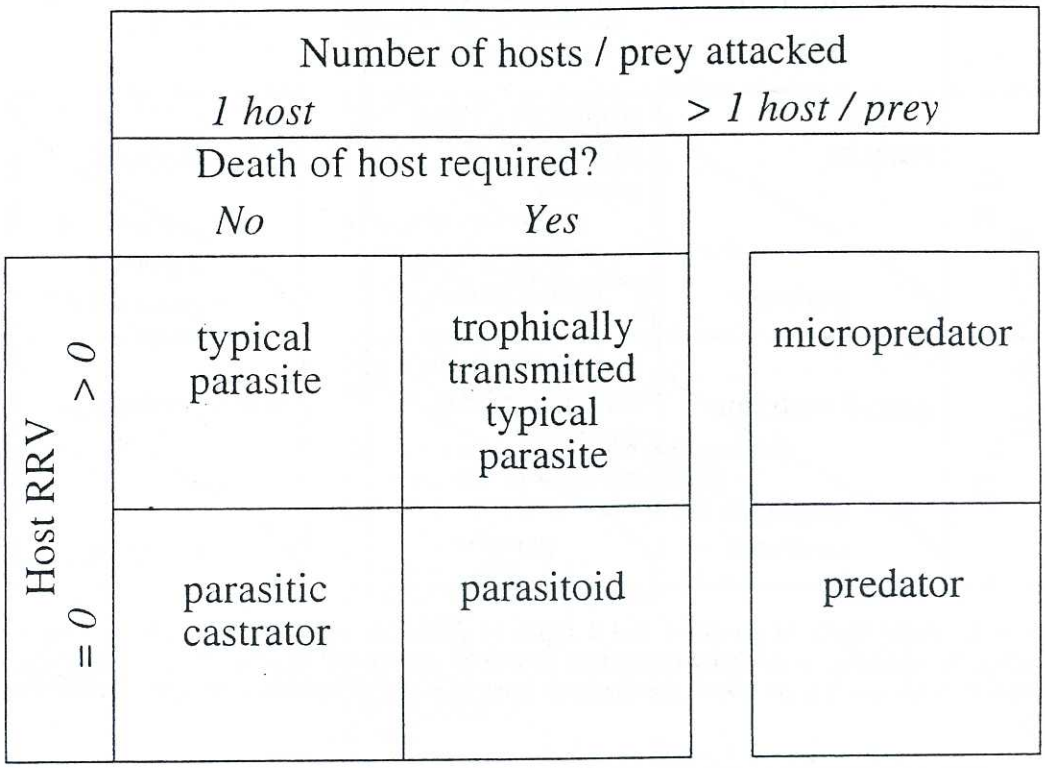

Figure 4. The 2x2 dichotomy of Table 1 is expanded, separating parasites according to whether host death is required. This allows the distinction between parasitoids and parasitic castrators and creates a new cell for trophically transmitted parasites.

6. INTENSITY DEPENDENCE

Returning to our initial goal to include the full range of consumer life histories, we now add the m/m intensity-dependence dichotomy to the six adaptive syndromes revealed by our evolutionary based analysis. Twelve types of antagonistic interspecific interactions can now be recognized, representing 6 pairs of intensity dependent and intensity-independent associations (Figure 5).

