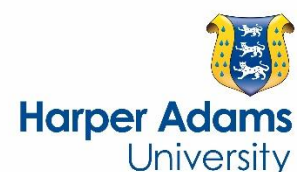


# Body condition is negatively associated with infection with *Syngamus trachea* in the ring-necked pheasant (*phasianus colchicus*)

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1 **Body condition is negatively associated with infection with *Syngamus trachea* in the Ring-**  
2 **necked Pheasant (*Phasianus colchicus*).**

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8 **Summary**

9 The role that parasites play in regulating animal populations is debated, however recent research hints  
10 at their pervasiveness among free-living animal populations. Parasites exert both direct and indirect  
11 effects on host populations, and can act to regulate populations. The Ring-necked pheasant is an  
12 important game-bird species in the UK, and large numbers of birds are released annually. The impact  
13 of the ubiquitous tracheal nematode, *Syngamus trachea* on pheasant populations through effects on  
14 host condition was assessed on two pheasant estates in the south west of England. Pheasants infected  
15 with *S. trachea* demonstrated a significant reduction in host condition compared with uninfected  
16 controls, with as few as one pair of worms per bird. Although there was no difference in worm  
17 burden between sexes, analysis of regression slopes revealed there was a significant difference  
18 between sexes in the magnitude of the effect of increasing worm burden on host condition, with  
19 detectable effects observed in hosts with one and three pairs of worms for males and females  
20 respectively. The observed reductions in host condition in birds with even sub-clinical infections  
21 could be the cause of poor reproductive success and survival of pheasants post-release.

22 **Keywords**

23 *Phasianus colchicus; Pheasant; Body condition; Gapeworm; Syngamus trachea; Reproduction*

## 24 **Introduction**

25 Parasites are well known to play an important role in regulating host population dynamics (Tompkins  
26 & Begon, 1999; Irvine *et al.*, 2006; Dunn *et al.*, 2012; Watson, 2013; Granoth-Wilding *et al.*, 2015),  
27 although there has been some debate as to the relative importance of predators compared with  
28 parasites (Irvine, 2000; Moss & Watson, 2001). Recent research however, has demonstrated that  
29 parasites are as, if not more, important than predators in regulating host populations (Watson, 2013).  
30 Parasites can have direct impacts on host populations through increases in morbidity and mortality,  
31 and they can also indirectly affect host populations through reductions in fecundity (Hudson, 1986;  
32 Dunn *et al.*, 2012; Granoth-Wilding *et al.*, 2015). Despite this, very little published research exists on  
33 the effects of parasite species on condition and host population dynamics in the ring-necked pheasant.  
34 One of the few studies by Draycott *et al.* (2002) assessed the effects of *Syngamus trachea* and  
35 *Heterakis gallinarum* on pheasant populations and concluded that infection with these species did not  
36 negatively affect host body condition. One major issue with this study is that body condition was  
37 assessed in April, whereas the infectious stages of *S. trachea*, and therefore infection pressure do not  
38 reach their peak until around June/July (Gethings *et al.*, 2015). A similar issue was observed by  
39 Irvine *et al.* (2006). Previous studies failed to find any effect of gastrointestinal nematodes on host  
40 condition in Reindeer populations in the High Arctic, however Irvine *et al.* (2006), through the use of  
41 delayed-release anthelmintic boluses, demonstrated reductions in host fitness in winter. Reindeer  
42 populations were sampled previously when populations were more accessible, which highlights the  
43 importance of timing research protocols to account for seasonal dynamics in the transmission of  
44 parasites, and therefore any parasite-mediated effects.

45

46 Reproductive success of released ring-necked pheasants is generally poor compared with their 'wild'  
47 counterparts (Leif, 1994), but it is currently unclear as to why this is the case (Leif, 1994; Draycott *et al.*  
48 *et al.*, 2000; Millan *et al.*, 2002; Draycott *et al.*, 2006; Villanua *et al.*, 2006). A number of factors such

