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Trophic strategies, animal diversity and body size

Kevin D. Lafferty and Armand M. Kuris

A primary difference between predators and parasites is the number of victims that an individual attacks throughout a life-history stage. A key division within natural enemies is whether a successful attack eliminates the fitness of the prey or the host. A third distinctive axis for parasites is whether the host must die to further parasite development. The presence or absence of intensity-dependent pathology is a fourth factor that separates macroparasites from microparasites; this also distinguishes between social and solitary predators. Combining these four dichotomies defines seven types of parasitism, seven corresponding parasites, three forms of predation and, when one considers obligate and facultative combinations of these forms, four types of predator. Here, we argue that the energetics underlying the relative and absolute sizes of natural enemies and their victims is the primary selective factor responsible for the evolution of these different trophic strategies.

Published online: 11 September 2002

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Although reproduction is the overriding theme of the drama of life, feeding and being fed on, are the key subplots. For this reason, ecologists and evolutionary biologists are well aware of the importance of predation and are increasingly considering the role of parasitism in the control of populations and the structure of communities. A barrier to fully incorporating concepts about parasitism has been the daunting diversity of parasitic strategies that exist; this can impair our modeling efforts, blur theoretical predictions and retard basic communication about parasitism.

Early population modelers recognized a need to define four types of distinct NATURAL ENEMY (see Glossary): PREDATORS, PARASITOIDS, MICROPARASITES and MACROPARASITES. It is about this diversity of trophic

strategies that we ask: are these distinct trophic strategies? Are there any other strategies that these modeling categories do not adequately cover? What life-history traits of a natural enemy define the axes of the adaptive landscape of trophic strategy? In addition, what are the ecological consequences and corollaries of these strategies? Our conclusion morphs the old adage 'you are what you eat' into 'you are how you eat'.

Evolutionary concepts of trophic strategies

Natural enemies take nourishment from a victim (host) or victims (prey) using a variety of trophic strategies. The particular strategy that an individual natural enemy uses can vary from one victim to the next (we treat asexually produced progeny in a host as equivalent to a single genetic individual). For instance, a PARASITE might interact very differently with an intermediate host compared with a definitive host. Our goal is to determine the underlying key factors that distinguish different trophic strategies used by natural enemies. We do this with a factorial application of four dichotomies, each of which describes an aspect of enemy–victim interactions (expanding here on earlier work [1]*). A logical trophic strategy emerges for ten of the potential 16 cells (Fig. 1). These dichotomies apply to all taxa, including animals, plants, PATHOGENS, phages, bacteria, microbes and helminths, enabling us, for example, to evaluate herbivores as types of parasites or predators of plants. Nonetheless, our approach does not supplant existing terminologies based on the taxonomy of the victim (herbivore or carnivore). We intentionally avoid creating new terminology by adapting existing terms that, although they might refer traditionally to specific taxa, apply conceptually to other organisms.

The first dichotomy: does the enemy attack more than one victim?

How do parasites differ from predators? In his authoritative work on parasite ecology and evolution, Combes [2] considers that it is the durability of the

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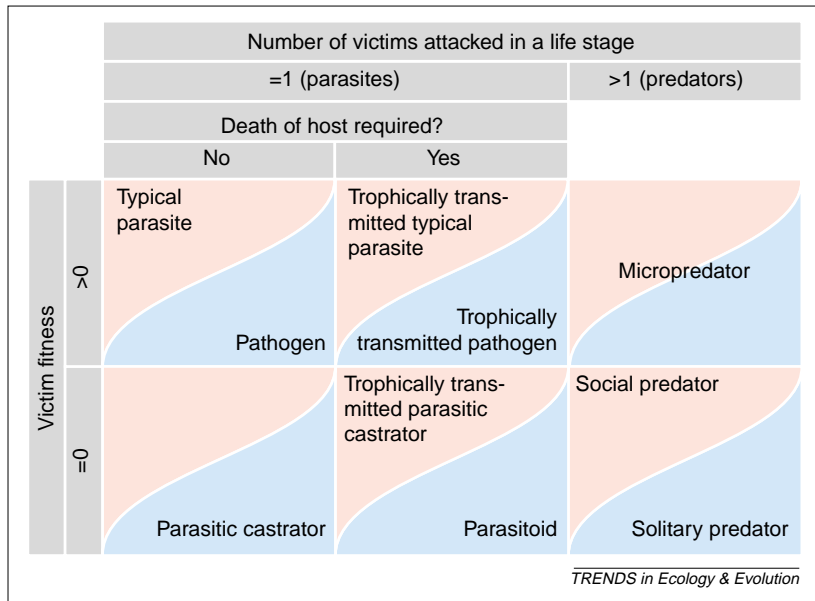


