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Magnitude and direction of parasite-induced phenotypic alterations: a meta-analysis in acanthocephalans

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ABSTRACT

Several parasite species have the ability to modify their host's phenotype to their own advantage thereby increasing the probability of transmission from one host to another. This phenomenon of host manipulation is interpreted as the expression of a parasite extended phenotype. Manipulative parasites generally affect multiple phenotypic traits in their hosts, although both the extent and adaptive significance of such multidimensionality in host manipulation is still poorly documented. To review the multidimensionality and magnitude of host manipulation, and to understand the causes of variation in trait value alteration, we performed a phylogenetically corrected meta-analysis, focusing on a model taxon: acanthocephalan parasites. Acanthocephala is a phylum of helminth parasites that use vertebrates as final hosts and invertebrates as intermediate hosts, and is one of the few parasite groups for which manipulation is predicted to be ancestral. We compiled 279 estimates of parasite-induced alterations in phenotypic trait value, from 81 studies and 13 acanthocephalan species, allocating a sign to effect size estimates according to the direction of alteration favouring parasite transmission, and grouped traits by category. Phylogenetic inertia accounted for a low proportion of variation in effect sizes. The overall average alteration of trait value was moderate and positive when considering the expected effect of alterations on trophic transmission success (signed effect sizes, after the onset of parasite infectivity to the final host). Variation in the alteration of trait value was affected by the category of phenotypic trait, with the largest alterations being reversed taxis/phobia and responses to stimuli, and increased vulnerability to predation, changes to reproductive traits (behavioural or physiological castration) and immunosuppression. Parasite transmission would thereby be facilitated mainly by changing mainly the choice of micro-habitat and the anti-predation behaviour of infected hosts, and by promoting energy-saving strategies in the host. In addition, infection with larval stages not yet infective to definitive hosts (acanthella) tends to induce opposite effects of comparable magnitude to infection with the infective stage (cystacanth), although this result should be considered with caution due to the low number of estimates with acanthella. This analysis raises important issues that should be considered in future studies investigating the adaptive significance of host manipulation, not only in acanthocephalans but also in other taxa. Specifically, the contribution of phenotypic traits to parasite transmission and the range of taxonomic diversity covered deserve thorough attention. In addition, the relationship between behaviour and immunity across parasite developmental stages and host–parasite systems (the neuropsychoimmune hypothesis of host manipulation), still awaits experimental evidence. Most of these issues apply more broadly to reported cases of host manipulation by other groups of parasites.

Key words: behaviour, helminth, host–parasite interaction, manipulation, multidimensionality, phylogenetic meta-analysis, publication bias, ribosomal DNA, trophic transmission

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CONTENTS

I. INTRODUCTION

Several parasites bring about phenotypic alterations in their hosts that appear to increase their own fitness at the expense of that of their hosts (Poulin, 1995; Moore, 2002; Thomas, Adamo, & Moore, 2005; Cézilly & Perrot-Minnot, 2010). Such parasite-induced phenotypic alterations (PIPAs) can take different forms, through affecting, for instance, the physiology (Plaistow, Troussard, & Cézilly, 2001; Tain, Perrot-Minnot, & Cezilly, 2006; Perrot-Minnot & Cezilly, 2013; Guler *et al.*, 2015; Kopp *et al.*, 2016; Perrot-Minnot, Maddaleno, & Cézilly, 2016), reproduction (Bollache, Gambade, & Cézilly, 2001; Bollache, Rigaud, & Cézilly, 2002; Rauque & Semenas, 2009; Bollache, 2016) or appearance (Lewis Jr, 1977; Camp & Huizinga, 1979; Oetinger & Nickol, 1981; Amato *et al.*, 2003; Wesołowska & Wesołowski, 2014) of infected hosts. However, most studies of PIPA concern the altered behaviour of host species. For instance, several species of ectoparasitoid wasps are known to modify the web-building behaviour of their spider hosts (Eberhard, 2000; Matsumoto, 2008; Korenko *et al.*, 2014; Takasuka *et al.*, 2015; Kloss *et al.*, 2017). Just before the wasp enters its final stage of development, the spider host builds a modified web in the form of a 'cocoon' (Eberhard, 2000) that appears to enhance the survival of the parasitoid pupae. Both rodents and chimpanzees infected with *Toxoplasma gondii* famously lose their innate aversion to the urine of feline predators (Berdoy, Webster, & Mcdonald, 2000; Dass & Vyas, 2014; Poirotte *et al.*, 2016), a phenomenon that presumably increases the transmission of the parasite to its final host. Similarly, several species of helminths with complex life cycles are known to alter the anti-predation behaviour of their intermediate arthropod hosts in ways that appear to enhance trophic transmission to final hosts (Hechtel, Johnson, & Juliano, 1993; Kaldonski, Perrot-Minnot, & Cézilly, 2007; Sánchez, Georgiev, & Green, 2007). For instance, whereas uninfected crustacean amphipods are significantly repulsed by the chemical cues originating from a fish predator, infected ones are significantly attracted to the odour (Baldauf *et al.*, 2007; Perrot-Minnot, Kaldonski, & Cézilly, 2007). Most of the time, such phenotypic alterations are interpreted as expressions of the extended phenotype (*sensu* Dawkins, 1982) of the parasite species, whose ability to 'manipulate' its host has evolved by natural selection (Moore, 2002; Thomas, Adamo, & Moore, 2005; Hughes, Brodeur, & Thomas, 2012). Alternatively, they could correspond to simple pathological effects (Chow & Mackauer, 1999; Edelaar, Drent, & De Goeij, 2003; Schutgens *et al.*, 2015) or to an adaptive host response (Smith Trail, 1980; Poulin, 1992; Poulin, Brodeur, & Moore, 1994). Whether the magnitude of parasite phenotypic alterations varies in relation to its consequences for the parasite and its host is poorly documented.

Although adaptive host manipulation has become a sort of paradigm in evolutionary parasitology and behavioural ecology (Poulin, 2000; Moore, 2002; Thomas, Adamo, & Moore, 2005; Bakker, Frommen, & Thuenken, 2017), growing evidence suggests that the 'purposive design' (*sensu* Poulin, 1995) of phenotypic alterations induced by parasites should be examined with more caution. A crucial step in validating the manipulation hypothesis is to show convincingly that a direct *causal* relationship exists between altered host phenotype and enhanced completion of the life cycle (Cézilly & Perrot-Minnot, 2010). Indeed, behavioural alterations observed in infected hosts that *seemingly* enhance the completion of the parasite's life cycle may not *actually* contribute to it. For instance, the behavioural alterations displayed by tenebrionid beetles infected with *Hymenolepis diminuta*, including reduced activity, concealment and photophobia (Hurd & Fogo, 1991; Robb & Reid, 1996), were initially interpreted as a case of manipulation. However, such phenotypic alterations do not necessarily result in a differential vulnerability of infected and uninfected beetles to predation by rodent final hosts (Webster *et al.*, 2000). Similar conclusions have been drawn from recent studies of two historical models of host manipulation. Crustacean amphipods serve as intermediate hosts for various acanthocephalan parasites that use different species of vertebrates as final hosts. Inside their intermediate hosts, larval acanthocephalans progressively develop into cystacanths, the infective stage for the definitive host. Cystacanths of several acanthocephalan species have a carotenoid-based, bright orange colouration (Gaillard *et al.*, 2004) that can be seen through the translucid cuticle of their hosts, such that infected hosts are particularly conspicuous, at least to the human eye. In addition, gammarids infected with acanthocephalans show altered behaviour, including reduced photophobia. Bethel & Holmes (1973, 1977) were the first to provide evidence for a causal link between the altered behaviour of gammarids infected with larval acanthocephalans and their increased vulnerability to predation, and the phenomenon was quickly regarded as a compelling example of host manipulation (Dawkins, 1982). Bakker, Mazzi, & Zala (1997) went further by arguing that both the modified appearance and the altered phototactic behaviour of *Pomphorhynchus laevis*-infected *Gammarus pulex* were responsible for their increased vulnerability to predation by three-spined sticklebacks, *Gasterosteus aculeatus*. However, more recent investigations using phenotypic engineering to manipulate one trait at a time (Kaldonski *et al.*, 2009; Perrot-Minnot *et al.*, 2012) demonstrated convincingly that neither parasite colour nor the altered phototactic behaviour of infected hosts *alone* contribute to the increased vulnerability of *P. laevis*-infected gammarids to fish predation. Therefore, several phenotypic changes might act synergistically to enhance trophic transmission.

