Dissecting the drivers of population cycles: Interactions between parasites and mountain hare demography

Sunny E. Townsend [a,*], Scott Newey [b], Simon J. Thirgood [b,1], Daniel T. Haydon [a]

[a] Boyd Orr Centre for Population and Ecosystem Health, College of Medical, Veterinary and Life Sciences, University of Glasgow, Glasgow G12 8QQ, United Kingdom
[b] The Macaulay Land Use Research Institute, Craigiebuckler, Aberdeen AB15 8QH, United Kingdom

ARTICLE INFO

ABSTRACT

There is a growing awareness that cyclic population dynamics in vertebrate species are driven by a complex set of interactions rather than a single causal factor. While theory suggests that direct host–parasite interactions may destabilise population dynamics, the interaction between host and parasite may also influence population dynamics through indirect effects that result in delayed responses to either density or to life-history traits. Using empirical data on mountain hares (*Lepus timidus*) infected with a nematode parasite (*Trichostrongylus retortaeformis*), we developed an individual-based model (IBM) that incorporated direct effects and delayed life-history effects (DLHEs) of a macroparasite, alternative transmission mechanisms and seasonality in host population dynamics. The full model describes mean characteristics of observed mountain hare time series and parasite abundance, but by systematically removing model structure we dissect out dynamic influences of DLHEs. The DLHEs were weakly destabilising, increasing the propensity for cyclic dynamics and suggesting DLHEs could be important processes in host–parasite systems. Further, by modifying model structure we identify a strong influence of parasite transmission mechanism on host population stability, and discuss the implications for parasite aggregation mechanisms, host movement and natural geographical variation in host population dynamics. The effect of *T. retortaeformis* on mountain hares likely forms part of a complex set of interactions that lead to population cycles.

© 2010 Elsevier B.V. All rights reserved.

1. Introduction

Cyclic population dynamics have captured the attention of ecologists since Elton's (1924) pioneering work, yet despite the intervening 85 years of research there is still no clear consensus as to what processes drive cyclic dynamics (Turchin, 2003). Single-factor hypotheses have been continually put forward since Elton's 1924 paper, as expressed by Lindstrom et al. (2001): "one can expect a hypothesis to be raised approximately every 4 years". The ability to explore and contrast multiple factors may certainly depend on the scale and scope of studies, and there are only a few studies to have successfully done so. One important example was the Klaune Boreal Forest Ecosystem Project (Krebs et al., 2001b) which used a factorial experimental design to disentangle the relative importance of some of the strongest candidates for control of the 10-year snowshoe hare cycles. Indeed, no one factor was singled out rather this experiment suggested that an interaction between nutrition and predation played a role in driving the hare cycles. Furthermore, a maternal effect has also been proposed to act synergistically with food and predation to drive cycles (Krebs et al., 2001a; Sinclair et al., 2003). There is a growing awareness that the causes of population cycles may include trophic interactions, individual variability, environmental variation and the complex interplay between these factors and population demography (Bjornstad and Grenfell, 2001; Lundberg et al., 2000).

The effects of trophic interactions may manifest themselves at the population level either directly or indirectly as a function of immediate and delayed responses to either density or to life-history traits (Beckerman et al., 2002). While an immediate life-history effect is a change in population demography in response to the current environment, delayed life-history effects (DLHEs) influence future population demography as a result of the current environment. Thus maternal effects, which transmit individual life-history responses between generations, can give rise to DLHEs (Beckerman et al., 2002), generating individual variability or a lag in the density dependence (delayed density dependence) with significant effects on the stability of population dynamics (Beckerman et al., 2002; Benton et al., 2001; Lindstrom and Kokko, 2002).

* Corresponding author at: Boyd Orr Centre for Population and Ecosystem Health, College of Medical, Veterinary and Life Sciences, University of Glasgow, Glasgow G12 8QQ, United Kingdom. Tel.: +44 0141 330 6638; fax: +44 0141 330 5971.
E-mail addresses: sunnytownsend@gmail.com (S.E. Townsend), s.newey@macaulay.ac.uk (S. Newey), daniel.haydon@glasgow.ac.uk (D.T. Haydon).
1 Deceased.

Although theoretical models suggest that host–parasite interactions may destabilise population dynamics and drive population cycles (May and Anderson, 1978), empirical support is limited (Hudson et al., 1998). Parasitic nematodes of the genus *Trichostrongylus* spp. have been implicated as drivers of cyclic population dynamics in red grouse and mountain hares. In red grouse, current hypotheses are multifactorial, based on interactions between parasitism and territoriality (Mougeot et al., 2005; Redpath et al., 2006) and productivity, harvesting and noise (Chapman et al., 2009). The role of parasites in mountain hare populations remains unclear (Newey et al., 2007a) as, although parasite mediated effects depress female fecundity (Newey and Thirgood, 2004), simple mathematical models suggest the direct parasite effect alone is too weak to account for sustained hare cycles and realistic parasite abundance (Townsend et al., 2009).

Here we develop a model to investigate multiple hypotheses for cyclic dynamics of mountain hare populations in Scotland. We include seasonal changes in host and parasite life-history traits in the model and explore how these alone, and in interaction with direct and delayed parasite mediated effects, influence host population dynamics and the propensity for population cycles. First, though, we present empirical evidence that DLHEs and seasonality are important structural elements of the mountain hare—*T. retortaeformis* system.

1.1. Ecology of the mountain hare—T. retortaeformis system

In a wide variety of organisms, the life-history traits of offspring, such as growth rate, survival, size, age at first reproduction and off-spring fecundity, depend on maternal condition and provisioning (Benton et al., 2001 and references therein). In mountain hares, the timing of breeding is important for reproductive success, as it is for many vertebrate species (e.g. Clutton-Brock et al., 1982). The mating season for the mountain hare in Scotland can begin as early as January although the timing of the first litter depends on winter temperature, female age, size and weight with older, larger and heavier females attempting to breed earlier (Flux, 1970; Hewson, 1976). Young born earlier in the year have a longer growing season, enter the winter at a heavier weight and larger than late born young and therefore have a higher over-winter survival and greater future fecundity when they enter the breeding population in the following year (Iason, 1989a,b, 1990). Although females may seek to breed earlier in the year to produce young with greater survival and reproductive potential, there is an associated cost on the size of later litters (Iason, 1990) such that females may have to trade-off the quantity and quality of their offspring.

Although it has not been studied for mountain hares, parasitic infections have been shown to influence the timing of breeding in a range of vertebrates (e.g. Allander and Bennett, 1995; Mulvey et al., 1994). If maternal parasite infection delayed the timing of breeding in mountain hares, the resulting maternal effect would constitute an additional 'indirect' parasite effect that may increase the overall impact of parasites. Since the abundance of nematode infections (parasite abundance is the number of parasites in a single host regardless of whether or not the host is infected, as defined by Bush et al., 1997) is thought to be dependent on host densities because of an increase in transmission rates (Arneberg et al., 1998), the result would be a delayed density dependent effect of the parasite on the host, with a tendency to destabilise population dynamics (Turchin, 2003).