Fig. 1. Seven types of parasitism and three types of predation separated by four life-history dichotomies. Intensity-dependent relationships are above the diagonal line; intensity-independent ones are below. This results in ten trophic strategies of natural enemies related to Box 3. Note that the three predation strategies combine to four types of predator: micropredators, facultative micropredators, solitary predators and facultative social predators. Victim fitness = 0 indicates that the interaction either kills or blocks victim reproduction, whereas victim fitness > 1 indicates that the victim typically survives the interaction and can reproduce.

enemy–victim interaction that provides the distinction (i.e. parasitic interactions are durable). Population models extend this perspective. In infectious disease models, a parasite exploits only one host during a particular phase of its ontogeny. By contrast, most population models in ecology incorporate a functional response [3] that considers how predators eat many prey. In other words, population modelers have established a well-accepted dichotomy between parasites and predators based on the number of hosts or prey attacked during a particular life-history stage. Attacking one (i.e. a parasite) or more than one (i.e. a predator) victim is easier to apply as a dichotomy than is durability of an attack, and we use the latter in our approach, noting that the two views are not in conflict (exploiting only one victim during a life-history stage is necessarily a durable attack).

The second dichotomy: does the enemy eliminate victim fitness?

Ecological modelers broadly classify parasites into parasitoids, macroparasites and microparasites [4,5]. Although parasitoids are undeniably parasitic according to our first dichotomy, they are also quite different from these other parasites, in that, similar to a predator, they must kill their host. Entomologists, seeking insight into biological control, have tailored predator–prey models to describe parasitoid insects [4,6,7]. The view of parasitoids as distinct from other parasites points to the effect that an individual parasite has on host fitness as a way to distinguish among parasites. The dichotomy we apply is whether

infection by a single natural enemy (usually a larval stage for parasitoids) necessarily takes victim fitness to zero. Several unrelated lineages have independently evolved this life history (Boxes 1,2).

This dichotomy, which separates parasitoids from parasites, also cleaves predation into two life histories. Mosquitoes represent a type of predator that does not reduce the fitness of prey to zero. Such a trophic strategy can be termed micropredation. Although many natural enemies are obligate MICROPREDATORS, others, such as vampire bats, lampreys, cookie-cutter sharks and many herbivores (e.g. deer), switch between micropredation and predation. For these FACULTATIVE MICROPREDATORS, the impact on an individual victim (either micropredation or predation) depends on the size of that individual. For example, pocket gophers nibble the roots of trees and shrubs but will pull small plants into the burrow and kill them. By combining the consequence of an attack with the number of victims attacked, a 2×2 matrix of natural enemy interactions produces four cells filled by: (1) predation; (2) micropredation; (3) typical parasitism; and (4) parasitoidism or parasitic castration (Box 1). We note that the second dichotomy could be expanded into an axis to allow mutualisms (positive effect on 'victim' fitness) and scavenging (no effect on 'victim' fitness).

The third dichotomy: does the enemy require the death of the victim?

