EVOLUTIONARY ECOLOGY OF TROPHICALLY TRANSMITTED PARASITES

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ABSTRACT: Trophic transmission is a distinctive parasitic life-history strategy. The evolution and ecology of trophically transmitted associations have been considered only recently. Its recognition as a distinctive adaptive peak has some important ecological consequences. Unlike typical parasites, death of the host is not necessarily unfortunate for its resident, trophically transmitted parasites (TTPs), for the following reasons. (1) Trophic transmission is widespread among helminth life histories and is also a feature of many protozoan life cycles. (2) Trophically transmitted parasites often, perhaps generally, modify the behavior of their prey hosts, making them more likely to be eaten by their predator hosts. Hence, they are potentially highly virulent to their prey hosts but may generally be avirulent to their predator hosts. (3) For TTPs, trade-offs between parasite reproductive rate and virulence may be less than for typical parasites. (4) Intensity-dependent competitive effects (crowding effects) may be less likely to occur, reducing the cost of aggregation. (5) In the prey host, the slope of the intensity–pathology curve is predicted to be greater than, or equal to, 1, and the shape of this curve is unlikely to be asymptotic.

TROPHIC TRANSMISSION AS AN ADAPTIVE PEAK

Parasites transmitted through an act of predation share a suite of life-history features providing them a distinctive life-history strategy. Here, I will offer a perspective on some of the notable selective forces distinguishing their life histories and discuss some of the ecological attributes of these life histories. Then, I will compare trophically transmitted parasites (TTPs) with other intimate and durable, i.e., parasitic, life histories that have been traditionally modeled as microparasites and macroparasites.

Sewall Wright (1931) recognized that distinctive ecologies select for a cluster of interacting traits and that these are maximized as adaptive peaks. Convergent life histories will share many of these traits. Hence, they can be recognized as adaptive syndromes. We have applied this concept to consumer strategies and have been able to distinguish major axes for these syndromes in the adaptive landscape (Kuris, 1974, 1997; Kuris and Lafferty, 2000; Lafferty and Kuris, 2002). As detailed in Kuris and Lafferty (2000), these adaptive peaks are determined by the nature of the individual interaction between a consumer and its host (or prey). In a complex life cycle, each consumer life-history phase may have a distinctive interaction with its host (or prey), and the progeny of asexual reproduction are evaluated as a single individual.

The first dichotomy separating adaptive syndromes is the number of hosts attacked per life-history phase. If more than 1 host (or prey) is attacked, then the adaptive syndromes of predation and micropredation (nonlethal feeders such as mosquitoes) can be distinguished from those life histories involving only 1 host. The latter are generally intimate and durable associations, as characterized by Combes (2001). Table I lists 7 intimate and durable adaptive syndromes and the 3 further dichotomies that unequivocally distinguish among them. These dichotomies include effects on host fitness, host death, and whether or not they are intensity dependent. Briefly, 1 dichotomy distinguishes whether the individual symbiont necessarily reduces host fitness to zero (parasitoids and parasitic castrators) or not (typical parasites and pathogens). Another dichotomy asks whether the death of the host is required for continuation of the life cycle of the symbionts or whether death of the host is not so required. This dichotomy recognizes parasitoids and TTPs as distinct from typical parasites, pathogens, and parasitic castrators. Finally, in the last dichotomy (see Table I), the intensity dependence of the

symbiont on the fitness of its host distinguishes intensity-dependent typical parasites, meeting the principal assumptions of macroparasite models (May and Anderson, 1979; Kuris and Lafferty, 2000), from intensity-independent pathogens, meeting the assumptions of microparasite models (Anderson and May, 1979; Kuris and Lafferty, 2000). Similarly, it separates trophically transmitted typical parasites from trophically transmitted pathogens, and trophically transmitted parasitic castrators from parasitoids and parasitic castrators.

COMPLEX LIFE CYCLES

Trophic transmission necessarily requires a complex (multiple host) life cycle. These complex life cycles are presumably derived from parasites with 1-host life cycles. Asymmetries in the outcome of a predatory event for the parasite and the host have selected for distinctive traits of the parasite in prey rather than in predator hosts (Kuris, 1997; Lafferty, 1999). I hypothesize that in either the prey or the predator host (or both), traits such as pathogenicity, intraspecific interactions, and virulence trade-offs may differ from those of an otherwise similar parasite that uses a single host.