Similarly, Worth, Lymbery, & Thompson (2013) questioned the adaptiveness of behavioural alterations induced by *T. gondii* in rodents, based on several lines of evidence. First, studies of mice and rats have resulted in conflicting results about what behaviours are or are not affected by infection. Second, behavioural alterations similar to those coincidental with *T. gondii* infection can also be induced by *Eimeria vermiformis*, a parasite that does not rely on trophic transmission to complete its life cycle [Kavaliers & Colwell, 1995; see also Cator *et al.*, 2013 for a related result in a markedly different host–parasite association]. Third, there is, surprisingly enough, no direct evidence that rodents infected with *T. gondii* are more vulnerable to predation by cats. Fourth, even if such evidence was available, it appears that cats and sexual reproduction might not be crucial for the survival, transmission, and maintenance of *T. gondii* in a population (Worth, Lymbery, & Thompson, 2013). The overall evidence thus suggests that the apparent 'purposive design' of parasite-induced phenotypic alterations does not guarantee a causal relationship between manipulation and enhanced trophic transmission. More to the point, it is still unclear to what extent the consequences of host manipulation, in terms of enhanced completion of the parasite's life cycle, depends on its magnitude.

In addition, although most studies have considered a single phenotypic alteration at a time, it is increasingly acknowledged that, most often, manipulative parasites affect more than one phenotypic dimension in their hosts (Gotelli & Moore, 1992; Cézilly & Perrot-Minnot, 2005; Cézilly, Favrat, & Perrot-Minnot, 2013). Such multidimensionality might be adaptive if, for instance, it allows the parasite to enhance the completion of its life cycle under a large range of ecological circumstances (Thomas, Poulin, & Brodeur, 2010). Under this scenario, multidimensionality may have arisen from the progressive addition of several phenotypic dimensions that are manipulated independently of each other through distinct physiological pathways. Alternatively, multidimensionality in manipulation may stem from the major disruption of some specific physiological mechanism, with cascading effects affecting various phenotypic dimensions (Cézilly & Perrot-Minnot, 2010). For instance, crustacean amphipods infected with fish acanthocephalans show a variety of modified phenotypic traits (Cézilly, Favrat, & Perrot-Minnot, 2013), including an increased serotonergic activity in the brain (Tain, Perrot-Minnot, & Cezilly, 2006). Interestingly, multidimensionality in manipulation as observed in *G. pulex* infected with *P. laevis* can be partly mimicked in uninfected individuals by the injection of serotonin (Perrot-Minnot, Sanchez-Thirion, & Cézilly, 2014), thus providing support for the second hypothesis. To what extent this finding applies to other cases of multidimensionality in manipulation remains an open question. In addition, whether the existence of a single mechanism would result in co-variation among individuals in the magnitude of the various phenotypic alterations simultaneously brought about by a parasite species remains unclear (see Bailly, Cézilly, & Rigaud, 2018).

The interest in manipulative parasites is however not limited to their value as a supposedly perfect example of an extended phenotype. Growing attention has been given to the role that such parasites play in ecosystems through their influence on the behaviour and trophic niches of their hosts and, ultimately, on trophic cascades (Thomas *et al.*, 1997, 1998; Lefèvre *et al.*, 2009; Lafferty & Kuris, 2012; Sato *et al.*, 2012; Boze & Moore, 2014; Britton & Andreou, 2016; Reisinger & Lodge, 2016). Still, the precise impact of manipulative parasites on ecosystem dynamics remains unclear, partly because the relationship between the magnitude of phenotypic alterations and their ecological consequences is difficult to assess. More to the point, the ability of parasites to manipulate their hosts might be modulated by various environmental variables. For instance, temperature recently has been shown to affect the extent of manipulation of phototaxis in amphipods infected by acanthocephalans, but not that of geotaxis or refuge use (Labaude, Cezilly, & Rigaud, 2017a). Environmental influences and infection with manipulative parasites may thus have interactive or additive effects on the phenotype of infected hosts (see Labaude, Rigaud, & Cézilly, 2017b) and, therefore contribute directly to the observed variation in the magnitude of manipulation within and among host–parasite associations, with potential consequences at the level of ecosystems.

Whether host manipulation is studied from the point of view of its evolutionary routes (Thomas, Rigaud, & Brodeur, 2012), its underlying mechanisms (Perrot-Minnot & Cezilly, 2013) or its ecological consequences (Lafferty & Kuris, 2012; Labaude, Rigaud, & Cézilly, 2015b), an important question is what causes variation at different levels in the magnitude of phenotypic alterations coincidental with infection by manipulative parasites. Variation in the extent of such phenotypic alterations exists both within and among infected individuals in a single host population, as well as among host populations (Thomas *et al.*, 2011; Fayard, Cezilly, & Perrot-Minnot, 2019) or among host species infected with the same parasite (Gotelli & Moore, 1992; Bauer *et al.*, 2000; Tain, Perrot-Minnot, & Cezilly, 2007). The relative importance of host and parasite phylogenies, the type of altered trait or the consequences in terms of enhanced completion of the parasite's life cycle remain however poorly documented, although a few attempts have been made to provide quantitative reviews of the existing literature on host manipulation (Moore & Gotelli, 1990; Poulin, 1994, 2000; McElroy & de Buron, 2014; Nakagawa *et al.*, 2015). In the latter approach, meta-analysis constitutes a valuable tool (Poulin & Forbes, 2012), particularly to quantify the heterogeneity observed in the magnitude of host manipulation. So far, however, meta-analysis has seldom been used for that purpose. Using a meta-analytic framework, Poulin (1994, 2000) and Nakagawa *et al.* (2015) provided valuable insights on the influence of parasite taxa and behavioural traits on the magnitude of the effect of parasites on their hosts. Interestingly, based on 137 comparisons between the behaviour of infected and uninfected hosts, Poulin (2000) found that the reported values of effect size indicating host manipulation tended to decrease over time. He further suggested that this may be due to the fact that most of the earlier investigations of host manipulation concerned acanthocephalan parasites, in which the ability to manipulate host phenotype is regarded as an ancestral, well-established character (Moore, 1984b), whereas, later on, evidence for manipulation was sought in a larger range of host–parasite associations (Poulin, 2000). The same, although non-significant, trend for effect size becoming smaller over time was reported in an updated analysis based on 202 effect sizes (Nakagawa *et al.*, 2015). As acanthocephalans tend to have marked effects on their hosts (Bakker, Frommen, & Thuenken, 2017), it might have been difficult to obtain similar results in other parasites with a relatively smaller ability to manipulate their hosts (Nakagawa *et al.*, 2015). Another recent analysis, focusing on host performance (defined as a physical quantity that measures how well an organism can execute a given behaviour or task) and considering the literature published until 2013, failed to detect the same effect, but found some evidence for an increase in the magnitude of the effect of parasites on their hosts with publication year (McElroy & de Buron, 2014). However, the final data set in that study was based on only 49 studies.

The use of meta-analysis to analyse both the direction and magnitude of parasite-induced phenotypic alterations introduces several difficulties. First, not all published articles provide enough statistical information to allow the computation of effect sizes, such that final data sets available for meta-analysis might be of reduced size, thus increasing the risk of type II error (Arnqvist & Wooster, 1995). Second, there exists an unequal representation of the various species of hosts and parasites in the scientific literature on host manipulation, and this taxonomic bias is likely to result in non-random data sets (Lajeunesse, 2010). This is why it is highly recommended to incorporate phylogenetic information in ecological meta-analyses (Chamberlain *et al.*, 2012).

Here, we provide a meta-analysis of the existing literature about the phenotypic alterations induced by acanthocephalan parasites in their intermediate hosts. Although around 1300 species of acanthocephalan parasites have been described, their phylogenetic relationships within Metazoa remain controversial (García-Varela & León, 2015). Based on morphological, ecological and molecular evidence, acanthocephalan parasites have been divided into four classes: Archiacanthocephala, corresponding to the most basal clade, and Palaeacanthocephala, Eoacanthocephala, and Polyacanthocephala, corresponding to three derived clades (Amin, 1987; Kennedy, 2006; García-Varela & León, 2015). Although the evolution of acanthocephalans is characterized by a multiplicity of host-switching events (García-Varela & León, 2015), they tend to have strong and diversified effects on the phenotype of their hosts (Cézilly, Favrat, &

Perrot-Minnot, 2013; Bakker, Frommen, & Thuenken, 2017). Palaeacanthocephalans represent the most diverse and best-studied class of acanthocephalans, while published studies of host manipulation in other acanthocephalan groups are scarce (see Section II).

