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Indirect effects of parasitism: costs of infection to other individuals can be greater than direct costs borne by the host

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Parasitic infection has a direct physiological cost to hosts but may also alter how hosts interact with other individuals in their environment. Such indirect effects may alter both host fitness and the fitness of other individuals in the host's social network, yet the relative impact of direct and indirect effects of infection are rarely quantified. During reproduction, a host's social environment includes family members who may be in conflict over resource allocation. In such situations, infection may alter how resources are allocated, thereby redistributing the costs of parasitism between individuals. Here, we experimentally reduce parasite burdens of parent and/or nestling European shags (*Phalacrocorax aristotelis*) infected with *Contracaecum* nematodes in a factorial design, then simultaneously measure the impact of an individual's infection on all family members. We found no direct effect of infection on parent or offspring traits but indirect effects were detected in all group members, with both immediate effects (mass change and survival) and longer-term effects (timing of parents' subsequent breeding). Our results show that parasite infection can have a major impact on individuals other than the host, suggesting that the effect of parasites on population processes may be greater than previously thought.

1. Introduction

Parasite infections impose a number of direct costs on their hosts that can limit resources available for other processes important to survival and reproduction [1]. There is increasing recognition that infection can also alter the way that hosts interact and share resources with other individuals in their social environment [2,3]. This can lead to additional, indirect costs of infection for individuals with which the host interacts, for example by altering host success in competitive interactions or influencing how hosts use or contribute to group resources [2–6]. The impact of both direct and indirect effects of parasitism are likely to become particularly acute during periods of reproduction, when adult and juvenile hosts are under additional nutritional stress and relatives may share limited resources. Optimal levels of resource allocation are likely to differ between family members; for example, in species with parental care, offspring may seek a greater share than is optimal for parents to provide as they balance investment in their offspring with self-maintenance and future reproductive attempts. Levels of allocation are influenced by a combination of parental provisioning decisions, offspring signals of need and the outcome of competitive interactions between siblings [7]. The costs of parasitism at this time may therefore have a substantial impact on social dynamics by altering how resources are partitioned between group members [8,9]. While social interactions are known

to play a major role in the spread of infection [10] and can influence host and non-host responses to infection in experimental settings [4], the relative impact of direct and indirect effects of parasitism on host traits in wild populations remains unclear.

The potential consequences of direct and indirect effects of parasitism may also persist across an individual's lifetime. Infection could have cumulative costs across breeding events, impairing future survival or breeding performance [11,12]. Alternatively, parasitism could alter a host's trade-off between current and future reproductive effort [13]: an infected parent may strategically reduce its investment in current reproduction to preserve its residual reproductive value [14] or increase it as a mechanism to ameliorate the effects of parasitism on the current breeding attempt [15]. Thus, the full influence of infection may not be captured by considering only its immediate consequences. Failure to account for both direct and indirect effects of infection, immediately and in the longer term, is therefore likely to underestimate the effect of parasitism on hosts' life-history decisions, performance of both hosts and non-hosts, and hence population processes.

Recent theoretical and empirical work has highlighted the importance of both parent and offspring phenotype in determining the outcome of resource distribution within the family [16]. Therefore, both parent and offspring responses to infection are likely to influence the impact of infection on any individual family member. There is considerable evidence that the infection status of parents can influence offspring growth and survival [2,9,17]. However, far fewer studies have examined how offspring infection affects other family members. Notable exceptions suggest that parasite infection in young can decrease parents' future breeding success [12] via mechanisms such as increasing parents' feeding effort [18], but many of these findings stem from studies of host–ectoparasite systems, where host-switching between family members is an essential part of the parasite's life cycle [19]. Effects observed in non-treated individuals may therefore in part be a direct effect of an associated change in their parasite load, if treatment causes parasites to redistribute themselves among the host group [12].