Application of the second dichotomy groups parasitoids and PARASITIC CASTRATORS together because both take host fitness to zero. However, these two natural enemies are sufficiently different that it would be useful to distinguish between them. Unlike the parasitic castrator that keeps its host alive to benefit from a long life, the parasitoid must kill its host to complete its lifecycle, indicating that requisite death of the host is a useful third dichotomy. This dichotomy does not add insight to predation, because it is redundant to the concept. However, when we apply this dichotomy to TYPICAL PARASITES, a new group of parasites emerge as a distinct trophic strategy. Some parasites are transmitted when a predatory host feeds on an infected intermediate host and, although these TROPHICALLY TRANSMITTED parasites do not kill their intermediate host, they do ultimately require its death [8].

The fourth dichotomy: does the enemy cause intensity-dependent pathology?

Anderson and May (Box 3) noted that an INTENSITY-DEPENDENT PATHOLOGY dichotomy divides pathogens (INTENSITY INDEPENDENT) from typical parasites (INTENSITY dependent). Superimposing a dichotomy of intensity-dependent/independent pathology on the other three dichotomies enables us to define ten types of antagonistic, interspecific interaction (Fig. 1). Doing so divides some natural enemies (trophically transmitted parasites, parasitic

Box 1. The first two dichotomies

The number of victims attacked combined with the fitness consequences to the victim of a successful attack enables us to separate typical parasitism, predation and parasitoidism. If we create a 2x2 table from these two dichotomies (Table I), we find an empty category for natural enemies that attack more than one victim but do not eliminate the fitness of that victim. The strategy of micropredation (e.g. as used by mosquitoes) fills this vacancy well because these predators take small, nonlethal meals from several victims. The ecology and evolution of these four strategies are familiar to ecologists [a–l]. However, ecologists are less familiar with the common trophic strategy of parasitic castration. From a fitness standpoint, parasitic castrators 'kill' a single victim by blocking all further reproduction of the host and, therefore, our 2x2 table includes them with parasitoids. Synonymizing these two distinctly different trophic strategies is not appropriate and we offer a third dichotomy (requirement of the death of the victim,) to distinguish between them. Similarly, Anderson and May (Box 3) recognized that typical parasites were better understood if one considered the extent to which they had intensity-dependent or intensity-independent pathology. This fourth dichotomy (intensity dependence) leads us to our final set of ten trophic strategies as depicted in Fig. 1 in the main text.

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Table I. Separation of typical parasitism, predation and parasitoidism

	Attacks one victim per life stage (Parasite)	Attacks more than one victim per life stage (Predator)
Victim fitness > 0	Typical parasite/pathogen A natural enemy attacks one victim during a life stage and does not necessarily eliminate its fitness (e.g. digene metacercaria, adult trematode, <i>Giardia</i> , scale insect or cold virus). Anderson and May (Box 2) usefully subdivide this category into microparasites and macroparasites according to whether pathology increases with the intensity of infection.	Micropredator A natural enemy attacks more than one victim during a life stage and does not necessarily eliminate its fitness (e.g. mosquito, leeches, lamprey, leaf hopper or koala). These take a small meal before moving on.
Victim fitness = 0	Parasitoid/parasitic castrator A natural enemy attacks one victim during a life stage and necessarily eliminates its fitness. Parasitoids (e.g. braconid wasps, tachinid flies, larval gordian worms, many hyperiid amphipods, bruchid beetle larvae or lytic phage) kill the host on emergence. Parasitic castrators (e.g. digene rediae and sporocysts, <i>Schistocephalus</i> tapeworm plerocercoids or Rhizocephala) usurp the reproductive efforts of the host but do not kill it.	Social/solitary predator A natural enemy attacks more than one victim during a life stage and necessarily eliminates its fitness (e.g. seastar, cat, tunicate, tigerbeetle or sparrow). Social predators cooperate to capture and share large prey (e.g. wolves).

castrators and parasitoids) into some previously unrecognized relationships.