We reviewed phenotypic alterations induced by acanthocephalan parasites by considering two features: (*i*) the alteration of mean trait value, measured as the increase or decrease in a host phenotypic trait value expected to increase parasitic transmission in infected hosts; (*ii*) the magnitude of alterations, quantifying the influence of the parasite on the host's phenotype irrespective of its consequence on parasite transmission. For the former, we used signed effect sizes measured at the last developmental stage infective to the final host (cystacanth). For the latter, we also included effect size estimates from larval stages not yet infective to final hosts (acanthella). We first examined the extent to which the alteration of mean trait value and the magnitude of alteration was affected by phylogeny. We then quantified the effects of acanthocephalans on their host phenotype to answer three questions: (*i*) how strong is the overall effect of infection? (*ii*) Following the parasite manipulation hypothesis, are these alterations of host trait value likely to enhance parasite transmission on average? (*iii*) How variable is alteration of the trait value according to several factors including trait categories (multidimensionality) and publication year?

II. METHODS

(1) Literature search

Studies on acanthocephalan-induced phenotypic alterations were searched in the the *Web of Science* and *Google Scholar* databases by using combinations of "acanthocephala*" and "behav*" or "physio*" or "morpho*" or "size" or "chang*" or "host" or "predat*" or "reproduct*" or "survival" or "mortality" key words. The search included studies published until January 2018. From 3531 studies, and after sequential removals because of lack of sufficient quantitative information, we obtained a database of 81 studies (PRISMA flow diagram, Fig. 1). All studies included in analyses are identified with an asterisk in the reference list.

(2) Data collection

For each study, we recorded the year of publication, parasite taxonomy (from the class to the species) and stage (non-infective acanthella or infective cystacanth), intermediate

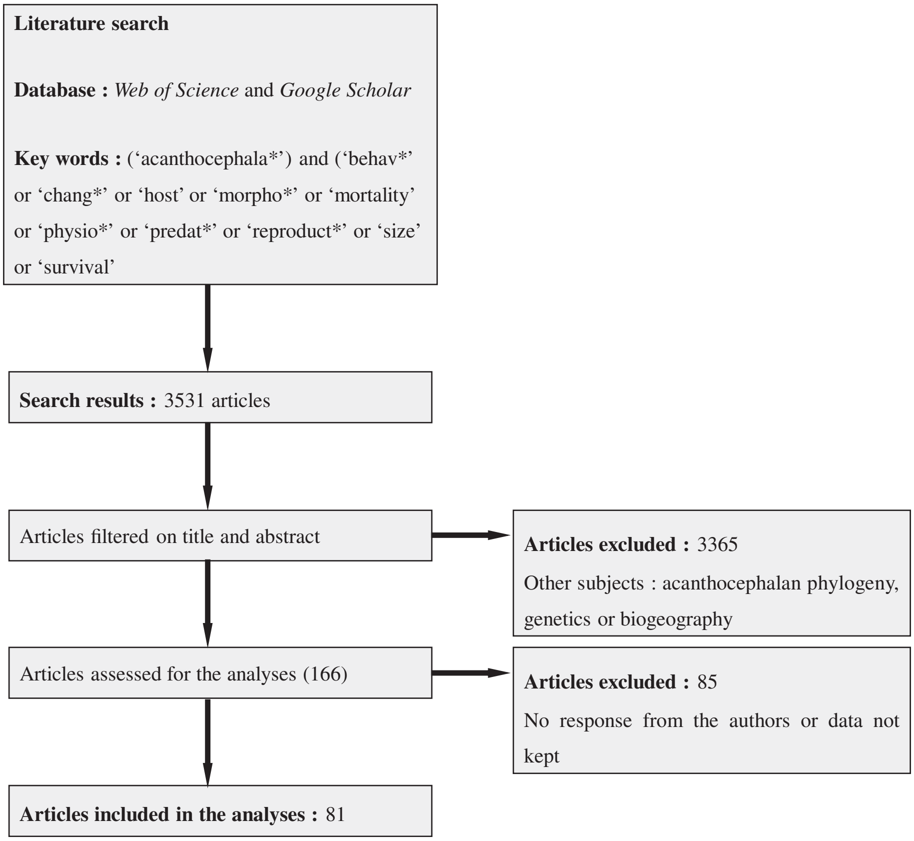

Fig. 1. Preferred reporting items for systematic reviews and meta-analyses (PRISMA) flow diagram (Liberati *et al.*, 2009; Nakagawa *et al.*, 2017) for this meta-analysis on variation in the intensity of host manipulation by acanthocephalans.

host taxonomy (class and species), phenotypic traits measured and the magnitude of alteration associated with infection (effect size), sample size (infected and uninfected individuals) and infection type (natural or experimental). Following the recommendations of Noble, Stenhouse, & Schwanz (2017), we sought for sources of non-independence stemming from within-study design, in addition to phylogeny-, species- and study-level non-independence. We identified two sources of within-study covariance: effect sizes estimated for different parasite species using the same controls ('shared treatment comparison' or 'shared controls'), and effect sizes measured on the same individual ('shared traits') (Noble, Stenhouse, & Schwanz, 2017).

(3) Categorization of host phenotypic traits

We categorized host phenotypic traits into five groups: 'behaviour', 'life history', 'morphology' and 'physiology' according to Mousseau & Roff (1987), and vulnerability to 'predation' (Table 1). We further subdivided each category into trait subcategories: for behaviour, 'activity', 'protection', 'response to stimuli' and 'taxis/phobia'; for life history, 'body condition', 'foraging', 'intraspecific interaction', 'reproduction' and 'survival'; for morphology, 'growth' and 'colour'. We subdivided the category physiology into 'immunity', 'energy reserves/metabolism', and 'neurophysiology'. Finally, within the trait category predation, we differentiated two types of predators, 'non-host' and 'suitable host'.

(4) Calculation of effect sizes

Following the recommendation of Nakagawa *et al.* (2017) for comparisons between two treatments (here, parasite infection and control), we used standardized effect sizes based on means and standard deviations, mostly with Cohens *d* (Cohen, 1988). In some cases, we also extracted this

Table 1. Categories of host trait altered by acanthocephalan parasites that were incorporated in the meta-analysis. The predicted direction of alteration under the hypothesis of increased trophic transmission of the infective cystacanth stage to definitive hosts is provided as the main hypotheses. The direction of alteration is predicted from either increased encounter rate between infected prey and predators (ERH: encounter rate hypothesis), or decreased energetic expenditure by intermediate host (ESH: energy-saving hypothesis). In the former case, parasite-induced phenotypic alteration (PIPA) results in predation bias towards infected hosts. Non-exclusively in the latter case, energy saving/reallocation increases host and parasite survival and/or parasite growth rate

Host trait category	Host trait subcategory	Main hypothesis	Competing hypothesis (opposite signed effect)
Behaviour	Activity	Higher activity increases conspicuousness; distance covered increases the probability of encounter (ERH)	Lower activity increases catchability (ERH) and saves energy (ESH)
	Protection	Decreased protective behaviour, increased exposure (ERH)	
	Response to stimuli	Decreased detection of stimulus or responsiveness to predator cues (ERH)	
	Taxis/phobia	Micro-habitat overlap with predators (decreased photophobia or negative geotaxis) (ERH)	
Life history	Body condition	Increased body condition (ERH and ESH)	
	Foraging	Higher exploration for resources (high food intake) increases prey exposure to predators (ERH)	
	Reproduction	Behavioural (male) and physiological (female) castration (ESH)	
	Intraspecific interaction (sociality)	Decreased agonistic behaviours (competition, cannibalism) (ESH)	
	Survival	Higher host survival increases the time frame for transmission (ERH and ESH)	
Morphology	Colour	Increased conspicuousness (ERH)	
	Growth	Higher growth/body size increases detection (ERH)	
Physiology	Immunity/resistance	Immunosuppression (ESH)	
	Energy reserves/metabolism	Higher energetic reserves (ERH and ESH) Lower metabolic rate (decreased oxidative stress: increased survival) (ERH and ESH)	Higher metabolic rate (sustains higher foraging and activity rate) (ERH and ESH)
	Neurophysiology	High serotonin level decreases anxiety (ERH) – associated with low dopamine level (5HT-DA opponency)	Low serotonin level impairs aversive learning, hence delays response to predation stimulus (ERH) – associated with high dopamine level (5HT-DA opponency)
Predation	Non-host	Decreased predation by non-hosts (ERH)	
	Suitable final host	Increased predation by suitable hosts (ERH)	

information from figures with the 'digitize' R package (Poisot *et al.*, 2016). When means and standard deviations were not available in the publication, we attempted to contact the authors directly. When proportions of individuals were given, we used the Odds ratio (Borenstein *et al.*, 2009). As not all studies reported the same effect size metrics, their direct comparison was not possible. We used conversions from Borenstein *et al.* (2009) to obtain a common metric of effect size, the correlation coefficient r, allowing comparison between studies. To conduct the analyses, we then converted each r into a Fisher Zr using the Fisher r to Z transformation (Borenstein *et al.*, 2009), with $Zr = 0.5 \times \ln((1 + r)/(1 - r))$. After the analyses, meta-analytic Zr means were back-transformed into meta-analytic r means. Values of 0.1, 0.3 and 0.5 were interpreted as low, moderate and strong effects, respectively (Cohen, 1988).

