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REVIEW

Parasite manipulation of host personality and behavioural syndromes

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Summary

The past decades have seen mounting evidence that parasites alter their host's behaviour in ways that benefit transmission, based on differences in the expression of behavioural traits between infected and control individuals, or on significant correlations between trait expression and infection levels. The multidimensional nature of host manipulation has only recently been recognised: parasites do not target single host traits, but instead suites of interrelated traits. Here, I use recent research on animal personality (behavioural differences among individuals consistent across time and situations) and behavioural syndromes (correlations at the population level among distinct behavioural traits, or between the same trait expressed in different contexts) to provide a framework from which simple testable patterns of host behavioural changes can be predicted. Following infection, a manipulative parasite could (i) change the temporal consistency of its host's behavioural responses, (ii) change the slope of a host reaction norm, i.e. the way host behavioural traits are expressed as a function of an environmental gradient, or (iii) decouple two or more host behavioural traits and/or change the way in which they correlate with each other. Two case studies involving trematode parasites and their freshwater hosts are used to provide empirical illustrations of the above scenarios. These clearly illustrate the full richness of behavioural alterations induced by parasites, and how these effects would go unnoticed using the classical trait-by-trait comparisons of mean values between parasitised and non-parasitised individuals. However, the power of animal personality and behavioural syndromes to inform research on host manipulation by parasites will only be fully realised when underlying mechanisms are elucidated and linked to their phenotypic impacts.

Key words: animal personality, behavioural syndrome, host manipulation, reaction norm, trematode.

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Introduction

The host–parasite interaction is often depicted as a conflict between two antagonists over resources that appear to 'belong' to the host. However, the conflict also extends to other levels. For instance, the way the host behaves has consequences for both itself and its parasites, as the host serves as a common vehicle for its genotype and that of all symbionts it harbours. In many situations, the behavioural decisions that benefit the host may differ from those that would benefit its parasites, i.e. those behavioural choices by the host that would facilitate parasite transmission to other hosts. Among the many possible outcomes of such a conflict, in numerous cases natural selection has favoured the manipulation of host behaviour by its parasites. Indeed, in many unrelated taxa of parasites exploiting a broad taxonomic range of hosts, following infection, the parasite alters the behaviour, or more generally the phenotype, of its host in ways that benefit its own replication or transmission (Poulin, 1995; Moore, 2002; Thomas et al., 2005). Theory predicts that this manipulative strategy should evolve under many different scenarios (Poulin, 1994; Parker et al., 2009; Vickery and Poulin, 2010), and, indeed, examples from the natural world now amount to a very long list.

Most of the early, and many of the recent, examples consist of documenting either a statistically significant difference in the expression of a given behavioural or morphological trait between infected and control host individuals, or a significant correlation between the expression of that trait and infection levels (reviewed in Moore, 2002). However, in recent years the recognition that parasite manipulation does not target single host traits, but instead a suite of more-or-less interrelated traits, has emerged to provide new research directions (Cézilly and Perrot-Minnot, 2005; Thomas et al., 2010). Host manipulation is clearly a multidimensional phenomenon that should be measured simultaneously along several phenotypic axes. For example, the parasitic nematode *Myrmeconema neotropicum*, which must be transmitted from ant intermediate hosts to frugivorous birds in order to complete its life cycle, alters a suite of phenotypic traits in infected ants. The parasite not only turns the ant's abdomen bright red but also causes the ant to find a patch of red berries on a tree branch, stay within that patch, and maintain its abdomen raised at an angle close to vertical, in order for it to convincingly mimic a small fruit and deceive a bird (Yanoviak et al., 2008). Similarly, the acanthocephalan *Acanthocephalus lucii* not only reduces the time spent in hiding by its intermediate isopod host but also increases the darkness of the host's abdominal coloration, a combination likely to increase the visibility of infected isopods to fish definitive hosts (Benesh et al., 2008). In these and many other instances, the successful outcome of the manipulation for the parasite may depend on the combined effects of several altered traits in the host. Therefore, hosts manipulated by parasites are often not just slightly different from non-parasitised ones but instead are radically modified in several phenotypic dimensions.

The burgeoning field of animal personality and behavioural syndromes provides an ideal framework in which to explore the multidimensionality of host manipulation by parasites. Animal

'personality' refers to the behavioural differences among individuals in a population that are consistent over time and across situations (Réale et al., 2010). The related notion of behavioural syndromes concerns the existence of correlations at the population level either among distinct behavioural traits or between the same behavioural trait expressed in two different contexts (Sih et al., 2004). Current research on behavioural syndromes has generated simple analytical tools and provided a context for the development of hypotheses about the ways in which parasites could alter the host phenotype as a whole instead of single host traits. Here, after a brief overview of key concepts and approaches in animal personality research, I will discuss how host manipulation can be integrated within the emerging behavioural patterns that make up behavioural syndromes, and present the types of predictions that arise from this integration and how they can be tested. Then, I will provide empirical evidence that host personality and behavioural syndromes may be affected by parasites, focusing on two recent case studies. Finally, I will conclude with an outlook on the future application of ideas from research on animal personality and behavioural syndromes to research on host manipulation by parasites.