Teasing apart the direct and indirect effects of different family members' infections is further complicated by an expected correlation in parasite load between family members. Parents and offspring are likely to have similar levels of parasite exposure owing to their shared environment and potential to act as infection sources for other family members [12,19]. Family members may also have comparable levels of immune defence because of their shared genetic background [20] and maternal transfer of antibodies to offspring [21]. Parental and offspring traits that govern how resources are distributed among the family are also likely to be coadapted [16], making within-family comparisons essential to understanding the relative impact of parasitism across the family unit. A powerful approach to investigate the relative roles of direct and indirect effects of parasitism in wild populations would therefore be to simultaneously manipulate the parasite load of different family members independently in a factorial design in a system where parasites cannot redistribute themselves between hosts. However, to our knowledge, the family-wide impact of parasitism has not yet been examined in a single experimental framework.

Here, we examine the impact of both direct physiological effects of infection on hosts and indirect effects on other individuals in the family unit across consecutive breeding seasons. We use the European shag, *Phalacrocorax aristotelis*, a seabird that is commonly infected through its fish diet by gastrointestinal nematodes [22–24], which are discretely distributed between hosts. Prevalence of nematodes in our study population is high [24] and infection has direct effects on parents and nestlings, particularly late in the breeding season and when breeding conditions are poor [8,25,26]. To assess the family-wide effect of parasitism, we treated parents and/or chicks with an anti-helminthic drug in a fully factorial experimental design. We measured the effects of treatment not only directly on the treated generation but also indirectly on all other family members, including longer-term effects beyond the contact period between parents and offspring.

2. Material and methods

(a) Study system

This study was conducted on the individually marked breeding population of shags on the Isle of May National Nature Reserve in southeast Scotland (56°11 N, 2°33 W) in 2011 and 2012. Shags are piscivorous seabirds infected through the fish they eat by larval gastrointestinal nematodes, predominantly *Contracaecum rudolphii*, which attach to the shags' stomach wall and become reproductively mature [22,23]. All adults and chicks over 10 days of age that have been sampled in this population are infected (68 adults endoscoped and 33 dead chicks dissected [24,27]). There is no known mechanism by which chicks can infect parents, and direct transmission of adult worms from parents to chicks does not appear to drive the establishment of infection in chicks [27], although parents act as vectors of larval worms to chicks via the regurgitated food they provide.

Treatment of shags with 1% w/v ivermectin (Panomec©, Merial, UK), a broad-spectrum anti-helminthic, reduces the number of worm eggs passed in faeces in chicks, removes worms from adult shags for at least three weeks at a high dose, and reduces costs associated with infection [24–26]. Treatment can increase chick growth, with a stronger effect in later-hatched siblings; it can increase chick survival and parental foraging, with greater effects on sons and mothers, respectively; and can increase breeding success, with a greater effect on birds breeding later in the season [8,24,25]. Shags' modal clutch size is three eggs, which hatch asynchronously creating a size hierarchy across the brood (the 'A' chick hatches first, 'B' within 24 h and 'C' *ca* 2 days later [28]), although siblings do not differ in nematode prevalence at age 10 days, when our treatment was administered [8]. Males are 22% heavier than females as adults and grow faster during the linear growth phase between the ages of 8 and 30 days [29]. The earliest breeders can lay in March and the latest in July, and earlier laying is associated with greater breeding success [28,30] and lower nematode burden in adults [24].

(b) Anti-parasite treatment experiment

We measured the direct and indirect effects of parasitism in all family members by treating parents and/or offspring with Panomec© in the 2011 breeding season and comparing their performance to equivalent sham-treated controls. Parents and/or offspring were treated in a two-by-two factorial design, which gave four treatment groups: parents control/chicks control, parents control/chicks drug-treated, parents drug-treated/chicks control and parents drug-treated/chicks drug-treated. Both parents were treated in the parent treatment and all chicks were treated in the chick treatment.

