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Commentary

Trophic transmission of parasites and host behavior modification

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Thomas et al. (2005) provide a wide-ranging and insightful review of issues surrounding the evolution and ecology of parasite manipulation of host behavior. To support their goal seeking future directions I offer three comments.

Firstly, several very different types of interactions are combined under the vague label "parasite". This obscures important differences with respect to host behavior modification. These host–parasite interactions are pathogens, parasitoids, parasitic castrators and trophically transmitted parasites (TTPs). All these infectious agents may modify host behavior, but under very different selective environments (Kuris, 1997). Applying a study of one of these relationships to another without an evaluation of its applicability can often be misleading or irrelevant. For example, behavior modification by parasitoids such as nematomorphs, and parasitic castrators such as trematode parthenitae (Thomas et al., 2002; Curtis, 1990) cannot be confused with host defensive behaviors. As O'Brien has succinctly put it, a parasitic castrator has a parasite genotype, but a host phenotype (O'Brien and Van Wyk, 1985). The parasitically castrated "host" is not being manipulated; in an evolutionary sense it is no longer present. Investigations of these relationships have scant bearing on the issue of adaptation since there is no countervailing host interest. However, they are of great interest as to mechanisms of host control with respect to the time and place of death (parasitoids), and longevity and risk aversive behavior (parasitic castrators).

Pathogens may well modify host behavior, often to disseminate dispersal stages. For these interactions, issues of non-adaptive pathology and host defensive responses are highly relevant. Further, these behavior modifications are usually not complex, so sophisticated molecular mechanisms seem unlikely. Hence, information from systems such as rabies must be critically examined before being applied to, say, TTPs.

Host behavior modifications by TTPs often meet the criteria for adaptation set out by Poulin (1995). They are often complex and usually seem "designed" to deliver prey to an appropriate predator (Kuris, 1997, 2003). Independent origin is a less relevant criterion (as long as selection can maintain the trait), and the likelihood that parasite fitness is enhanced is evident. The ability of some TTPs to mask the behavior modification from the predator is an important variant on the TTP strategy since it enables a relatively virulent parasite to enhance the probability of transmission (Kuris, 2003). Behaviour modification of ants by the lancet fluke, *Dicrocoelium dendriticum*, is a good example of masked behavior presumably enhancing access to grazing sheep.

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Secondly, the demand that the adaptive nature of behavior modification by TTPs be rigorously tested has developed an ecclesiastical tenor (forgive a Church metaphor in an evolutionary argument). Whether the modification arises early in a phylogeny, or has other pathological consequences, does not diminish the selective force exerted by the requirement that a TTP's prey host be consumed by an appropriate predator host. It is not surprising that the range of behaviors reported includes increased exposure, reduced alertness, reduced speed, reduced stamina and "odd" behaviors that potentially facilitate recognition and capture by the predator. These are often accompanied by changes in colour and sometimes by structural deformities. The dramatic findings concerning the trematode, *Ribeiroia ondatrae*, in frogs (Johnson and Sutherland, 2003) provide perhaps the most obvious link between a TTP, its induction of limb deformities, and increased susceptibility to avian predators.

Models of the three species interaction between a TTP, its prey and its predator hosts suggest that under a wide set of conditions the predator will benefit via easier food acquisition (Freedman, 1990; Lafferty, 1992), particularly if their pathology is limited in the predator. This condition is widely met (except for masked TTPs) (Kuris, 2003). Hence, for TTPs, a prima facie case for adaptation appears justified and more interesting issues merit attention.

Thirdly, future research emphasizing the physiological mechanisms of altered behaviors, and the role of these interactions in ecology, will provide the greatest gain to knowledge of behavior modification by TTPs. Consider one of the best documented cases, the metacercariae of *Euhaplorchis californiensis* encysted in the brain case of the killifish, *Fundulus parvipinnis*. In a field test of the susceptibility of parasitized versus unparasitized, killifish to avian predators, Lafferty and Morris (1996) showed that infected fish were 30X more likely to be eaten than were unparasitized fish. This effect was also intensity-dependent (as was the extent of behavior modification). Importantly, these parasitized fish were otherwise normal and healthy. Their weight-to-length and gonosomatic ratios were not significantly different from those of uninfected control fish. This implies that the manipulation by the parasite is physiologically subtle and sophisticated. Knowledge of this mechanism has the potential to reveal how complex behaviors are precisely controlled, and could ultimately lead to important pharmacological discoveries. In a similar vein, analysis of the teratogenic induction of limb deformities by *R. ondatrae* in frogs may also contribute to our understanding of limb morphogenesis.

The Lafferty and Morris (1996) study also points to major gaps in the ecological evaluation of host behavior modification by TTPs. The killifish is the most abundant fish in the estuaries and salt marshes of southern California and Baja California. The trematode, *E. californiensis*, is also ubiquitous in those habitats. The question arises as to whether the diverse and abundant assemblage of piscivorous birds on these coasts could be sustained in the absence of this enhanced food delivery system? The population level consequences of behavior modification need investigation to determine whether such effects are curiosities or can structure ecosystems.

To evaluate the role of TTPs in host–parasite dynamics, it will also be of considerable value to examine host specificity, site specificity, crowding effects, intensity-dependent pathology and the aggregation of TTPs among both prey and predator hosts. If the physiological intervention by the TTP is precise, then it is unlikely to be effective in all host species. Some studies suggest that host specificity of trematodes to second intermediate (prey) hosts is surprisingly host-specific (Reversat et al., 1989). In prey hosts, TTPs are often relatively small (e.g., metacercariae), hence a precise location should promote effective delivery of the modifying message. Yet, for some species site specificity is low (Martin, 1950; Torchin et al., in press). Crowding effects are widespread among macroparasites, parasitoids and parasitic castrators. In contrast, competition among behavior-altering TTPs should be negligible (Kuris, 2003) (an obvious exception, larval tapeworms such as *Schistocephalus* (Heins et al., 2002), are also parasitic castrators). This remains to be systematically investigated. As a corollary, intensity-dependent pathology should promote the success of the entire infrapopulation of TTPs in the prey host. This also merits examination. Finally, aggregation of TTPs in prey hosts would seem to facilitate their transmission (unlike aggregation in macroparasites models which selectively deletes large infrapopulations of parasites from a host population). Yet, intensity-dependent host behavior modification potentially truncates the aggregated distributions in prey hosts; making analysis of this gain to aggregated infrapopulations challenging.

How factors increase aggregation upon transmission to prey and then reduce it upon transmission to predator will inform models of these complex systems.

Host behavior modification is widespread and likely generally adaptive. Study of its physiological basis and of its ecology will illuminate our understanding of the role of parasites in ecosystem, models of host–parasite dynamics and perhaps contribute to our pharmacological tool kit for neurological and developmental maladies.

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