Animal personality and behavioural syndromes

The consistent differences in behavioural traits among individuals that define animal personalities, and the correlations among behavioural traits that constitute behavioural syndromes, are easily quantifiable properties of individuals and/or populations (see Fig. 1). They have now been documented across a wide range of taxa, and are increasingly believed to play important roles in evolution and ecology, from determinants of social organisation to key factors in dispersal and invasion (Sih et al., 2004; Réale et al., 2007). The existence of behavioural syndromes is no surprise, as genetic co-variance among traits has long been recognised in the field of quantitative genetics (Roff, 1997). Associations between traits arise from either pleiotropy (one gene influencing two or more traits) or linkage disequilibrium (non-random association of alleles at two or more loci). A full interpretation of phenotypic evolution requires measurement of the heritability not only of the traits themselves but also of the co-variance among traits (Stirling et al., 2002; Walsh and Blows, 2009). Co-varying traits can represent integrated modules that interact to perform a given function; if variation in performance impacts fitness, selection will further reinforce the integration of modular traits, such that they will tend to be inherited together. Although this is usually conceived in terms of morphological or life history traits (Roff, 1997; Klingenberg, 2010), the principle extends to behavioural traits.

In principle, animal personalities and behavioural syndromes can be measured along any suite of behavioural axes. In practice, however, most research has focused on five general areas of behaviour (Réale et al., 2007): (i) the shyness–boldness continuum, characterising an individual's reaction to a perceived risk, for example to a predator; (ii) the exploration–avoidance continuum, defining an individual's response to a novel situation, including food, habitat or objects; (iii) general activity level, but not in response to a novel habitat or situation; (iv) aggressiveness, i.e. an individual's agonistic reaction towards conspecifics; and (v) sociability, or an individual's reaction to conspecifics, but excluding aggressive behaviour. At the individual level, consistency in these behaviours may not always be beneficial. For example, a bolder individual could benefit in certain situations, such as when boldness allows for increased foraging time, but in other situations it might be disadvantaged, such as in the presence of predators (Smith and Blumstein, 2008). The persistence of consistent and repeatable behavioural responses, which may seem maladaptive compared with plastic responses, can be explained by linkages among different traits within behavioural syndromes (Bell, 2007; Dingemanse and Wolf, 2010).

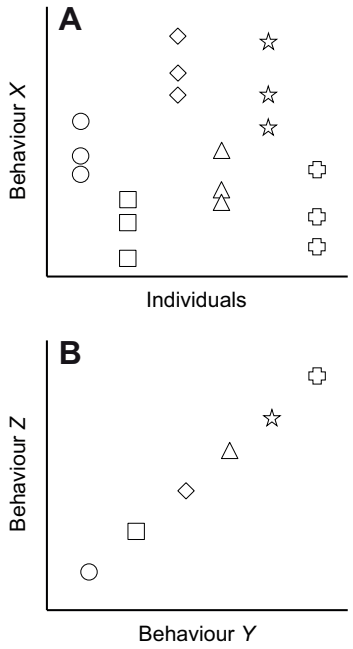

Fig. 1. Animal personalities and behavioural syndromes can be captured visually by two simple quantitative patterns. (A) For any behavioural trait, individuals show relatively consistent scores over time or across contexts, such that variance in their individual scores is less than the population variance. (B) There are significant correlations across individuals between pairs of behavioural traits forming a behavioural syndrome, with high values for one trait associated with high values for the other trait (or with low ones, in the case of negative correlations). Different symbols represent behavioural measurements taken on different individuals.

It is important to point out that behavioural plasticity itself, sometimes referred to as an individual's 'responsiveness', is itself a behavioural characteristic that varies among individuals in a population, and is therefore also a personality trait (Dingemanse and Wolf, 2010; Stamps and Groothuis, 2010). A promising avenue of research involves quantifying the variation in the expression of a particular behavioural trait in the same individual but across varying environmental conditions, using a reaction norm approach (Stamps and Groothuis, 2010). A reaction norm is the pattern of phenotypic expression of a given genotype across a spectrum of environmental conditions, usually visualised in bivariate phenotype-by-environment space. Some individuals' genotypes may be 'canalised' to produce a fixed trait value across all environments, whereas others may display great plasticity. In addition to highlighting differences among individuals in responsiveness, this approach has two advantages. Firstly, it can confirm that the relative ranking of individuals with respect to a given behavioural trait remains unchanged across circumstances, a confirmation of the consistent differences that determine personalities. Secondly, it can reveal different directions in the responses of individuals to environmental gradients (Fig. 2). For instance, two individuals with identical average behavioural scores and with the same variance among their scores, might nonetheless respond differently to environmental conditions. The slope of

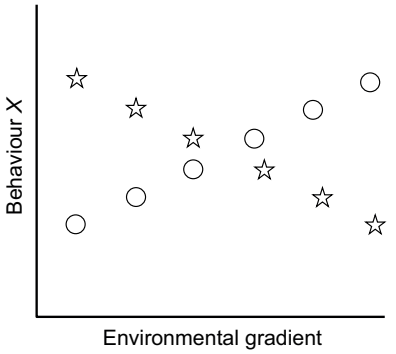

Fig. 2. Behavioural measurements on two different individuals (represented by different symbols) as a function of a gradient in environmental or ecological context. The reaction norm plot reveals that although the two individuals have the same average behavioural score, and the same variance, they differ in how the expression of the behaviour varies across contexts. The slope of the reaction norm therefore provides an additional dimension to animal personality. Modified from Dingemanse and Wolf (Dingemanse and Wolf, 2010).

