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The Evolution of Trophic Transmission

K.D. Lafferty

Parasite increased trophic transmission (PITT) is one of the more fascinating tales of parasite evolution. The implications of this go beyond cocktail party anecdotes and science fiction plots as the phenomenon is pervasive and likely to be ecologically and evolutionarily important. Although the subject has already received substantial review, Kevin Lafferty here focuses on evolutionary aspects that have not been fully explored, specifically: (1) How strong should PITT be? (2) How might sexual selection and limb autotomy facilitate PITT? (3) How might infrapopulation regulation in final hosts be important in determining avoidance of infected prey? And (4) what happens when more than one species of parasite is in the same intermediate host?

Some of the most compelling Nature documentaries are those that show hunting and feeding behavior, such as a lioness downing a gazelle, or an osprey snatching a fish from the water's surface. Hidden from view and never mentioned are the parasites that are experiencing transmission at that moment. Unfortunately, parasite transmission loses some of this drama when portrayed to undergraduates as arrows in a parasite life cycle. A perusal of life cycle diagrams in any parasitology text will reveal that many 'typical' (as in Ref. 1) parasites (many nematodes, most trematodes, most cestodes and all acanthocephalans) depend on a definitive host eating an intermediate host. Such trophic transmission is conspicuously absent in a few groups such as the monogenes, gyrocotylid Cestodaria, rhabditoid, oxyuroid and filarial nematodes and schistosomatid trematodes[2]. Trophic transmission might have evolved under the strong selective pressure to survive the death of the host by predation, a feat most easily accomplished by parasitizing the host's predator. In addition to surviving, parasites able to succeed at this would typically enter a larger and longer-living host.

Many parasites that achieve transmission via the food chain alter the behavior or appearance of intermediate hosts to increase their risk of being preyed upon by final hosts (reviewed in Refs 3–6). Broad categorical

Kevin D. Lafferty is with the US Geological Survey, Marine Science Institute, University of California, Santa Barbara, CA 93106, USA. **Tel: +1 805 893 8778, Fax: +1 805 893 8062, e-mail: Lafferty@lifesci.UCSB.edu**

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terms that include this phenomenon are variations of 'behavior modification' or 'manipulative parasites'. Recognizing that parasites manipulate hosts for reasons other than trophic transmission and noting that alterations to hosts were not strictly behavioral, Kuris[2] coined the more specific term 'parasite increased susceptibility to predation' to represent a parasite manipulation that increases predation on intermediate hosts. However, because increased predation is not necessarily adaptive (if, for example, the predator is not an appropriate definitive host), I will refine the term as 'parasite increased trophic transmission' (PITT). The evolution of this strategy is obvious from the parasite's perspective: in general, increased transmission will mean increased parasite fitness.

Although it is tempting to assume that all host behavioral changes are adaptations for transmission, such changes might also represent side effects of pathology or host defense[7]. Most reviews have stressed that further work needs to be carried out to distinguish between these alternatives. In addition, Moore and Gotelli[4] point out the alternative hypothesis that modification of some host behaviors may be consequences of constraints of the host's phylogeny. In other words, the ability to modify a host's behavior may have been adaptive in a host's ancestor, even though it does not act to increase transmission in the host today. In contrast, PITT might have evolved in an ancestral host, but continues to increase transmission in derived hosts. The same logic holds for the influence of parasite phylogeny on PITT[8]. As an example, for four trematode species infecting bivalves, PITT is arguably the result of convergent evolution in three; while for one species, there is evidence for a phylogenetic constraint[9]. It would be interesting to follow the evolution of PITT using both host and parasite phylogeny. Irrespective of phylogenetic constraints, to determine whether behavior modification is adaptive, one must demonstrate that the altered behavior increases parasite transmission.

How deep a PITT?

How dramatic should we expect PITT to be? If resources needed for PITT come at the expense of a parasite's future reproductive success, parasites should invest an optimal intermediate amount of energy in PITT[5,10]. An alternative hypothesis is that parasites will evolve to minimize energetic costs and that PITT will derive from mechanisms that are energetically efficient or are byproducts of infection or pathology.