(5) Signed effect sizes according to parasite transmission

We assigned a sign to each effect size according to the direction of alteration, whether an increase or a decrease in mean trait value, that was expected to enhance trophic transmission. Positive values of effect sizes were associated with alterations in a host trait expected to enhance parasite transmission by increasing encounter rate between infected prey and predators (ERH: encounter rate hypothesis) (Table 1). When the effect of PIPA on parasite transmission did not directly affect encounter rate, positive values were assigned to alterations that would favour host survival independently of predation, for instance by decreasing host energetic expenditure (ESH: energy-saving hypothesis) (Table 1). The rationale is that parasite transmission relies on host survival until predation, in part modulated by energy reserves invested in host maintenance (traded-off against other functions) and parasite growth. Therefore, the fitness of trophically transmitted parasites relies partly on the survival of intermediate hosts until predation. In some cases, the direction of alteration for optimal parasite transmission was ambiguous, as either an increase or a decrease in the expression of one trait could contribute to increased parasite transmission (Table 1). We therefore ran alternative analyses without these ambiguous traits, following Cally, Stuart-Fox, & Holman (2019), and present these additional results as online Supporting Information.

(6) Choice of moderators

Five factors were considered as fixed effects.

(*a*) *Category of traits*

As parasite transmission depends critically on prey–predator interactions, the behaviour of infected intermediate hosts is of prime importance relative to other phenotypic traits. In addition, behavioural traits are more plastic than, for instance, morphological traits (Price, Qvarnström, & Irwin, 2003). One may therefore expect behavioural traits to be more easily altered by 'manipulative' parasites than morphological traits, resulting in differences in effect size between behavioural and morphological traits.

(*b*) *Infection type and environmental conditions*

One criticism of experimental studies on host manipulation by parasites is that laboratory conditions imperfectly reflect natural ones. Experimental infection procedure and maintenance conditions (light intensity, temperature, host density, stress of handling and maintenance) could impact the expression of phenotypic traits, and thus affect the estimates of effect size.

(*c*) *Parasite developmental stage*

Two different phenomena with opposite effects could alter the vulnerability to predation and survival probability of infected intermediate hosts (Parker *et al.*, 2009). 'Predation suppression' is used to refer to manipulations by immature parasites that decrease the vulnerability to predation of their intermediate host. Conversely, 'predation enhancement' is used to refer to manipulations that increase vulnerability to predation of the intermediate host at a developmental stage at which the parasite is infective to its final host. In acanthocephalans, acanthella are developmental stages at which the parasite is unable to establish in an appropriate final host, while the cystacanth is the last developmental stage in the intermediate host and is infective to final host. Dianne *et al.* (2011) found experimental evidence for both effects in *G. pulex* infected with *P. laevis*. Although opposite effects between acanthella and cystacanth infections have been reported several times, it is not clear whether they are of the same magnitude.

(*d*) *Publication year*

Host manipulation by parasites has been actively investigated in the field of host–parasite interactions since the study of Holmes (1972) pointed out the adaptive value of manipulation. Several criticisms of this hypothesis and alternative explanations emerged almost 20 years later from the review of Moore & Gotelli (1990). The approach used to study a phenomenon can change according to current paradigms, and this may lead to different conclusions (Poulin, 2000). As a consequence of growing interest in the topic, and methodological and technical progress, it is possible that trends in magnitude of acanthocephalan-induced alterations reported in the literature could appear through time.

(*e*) *Sample size*

Confidence intervals vary with sample size, being larger for small sample sizes. Therefore, we included the effect of sample size as a source of heterogeneity among effect sizes, estimated here using within-study sample size.

(7) Meta-analyses

We ran multi-level/hierarchical models with the MCMCglmm (Markov chain Monte-Carlo general linear mixed models) function in the MCMCglmm package (Hadfield, 2010), to investigate several types of non-independence. The first is widespread in evolutionary biology, and stems from phylogenetic relatedness among species (Harvey & Pagel, 1991; Nakagawa & Santos, 2012). To control for the potential non-independence of species data points, we implemented phylogenetic information as a variance–covariance matrix in the meta-analyses. As the most recent phylogenetic tree based on 18S rRNA gene sequences comprises only 36 acanthocephalan species (Verweyen, Klimpel, & Palm, 2011), we constructed a new tree based on 59 species (including three new species sequences) (see online Supporting information, Table S1). We retrieved the distances between species from an ultrametric tree derived from Bayesian inference (see Table S1). In addition to phylogenetic non-independence between effect sizes, we accounted for species- and study-level non-independence by including parasite species and study ID as random factors (Nakagawa & Santos, 2012). Finally, we explored the consequences of violating assumptions of independence among effect size estimates at the individual level ('shared-measures' and 'shared-controls') by running a sensitivity analysis, following the recommendations of several authors (Koricheva & Gurevitch, 2014; Noble, Stenhouse, & Schwanz, 2017). Shared measures are effect sizes estimated for different traits in the same individuals, shared controls are effect sizes estimated for at least two parasite species using the same control (uninfected) group. We assessed the robustness of the meta-analysis on signed effects of cystacanth infection after controlling for these sources of non-independence, by running the same analysis on a subset of independent measures (Fig. S1B): (*i*) only one effect size was randomly chosen per individual whenever more than one trait was measured per individual within the same trait category or in two categories besides behaviour, (*ii*) when one or more behavioural traits were measured together with morphological, physiological or life-history traits on the same individual, we removed the behavioural trait(s) as this category was over-represented in the data set. This was a conservative approach, since behaviour was expected to be the trait category that was most impacted by infection.

Effect sizes (Zr) were used as the dependent variables and their variance was calculated using the formula: $1/(n - 3)$ (Borenstein *et al.*, 2009), where n is the sample size associated with each effect size. The analyses were based on Bayesian hierarchical models which impose definitions of priors (Gelman, 2006). A prior is the strength of belief in the parameter value associated with the variable affecting the observed data. It is represented by the distribution of the parameter based on previous experience. In the absence of information on prior distribution, we used non-informative priors ($nu = 0.002$ and $V = 1$). To assess the influence of these priors on the results, we repeated the analyses with expanded priors ($nu = 1$, $V = 1$, $alpha.mu = 0$, $alpha.V = 1000$), with no detectable effect on our results. For each model, we ran 500000 iterations, which was large enough to minimize the level of autocorrelation (non-independence) between successive iterations; we checked that the autocorrelation coefficient was below 0.10, as suggested by J.D. Hadfield (personal communication). Model convergence was verified according to Gelman & Rubin (1992). To evaluate the reliability of the meta-analytic mean, we also assessed consistency among studies by calculating I^2, which quantifies heterogeneity between effect sizes for each random factor (Nakagawa & Santos, 2012). I^2 represents the variance accounted for by each random factor relative to the total variance. Heterogeneity was considered as low, moderate and high when $I^2 = 0.25$, 0.50 and 0.75, respectively (Higgins *et al.*, 2003).

First, we performed a meta-analysis using signed effect sizes of cystacanth infection, to quantify overall alteration in mean trait value. We also estimated the average magnitude of alterations by estimating the meta-analytic mean of absolute effect sizes on the complete data set, including effect sizes of infection with acanthella. We could not run the meta-analysis directly on absolute values of effect sizes, as the distribution of absolute effect sizes is a folded normal distribution (Morrissey, 2016a). Therefore, we used the procedure recommended by Morrissey (2016a, 2016b), specifically the 'analyze-then-transform' approach. We first estimated the meta-analytic mean of all signed effect sizes (all infections with acanthella and cystacanth), and then derived the mean absolute value; we applied the formulae provided by Morrissey (2016a) to convert both the posterior mean and confidence interval. The different analysis and their purposes are summarized in Fig. S2.