Parasite-increased trophic transmission (PITT) is almost ubiquitously associated with complex life cycles involving predation on intermediate hosts. This type of intermediate host behavior modification is remarkable for its severe fitness consequences on these hosts. A developing body of theory (Holmes and Bethel, 1972; Moore, 1984; Holmes and Price, 1986; Dobson, 1988; Freedman, 1990; Combes 1991; Lafferty, 1992, 1999; Kuris, 1997; Choisy et al., 2003) supports the hypothesis that, for the TTP, there is a trade-off in fitness consequences for prey and predator hosts. A strong impact on the intermediate host, leading to ready capture by the definitive host, implies that such an infected host is detectable by the predator. If there were also a strong fitness decrement for the predator host, it would select for avoidance of such hosts. Consequently, several features associated with typical parasites in the predator host are hypothesized to be not only unlike the effects of the same parasite in its prey host but also unlike otherwise similar parasites that have direct transmission or have otherwise complex life cycles that do not involve trophic transmission.

The hypothesized life-history contrasts among intermediate host, final host, and non-TTP hosts are summarized in Table II. Digeneans are a taxonomic group suitable for such compari-

TABLE I. Parasite strategies or adaptive syndromes as recognized by dichotomies of effects on host fitness, whether host mortality is required to complete development, and whether pathology is density dependent. See Kuris and Lafferty (2000) and Lafferty and Kuris (2002) for an additional dichotomy distinguishing predator life histories.

Parasite strategy/adaptive syndrome	Host fitness	Is death of host required?	Is pathology intensity dependent?
Typical parasite	>0	No	Yes
Pathogen	>0	No	No
Trophically transmitted parasite	>0	Yes	Yes
Trophically transmitted pathogen	>0	Yes	No
Parasitic castrator	0	No	No
Trophically transmitted parasitic castrator	0	Yes	Yes
Parasitoid	0	Yes	No

sons. Most are trophically transmitted from the second intermediate host to the definitive host, e.g., heterophyids, microphallids, and echinostomes. However, others are not, e.g., schistosomes. Some are trophically transmitted but cannot alter host behavior, e.g., *Fasciola hepatica* (metacercariae encyst on plants), or *Parorchis acanthus* or *Himasthla rhigedana*, which encyst on the exterior surfaces of mollusk shells, operculae, or crab exoskeletons (Stunkard and Cable, 1932; Adams and Martin, 1963). There are also some TTPs that engage in strong prey–host behavior modification but whose altered behavior is masked so that it is unlikely to be detected by the definitive predator host, e.g., *Dicrocoelium* spp. In this case the infected ant clings to the tips of vegetation at midday, making it much more prone to be eaten by the grazing final host (sheep) than an uninfected ant (Anokhin, 1966)). Similar comparisons can be made for other taxa. Nematodes include species with direct life cycles, e.g., *Ascaris lumbricoides*, and related species having life cycles that involve trophic transmission, e.g., anisakids, *Baylisascaris procyonis*—facultatively (Kazacos and Boyce, 1989). Coccidia include species with direct life cycles (*Eimeria stiedae*) and those involving trophic transmission, e.g., *Toxoplasma gondii* and *Neospora caninum*.

The negligible pathogenicity of TTPs in their final (predator) hosts has been noted and briefly reviewed (Lafferty, 1992; Kuris, 1997). Related parasites with direct life cycles or transmission that does not require predation are often quite pathogenic and may alter behavior either as a pleiotrophic effect of their virulence or to aid transmission of the parasite. Although a comprehensive review of these comparisons is not available, it appears likely that the pattern holds. Species directly transmitted to final (or sole) hosts are often pathogenic, e.g., human schistosomes (Cheever, 1968), dog hookworms (Georgi et al., 1969), and rabbit coccidians (Dunsmore, 1971), whereas in the predatory hosts, their counterparts are not (or are less so), e.g., heterophyids (Kuris, 1997), raccoon roundworms (Kazacos, 2001), and *T. gondii*. All of these are notable behavior modifiers of their intermediate hosts (e.g., Kazacos et al., 1981; Berdoy et al., 1995; Lafferty and Morris, 1996).

