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Abstract

Parasites are known to manipulate the behavior of their hosts in ways that increase their probability of transmission. Theoretically, different evolutionary routes can lead to host manipulation, but much research has concentrated on the 'manipulation hypothesis' sensu stricto. Among the arsenal of host compensatory responses, however, some seem to be compatible with the parasite objectives. Another way for parasites to achieve transmission, therefore, would be to trigger specific host compensatory responses. In order to explore the conditions favoring this manipulative strategy, we developed a simulation model in which parasites may affect their hosts' behavior by using two nonmutually exclusive strategies: a manipulation sensu stricto strategy and a strategy based on the exploitation of host compensatory responses. Our model predicts that the exploitation of host compensatory responses can be evolutionary stable when the alteration improves the susceptibility to predation by final hosts without compromising host survival during parasite development. Inversely, when the behavioral modification resulting from a compensatory response conflicts with the host's interest we expect parasites to use both strategies. From this result, we conclude that the strategy based on the exploitation of host compensatory responses should be more common among nontrophically transmitted parasites. Furthermore, our findings indicate that the transmission rate of parasites in a definitive host is highest when each of the two strategies affects different traits, which supports the hypothesis that host manipulation is a multidimensional phenomenon in which each altered trait contributes independently to increase parasite transmission efficiency.

Introduction

Many parasitic organisms (e.g., virus, helminths, parasitoids) have evolved the capacity to alter phenotypic traits of their hosts, extending from color, morphology, and behavior, in order to either increase their probability of transmission and/or survival in a given host or insure that their propagules will be released in an appropriate habitat (see reviews by Moore 2002; Thomas et al. 2005; Poulin 2010; Hughes et al. 2012). For example, tropical ants parasitized with nematodes go perch and develop bright red abdomens (filled with nematode eggs) that resemble ripe fruits in the tropical rain forest canopy (Yanoviak et al. 2008). This drastic alteration of the ants' appearance increases their predation by frugivorous birds, which then pass the parasite eggs in their feces. The widespread protozoan parasite *Toxoplasma gondii*, which must be transmitted from a rodent (intermediate host) to a felid (definitive host), reverses the innate aversion of the rodent to cat odor into attraction, thereby increasing the probability of intermediate host predation (Berdoy et al. 2000). Some hairworm species parasitizing Orthoptera alter the behavior of their hosts in a way that forces them to jump into water, where the worms can emerge from the host and search for a sexual partner (Thomas et al. 2002a). Several parasitoids can even usurp host behavior after leaving it (Brodeur and Vet 1994), converting the latter into a bodyguard protecting developing pupae from approaching predators and hyperparasitoids (Grossman et al. 2008; Harvey et al. 2008; Maure et al. 2011).

Determining why and how host manipulation by parasites evolves is a fascinating but challenging question for

evolutionary biologists (Lefèvre et al. 2009; Thomas et al. 2012). The extended phenotype perspective (Dawkins 1982) postulates that host behavioral alteration should be regarded as the expression of the parasite's genes in the host phenotype. Natural selection is indeed expected to favor the ability of parasites to induce behavioral, morphological, or physiological alterations in their hosts that are beneficial for themselves, even though they are detrimental to the host's fitness. This scenario, also called manipulation sensu stricto, is a decidedly parasite-oriented view, and is traditionally considered as the main process used by parasites to manipulate their host's behavior. Recent studies, however, acknowledged that different evolutionary routes can lead to host manipulation, notably processes involving compromises between host and parasite strategies rather than a complete parasite takeover (Thomas et al. 2012). For instance, Lefèvre et al. (2008) proposed that parasites could theoretically achieve transmission by triggering host compensatory responses when these responses match, at least partially, with the transmission route. In this view, genes of the parasite are selected for their pathological effects that induce a host compensatory response. As behavioral changes both mitigate the costs of infection for the host and meet the objectives of the parasite in terms of transmission, natural selection is likely to favor the evolution of such interaction (Lefèvre et al. 2009). In accordance with this hypothesis, the sexually transmitted ectoparasite mite *Chrysomelobia labidomera* reduces the survival of its leaf beetle host (*Labidomera clivicollis*), and in response infected males exhibit increased sexual behavior before dying (Abbot and Dill 2001). This compensatory response from the host clearly benefits the sexually transmitted parasite as enhanced inter- and intrasexual contacts (i.e., copulation and competition) provide more opportunities for transmission (Drummond et al. 1989; Abbot and Dill 2001). Although a few other examples support the idea that parasites could indeed exploit host compensatory responses instead of manipulating sensu stricto their host (see Lefèvre et al. 2009), it remains unclear if this manipulative strategy is widespread or not. Issues of manipulation sensu stricto versus interactive scenarios have much to gain from a theoretical approach.