Three-egg nests were randomly assigned to treatment groups at laying. Groups were matched for lay date and clutch size. At 3–7 days prior to predicted hatching, both parents at each

Table 1. Sample sizes and hatch dates (median and inter-quartile range) for each treatment group used in the analysis. All nests had three eggs at the start of the experiment. Not all parents could be recaught to measure mass change, and some chicks died after the first weight measure at treatment so growth could not be calculated. Hence, not all manipulated nests were represented in all analyses. Final sample sizes were: for parent mass change, 106 parents in 58 nests; for chick survival measures, 189 eggs in 63 nests; for chick growth, 134 chicks in 59 nests; for subsequent parent breeding, 105 breeders from 60 initial nests, with hatch date available for 92 individuals in 55 nests.

	parent treatment	
chick treatment	control	drug-treated
monitored during breeding season		
control	17 nests	15 nests
	36 chicks, 31 adults	34 chicks, 26 adults
	14 May (12 May – 16 May)	18 May (14 May – 23 May)
drug-treated	14 nests	14 nests
	32 chicks, 23 adults	32 chicks, 26 adults
	19 May (14 May – 15 May)	18 May (12 May – 24 May)
failed before treatment	1 nest	2 nests
	0 chicks or adults	0 chicks or adults
adults that returned to breed		
control	30	27
drug-treated	24	24

study nest were caught, weighed and measured, and injected intramuscularly with either ivermectin or a saline control at a dose of 0.7 mg kg^{-1}. All individuals not already carrying a British Trust for Ornithology metal ring and field-readable Darvic ring were marked in this way as part of the long-term study on the island. Nests were visited daily to obtain accurate hatching dates for all chicks. Hatchlings were blood sampled for molecular sexing [31] and marked individually. When the oldest chick was 10–12 days old, all chicks in the brood were weighed and injected subcutaneously with 0.05 ml (mean 1.8 mg kg^{-1}) of either ivermectin or saline. Differences between siblings in mass at this point were too small to allow dose adjustments in relation to mass, but we have previously shown that individual chick responses to treatment are driven by rank rather than mass at treatment [8,26]. Chicks were subsequently weighed at ages 15, 22, 28 and 35 days old (all ± 1 day) and survival was recorded. Parents were caught and weighed at the end of the experimental period (chick age 30–35 days). Overwinter survival of parents was determined by examining whether individuals were resighted on the Isle of May in future breeding seasons (overall annual summer resighting probability under routine long-term monitoring is more than 95%; unpublished data from Isle of May long-term study, 2008 to 2014) and breeding dispersal is negligible in this population [32].

In the breeding season following the experiment (2012, henceforth 'subsequent' year), we recorded three aspects of reproduction of all parents from our four experimental groups: whether breeding was attempted, hatch date (by observation or calculated from chick wing length at ringing around age 20 days, a reliable indicator of chick age), and breeding success measured as the number of chicks fledged. Testing for longer-term effects on chicks was beyond the scope of this study as most shags do not recruit until aged at least 3 years [33].

In total, we manipulated 71 nests, but excluded one nest with related parents, three that were second clutches, and three with hatch dates more than 10 days after the latest nest in the main hatch date distribution (range 31 days) that had spuriously strong statistical leverage. We also excluded one nest where only one parent could be caught for ivermectin treatment, but retained two nests where only one parent could be caught for control treatment as previous studies have found no difference between unmanipulated and sham-treated controls [8,25]. These exclusions did not qualitatively change our main results. Final sample sizes are shown in table 1.

(c) Statistical analysis

We considered the effects of both parent and chick treatments on all family members. Immediate treatment effects on parents (i.e. the effect in the same breeding season as dosing occurred) were measured as change in mass over the experimental period. Longer-term treatment effects were measured as parents' overwinter survival, whether breeding was attempted in the subsequent year, shift in hatch date (measured as the absolute shift in hatch date from the experimental year, relative to the median in each year) and breeding success in the subsequent year (number of chicks fledged, including zero values for individuals who did not breed). Chicks' immediate responses to treatment were measured as growth rate (calculated by fitting a linear regression through the four masses during the linear growth phase) and survival to fledging from three stages: parent treatment (before hatching), hatching and chick treatment (aged 10–12 days). Survival from parent treatment reflects effects on offspring hatching success as well as post-hatching survival, but the effects of chick sex and rank, which were assigned at hatching, could only be assessed using post-hatching survival. For all response variables, parameter estimates are presented ± 1 s.e.

