International Journal for Parasitology: Parasites and Wildlife 2 (2013) 190–196

Contents lists available at SciVerse ScienceDirect

International Journal for Parasitology: Parasites and Wildlife

journal homepage: www.elsevier.com/locate/ijppaw

# What drives population-level effects of parasites? Meta-analysis meets life-history

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ARTICLE INFO

*Article history:*
Received 1 February 2013
Revised 7 May 2013
Accepted 8 May 2013

*Keywords:*
Cost of parasites
Lifespan
Meta-regression
Virulence

ABSTRACT

Parasites are considered drivers of population regulation in some species; unfortunately the research leading to this hypothesis has all been conducted on managed populations. Still unclear is whether parasites have population-level effects in truly wild populations and what life-history traits drive observed virulence. A meta-analysis of 38 data sets where parasite loads were altered on non-domesticated, free-ranging wild vertebrate hosts (31 birds, 6 mammals, 1 fish) was conducted and found a strong negative effect of parasites at the population-level ($g = 0.49$). Among different categories of response variables measured, parasites significantly affected clutch size, hatching success, young produced, and survival, but not overall breeding success. A meta-regression of effect sizes and life-history traits thought to determine parasite virulence indicate that average host life span may be the single most important driver for understanding the effects of parasites. Further studies, especially of long-lived hosts, are necessary to prove this hypothesis.

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## 1. Introduction

A central goal of population ecology is to identify factors controlling population dynamics. In wild populations, predation and competition are well studied, with some theoretical and empirical investigations focusing on the effects of parasites. Population regulation by parasites has been identified in Red Grouse *Lagopus lagopus scoticus* (Hudson et al., 1998), Svalbard Reindeer *Rangifer tarandus platyrhnchus* (Albon et al., 2002) and Soay Sheep *Ovis aries* (Gulland, 1992); unfortunately, these examples represent managed populations, and therefore may not reflect true effects of parasites on wild populations. Thus, the question remains—are parasites significant drivers of population-level effects and what host life-history traits drive observed virulence (*sensu lato* Casadevall and Pirofski, 1999—the capacity of a parasite to cause damage to a host)?

The modern view of parasitism is predicated on the assumption that 'every parasitic organism. . . imposes a cost on its host' because resources, however slight, are being diverted from host to parasite (Combes, 2005). These costs can be couched in two evolutionary trajectories: (1) the 'mutual aggression model, (Holmes, 1983) which suggests that parasites evolve to be as virulent as possible, and thus are a primary regulatory force; and (2) the 'prudent parasite model' (Holmes, 1983; Renaud and de Meeüs, 1991) which suggests that parasites evolve towards a balance between short- and long-term needs conferring a range of benefits to the infected host that may or may not offset the costs (Michalakis et al., 1992; Schmidt-Hempel, 2003).

Several researchers have argued that the only way to assess the true effects of parasites is by altering the parasite population of the host *in situ* (Møller, 2005). Alterations of parasite loads are easy to effect in domestic and laboratory animals, and even wild animals in the laboratory (Diamond, 1983; McCallum, 1995). However, relatively little parasite work on wild, free-ranging hosts incorporates this technique due to logistical difficulties surrounding field work and obtaining sufficient sample sizes to detect differences between infected and non-infected hosts. Therefore, much ecological work on the effects of parasites ends up being correlative (Poulin, 2007a). It is unclear if the differences detected between parasitized and non-parasitised hosts are due to indirect effects or pre-existing differences (i.e., prior to infection; Bize et al., 2008 or host-quality; Lailvaux and Kasumovic, 2011). Field experimentation is necessary to quantify actual costs of parasites on hosts due to the many problems associated with extrapolating laboratory results on individuals or populations to real effects in the field (Seitz and Ratte, 1991).

In order to understand if parasites are truly a driver of host populations, reviews of the effects of parasites to wild hosts need to be conducted. Reviews to date of both observational and

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http://dx.doi.org/10.1016/j.ijppaw.2013.05.001

experimental work on the cost of parasites to wild hosts (birds, mammals, fish and insects: Lehmann, 1993; birds: Møller, 1997; birds and mammals: Tompkins and Begon, 1999; mammals: Irvine, 2006) have implied that parasites are costly, but the implications of that cost are unreliable, due to the methods used to synthesize results (Stewart, 2010). A recent meta-analytical synthesis of parasite induced mortality of nestlings showed an overall small effect (12% mean parasite-induced mortality, range 0–89%, $n = 117$), with parasite-induced mortality determined by latitude, nesting site, probability of host survival and parasite prevalence (Møller et al., 2009). However, this meta-analysis only considered studies of nestling birds and may be fundamentally flawed because it includes observational data as well as experimental data (Borenstein et al., 2009).