To address the issue, we developed a simulation model that predicts the conditions under which parasites should benefit from using a strategy based on the exploitation of compensatory responses, either alone or in concomitance with a manipulation sensu stricto strategy. Because predation of the intermediate host by the definitive host is necessary for parasite transmission, parasites that manipulate the behavior of their host may benefit from increasing either the vulnerability of hosts to predation or their susceptibility to predation by suitable predators. In order to do that, parasites can use two nonmutually exclusive strategies: they can induce a compensatory response that reduces the negative effects of infection on host fecundity (thus providing a benefit to the host) but in turn renders their transmission more probable (thus providing a benefit to the parasite as well) and/or they can exert a certain manipulative effort to alter the behavior and/or appearance of infected hosts, making them more susceptible to predation by definitive hosts. More precisely, parasites that trigger host compensatory responses can, for instance, affect the energy requirement for reproduction, thereby causing hosts to increase their foraging activity so that they can acquire enough resources to reproduce, but also inevitably their vulnerability to predation. Conversely, parasites that use a manipulative strategy sensu stricto can modify the response of their hosts to predation risks by definitive hosts, for example, by turning their innate aversion into an imprudent attraction (Berdoy et al. 2000). Among other parameters, we expect the evolutionary stable strategy to depend critically on the probability that hosts possess in their repertoire a compensatory response that matches the transmission objectives of the parasites as well as on the benefits for the hosts of opposing manipulation or compensating. Therefore, because parasitized hosts may show different levels of tolerance/resistance to parasite-induced behavioral changes (Thomas et al. 2011), we also considered in our model two types of hosts that differ in their ability to suppress the manipulative efforts of the parasite (i.e., manipulatable and unmanipulatable hosts).

The model

To analyze the dynamics of hosts and parasites over time, we run simulations over consecutive generations until they reach equilibrium states. For a given simulation, all parameters remained fixed over time, except the relative proportion of unmanipulatable and manipulatable hosts (denoted x_t and $(1 - x_t)$, respectively) as well as the prevalence of hosts (i.e., the probability that a parasite finds a host) and parasites (i.e., the probability that a host becomes infected by a parasite) that were estimated at each time t from parameters p_t and q_t, respectively. For the first generation (i.e., at $t = 0$), we used the following default starting values: $x_0 = 0.5$, $p_0 = 0.5$, and $q_0 = 0.8$.

To find the optimal parasite strategy at each time t, we assume a two-step decision process: first both the parasites and their host decide, respectively, whether or not to induce a compensatory response and, if relevant, whether or not to compensate, and second the parasites decide how much effort they invest in manipulation to maximize their expected fitness.

Should parasites exploit host compensatory responses?

The passive expected fitness of a parasite that does not use any strategy to increase its transmission to a definitive host is denoted by $W\alpha\tau$. It depends on (1) W: the basic reproductive success of parasites; (2) τ: the predation rate of hosts whose behavior is not altered (which is assumed to be equal to that of uninfected hosts); and (3) α: the proportion of predatory events that are attributable to a suitable predator (i.e., a predator in which a parasite can complete its life cycle).

The induction of a compensatory response by a parasite not only reduces its fitness by C_{CR} (the cost of inducing a compensatory response) but it may also alter the behavior of its host in a way that increases either its rate of predation (by a factor τ_{CR}) or its susceptibility to predation by suitable predators (by a factor α_{CR}), provided that the host decides to compensate.

We assume that parasites decide to induce a compensatory response only if their expected fitness when they do so and the hosts compensate (i.e., $[W\alpha(\tau + \tau_{CR}) - C_{CR}]$ or$[W(\alpha + \alpha_{CR})\tau - C_{CR}]$ if the parasite-induced change in host behavior increases the rate of predation or the susceptibility to predation by suitable predators, respectively) is larger than their passive fitness. Therefore, we predict that parasites should induce a compensatory response only when the benefit of compensation (τ_{CR} or α_{CR}) is larger than: $C_{CR}/W\alpha$ or than: $C_{CR}/W\tau$, if the change in host behavior concerns the rate of predation or the susceptibility to predation by suitable predators, respectively. From these conditions, we can conclude that parasites should induce a compensatory response more frequently when the cost of doing so (C_{CR}) is relatively small and in parasite species in which individuals produce a large number of offspring (W). Furthermore, when hosts possess in their repertoire a compensatory response that increases their predation risk (τ_{CR}), parasites would benefit from exploiting this response only if there is a high probability that hosts are consumed by a suitable predator (i.e., high α). Conversely, when the available compensatory response affects the susceptibility to predation by suitable predators (α_{CR}), the likelihood that parasites induce a compensatory response should increase with the risk of predation incurred by hosts (τ).

