Investigating the loss of recruitment potential in red grouse (Lagopus lagopus scoticus): the relative importance of hen mortality, food supply, tick infestation and louping-ill

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Abstract Ticks and their pathogens cause significant disease and economic loss in many animal populations. Despite this, experiments that test the impact of ticks and tick-borne diseases on wild animal populations are rare. Here, we report on an experiment assessing the effect of ticks on red grouse productivity and chick growth in relation to other causes of poor recruitment at two sites in the Scottish uplands during 2005. Treated hens received two leg bands impregnated with the acaricide permethrin, while controls hens were untreated. Chicks were captured at c.2 weeks of age and fitted with a metal patagial tag, and chicks from treated hens also received a permethrin-impregnated strip. Mean tick burdens in treated chicks were close to zero compared with a mean of around 12 in the control group. Although treatment reduced tick infestations, it did not increase brood size. Growth rates in chicks from control and treated hens were similar during the first 10 days and comparable with chicks fed an ad-lib invertebrate-based diet. These results suggest that in this case, neither ticks (and the tick transmitted louping-ill virus) nor food shortages was the main cause of chick mortality. However, mortality in the adult hens was around 35 %, and predation accounted for 62 % of these losses before broods fledged. Our results indicate that on our study sites, predation may have a more important impact on grouse population dynamics than ticks and tick-borne disease. We suggest that it may be more cost effective to determine the causes of poor grouse population performance before implementing popular but expensive tick control measures such as the culling of alternative hosts and running acaracide treated sheep 'tick-mop' flocks.

Keywords Food supply · Louping-ill virus · Mortality · Recruitment · Red grouse · Ticks

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Introduction

Pathogen transmission between domestic animals and wildlife can be a source of human–wildlife conflict (Cleaveland et al. 2001). Pathogens transmitted by arthropod vectors such as ticks are known to cause significant disease in livestock and wildlife hosts in the tropics and temperate parts of the world (Sonenshine and Mather 1994) resulting in substantial economic loss (Sonenshine 1991). In Britain, the sheep tick (*Ixodes ricinus* L) is the vector for diseases such as Lyme borreliosis and louping-ill (Sonenshine 1993), which are of major pathogenic and economic importance.

In the Scottish uplands, the complexity of the system is illustrated by the wide range of tick hosts including sheep (*Ovis aries* L), red deer (*Cervus elaphus* L), mountain hare (*Lepus timidus* L) and red grouse (*Lagopus lagopus scoticus* L) (Gray et al. 1992; Hudson et al. 1997; Hudson et al. 2001; Laurenson et al. 2003). Of particular interest is the impact that the sheep tick may have on red grouse, an economically important gamebird, which has been in long term decline (Shaw et al. 2004). .

On many grouse moors, the failure of grouse populations to thrive is often attributed by managers to high chick mortality caused by tick infestation and the associated transmission of louping-ill virus (Hudson 1992)[1]. Clinical signs of this disease were present about 5 days post infection (Buxton and Reid 1975), and it has been shown to cause up to 78 % mortality in birds inoculated with the virus in the laboratory (Reid 1975) with high mortality indicated in the wild (Reid et al. 1978). In Scottish uplands, tick infestations of red grouse chicks (aged 1–40 days) increased between 1985 and 2003 from an average of 2.6–12.71 ticks per chick (Kirby et al. 2004). Suggested causes for this trend include increases in the red deer population (Clutton-Brock et al. 2004) and the warming climate which in the Scottish uplands (Barnett et al. 2006) may increase the tick questing season and altitudinal range (Gilbert 2010).

Because of the belief that controlling tick hosts and louping-ill virus (LIV) will enhance grouse populations, some grouse moor managers are increasingly treating and vaccinating moorland sheep flocks and culling wild tick hosts such as red deer and mountain hares. The sheep management aims to control ticks by treating animals with acaricides, which kill questing ticks on contact, and vaccinating ewes against LIV. However, ticks are likely to persist even if the hosts are at low densities as predicted in modelling studies of the use of acaricide-treated sheep as tick 'mops' which show that they may only be effective where deer numbers are reduced to very low densities (<6 per square kilometre) and acaricide efficacy is very high (>90 % of ticks killed—Porter et al. 2010). Mountain hares have also been implicated in the persistence of LIV through non-viraemic transmission between co-feeding ticks (Jones et al. 1997). Experimental reductions of hares have led to declines in tick abundance and LIV with corresponding increases in grouse populations (Laurenson et al. 2003) but only where sheep are well managed and red deer are absent. When deer are present, models predict that hare culls are unlikely to be effective because LIV will persist due to the combined effect of deer amplifying the tick population and grouse transmitting the virus (Gilbert et al. 2001; Harrison et al. 2010).