49 as increased parasitic worm infections and reduced food availability/quality are believed to be major  
50 components governing life-history traits in game birds (Hudson *et al.*, 1992). *Syngamus trachea*, for  
51 example, is a parasitic-tracheal nematode that commonly affects managed pheasant and poultry  
52 populations. Morbidity rates are generally very high, particularly when birds are managed under high  
53 stocking densities and when proper sanitary measures are not incorporated into management  
54 programs (Gethings *et al.*, 2015). There is marked pathology associated with syngamosis (Fernando  
55 *et al.*, 1971; Nevarez *et al.*, 2002; Atkinson *et al.*, 2008) and mortality rates of affected birds can be  
56 as high as 80 % (Wojcik *et al.*, 1999). Experimental infections with *S. trachea* have demonstrated  
57 negative associations between larval challenge and weight gain and condition. Hwang (1964)  
58 experimentally infected turkey poults with varying numbers of *S. trachea*-infected earthworms to  
59 observe their effect on weight gain and packed cell volume (PCV). Significant differences were  
60 identified in weight gain between birds infected with an average of 0.2 worms ( $n = 10$ ) compared  
61 with an average of 55 worms ( $n = 55$ ), with the heavily infected group gaining an average of 51g  
62 compared with 1482g for the lightly infected group (Hwang, 1964). This significant effect on weight  
63 gain and condition could potentially influence fecundity directly through parasite-mediated  
64 competition for resources or indirectly if the host invests more resources into mounting an immune  
65 response (Delahay *et al.*, 1995; Shutler *et al.*, 2012). Indeed, Draycott *et al.* (2006) demonstrated  
66 increased breeding success in pheasants treated with Flubendazole, which suggests a possible  
67 relationship between fecundity and parasitic infection in the ring-necked pheasant.

68         Similarly, other studies have demonstrated significant negative parasite-mediated effects on  
69 host fitness. Many of these associations are however, correlational, and it is often difficult to  
70 disentangle cause and effect in the parasite-host relationship. Tompkins *et al.* (2000), demonstrated a  
71 negative relationship between the caecal nematode *H. gallinarum* and body condition in partridges.  
72 More relevant perhaps, is the negative association identified between *H. gallinarum* and body mass,  
73 breast muscle mass and cloacal fat in ring-necked pheasants (Sage *et al.* 2002).

74           The aim of the present study was to evaluate what effect, if any, significant infections with  
75 the nematode, *Syngamus trachea* have on pheasant body condition under natural circumstances.

76

## 77 **2. Materials and method**

### 78 *2.1 Study sites*

79 Two pheasant estates were selected in the South West of England due to regular problems with  
80 clinical syngamosis, as reported by managers. Site 1 was located approximately at grid reference ST  
81 97502 39837 and consisted of seven release pens. Site 2 was situated approximately at grid reference  
82 SU 17769 30326 and similarly consisted of seven release pens. Both sites release ~15,000 birds  
83 annually, undertake thorough predator control measures and provide supplementary grain via feed  
84 hoppers. Anthelmintic treatment (Flubendazole – at manufacturers dosage recommendations) ceased  
85 after birds were released in June 2015. Sites were matched in order to ensure that any effects on body  
86 condition would be parasite-mediated and not a result of intra/inter specific competition for food  
87 resources or other environmental factors.

88

### 89 *2.2 Carcass recovery*

90 One hundred and eighty adult pheasants were recovered following release from June 2015 through  
91 April of 2016. Birds were recovered by professional game managers, either as part of crop-protection  
92 programs or were shot during the shooting season. Pheasants were either shot whilst flying, or  
93 occasionally found dead upon the estate ( $n = 4$ ). Carcasses that had been scavenged were not  
94 included in the analysis and recovered birds were examined for non-parasite related disease that  
95 could influence the results. Carcasses were processed immediately upon recovery and assessed for  
96 the presence of *S. trachea*, *Ascaridia galli* and *H. gallinarum* by dissection of the trachea,

97 gastrointestinal tract and abdominal cavity and caeca respectively. Other nematode species were  
98 recorded but were not differentiated by species, as they were too few in number. In two instances,  
99 severely emaciated pheasants with bulbous, fluid-filled intestines consistent with clinical  
100 hexamitiasis and confirmed by the presence of motile protozoa on wet slide preparation were  
101 recovered. These birds were excluded from the analyses as they were found to be free of nematode  
102 infection, but are mentioned in the discussion.

103

### 104 *2.3 Worm recovery and body condition assessment*

105 Adult pheasants were weighed to the nearest 0.1g using a digital weighing scales and tarsal length  
106 was measured using a digital calliper with accuracy to 0.01 mm. A body condition index was then  
107 obtained by dividing body mass by tarsal length (Yom-Tov, 2001), which controlled for body size.

