Journal of Animal Ecology

British Ecological Society

Journal of Animal Ecology 2014, **83,** 85–98 doi: 10.1111/1365-2656.12098

SYNTHESIS

Insights into population ecology from long-term studies of red grouse *Lagopus lagopus scoticus*

Jesus Martínez-Padilla[1]*†, Steve M. Redpath[2]†, Mohammed Zeineddine[2] and François Mougeot[3,4]

[1]*Museo Nacional de Ciencias Naturales, Department of Evolutionary Biology, José Guitérrez Abascal 2, 28006, Madrid, Spain;* [2]*ACES, Department of Biological Sciences, University of Aberdeen, Zoology Building, Tillydrone Av., Aberdeen AB24 2TZ, UK;* [3]*EEZA-CSIC, La Cañada de San Urbano, 04120, Almeria, Spain; and* [4]*IREC (CSIC-UCLM-JCCM), Ronda de Toledo s/n, 13005, Ciuada Real, Spain*

Summary

1. Long-term studies have been the backbone of population ecology. The red grouse *Lagopus lagopus scoticus* is one species that has contributed widely to this field since the 1950s. This paper reviews the trajectory and profound impact that these studies have had.
2. Red grouse research has combined long-term studies of marked individuals with demographic studies over wide geographical areas and replicated individual- and population-level manipulations.
3. A main focus has been on understanding the causes of population cycles in red grouse, and in particular the relative importance of intrinsic (behaviour) and extrinsic (climate, food limitation and parasite) mechanisms.
4. Separate studies conducted in different regions initially proposed either the nematode parasite *Trichostrongylus tenuis* or changes in male aggressiveness in autumn as drivers of population cycles.
5. More recent experiments suggest that parasites are not a necessary cause for cycles and have highlighted that behavioural and parasite-mediated mechanisms are interrelated. Long-term experiments show that parasites and aggressiveness interact.
6. Two outstanding questions remain to be tested experimentally. First, what intrinsic mechanism causes temporal variation in patterns of male aggressiveness? The current favoured mechanism is related to patterns of kin structuring although there are alternative hypotheses. Second, how do the dual, interacting mechanisms, affect population dynamics?
7. Red grouse studies have had an important impact on the field of population ecology, in particular through highlighting: (1) the impact of parasites on populations; (2) the role of intrinsic mechanisms in cyclic dynamics and (3) the need to consider multiple, interacting mechanisms.

Key-words: behaviour, host–parasite interactions, *Lagopus lagopus scoticus*, long-term studies, population cycles

Introduction

The field of population ecology is primarily concerned with explaining spatial and temporal variation in population size. Much of the initial research interest in understanding the factors limiting and regulating populations stemmed from observations of outbreaks of agricultural pests and the applied need to understand why populations of certain species erupted (Elton 1942; Berryman 2003; Turchin 2003). Similarly, managers of harvested species were keen to understand how they could maximize the abundance of their quarry and why certain species showed such a high degree of temporal variation. Studies on population size variation, particularly in species of economic relevance, have fed considerable controversy and argument over the last hundred years. Disagreements have erupted over the relative role of biotic and abiotic factors,

*Correspondence author. E-mail: j.mart@mncn.csic.es; jmartinezpadilla12@gmail.com
†Both authors contributed equally.

over whether the approach to understanding population regulation should be focused on variation in density or on the mechanisms that drives it and over the relative role of different processes (Sinclair 1989; Krebs *et al.* 1995; Sinclair & Arcese 1995; Krebs 2002; Sibly & Hone 2002).

Our understanding of population regulation has been illuminated by detailed, long-term studies of species with unstable dynamics such as moths, voles, lemmings, hares and grouse (Stenseth, Bjornstad & Falck 1996; Liebhold & Kamata 2000; Moss & Watson 2001; Berryman 2002; Korpimäki *et al.* 2002; Klemola *et al.* 2003; Krebs 2011). These studies have been complemented by increasingly sophisticated and elegant modelling approaches. This interest in unstable dynamics partly stems from an intrinsic curiosity in understanding the causes of cyclic dynamics and partly for the more prosaic reason that population size varies greatly, and this variation increases the strength of the signal in the data and thus the ability of unstable dynamics to reveal putative regulatory mechanisms.

Box 1 Definitions

Intrinsic

In the population ecology literature, intrinsic has two broad definitions. The first definition goes back to the work of Nicholson (1933) and Andrewartha & Birch (1954) and the subsequent debate on the role of intrinsic, or endogenous, factors as opposed to extrinsic, or exogenous, environmental variation in determining population dynamics. By this definition, intrinsic refers to any density-dependent process. A second, widely used definition refers to a subset of these exogenous factors. By this definition, intrinsic refers to processes acting within the population to influence dynamics, such as genetic structure or behaviour. Other trophic interactions are referred to as extrinsic factors. It is this latter definition we use here.

Territorial behaviour, spacing behaviour, aggression and aggressiveness

In this paper, we use the term 'aggressiveness' to indicate the frequency or intensity of aggressive behaviour by grouse towards other individuals (Moss *et al.* 1979). We use the term 'territorial behaviour' to refer to the behaviours used by grouse to obtain and maintain a territory. This territorial behaviour results in a spatial pattern of territories within a population and so is also sometimes referred to as spacing behaviour, but to prevent confusion, we avoid the use of this term in this paper.

Studies of unstable dynamics have focused largely on trophic interactions (Berryman 2003; Turchin 2003) and on the interaction between these and climate (Ranta, Lindstrom & Linden 1995; Grenfell & Finkenstadt 1998; Stenseth 1999; Cattadori, Merler & Hudson 2000; Bjornstad *et al.* 2002; Cattadori, Haydon & Hudson 2005). Whilst the focus has been revealing, it has led to intrinsic mechanisms (i.e. processes acting within the population to influence dynamics, such as genetic structure or behaviour; see Box 1) being ignored or dismissed. In two books reviewing case studies, the emphasis was on a predominant role of trophic mechanisms as drivers of population cycles (Berryman 2003; Turchin 2003). Indeed, Turchin (2003) stated: 'There is not a single case where an intrinsic hypothesis has provided a theoretically sound and empirically supported explanation of complex dynamics in nature'.

The red grouse *Lagopus lagopus scoticus* has provided a battleground for some of these ideas and debates. Arguments have stimulated a large body of work that has been sometimes contradictory but ultimately revealing. In this review, we briefly summarize the history of red grouse research, draw together this body of work, critically review alternative hypotheses put forward to explain unstable dynamics and consider the implications of this work for population ecology. Our focus is on population studies and in particular the mechanisms that regulate red grouse abundance. The aims of this review are to (1) briefly describe red grouse biology, (2) provide a historical overview of red grouse research from late 1950s, (3) examine the evidence for the hypotheses put forward to explain unstable population dynamics and (4) consider the impact on population ecology and the way forward.

Red grouse biology and population dynamics

Red grouse is a subspecies of the widely occurring Willow Ptarmigan (*Lagopus lagopus*). Willow ptarmigan are widely distributed in the arctic, boreal and temperate regions of the Northern Hemisphere, with 19 different subspecies (Watson & Moss 2008). Willow ptarmigan have been extensively studied World-wide (see Watson & Moss 2008), but for the purpose of this review, we will focus solely on the red grouse (*Lagopus lagopus scoticus*). This subspecies inhabits the heather *Calluna vulgaris* dominated moorlands of upland Britain, is territorial and generally monogamous and produces one brood a year of up to 14 young (Watson, Moss & Rae 1998; Hudson *et al.* 2002). Males establish territories in the autumn and defend them through the winter, unless forced to abandon the territory by adverse weather conditions. They attract females over the winter and pairs become settled on territories in early spring, with eggs being generally laid in April. Territories are vigorously defended during spring. Males are involved in chick rearing through the summer when the territory structure breaks down and broods wander more widely in search of food. Grouse predominantly feed on heather, although other plants such as Cotton Grass *Eriophorum spp.* and Bilberry *Vaccinium myrtilus* are seasonally important. Invertebrates are also

important during the first few weeks of the chicks' life. More detailed descriptions of red grouse biology can be found in Watson & Moss (2008) .