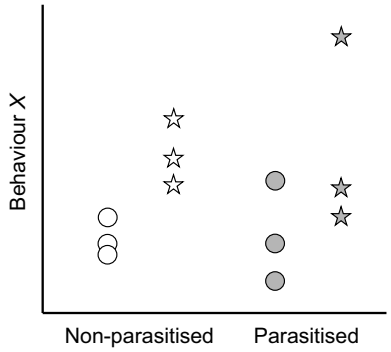

Fig. 3. Decreased consistency in the expression of a particular behaviour following parasitic infection in two different individuals (represented by different symbols). Although the average behavioural score of non-parasitised (open symbols) individuals does not change after they are parasitised (filled symbols), the variance among values obtained at different times or in slightly different contexts is increased.

reaction norms can therefore provide an additional dimension along which to characterise animal personalities.

Various behavioural traits like activity, boldness or sociability are no longer considered in isolation as personality characteristics but instead as suites of inter-related traits. The characteristic set of behaviours displayed by an individual represents its behavioural type, whereas correlations across all individuals between pairs of behavioural traits form the population's behavioural syndrome (Bell, 2007). These correlations are widespread; in particular, correlations between boldness, aggressiveness and exploration behaviour have been reported for several species (Bell, 2005; Johnson and Sih, 2005; Eriksson et al., 2010). These findings suggest that these behaviours evolve in tandem, i.e. as a whole inter-connected suite of traits, instead of evolving separately (Sih et al., 2004). Indeed, genetic linkages between traits may constrain their independent evolution and result in indirect selection of non-target traits or whole suites of behaviours as 'packages' (Bell, 2005).

Parasite manipulation of behavioural syndromes

In principle, coordinated changes in several aspects of host behaviour and phenotype could yield greater benefits to a manipulating parasite than changes to a single trait (Thomas et al., 2010). Consider for instance the case of a larval helminth parasite inside its intermediate host, awaiting transfer to its predatory definitive host through predation by the latter on the former host. Typically (though not always), these parasites exploit well-established predator–prey interactions, with the normal rates of predation providing a passage to the definitive host. However, as a result of the predator–prey co-evolutionary arms race, the intermediate prey host is probably well adapted to elude predators, with its anti-predator adaptations possibly including cryptic coloration along with several morphological and behavioural traits. It is therefore likely that, from the parasite's perspective, interfering with only one of these defences would not increase transmission rates to the definitive host as much as altering the whole suite of defensive traits all at once. Whether or not this is actually achievable depends greatly on the underlying physiological mechanisms. If the traits to be targeted are the visible products of independent mechanisms, then the challenge may be insurmountable. In many cases, however, it is possible that altering a single neurotransmitter or hormone can have cascading repercussions on several linked behavioural traits (Adamo, 2002; Shaw et al., 2009), or that inducing compensatory responses in the host can result in changes to several traits at little cost to the parasite (Lefèvre et al., 2008). In cases like these, behavioural syndromes could be prime targets for host manipulation.

Parasites impose fitness costs on animals, and several behaviours clearly affect the risk of exposure to parasite infection. For example, sociability can increase the acquisition of contagious pathogens and ectoparasites through contact with infected conspecifics, and more active and exploratory fish can expose themselves to more infection by skin-penetrating trematode larvae (Wilson et al., 1993). Parasites should therefore act as selection agents, shaping individual personalities and behavioural syndromes, just like predation risk or resource availability (Barber and Dingemanse, 2010; Kortet et al., 2010). From a physiological perspective, parasite infection can also change the energy status of the host, or some other state variable, with consequences for the expression of personality traits (Barber and Dingemanse, 2010; Kortet et al., 2010). Here, I am only concerned with the scenario in which parasites target some aspects of host personality as part of their manipulation of host phenotype (Poulin, 2010). I present several ways in which parasites could affect host personality or behavioural syndromes such that parasite transmission may be enhanced, using simple graphical illustrations of the sort of empirical patterns one might observe. This is not meant to represent an exhaustive list of possibilities but instead some of the most readily measurable ways in which infection may alter host personality. In addition, it is important to note that the effects of parasite infection on host personality may be indistinguishable from those manifested outside of an adaptive manipulation context. Indeed, pathological consequences of infection may disrupt host behaviour in similar ways, and as always the distinction between adaptation and by-products remains difficult (Poulin, 1995).

First, parasites could modify a central feature of host personality simply by decreasing or increasing the consistency of host behaviour (Fig. 3). In a particular context, and given an individual's other behavioural traits, the expression of a selected behaviour is typically restricted to a narrow range of values that yield the

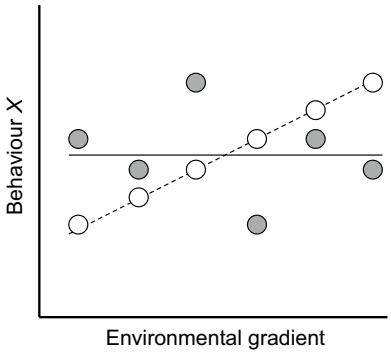

Fig. 4. Behavioural measurements on the same individual as a function of a gradient in environmental or ecological context, shown as a reaction norm plot. Neither the average behavioural score nor the variance across time or different contexts differs between measurements taken on the non-parasitised (open symbols) individual and those taken after it is parasitised (filled symbols). However, the slope of the reaction norm changes from positive to zero, indicating that parasitic infection makes the host's responses independent from the context in which they are measured.