108

### 109 *2.4 Statistical analysis*

110 To determine whether the data were aggregated, adult worm counts were compared with an  
111 estimated Poisson distribution ( $\mu = \sigma^2$ ) with n-1 d.f. using the chi.sq test function in R available in  
112 the MASS package. Data were then compared with both an estimated Poisson and a Negative  
113 Binomial distribution using the fitdistr function (fitdistrplus package) and goodness of fit was  
114 assessed using Maximum Likelihood Estimation using AIC as a determinant. As the number of  
115 factors in each model were equal, the model with the lowest AIC score was considered a better fit.  
116 All data were analysed using R for Macintosh. The effect of parasite burden on host body condition  
117 was assessed using ordinary least-squares regression using  $\log(n+1)$  transformed parasite count data.  
118 Though the inclusion of the four dead-found birds could be a potential confounder, there was no  
119 change in model accuracy when they were excluded. Non-constant error variance was assessed using  
120 the Breusch-Pagan test and 'ratio' data were transformed to the appropriate power transformation

121 ( $y^{0.15}$ ). The transformed data were then assessed for non-constant error variance, which confirmed  
122 that the power transformation was successful ( $x^2 = 0.54$ ,  $df = 1$ ,  $p = 0.51$ ). Differences in parasite  
123 burden between sexes (including zero counts) were assessed using Welch's *t-test* for unequal  
124 samples.

125

### 126 **3. Results**

#### 127 *3.1 Prevalence of S. trachea – Pheasants*

128 Parasite count data were significantly different from the estimated Poisson distribution ( $x^2 = 2175$ , d.f.  
129 = 153,  $p = <0.001$ ) and comparison of models demonstrated the data were aggregated, and consistent  
130 with the negative binomial distribution ( $x^2 = 4.87$ , d.f = 3,  $p = 0.18$ ). The overall prevalence of *S.*  
131 *trachea* within this study population was 33%, with 32% of males ( $n = 148$ ,  $n = 48$  infected) and 38%  
132 of females ( $n = 32$ ,  $n = 12$  infected) being infected with at least 1 pair of worms. Males had a mean ( $\pm$   
133 SEM) worm burden of  $3.01 \pm 0.54$ , and females had a mean ( $\pm$  SEM) worm burden of  $4.78 \pm 1.68$ ,  
134 however no significant differences were found between sexes in mean worm burden ( $t^{35.78} = -1.26$ ,  $p$   
135 = 0.21).

136

#### 137 *3.2 Effect of S. trachea on body condition*

138 Worm burden and the associated effects on pheasant body condition are presented in tables 1 and 2.  
139 The regression of log parasite burden on body condition revealed a significant inverse relationship  
140 between increasing worm burden and body condition in pheasants ( $F^{3,176} = 216.2$ ,  $R^2 = 0.78$ ,  $P = <$   
141  $0.001$ ). Similarly, the effect of 'sex' was significant (coef = 1.1002,  $t = 2.84$ ,  $p = 0.005$ ) with  
142 regression slopes of  $b = -4.18$  (95% CI = -4.034) and  $b = -3.23$  (95% CI = -2.67) for males and  
143 females respectively, with slopes being statistically different from each other ( $t^{176} = 2.38$ ,  $p = 0.01$ ).

144 Comparison of regression coefficients for minimum worm burden requirements for identifiable  
145 reductions in body condition revealed 1 and 3 pairs were required for males (coef = -1.73,  $t = -4.69$ ,  
146  $p = <0.001$ ) and females (coef = -3.24,  $t = -2.80$ ,  $p = 0.04$ ) respectively. Stepwise deletion of the  
147 lowest parasite burdens suggested a ‘flattening off’ in body condition reduction above 11 worms per  
148 bird.

149

#### 150 **4. Discussion**

151 Pheasants infected with *Syngamus trachea* demonstrated significantly reduced body condition when  
152 compared with uninfected birds, and a negative association was identified between the number of *S.*  
153 *trachea* pairs per bird and body condition. This is the first study to observe such profound reductions  
154 in host body condition in pheasants naturally infected with *S. trachea*. Although Hwang *et al.* (1964)  
155 found that infection with *S. trachea* negatively affected weight gain in turkey poults, this was a result  
156 of an experimental infection, which does not accurately represent conditions faced by wild birds in  
157 terms of encounter rates and parasite load. Similarly, the provision of feed *ad libitum* in an  
158 experimental setting could increase host fitness and enable the production of a stronger immune  
159 response compared with birds under natural conditions.