Therefore, the evidence that grouse production will increase when tick numbers are controlled is not strong, probably because other factors affecting grouse survival and breeding success, such as weather, food supply and predation, may be responsible for poor recruitment of individuals to the breeding population year on year. What is needed is an investigation into the causes and timing of chick losses and the determination of the extent to which ticks are responsible for these losses compared with other causes of poor recruitment. While studies have demonstrated that ticks on grouse chicks can be reduced by treating hens and chicks with acaricide (Laurenson et al. 1997; Mougeot et al. 2008), the consequences for survival and breeding success have yet to be satisfactorily quantified. In this study, we investigate the breeding success of red grouse on two grouse moors by following radio-tagged hens from the pre-laying period right through to the autumn, quantifying losses of potential recruits to the population and experimentally investigating whether these losses are associated with ticks, LIV or due to compromised growth rates.

Materials and methods

Study sites

The experiment was conducted on two grouse moors in north-east Scotland in 2005: Tullybeagles, Perthshire (moor 1) and Forest of Birse, Aberdeenshire (moor 2). Both sites had similar vegetation (heather *Calluna vulgaris* dominated moorland), were managed for grouse shooting (heather burning, predator control and nematode parasite control) and had low grouse densities and low productivity in recent years. On moor 1, mean tick counts (nymphs + larvae) in June on chicks around 7 days old varied between 5.3 and 41.1 between 1991 and 2000 (L. Gilbert, pers comm.) and LIV prevalence in sheep ranged between 4 and 32 % between 2002 and 2005 (Braan-Almond Grouse Management Group, pers comm).

[1] Game and Conservation Wildlife Trust http://www.gwct.org.uk/research/species/birds/red-grouse/

Catching, monitoring and treatment

Experimental work was conducted under the auspices of the UK Animals (Scientific Procedures) Act and approved by the local ethical committee. Sixty hens (40 on moor 1 and 20 on moor 2) were caught between 18 March and 6 April by lamping and netting at night (Hudson 1986). Half of the birds at each site were randomly assigned to the treatment group (i.e. given two acaricide leg bands, one on each leg; see Mougeot et al. 2008) or kept as control (untreated bands). Hens were fitted with radio collars (TW-3, Biotrack) to facilitate relocation. Birds were categorised as adult (greater than 1 year old) or juvenile (born the previous year) based on primary wing feather wear and toe claw marks (Watson and Moss 1979). Weight (g), wing length (mm), and body-condition score (based on the prominence of the sternum) were recorded. Nematode infestations are well known for their negative effects on grouse breeding success and survival (Hudson 1986) and are routinely treated for by grouse moor managers. Thus, we dosed all caught hens with an anthelmintic (Levamisole hydrochloride 3 %) at the time of catching. This treatment is highly effective at reducing nematode burdens in red grouse (Hudson 1986; Mougeot et al. 2005) and allowed us to standardise hens for the effect of this possible source of variation. We assumed that the probability of reinfection was likely to be randomly distributed between our control and treated hens and would therefore not systematically affect our analysis.

Nests were located by radio tracking and their location registered using GPS during the laying period. Clutch size was recorded, and every egg was weighed (±0.1 g) and measured (length×width, ±0.1 mm). Egg density was used to estimate hatch dates (Seivwright 2004). If clutches were incomplete at first visit, nests were revisited. At the expected hatching date, nests were relocated using GPS, and number of hatched and unhatched eggs were recorded. Hens found dead were assessed for the cause of death. If predation was involved, the type of predator was determined using visual signs (Thirgood et al. 1998).

At 2 weeks (±5 days) post-hatching, hens were located by radio tracking and pointer dogs were used to help locate and catch chicks. For each chick, we recorded body mass, wing length and tick numbers (nymphs plus larvae – adults are very rare on grouse), as per Kirby et al. (2004) and Mougeot et al. (2008). All chicks caught that were from treated hens were fitted with patagial wing tags that included a permethrin-impregnated plastic strip. At 1 month (±5 days) post-hatching, chicks were once again located and measured as above. Also at this stage, we located hens at night by radio tracking and counted the number of chicks in each brood using a lamp. This estimate was more effective than using dogs and was used in subsequent analyses. Hens were again located at around 45 days post-hatching when the chicks were able to fly short distances. Broods were then flushed, and the number of chicks was counted. Because chick counts at any one time could underestimate the number of chicks alive, earlier counts were revised upwards if later counts revealed more chicks than were previously detected.

Blood sampling and LIV prevalence testing

Chicks were sampled between 9 and 45 days of age. Those less than 9 days of age were not sampled in order to minimise trauma in small chicks. Blood samples were collected from the brachial vein of 75 chicks at time of the first capture using capillary tubes and kept in EDTA-coated eppendorfs (Moseley et al. 2007). Twenty-eight hens were caught and sampled in autumn (at the end of the experiment, when the radios where retrieved). Samples were centrifuged and the plasma separated and frozen within 6 h of collection before being transported to the Moredun Research Institute and stored at −70 °C. Plasma was used to determine seroprevalence for louping-ill virus using a haemagglutination-inhibiting antibody (HIA) test to detect antibodies to LIV in chicks and hens (Reid et al. 1978). Where sufficient sample remained ($n = 67$), a real-time reverse transcriptase PCR test (Marriott et al. 2006; Moseley et al. 2007) was conducted to detect the LIV virus directly.