Predatory hosts of TTPs that do not or cannot alter prey host behavior (because the host is a plant or because they are encysted on outer surfaces) may experience evident pathogenicity, e.g., *F. hepatica* (Martinez-Moreno et al., 1999). The effects of *H. rhigedana* and *P. acanthus* in their final hosts have been little studied, but these are relatively large and long-lived worms that may be pathogenic. Similarly, masked TTPs (such as *D. dendriticum*) are also pathogenic in their final hosts (Massoud, 1981).

INTENSITY AND PATHOLOGY

The effect of most typical parasites on their hosts is intensity dependent. Well-studied examples include the human schistosomes (Cheever, 1968) and whipworms (Gilman et al., 1983). Although the relationship between intensity and pathology is a key aspect of macroparasite models (May and Anderson, 1979), the shape of the intensity–pathology curve has been little investigated. Comparative studies are not available. Figure 1 depicts 3 possible intensity–pathology curves representing hypo-

TABLE II. Comparison of life-history traits and host–parasite relationships for TTPs in their intermediate hosts, as typical parasites in their final hosts, and for nontrophically transmitted typical parasites in their definitive hosts.*

Trait	Intermediate host	Final host	Nontrophically transmitted host
Behavior modification	High	None	Present, variable
	None (unable to alter)	Present	
	High (masked)	Present	
Intensity/pathology	High	None	Often present
Crowding effects	None	May be high, variation not examined	Present, varible
	Often present (TTPC)	May be high	
Individual virulence	High	None	Present, variable
Cost of virulence	None	May be high, variation not examined	Present, variable
Aggregation	High	Low	Variable

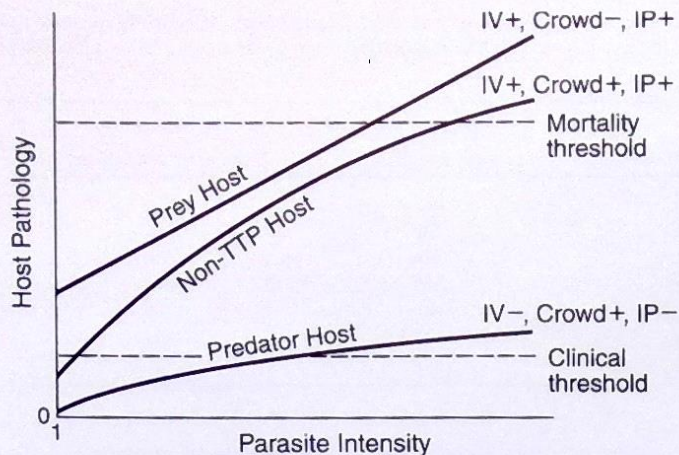* TTPC, trophically transmitted parasitic castrator.

FIGURE 1. Hypothetical relationships between parasite intensity and host pathology for a TTP in its intermediate (prey) host, in its final (predator) host, and in a typical parasite that is not trophically transmitted. TTP here excludes those whose PITT is masked and those that cannot alter host behavior. Lower and upper dashed lines represent the thresholds for clinical detection of the pathology and the threshold at which the parasite is a probable contributor to host mortality, respectively. The principal conditions generating such curves are given to the right of the respective curves: IV + and − represent parasites with high and low individual virulence, respectively, Crowd + and − represent parasites with strong as opposed to weak crowding effects, respectively, and IP + and − represent parasites with strong or weak individual incremental pathology effects, respectively. The position of the non-TTP curve is arbitrary when compared with the other 2 curves. The features captured by the depicted curve are the low (but not nil) per capita virulence and the asymptotic population effect.

