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1 **Indirect effects of parasitism: costs of infection to other individuals can be greater than direct**

2 **costs borne by the host**

3

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16

17 **Abstract**

18

19 Parasitic infection has a direct physiological cost to hosts but may also alter how hosts interact with

20 other individuals in their environment. Such indirect effects may alter both host fitness and the

21 fitness of other individuals in the host's social network, yet the relative impact of direct and indirect

22 effects of infection are rarely quantified. During reproduction, a host's social environment includes

23 family members who may be in conflict over resource allocation. In such situations, infection may

24 alter how resources are allocated, thereby redistributing the costs of parasitism between individuals.

25 Here we experimentally reduce parasite burdens of parent and/or nestling European shags

26 (*Phalacrocorax aristotelis*) infected with *Contracaecum* nematodes in a factorial design, then

27 simultaneously measure the impact of an individual's infection on all family members. We found no

28 direct effect of infection on parent or offspring traits but indirect effects were detected in all group

29 members, with both immediate effects (mass change and survival) and longer term effects (timing

30 of parents' subsequent breeding). Our results show that parasite infection can have a major impact

31 on individuals other than the host, suggesting that the effect of parasites on population processes

32 may be greater than previously thought.

33

34

35 **Keywords**

36 Endoparasite, life history decision, trade-off, anisakid, seabird, parent-offspring conflict

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

62 investment in current reproduction to preserve its residual reproductive value [14] or increase it as a

63 mechanism to ameliorate the effects of parasitism on the current breeding attempt [15]. Thus, the

64 full influence of infection may not be captured by considering only its immediate consequences.

65 Failure to account for both direct and indirect effects of infection, immediately and in the longer

66 term, is therefore likely to underestimate the effect of parasitism on hosts' life-history decisions,

67 performance of both hosts and non-hosts, and hence population processes.

68

69 Recent theoretical and empirical work has highlighted the importance of both parent and offspring

70 phenotype in determining the outcome of resource distribution within the family [16]. Therefore,

71 both parent and offspring responses to infection are likely to influence the impact of infection on

72 any individual family member. There is considerable evidence that the infection status of parents

73 can influence offspring growth and survival [2,9,17]. However, far fewer studies have examined

74 how offspring infection affects other family members. Notable exceptions suggest that parasite

75 infection in young can decrease parents' future breeding success [12] via mechanisms such as

76 increasing parents' feeding effort [18], but many of these findings stem from studies of host-

77 ectoparasite systems, where host-switching between family members is an essential part of the

78 parasite's life-cycle [19]. Effects observed in non-treated individuals may therefore in part be a

79 direct effect of an associated change in their parasite load, if treatment causes parasites to

80 redistribute themselves among the host group [12].

81

82 Teasing apart the direct and indirect effects of different family members' infections is further

83 complicated by an expected correlation in parasite load between family members. Parents and

84 offspring are likely to have similar levels of parasite exposure due to their shared environment and

85 potential to act as infection sources for other family members [12,19]. Family members may also

86 have comparable levels of immune defence because of their shared genetic background [20] and

4

87   maternal transfer of antibodies to offspring [21]. Parental and offspring traits that govern how

88   resources are distributed among the family are also likely to be coadapted [16], making within-

89   family comparisons essential to understanding the relative impact of parasitism across the family

90   unit. A powerful approach to investigate the relative roles of direct and indirect effects of parasitism

91   in wild populations would therefore be to simultaneously manipulate the parasite load of different

92   family members independently in a factorial design in a system where parasites cannot redistribute

93   themselves between hosts. However, to our knowledge, the family wide impact of parasitism has

94   not yet been examined in a single experimental framework.

95

96   Here, we examine the impact of both direct physiological effects of infection on hosts and indirect

97   effects on other individuals in the family unit across consecutive breeding seasons. We use the

98   European shag, *Phalacrocorax aristotelis*, a seabird that is commonly infected through its fish diet

99   by gastrointestinal nematodes [22–24], which are discretely distributed between hosts. Prevalence

100   of nematodes in our study population is high [24] and infection has direct effects on parents and

101   nestlings, particularly late in the breeding season and when breeding conditions are poor [8,25,26].