Much of the heather dominated moorland in northern England and southern, central and eastern Scotland is managed for the production and harvesting of red grouse. Private estates employ gamekeepers to maximize numbers of grouse and the birds are shot during the autumn, with the season starting on the 'Glorious 12th' of August. Grouse management involves three main activities (Hudson & Newborn 1995). The heather is burnt in strips to provide nutritious food, the predators are killed (primarily red fox *Vulpes vulpes,* stoat *Mustela erminea* and crow *Corvus corone*), and increasingly, parasites are controlled, either directly, through catching and treating grouse with anthelminthic, or indirectly through provision of 'medicated grit' (grit coated with anthelminthic) that the grouse eat (Newborn & Foster 2002). Red grouse is subject to a variety of parasites, including the sheep tick *Ixodes ricinus,* the nematode *Trichostrongylus tenuis,* the tapeworms *Hymenolopis microps* and *Divainea urogalli* as well as coccidia (Hudson 1986a; Delahay, Speakman & Moss 1995; Laurenson *et al.* 2004; Mougeot, Redpath & Piertney 2006; Martinez-Padilla *et al.* 2007). Ticks are vectors for the Louping ill virus which can cause up to 78% of chick mortality in red grouse (Reid 1975; Hudson *et al.* 1995; Laurenson *et al.* 2003; Moseley *et al.* 2007).

Trichostrongylus tenuis nematodes are virtually ubiquitous in adult grouse (Hudson, Dobson & Newborn 1992). Adult worms inhabit the large caeca of the grouse and lay eggs that are passed in the faeces. The eggs then hatch on the moor and the infective larvae migrate of the tips of the heather plant where they are eaten by the grouse. Transmission mode is therefore indirect, although there is no intermediate or alternative host in this system (Hudson 1986a; Watson & Moss 2008).

Bag records from red grouse shooting have been analysed in detail (Mackenzie 1952; Potts, Tapper & Hudson 1984; Williams 1985; Haydon *et al.* 2002; Shaw *et al.* 2004). These have shown that numbers of grouse shot have declined over the last 100 years, that average numbers shot vary consistently between estates and that populations of red grouse are inherently unstable (Fig. 1), with population size fluctuating over time (Haydon *et al.* 2002). It is this latter pattern that has been the focus of much of the research. Population cycles or quasi cycles (Potts, Tapper & Hudson 1984) are evident from most (63%) long-term time series in red grouse (Haydon *et al.* 2002). Within regions, population cycles are fairly synchronized (Cattadori, Haydon & Hudson 2005), but their shape and periodicity are more characterized by their dynamic heterogeneity than by their regularity (Haydon *et al.* 2002; Shaw *et al.* 2004). Specifically, there is a weak tendency for cycle period to increase with latitude (Haydon *et al.* 2002; Shaw *et al.* 2004) and for noncyclic

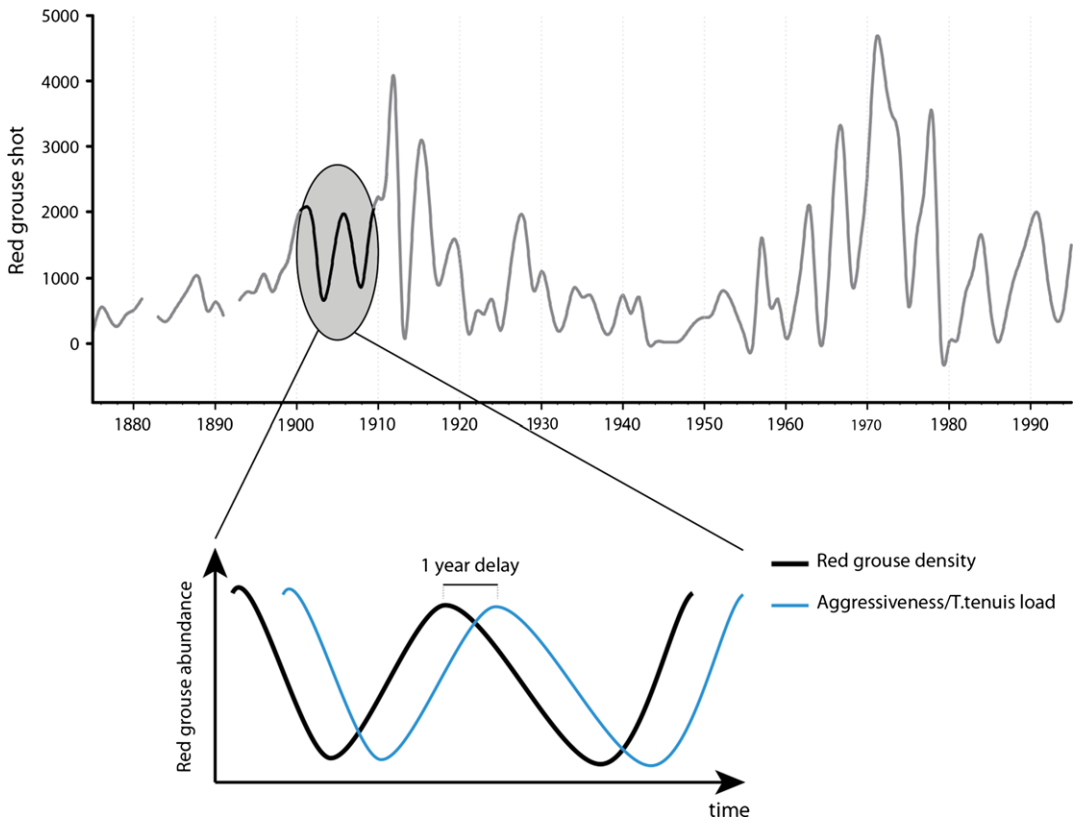

Fig. 1. Example of a typical long-term time series of red grouse harvest records, showing fluctuations in abundance (above), and of delayed density dependence in a regulatory mechanism (aggressiveness or parasites) during a population cycle.

populations to be more prevalent at high altitudes (Shaw *et al.* 2004). Red grouse cyclic fluctuations differ from those of arvicoline (microtine) rodents, a group traditionally targeted in studies of unstable population dynamics. Analyses of long-term field vole *Microtus agrestis* data, for example, suggest more temporally (periodicity and amplitude) and spatially consistent (travelling waves) population cycles than in red grouse (Lambin *et al.* 1998; Bjornstad, Ims & Lambin 1999; Hanski *et al.* 2001; Bierman *et al.* 2006).

Short history of red grouse research

Grouse population research started at Aberdeen University in north-east Scotland in the late 1950s, led by Vero Wynne-Edwards, David Jenkins and Adam Watson. A decade later, the group moved to Banchory (Scotland) along with Robert Moss, to be part of the Nature Conservancy, which became the Institute of Terrestrial Ecology (ITE) in 1972, and then the Centre for Ecology and Hydrology (CEH), before being shut in 2007. The long-term studies of grouse developed by this group were not carried out on a single site, but moved between different estates in Scotland (Millden, Angus 1956–1962, Glen Dye, Kerloch, 1962–78 Rickarton, 1979–1989 Glen Tanar, 1992–1993 Glas Choille, Edinglassie and Invercauld 1993–2003) and lately in northern England (Moorhouse and Catterick), managed or not for grouse shooting. Recent experiments, once again out of the University of Aberdeen, have been performed over shorter time periods on a variety of sites in Scotland and England. Dick Potts and Peter Hudson started working on red grouse in northern England in 1979 (Gunnerside in Swaledale) for the Game Conservancy Trust (now the Game and Wildlife Conservation Trust – GWCT). Their work subsequently moved to Scotland. The GWCT hold long-term, grouse bag records based on data sent in by estates from across upland Britain.