Here we develop a model to describe the population ecology of the mountain hare (offspring vital rates dependent on maternal condition through timing of breeding) that includes suspected indirect parasite effects, by making body size and parasite abundance proxies of maternal condition. We want to know if these maternal effects can generate DLHEs and, if so, whether the DLHEs

have an important impact on hare population dynamics. A previous simple model of the hare–parasite interaction (characterized in Fig. 1a) may have excluded important elements of the interaction which could contribute to the debate on whether parasites are the main drivers of mountain hare population cycles (Townsend et al., 2009). Here we explicitly add maternal effects to the hare–parasite interaction (characterized in Fig. 1b).

In host–parasite systems, seasonal variation in host demographics and parasite transmission can destabilise the population dynamics and increase the likelihood of cycles (Altizer et al., 2006). In mountain hare populations, reproduction is restricted to 9 months of the year and seasonal patterns have been recorded in host vital rates. Adult mortality peaks in late winter and early spring (Flux, 1970; Iason, 1989b), pregnancies peak in spring (Flux, 1970; Hewson, 1970), and mortality in leverets peaks with the onset of winter (Flux, 1970; Iason, 1989b). Transmission of *T. retortaeformis*, as with most direct life-cycle intestinal parasites, depends on the production, development, decay and host encounters with parasite infective stages in the environment (Altizer et al., 2006). For *T. retortaeformis* in Scotland, eggs mostly survive the winter resulting in a mass hatching in spring with large numbers maintained throughout the summer (Crofton, 1948). Infection rate is also expected to increase in spring and summer as a result of immunosuppression associated with raised testosterone levels in males, naïve immune systems of juveniles and a combination of the periparturient rise in females with shared foraging habitat with juveniles (Cattadori et al., 2005). We incorporate seasonality via a hare breeding season and pulses of hare mortality, recruitment and parasite transmission.

1.2. A tactical approach

Here we adopt an individual-based modelling (IBM) framework to develop a tactical model that encompasses a large degree of ecological detail. This approach allows us to incorporate leveret biology, suspected DLHEs and seasonality. An IBM approach requires being explicit about parasite transmission mechanisms. One of the key features of parasitic infection, especially of nematodes with a direct life cycle, is the aggregated distribution of parasites between hosts, such that a few hosts harbor the majority of parasites (Shaw et al., 1998). Several effects are thought to contribute to aggregation: host heterogeneities, clumping of infection events and the positive feedback of the reinfection process (Rosa and Pugliese, 2002; Shaw and Dobson, 1995). In an analytical framework, the effect of aggregation has been accounted for by assuming a negative binomial distribution of parasites between hosts (Anderson and May, 1978). In an IBM, however, the infection status of each host must be tracked explicitly. Although Crofton (1948) carried out intensive studies in Scottish grasslands on the availability of infective larvae of *T. retortaeformis* to hosts, no study has looked at transmission within and between its mountain hare host. We devise three parasite transmission mechanisms and test the impact on hare population dynamics (see Section 2.5).

Our first aim here is to present a mountain hare—*T. retortaeformis* IBM that incorporates maternal effects and is able to simulate mountain hare population cycles and levels of parasite abundance that are consistent with empirical observations (herein referred to as the 'full model'). Rather than reproduce the dynamics of a particular hare population, we judge model fit based on characteristic dynamic properties identified for Scottish populations (Newey et al., 2005, 2007b). Our second aim is to study the dynamic effect of removing or modifying structure in the full model. We focus these results around four specific questions about how parasites may interact with hare demography to drive hare cycles: (a) does a relationship between the timing of breeding and maternal body size and parasite abundance generate DLHEs? (b) Do DLHEs reduce the strength of direct parasite effects necessary to reproduce observed
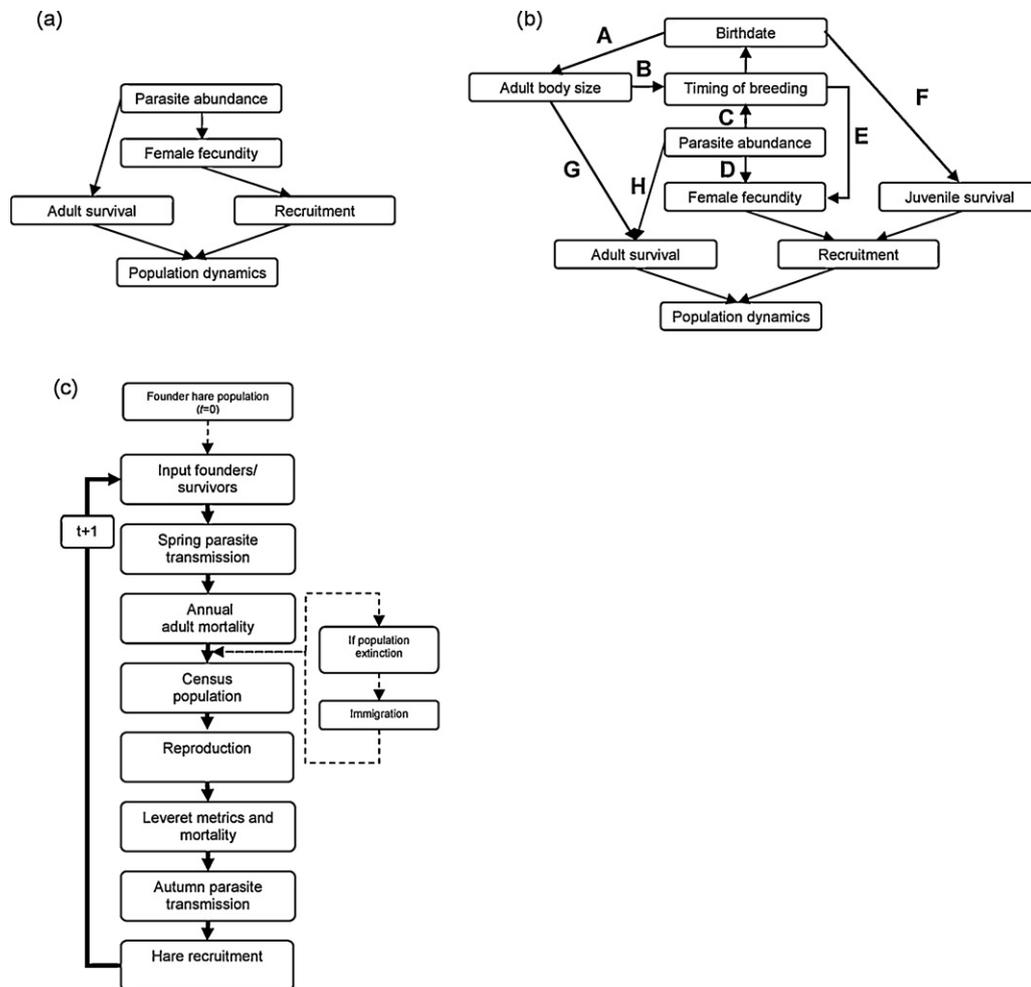
Fig. 1. Model configuration. Modelled links between individual hare attributes, demographic parameters and population dynamics in a model with (a) only direct parasite effects on adult survival and female fecundity and (b) additional effects of hare body size and parasite abundance on the timing of breeding, with subsequent indirect effects on juvenile survival, adult survival and female fecundity. Arrows indicate the direction of effects. In (b) the letters correspond to the modelled links (see Section 2.1). Link A: leverets born earlier in the breeding season attained a larger size before the onset of winter. Link B: an adult hare with a large body size was more likely to reproduce earlier in the year. Link C: an adult hare with few parasites was more likely to reproduce earlier in the year. Link D: females with fewer parasites tended to have more offspring. Link E: a female breeding earlier and with larger litters had smaller litters later in the year (Link F). Earlier born leverets had a lower chance of surviving to the onset of winter (Link G). An adult hare with a large body size was more likely to survive the winter (Link H). An adult hare with few parasites was more likely to survive the winter. (c) Outline of the order of events that the hare population experiences over one year in the IBM.