thetical relationships for a nontrophically transmitted typical parasite, a TTP in its prey host, and a TTP as a typical parasite in its predator host. It is hypothesized that these curves are derived from the interactions between 3 key parameters: the virulence (pathology-inducing potential) of an individual parasite, crowding effects (whether competitive intraspecific interactions or host-mediated defensive responses), and the slope of the additive effects of the pathological potential of accumulating individual parasites. First, for a non–trophically transmitted typical parasite, individual virulence is often low, increases strongly as intensity builds up, and may be ameliorated by crowding effects. *Haemonchus contortus* in sheep (LeJambre et al., 1971) and *Heligmosomoides polygyrus* in mice (Keymer and Hiorns, 1986) provide representative examples. Second, in an intermediate prey host, a TTP should have a very different relationship. There is no trade-off between longevity and parasite fitness in an intermediate host, but rather there is a potentially positive interaction as more parasites increase behavior modification (e.g., Lafferty and Morris, 1996). This will increase the likelihood of transmission (Lafferty and Morris, 1996). The intensity–pathology curve should not be asymptotic (on account of crowding effects) but should rather rise monotonically as parasites accumulate. Third, however, because of trade-offs associated with PITT, parasites that were trophically transmitted to their predator hosts will generally have very low individual virulence and may show a strong crowding effect and a very low incremental increase (low slope) for the intensity–pathology relationship. *Hymenolepis diminuta* in the rat provides a classic example (Roberts, 1961).

CROWDING

Decreases in body size, fecundity, or suboptimal site use have been frequently associated with increased intensity, e.g., *Ancylostoma caninum* (Krupp, 1961), *Ostertagia ostertagi* (Michel et al., 1978), for nontrophically transmitted parasites (Table II). These have been loosely termed as crowding effects. They may be due to intraspecific competitive interactions or may be host-mediated. In contrast, crowding effects are rarely reported for TTPs in their intermediate hosts (there is a dearth of data here for trematode metacercariae, acanthocephalan cystacanths, and tapeworm cysticercoids, perhaps because there was little interesting variation observed). I hypothesize that this is generally so, and it is adaptive because each of these larval stages can thereby add a proportional incremental impact on host behavior modification for PITT. In contrast, crowding effects are often noted for TTPs as typical parasites in their final predator hosts. *Hymenolepis diminuta* is a spectacular example in that 100 worms in a host weigh no more than a worm in a single infection (Read and Simmons, 1963). However, this is not always the case. Marcogliese (1997) did not detect a significant effect of intensity on growth or on fecundity of the sealworm, *Pseudoterranova decipiens*, in its final gray seal host. The overall pattern and the reasons for variation in crowding effects of TTPs in their final hosts merit further investigation.

There are also some interesting exceptions to the pattern for TTPs in their intermediate hosts. These involve representatives of the trophically transmitted parasitic castrator adaptive peak. The pseudophyllidean tapeworm, *Schistocephalus solidus*, exhibits a strong crowding effect in its stickleback intermediate host. Further, in a single infection, a plerocercoid worm may reach 30% of the weight of the host (Pennycuick, 1971). Perhaps this is due to the castration effect. Parasitic castrators are able to divert considerable host reproductive energy to their own growth (Kuris, 1974; Kuris and Lafferty, 2000). Under conditions of limited energy available for other worms, strong growth suppression of potential competitors would be predicted (Kuris, 1974). In fact, most of the growth of these worms occurs in the intermediate host. When consumed by the predatory avian final host, these precocious worms mature rapidly, commence and complete egg production, and die. The size differential initiated in the stickleback is merely passed along to the bird.

VIRULENCE

The concept of virulence is too complex to be fully considered here. Accepting the definition of Ebert and Hamilton (1996) and Combes (2001), a parasite-induced loss of host fitness is "virulence." I apply this explicitly on a per capita basis for individual parasites. It is an attribute of the parasite measured in host units. Importantly, virulence generally has a cost to the parasite. Too great an effect on the host might decrease the ultimate success (transmission) of the parasite (Combes, 2001).