The work of the Banchory team tended to focus on specific study areas, ranging in size from 140 ha (Glas Choille) to 460 ha (Glen Esk). GWCT worked intensively on 100 ha areas, but combined this with long-term monitoring data from a larger spatial scale from across the uplands. Although the initial impetus for the Banchory work was grouse management (Jenkins 1970), the research soon moved to addressing fundamental ecological questions. The focus for GWCT has always been primarily on red grouse management and partly as a result, the two teams explored different aspects of grouse biology. Stemming from their early observations, Banchory research focused primarily on the role of intrinsic mechanisms in explaining variation in spring breeding numbers, particularly of males and their overwinter survival. In contrast, GWCT research focused on the role of parasites in explaining variation in autumn numbers and numbers shot, with a focus on females and their reproductive success. Both groups developed theoretical modelling to explore the causes of variation of population cycles in red grouse (see for example, Dobson & Hudson 1992; Moss, Watson & Parr 1996; Matthiopoulos *et al.* 2003; further references below). This has produced supporting evidence and provided important insights for different ideas on population ecology, yet as we shall see the findings and conclusions of the two groups have contrasted dramatically. In summary, red grouse research has been carried out during the last 60 years mainly based on population dynamics. Still, even after that period, there is no clear consensus regarding the mechanisms that cause of population cycles in red grouse.

Why do red grouse populations cycle?

There are a variety of possible mechanisms that have been considered to explain why red grouse populations show cyclic dynamics. Over the last 60 years, all but two of these have now been dismissed (Moss & Watson 2001 – figure 2). Variation in predation has been rejected as a possible mechanism primarily because grouse populations cycle in the absence of predators (Watson & Moss 2008). However, under specific circumstances, increases in generalist predators can dampen cyclic dynamics and limit populations at low density (Thirgood *et al.* 2000). Food quality has also been rejected as grouse take a very small proportion of available food and experiments showed that improving food quality is unable to prevent a cyclic decline (Watson, Moss & Parr 1984). Similarly, changes in maternal nutrition have been rejected (Moss, Watson & Parr 1996). There is evidence to suggest that weather affects demographic traits (Watson & Moss 2008), spatial synchrony in population dynamics (Cattadori, Haydon & Hudson 2005) and may even lead to cycle entrainment in high altitude sites with poor soils (Watson, Moss & Rothery 2000). However, there is no evidence to suggest that variation in climate is the primary cause of cyclic dynamics. Hunters can shoot large numbers of grouse in the autumn, but although some of this mortality may be additive (Hudson 1986b) and high levels of harvesting may affect cycle amplitude (Bunnefeld *et al.* 2009; Chapman, Cornell & Kunin 2009a), there is no evidence to suggest that harvesting could be the cause of the instability in red grouse populations. Amongst other potential causes, two mechanisms have received empirical and experimental support – parasites and territorial behaviour (Fig. 2).

EXTRINSIC MECHANISM – PARASITES

The link between grouse disease and population crashes was long recognized by game managers, but it was Cobbold (1873) and later Lovat (1911) who associated grouse disease with the caecal threadworm *T. tenuis*. Much later, Wilson & Wilson (1978) infected captive red grouse with *T. tenuis* and found that infection led to lower egg production in red grouse and, at high intensi-

ties, death. Potts, Tapper & Hudson (1984) examined data from shot grouse and found an inverse correlation between adult worm burdens and the ratio of young to old grouse in August, a measure of productivity. They concluded that population cycles could be caused by *T. tenuis* operating together with stochastic factors.

At the same time as Hudson and colleagues started working on *T. tenuis* in northern England, Anderson and May published their influential papers on the population biology of infectious diseases (Anderson & May 1978; May & Anderson 1978; Hudson, Dobson & Newborn 1992). These papers greatly influenced subsequent work on the red grouse – *T. tenuis* system. The models of Anderson and May highlighted conditions under which transmitted parasites were likely to lead to population cycles: i) parasites should show relatively low aggregation within hosts, ii) parasite impact on host fecundity should be greater than its impact on host mortality and iii) transmission or reproduction of parasites should show a time-lag.

The red grouse has provided a valuable field test of these models. These conditions were examined using the data from Gunnerside and supporting evidence for each was found (Hudson, Dobson & Newborn 1992). The parasite showed low levels of aggregation (Hudson, Dobson & Newborn 1992), strongly affected grouse productivity (Hudson 1986a; Shaw & Moss 1990; Hudson, Dobson & Newborn 1992, 1998; Newborn & Foster 2002; Redpath *et al*. 2006) and showed evidence of arrested development leading to a time delay of up to six months (Shaw 1988; Shaw & Moss 1989b). The parasite also affected grouse survival (Hudson, Dobson & Newborn 1992, 1998), and a mathematical model suggested that the principal cause of the instability in grouse population dynamics was caused by the fact that *T. tenuis* had a greater impact on fecundity than mortality (Dobson & Hudson 1992). Thus, the empirical data and a modified version of the Anderson–May model were consistent with *T. tenuis* being a potential cause of red grouse population cycles in the north of England.

A population-level experiment was later performed to test the parasite hypothesis (Hudson, Dobson & Newborn 1998). This experiment was done at the scale (10s km^2) at which long-term indices of autumn density were recorded through harvested grouse and the nature of the population cycles described. The study involved six different estates monitored over 10 years and focused on the impact of parasites on grouse bags. Hudson and colleagues used long-term bag data to predict when population crashes would occur. They then worked with gamekeepers on these estates to catch and orally dose between an estimated 15–50% of the grouse with an anthelmintic to kill the parasites in late winter and early spring of the crash years. On two estates grouse were caught and dosed before the first crash, on two other estates grouse were dosed before both crashes, and the remaining two estates acted as controls. The experiment showed that the amplitude in the cyclic dynamics of the number of grouse shot in autumn was reduced, as predicted from a model and the authors concluded that the 'treatment of the grouse population prevented population crashes, demonstrating that parasites were the cause of the cyclic fluctuations' (Hudson, Dobson & Newborn 1998, page 2256). They went on to say that the findings: 'also show that intrinsic mechanisms do not need to be evoked as a cause of cyclic fluctuations in grouse abundance' (Hudson, Dobson & Newborn 1998, page 2258).

This experiment has received considerable interest and been widely cited. May (1999) commended the scale of the experiment and concluded that Hudson and colleagues had finally settled the debate about the causes of grouse cycles. Others were less enthusiastic. Indeed, there was substantial debate about the conclusions drawn from the experiment (Lambin *et al*. 1999; Tompkins & Begon 1999; Moss & Watson 2001; Turchin 2003). There were two main criticisms. First, grouse densities were estimated from harvest records, and in some years, no grouse were shot, so the magnitude of the treatment effect was less clear than at first seemed apparent (see Watson & Moss 2008). Second, cyclic fluctuations in treated populations still occurred, and it was not possible to determine whether the remaining parasites caused the cycles, as argued by the authors, or whether the underlying causes lay elsewhere. In any event, it seems clear that an alternative mechanism could not be so easily dismissed.