Dobson[11] provided one of the first reviews about the strength of behavior modification, noticing interesting variation among parasite taxa, with acanthocephalans being the most potent and consistent modifiers. More recently, Poulin[6] conducted a meta-analysis of 114 published comparisons of behavior modification and found that parasites (nematodes, acanthocephalans and cestodes) moderately, but significantly, alter host activity and habitat choice. Nematodes and cestodes alter host activity the most. This effect is strongest for non-trophically transmitted nematodes (consistent with the idea that behavior modification can be a strategy suitable for several modes of transmission). Nematodes and acanthocephalans alter host microhabitat choice the most. Little information was available for trematodes at that time. The strength of behavior modification does not differ between vertebrate and invertebrate hosts, suggesting that the size of the host does not represent an obstacle to behavior modification.

The moderate alterations seen in these comparisons deserve comment. Parasites may not alter behavior during the entire course of an infection (underestimating effect size), some taxa were disproportionately represented (unpredictably biasing average effect size) and there may have been a bias towards publishing significant effects (increasing average effect size)[6]. The latter problem means that it is difficult to tell what proportion of parasites have no effect on host behavior without a systematic study specifically designed to test this hypothesis. In addition, it is plausible that within-study effect sizes of PITT were systematically underestimated by projecting the effect merely from measurements of host behavior. For example, a trematode that alters the measured behavior of killifish fourfold, increases transmission 30-fold[12]. I suspect that this disparity is commonplace because most studies focus on a single behavior, quantified according to human interpretation. If the parasite alters host behavior to increase transmission, a human interpretation of a limited suite of behaviors is very likely to be a conservative estimate of a predator's reaction to an infected host. This will lead to a substantial underestimate of the parasite's ability to increase transmission. Even when one measures transmission directly, it is possible to underestimate PITT. Urdal *et al*.[13] found that a cestode strongly alters its copepod host's behavior, but they could not detect PITT in trials with predatory fishes. This might have been due to a weak experimental design or analysis that could not detect a statistical significance despite a potential effect on predation rates (on average, fish were 1.25 times more likely to eat infected than uninfected copepods). In addition, predation trials without prey replacement will underestimate the magnitude of PITT (unless one accounts for this analytically) because infected prey become relatively rare in the prey population over the course of the trial[12].

Intermediate hosts

If infection leads to increased predation, intermediate hosts should be under strong selective pressure to resist PITT[1,5,14]. Consequently, the magnitude of PITT might be the outcome of an evolutionary arms race between virulence and resistance. The literature is replete with examples of how parasites evade host defenses and how hosts respond to infection. Some parasites infect components of the vertebrate central nervous systems (CNS), perhaps because the CNS is poorly guarded by the host's immune system[15]. Exploiting this haven may have the additional effect of providing the parasite with a potent site from which to launch PITT. I propose that there is another possible evolutionary trajectory for PITT: to receive less resistance from its host, a parasite might alter behaviors that increase transmission more than they reduce host fitness. There are at least two host traits that parasites could exploit to accomplish this: secondary sex characteristics and limb autotomy (intentional release of a limb as seen in crab legs and lizard tails).

Much has been made of the hypothesis that male secondary sexual traits might be a way of advertising parasite resistance to prospective mates[16]. For example, Rosenqvist and Johansson[17] found that tattoos resembling the black epidermal metacercariae of *Cryptocotyle* make pipefish less attractive to mates. Möller[18] provided a counter example of a fungus that apparently enlarges a fly's abdomen, such that it appears swollen with eggs, a manipulation that stimulates mate choice in males.

Darwin's realization[19] that conspicuous sexually selected traits might increase predation risk suggests that 'love-potion' parasites could also increase trophic transmission. For example, male fiddler crabs spend much of their time courting females with a conspicuous claw-waving display, an action that probably puts them at greater risk to predation by shorebirds. Fiddler crabs also become infected by microphallid metacercariae[20], which should be under selection to increase risky crab behavior, because these parasites use shorebirds as definitive hosts. As with other parasite manipulations, increasing predation risk could increase parasite transmission. Increased mating success could also partially offset the fitness costs to the crab of increased predation. Some cestodes (*Diphyllobothrium*) may exploit this trade-off indirectly. They use copepods as first intermediate hosts, sticklebacks as second intermediate hosts and birds as final hosts. Copepods are a rich source of the carotenoids that male sticklebacks use to generate their red display[21]. The cestodes increase the susceptibility of copepod prey[22], which leads to redder sticklebacks[21]. In addition to making the stickleback sexier, red coloration may increase the risk that a final host eats a male stickleback[23] and transmits the tapeworm. The prediction that trophically transmitted 'love-potion' parasites might increase risky sexually selected behaviors or morphology should be testable in several systems.