(8) Meta-regressions

We ran a meta-regression to assess the contribution of fixed effects to variation in signed effect sizes of cystacanth infection. The category of trait, infection type (natural or experimental), sample size and year of publication were entered as fixed factors, and parasite species, study and parasite phylogeny as random factors within the model. In the analysis on the entire data set including both acanthella and cystacanth infection to derive the average magnitude of alterations (Fig. S2), parasite developmental stage was added as an additional fixed factor. Since parasite species was already taken into account as a random factor, and was associated with host species (Fig. S1A), neither the host nor the parasite species were considered as fixed factors. We chose to keep these as random factors to control for non-independence between effect sizes. Starting with a global model (including all fixed effects), we performed model selection with the MuMIn package (Bartoń, 2016) using the deviance information criterion (DIC) (Spiegelhalter *et al.*, 2002; Grueber *et al.*, 2011). For each factor level, the meta-analytic mean was estimated from the meta-regression.

(9) Analysis of parasite maturity

The aim of this analysis was to test whether the average magnitude and direction of parasite-induced phenotypic alterations varies according to whether parasite developmental stage is, or is not yet, infective to the final host (Fig. S2). First, we assessed to what extent parasite maturity could affect the overall meta-analytic mean of signed effect sizes, by comparing the output of two analyses, the main analysis based the data set restricted to the cystacanth stage, and the additional analysis based on the entire data set (acanthella and cystacanth stages) (Fig. S2). We expected the meta-analytic mean of signed effect sizes to be positive and larger when considering cystacanth infection only compared to both developmental stages. In addition, as for the overall mean absolute value, we derived the mean absolute values of effect sizes and their confidence intervals for each factor level, including parasite developmental stage. We used the 'analyze-then-transform' approach on the meta-analytic mean effect size of each factor level estimated from the meta-regression on signed effect sizes (both acanthella and cystacanth included).

(10) Publication bias

Statistically significant results are much more likely to be published than non-significant ones (Rosenthal, 1979). In addition, when published, studies reporting non-significant results tend to be based on large sample sizes, which is expected to increase their leverage on the meta-analytic mean. We identified potential publication biases using funnel plot (Sterne & Egger, 2001). We quantified the magnitude of these publication biases using both Egger's regression (Egger *et al.*, 1997) and trim-and-fill (Duval & Tweedie, 2000) methods. We estimated the number of missing studies using both L0 and R0 estimators (Duval & Tweedie, 2000). In order to remain conservative, we reported the estimator giving the largest number of missing studies. The associated correction was then applied to the first meta-analytic mean to see if the missing studies would have affected it significantly (Møller & Jennions, 2001; Rothstein, Sutton, & Borenstein, 2005).

All analyses were run using R software (version 3.4.3, R Core Team, 2018).

III. RESULTS

The full data set comprises 279 effect sizes obtained from 81 studies (Fig. 1), conducted on 13 species of acanthocephalan parasites (Table 2A), and 20 host species belonging to three orders of Crustacea and one order of Insecta (Fig. S1A). Our data set was composed of two phylogenetically different acanthocephalan classes: Archiacanthocephala and Palaeacanthocephala. Although these two classes were not equally represented (11 and 89%, respectively), we retained both in order to maximize statistical power given the variability in effect size estimates. The fish parasite *Pomphorhynchus laevis* accounted for 33% of the total number of effect size estimates, whereas estimates obtained for *Acanthocephalus anguillae*, *Hexaglandula corynosoma* and *Pseudocorynosoma constrictum* accounted for only 2.5% in total (Table 2A, Fig. S1A). Among the different trait categories, behaviour was the most frequently recorded trait, accounting for about 49% of all effect size estimates, whereas morphological traits represented only 9.7% (Table 2B, Fig. S1B). Effect size estimates of vulnerability to predation represented only 5.4% of the data set (Table 2B; Fig. S1B). Most effect sizes were estimated on intermediate hosts infected with the cystacanth stage (261 out of 279) as compared to acanthella (18).

Most effect size values were retrieved from studies reporting more than one estimate (93.5% of the overall data set), justifying the incorporation of study as a random factor in the model. Additionally, more than half the data set (58.8% of effect size values) comprised shared measures (55.9% of effect size values), with very few cases of shared controls (2.9%) (Fig. S1B). Shared measures were found in all trait categories except predation.

(1) Meta-analytic means and phylogenetic inertia

To incorporate phylogenetic information in the meta-analysis, we first estimated phylogenetic relationships among 59 acanthocephalan species using Bayesian inference of nuclear 18S rDNA sequences. The phylogeny was well resolved (Fig. 2), and our tree topology matches those published previously [see Verweyen, Klimpel, & Palm, 2011, and references therein]. We confirmed paraphyly of the orders Echinorhynchida and Polymorphida within the most diversified class Palaeacanthocephala, in agreement with Verweyen, Klimpel, & Palm (2011) but with a larger data set (59 species instead of 29 species) (Fig. 2).

Overall, heterogeneity due to phylogenetic inertia (I^2) accounted for about 12–13% of the variation in signed effect sizes of infection with the cystacanth stage only (Table 3). Incorporating the signed effect sizes of infection with acanthella slightly increased this phylogenetic signal to 19% of overall variation (Table S2A). The meta-analytic mean effect size of infection with the cystacanth stage was significantly positive (0.28 [0.05–0.49], with phylogenetic correction) (Fig. 3). However when incorporating the signed effect sizes of infection with acanthella (entire data set including cystacanth and acanthella infection), the meta-analytic mean was no longer significant (0.23 [−0.19–0.57], with phylogenetic correction) (Fig. S3). Finally, the average magnitude of alteration induced by acanthocephalan infection independently of parasite transmission (absolute mean value) was moderate to large (0.40 [0.34–0.60]) (Fig. 4).

We ran another analysis on a subset of 230 signed effect size estimates, after removing ambiguous phenotypic traits with respect to whether an increase or a decrease would enhance parasite transmission in cystacanth-infected hosts. In this analysis, the meta-analytic mean remained significant (0.32 [0.05–0.52]) (Fig. S4).

Table 2. Number of studies and number of effect size estimates including both cystacanth and acanthella infection stages (with number of effect sizes for acanthella infections alone shown in parentheses) included in our data set for (A) acanthocephalan parasite species and (B) categories and subcategories of host phenotypic traits

A

Parasite class	Parasite species	Number of studies	Number of effect sizes (acanthella only)
Archiacanthocephala	*Moniliformis moniliformis*	8	26 (0)
	Oncicola venezuelensis	2	5 (0)
Palaeacanthocephala	*Acanthocephalus anguillae*	1	2 (0)
	Acanthocephalus dirus	18	29 (2)
	Acanthocephalus lucii	12	25 (1)
	Echinorhynchus truttae	6	9 (0)
	Hexaglandula corynosoma	1	2 (0)
	Leptorhynchoides thecatus	1	8 (0)
	Plagiorhynchus cylindraceus	3	7 (0)
	Polymorphus minutus	27	56 (4)
	Pomphorhynchus laevis	39	92 (10)
	Pomphorhynchus tereticollis	7	15 (0)
	Pseudocorynosoma constrictum	2	3 (1)
TOTAL			279 (18)

B

Trait category	**Trait subcategory**	**Number of studies**	**Number of effect sizes (acanthella only)**
Behaviour	Activity	17	19 (1)
	Protection	15	23 (2)
	Response to stimuli	22	43 (4)
	Taxis/phobia	26	53 (2)
Life history	Body condition	4	4 (0)
	Foraging	4	8 (1)
	Reproduction	10	25 (3)
	Intraspecific interaction	1	1 (0)
	Survival	5	6 (1)
Morphology	Colour	5	7 (0)
	Growth	14	20 (3)
Physiology	Immunity/resistance	8	27 (0)
	Energy reserves/metabolism	12	19 (0)
	Neurophysiology	5	9 (0)
Predation	Suitable final host	10	13 (0)
	Non-host	2	2 (0)
TOTAL			279 (18)

Finally, to account for non-independence among effect size estimates caused by shared measures and shared controls, we ran a sensitivity analysis on a subset of 143 independent effect size estimates: the meta-analytic mean of infection with cystacanths was still significant (0.31 [0.01–0.52]) (Fig. S5).

(2) Meta-regressions

To assess whether PIPAs enhance parasite transmission, we focused the meta-regression analysis on signed effect sizes including the cystacanth stage only (Fig. S2). The meta-regression revealed that trait category and sample size were the first factors driving variation in the magnitude of effect sizes, regardless of the incorporation of ambiguous traits (Fig. 3; Table 3), or not (Fig. S4; Table S2B). Specifically, two behavioural traits (response to stimuli and taxis/phobia), one life-history trait (reproduction), one morphological trait (colour), one physiological trait (immunity), and the vulnerability to predation of suitable final hosts, were significantly and positively affected by infection with cystacanths (Fig. 3). Here also, heterogeneity arising from random effects was consistent across models, regardless of the inclusion of fixed effects and their total number in the analyses (Table 3). Among the random effects, study ID accounted for about 32% of heterogeneity in signed effect sizes, whereas parasite species accounted for only 7% (Table 3).