Nontrophically transmitted typical parasites are virulent on a per capita basis. However, this may vary because virulence may come at a wide range of costs. They are, however, rarely very virulent on a per capita basis (as shown in Fig. 1). The cost (death of their host) is generally too great. Consequently, it is
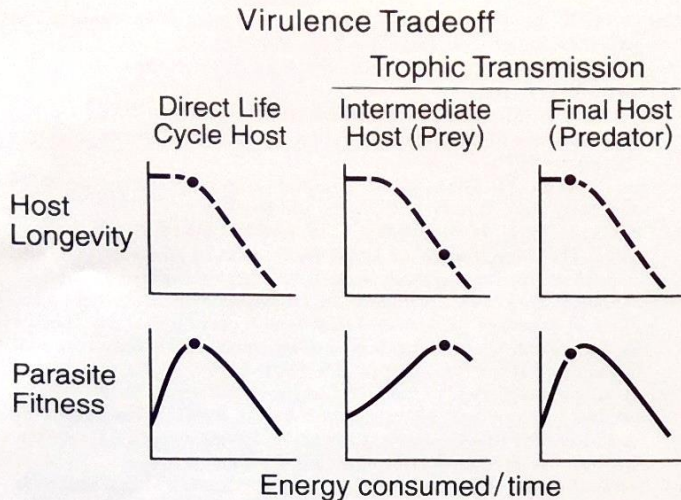
Virulence Tradeoff

FIGURE 2. A comparison of virulence trade-offs with host longevity and parasite fitness with respect to energy consumed by the parasite per unit time. The model is for phylogenetically similar parasites in similar hosts. Trade-offs are shown for parasites with direct life cycles, for TTPs in their prey hosts, and for TTPs acting as typical parasites in their predator hosts.

countered by selection. However, TTPs in their prey host must be considered quite virulent (because of PITT); the cost of virulence is probably quite low but not nil if they increase risk of death due to reasons other than predation by an appropriate definitive host. Consequently, the easy delivery of these parasites to final predator hosts will generally select for low virulence in the predator hosts. Although they are generally avirulent (often small and ephemeral), e.g., avian hymenolepidid cestodes and heterophyid trematodes, they can also be large and avirulent, e.g., *H. diminuta*. Presumably, the cost of virulence is so great that selection against virulence is very strong in the predator host. Thus, the relationship between the cost of virulence and the fitness gain to the TTP leads to opposite effects on their prey as compared to their predator hosts.

Figure 2 summarizes the virulence trade-off. In a parasite that is not trophically transmitted, such as one with a direct life cycle, host longevity will decline if parasites consume too much energy. Hence, parasite fitness is maximized near the consumption rate that does not cost an excessive decrease in host longevity. In the intermediate prey host, much more energy can be consumed by the parasite if it can guide the host to an appropriate death (predation by a suitable final host). So, it follows that in the final host little energy can be consumed (which would avoid the often detectable behavior modifications yielding PITT). Hence, host longevity will be minimally affected.

AGGREGATION

It is widely recognized that parasites are often highly aggregated among their hosts (Crofton, 1971; May and Anderson, 1979), although their distribution tends to be more uniform for parasitoids and parasitic castrators, and for some typical parasites, e.g., species of *Gyrocotyle* cestodarians (Simmons and Laurie, 1972), and insect pinworms (Adamson and Noble, 1993). Crowding effects and intensity–pathology relationships potentially limit aggregation. Hence, I hypothesize that infec-

tion dynamics may aggregate TTPs in their prey hosts (it is to their potential mutual benefit to increase their impact on particular hosts), but aggregation can be greatly decreased by PITT effects on heavily parasitized hosts. Because they are often ephemeral in their final hosts, they will not experience the accumulating aggregative effects of time. It would be of interest to review the literature on aggregation to determine whether these hypotheses are supported.

CONCLUSIONS

Several hypotheses concerning the life history of TTPs have been proposed and contrasted with those of nontrophically transmitted typical parasites. Data are available in the literature to test some of these hypotheses.

Consideration of the differential selection of a parasite in the different hosts traversed along a complex life cycle strongly suggests that to model such parasites requires equivalent attention to all hosts in such a life cycle. It is simply, and perhaps dangerously, unrealistic to black box 1 of the hosts (usually the invertebrate intermediate host), seemingly as a vertebrate conceit.

Finally, knowledge of these behavioral and life-history features may provide insights into the improved management of trophically transmitted diseases of concern for human health, animal husbandry, and wildlife diseases.

LITERATURE CITED

ADAMS, J. E., AND W. E. MARTIN. 1963. Life cycle of *Himasthla rhigedana* Dietz, 1909 (Trematoda: Echinostomatidae). Transactions of the American Microscopical Society 82: 1–6.