We used backwards stepwise model selection, beginning with a maximal model including all candidate main effects and interactions and eliminating the least significant effect in turn, removing all non-significant interactions before removing main effects. In all response variables, we tested for effects of parent and chick treatment as independent main effects, interacting with each other, and each interacting with traits previously found to affect shags' responses to infection (hatch date, sex and chick rank (A, B or C) [8,24–26]). Treatment effects were tested with factors known to influence each response and

treatment interactions with these variables: for chick survival, hatch date and chick rank [25,30,34]; for chick growth, chick rank and sex [8,29]; for parent mass change, sex to account for sexual size dimorphism; and for subsequent timing of breeding, sex to allow for differences between males and females in over-winter behaviour and previous hatch date to account for individual repeatability in phenology [35,36]. Interactions of chick and parent treatments with these variables were examined in separate models to limit the number of terms; all models included main effects of both treatments and an interaction between them (see the electronic supplementary material).

All analysis was conducted in R v. 2.15.1 [37] with packages nlme [38] and lme4 [39], fitting nest as a random factor to account for non-independence of siblings and of parent pairs. Parental mass change, chick growth and subsequent hatch date shift were modelled as continuous Gaussian responses; chick survival, over-wintering parent survival and whether parents attempted subsequent breeding as binary responses with binomial errors and a logit link; and number of chicks fledged with Poisson errors and a log link. Because of limited variation in these binary and Poisson variables, we fitted hatch date as a two-level categorical variable (early, i.e. hatched on or before the median hatch date, or late, i.e. hatched after the median) when modelling these responses.

3. Results

(a) Direct effects of parent treatment

We found no detectable effect of parent treatment on their mass change or overwinter survival, either overall or varying with hatch date, sex or chick treatment (all parent treatment terms dropped during model selection at $p > 0.1$; minimal models in table 2, model 1; model selection for all response variables in electronic supplementary material). Parent treatment also had no effect on their subsequent breeding probability, timing or success (all parent treatment terms dropped during model selection at $p > 0.2$; minimal models in table 2, models 2–4).

(b) Direct effects of chick treatment

Similarly, we found no direct effect of chick treatment on chick survival, either overall or interacting with chick sex, rank or parent treatment (all chick treatment terms dropped during model selection at $p > 0.1$; minimal models in table 2, model 5c), though mortality after chick treatment was low overall (11 deaths, 134 survivors). Chick treatment had a marginal but non-significant effect on chick mass change (growth rate), irrespective of sex, rank or parent treatment (in minimal model, treatment effect -1.3 ± 0.7 g d^{-1}, $t = -1.83$, $p = 0.073$; table 2, model 6). An illustration of all responses across the four treatment groups is given in electronic supplementary material, figure S1.

(c) Indirect effects of parent treatment

Treatment of parents had no overall effect on chick survival from the point of treatment; however, parent treatment affected chick survival differently in early and late nests (hatch date × parent treatment interaction: effect size 2.1 ± 0.9 (not back-transformed), $z = -2.42$, $p = 0.016$; table 2, model 5*a*). For parents that bred before the median hatch date, treatment slightly increased chick survival, but after the median, parent treatment decreased chick survival (figure 1).

Last-hatched siblings had lower survival than A and B chicks (mean survival probability from hatch: A chicks, $85 \pm 4\%$ of 63 chicks; B chicks, $84 \pm 5\%$ of 62 chicks, C chicks, $67 \pm 7\%$ of 42 chicks; difference between A and C chicks, $z = -2.66$, $p = 0.008$), but neither chick rank nor sex influenced responses to parent treatment (interactions dropped at $p > 0.3$; table 2, model 5*b*).