The average magnitude of alteration, estimated for each factor as the mean absolute effect size, was comparable between developmental stages. In addition, behavioural and life-history traits seemed to be the most strongly affected (Fig. 4).

(3) Publication bias

Based on Egger's regression, there was no significant evidence for a publication bias (intercept = 0.05, 95% CI = −0.02–0.11). This was further confirmed by the trim-

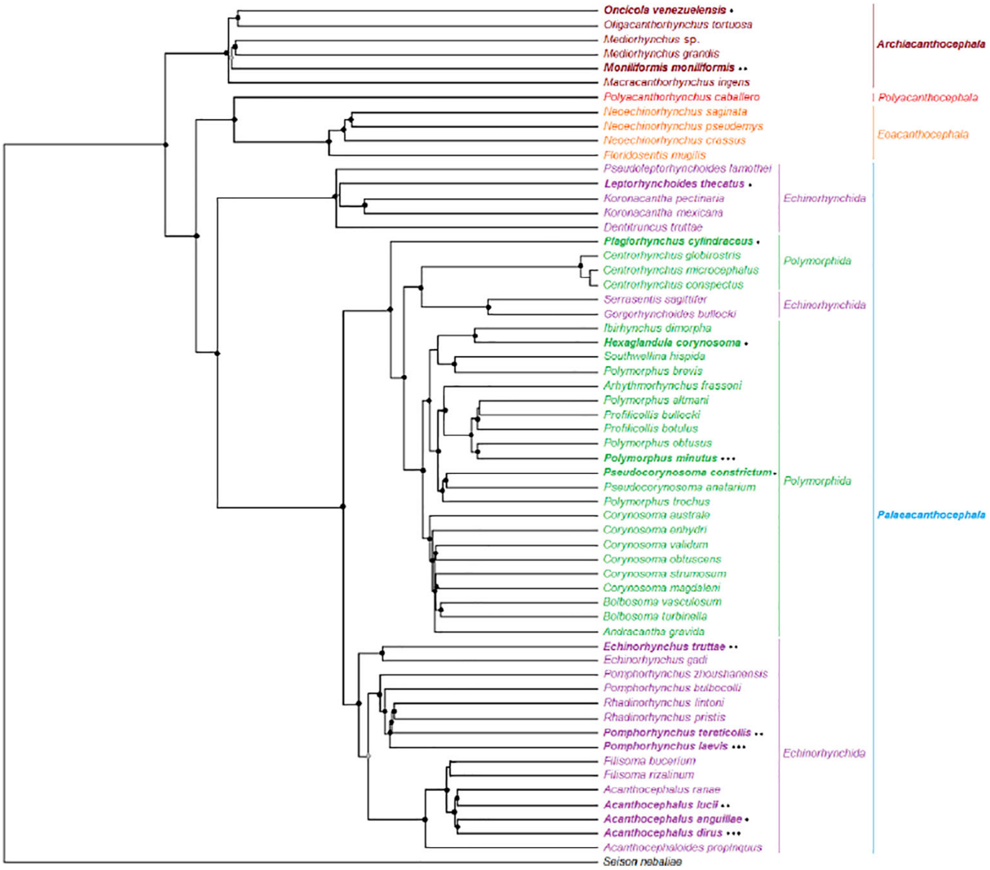

Fig. 2. Phylogenetic reconstruction of the phylum Acanthocephala based on 18S rRNA sequences from 59 species, and one outgroup species of Rotifera (in black), using Bayesian MCMC algorithms with MrBayes. Species included in the meta-analyses belong to the classes Archiacanthocephala (in brown, two species) and Palaeacanthocephala (in blue, 11 species). Species identified with one, two or three asterisks are represented in the data set by less than 5, between 5 and 15, or more than 15 estimates, respectively. Black and grey dots represent values of posterior probabilities higher than 0.90 and 0.80, respectively.

and-fill analysis. Although 48 effect size estimates were likely to be missing on the left side of the funnel plot (Fig. 5), the funnel plot was almost symmetrical and the correction of −0.09 from the trim-and-fill did not alter the meta-analytic mean significantly (0.26, 95% CI = 0.03–0.45).

IV. DISCUSSION

The aim of our study was to undertake a critical review on host manipulation by acanthocephalan parasites, in the framework of phylogenetic meta-analysis. We believe the

Table 3. Composition of the meta-analytic models run to explain variation in signed effect sizes of infection with cystacanths only. The corresponding deviance information criterion (DIC), and heterogeneity (I^2) arising from the random factors (study, parasite species and parasite phylogenetic distance) are provided. The best model, according to the lowest DIC, is shown in bold type

			Heterogeneity I^2 (%) (random effects)		
Model	Moderators (fixed effects)	DIC	Parasite species	Study	Parasite phylogenetic distance
1	Intercept only	215.36	6.55	31.18	11.84
2	Category of traits	208.20	6.45	32.78	13.60
3	Infection type	215.77	6.61	30.56	13.12
4	Publiation year	216	7.05	30.38	12.39
5	Sample size	209.80	6.40	32.70	11.86
6	**Category of traits + sample size**	**202.99**	**6.42**	**34.74**	**13.20**
7	category of traits + infection type + publication year + sample size	204.68	6.71	34.62	14.04

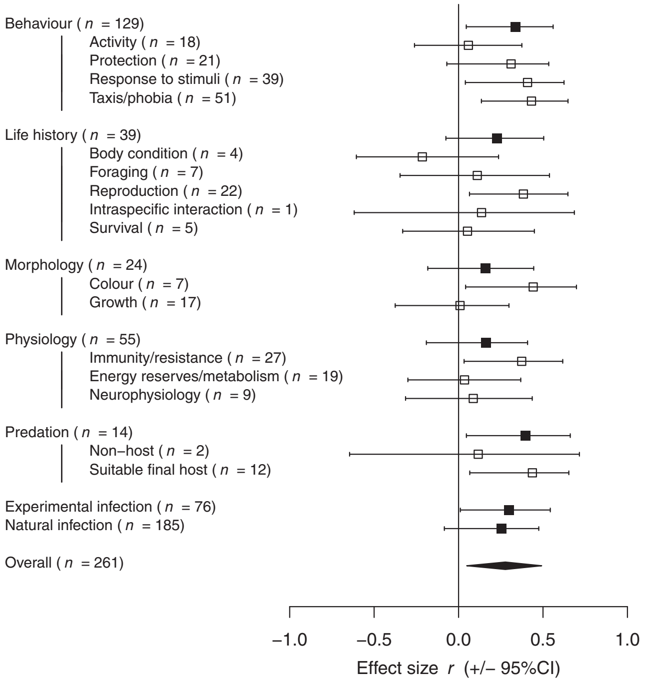

Fig. 3. Forest plots of the global meta-analytic mean of signed effect sizes (overall) based on cystacanth-induced alterations in host phenotype, and the meta-analytic mean for each moderator (categories and subcategories of traits and type of infection). Positive effect sizes represent infection-induced alterations of trait value expected to increase trophic transmission, whereas negative effect sizes represent infection-induced alterations expected to decrease trophic transmission. *n*, sample size.