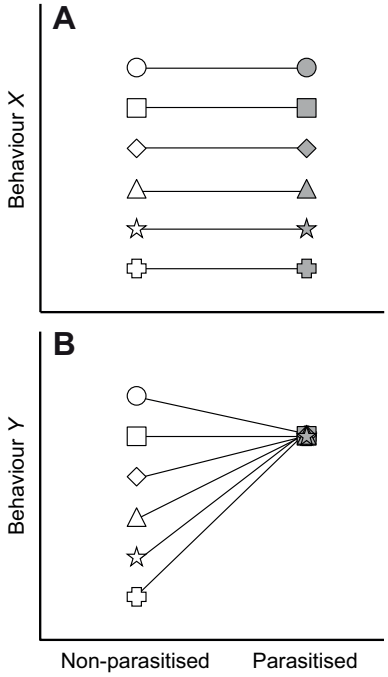

Fig. 5. Behavioural measurements on different individuals (represented by different symbols), taken both when they are non-parasitised (open symbols) and after they are parasitised (filled symbols). The lines connecting the points for the same individual help to visualise the effect of infection on host behaviour. (A) The expression of behaviour *X* is unaffected by parasite infection. (B) In contrast, the expression of behaviour *Y* shows similarly high values for all individuals following parasite infection. Modified from Barber and Dingemanse (Barber and Dingemanse, 2010).

greatest benefit for the individual. Faced with a predator, only a narrow spectrum of responses along the shyness–boldness continuum can help the individual escape. Any trophically transmitted parasite, such as larval helminths using prey organisms as intermediate hosts, capable of generating variance in its host's behavioural responses might increase its own probability of reaching its predatory definitive host. Decreased consistency in host behaviour would amount to a greater probability of a mismatch between stimulus and response.

Second, parasites could change the slope of a behavioural reaction norm without necessarily changing the average value of a behavioural trait or the variance in its expression (Fig. 4). By making the response of the host completely independent from the context in which it is expressed, or by reversing the trend in the response as a function of the environmental context, the parasite could achieve greater transmission success. For instance, any change to the reaction norm characterising the expression of anti-predator responses by the host in relation to hunger or perceived risk should benefit trophically transmitted parasites.

Third, parasites may have specific effects on a particular behavioural trait without affecting others, therefore decoupling traits and/or changing the way in which they correlate with each other (Fig. 5). Consider the case of a prey using crypsis to avoid predation. For cryptic coloration to be an efficient defence, it much be coupled with the selection of microhabitats providing background colour matching that of the prey's body, and with a tendency to freeze when perceiving a risk. These traits work only in conjunction with each other; any trophically transmitted parasite capable of uncoupling them could achieve a higher probability of transmission. We should therefore expect correlations between pairs of traits to differ between parasitised and non-parasitised conspecifics (Barber and Dingemanse, 2010).

Correlations between host traits are probably frequently altered by parasite infection, as many behavioural traits are often the outcome of the same chain of endocrine or physiological phenomena (Benesh et al., 2008). These changes have gone undetected because the vast majority of past studies, even when focusing on multiple host traits, have tended to only compare average (or median) trait values between parasitised and non-parasitised individuals, and not the relationships between different traits (Poulin, 2010). There are many ways in which the correlation between two behavioural traits across individuals can be altered by parasitic infection without the mean value of either trait differing between parasitised and non-parasitised individuals (Fig. 6). The next section provides case studies demonstrating how parasites do indeed affect host personalities and behavioural syndromes in some of the ways described above.

Empirical examples

In recent years, the influence of parasites on human personality has been highlighted in a series of intriguing studies into the effects of the protozoan parasite *Toxoplasma gondii* (Flegr et al., 1996; Flegr et al., 2000; Flegr, 2007). Apart from these mostly psychological studies, several animal personality traits, when considered one at a time, have been shown to differ significantly between parasitised and non-parasitised individuals within a population (reviewed in Moore, 2002; Barber and Dingemanse, 2010). For example, trematode infection reduces sociability in killifish *Fundulus diaphanus* (Krause and Godin, 1994), cestode infection increases boldness in sticklebacks *Gasterosteus aculeatus* (Giles, 1983), and malaria infection increases exploratory behaviour in female great tits, *Parus major* (Dunn et al., 2011). However, there are still very few studies attempting to link parasitic infection to either of the fundamental properties of personality or behavioural syndromes, i.e. consistency of behavioural traits across context and correlations between traits across individuals.