In the light of these concerns, a similar, replicated population experiment was later carried out on four estates; two in England and two in Scotland (Redpath *et al*. 2006). The main differences here were that the experiment was based on count data and done over 100 ha areas, rather than whole estates. This time the experiment was initiated at peak density and up to 81% of birds were treated with anthelmintic in each winter of the predicted decline (during two years in England and four years in Scotland). The findings showed that whilst parasite reductions increased grouse productivity and postbreeding densities overall, they were unable to prevent a decline in breeding density the following year. The treatment did reduce the extent of the decline in autumn density (which mostly depends on productivity) in England, but not in Scotland. The inference drawn from this experiment was that parasites alone were not responsible for the cyclic declines witnessed in our study areas – some other factor was operating, influencing changes in density between autumn and spring. The findings also pointed to the fact that parasites had more effect on numbers in autumn than spring and that parasites may have a greater role to play in England than in north-east Scotland.

The limited impacts of parasites have also been highlighted in other studies and suggest that parasites do not seem necessary for cycles to occur. Both temperature and humidity are known to affect worm fecundity, the survival of infective larvae and the effectiveness of parasite transmission (Shaw 1988; Shaw & Moss 1989a; Shaw, Moss & Pike 1989; Moss *et al*. 1990). Temperature has been shown

to affect the proportion of parasite eggs that hatch and survive (Shaw & Moss 1989b), and humidity affects the ability of parasite larvae to move freely (Shaw 1988). Moss *et al.* (1993), Moss, Watson & Parr (1996) showed that variation in parasite transmission was a function of rainfall rather than grouse density and that a population cycle occurred in the presence of relatively low numbers of parasites with no evidence of delayed density dependence in parasite abundance. Hudson, Dobson & Newborn (1985) highlighted that levels of infection were associated with rainfall in northern England and that grouse populations on the drier moors in north-east England showed a reduced propensity to cycle. More recently Cattadori, Haydon & Hudson (2005) examined bag data in relation to weather and their model suggested that summer temperature and rainfall can play an important role in synchronizing population cycles within regions through their impact on parasites. There is, however, another test of the parasite hypothesis currently being carried out across the grouse moors of upland Britain due to changes in management practices. As part of their management, gamekeepers now distribute medicated grit in small piles across the moorland. This is now effective at reducing parasites and improving grouse breeding success (Newborn & Foster 2002) and is occurring at very large scales. We wait to see its impact on red grouse population dynamics.

In conclusion, parasites clearly have a destabilizing role in grouse population dynamics, primarily through their impact on female reproductive success. There is evidence to suggest that their effects on dynamics will be greater in autumn than spring and be greater in warmer, more humid conditions (such as on the blanket bogs of northern England). However, there is also strong evidence to suggest that parasites are not necessary for grouse population cycles.

INTRINSIC MECHANISM – TERRITORIAL BEHAVIOUR

The early ideas formulated on the role of intrinsic mechanisms in red grouse populations followed the work of Errington (1945, 1956), Chitty (1960), Jenkins, Watson & Miller (1963) and later Charnov & Finnerty (1980). Jenkins, Watson & Miller (1963) examined the relationship between parasites and grouse abundance at Glen Esk, but concluded that disease did not regulate breeding numbers, as parasites were considered to kill mainly surplus, nonterritorial birds. Parasites were seen as a consequence of cycles, rather than their cause. Instead, the authors inferred that breeding numbers in spring were limited by territorial behaviour during the autumn (Watson & Jenkins 1968). This idea was further supported by removal experiments performed at Glen Esk and Kerloch which showed that a territory was essential for breeding (see also Watson & Jenkins 1968; Watson 1985), although this was later disputed by Hudson, Dobson & Newborn 1992; examining data from low-density grouse populations. Jenkins, Watson & Miller (1963) also put forward the idea that the aggressiveness involved in territorial defence may vary between years (also known as spacing behaviour see Box 1). Over the next 47 years, this research group focused primarily on understanding cause and effect in these interannual variations in aggressiveness and sought to refute their putative ideas through experimentation.

The initial idea that aggressiveness can have fundamental effects on population dynamics has been explored through a series of later papers. The role of aggressiveness in limiting density was tested by Moss, Parr & Lambin (1994). They caught territorial males in spring and implanted a small amount of testosterone into some males to increase their aggressiveness. They found that those males then took larger territories, evicted neighbouring males and thereby reduced density, providing clear evidence that aggressiveness limits density by affecting territory size. Whilst this finding is relatively uncontroversial, the idea that changing patterns of aggressiveness can regulate populations and lead to cycles has been contested (Turchin 2003).

Evidence for delayed density dependence in aggressiveness was initially given by Watson *et al.* (1994), who analysed field data over two population fluctuations. These analyses suggested that aggressiveness (measured as numbers of song flights and territorial boundary disputes) varied with density, but was delayed by a year. Thus, male aggressiveness tended to reach its maximum a year after peak densities. This pattern was later supported by Moss, Watson & Parr (1996), who measured the size of the androgen dependent, supra-orbital combs of male grouse. Similar changes in autumn comb size during a population cycle were found in a later study (Piertney *et al.* 2008). These observations supported the idea that there was delayed density dependence in the levels of aggressiveness using comb size as a proxy in male grouse during the autumn territorial contests. The next question was whether or not these changes in aggressiveness were regulating density and causing cycles. This was tested in a replicated population-level experiment (Mougeot *et al.* 2003a, b) and modelled (Matthiopoulos *et al.* 2003). Adult territorial males were caught during autumn and implanted with testosterone to increase their aggressiveness for 2–3 months. The experiment demonstrated that increased aggressiveness reduced recruitment of young males into the population and led to a decline in breeding density the following spring. Moreover, males continued to be aggressive and maintain large territories for at least a further year, showing that there was memory or time-lag in the system (Mougeot *et al.* 2003b). Thus, changes in autumn aggressiveness not only affected breeding density the following spring but aggressiveness in the following autumn, supporting the idea that delayed-density-dependent changes in aggressiveness can lead to population cycles.

Although the evidence that aggressiveness has fundamental effects on grouse population dynamics is strong, the question of what specific mechanism causes these

changes in aggressiveness remains open. Dennis Chitty (1967) proposed that at high densities genetically more aggressive animals should be at an advantage and therefore selected for. Increased competition at high density would lead to reduced recruitment and a declining population of aggressive animals. At lower density, there is then an advantage for subordinate individuals, eventually leading to population increase. Robert Moss and colleagues tested this idea in a 9-year study at Kerloch from 1969 to 1977 (Moss & Watson 1980; Moss, Watson & Rothery 1984). They monitored a population through a cycle and took eggs into captivity in each year, which they hatched and raised to produce chicks in the following year. Their data were at odds with the Chitty hypothesis. The evidence suggested that there was selection for subordinate types during the increase and peak phases, reaching a maximum one year after peak density. Selection for dominant types then occurred during the decline. The authors concluded that variation in tolerance behaviour was important. During the increase phase, birds were tolerant of each other, enabling recruitment of subordinate types, but birds then switched to being intolerant causing population declines attributed to reduced recruitment and increased emigration.

From this study, Moss, Watson & Rothery (1984) developed the idea that 'Tolerance is thought to change to intolerance above a certain threshold of density, when the strategy of taking smaller territories becomes maladaptive; the rate of emigration then increases and numbers decline'. They devised an elegant experiment to explore this idea at Rickarton (Moss, Watson & Parr 1996). They used previous data on grouse numbers to predict future changes in the grouse population on an experimental and a control area. The year before the predicted peak in abundance and all subsequent years they removed a few male territorial grouse from the population to prevent peak densities from being reached. Changes in the numbers of birds on the control area were very similar to the predictions. However, the experimental area did not enter a cyclic decline, but stayed at a similar density for five years, until the removals stopped. The simple inference was that some process was acting in a delayed-density-dependent fashion to cause cyclic changes in abundance. By preventing the population reaching the peak density, the delayed-density-dependent process did not act on the population to drive it down. So what was the suspected process? Moss, Watson & Parr (1996) showed data on parasite numbers which showed no evidence of tracking numbers of grouse, and they inferred that the process was most likely an intrinsic one. The demographic patterns observed during the experiment suggested that the main cause of population change was variation in the recruitment of young cocks.