102   To assess the family-wide effect of parasitism, we treated parents and/or chicks with an anti-

103   helminthic drug in a fully factorial experimental design. We measured the effects of treatment not

104   only directly on the treated generation but also indirectly on all other family members, including

105   longer-term effects beyond the contact period between parents and offspring.

106

107

108   **Methods**

109

110   *Study system*

111   This study was conducted on the individually-marked breeding population of shags on the Isle of

112   May National Nature Reserve in south-east Scotland (56º11 N, 2º33 W) in 2011 and 2012. Shags

113   are piscivorous seabirds infected through the fish they eat by larval gastrointestinal nematodes,

114   predominantly *Contracaecum rudolphii*, which attach to the shags' stomach wall and become

115   reproductively mature [22,23]. All adults and chicks over 10 days of age that have been sampled in

116   this population are infected (68 adults endoscoped and 33 dead chicks dissected [24,27]). There is

117   no known mechanism by which chicks can infect parents, and direct transmission of adult worms

118   from parents to chicks does not appear to drive the establishment of infection in chicks [27],

119   although parents act as vectors of larval worms to chicks via the regurgitated food they provide.

120

121   Treatment of shags with 1% wt/vol ivermectin (Panomec©, Merial, UK), a broad-spectrum anti-

122   helminthic, reduces the number of worm eggs passed in faeces in chicks, removes worms from

123   adult shags for at least three weeks at a high dose, and reduces costs associated with infection [24–

124   26]. Treatment can increase chick growth with a stronger effect in later-hatched siblings; it can

125   increase chick survival and parental foraging, with greater effects on sons and mothers respectively;

126   and can increase breeding success, with a greater effect on birds breeding later in the season

127   [8,24,25]. The modal clutch size is three eggs, which hatch asynchronously creating a size hierarchy

128   across the brood (the "A" chick hatches first, "B" within 24 hours and "C" ca. 2 days later [28]),

129   although siblings do not differ in nematode prevalence at age 10 days, when our treatment was

130   administered [8]. Adult males are 22% heavier than females and grow faster during the linear

131   growth phase between the ages of 8 and 30 days [29]. The earliest breeders can lay in March and the

132   latest in July, and earlier laying is associated with greater breeding success [28,30] and lower

133 nematode burden in adults [24].

134

135 *Anti-parasite treatment experiment*

136 We measured the direct and indirect effects of parasitism in all family members by treating parents

137 and/or offspring with Panomec© in the 2011 breeding season and comparing their performance to

138 equivalent sham-treated controls. Parents and/or offspring were treated in a two-by-two factorial

139 design, which gave four treatment groups: parents control/chicks control, parents control/chicks

140 drug-treated, parents drug-treated/chicks control and parents drug-treated/chicks drug-treated. Both

141 parents were treated in the parent treatment and all chicks were treated in the chick treatment.

142

143 Three-egg nests were randomly assigned to treatment groups at laying. Groups were matched for

144 lay date and clutch size. At 3–7 days prior to predicted hatching, both parents at each study nest

145 were caught, weighed and measured, and injected intramuscularly with either ivermectin or a saline

146 control at a dose of 0.7mg/kg. All individuals not already carrying a British Trust for Ornithology

147 metal ring and field-readable Darvic ring were marked in this way as part of the long-term study on

148 the island. Nests were visited daily to obtain accurate hatching dates for all chicks. Hatchlings were

149 blood sampled for molecular sexing [31] and marked individually. When the oldest chick was 10–

150 12 days old, all chicks in the brood were weighed and injected subcutaneously with 0.05ml (mean

151 1.8mg/kg) of either ivermectin or saline. Differences between siblings in mass at this point were too

152 small to allow dose adjustments in relation to mass, but we have previously shown that individual

153 chick responses to treatment are driven by rank rather than mass at treatment [8,26]. Chicks were

154 subsequently weighed at age 15, 22, 28 and 35 days old (all ±1 day) and survival was recorded.

155 Parents were caught and weighed at the end of the experimental period (chick age 30–35 days).

156 Overwinter survival of parents was determined by examining whether individuals were resighted on

157 the Isle of May in future breeding seasons (overall annual summer resighting probability under

158 routine long-term monitoring is >95%, unpublished data from 2008-2014) and breeding dispersal is

159 negligible in this population [32].