Statistical analyses

The hens and their broods were divided into three groups. The control group consisted of untreated hens with all their chicks untreated. The first treated group comprised treated hens with all their chicks untreated, and the second treated group comprised treated hens and treated chicks. Because not all chicks could be captured, not all the chicks from the second treated groups could be treated. Thus, it is not possible to analyse individual chick survival particularly in the later stages when broods were only flushed and not handled. Mean tick numbers per chick were modelled using generalised linear mixed models with hen identity as a random effect and using a Poisson error distribution (Elston et al. 2001). The fixed effects that were explored included treatment, chick age (days since hatching), time of year (julian day) and study site (moor). The seasonal pattern of tick infestation was investigated by fitting time as julian day (days from 1st Jan). In addition, we tested if using a quadratic function of julian day was better at capturing the pattern in the data. The interaction between time of year and treatment was used to determine if the pattern of tick burden varied between treatments. Differences between the two study sites were controlled for by fitting moor and testing the interactions terms moor*treatment and moor*chick age. The relationship between tick burden and age was investigated as

above but using chick age instead of julian day. We also tested if using a quadratic function of chick age was better at capturing the pattern in the data. The effect of treatment on hatching success was modelled using logistic regression (binomial error structure). The response variable was the number of eggs that hatched (events) divided by the total clutch size (trials) for each brood and fixed effects included moor, maternal body mass and treatment. In the results we describe the season and age dependant patterns of tick infestation and then go on to present (1) the analysis of the control group versus the treated hens and chicks, (2) an analysis of the effect of treating hens only compared with treating hens and chicks on grouse chick tick infestation and (3) the effect of hen LIV seroprevalence on brood survival modelled using logistic regression (number of chicks alive at each period divided by the number of chicks that hatched). We then tested whether there were any maternal effects on brood size by fitting hen mass, treatment, hen age and moor as fixed effects in a generalised linear mixed model with hen id as a random effect.

The effect of treatment and hen LIV seroprevalence on chick growth rate was analysed by testing for differences in the rate of increase in body mass and wing length with chick age using generalised linear models. Body mass increased non-linearly, and we fitted an exponential curve to analyse the data using PROC NLIN in SAS (SAS Institute Inc, Cary, North Carolina, USA). Wing length increased linearly, and we analysed the variation using a linear model. In order to evaluate whether food limitation might have been a problem for chick growth, we visually compared the growth data from both moors with those from experiments on captive reared chicks that were fed *ad libitum* invertebrates (data in Park et al. 2001) but were unable to statistically test for a difference without access to the raw data.

Results

Tick infestation in relation to time of year and chick age

In the control group, tick counts varied with time of year and were very low in the first chicks caught in late May. They peaked in mid-June with a mean of around 12 ticks per chick and declined markedly by mid-July (Fig. 1a). A similar pattern was found when considering changes in tick abundance according to chick age (Fig. 1b). Chicks showed increasing tick infestation rates, which peaked at about 4 weeks of age with a mean of 10.75 (se=2.92) ticks per chick. The most infested chicks had up to 57 ticks on moor 1 and 45 ticks on moor 2. There was no difference between moors in relation to the pattern of tick infestation over time ($F_{1,51.4}=1.55$, $p>0.21$) or in relation to chick age ($F_{1,58.2}=0.26$, $p>0.61$).

Effect of treatment on tick infestation rates and LIV in chicks

(a) The effect of acaricide treatment of chicks on average brood tick infestation rates (treatment group 2). Taking into account the seasonal pattern (time of year) in tick abundance, mean tick counts on treated chicks (1.97, range=0–6) were significantly lower than those of control chicks (12.28, range=3–57; $F_{1,91.9}=44.27$, $p<0.01$, Fig. 1a). The same pattern was found when considering differences in tick abundance in relation to chick age instead of time of year: treated chicks had significantly lower tick numbers than controls ($F_{1,117}=32.47$, $p<0.01$; Fig. 1b) throughout the age range (0–55 days of age) with maximum mean infestation rates in the treated group of less than two ticks per chick (Fig. 1b) compared with 11.38 for control chicks.

(b) The effect of acaricide treatment of the hen on tick abundance in young chicks (treatment group 1). Average numbers of ticks per chick in untreated chicks from treated hens were significantly lower than those of chicks from control broods ($F_{1,68}=19.8$, $p<0.01$, Fig. 1a) and was higher than those of treated chicks ($F_{1,275}=7.99$, $p>0.01$, Fig. 1a) taking into account time of year. Peak tick numbers in the untreated chicks from treated hens were around four ticks per chick, about one-third of that found in chicks from control hens. The same pattern was observed in relation to chick age instead of time of year: tick infestation in untreated chicks from treated hens was significantly lower than those found in controls ($F_{1,117}=17.57$, $p>0.01$, Fig. 1b). However, there was no significant difference in tick infestation rates between treated chicks and untreated chicks ($F_{1,117}=2.1$, $p>0.14$, Fig. 1). Thus, treating hens only provided a reduction in tick infestation that was similar to that found in treated chicks when compared age for age (Fig. 1b) but did not provide quite as good protection when comparing over the same period (Fig. 1a). The interaction between site and either time or chick age was not significant ($p>0.1$).