Here, I use two case studies involving trematodes and their hosts from New Zealand freshwater ecosystems as empirical examples

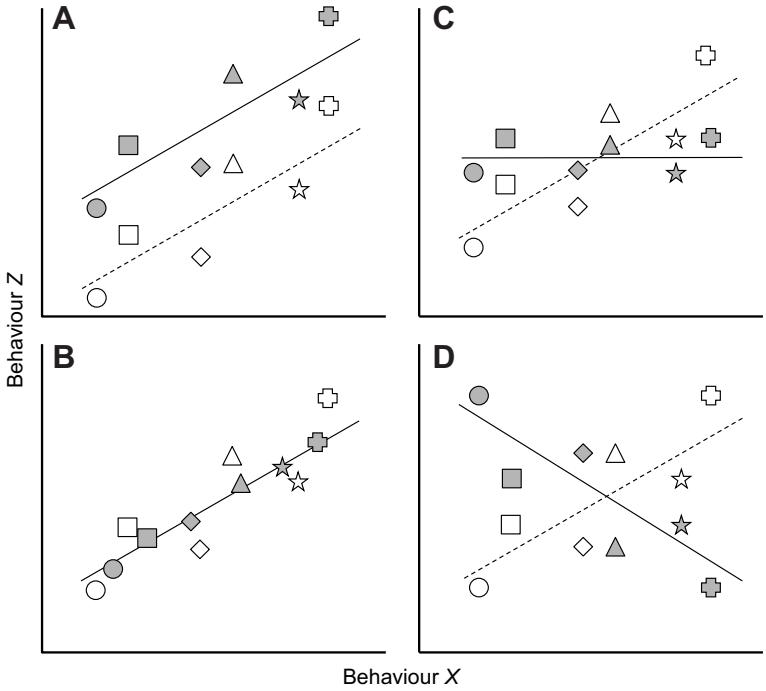

Fig. 6. Possible effects of a manipulative parasite on the correlation between two host traits. Behavioural measurements from different individuals and their correlation are represented by different symbols, taken both when they are non-parasitised (open symbols, broken line) and after they are parasitised (filled symbols, solid line). (A) The manipulation results in an increase of values for behaviour *Z* only, but no change in the correlation between traits. (B) The manipulation results in a strengthening of the correlation between traits, i.e. tighter clustering of points around the trend line. (C) The manipulation results in an uncoupling of the association between the two traits, such that their values are no longer significantly correlated. (D) The manipulation results in a reversal in the direction of the correlation between traits, from positive for non-parasitised hosts, to negative for parasitised ones. Note that in scenarios B, C and D, the parasite induces no change in the average trait values, only a change in how they correlate with each other; therefore, a comparison of mean trait values between parasitised and non-parasitised hosts would completely miss the effect of manipulation. Modified from Poulin (Poulin, 2010).

of the disruption, whether adaptive or not, to host personality and behavioural syndromes caused by infection. Trematodes have complex life cycles, involving three hosts (Fig. 7). The first intermediate host is usually a snail, in which the parasite multiplies asexually to produce large numbers of cercariae, which are motile infective stages responsible for transmission to the next host. After leaving the snail host, cercariae seek and penetrate the second intermediate host, through its skin or cuticle. Depending on the trematode species, the second intermediate host can be an invertebrate, an amphibian or a small fish; in this host, the cercariae grow to some extent and encyst as metacercariae, to await ingestion by their definitive host. The latter is typically a vertebrate predator of the second intermediate host. In the definitive host, the parasites excyst and settle in their target tissue, usually a part of the digestive tract, to reproduce sexually and release eggs. When eggs are released in the definitive host's faeces, they hatch into a ciliated larva that will seek and infest a snail, thereby completing the cycle. It is at the stage of trophic transmission, requiring predation of the second intermediate host by the definitive host (Fig. 7), that manipulation of host behaviour is most likely to be manifested. Indeed, many trematodes are known to modify the behaviour or morphology of their second intermediate hosts in ways that can increase transmission success (reviewed in Moore, 2002).

The first case study is an investigation into the behaviour of the amphipod *Paracalliope fluviatilis*, which serves as second intermediate host for the trematode *Microphallus* sp. (Coats et al., 2010). This parasite reaches its definitive host when ducks feed on infected amphipods or other small crustaceans also suitable as second intermediate hosts. Like other members of its genus (e.g. Helluy, 1983), this parasite alters the behaviour of at least one of its second intermediate host species: infected isopods, *Austridotea annectens*, swim more actively and show weaker evasive responses when exposed to a simulated predator than uninfected conspecifics (Hansen and Poulin, 2005). In their study, Coats and colleagues focused on three behavioural traits, each measured three times on parasitised and non-parasitised amphipods (Coats et al., 2010). These traits can, in principle, affect the susceptibility of amphipods to predation by ducks. Phototaxis was measured as the amphipods' response to light, by scoring the frequency of their visits to well-lit areas within a horizontal tube with light coming from one end. Swimming activity was scored as the spontaneous activity of the amphipod in the absence of stimulation, measured as the frequency of swimming bouts out of a fixed observation period. Finally, vertical distribution was quantified in a glass cylinder with uniform light coming from the side and not from above (to distinguish geotaxis from phototaxis), using a scale reflecting height in the water column. The experiments used naturally infected amphipods; upon dissection after the behavioural tests, all individuals found to harbour parasites other than *Microphallus* were treated separately. For the present purposes, amphipods could therefore be split into two groups, non-infected individuals and those parasitised by *Microphallus*, with the vast majority harbouring a single metacercaria and no amphipod harbouring more than four.