dynamics? (c) How do different parasite transmission mechanisms affect the host–parasite dynamics? (d) Can these models account for the wide geographical diversity of observed dynamics across Scottish populations?

2. Methods

2.1. The model

The model is initiated with a founder population of mountain hares that are allocated a random parasite abundance. In following years, the model runs on an annual cycle of eight principal steps with the chronological order of events chosen to reflect the natural sequence of identified seasonal pulses in hare adult mortality, reproduction, juvenile mortality, recruitment and parasite transmission (Fig. 1c). Reproduction takes place within a breeding season, prior to which the hare and parasite populations are "censused" close enough to harvesting time (often in December after the close of the grouse season (Hewson, 1970)) to be comparable to hare bag data used in mountain hare time series analysis (2007b). Hare adult mortality, reproduction, juvenile mortality, recruitment and parasite transmission are governed by simple rules

and mathematical formulas, the formulation of which depends on the presence of maternal effects and the parasite transmission mechanism. For the full model, the methods are described below with the corresponding model structure and parameterisation summarized in the table in Appendix F. For all models (full and modified), the structure and parameterisation are presented in greater detail in Appendices A–E. The models were programmed in MatLab (version 7 release 14, The MathWorks Inc.).

The full model links individual hare attributes to survival, fecundity, time of breeding and the vital rates of offspring (Fig. 1b). An adult hare with few parasites and a large body size was more likely to survive the winter (Fig. 1b, links G and H, see Appendix A) and reproduce earlier in the year (links B and C, see Appendix B). Females could have up to three litters and the birthdates of second and third litters depended on the birthdates of the first litter, the gestation period and a randomly determined additional inter-litter period (see Appendix B). Females with fewer parasites tended to have more offspring (link D, see Appendix C) but having larger litters early in the year reduced the size of later litters (link E, see Appendix C).

Leverets born earlier in the breeding season had longer to grow before winter and attained a larger size (link A, see Appendix D).

When they were reclassified as adults in the following year, their larger size increased their chances of surviving the winter (link G, see Appendix A). However, leveret survival was calculated as an accumulation of daily survival rates and therefore earlier born leverets had a lower chance of surviving to the onset of winter (link F). Related to this, a female that began breeding later had smaller leverets but was able to produce larger litters later in the year with a higher chance of surviving to the onset of winter. As data were lacking on leveret biology, the value for daily leveret survival rate (DLS) was reverse engineered (determined on the basis of match of the simulated dynamics to mean observed values (see Section 2.4)) for the full model.

Parasite transmission was controlled by parasite fecundity (λ), transmission inefficiency (H_0, reflects the proportion of eggs that do not re-enter the host) and the mechanism of transmission (described in Section 2.5 and presented in detail in Appendix E). For the full model, the transmission mechanism was reverse engineered (along with associated parameters).

2.2. Characterizing real hare time series and parasite abundance

We summarized the dynamics of hare and parasite populations in Scotland using the mean and range of four characteristics: the period of hare cycles, the amplitude of hare cycles, mean parasite abundance (averaged across whole population including non-infecteds, as defined by Bush et al., 1997) and the extent of statistical over-dispersion in the distribution of parasites between hares (as summarized by the relevant parameter of the negative binomial distribution, k) (Table 1). Empirical estimates for period and amplitude were taken from analyses by Newey et al. (2007b) of hare game bag time series ($n = 56$, median length = 37 years). For statistics on parasites, we compiled a dataset of parasite abundance for 654 hares sampled over 4 years from cross-sectional surveys conducted on 29 estates (Newey et al., 2005) and data presented in Boag and Iason (1986). The parasite abundance distribution from each of the 29 estates was fitted with a negative binomial distribution, the parameters of which are mean parasite abundance and k. By assuming that different estates have separate hare populations and their parasite infections are not synchronized, the mean and range of mean parasite abundance and k for the 29 estates provided an estimate of the variation in Scottish populations.

2.3. Characterizing simulated hare time series and parasite abundance

Characteristics of simulated hare time series and parasite abundance were estimated as for empirical data (Section 2.2). Where variation in empirical and simulated data characteristics were compared, mean parasite abundance and dispersion (k) were estimated by randomly sampling an individual year within a time series. Otherwise time series were characterized from more than just a single sample year, with estimates taken at 5 yearly intervals. Time series simulations ran for 37 years (after a minimum burn-in of 50 years to remove transient dynamics), the mean and range for period and amplitude was estimated from 56 simulated time series and the mean and range of mean parasite abundance and parasite dispersion (k) from 29 time series.

Simulations of model mountain hare populations could also, unlike real time series, be classified as implausible. Given that the highest observed hare density is around 200 km^{-2} (Watson et al., 1973), simulations which reached hare densities of greater than 400 km^{-2} were considered implausible. Extinction at the trough of oscillations resulting from demographic stochasticity was prevented by permitting some immigration (if there were less than 2 hares remaining, the model population was augmented by 10

'immigrants'), but simulations which exhibited near annual extinction frequencies were judged implausible. In this way we were able to distinguish between cyclic dynamics where the abundance at the trough was very small and unstable dynamics where the trajectory was persistently drawn to zero. A transition from equilibrium dynamics to sustained cycles or an increase in the amplitude of sustained cycles was regarded as a reduction in stability.

2.4. Full model performance

Performance of the full model was judged on the match of the four characteristic properties of hare time series and parasite abundance (hare cycle period, mean parasite abundance, parasite dispersion and hare cycle amplitude) between observed (Section 2.2) and simulated (Section 2.3) population dynamics. We took into account demographic stochasticity by running the model 200 times to quantify the 95% confidence interval for each characteristic estimated from the simulated data. The mean observed values of the four characteristics were required to fall within these intervals. The full model was also required to exhibit sustained cycles and have realistic minimum and maximum hare densities, overall parasite effect on fecundity (δ), mean annual values of hare mortality rate, hare recruitment rate, litter sizes and parasite abundances in juvenile hares.