160

161 In the breeding season following the experiment (2012, henceforth "subsequent" year), we recorded

162 three aspects of reproduction of all parents from our four experimental groups: whether breeding

163 was attempted, hatch date (by observation or calculated from chick wing length at ringing around

164 age 20 days, a reliable indicator of chick age), and breeding success measured as the number of

165 chicks fledged. Testing for longer-term effects on chicks was beyond the scope of this study as most

166 shags do not recruit until aged at least 3 years [33].

167

168 In total, we manipulated 71 nests, but excluded one nest with related parents, three that were second

169 clutches, and three with hatch dates >10 days after the latest nest in the main hatch date distribution

170 (range 31 days) that had spuriously strong statistical leverage. We also excluded one nest where

171 only one parent could be caught for ivermectin treatment, but retained two nests where only one

172 parent could be caught for control treatment as previous studies have found no difference between

173 unmanipulated and sham-treated controls [8,25]. These exclusions did not qualitatively change our

174 main results. Final sample sizes are shown in table 1. All data used in this paper are available from

175 the Dryad repository, doi xxxxx.

176

177 *Statistical analysis*

178 We considered the effects of both parent and chick treatments on all family members. Immediate

179 treatment effects on parents (i.e. the effect in the same breeding season as dosing occurred) were

180 measured as change in mass over the experimental period. Longer-term treatment effects were

181 measured as parents' overwinter survival, whether breeding was attempted in the subsequent year,

182 shift in hatch date (measured as the absolute shift in hatch date from the experimental year, relative

183   to the median in each year) and breeding success in the subsequent year (number of chicks fledged,

184   including zero values for individuals who did not breed). Chicks' immediate responses to treatment

185   were measured as growth rate (calculated by fitting a linear regression through the four masses

186   during the linear growth phase) and survival to fledging from three stages: parent treatment (before

187   hatching), hatching, and chick treatment (aged 10-12 days). Survival from parent treatment reflects

188   effects on offspring hatching success as well as post-hatching survival, but the effects of chick sex

189   and rank, which were assigned at hatching, could only be assessed using post-hatching survival. For

190   all response variables, parameter estimates are presented ±1 standard error.

191

192   We used backwards stepwise model selection, beginning with a maximal model including all

193   candidate main effects and interactions and eliminating the least significant effect in turn, removing

194   all non-significant interactions before removing main effects. In all response variables, we tested for

195   effects of parent and chick treatment as independent main effects, interacting with each other, and

196   each interacting with traits previously found to affect shags' responses to infection (hatch date, sex

197   and chick rank (A, B or C) [8,24–26]). Treatment effects were tested with factors known to

198   influence each response and treatment interactions with these variables: for chick survival, hatch

199   date and chick rank [25,30,34]; for chick growth, chick rank and sex [8,29]; for parent mass change,

200   sex to account for sexual size dimorphism; and for subsequent timing of breeding, sex to allow for

201   differences between males and females in overwinter behaviour and previous hatch date to account

202   for individual repeatability in phenology [35,36]. Interactions of chick and parent treatments with

203   these variables were examined in separate models to limit the number of terms; all models included

204   main effects of both treatments and an interaction between them (see ESM).

205

206   All analysis was conducted in R 2.15.1 [37] with packages nlme [38] and lme4 [39], fitting nest as a

207   random factor to account for non-independence of siblings and of parent pairs. Parental mass

9

208   change, chick growth and subsequent hatch date shift were modelled as continuous Gaussian

209   responses; chick survival, over-wintering parent survival and whether parents attempted subsequent

210   breeding as binary responses with binomial errors and a logit link; and number of chicks fledged

211   with Poisson errors and a log link. Because of limited variation in these binary and Poisson

212   variables, we fitted hatch date as a two-level categorical variable (early, i.e. hatched on or before the

213   median hatch date, or late, i.e. hatched after the median) when modelling these responses.

214 **Results**

215

216 *Direct effects of parent treatment*

217 We found no detectable effect of parent treatment on their mass change or overwinter survival,

218 either overall or varying with hatch date, sex or chick treatment (all parent treatment terms dropped

219 during model selection at p > 0.1; minimal models in table 2, model 1; model selection for all

220 response variables in ESM). Parent treatment also had no effect on their subsequent breeding

221 probability, timing or success (all parent treatment terms dropped during model selection at p>0.2;

222 minimal models in table 2, models 2-4).