The results show two interesting associations between parasitism and the personality and behavioural syndromes of amphipods. Firstly, in comparisons of trait values between parasitised and non-parasitised individuals, a difference was found for phototaxis but not for swimming activity or vertical distribution (Coats et al., 2010). However, there was a difference between parasitised and non-parasitised individuals with respect to how these behavioural traits correlate with each other across individuals (Fig. 8). Phototaxis correlated positively with both swimming activity and vertical distribution only among parasitised amphipods, not among non-parasitised ones. In simple terms, these correlations mean that amphipods harbouring *Microphallus* that are attracted to light also show more active swimming and spend more time closer to the water surface. From an adaptive perspective, the linkages among these behavioural traits might reinforce each other, such that the outcome is an even greater probability of transmission to ducks than without the correlations. However, the benefits of these correlated behaviours remain to be tested. Nevertheless, these findings indicate that behavioural syndromes may only be

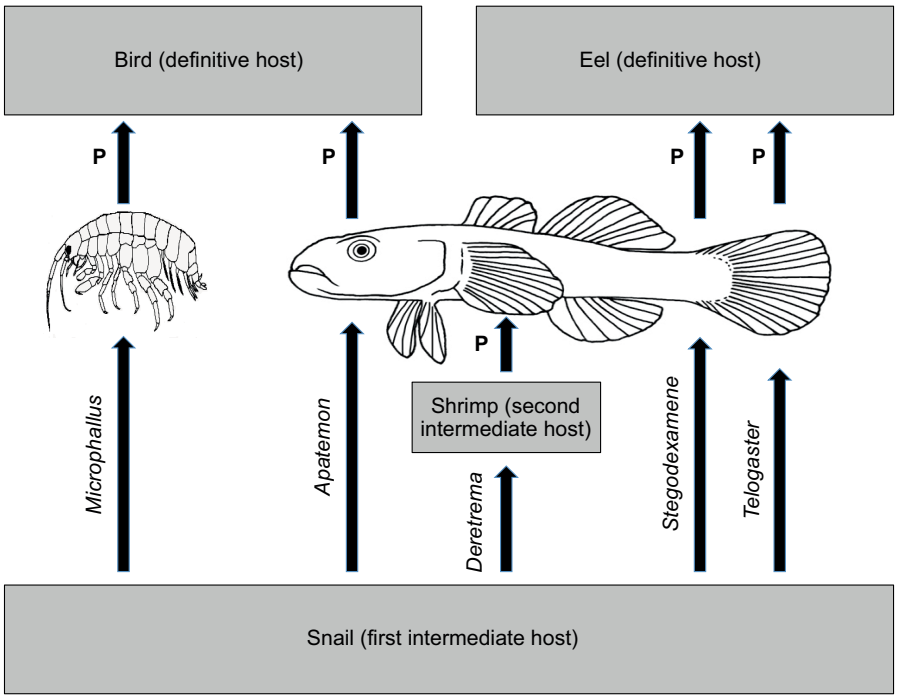

Fig. 7. Schematic summary of the life cycles of five trematode species from New Zealand freshwater systems used in the case studies. The two focal host species, the amphipod *Paracalliope fluviatilis* and the fish *Gobiomorphus cotidianus*, are shown in full, with other hosts shown as boxes. The trematode *Microphallus* sp. uses the amphipod as its second intermediate host, whereas *Apatemon* sp., *Stegodexamene anguillae* and *Telogaster opisthorchis* use the fish as second intermediate host; in contrast, *Deretrema minuta* uses the fish as definitive host. For simplicity, only genus names of the parasites are used in the figure and in the text. The stage of the life cycle where transmission from one host to the next occurs *via* predation is indicated by a P.

manifested following parasite infection, whether due to parasite manipulation or mere physiological stress. Thus, simply comparing mean trait values between parasitised and non-parasitised individuals is insufficient to reveal the full range of parasite effects on host behaviour.

Secondly, Coats and colleagues also found that the repeatability of phototaxis scores for amphipods infected with *Microphallus* was significantly lower than that for non-parasitised amphipods (Coats et al., 2010). This reduction was due more to a decreased variance in response to light among infected individuals than to an increase in within-individual variance. Such a pattern would be consistent with parasite manipulation, with infection homogenising behavioural responses toward some value favouring increased parasite transmission. Whatever the underlying explanation, this finding was the first demonstration that parasitism can change population-level behavioural repeatability values.