2.5. Structural changes to the full model

We investigated the DLHEs on model hare population dynamics in three ways. First, by comparing the full model with models where the links between female body size and parasite abundance and timing of breeding were removed. Removal of the body size-timing link (link B in Fig. 1b) would leave an indirect effect of parasite abundance on size (through links C–A) which could complicate interpretation of results, therefore we investigated models where the parasite-timing link (link C, abbreviated to '-PT') was removed and where both links were removed ('-PTST'). Second, we measured the increase in the overall parasite effect on fecundity (δ) that was required to recover observed dynamics. δ was estimated from the simulated data as the slope of the relationship between parasite abundance and ova shed, and was increased by manipulating the direct parasite effect on fecundity (link D in Fig. 1b) via the parasite abundance thresholds for allocating ova shed in the second litter (see Appendix C). Third, by comparing the full model with a model without a direct parasite effect (removed link D, 'DFP = 0'), thus leaving only the DLHEs to impact female hare fecundity (link E).

Three mechanisms of parasite transmission among hosts were devised (see Appendix E). In the first mechanism, parasite recruits (defined here as new adult parasites within the host population) entered a pool from which they were allocated among the whole hare population ('Global') in each transmission pulse. The second mechanism represented local transmission ('Local'), recognizing that hares maintain home ranges (Flux, 1970) and therefore parasite recruits may be more likely to reinfect the same host, or the offspring they produce. In the third mechanism, hares were given a lifetime dose of parasites as leverets rather than annual augmentation of infection ('Once'). This was considered a plausible governing mechanism because hares tend to live less than 3 years (Hewson, 1976) and added infections after the first year may be relatively low in fertility (Skorping et al., 1991). The third mechanism 'Once' could not be mixed with other mechanisms, but it was possible to model transmission as a mix of the 'Local' and 'Global' mechanisms. To investigate the dynamic effects of each mechanism, we compared the full model with models where parasite transmission was governed solely by each mechanism.

Table 1

Mean (and range) of four characteristic properties of real and simulated mountain hare time series and parasite abundance. In order to assess whether the full model could capture the mean observed values and Scottish geographical diversity in the characteristic properties, the table presents the mean and range of these properties from (i) empirical sources (range visualized in Fig. 3), (ii) repeated simulations of the full model (Appendix G) and (iii) repeated simulations of the full model where parameters were varied within plausible ranges. See Fig. 3 for a visual comparison of the empirical range and parameter generated variation.

Property	Units	Observed time series		Simulated time series from best fit model	
		(i) Empirical estimate	Source	(ii) Stochastically generated variation	(iii) Parameter generated variation
Period of hare cycles	Years	9.2 (4–15)	Newey et al. (2007b)	9.5 (9–13)	12.3 (7–24)
Amplitude of hare cycles (coefficient of variation)		0.81 (0.39–1.80)	Newey et al. (2007b)	0.77 (0.58–1.41)	0.79 (0.05–1.72)
Mean annual adult hare abundance of *T. retortaeformis*	Hare^{-1}	1936 (190–4957)	Based on reanalysis of datasets described in Newey et al. (2005) and Boag and Iason (1986). See Section 2.2.	2759 (42–7705)	20186 (140–164460)
Parasite dispersion (k)		1.16 (0.19–5.55)		0.75 (0.46–1.26)	0.60 (0.28–1.36)

2.6. Changes to the parameterisation of models

The characteristics of hare time series and parasite abundance from the full model and models with structural changes were examined across plausible ranges of parameters (see Appendix F). Parameter combinations were generated using a Sobol' sequence which allowed us to sample parameter space more efficiently than if samples were taken at random (Press et al., 1992). Elasticity analysis was performed on the full model to compare the proportional effects of changing parameters on the characteristic properties of simulated hare time series and parasite abundances. Given the large number of parameters in the model, the elasticity analysis was limited to five parameters of interest: one emergent parameter (overall parasite effect on fecundity (δ)) and four controlled parameters (direct parasite effect on mortality (α), parasite transmission parameters (λ, H_0) and leveret survival LS (DLS multiplied by the length of the breeding season)).

3. Results

The structure and parameterisation of the full model are reported in Appendix F. Mean empirical values for the four characteristic properties of hare time series and parasite abundance (period of hare cycles, amplitude of hare cycles, mean parasite abundance and parasite dispersion) all fell within the 95% confidence interval generated from 200 simulations (Appendix G) and corresponded well to the mean simulated values (Table 1). An example simulation of the hare and parasite time series generated by the full model shows sustained cycles (Fig. 2), realistic minimum and maximum hare densities (4–262 km^{-2}), overall parasite

effect on fecundity (δ, 0.00045 hare parasite^{-1}), and mean annual values of hare mortality rate (0.5 year^{-1}), hare recruitment rate (2.7 year^{-1}), litter sizes (0.7, 2.3 and 3.9 leverets for the 1st, 2nd and 3rd litter, respectively) and parasite abundances in juvenile hares (882) parasites.

3.1. Does introducing a relationship between the timing of breeding and maternal body size and parasite abundance generate delayed life-history effects (DLHEs)?

We found no major differences in either the plausibility of simulated dynamics or the propensity of cyclic dynamics (Appendix I) between the full model and models where the maternal effect links were removed (abbreviated as -PT and -PTST) suggesting there was not a strong effect of removing the links on the stability of the dynamics. Simulations of hare and parasite population dynamics with the parasite abundance-timing of breeding link (Fig. 1b, link C) removed showed reduced amplitude of hare density fluctuations (amplitude = 0.73, Appendix H (a)). Removing the body size-timing link (Fig. 1b, link B) reduced the amplitude further (amplitude = 0.63, Appendix H (b)). The distributions of dynamic characteristics for simulations where parameters were sampled across plausible parameter space suggest that, compared to the full model, when links were removed amplitudes tended to be smaller (Fig. 3b) and parasites were more dispersed (Fig. 3d), while parasite abundance (Fig. 3c) and period (Fig. 3a) tended to be larger. In summary, removal of the links between maternal body size and parasite abundance and the timing of breeding suggested that they generated DLHEs with similar, weak, destabilising effects on the model hare population dynamics.
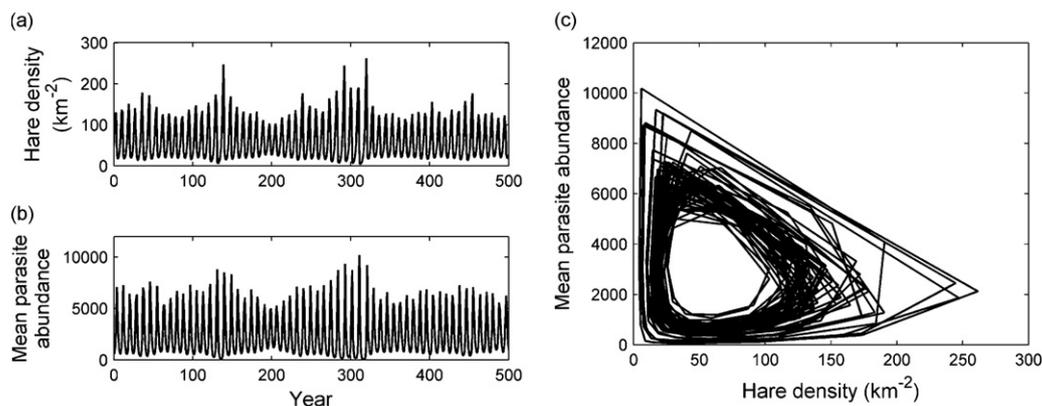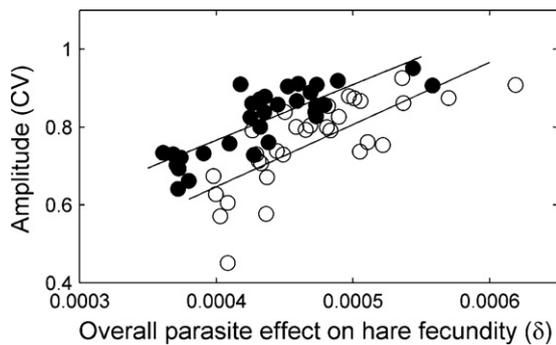

