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Infection syndrome and multidimensionality: two terms for two different issues

Frédéric Thomas, Robert Poulin and Jacques Brodeur

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Multidimensionality is an emerging topic in the study of host manipulation by parasites (Thomas et al. 2010), and it is therefore timely and important to lay down definitions. We fully sympathize with the idea proposed by Cézilly and Perrot-Minnot (2010) that some parasites, through their manipulative activities, are likely to induce infection syndromes in their hosts. However, we believe that it is better to restrict the label of multidimensional manipulation only to traits that have a significant, direct or indirect (Thomas et al. 2010), value for the transmission or survival of the parasite.

Virtually all parasites (thus, including manipulative ones) induce more or less conspicuous infection syndromes in their host, because host exploitation is likely to interfere concomitantly with several functions, and generates infection syndromes through cascading effects and/or correlations between traits. An important question in our opinion is not to determine if manipulative parasites induce infection syndromes, but to determine whether or not we should include assemblages of collateral effects with no function for parasite transmission under the umbrella of multidimensional manipulation. The majority of the scientific community now recognizes that when studying host manipulation by parasites, it is crucial to distinguish between traits that truly favour transmission from those that are by-products without adaptive value (Poulin 1995, Moore 2002); the ‘manipulation’ label being traditionally exclusively restricted to the first category of traits. Traits with no function for parasite transmission or survival within an infection syndrome are in fact by-products, generated by physiological correlations between traits and/or by other processes. They exist, we cannot ignore them, we should even do our best to document their nature and incidence and to understand how they arise during the infection process, but they are not involved in the host manipulation process.

Cézilly and Perrot-Minnot (2010) also suggest that restricting multidimensionality to alterations that appear to increase parasite transmission is often problematic in practice, because demonstrating that a particular phenotypic alteration is *directly* responsible for increased parasite transmission remains difficult. We agree that such a demonstration is not simple, but we disagree with the logic according to which technical difficulties should decide whether a research direction is legitimate or not. Instead, we believe that the true challenge for scientists facing such complex questions is to develop sophisticated tools appropriate for the task at hand. For instance, gene silencing should allow in the near future the separate exploration of the role of different host phenotypic alterations induced by parasites on their own transmission success.

Even if we do not share all opinions expressed by Cézilly and Perrot-Minnot (2010), we think that their contribution is a very stimulating one, useful to the debate. It reflects the diversity of views and the high enthusiasm of researchers for the fascinating world of manipulative parasites.

References

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