223

224 *Direct effects of chick treatment*

225 Similarly, we found no direct effect of chick treatment on chick survival, either overall or

226 interacting with chick sex, rank or parent treatment (all chick treatment terms dropped during model

227 selection at p > 0.1; minimal models in table 2, model 5c), though mortality after chick treatment

228 was low overall (11 deaths, 134 survivors). Chick treatment had a marginal but non-significant

229 effect on chick mass change (growth rate), irrespective of sex, rank or parent treatment (in minimal

230 model, treatment effect -1.3 ± 0.7 g/day, t = -1.83, p = 0.073; table 2, model 6). An illustration of

231 all responses across the four treatment groups is given in the ESM (fig. S1).

232

233 *Indirect effects of parent treatment*

234 Treatment of parents had no overall effect on chick survival from the point of treatment; however,

235 parent treatment affected chick survival differently in early and late nests (hatch date * parent

236 treatment interaction: effect size 2.1 ± 0.9 (not back-transformed), z = -2.42, p = 0.016; table 2,

237 model 5a). For parents that bred before the median hatch date, treatment slightly increased chick

238 survival, but after the median, parent treatment decreased chick survival (fig. 1).

11

239

240 Last-hatched siblings had lower survival than A and B chicks (mean survival probability from

241 hatch: A chicks, 85 ± 4% of 63 chicks; B chicks, 84 ± 5% of 62 chicks, C chicks, 67 ± 7% of 42

242 chicks; difference between A and C chicks, z = -2.66, p = 0.008), but neither chick rank nor sex

243 influenced responses to parent treatment (interactions dropped at p>0.3; table 2, model 5b).

244

245 Parent treatment did not affect their chicks' mass change (all parent treatment terms dropped at

246 p>0.2; table 2, model 6).

247

248 *Indirect effects of chick treatment*

249 Anti-helminthic treatment of chicks had a significant impact on their parents' mass change.

250 Mirroring the indirect effects of parent treatment on chick survival, opposite effects were found in

251 early and late breeders (chick treatment * hatch date term in minimal model: effect size −8.7 ± 3.6

252 g, t = −2.81, p = 0.018; table 2, model 1). In earlier nests, parents of treated chicks gained weight

253 compared to controls, but in later nests, parents of treated chicks lost weight (fig. 2). Mothers and

254 fathers did not differ in this relationship, nor did parents' own treatment change the way they

255 responded to chick treatment (all parent treatment terms dropped at p > 0.1).

256

257 While chick treatment did not affect parents' over winter survival or likelihood of breeding in the

258 subsequent year (all chick treatment effects dropped at p > 0.4; table 2, models 2 and 4), parents of

259 drug-treated chicks bred almost a week earlier than the previous year compared to parents of control

260 chicks, with a marginally greater effect in fathers (in minimal model, chick treatment * parent sex

261 term: effect size −5.6 ± 2.8 days, t = −2.01, p = 0.052, table 1, model 3). Removing this interaction

262 term demonstrated a persistent influence of chick treatment on parents' subsequent hatch date (chick

263 treatment main effect: −6.04 ± 2.1 days, $F_{1, 53} = 8.80$, p = 0.005; fig. 3). In contrast to the more

12

264 immediate indirect effects of parasitism, chick treatment affected subsequent breeding in the same

265 way for early and late experimental parents (chick treatment by hatch date interaction dropped from

266 model at p = 0.270; fig. 3). Subsequent breeding success declined through the season overall (hatch

267 date main effect on number of chicks fledged, effect size (not back-transformed) -0.4 ± 0.2, z = -

268 2.68, p = 0.007) but was not affected by chick treatment (main effect and interaction dropped at p >

269 0.5; table 2, model 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 parents' 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 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 coinfecting *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

14

295 different allocation strategies to optimise reproductive outcome given the current breeding

296 conditions: experiments in European starlings (*Sturnus vulgaris*) and Alpine swifts (*Apus melba*)

297 have found that early-breeding parents favoured chicks in poor condition while late-breeding

298 parents favoured high-quality chicks [41], which parallels our results if parents perceive parasitised

299 chicks as being of lower value.