Should hosts compensate?

If parasites induce a compensatory response, then hosts can also decide to compensate or not. We assume that uninfected hosts produce on average w offspring. Thus, as their mortality rate due to predation is τ, their mean breeding success equals $(1 - \tau)w$.

When hosts are infected by a parasite, the fecundity of female hosts is reduced, and their expected breeding success then becomes $\sigma(1 - \tau)w$. This detrimental consequence of infection, however, can be reduced through compensatory responses. In that case, female hosts suffer less fecundity reduction than those that do not compensate (σ_{CR} and σ respectively, with $\sigma_{CR}>\sigma$), but in turn they become more vulnerable to predation or more susceptible to suitable predators. The fitness of hosts that do compensate therefore is: $\sigma_{CR}(1 - \tau - \tau_{CR})w$ if the parasite-induced change in host behavior affects the rate of predation, or $\sigma_{CR}(1 - \tau)w$ if the parasite-induced change in host behavior affects the susceptibility to predation by suitable predators. Note that in that latter case, the mortality rate of infected hosts is not reduced and hosts, therefore, only receive benefits from compensating. As above, we assume that hosts compensate only if the benefits of compensation in terms of increased fecundity outweigh the costs. As host females that compensate have a higher fecundity compared to those that do not (i.e., $\sigma_{CR} > \sigma$), we predict that hosts should always compensate when the parasite-induced change increases the susceptibility to predation by suitable hosts (τ_{CR}). Indeed, in that case the induced compensatory response only increases the proportion of suitable versus unsuitable predators that do consume the hosts, but not the overall rate of predation. Conversely, when the change in host behavior increases the rate of predation, one would expect hosts to compensate only when the benefit of compensation in terms of increased predation risk (τ_{CR}) is smaller than: $\frac{[\sigma_{CR}-\sigma]\times(1-\tau)}{\sigma_{CR}}$. From this condition, we can conclude that hosts should compensate with a higher probability when (1) the reduction in fecundity in infected host females is important (i.e., small values of σ); (2) the benefits of compensating are large (i.e., high values of σ_{CR}); and (3) the predation rate of uninfected hosts (τ) is small.

How much effort should parasites invest in manipulation?

The last step of the decision process consists in determining how much energy parasites invest in manipulation, in order to maximize their expected fitness. Although we allow parasites to adjust their manipulative effort to conditions, we are not interested in exploring how it should vary in relation to various factors (see Poulin 1994a). Instead the aim of our model is to predict the conditions that should favor the use of a strategy based on the exploitation of compensatory responses either alone or in concomitance with a manipulation sensu stricto strategy. This is the reason why the benefit and the cost of manipulation, that are both functions of the amount of manipulative effort invested (ME), were kept constant. More specifically, we fixed the cost of manipulation C_{ME} to ME^2 and, as for

the strategy based on the exploitation of compensatory responses, we assumed that the parasite-induced change in host behavior resulting from manipulation may affect either the rate of predation (with $\tau_{ME} = 0.2ME$) or the susceptibility to predation by suitable predators (with $\alpha_{ME} = 0.2ME$). In the case of an unmanipulatable host the benefits of manipulation are also proportional to its efficiency in suppressing the parasite efforts (ε) and are then equal to $(1 - \varepsilon)\tau_{ME}$ or $(1-\varepsilon)\alpha_{ME}$ depending on whether the manipulation increases the rate of predation or the susceptibility to predation by suitable predators. To determine the optimal manipulative effort invested, we estimated the average fitness expected by parasites for each value of ME, and then we retained the value ME* for which the fitness is maximal (see Fig. 1 for an example of the gain expected by the parasite for each of the four possible cases when the host is manipulatable). Once we have retained the value of ME*, we can estimate the fitness of parasites as well as that expected by unmanipulatable and manipulatable hosts for the generation t. Figure 2 gives an example of the gain expected by a manipulatable host in each of the four possible cases. In the case of an unmanipulatable host, their expected fitness is reduced by C_R (the cost of host resistance incurred by unmanipulatable hosts to suppress parasite manipulative efforts). The gains expected by hosts and parasites in each possible situation are given in Tables S1 and S2.