Watson, Moss & Parr (1984) took up the ideas first raised by Charnov & Finnerty (1980) to provide a mechanism to explain red grouse population cycles. They explicitly suggested that kin selection involved differential aggressive behaviour towards kin and nonkin, influencing recruitment leading to population cycles – later termed the kinship hypothesis. Put simply, the hypothesis states that during the increase phase of a cycle, cocks are less aggressive towards kin, so clusters of related individuals build up and facilitate recruitment. As numbers rise, space becomes limited; tolerance amongst relatives declines, and recruitment is reduced until it no longer compensates for mortality and the population declines. The degree of relatedness between neighbours declines, maintaining high levels of aggressiveness and driving density into a trough and so the cycle continues. Using previously collected data from Glen Esk and Kerloch, Watson, Moss & Parr (1984) found that sons tended to settle close to their fathers, especially during years of population increase, and that there was less aggression (territorial song flights and fighting) between kin than between nonkin. As they pointed out, these observations were consistent with the kinship hypothesis, but they did not prove it. Modelling by Mountford *et al.* (1990), Hendry *et al.* (1997) and Matthiopoulos, Moss & Lambin (1998, 2000, 2002) and field-based data analyses from MacColl *et al.* (2008), Mougeot *et al.* (2005a) and Piertney *et al.* (2008) supported the idea that kinship could lead to realistic cyclic population dynamics. The kinship hypothesis has a number of population-level testable predictions, which Piertney *et al.* (2008) verified through field observational data from one population at Glas Choille over the course of an 8-year cycle. Consistent with the hypothesis, the size of related groups of males (kin clusters) built up during the increase phase of a cycle, but broke down prior to maximum density. In addition, the size of combs in autumn was negatively related to kin-clustering levels, and recruitment was positively related to kin clusters (Piertney *et al.* 2008). However, not all evidence is supportive of the hypothesis. In a recent study, Vergara *et al.* (2012) found that mean comb size and population density across populations can sometimes be negatively correlated, whereas according to the kinship hypothesis, the two should be positively associated with a time-lag. This may be in part because testosterone levels and comb size are not always directly related. In populations where males are more aggressive (high density populations), there is evidence to suggest that subordinate individuals may increase their testosterone levels, but not their comb size (Vergara & Martinez-Padilla 2012). Within the kinship hypothesis, it suggests that caution should be taken when suggesting that comb size is a proxy of aggressiveness because smaller combs might not mean a low level of testosterone and aggressive behaviour.

Population-level experiments also showed patterns consistent with the kinship hypothesis (Mougeot *et al.* 2005a). In the spring following the experimental increase in aggressiveness, levels of kinship were reduced, and in the following autumn, recruitment was still depressed. Therefore, males continued to be more aggressive long after the initial manipulation, and this 'memory' of previous territorial

contests could be held in the kin structure of male populations. With this wealth of theoretical and observational support, the hypothesis would seem to be compelling. However, the ultimate experimental manipulation of kin groups to test the kinship hypothesis is still awaited.

In conclusion, there is persuasive evidence that aggressiveness provides us 'a theoretically sound and empirically supported explanation of complex dynamics in nature'. We therefore disagree with Turchin's (2003) conclusion. However, we still await a population-level experimental test of the precise mechanism that causes the changing patterns in aggressiveness. The evidence so far suggests that an experiment aimed at refuting the kinship hypothesis would be most fruitful. In particular, individual-level-based studies should explore the fitness consequences for young individuals of being established besides their first-order relatives (i.e. inclusive fitness) and experimentally explore how aggression levels of given individuals change when their sons are removed (and not aggressiveness modified by means of testosterone implants). In addition, parasite susceptibility increases with aggressive behaviour (Mougeot *et al*. 2005b), so it is possible that kin-mediated aggressiveness may increase parasite susceptibility of young individuals, giving scope for potential interactive effects between intrinsic and extrinsic mechanisms. It would shed new light onto the reasons why males switch behaviour, from tolerant to aggressive and back, during the course of cycles.

INTERACTIONS BETWEEN INTRINSIC AND EXTRINSIC MECHANISMS

Intrinsic and extrinsic processes do not operate in isolation (Fig. 2). Not only is there good evidence that both parasites and aggressiveness are operating in natural populations, but there is broad theoretical and empirical support for interactions between aggressiveness and parasites (Folstad & Karter 1992; Hillgarth & Wingfield 1997). Four separate experiments in red grouse have now shown that parasites and aggressiveness do indeed interact and interestingly they do so in two opposing ways (Fox & Hudson 2001; Mougeot, Evans & Redpath 2005; Seivwright *et al*. 2005).

Fox & Hudson (2001) manipulated parasite intensities in males in early winter, through dosing with anthelmintic, and monitored their aggressiveness in the following spring. They found that treated males won more contests and were more aggressive to playbacks than control birds. These findings were partly supported by a later, similar experiment, although this time done in autumn, during the period of territory establishment (Mougeot, Evans & Redpath 2005). Experimental data also pointed to increased aggressiveness in parasite-purged birds, but only in old red grouse. Young males held lower intensities of *T. tenuis*, suggesting that their autumn territorial behaviour was not as limited by parasites. During years of high parasite numbers, old males may be more affected than young males, and this could facilitate the recruitment of young males.

Seivwright *et al*. (2005) manipulated aggressiveness in autumn using testosterone implants, removed parasites and then re-infected males with a standard dose of larvae. They then compared subsequent parasite intensities over the following year and found that males with increased testosterone ended up with almost twice the number of parasites as control birds, a difference only apparent after one year. The authors speculated that the delay in response was due to larvae delaying development until the following spring/summer. Mougeot, Evans & Redpath (2005) then showed that variation in susceptibility to the larvae, rather than increased exposure, was the likely cause of the difference in parasite intensities. What remains untested, however, is the effect of parasites on testosterone levels, the triggering mechanism assumed for aggressiveness in red grouse. A recent meta-analysis reviewing evidence on captive animals sup-

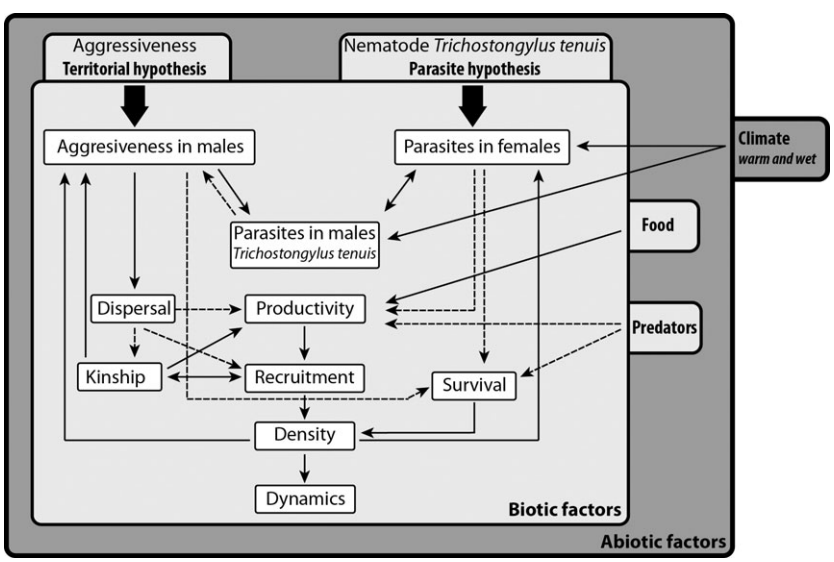

Fig. 2. Summary of how the two destabilizing processes act on red grouse populations. The intrinsic process (male aggressiveness) and the extrinsic process (parasites) have direct effects on demographic processes and also interact. Bold lines indicate positive effects and dashed lines negative ones (see text for further details).

ports the idea that immune suppression may indeed lower testosterone levels (Verhulst, Dieleman & Parmentier 1999).