Fig. 2. Simulated population dynamics from a single run of the full model (see Appendix F for parameters) of (a) hares and (b) parasites. (c) The sustained cycles in state space. Hares cycled with a 10 year period and amplitude of 0.79. Parasites were distributed between hares with a mean abundance of 2400 worms and parasite dispersion, k, of 0.80.
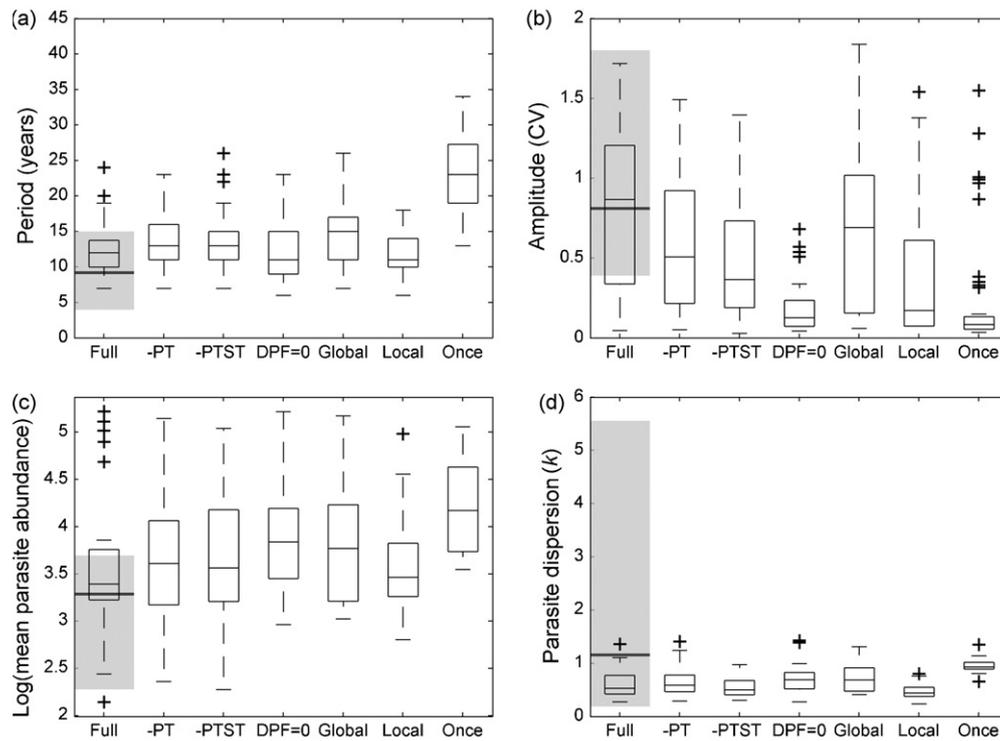
Fig. 3. The effect of structural and parameter changes to the full model on four characteristic properties of mountain hare time series and parasite abundance (a) period of hare cycles (for cyclic time series), (b) amplitude of hare cycles (for cyclic time series), (c) mean parasite abundance and (d) parasite dispersion k. Parameters were varied within the plausible parameter envelope (see Appendix F). Model structures abbreviate as: full (Full), parasite-timing link removed (-PT), parasite-timing and size-timing links both removed (-PTST), direct parasite effect on fecundity removed (DPF = 0), population-wide parasite transmission (Global), local transmission (Local), parasite abundance allocated once during lifetime (Once). The variation generated by plausible parameter changes to the full model was also compared with observed variation (range covered by light grey boxes, mean marked with dark grey line). Parameters that were not varied were kept at values set for the full model (Appendix F).

3.2. Do DLHEs reduce the strength of direct parasite effects necessary to reproduce observed dynamics?

With the parasite-timing link removed, increasing hare cycle amplitude from 0.73 (as estimated in the absence of this link) to the observed mean 0.81 required an increase in the overall parasite effect on fecundity (δ) of 0.00006 year^{-1}, while in the absence of both links δ was required to be increased by almost twice this amount (0.00011 year^{-1}) to increase amplitude from 0.63 (estimated in the absence of both these links) to 0.81 (Fig. 4). A simulation in which the direct effect of parasites on female fecun-

dity was removed and only DLHEs affected fecundity showed hare density fluctuating close to equilibrium (Appendix H (c)). Sampling across parameter space, more time series were plausible and the highest percentage for all models (10%) were non-cyclic (Appendix I). Compared to the full model, time series exhibited low amplitudes (Fig. 3b, 'DPF = 0') and mean parasite abundance tended to be higher (Fig. 3c). These results imply that the direct parasite effect on fecundity had a strongly destabilising effect on hare population dynamics, in contrast to the weaker, indirect parasite effects on fecundity caused by the DLHEs.

3.3. How do different transmission mechanisms affect the dynamics?

The full model allocated 90% of parasite recruits to be transmitted locally ('Local' mechanism) and 10% to population-wide transmission '(Global' mechanism). A simulation where parasite recruits were allocated to hares solely on a population-wide scale ('Global') fluctuated around a relatively small equilibrium hare density and ranged from non-cyclic to small amplitude 10 year period cyclic dynamics (Appendix H (d)). In contrast, when parameters were allowed to vary within plausible limits, amplitudes ranged widely (Fig. 3b). However, 95% of runs generated plausible dynamics compared to 83% for the full model (Appendix I), suggesting that global distribution of parasites generally had a stabilising effect on model hare dynamics, and generated dynamics which tended to have longer periods and larger parasite abundance than the full model (Fig. 3a and c).