(c) LIV prevalence. LIV prevalence was zero in tested chicks at moor 2 and 3 % (2/67) using real-time polymerase chaion reaction (RT-PCR) at Moor 1 (Moseley et al. 2007). Therefore, it was not possible to test the impact of acaricide treatment on LIV prevalence.

Effect of treatment on breeding parameters and chick growth

Laying date and clutch size did not differ between treatment groups or moors (Table 1). There was no effect of hen treatment or site on the hatched brood size as a proportion of the clutch size (Table 1; Fig. 2). The greatest losses of hatched chicks occurred between hatching and first capture at

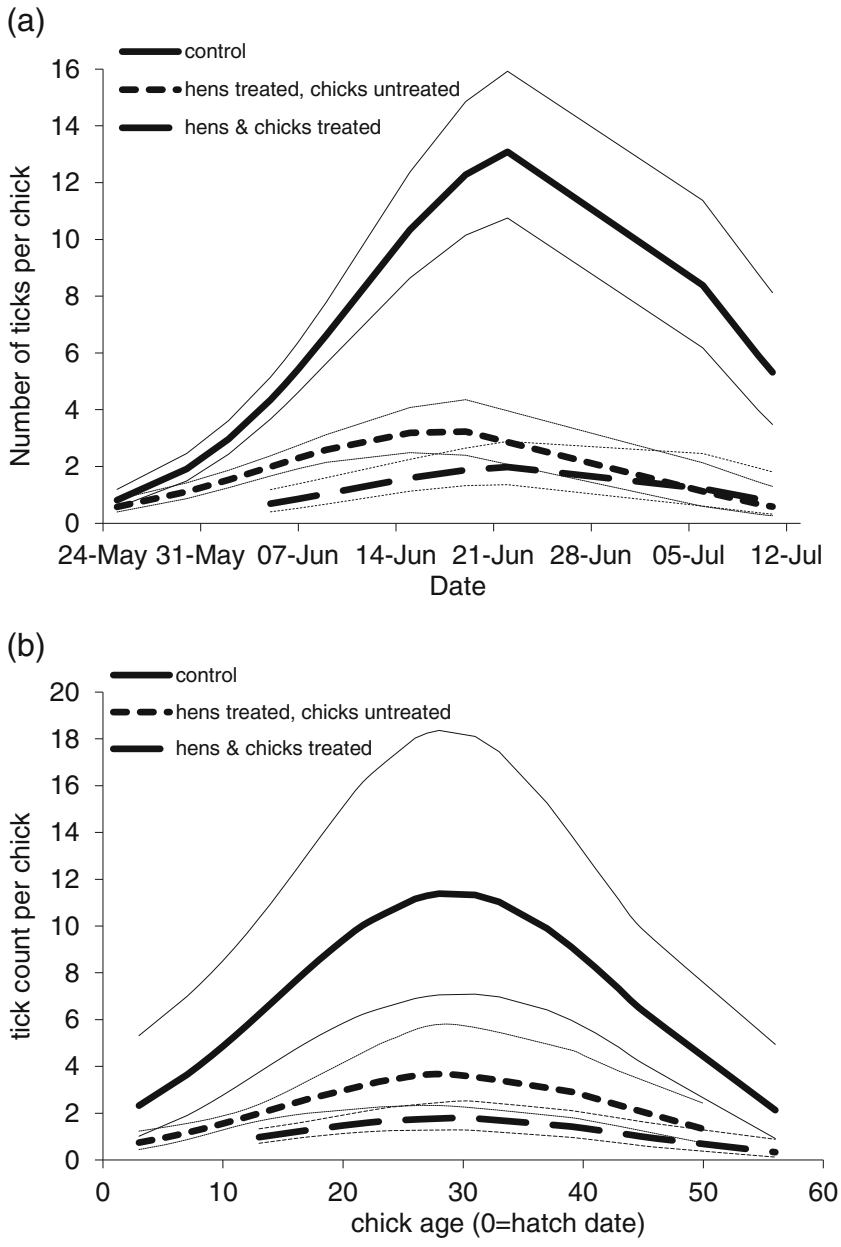

Fig. 1 Average number of ticks (larvae+nymphs) counted on red grouse chicks in relation to **a** date of sampling and **b** chick age (days since hatching). *Thick lines* represent mean counts and *thinner lines* represent the 95 % confidence limits from a model using a Poisson distribution and log link

approximately 2 weeks of age (Table 1). Brood size was smaller on moor 2 at 2 weeks of age, but the difference between moors was not significant at 1 month or at 45 days post-hatching (Table 1; Fig. 2). Less than 30 % of the hatched chicks were still alive at moor 1 at 45 days post-hatching. There was no effect of treatment on brood size at 2 weeks of age, 1 month of age or at 45 days post-hatching (Table 1). After taking into account the site differences, there was no effect of hen mass or age on brood size at time of first capture.

Growth rates in either wing length or body mass did not differ between moors 1 and 2 or between control and treated broods in either moor, so we pooled the data across moors and treatment groups and used average body mass at a given chick age (Fig. 3). Body mass of chicks during the first 10 days was similar to those of captive chicks fed with *ad libitum* heather and invertebrates (Fig. 4).