Analysis

The expected fitness of parasites and hosts at each time (generation) t depend on the proportion of hosts infected by a parasite (q_t), on the probability that a parasite finds a host (p_t), as well as on the relative proportion of unmanipulatable hosts (x_t). For the first generation (i.e., at $t = 0$), we use the initial starting values of x_0, p_0, and q_0 to determine the success of each strategy. Then for the subsequent generations, the values of these three parameters have to be estimated at the beginning of each generation before we can evaluate the average success expected by hosts and parasites. For sake of simplicity, we assume that p_t and q_t are determined solely by the population sizes of hosts and parasites (and not, for instance, by the density of individuals), and that population sizes are limited to K_P and K_H individuals, for parasites and hosts, respectively (with $K_P = K_H = 500$). Also, for convenience, we hypothesize that all individuals die at the end of each generation t and are replaced by their offspring at generation $(t + 1)$.

In order to evaluate the prevalence of hosts and parasites, we consider that each host can be infected by only one parasite and that parasites detect potential hosts (i.e., uninfected hosts) with a probability λ that represents their searching efficiency.

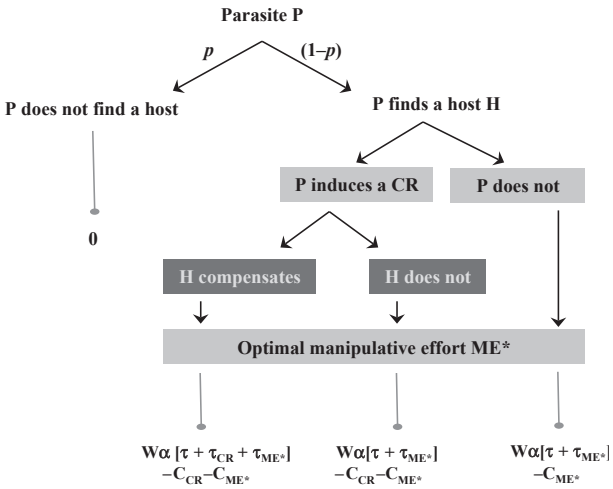

Figure 1. Possible expected gains of a parasite. The equations correspond to the case where the host is manipulatable and where both strategies increase the predation rate (i.e., $\tau_{CR} > 0$, $\alpha_{CR} = 0$, $\tau_{ME} > 0$, and $\alpha_{ME} = 0$).

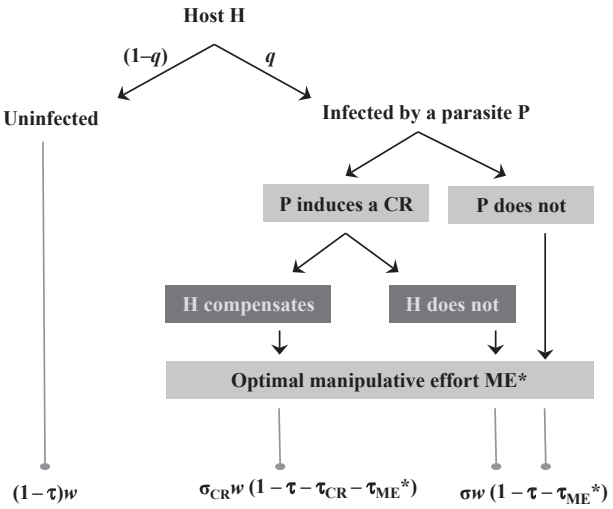

Figure 2. Possible expected gains of a manipulatable host. The equations correspond to the case where both strategies increase the predation rate (i.e., $\tau_{CR} > 0$, $\alpha_{CR} = 0$, $\tau_{ME} > 0$, and $\alpha_{ME} = 0$).

Finally, the proportions of unmanipulatable and manipulatable hosts at generation $(t + 1)$ are proportional to their relative success at generation t.

Results and Discussion

The benefits and costs of compensation for parasites and their hosts

Our model predicts that the probability that parasites induce a compensatory response should increase with the

benefits of compensation (τ_{CR} or α_{CR}). Logically, we would then expect this strategy to be more frequently used by parasites whose hosts possess in their phenotypic repertoire a compensatory response that matches the parasites' transmission objectives by causing either an increased generalized susceptibility to predation or a more targeted susceptibility to predation by final hosts.