ADAMSON, M. L., AND S. J. NOBLE. 1993. Interspecific and intraspecific competition among pinworms in the hindgut of *Periplaneta americana*. Journal of Parasitology 79: 50–56.

ANDERSON, R. M., AND R. M. MAY. 1979. Population biology of infectious diseases. Part 1. Nature 280: 361–367.

ANOKHIN, T. A. 1966. Daily rhythm in ants infected with metacercariae of *Dicrocoelium lanceolatum*. Doklady Akademia Nauk SSSR 166: 75–79.

BERDOY, M., J. P. WEBSTER, AND D. W. MACDONALD. 1995. Parasite-altered behaviour: Is the effect of *Toxoplasma gondii* on *Rattus norvegicus* specific? Parasitology 111: 403–409.

CHEEVER, A. W. 1968. A quantitative post-mortem study of schistosomiasis mansoni in man. American Journal of Tropical Medicine and Hygiene 17: 38–64.

CHOISY, M., F. THOMAS, S. BROWN, AND K. D. LAFFERTY. 2003. The evolution of complex multi-host life cycles. American Naturalist. [In press.]

COMBES, C. 1991. Ethological aspects of parasite transmission. American Naturalist 138: 866–880.

———. 2001. Parasitism: The ecology and evolution of intimate interactions. University of Chicago Press, Chicago, Illinois, 728 p.

CROFTON, H. D. 1971. A model of host-parasite relationships. Parasitology 63: 343–364.

DOBSON, A. P. 1988. The population biology of parasite-induced changes in host behavior. The Quarterly Review of Biology 63: 139–165.

DUNSMORE, J. D. 1971. A study of the biology of the wild rabbit in climatically different regions in eastern Australia: IV. The rabbit in the south coastal region of New-South-Wales an area in which par-

asites appear to exert a population regulating effect. Australian Journal of Zoology **19**: 355–370.

EBERT, D., AND W. D. HAMILTON. 1996. Sex against virulence: The coevolution of parasitic diseases. Trends in Ecology and Evolution **11**: 79–82.

FREEDMAN, H. I. 1990. A model of predator-prey dynamics as modified by a parasite. Mathematical Biosciences **99**: 143–155.

GEORGI, J. R., L. F. LEJAMBRE, AND L. H. RACTLIFF. 1969. *Ancylostoma caninum* burden in relationship to erythrocyte loss in dogs. Journal of Parasitology **55**: 1205–1211.

GILMAN, R. H., Y. H. CHONG, C. DAVIS, B. GREENBERG, H. K. VIRIK, AND H. B. DIXON. 1983. The adverse consequences of heavy *Trichuris* infection. Transactions of the Royal Society of Tropical Medicine and Hygiene **77**: 432–438.

HOLMES, J. C., AND W. M. BETHEL. 1972. Modification of intermediate host behaviour by parasites. *In* Behavioural aspects of parasite transmission, E. U. Canning and C. A. Wright (eds.). Academic Press, London, U.K., p. 123–149.

———, AND P. W. PRICE. 1986. Communities of parasites. *In* Community ecology: Pattern and process, J. Kikkawa and D. J. Anderson (eds.). Blackwell Scientific, Boston, Massachusetts, p. 187–213.

KAZACOS, K. R. 2001. *Baylisascaris procyonis* and related species. *In* Parasitic diseases of wild mammals, 2nd ed., W. M. Samuel, M. J. Pybus, and A. A. Kocan (eds.). Iowa State University Press, Ames, Iowa, p. 301–341.

KAZACOS, K. R., G. O. APPEL, AND H. L. THACKER. 1981. Cerebrospinal nematodiasis in a woodchuck suspected of having rabies. Journal of the American Veterinary Medical Association **179**: 1102–1104.

———, AND W. M. BOYCE. 1989. *Baylisascaris* larval migrans. Journal of the American Veterinary Medical Association **195**: 894–903.

KEYMER, A. E., AND R. W. HIORNS. 1986. Faecal egg counts and nematode fecundity: *Heligmosomoides polygyrus* and laboratory mice. Parasitology **93**: 189–203.