Finally, the interaction between testosterone and *T. tenuis* parasites was tested by measuring gene expression levels. After describing which genes were normally expressed by red grouse in response to *T. tenuis* infection (Webster *et al.* 2011a), a factorial experiment was conducted on males in autumn to test whether the genes normally expressed in response to the parasite were also suppressed by testosterone (Webster *et al.* 2011b). Indeed, almost all (51 out of 52) of the transcripts expressed in response to the parasite were down-regulated at high testosterone titres. Therefore, the normal response that a grouse would raise to a challenge by *T. tenuis* is not achieved under conditions of high testosterone titre. When testosterone levels are low, males should therefore be better able to raise an appropriate response to the parasite. In contrast, at higher testosterone levels, when densities are high and there are usually more parasites, males will be less capable of raising the necessary response, which may cause further increases in parasite burdens (Seivwright *et al.* 2005).

Whilst there is good evidence for testosterone–parasite interactions in individual males, what has been missing for coupling both the intrinsic and extrinsic hypotheses is a link between parasite abundances in males and in females. The 'parasite hypothesis' suggests that *T. tenuis* parasites reduce female fecundity and recruitment (Hudson 1986a; Hudson, Dobson & Newborn 1998; Redpath *et al.* 2006). The 'territorial behaviour' hypothesis suggests that male aggressiveness increases the probability of territory establishment in young males (Mougeot *et al.* 2003a,b). We know that there are important interactions between the two processes in males (Fox & Hudson 2001; Mougeot, Evans & Redpath 2005; Seivwright *et al.* 2005). We also know that there is a positive association between parasite abundance in males and females within pairs in spring (Martinez-Padilla *et al.* 2012). In addition, experimental manipulation of parasite burdens in one member of the pair influences parasite intake rate in its mate during the breeding season, most likely via territory sharing and shared exposure to infective larvae (Martinez-Padilla *et al.* 2012). This finding suggests that males should be considered in the parasite hypothesis, because their parasite abundance can be directly affected by the parasite abundance of their mates. Indeed, males paired with highly parasitized females may amplify parasite abundance in the population and thereby alter male aggressive behaviour. This work suggests that an exploration of the sex-dependent roles in driving cyclic dynamics might prove fruitful.

In conclusion, there is now clear evidence that the two mechanisms, parasites and aggressiveness, are operating and can destabilize the dynamics in this system. It is also clear that the two mechanisms are interacting. Ultimately, further experiments will be required to quantify the impact of single vs. dual, interacting processes on population dynamics.

POPULATION MODELS

A variety of grouse population models have been developed to explore parasite-regulated birth and death rates (Dobson & Hudson 1992), aggression-dependent recruitment rates (Matthiopoulos, Halley & Moss 2005), kin-structuring dynamics (Mountford *et al.* 1990; Matthiopoulos, Moss & Lambin 1998, 2002; Matthiopoulos *et al.* 2003), environmental stochasticity (Chapman *et al.* 2009b) and effects of combined parasite and aggressiveness mechanisms (New *et al.* 2009).

These models have been parameterized with empirical measurements and have provided valuable insights and guided a series of important experiments, as outlined above. However, there are some important distinctions between approaches. Dobson & Hudson (1992) developed a continuous-time parasite model focused on female grouse. In contrast, the other models have been in discrete time and focused on male density. The parasite model has been criticized for having some unrealistic assumptions and for being highly sensitive to initial conditions (Matthiopoulos, Moss & Lambin 1998; Moss & Watson 2001). Similarly, a central assumption of the territorial behaviour models, that of very low overwinter survival of nonterritorial birds, has also been disputed (Hudson, Renton & Dalby 1988; Hudson & Newborn 1990). One other distinction is that the models have been parameterized using data from distinct regions, which highlights a limitation to models based on data from one area in that they are suited well to the environments they are developed in, but may lack the flexibility to accurately account for or capture the full range of dynamics that is observed in grouse populations across the UK.

One of the challenges facing ecologists today lies in explaining the spatial diversity in dynamics. In the case of the grouse system, we know from analyses of long-term time series that dynamics vary substantially across space (Haydon *et al.* 2002). What is also clear is that current single-process models are poor at explaining this variation, as they were not developed to address this question. We hypothesize that incorporating the two mechanisms and taking into account how they interact may better explain the spatial variation in grouse dynamics.

Discussion

Long-term, individual-based studies offer enormous insight into ecology and evolutionary biology (Clutton-Brock & Sheldon 2010). They provide in-depth understanding of demography and the role of specific mechanisms in affecting abundance. They also permit questions in population ecology to be addressed alongside shorter-term questions in fields such as behavioural, physiological or molecular ecology (Moss & Watson 1991). Yet, such longitudinal, in-depth studies do not sit easily with the type of population-level experiments required to understand the role of different mechanisms in limiting

and regulating abundance. Red grouse studies instead have combined individual-level studies with large-scale, replicated population-level experiments manipulating putative causal factors (Watson, Moss & Parr 1984; Moss, Parr & Lambin 1994; Moss, Watson & Parr 1996; Hudson, Dobson & Newborn 1998; Mougeot *et al*. 2003a; Redpath *et al*. 2006). These experiments have contributed substantially to our understanding of the roles of parasites and behaviour in regulating populations, as well as the role of food in population limitation, whilst at the same time providing insights into fields as diverse as endocrinology, physiology, epidemiology, predation, behavioural ecology, conservation biology and genetics.

A characteristic feature of the red grouse work has been the focus on the relative importance of extrinsic and intrinsic processes in driving changes in abundance. These arguments are of fundamental interest to population ecology because both parasites and territorial competition are two well-known mechanisms in a wide variety of field systems. However, the focus of research in population ecology has primarily been focused on trophic interactions rather than exploring destabilizing effects of intrinsic mechanisms, despite some notable exceptions (Krebs *et al*. 1995; Sinclair & Arcese 1995; Lambin, Petty & Mackinnon 2000; Sinervo, Svensson & Comendant 2000). After 60 years of grouse research, it is clear that parasites have regulatory and destabilizing effects that can explain the cyclic dynamics witnessed in long-term bag records. Indeed, the grouse system has been held up as an example of the importance of parasites in natural systems and their role in causing population cycles (May 1999; Albon *et al*. 2002; Berryman 2002, 2003; Stien *et al*. 2002; Turchin 2003, 2003; Newey & Thirgood 2004). However, one lesson from the grouse research has been that caution should be applied before dismissing alternative mechanisms.

The idea that changing patterns of aggressiveness are underpinned by changing patterns of kinship (Piertney *et al*. 2008) may not just be restricted to grouse. With our rapidly improving ability to look at the fine-scale genetic structuring of animal populations, it is increasingly apparent that kin structuring is common in nature, and not only in social animals, but also many territorial species (see Hatchwell 2010 for a review). Dynamic kin structuring has been shown not only in red grouse (Piertney *et al*. 2008) but also in cyclic root vole *Microtus oeconomus* populations (Pilot *et al*. 2010). It is hoped that these studies will stimulate more research on the dynamic consequences of kinship in other systems.