Whether the compensatory response affects the predation rate of the hosts or their susceptibility to specific predators, however, directly influences the hosts' decision to compensate or not, thereby determining the benefits that parasites can obtain from this strategy. More precisely, when parasites induce a compensatory response that makes the hosts more vulnerable to predation, they should cease to compensate when the increased rate in predation due to compensation exceeds a threshold value, because the cost of compensating then becomes larger than the expected benefit in terms of increased fecundity. In that case, the occurrence of compensatory responses, and hence the benefits obtained from this strategy, are then highest for intermediate values of τ_{CR} (Figs 3A, 4A, 4B). Conversely, when the compensatory response affects the proportion of predatory events that are attributable to a suitable predator (α_{CR}) but without reducing host survival (i.e., without affecting the rate of predation), hosts should always benefit from compensating, regardless of the benefits gained by the parasites (Figs. 3B, 4C, 4D). Such a situation may arise when the induction of a compensatory response changes the host feeding preferences (see Moore 2002) and causes them to move in a different habitat in which the predation pressure is the same but the proportion of predators that are suitable definitive hosts is higher.

This prediction suggests that the exploitation of compensatory responses should be more common in nontrophically transmitted parasites (such as parasitoids, contact-transmitted parasites, or vector-borne parasites) whose objectives do not conflict with the interests of their hosts. Conversely, in trophically transmitted parasites, we predict that natural selection should favor parasites that induce only moderate (and hence a priori less efficient) compensatory responses, because otherwise infected hosts would never benefit from compensating. Consistent with this expectation, Poulin (1994b) demonstrated through a meta-analysis that most parasites, and especially those that rely on host predation for transmission, induce only small changes in the behavior of their hosts, which could reflect a compromise between host and parasite needs.

Evolutionary stable strategy and parasites' transmission success

Results from the model indicate that both strategies can be evolutionary stable (Fig. 3), and so there are conditions under which parasites should only rely on host compensatory responses to favor their transmission. This is likely to occur when parasites can obtain large benefits from the exploitation of host compensatory responses and, consequently, when parasites alter the behavior of their hosts in a way that increases the probability of transmission to a definitive host, though without compromising the host survival during parasite development.

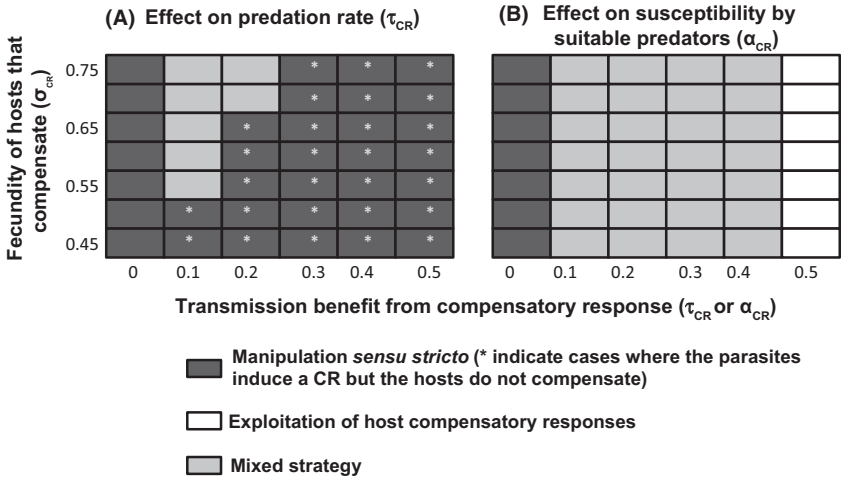

Figure 3. Expected evolutionary stable strategy in relation to the benefits of compensating in terms of (i) fecundity (*y* axis) and (ii) transmission rate (*x* axis). Compensatory responses affect either the rate of host predation (panel A) or the susceptibility to predation by suitable predators (panel B). We assume that both strategies affect the same trait (i.e., both strategies tend to increase either the rate of predation or the susceptibility to predation by suitable predators). In this figure: W = 20, w = 20, λ = 0.8, ε = 0.5, τ = 0.5, σ = 0.4, α = 0.5, C_{CR} = 0.2, C_R = 0.5.