Limb autotomy is a way for parasites to achieve trophic transmission without killing their hosts. The spiny sand crab *Blepharipoda occidentalis*, like most other crabs, has the ability to autotomize a limb to escape. Metacercariae disproportionately infect the crab's claws, suggesting that they might become transmitted during one of these successful escapes from an attack. Claws released by disturbed crabs have three times more metacercariae than do retained claws, suggesting that the parasite can influence autotomy behavior to its advantage (K.D. Lafferty and M. Torchin, unpublished). Parasites in other hosts that autotomize and regenerate parts of their bodies might evolve similar strategies. For example, *Sarcocystis gallotiae* is transmitted among lizards of the Canary Islands by cannibalistic tail predation[24]. Lizards generally autotomize tails as an escape response and it is possible that *Sarcocystis* could evolve to increase the propensity of tail autotomy.

A related scenario might occur in hosts that are colonial. But in this case, infected intermediate hosts might actually benefit from PITT because natural selection acts more on the colony or genet than on the individual. In this sense, predation on an infected individual might be analogous to predation on a body part. Aeby[25,26] studied a trematode metacercaria that infects coral, resulting in pink, swollen polyps. Infected colonies grow slower. Butterflyfishes prefer to eat infected polyps, presumably because they are relatively easy prey. The removal of an infected polyp benefits the coral because the colony is able to replace dead polyps with new ones but cannot replace the infected, living, functionless polyps. This, of course, assumes that the transmission from coral to fish does not, in time, predictably result in higher rates of transmission (fish to snail to coral) back to the same colony. Other colonial organisms, including social insects, serve as second intermediate hosts for parasites. These might provide other seemingly unusual cases, where second intermediate hosts can ironically benefit from a seemingly costly parasite-induced modification.

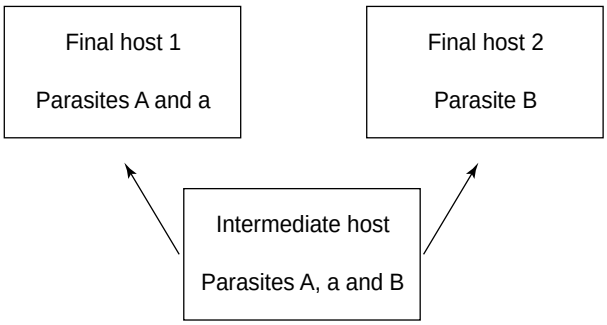
Definitive hosts

In addition to modifying intermediate host behavior, the parasite must enlist the participation of the definitive host, who risks the consequence of becoming sick by eating infected prey. Thus, the evolution of PITT requires either that the parasite should be cryptic or that feeding on parasitized prey becomes less costly than avoiding such prey[5,14]. Why do predators choose to feed on infected prey? It may simply be that predators are unable to distinguish between infected and uninfected prey. Alternatively, enhanced capture of infected prey might outweigh the costs of parasitism[3,14,27]. If so, PITT might increase a predator's energy intake[14]. To determine whether definitive hosts benefit from the parasites they ingest requires weighing the energetic gains of increased foraging success against the energetic costs of parasitism.

Typically, the cost of parasitism within hosts is a function of both the numbers of parasites residing in the host and the cost per parasite. Therefore, mechanisms that regulate infrapopulations of parasites, such as the immune system, would be important in determining the resultant costs of parasitism[14]. Acquired immunity to a parasite would allow the host to exploit a potential resource (infected prey) without accumulating repeated costs. Regulation of parasite infrapopulations could also be the result of intra- or interspecific competition among parasites. Strong intraspecific crowding effects are often seen among adult trophically transmitted parasites such as tapeworms. To understand the maintenance of PITT, it is important to elucidate the mechanisms that might contribute to the regulation of parasite infrapopulations. Although theoretical consideration has been given to the possible mechanisms[14], little empirical evidence exists to test these hypotheses.