Effect of treatment on hen survival

Hen survival before offspring independence was 65 % (27 out of 40 hens on moor 1 and 12 out of 20 at moor 2). Overall, survival did not differ between treated and control hens (Fig. 5). The cause of death could be determined for 18 out of 21 hens found dead, and of these, predation was the cause in 13 cases (61.9 %, Table 2). One treated hen on moor 2 was found dead with no obvious signs of predation, but intact, and LIV was detected using RT-PCR on recovered brain tissue (Moredun Research Institute pers comm).

Table 1 Summary of breeding parameters for control and treated hens on each moor (mean±standard error, sample size in brackets)

	Moor 1		Moor 2		Moor	Treatment	Moor×treatment
	Control	Treated	Control	Treated			
Number of hens caught	19	21	10	10			
Number of hens that laid	17	19	8	8			
Clutch size	9.0±1.8 (17)	9.8±1.3 (19)	10.2±1.3 (6)	9.2±2.5 (5)	$F_{1,43}=0.26$ $P=0.61$	$F_{1,43}=0.02$ $P=0.88$	$F_{1,43}=2.37$ $P=0.13$
Hatch date	28.9±5.6 (15)	28.2±6.2 (19)	26.5±2.2 (8)	27.4±4.7 (8)	$F_{1,46}=0.98$ $P=0.33$	$F_{1,46}=0.00$ $P=0.96$	$F_{1,46}=0.23$ $P=0.63$
Hatched brood size	8.2±3.1 (17)	8.5±3.2 (19)	9.4±1.5 (5)	8.1±2.4 (8)	$F_{1,46}=0.21$ $P=0.65$	$F_{1,46}=0.26$ $P=0.61$	$F_{1,46}=0.63$ $P=0.43$
Brood size at 2 weeks	5.2±2.4 (13)	6.0±2.7 (16)	2.9±2.1 (7)	3.4±1.0 (7)	$F_{1,39}=9.24$ $P<0.01$	$F_{1,39}=0.68$ $P=0.42$	$F_{1,39}=0.01$ $P=0.90$
Brood size at 1 month	3.5±2.8 (14)	3.6±2.9 (16)	2.0±2.5 (7)	2.0±2.0 (5)	$F_{1,38}=2.71$ $P=0.11$	$F_{1,38}=0.00$ $P=0.97$	$F_{1,38}=0.00$ $P=0.97$
Brood size at 45 days post-hatching	2.3±2.8 (14)	2.7±2.9 (15)	1.9±2.3 (7)	1.0±1.2 (5)	$F_{1,37}=1.30$ $P=0.26$	$F_{1,37}=0.07$ $P=0.80$	$F_{1,37}=0.46$ $P=0.50$
Breeding success (%)[a]	37.2±27.4 (14)	35.9±27.2 (16)	29.5±28.0 (5)	12.8±13.7 (5)	$F_{1,34}=1.97$ $P=0.17$	$F_{1,34}=0.68$ $P=0.42$	$F_{1,34}=0.49$ $P=0.49$
Breeding potential=Total number of eggs laid	153	186	61	46			
Breeding outcome=Total number of young fledged (success %)	32 (21.0 %)	40 (21.5 %)	13 (21.3 %)	5 (10.9 %)			
% hens alive after 6 months	70 %	60 %	50 %	68 %			

[a] Calculated using only the hens alive at the end of breeding season

Hen LIV seroprevalence and breeding success

Seven of the 28 hens tested in the autumn were seropositive for LIV. Two of the seven positive and six of the 18 negative were juvenile birds. For moor 1, 30 % of hens ($n=4$ treated; $n=2$ control) were positive. Only one of six was positive on moor 2. For these 28 hens, we investigated whether breeding productivity differed depending on hen seroprevalence. Brood size at 2 weeks post-hatching was not different between seropositive and naïve hens after taking into account the difference in brood size between sites ($\chi^2=2.59$, $p=0.107$, df=1, mean for negative hens=4.47, SD=2.853, mean for positive hens=5.86, SD=2.34). However, brood size at 45 days post-hatching was significantly higher in seropositive hens ($\chi^2=7.62$, $p<0.01$, df=1, mean for negative hens=2.00, SD=2.95, mean for positive hens=4.17, SD=2.99), and there was a tendency towards a positive relationship with maternal condition which indicated that brood size increased by 0.2 % for every gramme of hen body mass in spring ($F=0.25$, $p=0.0621$).