The intensity-dependent pathology dichotomy distinguishes between trophically transmitted typical parasites and the less common trophically transmitted pathogens. It does not divide parasitic castrators into relevant subclasses because the castration effect is intensity independent (simple models suggest that parasites that reduce reproduction should be selected to fully castrate the host [9]; examples of intensity-dependent partial castrators are few and these are treated more adequately as typical parasites). Because parasitoids

also induce intensity-independent pathology, it is not immediately clear which parasites meet the conditions of reducing host fitness to zero and requiring host death in an intensity-dependent fashion. This life history is not widespread across taxa, but some trophically transmitted larval tapeworms and acanthocephalans castrate their intermediate hosts [10,11]. Via an intensity-dependent effect, they also appear to modify host behavior to increase the probability of transmission to a predatory host.

Applying an intensity-dependent pathology dichotomy to predation makes little sense unless one

Box 2. Evolution of trophic strategies

Among the animals, there are nine entirely parasitic phyla and 22 predominantly (>99%) predatory phyla [a]. For the remaining 11 phyla that have a mix of parasitic and free-living species (e.g. Nematoda, Platyhelminthes and Arthropoda), clades within a phylum are often either entirely parasitic or predatory. For example, the parasitoid strategy in wasps appears to have a single origin [b] followed by one of the most spectacular radiations in evolutionary history. Some parasitic lineages produce free-living species, but these examples are rare [c]. The upshot is that the evolution of a parasitic strategy appears to be a cladogenic event. For instance, recent molecular phylogenies suggest that at least nine nematode clades arose through the evolution of parasitism [d]. In spite of the link between parasitism and cladogenesis, none of the ten trophic strategies we describe here (Fig. 1 in main text) has a single evolutionary origin and all are spread among phyla. This pattern fits Sewall Wright's [e] concept of adaptive peaks: among species, selection for similar optima for the interacting cluster of traits that comprise a life-history strategy will yield analogous adaptive peaks; that is, an adaptive syndrome across species as opposed to strategies that grade into one another along a continuum.

When a victim evolves a larger or smaller body size, changes in the body size of its natural enemies could follow in step, or the natural enemies could evolve other strategies or switch to other victim species. Once in a new host, enemy body size should evolve to maximize fitness according to the initial trophic strategy. But evolving body size based on available energetics could eventually result in a change in the optimal trophic strategy. For example, it seems plausible that the ancestors of some parasitic castrators were smaller, typical parasites that eventually evolved to the size that allowed them to consume the maximum amount of host possible without seriously compromising the longevity of the host. That is, they became parasitic castrators.

There is some evidence for evolution between trophic strategies. Wasps and flies that are parasitoids of insects may have separately evolved from predators of insects, predators of fungi and scavengers of insects [b]. Two clades of intestinal nematodes in vertebrates might have evolved independently from two clades of nematode parasitoids of

insects [4], suggesting that switching from a small host to a large host selected for the more appropriate typical parasite strategy instead of the ancestral parasitoid strategy.

For predators, there is often room for plasticity between some strategies, depending on the relative body sizes of a particular predator-prey interaction. For instance, a leech might prey on a small insect for breakfast and be a micropredator on a large fish for dinner. Plastic trophic strategies in parasites are rare. One example of a context dependent switch from parasitoid to typical parasite may be the little trichopsid flies that are similar to parasitoids except that, when they infect large locusts, they do not kill them upon emergence [f].

In addition to evolving a new strategy, a parasite can add strategies to its life cycle. For example, it seems plausible that trophically transmitted parasites evolved from typical parasites that managed to turn the otherwise unfortunate death of their host by predation into an opportunity to parasitize the predator [g]. Trematodes are extreme examples of strategy accumulators. Most species start out as a parasitic castrator, switch hosts to become trophically transmitted parasites and end their lives as typical parasites.

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Box 3. A history of macroparasite–microparasite models

Simple mass-action equations [a] used in disease models track susceptible, exposed, infectious and recovered hosts (SEIR models) but not parasite intensities within the host [b–d]. The protozoans, bacteria and viruses that inspired SEIR models have short generation times, rapid reproduction inside the host, a tendency to induce immunity in surviving hosts and a short duration of infection. Most parasites and pathogens that fit these assumptions are relatively small in size. Anderson and May recognized this pattern and termed these 'intensity-independent' SEIR models 'microparasite' models [e,f].