Local transmission tended to have a destabilising effect on hare dynamics. A model with locally distributed parasites generated time series that varied from non-cyclic to high amplitude 10-year cycles (Appendix H (e)). When parameters were allowed to

Fig. 4. The relationship between the amplitude of hare cycles and the 'overall' parasite effect on hare fecundity (δ) when maternal effects on leveret birthdate were removed from the full model. δ takes a negative value, therefore to ease interpretation the strength (magnitude) of the effect is graphed. When the parasite-timing link was removed (filled circles) the regression coefficient for the slope was -1428 year ($t_{2,29} = -7.61$, $p < <0.001$) and intercept 0.195 ($t_{2,29} = -2.37$, $p = 0.03$). When both the parasite-timing and size-timing links were removed (open circles) the regression coefficient for the slope -1598 year ($t_{2,29} = -6.26$, $p \ll 0.001$).

vary within plausible limits, the lowest percentage of time series were plausible (45%, Appendix I) and parasites tended to be more overdispersed (lower k) than the full model (Fig. 3d).

A model developed to allocate lifetime parasite abundance to leverets ('Once') generated dynamics that were starkly different from other models considered. When parameterised with full model parameters, the model exhibited non-cyclic dynamics at a high equilibrium hare density (Appendix H (f)). In contrast, across parameter space, plausible dynamics were generally found to be cyclic (Appendix I, although they appeared more irregular than the smooth cycles seen for other model structures) with large periods and mean parasite abundance (Fig. 3a and c) and low amplitudes (Fig. 3b).

3.4. Can the wide geographical diversity of observed dynamics across Scottish populations be recovered?

Variation generated by demographic stochasticity in repeated simulations matched reasonably well with the observed range in hare cycle amplitudes and mean parasite abundance, but not period and parasite dispersion (Table 1). Variation generated by allowing parameters to vary within plausible ranges improved the match to real ranges of period and amplitude but worsened the match to the observed range of mean parasite abundance (Table 1).

The variability in period is a distinctive feature of Scottish mountain hare population dynamics (Newey et al., 2007b). Elasticity analysis suggested that period was particularly sensitive to leveret survival and the direct parasite effects on hare fecundity and survival (Appendix J). The variability in mean parasite abundance was unrealistic when parameters were allowed to vary, since abundances of 10^5 parasites were being reached (Table 1). We found that lower mean parasite abundance was associated with poor leveret survival and strong parasite-induced hare mortality, α (Appendix J), the local parasite transmission mechanism and presence of DLHEs (Fig. 3c, compare 'Full' with '-PT' or 'PTST').

4. Discussion

We explored the direct and delayed effects of a macroparasite on host population dynamics using the host–parasite interaction between mountain hares and *T. retortaeformis* as a model system. We developed an IBM of an infected host population which could reproduce host time series and parasite abundance distributions with mean characteristics taken from empirical studies of mountain hares in Scotland. The model was structured to allow delayed responses to life history by linking maternal body size and parasite abundance to the future survival and fecundity of offspring. We found these maternal effects could generate DLHEs that had a weak destabilising effect on hare population dynamics. The nature of individual-based modelling required formulating explicit mechanisms of parasite transmission. As this mechanism was unknown, we devised and compared three different transmission processes and found the best fit was a combination of local and population-wide transmission. While the full model was able to reproduce the mean dynamic behavior, an adequate model of mountain hare population dynamics should also be capable of replicating the natural diverse array of behaviors. We found that, with realistic variation in parameter values, the full model could capture the observed variation in three of the four studied dynamic characteristics.

DLHEs can increase the propensity for complex dynamics by generating delayed density dependence and individual variability (Beckerman et al., 2002). In simple dynamic models, the transmission via maternal inheritance of average individual 'quality' has been shown to be a plausible cause of forest lepidopteran cycles (Ginzburg and Taneyhill, 1994) and microtine rodent cycles

(Inchausti and Ginzburg, 1998) by causing delayed density dependence. Delayed density dependence is detectable using time series analysis and has been identified in cyclic Scottish mountain hare time series (Newey et al., 2007b). However, delayed density dependence has many potential sources: it is classically associated with endogenous factors including direct effects of trophic interactions (Turchin, 2003), but can also be generated by temporally autocorrelated environmental noise (Lundberg et al., 2000). It is currently almost impossible for time series analysis to distinguish among competing potential causes (Beckerman et al., 2002). While this work does not prove that DLHEs are acting in mountain hare populations, we found that DLHEs could have a destabilising effect on the dynamics. A previous model required an increase in the direct effect of parasites on fecundity outside of the empirically estimated envelope to generate hare population cycles (Townsend et al., 2009), while the presence of DLHEs reduced the increase (by 1×10^{-4} year^{-1}) in this parameter necessary to generate realistic dynamics—a relatively large reduction given the 'combined' parasite effect on fecundity (δ) has been empirically estimated in the order of 10^{-4} to 10^{-5} year^{-1} (Newey and Thirgood, 2004; Townsend et al., 2009). When DLHEs were included in the model, parameter values required to generate observed hare population cycles were closer to empirical estimates, suggesting that DLHEs could therefore be important processes in this host–parasite system.

The DLHEs could have a stronger effect on the propensity for cyclic dynamics than estimated here. In comparison to the direct parasite effect on fecundity, the DLHEs had a relatively weak destabilising impact. We note, however, that their impact may have been greater if the effect on time of breeding was made more sensitive to female fitness through a more biologically realistic mechanism. In the IBM, the timing of litters subsequent to the first was determined by an inter-litter period which was drawn from a uniform random distribution of between 1 and 60 days. While in principle postpartum estrus allows female hares to copulate within just a few hours of parturition (Höglund, 1957), the 60-day upper limit was reverse engineered to generate distributions of pregnancies and birthdates that were realistically spread across the breeding season (Flux, 1970; Hewson, 1970). While some variability is expected in inter-litter period, making it strongly stochastic may have effectively decoupled the impact of timing of the first litter on the timing of subsequent litters.

It is common practice in the theoretical investigation of complex population dynamics to compare the match of modelled to real time series using dynamic characteristics. This method of goodness of fit is open to criticism especially where just a single aspect of the time series, such as the period, is abstracted (Kendall et al., 1999). We have taken this approach but used two characteristics of hare populations, period and amplitude of cycles, and two characteristics of parasite distributions between hares, mean abundance and level of aggregation. We have generally found during our investigations that, while it is relatively easy to obtain realistic periods, it is most difficult to capture realistic mean parasite abundance. Both the elasticity analysis performed here on the IBM and on a previous analytical model (Townsend et al., 2009) agree that parasite abundance is strongly influenced by the parasite's effect on host mortality, but experimental studies do not detect a strong mortality effect (Newey and Thirgood, 2004). The model presented here introduced leveret survival and we identified it as a potentially important determinant of parasite abundance in adult hares. This suggests that host age-structure may be an important aspect of this host–parasite system and future models should separate leveret and adult demography.