Parent treatment did not affect their chicks' mass change (all parent treatment terms dropped at $p > 0.2$; table 2, model 6).

(d) Indirect effects of chick treatment

Anti-helminthic treatment of chicks had a significant impact on their parents' mass change. Mirroring the indirect effects of parent treatment on chick survival, opposite effects were found in early and late breeders (chick treatment × hatch date term in minimal model: effect size -8.7 ± 3.6 g, $t = -2.81$, $p = 0.018$; table 2, model 1). In earlier nests, parents of treated chicks gained weight compared with controls, but in later nests, parents of treated chicks lost weight (figure 2). Mothers and fathers did not differ in this relationship, nor did parents' own treatment change the way they responded to chick treatment (all parent treatment terms dropped at $p > 0.1$).

While chick treatment did not affect parents' overwinter survival or likelihood of breeding in the subsequent year (all chick treatment effects dropped at $p > 0.4$; table 2, models 2 and 4), parents of drug-treated chicks bred almost a week earlier than the previous year compared with parents of control chicks, with a marginally greater effect in fathers (in model selection, chick treatment × parent sex term: effect size -5.6 ± 2.8 days, $t = -2.01$, $p = 0.052$, table 1, model 3). Removing this interaction term demonstrated a persistent influence of chick treatment on parents' subsequent hatch date (chick treatment main effect: -6.04 ± 2.1 days, $F_{1,53} = 8.80$, $p = 0.005$; figure 3). In contrast to the more immediate indirect effects of parasitism, chick treatment affected subsequent breeding in the same way for early and late experimental parents (chick treatment by hatch date interaction dropped from model at $p = 0.270$; figure 3). Subsequent breeding success declined through the season overall (hatch date main effect on number of chicks fledged, effect size (not back-transformed) -0.4 ± 0.2, $z = -2.68$, $p = 0.007$) but was not affected by chick treatment (main effect and interaction dropped at $p > 0.5$; table 2, model 4).

4. Discussion

Our study highlights that the indirect effects of parasitism on individuals in a population may be as important as the direct physiological costs of infection experienced by a host. To our knowledge, this is the first time that both the direct and indirect consequences of parasitism have been simultaneously investigated for different family members in a wild population of naturally infected animals where it is possible to isolate such effects. Using experimental reduction of gastrointestinal nematodes in families of shags, we could not detect any strong direct effects of infection in parents or offspring in the current year, nor for parents in the subsequent breeding season. However, indirect effects were detected, both in terms of the consequences of a parent's infection for their offspring and the consequences of the offspring's infection for their parents. Moreover, there were both immediate indirect effects in the year of parasite removal and long-term indirect effects that persisted to affect subsequent breeding events. Our results indicate that the full influence of parasitism

Table 2. Minimal models explaining variation in all response variables tested. Parents' overwinter survival was best explained by an intercept-only model that is not presented here. Otherwise, models are presented and numbered in the order they appear in the results. Test statistics are *t*-values for continuous response variables (parents' mass change and subsequent breeding timing and chick growth rate) and *z*-values for binary and Poisson response variables (subsequent breeding attempted, breeding success and chick survival). Effect sizes are given in the following terms: for hatch date, the gradient of its relationship with the response variable; for categorical hatch date, late birds compared to late breeders; for sex, males compared to females; for treatment, ivermectin-treated birds compared to control birds; and for rank, B and C chicks (as indicated in the table) compared to A chicks. For binary and Poisson variables, effect sizes are not back-transformed from the link function.