The second case study is an investigation into the behaviour of a small benthic fish, the common bully *Gobiomorphus cotidianus*, which serves as second intermediate host for three trematodes and as definitive host for a fourth one (Fig. 7). The three species using bullies as intermediate host can induce phenotypic alterations in the fish that range from subtle to dramatic, depending on the age/size of the host at infection. In larval fish, infection by skin-penetrating trematodes can cause severe malformations and high mortality (Kelly et al., 2010); however, if fish are older when infected, these trematodes cause only small or no changes in behaviours such as responsiveness to the presence of a predator (Poulin, 1993) or aggressiveness towards conspecifics (Hamilton and Poulin, 1995). Using naturally infected fish, Hammond-Tooke and colleagues (Hammond-Tooke et al., 2012) focused on three behavioural traits now widely used in research on animal personality and behavioural syndromes (see earlier), measured using standard procedures for fish (e.g. Eriksson et al., 2010). Aggressiveness was measured as the time spent by each fish attacking a same-sized conspecific, simulated using a mirror in the test tank. Boldness was quantified as the time needed to emerge from a refuge following a simulated predator attack. Finally, activity was measured as the proportion of time spent swimming spontaneously in the absence of any stimuli. These behaviours were measured on six separate occasions for each fish, including twice during exposure to odours from predators (eels) that are also definitive hosts for two of the trematodes, *Telogaster* and *Stegodexamene*. Following dissection, practically all fish harboured the three trematodes *Apatemon*, *Telogaster* and *Stegodexamene*, which used them as intermediate hosts, though infection levels varied widely; in contrast, only a little over half the fish were infected by *Deretrema*, and only at low levels (Hammond-Tooke et al., 2012).

In the present context, Hammond-Tooke and colleagues' most interesting result is the finding that different parasite species had different effects on the within-individual consistency in values obtained for the different behavioural traits (Hammond-Tooke et al., 2012). The extent to which a particular behaviour varies in a given fish was estimated as the coefficient of variation, i.e. the standard deviation of the six behavioural values obtained divided by their mean. For any individual fish, the lower the coefficient of variation, the higher the consistency in its behaviour. Of the three behavioural traits considered, the consistency of boldness was most affected by parasitic infection: it correlated negatively with infections by *Telogaster*, positively with high *Deretrema* infections, and positively though not quite significantly with infections by *Apatemon* (Hammond-Tooke et al., 2012). However, the actual relationship with parasite load is more complicated (Fig. 9). Thus, fish with more *Telogaster* tended to show lower consistency in their boldness responses; in contrast, in the case of *Deretrema* the reduced consistency in boldness responses was only seen in fish harbouring a single worm, with those carrying more severe infections having a similar consistency to that of uninfected fish (Fig. 9). Activity was also significantly related to infection by *Deretrema*, but there was no relationship between parasitism and the consistency of aggression levels (Hammond-Tooke et al., 2012). It is possible to speculate and weave adaptive stories around these findings. For example, *Telogaster* could benefit by increasing

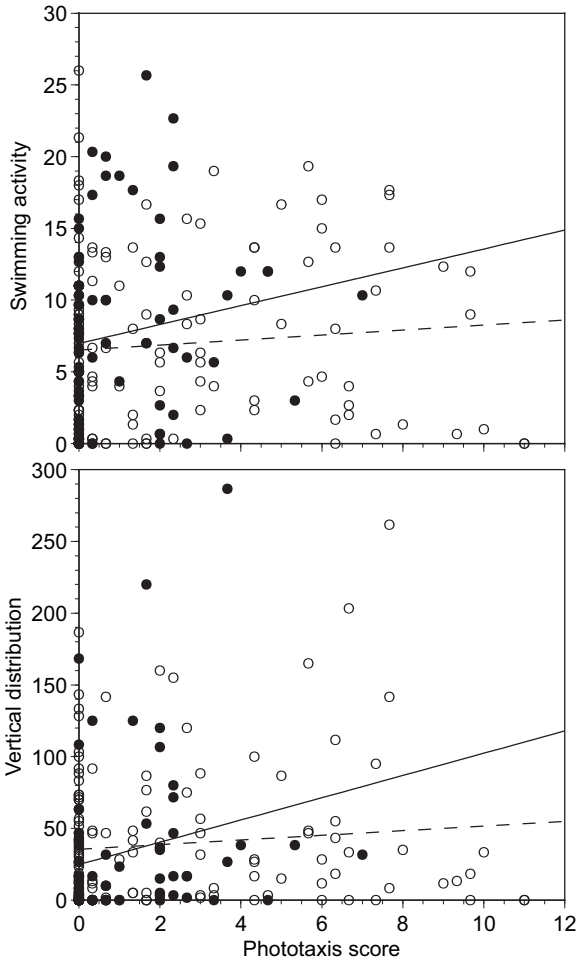

Fig. 8. Correlation between phototaxis and both swimming activity and vertical distribution of the amphipod *Paracalliope fluviatilis*, shown separately for individuals parasitised by the trematode *Microphallus* sp. (*N*=69, filled symbols, solid line) and non-parasitised individuals (*N*=138, open symbols, broken line). Each point is an average of three separate measurements per individual amphipod, with higher values representing stronger phototaxis, greater activity and higher vertical position in the water column. The two correlations involving parasitised individuals are statistically significant (Spearman rank correlation coefficients: phototaxis *versus* swimming activity, r_s=0.27; phototaxis *versus* vertical distribution, r_s=0.29; both $P \leq 0.03$), whereas neither correlation involving non-parasitised amphipods is significant ($P \geq 0.15$). Data from Coats et al. (Coats et al., 2010).