The population dynamics of most parasitic worms are not well described by SEIR models. Because pathology increases with the number of worms in an infection (or intensity), and worms typically aggregate among hosts, pathology varies considerably from host to host. To model such worms more appropriately, Crofton constructed the first model that accounted for parasite intensity and aggregation [g]. May and Anderson [e,f] further developed this approach using more sophisticated mathematics, specifically keeping 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 regulate a parasite population because mortality will be higher for heavily infected hosts which, when they die, claim the lives of a disproportionately large number of parasite individuals [h]. These intensity-dependent models were better able to accommodate the biology of many adult parasitic worms. Because helminths are much larger in body size than are protozoans, bacteria and viruses, Anderson and May termed their intensity-dependent models 'macroparasite' models [e,f].

Over time, the dichotomy of macro- and microparasites often shifted from a mathematical convenience based on intensity and associated with size, to a categorical classification scheme for parasites based on

size. This was because the prefixes macro- and micro- focus attention on the body sizes of the parasites and away from the issue of intensity. Beyond the modeling literature, the terms macroparasite and microparasite have devolved to a coarse taxonomy. Protozoa and smaller microbes are 'microparasites' and helminths and arthropods are 'macroparasites' even though some small parasites might be better modeled as macroparasites (e.g. *Ichthyophthirius multifiliis* ciliates or *Eimeria tenella* coccidians) and many large parasites might 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 or independent.

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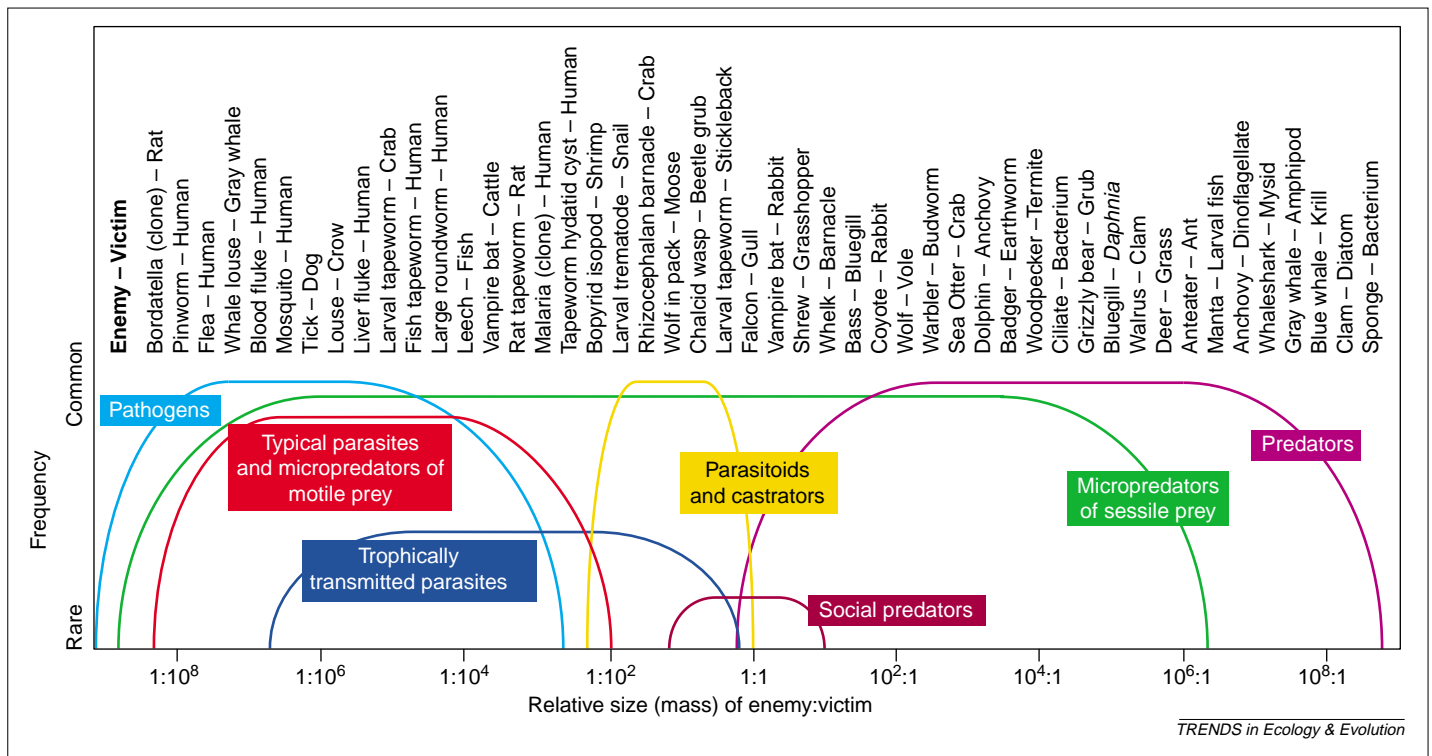