160 The presence of parasites within these study populations, and the apparent parasite-mediated  
161 effects on body condition in post-release adult pheasants could have far-reaching ecological  
162 consequences (Delahay *et al.*, 1995). It is well documented that reared pheasants have reduced  
163 survival and reproductive success compared with their wild counterparts (Leif, 1994; Draycott *et al.*,  
164 2000; Millan *et al.*, 2002; Draycott *et al.*, 2006; Villanua *et al.*, 2006), and the high occurrence of *S.*  
165 *trachea* on pheasant estates could be a significant limiting factor on populations; especially  
166 considering that even relatively low numbers of adult worms (well below those at which clinical  
167 effects would be observed) are sufficient to produce statistically-significant reductions in host



168 condition. Infection with *S. trachea* could affect host populations directly; though parasite or host-  
169 mediated reductions in fecundity via responses to developing and established worms; direct  
170 competition for resources; or via (hypothetically) disruption of yolk proteins synthesised in the liver.  
171 Similarly, indirect effects such as increased predation rates in infected birds could significantly affect  
172 population structure and size (Hudson *et al.*, 1992). Though the overall prevalence of disease was  
173 low, it may still be exerting a negative pressure on population density if infected hosts have reduced  
174 life expectancy compared with uninfected hosts (Anderson, 1995). Indeed, *S. trachea* has a high  
175 mortality rate among juvenile, immunologically-naïve birds (Wojcik *et al.*, 1999) and an increase in  
176 parasite-induced mortality in juvenile birds could stabilise the parasite-host interaction by a net loss  
177 of parasites from the system (Anderson & May, 1978). Similarly, as pheasants are extremely  
178 susceptible to infection with *S. trachea*, the small proportion of the population susceptible to  
179 infection could ensure the persistence of the disease through constant reseeded of infectious stages  
180 (Anderson, 1995).

181         It has been suggested that in order to initiate egg laying, birds must reach a body condition  
182 threshold, and that individual host body condition necessarily delays or advances threshold  
183 attainment (Drent & Daan, 1980). For example, parasites that undergo hepato-pulmonary migration  
184 and/or cause anaemia via exsanguination compete with the host for protein during the time when  
185 energy input is concentrated on egg production (Allander & Bennett, 1995). Proteins required for  
186 yolk production are synthesised within the liver, and the migration of *S. trachea* larvae across the  
187 liver parenchyma could impair the production of these proteins (Allander & Bennett, 1995) thus  
188 potentially affecting the onset of laying. Indeed, Jones and Ward (1976) demonstrated that reduced  
189 yolk proteins delayed the onset of breeding in Red-Billed Quaeleas. Female pheasants generally lay  
190 their eggs between April and June, incidentally when *S. trachea* larval availability is increasing  
191 (Gethings *et al.*, 2015). The development and migration of *S. trachea* across the liver parenchyma  
192 could disrupt the formation of vital proteins responsible for chick development directly, or indirectly

193 through competition for host-resources during a period when hen body condition is already reduced  
194 (Breitenbach & Mayer, 1959). Indeed, a number of empirical studies have demonstrated increased  
195 survival and reproductive success of birds treated with anthelmintics compared with control birds  
196 (Hudson, 1986; Draycott *et al.*, 2006). Woodburn *et al.* (2002) demonstrated that birds dosed with  
197 anthelmintics reared twice as many chicks as un-dosed controls. It is unknown however whether the  
198 anthelmintic had a direct effect on breeding success by reducing parasite challenge, or because the  
199 treatment was associated with greater bird survival due to reduced predation (Hudson, *et al.*, 1992;  
200 Millan, *et al.*, 2002; Woodburn *et al.*, 2002). Similarly, It has been shown in Red Grouse populations,  
201 that the number of eggs laid is directly related to host body condition and energy intake in the  
202 preceding weeks (Delahay *et al.*, 1995). Delahay *et al.* (1995) showed that infection with  
203 *Trichostrongylus tenuis* reduced host body condition and could explain poor breeding performance of  
204 wild birds. Furthermore, Newborn and Foster (2002) demonstrated that birds with access to grit  
205 medicated with Fenbendazole had lower *T. tenuis* burdens and higher body condition scores than  
206 control birds. Interestingly, birds from the treated plots had significantly higher breeding success and  
207 reared twice as many chicks as birds from control plots. Chick survival was also significantly greater  
208 in treated plots compared with control (Newborn & Foster, 2002). This, in conjunction with the  
209 findings of the present study appear to suggest that parasite infection does indeed have some  
210 measurable effect on host populations, whether that be through parasite-mediated competition,  
211 reductions in host fecundity mediated by effects on body condition, or other factors is currently  
212 unclear. Although currently only speculative, the tentative link between *S. trachea* infection and  
213 pheasant fecundity warrants further consideration. Indeed, Holand *et al.* (2015), found that house  
214 sparrows (*Passer domesticus*) infected with *S. trachea* demonstrated reduced reproductive success  
215 compared with uninfected controls. They found a reduction in the proportion of eggs within a nest to  
216 hatch as faecal egg counts of mothers increased. Similarly, juvenile females with high faecal egg  
217 counts demonstrated significantly reduced lifetime reproductive success compared with uninfected  
218 birds.