Host population dynamics depend on the mechanism causing aggregation in parasites between hosts (Rosa and Pugliese, 2002). Aggregation is a characteristic feature of macroparasite distribu-

tions (Shaw et al., 1998) and a pattern which is likely to result from several factors, most notably host heterogeneities, clumping of infection events and the positive feedback of the reinfection process (Rosa and Pugliese, 2002; Shaw and Dobson, 1995). Here, we explicitly modelled mechanisms of parasite transmission. A population-wide ('Global') mechanism was the IBM equivalent of the multiple infections term in the model of Pugliese et al. (1998), while the local transmission mechanism combined all three aggregation factors to some degree. A previous study has compared the effect of host heterogeneity (in immunity) and clumped infections and found the model with clumped infections tended to be less stable (Rosa and Pugliese, 2002). We have found that the model which included more aggregation effects tended to have less stable host population dynamics.

We found a mixture of local (90%) and population-wide parasite (10%) transmission was optimal (under the full model structure and parameterisation), and this could have interesting implications for the effects of host movement on population dynamics if we assume that the full model is a reasonable reflection of reality. The predominance of local transmission suggests the observed distribution of parasite abundance among hosts arises largely from individual differences between hosts, their local environment and/or infection pressure. This is consistent with what is known about the local movements of mountain hares which maintain non-exclusive home ranges and often feed alongside other hares (Flux, 1970). Any factors influencing host heterogeneities, hare movement or the longevity of *T. retortaeformis* free-living stages could result in variation in the balance of local–global transmission. Varying degrees of locally and globally distributed infections between mountain hare populations in the UK, or within or across years in the same population, is a plausible mechanism for generating the wide range of hare population dynamics observed.

In contrast, allocating lifetime parasite infections to newborn hosts gained limited support as the governing mechanism of transmission in the mountain hare–parasite system. The lifetime parasite abundance allocation mechanism was proposed because of the short lifespan of hares and the lower fertility of more recently acquired parasites (Hewson, 1976; Skorping et al., 1991). The starkly different dynamics resulting from this transmission mechanism suggests that the additional infections picked up by adult hares through their lifetime are important to the parasite population.

The model presented here captured mean characteristics of real mountain hare time series and abundance of the nematode parasite *T. retortaeformis*, and much of the empirically observed diversity in these characteristics. Further natural variation could be accounted for by population differences in the balance of local and population-wide parasite transmission. The model included direct parasite effects on the fecundity and survival, links between maternal body size and parasite abundance on the timing of breeding, leveret biology, seasonality in hare reproduction, recruitment, mortality and parasite transmission, and modelled mechanisms for parasite transmission. As has been shown for red grouse, snowshoe hares, Soay sheep and Svalbard Reindeer (Albon et al., 2002; Gulland et al., 1993; Krebs et al., 2001a; Mougeot et al., 2003), the effect of parasites on mountain hares likely forms part of a complex set of interactions that lead to population cycles. Further work is needed to provide better parameter estimates and to provide empirical estimates for important life-cycle stages. For example, we included immigration in the model to prevent stochastic extinctions, although this was not based on empirical data. Mountain hare populations in Scotland are harvested for sport and increasingly to attempt to control tick-borne disease (Harrison et al., 2010; Patton et al., 2010). Dispersal, harvesting and population control likely have significant effects on hare populations and ongoing field and modelling work are exploring these issues.

Appendix. Electronic supplementary information

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.ecolmodel.2010.08.033.

References

Albon, S.D., Stien, A., Irvine, R.J., Langvatn, R., Ropstad, E., Halvorsen, O., 2002. The role of parasites in the dynamics of a reindeer population. Proc. R. Soc. Lond. B: Biol. Sci. 269, 1625–1632.

Allander, K., Bennett, G.F., 1995. Retardation of breeding onset in great tits (*Parus major*) by blood parasites. Funct. Ecol. 9, 677–682.

Altizer, S., Dobson, A., Hosseini, P., Hudson, P., Pascual, M., Rohani, P., 2006. Seasonality and the dynamics of infectious diseases. Ecol. Lett. 9, 467–484.

Anderson, R.M., May, R.M., 1978. Regulation and stability of host–parasite population interactions. 1. Regulatory processes. J. Anim. Ecol. 47, 219–247.

Arneberg, P., Skorping, A., Grenfell, B., Read, A.F., 1998. Host densities as determinants of abundance in parasite communities. Proc. R. Soc. Lond. B: Biol. Sci. 265, 1283–1289.

Beckerman, A., Benton, T.G., Ranta, E., Kaitala, V., Lundberg, P., 2002. Population dynamic consequences of delayed life-history effects. Trends Ecol. Evol. 17, 263–269.

Benton, T.G., Ranta, E., Kaitala, V., Beckerman, A.P., 2001. Maternal effects and the stability of population dynamics in noisy environments. J. Anim. Ecol. 70, 590–599.

Bjornstad, O.N., Grenfell, B.T., 2001. Noisy clockwork: time series analysis of population fluctuations in animals. Science 293, 638–643.

Boag, B., Iason, G., 1986. The occurrence and abundance of helminth-parasites of the mountain hare *Lepus timidus* (L) and the wild rabbit *Oryctolagus cuniculus* (L) in Aberdeenshire, Scotland. J. Helminthol. 60, 92–98.

Bush, A.O., Lafferty, K.D., Lotz, J.M., Shostak, A.W., 1997. Parasitology meets ecology on its own terms: Margolis et al. revisited. J. Parasitol. 83, 575–583.

Cattadori, I.M., Boag, B., Bjornstad, O.N., Cornell, S.J., Hudson, P.J., 2005. Peak shift and epidemiology in a seasonal host–nematode system. Proc. R. Soc. Lond. B Biol. Sci. 272, 1163–1169.

Chapman, D.S., Cornell, S.J., Kunin, W.E., 2009. Interactions between harvesting, noise and territoriality in a model of red grouse population cycles. J. Anim. Ecol. 78, 476–484.

Clutton-Brock, T.H., Guinness, F.E., Albon, S.D., 1982. Red Deer: Behavior and Ecology of Two Sexes. Edinburgh University Press, Edinburgh.

Crofton, H.D., 1948. The ecology of immature phases of *Trichostrongyle* nematodes 2. The effect of climatic factors on the availability of the infective larvae of *Trichostrongylus retortaeformis* to the host. Parasitology 39, 26–38.

Flux, J.E.C., 1970. Life history of mountain hare (*Lepus timidus scoticus*) in northeast Scotland. J. Zool. 161, 75–123.

Ginzburg, L.R., Taneyhill, D.E., 1994. Population cycles of forest lepidoptera—a maternal effect hypothesis. J. Anim. Ecol. 63, 79–92.