Aeby (pers. commun.) has proposed a cost–benefit evaluation of the butterflyfish–coral–trematode system. By preferentially feeding on infected coral, butterflyfish are able to obtain more coral tissue per bite and, thus, are able to enhance their foraging efficiency[25,26]. At the same time, the cost of the parasite may be minimal because of its small size and low intensity in the fish. Preliminary studies have found that butterflyfish can ingest high numbers of metacercariae when feeding on infected coral (up to 340 metacercariae in 30 min) but fewer than 10% of ingested metacercariae are able to establish as juveniles. These observations suggest there may be mechanisms that limit trematode intensities in the fish host.

Some parasite life cycles require more than one trophic transmission event (eg. *Ligula*, *Alaria* and *Diphyllobothrium*), providing the possibility for PITT to evolve more than once within a single parasite's life cycle. However, assuming that: (1) parasites modify intermediate host behavior in ways that strongly reduce host fitness and (2) predators can distinguish between infected and uninfected prey, Kuris postulated[2] that PITT should evolve only once in a complex life cycle. This is because a predator should avoid prey infected with parasites that will, in turn, subject the predator to increased risk of predation. Kuris provides[2] a possible example of this[13,28], and suggests that pseudophyllidean tapeworms provide tractable systems for testing the prediction.



In intermediate	Analogy	Expected adaptation
A and a	Lucky passenger (a)	Hitchhiking (a)
A and A, B and B	Co-pilots	Hitchhiking
B and a	Unlucky passenger (a)	Soloing (a)
B and A	Unlucky pilot, back seat driver	Soloing, hijacking

Fig. 1. A host-as-vehicle analogy to illustrate PITT (parasite increased trophic transmission). Capital letters (A, B) are manipulator parasites and lower case letters (a) are nonmanipulators. Parasites 'A' and 'a' use final host 1, while parasite 'B' uses final host 2. By analogy, within an intermediate host 'vehicle', manipulator parasites are 'pilots' and nonmanipulators are 'passengers' with a specific final host 'destination'. I have stretched this analogy to cover several qualitative interactions that depend on whether the parasites involved: (1) can manipulate the intermediate host, (2) share the same final host, and (3) have evolved adaptations to associate or disassociate selectively with other parasites in the intermediate host. If both parasites have the same final host, they have a shared interest in transmission. When a nonmanipulator shares a host with a pilot, it is a 'lucky passenger' (in that the pilot might act to increase transmission)[29]. If both parasites are pilots, there may be a synergism between the two such that they act as 'co-pilots'. Parasites could evolve 'hitchhiking' if they acquire a strategy to associate with a pilot[29]. The possibility that parasites in the same intermediate host might use different final hosts adds additional 'conflict of interest' scenarios to the host-as-vehicle analogy. A nonmanipulator might be an 'unlucky passenger' if it shares a host with a pilot whose destination is a different final host. If both parasites in conflict are manipulators, the stronger manipulator is an 'unlucky pilot', while the weaker is an annoying 'back seat driver'. We might expect conflict of interest to select for 'soloing' (avoiding pilots) or 'hijacking' (overpowering the pilot). There are some limitations to these analogies. The first is that the final outcome of the interaction might be modified if the parasites compete within the final host. Also, the analogy assumes that PITT is targeted to a specific final host. If PITT is general, the distinction between some of the qualitative outcomes (particularly for conflict of interest) might be blurred. Finally, the predictions stemming from the adaptive analogies (positive associations between parasites with shared interest and negative associations between parasites with conflict of interest) have plausible alternative explanations, so that evidence for adaptation is likely to be speculative.

Co-occurring parasites

In some cases, more than one trophically transmitted parasite may infect the same intermediate host. This might lead to a diversity of PITT strategies. For example, several trematodes use the California killifish as an intermediate host. *Euhaplorchis californiensis* alters killifish behavior while the others, such as *Renicola buchanani*, seem not to[12]. All are probably able to use the same definitive host bird. In multiple infections, *R. buchanani* clearly benefits from increased transmission resulting from the PITT of *E. californiensis*. Intensities of the two species are associated positively with each other, suggesting that *R. buchanani* has found an alternative strategy to PITT. Such a strategy, termed 'hitchhiking'[29], has been investigated in more detail in the trematode *Microphallus subdolum*, which infects amphipods as second intermediate hosts. *Microphallus subdolum* does not alter the amphipod's behavior, yet it is positively associated with *M. papillorobustus*[30], which infects the amphipod's brain, makes it swim closer to the water's surface and increases its susceptibility to predation by birds[31,32]. Thomas *et al*.[29] argue that this positive association is not accidental because *M. subdolum* cercariae (the free-living stage that infects amphipods) actively swim closer to the surface where they seem more likely to penetrate amphipods already modified by *M. papillorobustus*. Other trematodes in this community, however, are randomly associated with *M. papillorobustus* and, therefore, are simply 'lucky passengers' when they find themselves in a modified host[33].