219           Visually, infected birds were emaciated with reduced breast muscle mass and prominent keel  
220 bones, however no quantitative measurements were taken. Although it is not overly surprising, given  
221 the highly pathogenic nature of this parasite, that reductions in host body condition were observed, it  
222 is surprising that just one pair of worms was associated with an 11% reduction in body condition  
223 compared with uninfected birds. Similarly, these effects were observed in immunologically naïve  
224 birds and birds with evidence of previous exposure. The threshold for detectable reductions in host  
225 body condition in the present study was particularly low, which could implicate sub-clinical  
226 infections as a causal factor of the poor breeding status of released pheasants.

227           Although there was no detectable difference in mean worm burden between males and  
228 females, there was a significant difference in the magnitude of the effect of increasing worm burden.  
229 Females, in contrast to males, appear to be able to withstand relatively low worm burdens not suffer  
230 any negative effect on body condition below three pairs of worms per host. In contrast, males were  
231 often found with single pairs of worms (whereas single infections were not identified in females in  
232 this study) and that level of infection already began to affect body condition. The differences between  
233 sexes could be explained by differences in resource allocation, and it has been demonstrated that  
234 immunocompetence is often sacrificed in favour of the expression of sexual ornaments, particularly  
235 in males (Hamilton & Zuk, 1982; Verhulst *et al.*, 1999). Whether females are able to successfully  
236 mount an immune response in the presence of one or two pairs of worms requires further  
237 investigation. The apparent “flattening off” of parasite-mediated reductions in body condition above  
238 eleven worms per bird can perhaps be attributed to the density-dependent reductions in worm length  
239 observed at higher densities (Gethings *et al.*, 2016). Density-dependent reductions in worm length  
240 peaked at eleven worms per bird before flattening off. This appears to provide evidence of a reduced  
241 *per capita* effect above the density threshold.

242           The findings presented here are in stark contrast to the results of Draycott *et al* (2002), who  
243 found that *S. trachea*, along with *H. gallinarum* and *Capillaria* spp. had no real observable effect on

244 pheasant body condition. This can, however, be explained by the fact that body condition in the  
245 Draycott *et al* (2002) paper was assessed in spring, whereas *S. trachea* larval availability, and  
246 therefore clinical cases of syngamosis, generally do not reach their peak until June/July (Gethings *et*  
247 *al.*, 2015). All birds used in the present study were recovered between March and October, when  
248 stress levels are likely to be elevated due to release (Villanua *et al.*, 2006), which also coincides with  
249 peak larval availability (Gethings *et al.*, 2015). These conditions are typical of the vast majority of  
250 pheasant estates, so results presented here are likely to be comparable with and representative of  
251 other intensively reared pheasant populations. Although other parasite species were quantified, there  
252 were no similar reductions in pheasant body condition: with the exception of *Hexamitia* spp.,  
253 *Heterakis gallinarum*, along with a few cestoda, were the only other parasite species recovered from  
254 these pheasant populations; however, no effect was observed between their densities and body  
255 condition, even when total worm burden included *S. trachea*.

256           Although it is intuitively likely that the reduction in body condition was a result of significant  
257 *S. trachea* infections, it is difficult to disentangle cause and effect. The problem with cross-sectional  
258 studies is that it is difficult to establish whether these negative effects were a result of *S. trachea*  
259 infection as opposed to other forms of competition, or, whether birds acquired these parasites because  
260 they had reduced condition (Irvine, 200) and less ability to mount an effective immune response. The  
261 abundance of supplementary feed, predator control and reduced stocking densities suggests however,  
262 that parasites may have been the underlying cause of the observed reductions in body condition.  
263 Birds are known to lose a considerable amount of body condition during egg laying and incubation  
264 (Breitenbach & Mayer, 1959), however, the reductions in body condition in infected birds were still  
265 apparent when compared with uninfected birds, which would likely be facing similar environmental  
266 stressors.

267

## 268 **5. Conclusion**

269           The findings presented here appear to suggest a difference in the magnitude of the effect of  
270 worm burden on adult pheasant condition, with females able to withstand higher worm burdens  
271 before suffering any negative effects. Similarly, the results of the present study, in conjunction with  
272 the findings of Sage *et al.* (2002) demonstrate significant parasite-mediated effects on pheasant  
273 condition in birds following release and could be the cause for poor breeding success. However, it  
274 should be noted that these birds were examined out of the breeding season, and subsequent stress and  
275 alternative resource allocation during the breeding season could reduce this threshold in female  
276 pheasants.

277

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282

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