model and terms	effect size	test statistic	*p*
(1) parents' mass change (g)			
intercept	−396.1 ± 390.5	−1.01	0.315
sex	68 ± 22.4	3.04	0.004
hatch date in 2011	2.7 ± 2.9	0.93	0.358
chick treatment	1176.1 ± 492.6	2.39	0.021
hatch date × chick treatment	−8.7 ± 3.6	−2.43	0.018
(2) subsequent breeding attempted			
intercept	1.9 ± 0.8	2.35	0.019
sex	1.8 ± 0.8	2.27	0.023
(3) subsequent breeding timing (hatch date shift 2011–2012)			
intercept	39.7 ± 20.5	1.93	0.059
hatch date	−0.3 ± 0.2	−2.11	0.039
parent sex	5.5 ± 1.8	3.05	0.004
chick treatment	−2.1 ± 2.7	−0.76	0.451
chick treatment × parent sex	−6.0 ± 2.9	−2.06	0.047
(4) subsequent breeding success			
intercept	0.6 ± 0.1	6.80	<0.001
hatch date (categ.)	−0.4 ± 0.2	−2.68	0.007
(5*a*) chick survival from parent treatment			
intercept	1 ± 0.4	2.91	0.004
hatch date (categ.)	0 ± 0.6	0.03	0.975
parent treatment	1.3 ± 0.7	1.97	0.049
hatch date × parent treatment	−2.1 ± 0.9	−2.42	0.016
(5*b*) chick survival from hatching			
intercept	2.5 ± 0.8	3.21	0.001
hatch date (categ.)	0 ± 0.8	0.05	0.961
rank (B)	−0.2 ± 0.6	−0.30	0.764
rank (C)	−1.8 ± 0.7	−2.66	0.008
parent treatment	2.5 ± 1.2	2.05	0.040
hatch date × parent treatment	−3.6 ± 1.5	−2.43	0.015
(5*c*) chick survival from chick treatment			
intercept	6.4 ± 2.4	2.64	0.008
hatch date (categ.)	−2.8 ± 1.4	−2.06	0.040
rank (B)	−1.1 ± 1.2	−0.94	0.348
rank (C)	−3.6 ± 1.5	−2.36	0.018
(6) chick growth rate (g d^{-1})			
intercept	57 ± 0.6	91.04	0.000
sex	3.3 ± 0.5	6.16	0.000
rank (B)	0 ± 0.5	−0.08	0.936
rank (C)	−1.9 ± 0.7	−2.89	0.005
chick treatment	−1.3 ± 0.7	−1.83	0.073

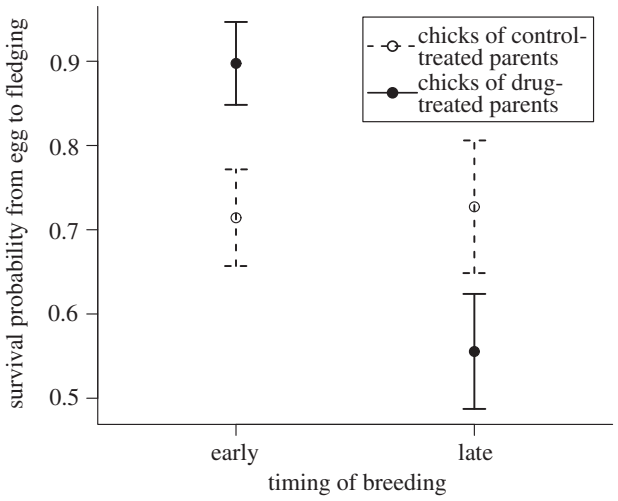

Figure 1. The effect of anti-nematode treatment of parents on the survival of their chicks, from the point of parent treatment (before hatching) to fledging, for individuals breeding before or on the median (early) or after the median (late) hatch date. Points show the group mean and error bars 1 s.e.