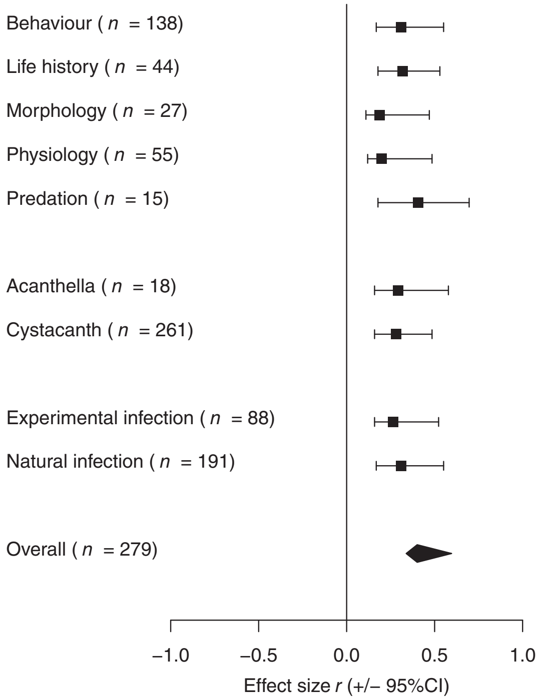

Fig. 4. Forest plots of the global meta-analytic mean of absolute effect sizes (overall), and the meta-analytic mean for each moderator (categories of traits, parasite maturity and type of infection) representing the magnitude of alterations induced by infection with acanthocephalans (both acanthella and cystacanth) on host phenotype. *n*, sample size.

originality and strength of our analysis lies in several features. First, we ran a phylogenetically corrected meta-analysis using a more exhaustive and multidimensional data set ($N = 279$ estimates of effect on a wide range of phenotypic traits) than in previous meta-analyses on the impact of parasites, which focused on the magnitude of behavioural alterations (Nakagawa *et al.*, 2015: $N = 202$, including 92 effect sizes from nine acanthocephalan species), on body condition (Sánchez, 2018; $N = 553$), on the relationship between infection and social status in vertebrates (Habig *et al.*, 2018, $N = 128$), or on the relationship between infection and group size (Patterson & Ruckstuhl, 2013; $N = 70$). A key feature of our study is that it incorporated all phenotypic traits reported in order to (*i*) broaden our understanding of multidimensionality in manipulation by acanthocephalans, and (*ii*) avoid potential bias resulting from inclusion only of behavioural traits [as in previous studies (Poulin, 1994, 2000; Nakagawa *et al.*, 2015)], given that they are more likely to be involved in parasite transmission. Second, we quantified the overall effect of these multiple phenotypic alterations induced by acanthocephalans on parasite transmission by assigning a benefit in terms of increased encounter rate with the final host or decreased energetic expenditure by the intermediate host. We also considered the magnitude of alterations independently from increased parasite transmission. Third, we addressed whether the effect size of infection differs according to trait category and parasite stage, as a way to address how fine-tuned PIPAs are.

(1) How strong is the general effect of infection, independent of parasite phylogeny?

We found little evidence for a phylogenetic signal. Relatedness between acanthocephalan species accounted for only a small proportion of the heterogeneity of all effect size estimates. The negligible effect of phylogenetic distance suggests that Acanthocephala is a homogeneous taxon in terms of phenotypic alterations induced in intermediate hosts. This conclusion must however be made with caution, as the class Archiacanthocephala is under-represented in the data set.

Overall, acanthocephalan parasites induce low to moderate alterations in their host phenotype, a result in agreement with Poulin, Brodeur, & Moore (1994). The phylogenetic mean ranges from $r = 0.23$ to 0.40, depending on correction for phylogeny and on the use of absolute or signed effect sizes.

The meta-regression analysis on signed effect sizes revealed no effect of the type of infection (experimental or natural) on overall intensity of manipulation (Fig. 4). We can therefore confidently rely on results from experimental infections in studies investigating the role of parasite stage

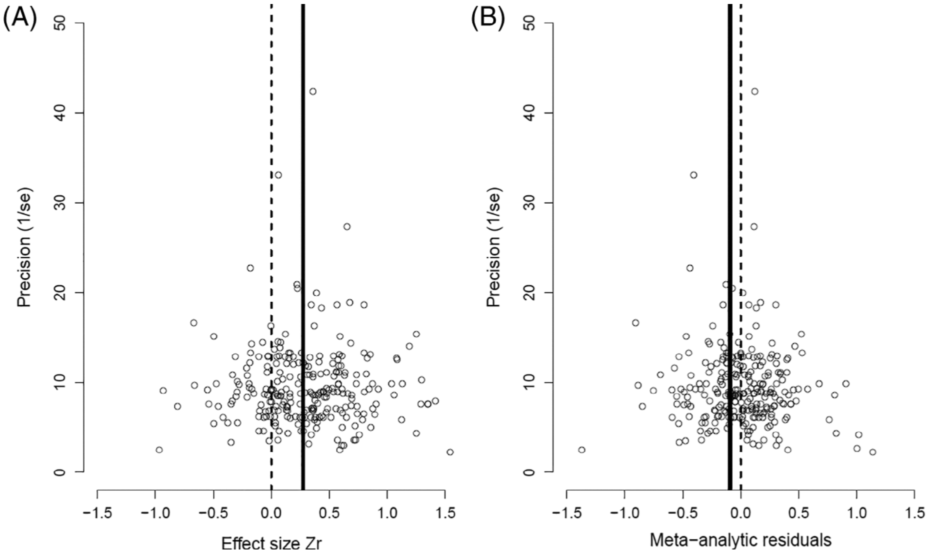

Fig. 5. Funnel plots of (A) original data points (effect size estimates) and (B) residuals from model 1 (Table 3), plotted against precision (the inverse of standard error). Bold lines represent the meta-analytic mean in A and the correction (calculated using the trim-and-fill method) in B.

(Dianne *et al.*, 2011), parasite age (Franceschi *et al.*, 2008), parasite and host populations (Franceschi *et al.*, 2010b), abiotic factors (Labaude, Cezilly, & Rigaud, 2017a), and biotic factors (no study to date, but see Fayard, Cezilly, & Perrot-Minnot, 2019) in modulating the intensity of PIPAs. In addition, the intensity of PIPAs decreased with increasing sample size. This highlights the importance of the number of replicates within a study in estimating the magnitude of PIPA.

Among random factors, the meta-regression analysis on signed effect size revealed a negligible effect of parasite species on overall intensity of manipulation, but a more important effect of study. This study effect highlights the possible impact of differences in experimental designs in estimating the type and magnitude of PIPAs.

(2) Is there evidence for adaptive manipulation? Trophic transmission and parasite stage

For the mature parasite stage (cystacanth only), acanthocephalans do induce a moderate increase in traits affecting parasite transmission to the definitive host. However, there are still too few studies quantifying trophic transmission (only 5.4% of the cystacanth data set), in comparison to those reporting PIPAs. In addition, even fewer studies have actually attempted to estimate the contribution of a given altered trait to trophic transmission (Kaldonski *et al.*, 2009; Perrot-Minnot *et al.*, 2012; Jacquin *et al.*, 2014). This limitation should not be overlooked when reviewing evidence for adaptive manipulation.

Another line of evidence for adaptive manipulation is a trend for reversed parasite-induced alterations in the intermediate host between parasite developmental stages, predicted theoretically to enhance parasite infectivity to the final host (Parker *et al.*, 2009). The average magnitude of alterations induced by infection with acanthella appears to be comparable to that induced by infection with the cystacanth stage, but in the opposite direction (Fig. 4; Fig. S3). This suggests that the acanthella stage could have a real and opposite impact on host phenotype compared to the cystacanth stage, in ways that are likely to decrease the vulnerability to predation of the infected intermediate host (Parker *et al.*, 2009; Dianne *et al.*, 2011). This result must be considered with caution, however, given the low number of effect size estimates for the acanthella stage ($n = 18$) compared to the cystacanth stage ($n = 261$), and the low number of parasite species for which estimates were available (five). In addition, half of these effect sizes were estimated for behavioural traits (taxis/phobia, protection, and response to stimuli), which might lead to overestimated differences between acanthella infection and cystacanth infection. Indeed, while acanthella and cystacanths are theoretically likely to have opposing effects in terms of behavioural alterations (Parker *et al.*, 2009), energy-saving strategies, such as physiological or behavioural castration or immunosuppression, are expected to be shared by both parasite stages to some extent. However, while immunosuppression may allow energy conservation, it may also compromise the survival of infected hosts in response to other pathogens (Cornet *et al.*, 2009). Therefore, immunosuppression might represent a more costly strategy for the acanthella than for the cystacanth stage, given the longer developmental time required to reach the stage infective for the final host (Crompton & Nickol, 1985). Unfortunately, there have been no studies that quantify acanthella-infected host immunocompetence. Finally, there is only mixed evidence in support of an energy-saving strategy by depressing host reproduction at the acanthella stage (Bailly, Cézilly, & Rigaud, 2018).

(3) Is there evidence for multidimensional alterations?

Overall, all trait categories were impacted by cystacanth infection. Behaviour was the trait category that was most significantly impacted, and hence is the category expected to contribute the most to acanthocephalan transmission (Fig. 3). Taxis/phobia was the most strongly impacted subcategory, followed by response to stimuli. If reversed taxis can drive alterations in microhabitat preferences through alterations in reactions to light, gravity, air or water velocity, or substrate, the observed pattern is likely to increase the encounter rate of the cystacanth with final hosts. These findings confirm that altering the host's microhabitat preference is an important feature of manipulation by acanthocephalans compared with other trophically transmitted parasites, whereas activity is not significantly affected (Lafferty & Shaw, 2013). Changes in responses to stimuli are also expected to modulate the encounter rate of infected prey and the final hosts. More surprising is the non-significant effect of cystacanth infection on protection behaviour (i.e. on exposure). However, we included studies that scored protection/exposure behaviour under simulated predation threat in the 'response to stimuli' subcategory, meaning that those in the 'protection/exposure' subcategory of behaviour reported alterations in protective behaviour solely in the absence of predation risk. The mechanisms by which acanthocephalans alter these context-dependent traits may thus rely on stimulus perception/response, rather than on avoidance or defensive behaviour itself.