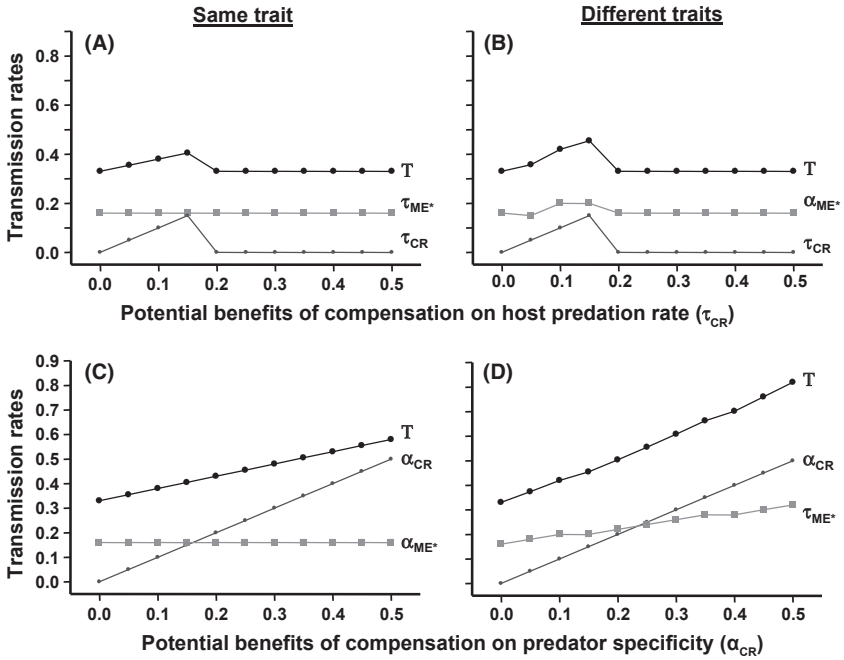

Figure 4. Expected parasite transmission rates in relation to the potential transmission benefits from compensation (τ_{CR} or α_{CR}) and depending on whether the manipulation sensu stricto strategy affects either the same trait (panels A and C) or a different trait (panels B and D) than the one involved in the compensatory response. The dark points correspond to the estimated parasite transmission in a definitive host (T). In this figure: W = 20, w = 20, λ = 0.8, ε = 0.5, τ = 0.5, σ = 0.4, σ_{CR} = 0.6, α = 0.5, C_{CR} = 0.2, and C_R = 0.5.

Conversely, when parasites can only obtain small benefits from host compensatory responses, because either the change in host behavior is small or hosts do not benefit from compensating, they should use a mixed strategy.

Which strategy is evolutionary stable profoundly affects the transmission success of parasites. Indeed, our results indicate that the parasites achieve greater transmission success when they can receive benefits from the two strategies rather than from only one strategy (Fig. 4). This is because the effect of parasite manipulation on host behavior is generally limited both because parasites relying on a manipulative strategy must exert a certain effort that reduces their fitness and hosts may develop resistance against manipulation. For that reason, the amount of effort invested in manipulation and/or the transmission benefits gained from this strategy is generally independent of whether the parasite uses the manipulation strategy alone or in concomitance with the strategy based on the exploitation of compensatory responses (Fig. 4), in which case parasites can then achieve greater transmission success. This finding supports the view that parasites most often alter multiple dimensions in their hosts, including behavioral, physiological, and morphological traits that contribute to increase parasite transmission (see Moore 2002; Thomas et al. 2010). This is the case, for example, in the amphipod *Gammarus insensibilis*: individuals parasitized by the trematode *Microphallus papillorobustus* display three behavioral changes (i.e., negative geotaxis, positive phototaxis, and aberrant escape behavior), and each of them contributes to increase the risk of predation by the definitive host (Helluy 1984).

Synergistic or independent mechanisms of manipulation?

Although there is increasing evidence for multidimensional manipulations, the questions of why and how they have evolved still remain unanswered (Cézilly and Perrot-Minnot 2005, 2010; Thomas et al. 2010). One hypothesis states that several traits would have evolved as secondary manipulations, because they improve the efficiency of the original one. Alternatively, each dimension could have evolved independently from each other. Our results tend to support this latter hypothesis. Indeed, results from our model indicate that parasites can benefit from inducing a compensatory response only when the trait which is not affected by the compensatory response is sufficiently high so that the benefits in terms of transmission outweigh the costs. Thus, even if hosts possess in their phenotypic repertoire a compensatory response that makes them more susceptible to predation by suitable predators, thereby serving the interests of the parasites, we predict that parasites should not

systematically exploit this response but do so only when the frequency of predation events is relatively high or alternatively when parasites can increase the risk of predation through manipulating their host behavior.

Under most conditions, one would then expect parasites to achieve greater transmission success when they use a mixed strategy rather than only one strategy and when the manipulation sensu stricto strategy affects another trait than the one involved in the compensatory response. Accordingly, we found that the expected percent of parasites that are transmitted to a definitive host is maximal when hosts that compensate become more susceptible to predation by suitable predators (Fig. 4). This is because the probability that hosts benefit from compensating, and hence the expected parasite benefits from compensation, are highest when the compensatory response matches the objectives of both the parasites and their hosts (i.e., when the compensatory response increases the susceptibility to predation by suitable predators and hence may provide the hosts only with benefits without compromising their survival). Furthermore the success of the parasites is further improved when the manipulation strategy affects another trait that makes parasites more vulnerable to predators. Thus, our finding suggests that selection should favor independent mechanisms of manipulation, rather than acting in a synergistic fashion, each dimension having its own efficiency in enhancing transmission. In accordance with this conclusion, Benesh et al. (2008) reported that isopods infected by the acanthocephalan *Acanthocephalus lucii* hide less and have darker abdominal coloration compared with uninfected isopods, but these two modified traits are uncorrelated. Multidimensional manipulation based on independent mechanisms could be particularly advantageous for parasites' transmission, as it could allow them to adjust the amount of effort invested in each mechanism depending on the characteristics of the environment (e.g., the predation pressure) or the quality or age of the female host (Thomas et al. 2002b, 2010).