300

301 Regardless of the mechanism driving the different responses to treatment across the season, it is

302 important to note that, firstly, late breeders were not driving the relative importance of indirect

303 effects (our results were qualitatively robust to removal of late nests) and secondly, we did not

304 observe a directly mirrored response in the subsequent breeding season. Rather, the indirect effect

305 of parasite removal on parents' timing of breeding the following year was the same across all

306 individuals, irrespective of when they bred in the season in which they were treated. This suggests

307 that immediate and long term indirect responses to infection may be governed by different

308 mechanisms and that breeding phenology in the subsequent season could be a strategic response to

309 costs of infection, rather than simply a carry-over effect arising from physiological condition

310 affecting performance from one season to the next [42,43]. It is notable that we detected these likely

311 behaviourally-mediated indirect effects in the absence of direct effects of treatment, which may be

312 due to particularly good breeding in the experimental year (average population breeding success of

313 1.54 chicks fledged per pair, compared to the 1985-2010 long-term average of 1.01). This longer-

314 term indirect effect on timing of subsequent breeding is one that can have crucial fitness

315 implications, as earlier breeding is generally associated with increased fledging success [28,30], and

316 chicks of earlier breeders are more likely to recruit into the breeding population [33]. Our results

317 therefore suggest that indirect effects of parasitism may be an important demographic driver that

318 has thus far been overlooked.

319

320 While it is becoming widely recognised that the social environment in which parasitism occurs is

321 key to both host and parasite fitness, the integration of indirect effects to these studies has received

322 less attention. The importance of indirect effects have previously been demonstrated between hosts

323 and non-hosts of different species and of the same species even where there is little contact between

324 family members [4,6]. However, Larcombe *et al.* [4] recently highlighted that such effects could be

325 mediated by the social relationships between individuals in group, with dominance status playing a

326 key role in the impact of parasitism both on host traits related to fitness and parasite traits related to

327 virulence. Family relationships are likely to play a stronger role, particularly in species with

328 parental care, as individuals are related. In behavioural ecology, traits of other family members are

329 typically seen as part of a focal individual's inclusive fitness [44] and parasite-mediated changes in

330 individual family members' resource investment priorities might therefore be viewed as having the

331 potential to impact on both personal and inclusive fitness of both the focal host and its family

332 members. However, allocating shared costs to fitness within this framework is challenging. An

333 alternative approach is to view the family as a series of interacting phenotypes [45]: quantifying the

334 direct and indirect effects of parasitism on a given trait then allows the full effect of parasitism on

335 both parent and offspring to be apportioned appropriately. Within this interacting phenotype

336 framework the importance of kinship in the potential to accelerate trait evolution has recently been

337 demonstrated [46]; relatedness is likely to increase the potential for selection on shared or covarying

338 traits such as those governing parent provisioning and offspring demand [16,46]. The indirect

339 effects of parasitism are therefore also likely to be particularly important for the evolutionary

340 potential of hosts to respond to costs associated with parasitism, particularly within a family setting.

341

342 In summary, we have shown that indirect effects of parasitism can have a major impact on

343 individuals other than the immediate host in a natural host-parasite system in the wild, with

344 consequences that persist beyond the period of the shared social environment within a single

345   breeding season. Our results represent a major step towards being able to capture the evolutionary

346   and demographic consequences of infection, increasing our understanding of the broader effects of

347   parasitism that extend beyond the infected individual.

348

349

350

Table and figure captions

352

353   Table 1: Sample sizes and hatch dates (median and inter-quartile range) for each treatment group

354   used in the analysis. All nests had three eggs at the start of the experiment. Not all parents could be

355   recaught to measure mass change, and some chicks died after the first weight measure at treatment

356   so growth could not be calculated. Hence, not all manipulated nests were represented in all

357   analyses. Final sample sizes were: for parent mass change, 106 parents in 58 nests; for chick

358   survival measures, 189 eggs in 63 nests; for chick growth, 134 chicks in 59 nests; for subsequent

359   parent breeding, 105 breeders from 60 initial nests, with hatch date available for 92 individuals in

360   55 nests.