Fig. 2. Conceptual diagram and examples of the relative size/mass of natural enemies and victims for several different trophic strategies. The horizontal axis represents the relative mass of individual (or clonal) natural enemies to (individual) victims along a log scale. Colored lines indicate locations along this axis where different strategies tend to lie. Examples of these strategies are indicated above.

considers the probability of a successful attack, rather than pathology, as the relevant criterion. This reveals SOCIAL PREDATION as an intensity-dependent phenomenon that is an important functional relationship for a few major taxa (mammals and arthropods). Because social predation is carried out by predators that can also kill by themselves (SOLITARY PREDATORS), perpetrators of this trophic strategy are FACULTATIVE SOCIAL PREDATORS. For example, the spider *Parawixia bistriata* cooperates with neighbors to subdue large insects but handles small prey alone [12].

The effect of micropredation is clearly intensity dependent (1000 mosquito bites are worse than one). However, micropredators can also feed intensity independently if they are larger than their victims and multiple individuals cannot generally attack the same host individual. For example, some herbivores eat the aboveground mass of a plant in a single bite, leaving nothing for a second micropredator to consume. We do not, however, see a compelling need to divide micropredation along the dichotomy of intensity dependence.

Body size and trophic strategies of natural enemies

Anderson and May argue that enemy body size is associated with the trophic strategies of parasites (Box 3). Size is a surrogate for energetics [13] and clearly also constrains predator-prey interactions. Although body size is not a defining characteristic of trophic strategies, we hypothesize that it is the key to understanding their evolution. Figure 2 conceptually

scales the relative sizes of enemy versus victim and provides several examples [1]. Although 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), limits on the minimum relative size of predators and the maximum relative size of parasites are less obvious.

Within a species, growth enables body size to vary greatly among individuals (and with age). The importance of relative size to predators is seen when larger (and older) predators attack prey over a broader size range than do smaller conspecific predators [14,15]. Studies within and among species show how this leads to a rough isometric positive relationship between maximum prey size and predator size [16]. Trophic morphology of a predator, prey shape and anti-predation adaptations can lead to deviations from this relationship [17]. Some typical parasites show a host-parasite size correlation for interspecific comparisons [18,19]. Similarly, the size of the host can limit the upper size of an individual parasitoid or castrator within that host, forming an allometric association between parasite size and host size as the host grows [1].