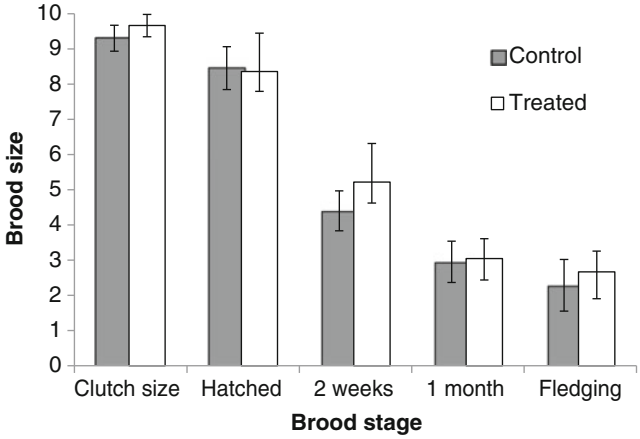

Fig. 2 Mean (±S.E.M) brood size at different stages for control and treated hens (data for both sites combined)

Discussion

The aim of this study was to investigate the relative importance of tick infestation, louping-ill, food supply (as measured through growth rates) and hen mortality on recruitment (brood size at fledging) in two red grouse populations. Whilst treating grouse with permethrin-impregnated leg bands was enough to reduce tick burdens on chicks (Mougeot et al. 2008), the effect of this treatment did not translate into increased brood size up to 45 days post-hatching. The observed high mortality in chicks was also unlikely to be due either to LIV as prevalence was less than 3 % as measured by RT-PCR and even less using the HIA test (Moseley et al. 2007) or to the food supply as

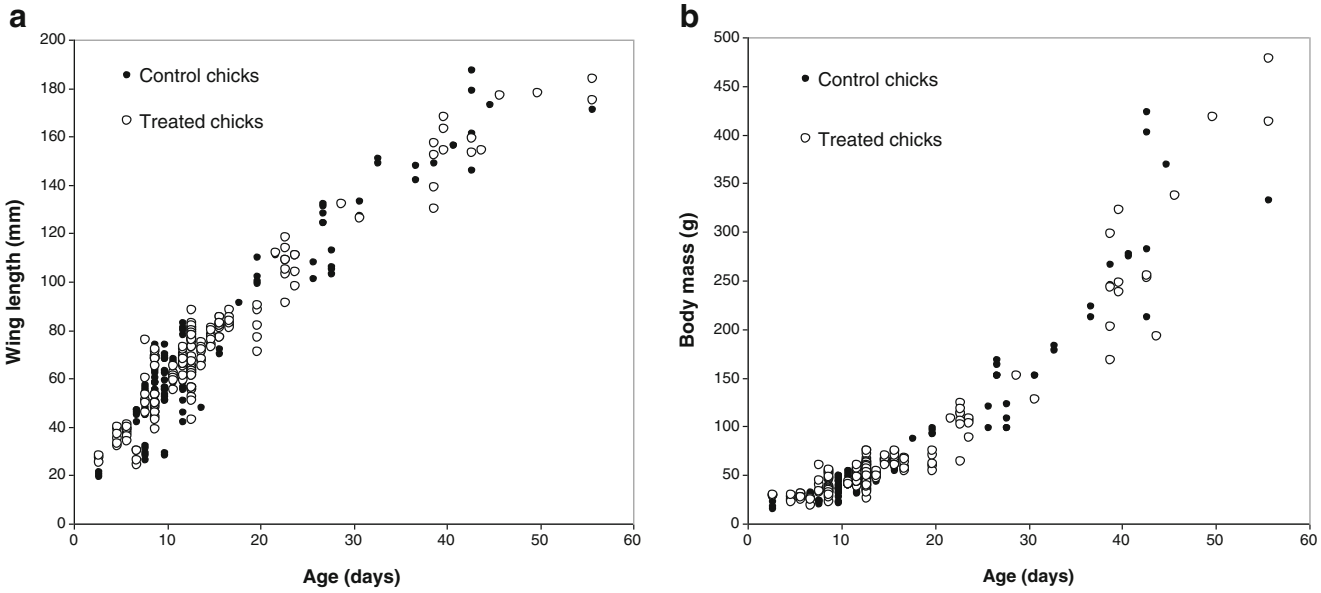

Fig. 3 Growth of **a** wing length and **b** body mass of chicks from control (*open symbols*) and treated broods (*filled symbols*). Data for both sites combined

growth rates were similar to that of chicks fed with an optimal diet (as in Park et al. 2001). In contrast, there is some evidence that hens that have been exposed to LIV and survived tended to have larger broods by the end of the study. Chick mortality was high in this study, and the biggest losses occurred in the first few days of life when tick infestation rates in untreated chicks were still very low. Chick mortality may be related to other causes, such as rainfall events around hatching or predation which was the cause of death in a high proportion of hens before and during the chick rearing period.