Until the proximate mechanisms responsible for host manipulation remain unclear, it will be difficult to differentiate between manipulation sensu stricto and the exploitation of host compensatory responses in empirical systems. Yet, despite the fact that clear demonstrations are lacking at the moment, there are systems for which it could be a priori beneficial for parasites to use both strategies and to adjust the effort allocated in each of them depending on some hosts' characteristics. For instance, *G. insensibilis* males parasitized by *M. papillorobustus* suffer a reduced pairing success when in competition for females with uninfected males (Thomas et al. 1996). For this reason, infected males would probably enjoy a reproductive advantage from moving toward the surface, where predation pressure is high but uninfected males are absent: despite the fact that most females found near the surface are parasitized and hence have a reduced fecundity, it is certainly better for less competitive males to reproduce with those females rather than risking not reproducing at all. Said differently, we would then expect *M. papillorobustus* to manipulate females more strongly than males to move toward the surface because for the latter, a compensatory response mimicks the effects of the manipulation sensu stricto.

Conclusion

Our model predicts that exploitation of host compensatory responses should be a widespread strategy, particularly in nontrophically transmitted parasites, and suggests that both strategies (manipulation sensu stricto and exploitation of compensatory responses) would have evolved independently. Further experimental work, therefore, is needed to test our model's predictions and improve our understanding of multidimensional manipulation. Furthermore, hosts in nature are usually parasitized by a diverse array of parasites with shared or conflicting interests (depending on whether they require the same or different definitive hosts), rather than by a single parasite as hypothesized in the present study. As a consequence, additional theoretical work will be necessary too, in order to explore how inter- (e.g., Lafferty et al. 2000; Thomas et al. 2002c; Haine et al. 2005) and intraspecific (e.g., Dianne et al. 2010) conflicts between parasites sharing a common intermediate host could influence our results and affect the use of both manipulative strategies.

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Conflict of Interest

None declared.

References

Abbot, P., and L. M. Dill. 2001. Sexually transmitted parasites and sexual selection in the milkweed leaf beetle, *Labidomera clivicollis*. Oikos 92:91–100.

Benesh, D. P., E. T. Valtonen, and O. Seppaelae. 2008. Multidimensionality and intra-individual variation in host manipulation by acanthocephalan. Parasitology 135:617–626.

Berdoy, M., J. P. Webster, and D. W. Macdonald. 2000. Fatal attraction in rats infected with *Toxoplasma gondii*. Proc. R. Soc. Lond. B 267:1591–1594.

Brodeur, J., and L. E. M. Vet. 1994. Usurpation of host behaviour by a parasitic wasp. Anim. Behav. 48:187–192.

Cézilly, F., and M.-J. Perrot-Minnot. 2005. Studying adaptive changes in the behaviour of infected hosts: a long and winding road. Behav. Process. 68:223–228.

Cézilly, F., and M.-J. Perrot-Minnot. 2010. Interpreting multidimensionality in parasite-induced phenotypic alterations: panselectionism versus parsimony. Oikos 119:1224–1229.

Dawkins, R. 1982. The extended phenotype. Oxford Univ. Press, Oxford, U.K.

Dianne, L., T. Rigaud, S. Léger, S. Motreuil, A. Bauer, and M. J. Perrot-Minnot. 2010. Intraspecific conflict over host manipulation between different larval parasites of an acanthocephalan parasite. J. Evol. Biol. 23:2648–2655.

Drummond, F. A., R. A. Cassagrande, and P. A. Logan. 1989. Population dynamics of *Chrysomelobia labidomerae* Eickwort, a parasite of the Colorado potato beetle. Int. J. Acarol. 15:31–45.

Grossman, A. H., A. Janssen, E. F. De Brito, E. G. Cordeiro, F. Colares, J. O. Fonseca, et al. 2008. Parasitoid increases survival of its pupae by inducing hosts to fight predators. PLoS ONE 3:e2276.