Fitness correlates that vary with natural enemy and/or victim body sizes include victim encounter rate, attack success, nutrition, lifespan and proportion of mass allocated to reproductive tissue [20]. Models indicate that the optimal life histories for a particular enemy-victim, body-mass combination fall into discrete ADAPTIVE PEAKS that depend on both relative and absolute body masses; predation is best when victims are relatively small, and typical parasitism (and micropredation) is more profitable for relatively large victims [1]. Evolutionary changes

Box 4. Outstanding questions

- How do trophic strategies evolve?
- How do trophic strategies affect the evolution of other life-history traits?
- How do different trophic strategies affect victim and enemy population dynamics?
- Under what conditions are strategies likely to be plastic?
- Can changes in strategies over ontogeny lead to complex life cycles?
- What kinds of consumer strategy are not well defined by the four dichotomies?
- How might the four dichotomies apply to the trophic dynamics of consumers that don't have living victims (scavengers and detritivores)?
- If pathology is higher when multiple strains of a pathogen infect a host, is the pathogen better defined and modeled as intensity dependent?

in the body size of a natural enemy or victim might lead to other trophic strategies being more profitable (Box 2). Predictions from this approach also correspond to observations that predators and micropredators can use all but the largest victims and typical parasites and pathogens cannot use the smallest victims [1].

Micropredators of motile prey, typical parasites and predators are relatively absent when the CONSUMER:victim ratio ranges from 1:100 to 1:1, because it is a challenge to feed on a motile prey with a similar body size to oneself. Pathogen infections, although composed of many single-celled or viral individuals, reach only a small fraction of the biomass of the host. For example, malaria, although it fills most of an infected human's blood cells, amounts to <1% of the weight of the host. Similarly, a relatively large typical parasite, such as an adult *Hymenolepis diminuta* tapeworm living in a rat's gut, reaches only ~1% of the weight of a host.

In spite of the difficulty, a diverse group of natural enemies (four of the ten proposed trophic strategies) feed on prey that are similar in size to themselves. Micropredators that feed on sessile prey, freed from the need to use stealth or a size advantage, have

fewer constraints to feeding on similar sized prey than do micropredators that feed on motile prey. Trophically transmitted parasites can actively compromise the viability of an intermediate host to improve transmission. Growing large might help some of these parasites to debilitate the host, making it more susceptible to predation by the next host in the life cycle [8]. Several larval tapeworms, for example, grow nearly to the mass of an uninfected host. Parasitoid and parasitic-castration life histories also enable a parasite to feed on a host similar in size to itself. Such intimate associations require remarkably efficient resource utilization if the host is to remain viable [10].

The absolute size of the victim can also constrain trophic strategies. Parasitoids infect larval insects and other small hosts, but infect less often those hosts weighing greater than a few grams. The longer life spans of larger hosts might make the parasitoid strategy a relatively inefficient life history compared with parasitic castration. Parasitic castrators, although parasitizing a wider range of host body sizes than do parasitoids (from tiny snails to large crabs and fish), are essentially absent from hosts weighing greater than a kilogram. Hosts with very large body sizes, although long lived, tend to invest relatively little in reproductive tissue [21], presumably making parasitic castration a less efficient trophic strategy. For these reasons, large hosts, such as humans, appear unsuitable for parasitic castrators and parasitoids, a constraint that we are pleased Hollywood ignored when it portrayed the second-instar parasitoid in the 1979 film *Alien* bursting from the chest of Executive Office Kane (played by John Hurt).

Although the attack success of most predators is predicated on having a substantial size advantage over their prey, highly specialized solitary killers

Glossary

Adaptive peak: a life-history strategy resulting from an interacting cluster of traits. When reviewed across taxa, adaptive peaks are recognized as an adaptive syndrome or as a categorical life-history strategy (i.e. one that is not well described by a continuum).

Consumer: an individual that feeds on another organism.

Facultative micropredator: a predator that switches between solitary predation to micropredation, depending on the size of the prey attacked.

Facultative social predator: a predator that switches from solitary predation to social predation, depending on the size of the prey attacked.

Intensity dependent: an effect (such as on victim fitness) that changes with intensity.

Intensity independent: an effect (such as on victim fitness) that does not change with intensity.

Intensity*: the number of natural enemies per victim at the time of attack.