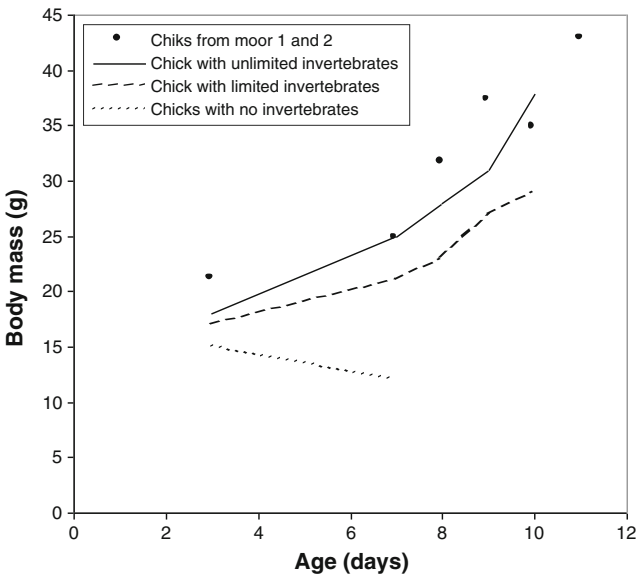

Fig. 4 Growth curves of chicks from both sites in their first 10 days compared with the growth from chicks reared in captivity under three diet types (data from Park et al. 2001): (1) heather and unlimited invertebrates (*solid line*); (2) heather and limited invertebrates (*long dashed line*) or (3) heather and no invertebrates (*short dashed line*)

Efficacy of the treatment

At 1 month of age, control broods had tick infestations equivalent to the levels found by Kirby et al. (2004). Treating hens with permethrin-impregnated leg bands did reduce tick burdens on their chicks, even if the chicks were not treated themselves, possibly because they gain protection from contact with the permethrin legs bands of females during brooding (Mougeot et al. 2008). In addition, we have shown that directly treating chicks with acaricide reduces chick tick infestations to very low levels. Despite preventing tick infestations, there was no beneficial effect on chick survival rates up to 45 days post-hatching (Table 1). In contrast, the greatest losses of chicks occurred between hatching and 10 days of age, that is, before peak tick infestation rates occurred. This contrasts to the work on pheasants *Phasianus colchicus* where acaricide reduced tick infestations had the effect of increasing survival of both hens and chicks and the reproductive success of males (Hoodless et al. 2002, 2003).

Hen mortality

A major loss of recruitment potential was hen mortality before hatching, or when chicks were still young (between 20 and 40 %, Fig. 5). This compares with typical hen mortality in summer of around 30 % (Thirgood et al. 2000a, b). Estimates from this experiment indicate that brood sizes at 45 days post-hatching for the remaining hens were between 2.3 and 2.7 (± 0.75) at moor 1 (Table 1) which is barely enough for the population to remain stable. On moor 2, the brood sizes were even lower, which has implications for population recovery at this site. Raptors contributed to some of the hen losses, but

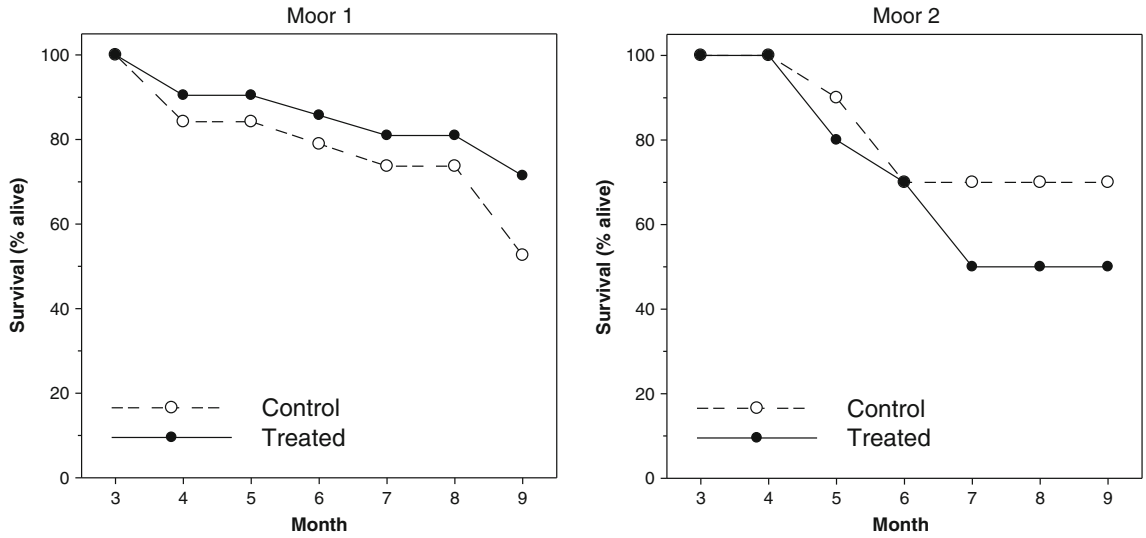

Fig. 5 Survival (proportion alive) of treated and control females on each study site. For sample sizes, see Table 1

mammal predation also had a significant impact (mainly by stoat and less often by foxes).

The post-mortem detection of LIV in the brain of a treated hen in early July suggests that acaracide application alone may not protect grouse from LIV infection because grouse can become infected by ingestion of infected ticks. This route could account for 73–98 % of LIV infections (Gilbert et al. 2004). Although other dead hens could not be tested for LIV, it is less likely that they died as a result of LIV infection as the majority died before the seasonal rise in tick numbers.