Haine, E. R., K. Boucansaud, and T. Rigaud. 2005. Conflict between parasites with different transmission strategies infecting an amphipod host. Proc. R. Soc. Lond. B 272:2505–2510.

Harvey, J. A., M. Kos, Y. Nakamatsu, T. Tanaka, M. Dicke, L. E. M. Vet, et al. 2008. Do parasitized catterpillars protect their parasitoids from hyperparasitoids? A test of the'usurpation hypothesis'. Anim. Behav. 76:701–708.

Helluy, S. 1984. Relations hôtes-parasites du trématode *Microphallus papillorobustus* (Rankin, 1940). III. Facteurs impliqués dans les modifications du comportement des *Gammarus* hôtes intermédiaires et tests de prédation. Ann. Parasitol. Hum. Comp. 59:41–56.

Hughes, D., J. Brodeur, and F. Thomas. 2012. Host manipulation by parasites. Oxford Univ. Press, Oxford, U.K.

Lafferty, K. D., F. Thomas, and R. Poulin. 2000. Evolution of host-phenotype manipulation by parasites and its consequences. Pp. 117–127 *in* R. Poulin, S. Morand and A. Skorping, eds. Evolutionary biology of host-parasite relationships: theory meets reality. Elsevier, Amsterdam.

Lefèvre, T., B. Roche, R. Poulin, H. Hurd, F. Renaud, and F. Thomas. 2008. Exploitation of host compensatory responses: the 'must' of manipulation? Trends Parasitol. 24:435–439.

Lefèvre, T., S. A. Adamo, D. G. Biron, D. Missé, D. Hughes, and F. Thomas. 2009. Invasion of the body snatchers: the diversity and evolution of manipulative strategies in host-parasite interactions. Adv. Parasitol. 68:45–83.

Maure, F., J. Brodeur, N. Ponlet, J. Doyon, A. Firlej, E. Elguero, et al. 2011. The cost of a bodyguard. Biol. Letters 6:843–846.

Moore, J. 2002. Parasites and the behavior of animals. Oxford series in ecology and evolution. Oxford Univ. Press, Oxford, U.K.

Poulin, R. 1994a. The evolution of parasite manipulation of host behaviour: a theoretical analysis. Parasitology 109:S109–S118.

Poulin, R. 1994b. Meta-analysis of parasite-induced behavioural changes. Anim. Behav. 48:137–146.

Poulin, R. 2010. Parasite manipulation of host behavior: an update and frequently asked questions. Adv. Stud. Behav. 41:151–186.

Thomas, F., F. Renaud, and F. Cézilly. 1996. Assortative mating by parasitic prevalence in *Gammarus insensibilis* (Amphipoda): patterns and processes. Anim. Behav. 52:683–690.

Thomas, F., A. Schmidt-Rhaesa, G. Martin, C. Manu, P. Durand, and F. Renaud. 2002a. Do Hairworms (Nematomorpha) manipulate their terrestrial host to seek water? J. Evol. Biol. 15:356–361.

Thomas, F., S. P. Brown, M. Sukhdeo, and F. Renaud. 2002b. Understanding parasite strategies: a state-dependent approach? Trends Parasitol. 18:387–390.

Thomas, F., J. Fauchier, and K. D. Lafferty. 2002c. Conflict of interest between a nematode and a trematode in an amphipod host: test of the sabotage hypothesid. Behav. Ecol. Sociobiol. 51:296–301.

Thomas, F., S. A. Adamo, and J. Moore. 2005. Parasitic manipulation: where are we and where should we go? Behav. Process. 68:185–199.

Thomas, F., R. Poulin, and J. Brodeur. 2010. Host manipulation by parasites: a multidimensional phenomenon. Oikos 119:1217–1223.

Thomas, F., J. Brodeur, F. Maure, N. Franceschi, S. Blanchet, and T. Rigaud. 2011. Intraspecific variability in host manipulation by parasites. Infect. Genet. Evol. 11:262–269.

Thomas, F., T. Rigaud , and J. Brodeur. 2012. Evolutionary routes leading to host manipulation by parasites. Pp. 16–34 *in* D. Hughes, J. Brodeur, F. Thomas, eds. Host manipulation by parasites. Oxford Univ. Press, Oxford, U.K.

Yanoviak, S. P., M. Kaspari, R. Dudley, and G. Poinar Jr.,. 2008. Parasite-induced fruit mimicry in a tropical canopy ant. Am. Nat. 171:536–544.

Supporting Information

Additional Supporting Information may be found in the online version of this article:

Table S1. Compensatory responses affect the rate of predation (τCR).

Table S2. Compensatory responses affect the susceptibility to predation by suitable predators (αCR).