Macroparasite: a parasite with intensity-dependent pathology (e.g. an adult trematode in a vertebrate).

Microparasite: a parasite with intensity-independent pathology (e.g. larval trematode redia in a snail).

Micropredator: a predator that does not kill its prey (by eating only part of it; e.g. mosquito).

Natural enemy*: a consumer that harms its victim (used commonly in the biological control literature to describe predators, parasitoids and parasites).

Parasite*: a natural enemy that feeds intimately on one host.

Parasitic castrator: a parasite that blocks host reproduction, sometimes called a sterilizing pathogen in the plant literature.

Parasitoid*: a parasite that kills its host as a normal and required part of its development.

Pathogen*: parasites (e.g. viruses, bacteria and protozoans) that reproduce asexually within the host but do not necessarily kill or castrate the host as part of their life cycle. The effect on the host is not intensity dependent (these are microparasites in the Anderson and May models).

Pathology: the nature of the effect of a natural enemy on a victim (virulence includes the aspects of pathology that increase parasite fitness).

Predator*: a natural enemy that feeds on more than one victim.

Social predation: when a predator feeds in groups and shares prey (attack success is intensity dependent).

Solitary predator: a predator that feeds alone on prey.

Trophically transmitted: parasitic castrator, pathogen or typical parasite is transmitted via predation to the next host.

Typical parasite: a parasite whose effect on the host is intensity dependent.

*Some of these terms are used less precisely in the vernacular (e.g. parasite or pathogen). Others, as technical terms, we define more precisely (e.g. intensity or typical parasite), and others are applied conceptually although they were originally associated with a limited set of taxa [e.g. parasitoid (insects), predator (e.g. carnivores) or pathogen (e.g. viruses, bacteria, fungi or protozoa)].

(e.g. weasels, vipers and wind scorpions) can be just one third the mass of their prey. Facultative social predators compensate for a size disadvantage with a numerical advantage. Canine packs, lion prides, killer whale pods, army ant colonies and aboriginal human tribes can capture and share prey significantly larger than themselves because of their social organization.

Conclusions and future directions

Placing concepts developed for population modeling in an evolutionary context has enabled us to recognize four life-history dichotomies, distinguishing ten trophic strategies. Although any such scheme might put simplification at odds with reality, we find only a few natural enemies that are not well described by this approach (Box 4). Hence, this approach provides a transparent means with which to describe, define and relate the bulk of the diversity of trophic strategies used by animals.

Most animals are parasites that, at some point in their lineage, evolved from a predator. Yet, surprisingly little attention has been paid to the selective pressures leading to different trophic strategies and the means by which a species might evolve a different strategy, develop a plastic strategy, or shift from one strategy to another within a lifecycle.

We hypothesize that the evolution of body size plays a large role in the evolution of trophic strategies. Furthermore, we hypothesize that trophic strategies can drive the evolution of body size in natural enemies and victims.

The most tangible outcome of our approach is that recognition of a diversity of distinct trophic strategies should greatly improve efforts to model population dynamics. This approach should also better enable predictions of the role of natural enemies in ecosystems. For example, tapeworms, fleas, foxes and hares differ sufficiently in their feeding strategies to merit different population modeling approaches. In addition, the tapeworm, being a trophically transmitted parasite in the flea and a typical parasite in the fox, deserves a two-stage model. As a next step, we should explore how the ten trophic strategies of natural enemies differ from each other in their population dynamics. Such systematic assessments are rare in theoretical population biology but could reveal the independent and interdependent effects of the four dichotomies on population dynamics. Anderson and May's (Box 3) fruitful efforts to compare and contrast the effects of the intensity-dependence dichotomy, distinguishing typical parasites from pathogens, illustrate the promise of this approach.

Acknowledgements

We would like to thank Andy Dobson, John Endler, Robert Warner, Karen Phillips and our graduate students for assistance with this article, which was prepared with funds from NSF through the NIH/NSF Ecology of Infectious Disease Program (DEB-0224565).

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