Gulland, F.M.D., Albon, S.D., Pemberton, J.M., Moorcroft, P.R., Cluttonbrock, T.H., 1993. Parasite-associated polymorphism in a cyclic ungulate population. Proc. R. Soc. Lond. B: Biol. Sci. 254, 7–13.

Hewson, R., 1970. Variation in reproduction and shooting bags of mountain hares on 2 moors in north-east Scotland. J. Appl. Ecol. 7, 243–252.

Hewson, R., 1976. Population study of mountain hares (*Lepus timidus*) in northeast Scotland from 1956–1969. J. Anim. Ecol. 45, 395–414.

Harrison, A., Newey, S., Gilbert, L., Haydon, D.T., Thirgood, S., 2010. Culling wildlife hosts to control disease: mountain hares, red grouse and louping ill virus. J. Appl. Ecol. 47, 926–930.

Höglund, N.H., 1957. Fortplantningen hos skogsharen (*Lepus t. timidus* Lin.). Viltrevy, 267–282.

Hudson, P.J., Dobson, A.P., Newborn, D., 1998. Prevention of population cycles by parasite removal. Science 282, 2256–2258.

Iason, G.R., 1989a. Growth and mortality in mountain hares—the effect of sex and date of birth. Oecologia 81, 540–546.

Iason, G.R., 1989b. Mortality of mountain hares in relation to body size and age. J. Zool. 219, 676–680.

Iason, G.R., 1990. The effects of size, age and a cost of early breeding on reproduction in female mountain hares. Holarct. Ecol. 13, 81–89.

Inchausti, P., Ginzburg, L.R., 1998. Small mammals cycles in northern Europe: patterns and evidence for a maternal effect hypothesis. J. Anim. Ecol. 67, 180–194.

Kendall, B.E., Briggs, C.J., Murdoch, W.W., Turchin, P., Ellner, S.P., McCauley, E., et al., 1999. Why do populations cycle? A synthesis of statistical and mechanistic modeling approaches. Ecology 80, 1789–1805.

Krebs, C.J., Boonstra, R., Boutin, S., Sinclair, A.R.E., 2001a. What drives the 10-year cycle of snowshoe hares? Bioscience 51, 25–35.

Krebs, C.J., Boutin, S., Boonstra, R. (Eds.), 2001b. Ecosystem Dynamics of the Boreal Forest: The Kluane Project. Oxford University press, New York, p. 511.

Lindstrom, J., Kokko, H., 2002. Cohort effects and population dynamics. Ecol. Lett. 5, 338–344.

Lindstrom, J., Ranta, E., Kokko, H., Lundberg, P., Kaitala, V., 2001. From arctic lemmings to adaptive dynamics: Charles Elton's legacy in population ecology. Biol. Rev. 76, 129–158.

Lundberg, P., Ranta, E., Ripa, J., Kaitala, V., 2000. Population variability in space and time. Trends Ecol. Evol. 15, 460–464.

May, R.M., Anderson, R.M., 1978. Regulation and stability of host–parasite population interactions. 2. Destabilizing processes. J. Anim. Ecol. 47, 249–267.

Mougeot, F., Redpath, S.M., Leckie, F., Hudson, P.J., 2003. The effect of aggressiveness on the population dynamics of a territorial bird. Nature 421, 737–739.

Mougeot, F., Piertney, S.B., Leckie, F., Evans, S., Moss, R., Redpath, S.M., et al., 2005. Experimentally increased aggressiveness reduces population kin structure and subsequent recruitment in red grouse *Lagopus lagopus scoticus*. J. Anim. Ecol. 74, 488–497.

Mulvey, M., Aho, J.M., Rhodes, O.E., 1994. Parasitism and white-tailed deer—timing and components of female reproduction. Oikos 70, 177–182.

Newey, S., Dahl, F., Willebrand, T., Thirgood, S., 2007a. Unstable dynamics and population limitation in mountain hares. Biol. Rev. 82, 527–549.

Newey, S., Shaw, D.J., Kirby, A., Montieth, P., Hudson, P.J., Thirgood, S.J., 2005. Prevalence, intensity and aggregation of intestinal parasites in mountain hares and their potential impact on population dynamics. Int. J. Parasitol. 35, 367–373.

Newey, S., Thirgood, S., 2004. Parasite-mediated reduction in fecundity of mountain hares. Proc. R. Soc. Lond. B Biol. Sci. 271, S413–S415.

Newey, S., Willebrand, T., Haydon, D.T., Dahl, F., Aebischer, N.J., Smith, A.A., et al., 2007b. Do mountain hare populations cycle? Oikos 116, 1547–1557.

Press, W., Teukolsky, S., Vetterling, W., Flannery, B., 1992. Quasi- (that is, Sub-) Random Sequences. Numerical Recipes in C: The Art of Scientific Computing. Cambridge University Press, Cambridge, pp. 309–315.

Patton, V., Ewald, J.A., Smith, A.A., Newey, S., Iason, G.R., Thirgood, S.J., et al., 2010. Distribution of mountain hares *Lepus timidus* in Scotland: results from a questionnaire. Mammal Rev., 1–14.

Pugliese, A., Rosa, R., Damaggio, M.L., 1998. Analysis of a model for macroparasitic infection with variable aggregation and clumped infections. J. Math. Biol. 36, 419–447.

Redpath, S.M., Mougeot, F., Leckie, F.M., Elston, D.A., Hudson, P.J., 2006. Testing the role of parasites in driving the cyclic population dynamics of a gamebird. Ecology Letters 9, 410–418.

Rosa, R., Pugliese, A., 2002. Aggregation, stability, and oscillations in different models for host–macroparasite interactions. Theor. Popul. Biol. 61, 319–334.

Shaw, D.J., Dobson, A.P., 1995. Patterns of macroparasite abundance and aggregation in wildlife populations: a quantitative review. Parasitology 111, S111–S133.

Shaw, D.J., Grenfell, B.T., Dobson, A.P., 1998. Patterns of macroparasite aggregation in wildlife host populations. Parasitology 117, 597–610.

Sinclair, A.R.E., Chitty, D., Stefan, C.I., Krebs, C.J., 2003. Mammal population cycles: evidence for intrinsic differences during snowshoe hare cycles. Can. J. Zool. 81, 216–220.

Skorping, A., Read, A.F., Keymer, A.E., 1991. Life-history covariation in intestinal nematodes of mammals. Oikos 60, 365–372.

Townsend, S.E., Newey, S., Thirgood, S.J., Matthews, L., Haydon, D.T., 2009. Can parasites drive population cycles in mountain hares? Proc. R. Soc. Lond. B Biol. Sci. 276, 1611–1617.

Turchin, P., 2003. Complex Population Dynamics: A Theoretical/Empirical Synthesis. Princeton University Press, Princeton.

Watson, A., Hewson, R., Jenkins, D., Parr, R., 1973. Population-densities of mountain hares compared with red grouse on Scottish moors. Oikos 24, 225–230.