Other, nonmutually exclusive, mechanistic explanations remain to be tested in red grouse. It would be particularly interesting to study how maternal allocation of testosterone to eggs may mediate the intrinsic or extrinsic mechanisms described before. If testosterone levels lead to an increase in aggressiveness and thereby modify the social environment, it might also influence differential maternal allocation of testosterone to eggs, as shown in other species (Mazuc *et al*. 2003). Increased maternal investment in egg yolk testosterone may compromise chick survival, but boost aggressive behaviour in offspring to facilitate territory establishment in autumn or influence their parasite susceptibility. This remains as an open question but might be particularly relevant given that maternal effects are known to affect population dynamics in general (Benton *et al*. 2005) and population cycles in particular (Ginzburg 1998; Inchausti & Ginzburg 1998).

A variety of studies have also considered the role of stress and its direct and indirect effects on the demographic parameters leading to population cycles (Charbonnel *et al*. 2008; Sheriff, Krebs & Boonstra 2010; Krebs 2011). There is some evidence that the stress response may play a role in mediating the interaction between parasites and aggressiveness (as indexed by comb size) in the red grouse system (Bortolotti *et al*. 2009). A high population density is associated with increased aggressiveness that leads, either directly or indirectly, to immunosuppression (Webster *et al*. 2011a). Individuals would then be more susceptible to the effects of *T. tenuis*, which are already at their highest burdens due to enhanced transmission at high host density. In other words, prior to a population decline, grouse are attempting to combat their greatest immune insult when they are also immune compromised (Webster *et al*. 2011a,b). An inevitable consequence would be a reduction in fitness contributing to a population crash (Lochmiller 1996). The negative effect of testosterone on parasites, and ultimately on fitness, may be context dependent and, more specifically, stress dependent (Bortolotti *et al*. 2009). Future studies should consider how variation in stress responses affects testosterone–parasite interactions in this system.

Long-term red grouse research has revealed some of the challenges we face in teasing apart the mechanisms underpinning complex dynamics. In this case, the protagonists of the two hypotheses have been competing for dominance over ideas and downplaying the importance of the other group's ideas and data. For example, Hudson, Dobson & Newborn (1998) stated that '*..intrinsic mechanisms do not need to be evoked as a cause of cyclic fluctuations in grouse abundance*', whereas Moss & Watson (2001) claimed that 'The evidence is that more typical cycles..… are not due to parasites'. Whilst such competition can stimulate rigour and new work, it can also hinder progress by preventing collaboration. Associated with this has been the focus on single, rather than multiple hypotheses. This is in line with the broader pattern in population ecology to focus on single mechanisms, despite the fact that multiple factors are known to be operating in the field. This has led some authors to suggest that we should be considering multiple factors and their interactions (Holmes 1995; Lochmiller 1996; Dwyer, Dushoff & Yee 2004).

This focus on single hypotheses is partly due to the logistical challenges of working on potentially interacting mechanisms and partly due to mindset. Moss & Watson (2001) outline their philosophical approach to refute

single hypotheses through experimentation. Using this approach, they have been successful at refuting hypotheses related to food (Watson, Moss & Parr 1984) and genetic determinants of aggressiveness (Moss & Watson 1980). Whilst such a Popperian approach is clearly powerful, the latter example reveals some of its limitations. In attempting to refute Chitty's hypothesis (see above), Moss *et al*. carried out an experiment over 9 years that in the end only refuted one version of the hypothesis, but 'left open other interpretations of Chitty's general hypothesis' (Moss & Watson 2001). There is a risk that such an approach may lead to the discarding of a valuable hypothesis. An additional problem is one of time. It has been 50 years since Jenkins, Watson & Miller (1963) first suggested that aggressiveness may be driving cycles. Since then there has been no experimental evidence that has revealed the underlying mechanism. An alternative approach is to design experiments that attempt to compare between competing hypotheses to see where the balance of evidence lies (Krebs 2002, 2010). Thus, any future test of any hypothesis should also consider alternative hypotheses in their design and explicitly state alternative predictions.

Experiments are ultimately what ecologists should be striving towards (Krebs 2010). There have been outstanding population-level experiments done in other systems (Graham & Lambin 2002; Krebs 2010) despite the fact that such experiments are challenging and fraught with difficulties (Lambin & Graham 2003). The real challenge now lies in designing (and funding) appropriate population-level experiments that allow us to test between multiple hypotheses (Krebs 2010). In the case of red grouse, this means an individually based replicated, factorial, population-level experiment manipulating parasites and aggressiveness. A challenge, certainly, but not impossible.

Acknowledgements

We thank all the landowners, gamekeepers, fieldworkers, laboratory collaborators, scientists and funding bodies that have worked hand by hand since the very beginning of red grouse research. Natural Environmental Research Council has been the main funder of this research through different grants in the last few years (NE/D014352/1; NE/D000602/1; NER/A/S/1999/00074). We thank Prof. C. Krebs, Prof. K. Wilson and an anonymous referee for helpful comments on the manuscript.

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. *Proceedings of the Royal Society of London Series B-Biological Sciences*, **269**, 1625–1632.

Anderson, R.M. & May, R.M. (1978) Regulation and stability of host-parasite population interactions. 1. Regulatory processes. *Journal of Animal Ecology*, **47**, 219–247.

Andrewartha, H.G. & Birch, L.C. (1954) *The Distribution and Abundance of Animals*. The University of Chicago Press, Chicago.

Benton, T.G., Plaistow, S.J., Beckerman, A.P., Lapsley, C.T. & Littlejohns, S. (2005) Changes in maternal investment in eggs can affect population dynamics. *Proceedings of the Royal Society B-Biological Sciences*, **272**, 1351–1356.

Berryman, A.A. (2002) Population Cycles: Causes and Analysis. *Population Cycles: The Case for Trophic Interactions* (ed. A.A. Berryman), pp. 3–28. Oxford University Press, Oxford.

Berryman, A.A. (2003) On principles, laws and theory in population ecology. *Oikos*, **103**, 695–701.

Bierman, S.M., Fairbairn, J.P., Petty, S.J., Elston, D.A., Tidhar, D. & Lambin, X. (2006) Changes over time in the spatiotemporal dynamics of cyclic populations of field voles (Microtus agrestis L.). *The American Naturalist*, **167**, 583–590.

Bjornstad, O.N., Ims, R.A. & Lambin, X. (1999) Spatial population dynamics: analyzing patterns and processes of population synchrony. *Trends in Ecology & Evolution*, **14**, 427–432.

Bjornstad, O.N., Peltonen, M., Liebhold, A.M. & Baltensweiler, W. (2002) Waves of larch budmoth outbreaks in the European Alps. *Science*, **298**, 1020–1023.

Bortolotti, G.R., Mougeot, F., Martinez-Padilla, J., Webster, L.M.I. & Piertney, S.B. (2009) Physiological Stress Mediates the Honesty of Social Signals. *PLoS ONE*, **4**, e4983.

Bunnefeld, N., Baines, D., Newborn, D. & Milner-Gulland, E. (2009) Factors affecting unintentional harvesting selectivity in a monomorphic species. *Journal of Animal Ecology*, **78**, 485–492.

Cattadori, I.M., Haydon, D.T. & Hudson, P.J. (2005) Parasites and climate synchronize red grouse populations. *Nature*, **433**, 737–741.

Cattadori, I.M., Merler, S. & Hudson, P.J. (2000) Searching for mechanisms of synchrony in spatially structured gamebird populations. *Journal of Animal Ecology*, **69**, 620–638.

Chapman, D.S., Cornell, S.J. & Kunin, W.E. (2009a) Interactions between harvesting, noise and territoriality in a model of red grouse population cycles. *Journal of Animal Ecology*, **78**, 476–484.