Chick losses

Our study supports earlier work which indicates that the first 2 weeks of life is the time when the main mortality of chicks occurs (Hudson 1986; Watson and Moss 1979). Potential causes include weather, diet quality, predation, ticks and LIV (Jenkins et al. 1963). The high levels of losses (between 70 and 90 %) compared with the 45 % losses were reported by Thirgood et al. (2000a, b). The weather may have played a role in our study because rain storms were recorded around hatching, and one nest was found abandoned with water running through it. However, diet quality on our sites did not seem to be limiting because chick growth rates were comparable with studies where chicks were fed *ad libitum* invertebrates (Park et al. 2001) indicating that chick diet quality was not compromised on these two moors. Furthermore, chick losses were unlikely to be due to ticks during this period because tick infestation rates were low early in the season and in young chicks less than 10 days of age whenever they are born (Fig. 1). Tick infestation rates peaked around a mean of 12 per chick in the control chicks, which is comparable with the more recent infestation rates reported in Kirby et al. (2004).

Table 2 Causes of hen mortality on the two study sites

	Moor 1	Moor 2	All
Predation by fox	0 (0 %)	1 (12.5 %)	1 (4.8 %)
Predation by stoat	3 (23.1 %)	1 (12.5 %)	4 (19 %)
Predation by raptors	3 (23.1 %)	2 (25 %)	5 (23.8 %)
Predation (unknown predator)	1 (7.7 %)	2 (25 %)	3 (14.3 %)
Disease[a]	2 (15.4 %)	1[b] (12.5 %)	3 (14.3 %)
Unknown	4 (30.8 %)	1 (12.5 %)	5 (23.8 %)
Total	13	8	21

[a] Bird found dead but intact

[b] Tested positive for LIV

Louping-ill virus

Tick transmitted LIV can be a major cause of mortality for sheep and red grouse (Reid 1975; Reid et al. 1978). Prevalence in sheep with grouse present can vary between 16 and 45.6 % (Gilbert et al. 2000) and on grouse chicks in Scotland between 0 and 36 % (Reid et al. 1978). The effect of acaricide treatment on LIV is difficult to determine in this study because LIV prevalence in grouse chicks was only 3 % (2/67) on moor 1 and zero on moor 2 (Moseley et al. 2007). The two chicks that tested positive weighed substantially less than uninfected chicks of the same age, consistent with the earlier findings (Reid et al. 1978). For the hens where we were able to assess LIV seroprevalence, our results suggest that brood size at 45 days post-hatching was significantly higher for the LIV seropositive hens compared with LIV naïve hens.

This effect is mostly marked between 2 weeks of age and 45 days post-hatching coinciding with the peak in tick numbers on chicks. Although the sample size is small, it may be worth investigating how management can capitalise on this effect, although more research is needed to determine the nature of this protection and investigate whether antibodies to louping-ill can be transferred to the eggs.

Our experiment demonstrated that the acaricide treatment was very effective at reducing tick burdens of chicks (even when only the hen was treated: Mougeot et al. 2008). Therefore, it may well prove effective in reducing losses due to tick-worry in some situations. However, treating grouse with topical acaricide does not prevent the possibility of them becoming infected from ingesting infected ticks. It is also possible that ticks and LIV will cause mortality in other years or at other sites when tick infestations and LIV prevalence are much higher (for an overview, see Scharlemann et al. 2008), but it seems unlikely that they were the major cause of recruitment loss at these two sites in 2005 (although they were perceived as such by local managers prior to conducting this experiment). Our findings need to be tested by repeating this experiment at other sites, with both higher tick infestations and higher LIV prevalences.

Our study also demonstrated that hen mortality during breeding (largely due to predation) and early chick mortality were the major causes of reduced recruitment to the red grouse populations, and that this was unlikely to be due to ticks or limited food supply. Indeed, predation has also been shown to be the major cause of mortality in pheasant (Hoodless et al. 2002, 2003).

We argue that our results should be taken into account when considering the cost effectiveness of tick control management strategies when losses may well be due to other causes. Even when ticks and LIV are implicated, their control by large scale reductions in tick hosts such as mountain hares are only likely to work if no other alternative hosts such as wild deer are present (Harrison et al. 2010). Although culling of wildlife hosts to control disease is a widely practised management strategy, it is usually only successful in controlling focal outbreaks rather than eliminating a disseminated disease (Wobeser 2002) and can have unpredictable consequences. In addition, the economic and practical implications of culling wildlife hosts are significant. For instance, as the population is reduced, the amount of time and effort required to continue culling increases exponentially (Wobeser 2002). It seems unlikely, therefore, that culling wildlife hosts in an effort to control ticks and LIV is sustainable or desirable in the long term.

Similarly, the effectiveness of sheep tick-mop flocks is only likely to be effective when alternative hosts such as red deer are reduced to very low levels (Porter et al. 2011). While vaccinating sheep against LIV can be effective in reducing LIV in grouse (GWCT 2011); this has also only been successful where alternative tick hosts are at low densities[2].

Although ticks and LIV may be an important issue in many areas used for red grouse shooting (for an overview, see Scharlemann et al. 2008), it is not clear that this is necessarily the case wherever ticks are found. This study indicates that other causes may also be important such as predation and weather events, which should be investigated before expensive tick control strategies such as sheep tick mops and the culling of other wild host species are embarked upon.

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