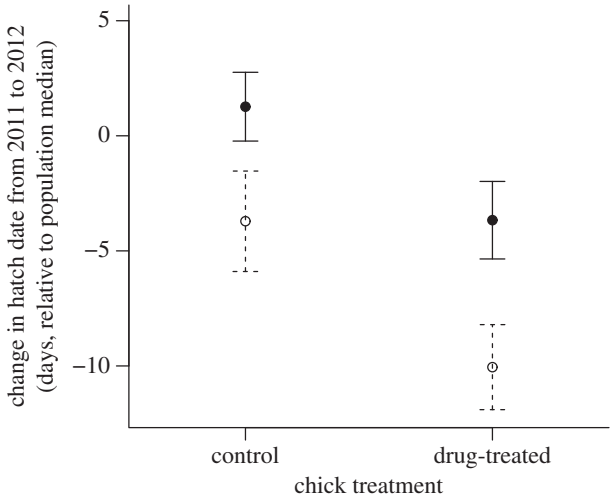

Figure 3. The effect of chick treatment on the timing of breeding of parents in the subsequent year for those with early initial timing of breeding (filled symbols and solid lines) and late initial breeding (open symbols and dashed lines). Early and late breeders are shown as separate categories for ease of representation; the analysis fitted continuous hatch date. Points show means ±1 s.e.

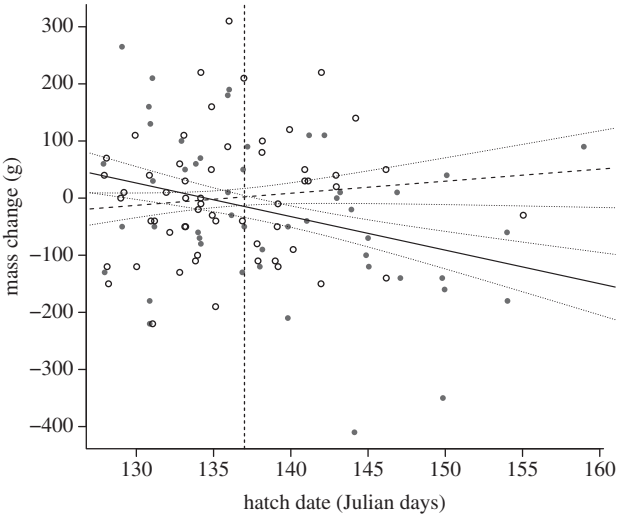

Figure 2. Parental mass change over the experimental period for parents of control (dashed line, open symbols) and drug-treated (solid line, filled symbols) chicks, in relation to hatch date. Points are jittered around hatch date for clarity. The fine-dotted lines show 1 s.e. around the fitted relationship, and the dashed vertical line shows median hatch date on 17 May. Elimination of nests past 145 days did not substantially alter treatment effects.

on individual fitness and host demography may be underestimated if indirect effects beyond the host and beyond the short-term experimental period are not accounted for.

The immediate indirect effects on both chicks and parents varied with hatch date, with treatment having positive consequences for early breeders and negative consequences for late breeders. This counters the expectation that anti-parasite treatment should benefit later breeders more (as found in [25]), which tend to be young and inexperienced individuals [35]. One potential mechanism could be that these young, late breeders suffer disproportionately from increases in co-infecting *Eimeria* species as a result of drug treatment very late in the season (*Eimeria* is the cause of avian coccidiosis, which occurs when burdens are high). Ivermectin treatment has similar effects in wild mice (*Peromyscus leucopus* and *P. maniculatus*), reducing nematode burden but increasing burdens of coccidia and cestodes under certain conditions [40]. Alternatively, later breeders may employ different allocation strategies to optimize reproductive outcome given the current breeding conditions: experiments in European starlings (*Sturnus vulgaris*) and Alpine swifts (*Apus melba*) have found that early-breeding parents favoured chicks in poor condition, whereas late-breeding parents favoured high-quality chicks [41], which parallels our results if parents perceive parasitized chicks as being of lower value.