The objectives of the present analyses were to review quantitatively experimental studies of wild, free-ranging hosts that measure parasite-induced changes in population-level traits (i.e. measures of fecundity and mortality); then, to evaluate this effect of parasites using life-history traits. Based on those host life-history traits that Møller et al. (2009) found to be significant, the following predictions are made: (1) cavity-nesting species (includes burrowing mammals as well as hollow nesting birds) will experience increased parasite density and intensity and thus more virulent effects than ground or open nesting species (Ewald, 1983); (2) colonial species will experience increased parasite density and intensity and thus more virulent effects than less gregarious species (Ewald, 1983); (3) tropical species will encounter more virulent parasites than temperate species because the absence of seasonality maintains higher parasite abundance (Møller, 1998); and (4) higher virulence will evolve in hosts with shorter life-spans because of the fewer opportunities there are for dispersal to a new host in search of a mate, and as a consequence, the parasites become more virulent (Lehmann, 1993; Nidelet et al., 2009).

## 2. Materials and methods

### 2.1. Data collection and inclusion criteria

The studies considered for use in the meta-analysis were obtained from a survey of the primary literature. The initial search was directed using reviews by Møller (1997), Newton (1998), Tompkins and Begon (1999) and Irvine (2006) followed by a comprehensive search of ISI Web of Science and Google Scholar up to and including January 2012. The following search terms and their combinations were used: "parasite*", "experiment*", "manipulation", "cost*", "effect*", "mortality", "survival", "fitness", "host*", "life-history". Older literature (pre-1985) was identified through Literature Cited sections of recent papers and unpublished theses (the same search terms were used in ProQuest Dissertations and Theses, Theses Canada and Trove). Only papers written in English were included. When reference to unpublished work was encountered, attempts were made to solicit raw data from the author(s). A large number of studies were screened using abstracts only (<2000); 89 full-text articles were assessed for eligibility. Of these, 51 were excluded due to a lack of numerical data, lack of sample size and/or variance, untranslatable test statistics, duplication of dataset from a previous paper or reported results not relevant to the selection criteria (e.g. behavioural or physiological/individual responses). Studies were selected if (a) host species were wild (not domesticated), free-ranging (not held in captivity) and the study was conducted under field-conditions (not laboratory conditions); (b) parasite species were experimentally manipulated (increased or decreased); and (c) the parasite was naturally occurring and not introduced, thus avoiding the 'suicide king' issue of parasites infecting hosts outside their normal range and becoming more virulent in the process (Dybdahl and Storfer, 2003). Of these, a study was included in the final meta-analyses if it provided (a) the means and standard errors or standard deviations (or any other statistic whereby means and standard errors could be derived) of at least one population-level parameter measuring the cost of parasitism for experimental and control groups, and (b) the sample sizes associated with the means.

### 2.2. Response variables and calculation of effect sizes

Response variable and effect size data were extracted from the text and tables for all studies except Cheney and Côté (2003), Fitze et al. (2004), Pap et al. (2005), Slomczyński et al. (2006) (additional information requested and received from the authors); and Bize et al. (2004) and Hillegass et al. (2010) (data extracted from graphs using DataThief; Tummers, 2006). Statistics were converted to effect sizes in the form of Hedges' $g$ (Hedges and Olkin, 1985) in the program Comprehensive Meta-analysis (CMA; Borenstein, 2006). Hedges' $g$ was chosen as the effect size over the more commonly used Cohen's $d$ because Hedges' $g$ pools variance using $n - 1$ instead of n and thus provides a better estimate for smaller sample sizes (Grissom and Kim, 2005). Studies that reported only $F$ statistics (Møller, 2002; Vandegrift et al., 2008) were not converted to effect sizes due to issues surrounding the overestimation of effect sizes identified by Hullett and Levine (2003) and lack of accurate sample size data in the respective articles. The type of response variable was coded into the data set to enable subgroup analyses. The response variables used were: clutch size (number of eggs in the clutch), percent hatching success (percentage of eggs that hatched from a single clutch), number of young produced (total brood size), percent breeding success (percentage of young produced, fledged or survived during the study period), and survival rate (survival during the study period or between one breeding season and the next).

### 2.3. Meta-analytic procedures

All meta-analyses were performed in CMA (Borenstein, 2006). A random-effects model was used for all tests because variability was expected in the effects being measured across different species and hosts. Many articles included multiple effect sizes from different measures of the effects of parasitism, so rather than combining all the effect sizes within a study (which may have obfuscated the true effect), in the overall meta-analysis one effect size was chosen at random from each of the forty-three studies (Gurevitch and Hedges, 1999). Separate random-effects meta-analyses were conducted grouped by effect being studied—so any given study might have data in several meta-analyses (sub-analyses) thus maintaining the independence of the data (Gurevitch and Hedges, 1999). One study (Roby et al., 1992) considered the responses of two host species to the same anti-parasitic treatment, so the two hosts were considered as independent studies.

### 2.4. Heterogeneity and publication bias

Publication bias, or the 'file drawer problem', where non-significant results are relegated to the file drawer rather than to the published literature (Rosenberg, 2005), is an ongoing issue affecting meta-analyses, leading to bias via the selective publication of statistically significant results (Hedges and Olkin, 1985). To guard against this issue, publication bias was assessed using three methods: funnel plot (plot of effect size and precision to search for asymmetry), $Q$-rank correlation (a test for publication bias; Begg and Mazumdar, 1994), and trim-and-fill (Duval and Tweedie, 2000). Heterogeneity indicates the presence of effect-modifiers, and the $Q$-test for heterogeneity was calculated for the overall

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