The parasites of sand crabs (*Emerita analoga* and *Blepharipoda occidentalis*) provide an example of even more complex interactions between parasite communities and PITT. Each crab species is parasitized by larval acanthocephalans, trematodes, nematodes and tapeworms. The acanthocephalans and trematodes are transmitted to birds, while the nematodes and tapeworms are transmitted to elasmobranchs. Although the effects of these parasites on host behavior are unknown, there is the potential for hitchhiking to occur in cases where different parasites infect the same host individual. There is also the potential for a conflict between parasites. For example, if a larval nematode and larval acanthocephalan share the same intermediate host, only one can expect to survive transmission. The other is an 'unlucky passenger'. Thus, there may be selection to avoid hosts infected with conflicting parasites. There might also be selection for competitive processes to gain control of PITT in hosts where two conflicting parasites share the same hosts, something that might be described as 'hijacking'. However, a preliminary survey of the parasite communities in sand crabs indicates that instances of hitchhiking and lucky passengers are no more frequent than expected, while hijacking and unlucky passengers are no less so (K.D. Lafferty and M. Torchin, unpublished). The potential for conflict of interest occurs in other systems as well. Bird acanthocephalans and fish acanthocephalans use the same amphipod intermediate host[3,33]. The rat tapeworm *Hymenolepis diminuta* and the chicken tapeworm *Raillietina cesticillus* use the same beetles for intermediate hosts and both alter beetle behavior in seemingly similar ways[34,35]. *Raillietina cesticillus* appears to prevent the establishment of *H. diminuta*[36], suggesting that a potential conflict of interest may have led to a hijacking defense. In any case, it is important that future studies of hitchhiking and hijacking demonstrate both preferential infection and benefits for transmission[37]. Figure 1 illustrates these concepts in a 'host-as-vehicle' analogy.

PITT as an adaptive strategy

I have taken a different approach from other recent reviews of behavior modification by concentrating on PITT as an adaptive strategy and asking how it might evolve under different conditions. If PITT is a consequence of pathology, or if parasites are able to modify

host neurobiology directly, PITT is likely to be strong. Alternatively, PITT should be more moderate if it requires an energy investment, or if intermediate hosts are able to mount a successful defense. Resistance of intermediate hosts to parasites may push PITT in ways that are less costly for intermediate hosts. Possibilities include the compensatory benefits of sexual selection or sacrificing body parts (or clonal units) instead of life. The need to enlist the participation of definitive hosts might require parasites to mask their presence so as not to be avoided. Alternatively, parasites might tip the cost–benefit balance in such a way that it does not pay predators to avoid parasitized prey. A key factor that might alleviate the costs of parasitism for predators is the existence of parasite infrapopulation regulation due to crowding effects or the host immune response[2,12]. This mechanism might allow definitive hosts to benefit from PITT. In cases where many larval parasites exploit the same intermediate host, there may be shared and conflicting interests depending on the strength of PITT and whether the parasites have the same final hosts or not. In conclusion, the evolution of PITT is subject to several conditions that might alter its expression. Much work remains to be done to investigate the growing number of fascinating hypotheses for how parasites have evolved to facilitate their own transmission.

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Proposed Unified Genetic Nomenclature for *Trypanosoma* and *Leishmania*

The increasing availability of kinetoplastid gene sequences and mutants, along with the wide use of genetic manipulation to create progressively more complex strains, has made the development of a unified genetic nomenclature imperative. C. Clayton *et al*., in *Molecular and Biochemical Parasitology* 97, 221–224 (1998), have proposed a nomenclature system. This follows discussion at a workshop at the Woods Hole Molecular Parasitology Meeting in September 1996, and again at a WHO-sponsored workshop for the *Trypanosoma brucei* and *Leishmania* Genome Projects (Arcachon, France) in April 1998.