Regardless of the mechanism driving the different responses to treatment across the season, it is important to note that, firstly, late breeders were not driving the relative importance of indirect effects (our results were qualitatively robust to removal of late nests) and secondly, we did not observe a directly mirrored response in the subsequent breeding season. Rather, the indirect effect of parasite removal on parents' timing of breeding the following year was the same across all individuals, irrespective of when they bred in the season in which they were treated. This suggests that immediate and long-term indirect responses to infection may be governed by different mechanisms and that breeding phenology in the subsequent season could be a strategic response to costs of infection, rather than simply a carry-over effect arising from physiological condition affecting performance from one season to the next [42,43]. It is notable that we detected these likely behaviourally mediated indirect effects in the absence of direct effects of treatment, which may be owing to particularly good breeding in the experimental year (average population breeding success of 1.54 chicks fledged per pair, compared with the 1985–2010 long-term average of 1.01). This longer-term indirect effect on timing of subsequent breeding is one that can have crucial fitness implications, as earlier breeding is generally associated with increased fledging success [28,30], and chicks of earlier breeders are more likely to recruit into the breeding population [33]. Our results therefore suggest that indirect effects of parasitism may be an important demographic driver that has thus far been overlooked.

While it is becoming widely recognized that the social environment in which parasitism occurs is key to both host

and parasite fitness, the integration of indirect effects to these studies has received less attention. The importance of indirect effects has previously been demonstrated between hosts and non-hosts of different species and of the same species even where there is little contact between family members [4,6]. However, Larcombe *et al.* [4] recently highlighted that such effects could be mediated by the social relationships between individuals in a group, with dominance status playing a key role in the impact of parasitism both on host traits related to fitness and parasite traits related to virulence. Family relationships are likely to play a stronger role, particularly in species with parental care, as individuals are related. In behavioural ecology, traits of other family members are typically seen as part of a focal individual's inclusive fitness [44] and parasite-mediated changes in individual family members' resource investment priorities might therefore be viewed as having the potential to impact on both personal and inclusive fitness of both the focal host and its family members. However, allocating shared costs to fitness within this framework is challenging. An alternative approach is to view the family as a series of interacting phenotypes [45]: quantifying the direct and indirect effects of parasitism on a given trait then allows the full effect of parasitism on both parent and offspring to be apportioned appropriately. Within this interacting phenotype framework, the importance of kinship in the potential to accelerate trait evolution has recently been demonstrated [46]; relatedness is likely to increase the potential for selection on shared or covarying traits such as those governing parent provisioning and offspring demand [16,46]. The indirect effects of parasitism are therefore also likely to be particularly important for the evolutionary potential of hosts to respond to costs associated with parasitism, particularly within a family setting.

In summary, we have shown that indirect effects of parasitism can have a major impact on individuals other than the immediate host in a natural host–parasite system in the wild, with consequences that persist beyond the period of the shared social environment within a single breeding season. Our results represent a major step towards being able to capture the evolutionary and demographic consequences of infection, increasing our understanding of the broader effects of parasitism that extend beyond the infected individual.

Ethics. All treatment doses were within an empirically established safe range for adult shags [24,25] and have been previously used on chicks with no negative consequences for survival or growth rate [8,26]. All drug treatment and blood sampling were carried out under UK Home Office licence (project licence PPL 60/3444), ringing under licence from the British Trust for Ornithology and experiments under a National Nature Reserve research licence from Scottish Natural Heritage, with full ethical approval.

Data accessibility. All data used in the analyses presented here are available at the Dryad repository: http://dx.doi.org/10.5061/dryad.8kc81.

Authors' contributions. E.J.A.C. and F.D. conceived and designed the study, contributed to interpretation and critically revised the manuscript; H.M.V.G.-W. carried out field and laboratory work, analysed the data, contributed to study design and drafted the manuscript; S.J.B. helped develop the study design, contributed to fieldwork, analysis and interpretation and critically revised the manuscript; S.L. contributed to study design, interpretation and revisions of the manuscript; K.A.H. contributed to fieldwork and manuscript revisions; E.A.T. carried out field and laboratory work and contributed to manuscript revisions. All authors have approved the manuscript for publication.

Competing interests. We declare we have no competing interests.

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