Among physiological and life-history-related traits, only host immunity and reproduction were significantly impacted by infection with cystacanth stages (Fig. 3). We interpret immunosuppression and castration as part of an energy-saving strategy to support both host maintenance and parasite growth, thereby increasing host survival and indirectly, parasite transmission. Alternatively and non-exclusively, alterations in host reproductive and immune system traits could be linked to behavioural alterations, and thereby to parasite transmission. The immune and nervous systems are connected through several different pathways in animals (Dantzer *et al.*, 2008; Adamo, 2013). Neurological functions can be modulated by immune factors such as cytokines by means of specific neuronal receptors. Cytokines released by the immune system act as signalling molecules to the central nervous system, and can result in sickness behaviour: a set of physiological and behavioural alterations that promote the survival of infected individuals (Dantzer, 2004; Dantzer & Kelley, 2007). Adamo (2013) postulated that if parasites could alter the amount or the type of cytokines released by the host immune system, then this could result in abnormal behaviour. Although highly interesting, this neuropsychoimmune hypothesis has not yet been addressed.

(4) Recommendations for future research

Our findings highlights several ways to improve our understanding of the adaptive significance of host manipulation. First, for future meta-analysis, researchers should attempt to increase the power and functionality of the metrics used to quantify phenotypic alterations. This could be achieved by increasing sample size, and by reporting effect sizes rather than statistical metrics. Indeed, 85 studies had to be excluded (Fig. 1) from the present analysis because suitable data were not provided or were no longer available. Second, as a consequence of the historical focus on behavioural trait alterations expected to increase trophic transmission of the infective stage, traits not directly related to predator–prey interactions have received little attention in acanthocephalans (Cézilly & Perrot-Minnot, 2010), including phenotypic alterations induced by acanthella. Yet, these remain crucial to developing a better understanding of whether PIPAs constitute a 'manipulation syndrome', and whether the adaptive value of PIPAs extends to developmental stages not infective to the final host (protective manipulation). Third, studies quantifying actual trophic transmission are still rare (Poulin & Maure, 2015). This is likely due to the fact that designing studies to quantify trophic transmission raises practical challenges, in particular under field conditions, as either prey choice or the diet of final hosts needs to be analysed [see Cézilly *et al.* (2010), for a recent review]. The study of proximate mechanisms, in particular the neuropsychoimmune hypothesis of parasite manipulation, also requires attention (Poulin & Maure, 2015). Finally, taxonomic bias may arise from focusing on only a small set of model species (Poulin & Maure, 2015). In our data set, the most diverse and derived class Palaeacanthocephala was over-represented, while the more ancient class Archiacanthocephala was represented by only two species (*Moniliformis moniliformis* and *Oncicola venezuelensis*). This prevented a detailed comparison between these two classes, for example to investigate whether host manipulation increases over evolutionary time.

Finally, another stimulating area in the study of parasite manipulation from an evolutionary point of view is to investigate not only the magnitude of parasite manipulation (changes in trait means) but also alterations in trait variability. Behavioural variability is predicted to decrease in infected hosts, making them more susceptible to predators as part of the manipulation strategy (Nakagawa *et al.*, 2015). Alternatively, behavioural variability in infected hosts could increase as a consequence of parasite-induced disruption of regulatory pathways controlling behaviour. To our knowledge, only one meta-analytic study has quantified the effect size of infection on behavioural variability and they failed to find a significant effect (Nakagawa *et al.*, 2015). However, their study was not restricted to acanthocephalans, and it remains possible that other taxa of parasites could respond differently, both in mean host traits and also their variance.

V. CONCLUSIONS

(1) Overall, infection with acanthocephalans induces low to moderate phenotypic alterations in their hosts.

The magnitude of alterations induced by the infective stage was highest for behavioural traits related to microhabitat choice and response to stimuli, and for immunity and reproduction. Although a trend for opposite effects of infection with acanthella was detected, a thorough analysis of the 'predation suppression then predation enhancement' strategy is still limited by the lack of data at the acanthella stage of development. Furthermore, testing for publication bias showed that 48 data points were lacking, corresponding to negative effects (opposing parasite-induced transmission facilitation), although no significant publication bias was detected overall. Future studies should be careful not to censor negative evidence for the host manipulation hypothesis.

(2) Multidimensionality of parasite manipulation was indicated in the significant effect of infection on all trait categories. Questions remain regarding the links between behavioural, life-history, and physiological traits. For instance, testing of the neuropsychoimmune hypothesis has so far been restricted to establishing correlations between phenotypic responses (phototaxis and immunity) in few acanthocephalan species (Cornet *et al.*, 2009). Although informative from an ecological point of view, this is not a powerful mechanistic approach since the absence of a phenotypic correlation does not prove the existence of independent modulation of these traits. Manipulating the level of immunocompetence, and monitoring any resulting alterations in levels of brain neuromodulators, neurogenesis or neuronal apoptosis, would be a more promising way to decipher the interrelationships between the immune and neural systems, and any consequences on behaviour.

(3) Although we were able to detect low to moderate increases in traits promoting parasite transmission to the definitive host, there are still too few studies that actually quantify trophic transmission. Even fewer have attempted to understand the relationship between multidimensional phenotypic alterations and parasite transmission success (discussed in Cézilly & Perrot-Minnot, 2010; Thomas, Poulin, & Brodeur, 2010).

(4) To allow comparison of effect sizes between trait categories, we combined traits that were functionally comparable from an ecological viewpoint. The criteria used here to assign phenotypic traits to different categories may be more broadly applicable to a wide range of host species. As a theoretical approach to host manipulation by parasites is relevant across a diverse range of taxonomic groups (Thomas, Rigaud, & Brodeur, 2012; Lafferty & Shaw, 2013), our method may be applicable to many other parasites engaged in host manipulation.

(5) The past 10 years has seen a decreasing number of empirical studies relative to theoretical analyses and reviews, creating an "imbalance between facts and ideas" (Poulin & Maure, 2015). This review provides quantitative evidence that the fascinating phenomenon of host manipulation has solid theoretical and empirical foundations, but also raises challenging questions about the underlying proximate and ultimate mechanisms that call for broader methodological and taxonomic coverage.

VI. ACKNOWLEDGMENTS

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VII. AUTHOR CONTRIBUTIONS

M.F. and M.-J.P.-M. designed project goals, trait categorization, and collected the data. M.F. and F.-X.D.-M. ran the meta-analysis, R.W. and M.-J.P.-M. collected and analysed molecular data for phylogenetic reconstruction, and drafted the relevant section. M. F., F.-X.D.-M and M.-J.P.-M. drafted the manuscript. All authors revised the manuscript.

VIII. DATA ACCESSIBILITY STATEMENT

Data and R script for the meta-analysis are available in the Mendeley repository (DOI: 10.17632/k5y4g5nfwy.2).

IX. REFERENCES

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X. Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Table S1. List of acanthocephalan species included in the phylogenetic analysis, and the outgroup species (*Seison nebaliae*, Rotifera), with their accession numbers.

Fig. S1. Number of effect size estimates retrieved from published studies on the impact of acanthocephalan infection on their intermediate invertebrate hosts.

Appendix S1. Phylogenetic relationship of acanthocephalans based on 18S ribosomal gene sequences.

Fig. S2. Road map of the analyses.

Table S2. Composition of the meta-analytic models in the complementary analysis.

Fig. S3. Parasite-induced alterations of mean trait value, incorporating all parasite developmental stages: forest plot from the complementary analysis with global meta-analytic mean of signed effect sizes (overall), and the meta-analytic mean for each moderator (categories and subcategories of traits, parasite maturity and type of infection).

Fig. S4. Ambiguous traits: forest plot with global meta-analytic mean of signed effect sizes (overall), and the meta-analytic mean for each moderator (categories and subcategories of traits and type of infection).

Fig. S5. Sensitivity analysis to account for the non-independence of effect sizes: forest plot with global meta-analytic mean of signed effect sizes (overall), and the meta-analytic mean for each moderator (categories of traits and type of infection).

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