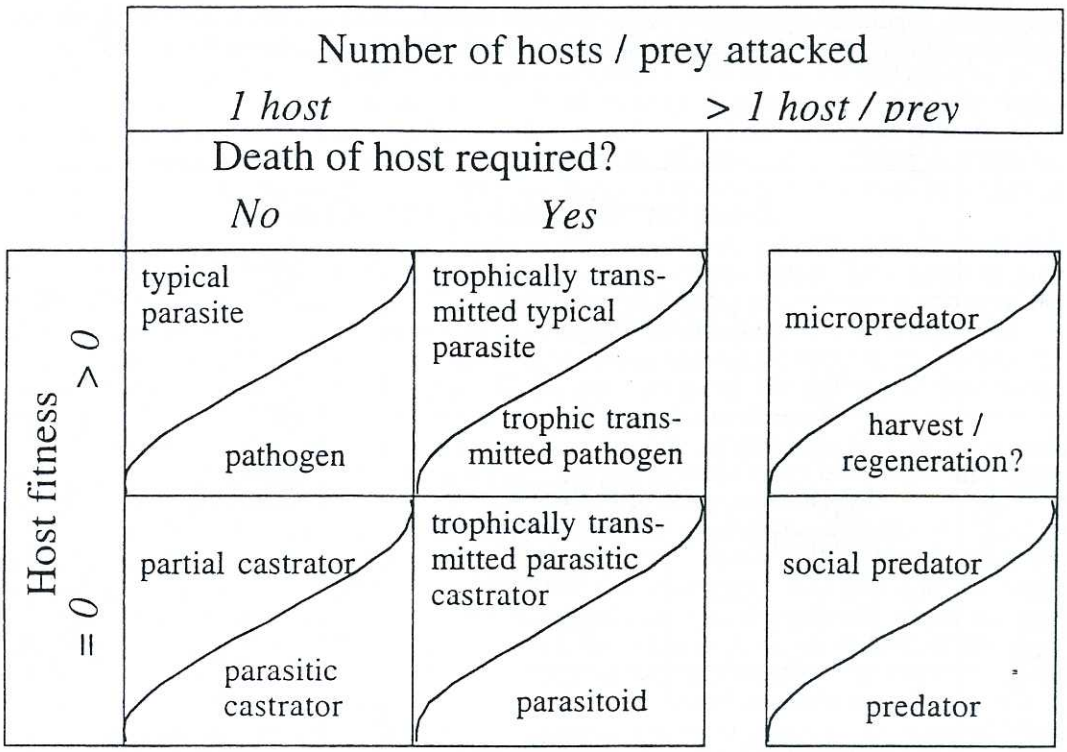

Figure 5. Eight types of parasites and 4 types of predators separated by 4 axes. This is achieved by applying an intensity-dependent pathology axis to all cells in figure 4. Intensity-dependent relationships are above the diagonal line; intensity-independent ones are below the line.

While the concept of adaptive syndrome seems to apply to some of these categories, particularly those for which there are many examples in the natural world (Table 2), a few are uncommon, and may most often represent transitions between more highly constrained adaptive peaks. The full elaboration of the evolution, ecology and modeling of these types of interactions lies beyond the scope of this paper (Kuris & Lafferty in prep.). Some of the most evident features will be noted here and their relation to the m/m models will be highlighted.

As Anderson and May intended, the intensity dichotomy cleaves pathogens (intensity independent) from typical parasites (intensity dependent). Both are widespread interactions in nature. It is these two categories that are fully appropriately modeled as, respectively, microparasites and macroparasites. The ability of pathogens to multiply within a host effectively diminishes the importance of multiple infections. The multiplicative effect generally overshadows small differences in the infective dosage. Further, for vertebrate hosts, long-lasting immunity prevents the accumulation of parasites over time. The host-parasite dynamic

Table 2. Examples of adaptive peaks for the trophic relationships between individual consumers and their hosts (or prey) in Figure 5 according to: (1) the number of hosts (or prey) attacked, (2) whether death of the host is required, (3) the impact on host fitness, and (4) if the relationship is intensity dependent.

Adaptive Peak	Examples
Typical parasite	Adult trematodes Monarch butterfly caterpillars *Ichthyophthirius* ciliates
Pathogen	*Giardia* Scale insects cold virus
Trophically transmitted typical parasite	Digene metacercariae *Anisakis* nematode larvae Unilocular hydatid cysts
Trophically transmitted pathogen	Multilocular hydatid cysts *Toxoplasma* sporozoites Trypanosomes (to the vector)
Partial castrator	Most bopyrid isopods *Hymenolepis diminuta* cysticercoids Some water mites on insects
Parasitic castrator	Rhizocephala Strepsiptera Digene rediae and sporocysts
Trophically transmitted parasitic castrators	*Schistocephalus* tapeworm plerocercoids Some acanthocephalan cystacanths Dilepidid tapeworm cysticercoids in ants
Parasitoids	Braconid wasps Bruchid beetle larvae in seeds Lytic phage
Micropredators	Mosquitos Leaf hoppers Koalas
Harvest / regeneration	Deer mowing grass Rays cropping clam siphons Snakes eating lizard tails
Social predators	Wolves Killer whales *Deinonychus* dinosaurs
Predators	Starfish Tunicates Sparrows

generally becomes an arms race as the host attempts to limit the damage caused by the pathogen by mounting an effective host response. It is worth noting that in well studied cases (e.g., malaria, Greenwood et al. 1991, Lines and Armstrong 1992; trypanosomes, Welburn and Maudlin 1999; HIV, McLean 1993), there is still a contribution of pathogen intensity to the reduction in host fitness as a multiple strain infection generally causes more pathology than does infection by a single strain. Intensity dependence, in addition to describing helminths (as typical parasites), creates new categories for predation such as social hunters and facultative micropredators.

Trophically transmitted pathogens can be distinguished from trophically transmitted typical parasites because the former increase within the host via reproduction, while the latter do not. Trophically transmitted typical parasites are a very common type of interaction; trophically transmitted pathogens are relatively uncommon, though we don't know why.