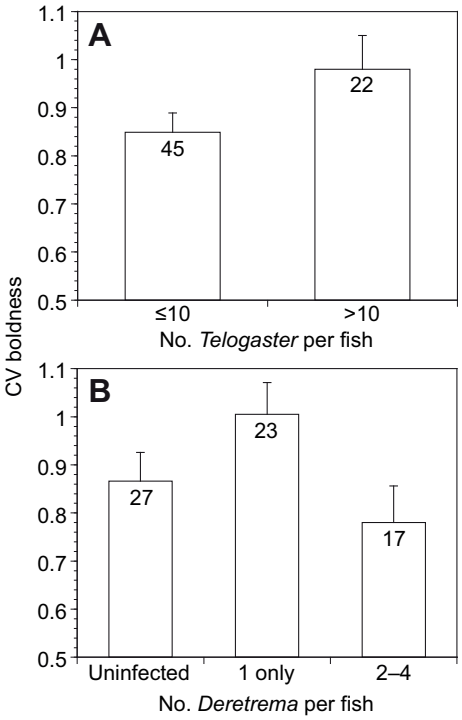

Fig. 9. Mean (±s.e.m.) coefficient of variation (CV) in boldness scores of individual *Gobiomorphus cotidianus* fish parasitised with different numbers of the trematodes *Telogaster opisthorchis* (A) and *Deretrema minuta* (B). Each individual fish was scored for boldness on six separate occasions; numbers on the bars indicate the number of individual fish in each group. Data from Hammond-Tooke et al. (Hammond-Tooke et al., 2012).

the heterogeneity of its host's responses to threats if this leads to a higher probability of predation. The apparent effect of *Deretrema*, which is not transmitted *via* predation, might be due to the host being unable to make fine adjustments to its responses as a consequence of physiological stress induced by infection, though the influence of parasite load remains to be explained. Whatever the underlying mechanisms, these results emphasise again the need to look at more than the average behavioural scores when attempting to relate infection and host behaviour, whether or not in an adaptive context.

These two case studies illustrate clearly the additional insights into host behavioural changes made possible by the general approach used in research on animal personality and behavioural syndromes. However, the two case studies suffer from the same weakness. Naturally infected hosts were used instead of experimentally infected ones, making it impossible to demonstrate beyond doubt that infection causes behavioural patterns, and not the other way around. This cause *versus* consequence dilemma is a common 'chicken-and-egg' problem plaguing research on parasite manipulation of host behaviour (Blanchet et al., 2009; Poulin, 2010). It does not invalidate the sorts of tests for behavioural consistency and correlations among behaviours presented above; it just means that future studies will need to improve on past ones.

Looking ahead

The increasing recognition that parasite manipulation does not target single host traits but suites of interrelated traits (Thomas et al., 2010) is set to meet the growing field of research on animal personality and behavioural syndromes. The latter provides the conceptual framework and simple analytical tools needed to make sense of the former (Barber and Dingemanse, 2010; Poulin, 2010). How can we take full advantage of the intersection between these two research programmes?

First, defining, measuring and choosing traits at the outset of a study, whether these are behavioural traits or other types of trait, are far from trivial matters. Including as many measurable traits as possible for the sake of completeness is not necessarily a useful strategy [see Blows for a relevant discussion (Blows, 2007)]. There exist methods for quantifying selection on multiple traits, and measuring their contributions to fitness (Lande and Arnold, 1983; Shaw and Geyer, 2010). However, these are problematic in situations such as host–parasite interactions where any given trait

may have a fitness impact on one organism or the other, on both organisms or on neither. Trait choice must be based on careful study design aimed at testing *a priori* hypotheses if the approach used in research on animal personality and behavioural syndromes is to benefit the study of host manipulation by parasites.

Second, as alluded to at the end of the previous section, future tests must involve animals infected experimentally under controlled conditions in order to resolve the cause-and-effect issue. We also need to expand these tests to other host–parasite systems, in particular those not only amenable to experimental manipulation but also currently held as paradigm or textbook examples of host manipulation by parasites. By applying a standard set of analyses to behavioural data (estimates of behavioural repeatability, relationship between within-individual consistency in behaviour and infection status, correlations among behaviours for both parasitised and non-parasitised individuals, etc.), we will be able to uncover the many nuances of host behavioural manipulation that have eluded past studies focused on single traits or on average behavioural scores.

Third, and most important, we need a greater and concerted effort to link whole-animal phenotypic effects with their underlying mechanisms. This call to arms has been made before (e.g. Poulin, 1995), but few have rallied behind it. If parasites target suites of inter-connected behavioural traits instead of single traits, the mechanisms linking different behaviours become as important as those linking infection and behaviour. Simple alterations to the relative levels of specific neuromodulator molecules, induced directly or indirectly by parasitic infection, can have cascading effects manifested through several seemingly independent behaviours (Adamo, 2002). The influence of parasitic infection can start all the way up the chain of physiological events leading to the manifestation of behavioural traits, i.e. at the stage of gene expression and protein synthesis (Biron et al., 2005; Poulin and Thomas, 2008). Identifying the host pathways targeted by parasites is essential to distinguish between 'hardwired' correlations between any two behavioural traits, and those resulting from trade-offs occurring post-infection through other pathways. We also need to determine what physiological processes result in the slight variability observed in the behavioural responses of an individual animal to the same stimulus, and how parasites can either increase or decrease this variability. Without a more solid mechanistic foundation, we may succeed in revealing the full richness of subtle behavioural changes occurring in an animal following parasitic infection, but we will not be able to discern between adaptive manipulation by the parasite, neutral but coincidental changes, compensatory responses by the host, and mere pathology.

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