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Parasite manipulation: stretching the concepts

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The parasitological literature is rife with examples of the phenotypic changes that hosts incur as a result of parasitic infections. Some of these changes affect host behaviour in dramatic and bizarre ways that have caught the attention of text book authors and are beloved of both teachers and students of introductory courses in parasitology. Many of these examples are cited by Thomas et al. (2005), who succinctly air the current concerns regarding the interpretation of these observations as examples of parasite manipulation.

The debate concerning the adaptive nature of host manipulation, the origin of drive for trait changes, be it parasite or host, and the ancestral history of manipulation is, I agree, difficult to resolve from an experimental standpoint. The design and interpretation of investigations is hampered by our inadequate understanding of the physiological basis of host behaviour even when uninfected. It is thus a pity that the theoretical analysis of host manipulation has focussed to such an extent upon parasite-induced changes in behaviour. Morphological and/or physiological alteration of hosts is so common place as to be almost ubiquitous and we often see a suite of changes associated with the presence of a parasite as, for example, the development of the nurse cell in the cyst of *Trichinella spiralis* (Despommier, 1993) and the changes in an erythrocyte when infected with the malaria parasite (Paul and Brey, 2003). It may thus be more illuminating to examine parasitic manipulation in the context of physiological changes to the host. I believe that the criteria originally devised by Poulin (1995) can equally be used in the latter context. Furthermore, given the present state of knowledge, it is likely to be much easier to determine the mechanism underlying changes in physiology than behaviour. As with other aspects of the study of host-parasite interactions, an integration of studies concerning the molecular, biochemical and physiological aspects of infection with the evolutionary, ecological and behavioural would be beneficial.

I welcome the author's recognition that host changes that seemingly result from infection by a manipulative parasite may actually be host responses to infection. Parasitologists tend to focus upon the combative nature of the host/parasite relationship that is enshrined in such concepts as the red queen hypothesis. However, in some circumstances selection may favour parasite and host strategies that change the host in a synergistic manner (Hurd, 1998, 2001). We have shown this to occur in the *Hymenolepis diminuta/Tenebrio molitor* model where infection with a metacystode induces fecundity reduction in the beetle intermediate host. Inhibition of the synthesis of the yolk protein, vitellogenin, is caused directly by a molecule secreted by the tapeworm but uptake of vitellogenin by developing oocytes is inhibited by a molecule of host origin (Cole et al., 2002). Both of these changes result in a reduction in the resources devoted to egg production. Host life span is significantly increased when infected, possibly as a result of repartitioning of resources away from reproduction. The parasite therefore gains an extended period for potential transmission and the host may be capable of replacing lost investment in reproduction by extending the reproductive period (Hurd et al.,

2001). In circumstances such as this, where the parasite can successfully evade the host defence system, tolerance on the part of the host is witnessed and strategies may evolve that make the best of the situation.

Finally, I firmly endorse the views of the authors that there is still much to be gained from examining host parasite interactions from the standpoint of manipulation, whether it be by the parasite, the host or both partners.

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