KRUPP, I. M. 1961. Effects of crowding and of superinfection on habitat selection and egg production in *Ancylostoma caninum*. Journal of Parasitology **47**: 957–961.

KURIS, A. M. 1974. Trophic interactions: Similarity of parasitic castrators to parasitoids. The Quarterly Review of Biology **49**: 129–148.

———. 1997. Host behavior modification: An evolutionary perspective. *In* Parasites and pathogens: Effects on host hormones and behavior, N. E. Beckage (ed.). Chapman and Hall, New York, New York, p. 293–315.

———, AND K. D. LAFFERTY. 2000. Parasite-host modeling meets reality: Adaptive peaks and their ecological attributes. *In* Evolutionary biology of host-parasite relationships: Theory meets reality, R. Poulin, S. Morand, and A. Skorping (eds.). Elsevier Science B. V., Amsterdam, The Netherlands, p. 9–26.

LAFFERTY, K. D. 1992. Foraging on prey that have been modified by parasites. American Naturalist **140**: 854–867.

———. 1999. The evolution of trophic transmission. Parasitology Today **15**: 111–115.

———, AND A. MORRIS. 1996. Altered behavior of parasitized killifish increases susceptibility to predation by bird final hosts. Ecology **77**: 1390–1397.

———, AND A. M. KURIS. 2002. Trophic strategies, animal diversity and body size. Trends in Ecology and Evolution **17**: 507–513.

LEJAMBRE, L. F., L. H. RACTLIFFE, L. S. UHAZY, AND J. H. WHITLOCK. 1971. Fecal egg output of lambs in relation to *Haemonchus contortus* burden. International Journal for Parasitology **1**: 157–160.

MARCOGLIESE, D. J. 1997. Fecundity of sealworm (*Pseudoterranova decipiens*) infecting grey seals (*Halichoerus grypus*) in the Gulf of St. Lawrence, Canada: Lack of density-dependent effects. International Journal for Parasitology **27**: 1401–1409.

MARTINEZ-MORENO, A., V. JIMENEZ-LUQUE, T. MORENO, E. S. H. REDONDO, J. M. DE LAS MULAS, AND J. PEREZ. 1999. Liver pathology and immune response in experimental *Fasciola hepatica* infections of goats. Veterinary Parasitology **82**: 19–33.

MASSOUD, J. 1981. Histopathology of liver in Iranian sheep naturally infected with *Dicrocoelium dendriticum*. Annals of Tropical Medicine and Parasitology **75**: 293–298.

MAY, R. M., AND R. M. ANDERSON. 1979. Population biology of infectious diseases. 2. Nature **280**: 455–461.

MICHEL, J. F., M. B. LANCASTER, AND C. HONG. 1978. The length of *Ostertagia ostertagi* in populations of uniform age. International Journal for Parasitology **8**: 437–441.

MOORE, J. 1984. Altered behavioral responses in intermediate hosts: An acanthocephalan parasite strategy. American Naturalist **123**: 572–577.

PENNYCUICK, L. 1971. Frequency distribution of parasites in a population of three-spined sticklebacks, *Gasterosteus aculeatus*, with special reference to the negative binomial distribution. Parasitology **63**: 389–406.

READ, C. P., AND J. E. SIMMONS. 1963. Biochemistry and physiology of tapeworms. Physiological Reviews **43**: 263–305.

ROBERTS, L. S. 1961. The influence of population density on patterns and physiology of growth in *Hymenolepis diminuta* (Cestoda: Cyclophyllidea) in the definitive host. Experimental Parasitology **11**: 332–371.

SIMMONS, J. E., AND J. S. LAURIE. 1972. A study of *Gyrocotyle* in the San Juan Archipelago, Puget Sound, U.S.A., with observations on the host, *Hydrolagus colliei* (Lay and Bennett). International Journal for Parasitology **2**: 59–77.

STUNKARD, H. W., AND R. M. CABLE. 1932. The life history of *Parorchis avitus* (Linton) a trematode from the cloaca of the gull. Biological Bulletin **42**: 328–338.

WRIGHT, S. 1931. Evolution in Mendelian populations. Genetics **16**: 97–159.