361

362   Table 2: Minimal models explaining variation in all response variables tested. Parents' overwinter

363   survival was best explained by an intercept-only model which is not presented here. Otherwise,

364   models are presented and numbered in the order they appear in the results. Test statistics are t-

365   values for continuous response variables (parents' mass change and subsequent breeding timing and

366   chick growth rate) and z-values for binary and Poisson response variables (breeding attempted in

367   2012, fledging success, and chick survival). Effect sizes are given in the following terms: for hatch

368   date, the gradient of its relationship with the response variable; for categorical hatch date, late birds

369   compared to late breeders; for sex, males compared to females; for treatment, ivermectin-treated

370   birds compared to control birds, and for rank, B and C chicks (as indicated in the table) compared to

371   A chicks. For binary and Poisson variables, effect sizes are not back-transformed from the link

372   function.

373

374   Figure 1: The effect of anti-nematode treatment of parents on the survival of their chicks, from the

375   point of parent treatment (before hatching) to fledging, for individuals breeding before or on the

18

376   median (early) or after the median (late) hatch date. Points show the group mean and error bars 1

377   standard error. Chicks of control parents are shown with open symbols and a dashed line, and chicks

378   of drug-treated parents with filled symbols and a solid line.

379

380   Figure 2: Parental mass change over the experimental period for parents of control (dashed line,

381   open symbols) and drug-treated (solid line, filled symbols) chicks, in relation to hatch date. Points

382   are jittered around hatch date for clarity. The fine-dotted lines show 1 standard error around the

383   fitted relationship, and the dashed vertical line shows median hatch date on 17th May. Elimination

384   of nests past 145 days did not substantially alter treatment effects.

385

386   Figure 3. The effect of chick treatment on the timing of breeding of parents in the subsequent year

387   for those with early initial timing of breeding (solid symbols and lines) and late initial breeding

388   (open symbols and dashed lines). Early & late breeders are shown as separate categories for ease of

389   representation; the analysis fitted continuous hatch date. Points show means ± 1 standard error.

390

Ethics statement

391

392

393   All treatment doses were within an empirically established safe range for adult shags [24,25] and

394   have been previously used on chicks with no negative consequences on survival or growth rate

395   [8,26]. All drug treatment and blood sampling was carried out under UK Home Office licence

396   (project licence PPL 60/3444), ringing under license from the British Trust for Ornithology, and

397   experiments under a National Nature Reserve research licence from Scottish Natural Heritage, with

398   full ethical approval.

399 **Data accessibility**

400

401 All data used in the anlayses presented here are available at the Dryad repository, doi xxxxxxxxx.

402 **Competing interests**

403

404 We have no competing interests.

Authors' contributions

405 **Authors' contributions**

406

407 EC and FD conceived and designed the study, contributed to interpretation, and critically revised

408 the manuscript; HGW carried out field and laboratory work, analysed the data, contributed to study

409 design and drafted the manuscript; SB helped develop the study design, contributed to field work,

410 analysis and interpretation, and critically revised the manuscript; SL contributed to study design,

411 interpretation, and revisions of the manuscript; KH contributed to fieldwork and manuscript

412 revisions; ET carried out field and lab work and contributed to manuscript revisions. All authors

413 have approved the manuscript for publication.

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425

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<div align="center">Table1</div>

Chick treatment	Parent treatment	
	Control	*Drug-treated*
Monitored during breeding season		
Control	17 nests 36 chicks, 31 adults 14th May (12th May – 16th May)	15 nests 34 chicks, 26 adults 18th May (14th may – 23rd May)
Drug-treated	14 nests 32 chicks, 23 adults 19th May (14th May – 15th May)	14 nests 32 chicks, 26 adults 18th may (12th May – 24th May)
Failed before treatment	1 nest 0 chicks or adults	2 nests 0 chicks or adults
Adults that returned to breed		
Control	30	27
Drug-treated	24	24

Table 2

Model & 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. *Hatch date shift 2011-2012*			
Intercept	40.9 ± 40.9	1.91	0.061
Chick treatment	-0.3 ± -0.3	-2.03	0.048
Hatch date	5 ± 5	2.85	0.008
Parent sex	-2.5 ± -2.5	-0.93	0.358
Adult treatment	-1.1 ± -1.1	-0.49	0.623
Chick treatment * parent sex	-5.6 ± -5.6	-2.01	0.052
4. *Subsequent breeding success*			
Intercept	0.6 ± 0.1	6.80	<0.001
Hatch date (categ.)	-0.4 ± 0.2	-2.68	0.007
5a. *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
5b. *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
5c. *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/day)*			
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