At very high intensities, many types of typical parasites may have an effect on host reproduction. As most typical parasites are highly aggregated, this outcome is a rare event. Intensity dependent parasites that block reproduction at relatively low intensities are infrequently reported. The few examples known seem to have a partial castration effect, even in single infections (e. g. Lanciani 1982). This type of host-parasite interaction may represent a transition for associations evolving towards parasitic castration from a typical parasite adaptive peak. Most female bopyrid parasites regularly greatly reduce host fecundity and are partial castrators (Van Wyk 1982). Double infections (the maximum possible intensity) are very rare, so the additive effect of pathology has not been investigated. It seems likely that this is a variant of a parasitic castrator strategy, rather than that of a typical parasite, since bopyrids are within the relative size range of parasitic castrators, and are related to other isopods that are parasitic castrators. Avian brood parasites, such as cowbirds and cuckoos, have a unique trophic interaction with their hosts where a parent host feeds a parasite nestling at the expense of the parent's own young. This might be considered partial castration (from the parent bird's perspective), as the brood parasite reduces the success (often to zero) of each host clutch that is parasitized. In this sense, brood parasites have a sort of temporal intensity-dependent effect on host reproduction. Nonetheless, this type of trophic interaction is not readily characterized by our scheme and requires further consideration.

The characteristics of trophically transmitted parasitic castrators become evident when their intensity dependence is considered. In this, they are unlike both parasitoids and parasitic castrators. It is notable that it is their effect on host viability, PITT, that appears to be intensity dependent. In contrast, the castration effect has generally been reported even for single infections as is the case for parasitic castrators. This adaptive syndrome is not widespread across taxa, but there are several well-studied examples among the larval tapeworms and acanthocephalans (Arme and Owen 1967, Plateaux 1972). It is probably most easily reached by trophically-transmitted parasites which evolve into the parasitic castration adaptive peak.

For predators and micropredators , size and intensity-dependent effects are linked (Fig 3). Predators sometimes hunt more effectively in groups and social hunters are a manifestation of intensity-dependent predation. Social hunting makes it possible for predators to capture prey much larger than they are. Lampreys, some leeches and many large herbivores may or may not kill their prey depending on the size of the prey and the size of the bite taken. The ability to transition between predation and micropredation might be termed facultative micropredation. Some grazers appear to feed intensity independently, forming a unique category related to micropredators which we have identified, tentatively, as harvest / regeneration. Unlike most micropredators, these consumers are relatively large compared to their hosts/prey and so it is difficult to imagine multiple individuals attacking the same host individual. For herbivores that feed on grasses, the above-ground mass of the prey is consumed in a single bite, but can re-sprout from the root. The prey adaptation of evasive limb autotomy, seen in crustaceans and lizards, provides an analogous interaction when the predator eats the limb, but not the prey. Among animals, harvest/regeneration has been reported for young *Corambe pacifica* (a nudibranch) (Ryland 1970). They attack the bryozoan,

Membranipora, devouring the polypide; leaving the cystid to regenerate another polypide. There may be other specialized consumers that feed on phoronid lophophores or bryozoan polypides that best satisfy these conditions because these structures are readily regenerated and each individual prey has but one of them.

7. CONCLUSION

Population modeling has unintentionally created several dichotomous life-history axes (viewed by us as adaptive syndromes) to distinguish among consumers. We have used these dichotomies to construct matrices of predatory and parasitic consumers. The resulting recognition of twelve consumer strategies brings us closer to capturing the richness of nature.

Our approach interfaces well with the microparasite/macroparasite dichotomy that is the main paradigm used to describe parasites. A causal relationship between intensity and pathology (macroparasite models) is seen in typical parasites, trophically transmitted typical parasites, trophically transmitted parasitic castrators, partial parasitic castrators, micropredators and social hunters. Typical parasites and micropredators are well suited for modeling as macroparasites, even when they are taxonomically microbial. Micropredators, because of their role as pathogen vectors, are often important components of microparasite models, but our understanding of their ecology might benefit from the further development of explicit micropredator models. Intensity-independent pathology (microparasite models) is characteristic of pathogens, trophically transmitted pathogens, parasitic castrators, parasitoids, predators and facultative micropredators and this key ecological feature should be a modeling component for these relationships. Microparasite models were developed and are appropriate for pathogens. There is, of course, an extensive family of models for parasitoids that has a largely independent intellectual history from epidemiological and m/m models. Much of this has been in the context of insect pest biological control and focuses on host population regulation (Hassell 1978, Chesson and Murdoch 1986, Hochberg and Holt 1999). A few microparasite models for parasitic castrators are available (Blower and Roughgarden 1987, Lafferty 1991). Dobson (1988) has adapted a macroparasite model for trophically transmitted parasites.

Theoretical issues revealed by our approach may require development of new models and associated theory. This may improve the development of a generalized, reality-based, comprehensive set of models that would include parasitoids, parasitic castrators, micropredators, PITT and a number of other consumers. It will be challenging and important to link such models to accommodate the many parasites with complex life cycles as Morand et al. (1999) have done for *Schistosoma mansoni* in the black rat.

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