Chapman, D.S., Termansen, M., Quinn, C.H., Jin, N.L., Bonn, A., Cornell, S.J., Fraser, E.D.G., Hubacek, K., Kunin, W.E. & Reed, M.S. (2009b) Modelling the coupled dynamics of moorland management and upland vegetation. *Journal of Applied Ecology*, **46**, 278–288.

Charbonnel, N., Chaval, Y., Berthier, K., Deter, J., Morand, S., Palme, R. & Cosson, J.F. (2008) Stress and demographic decline: A potential effect mediated by impairment of reproduction and immune function in cyclic vole populations. *Physiological and Biochemical Zoology*, **81**, 63–73.

Charnov, E.L. & Finnerty, J.P. (1980) Vole population cycles: a case for kin selection. *Oecologia*, **45**, 1–2.

Chitty, D. (1960) Population processes in the vole and their relevance to general theory. *Canadian Journal of Zoology*, **38**, 99–113.

Chitty, D. (1967) The natural selection of self-regulatory behaviour in animal populations. *Proceedings of the Ecological Society of Australia*, **2**, 51–78.

Clutton-Brock, T. & Sheldon, B.C. (2010) Individuals and populations: the role of long-term, individual-based studies of animals in ecology and evolutionary biology. *Trends in Ecology & Evolution*, **25**, 562–573.

Cobbold, T.S. (1873) , *The grouse disease*. The Field, London, UK.

Delahay, R.J., Speakman, J.R. & Moss, R. (1995) The energetic consequences of parasitism - effects of a developing infection of *Trichostrongylus tenuis* (Nematoda) on Red grouse (*Lagopus lagopus scoticus*) energy balance, body weight and condition. *Parasitology*, **110**, 473–482.

Dobson, A.P. & Hudson, P.J. (1992) Regulation and stability of free-living host-parasite system: *Trichostrongylus tenuis* in red grouse. II. Population models. *Journal of Animal Ecology*, **61**, 487–498.

Dwyer, G., Dushoff, J. & Yee, S.H. (2004) The combined effects of pathogens and predators on insect outbreaks. *Nature*, **430**, 341–345.

Elton, C.S. (1942) *Voles, Mice and Lemmings: Problems in Population Dynamics*. The Clarendon Press, Oxford, UK.

Errington, P.L. (1945) Some contributions of a fifteen-year local study of the northern bobwhite to a knowledge of population phenomena. *Ecological Monographs*, **15**, 2–34.

Errington, P.L. (1956) Factors limiting higher vertebrate populations. *Science*, **124**, 304–307.

Folstad, I. & Karter, A.J. (1992) Parasites, bright males, and the immunocompetence handicap. *American Naturalist*, **139**, 603–622.

Fox, A. & Hudson, P.J. (2001) Parasites reduce territorial behaviour in Red grouse (*Lagopus lagopus scoticus*). *Ecology Letters*, **4**, 139–143.

Ginzburg, L.R. (1998) Inertial growth: population dynamics based on maternal effects. *Maternal Effects as Adaptations* (eds T.A. Mousseau & C.W. Fox), pp. 42–53. Oxford University Press, New York.

Graham, I.M. & Lambin, X. (2002) The impact of weasel predation on cyclic field-vole survival: the specialist predator hypothesis contradicted. *Journal of Animal Ecology*, **71**, 946–956.
Grenfell, B. & Finkenstadt, B.F. (1998) Seasonality, stochasticity and population cycles. *Researches on Population Ecology*, **40**, 141–143.
Hanski, I., Henttonen, H., Korpimaki, E., Oksanen, L. & Turchin, P. (2001) Small-rodent dynamics and predation. *Ecology*, **82**, 1505–1520.
Hatchwell, B.J. (2010) Cryptic Kin selection: Kin structure in vertebrate populations and opportunities for Kin-Directed cooperation. *Ethology*, **116**, 203–216.
Haydon, D.T., Shaw, D.J., Cattadori, I.M., Hudson, P.J. & Thirgood, S.J. (2002) Analysing noisy time-series: describing regional variation in the cyclic dynamics of red grouse. *Proceedings of the Royal Society of London Series B-Biological Sciences*, **269**, 1609–1617.
Hendry, R., Bacon, P.J., Moss, R., Palmer, S.C.F. & McGlade, J. (1997) A two-dimensional individual-based model of territorial behaviour: possible population consequences of kinship in red grouse. *Ecological Modeling*, **105**, 23–39.
Hillgarth, N. & Wingfield, J.C. (1997) Parasite-mediated sexual selection: endocrine aspects. *Host-Parasite Evolution General Principles & Avian Models* (eds D.H. Clayton & J. Moore), pp. 78–104. Oxford University Press, Oxford.
Holmes, J. (1995) Population regulation: a dynamic complex of interactions. *Wildlife Research*, **22**, 11–19.
Hudson, P.J. (1986a) The effect of a parasitic nematode on the breeding production of red grouse. *Journal of Animal Ecology*, **55**, 85–92.
Hudson, P.J. (1986b) *The red Grouse: The Biology and Management of a Wild Gamebird*. The Game Conservancy Trust, Fordingbridge.
Hudson, P.J., Dobson, A.P. & Newborn, D. (1985) Cyclic and non-cyclic populations of red grouse: a role for parasitism? *Ecology and Genetics of Host-Parasite Interactions* (eds D. Rollinson & R.M. Anderson), pp. 77–86. Academic Press, London, UK.
Hudson, P.J., Dobson, A.P. & Newborn, D. (1992) Regulation and stability of a free-living host-parasite system – *Trichostrongylus tenuis* in red grouse. I. Monitoring and parasite reduction experiments. *Journal of Animal Ecology*, **61**, 477–486.
Hudson, P.J., Dobson, A.P. & Newborn, D. (1998) Prevention of population cycles by parasite removal. *Science*, **282**, 2256–2258.
Hudson, P.J. & Newborn, D. (1990) Brood defense in a precocial species – variations in the distraction displays of red grouse, *Lagopus-Lagopus-Scoticus*. *Animal Behaviour*, **40**, 254–261.
Hudson, P.J. & Newborn, D. (1995) *A Manual of Red Grouse and Moorland Management*. The Game Conservancy Trust, Fordingbridge, UK.
Hudson, P.J., Renton, J. & Dalby, G. (1988) Red grouse homing for 35 kilometres. *Scottish Birds*, **15**, 90–91.
Hudson, P.J., Norman, R., Laurenson, M.K., Newborn, D., Gaunt, M., Jones, L., Reid, H., Gould, E., Bowers, R. & Dobson, A. (1995) Persistence and transmission of tick-borne viruses: Ixodes ricinus and louping-ill virus in red grouse populations. *Parasitology*, **111**, S49–S58.
Hudson, P.J., Dobson, A.P., Cattadori, I.M., Newborn, D., Haydon, D.T., Shaw, D.J., Benton, T.G. & Grenfell, B.T. (2002) Trophic interactions and population growth rates: describing patterns and identifying mechanisms. *Philosophical Transactions of the Royal Society of London Series B-Biological Sciences*, **357**, 1259–1271.
Inchausti, P. & Ginzburg, L.R. (1998) Small mammals cycles in northern Europe: patterns and evidence for a maternal effect hypothesis. *Journal of Animal Ecology*, **67**, 180–194.
Jenkins, D. (1970) Practical results of research for management of red grous. *Biological Conservation*, **2**, 266–272.
Jenkins, D., Watson, A. & Miller, G.R. (1963) Population studies on red grouse, *Lagopus lagopus scoticus* (Lath.) in North-east Scotland. *Journal of Animal Ecology*, **32**, 317–376.
Klemola, T., Hanhimaki, S., Ruhomaki, S., Senn, J., Tanhuanpaa, M., Kaitaniemi, P., Ranta, H. & Haukioja, E. (2003) Performance of the cyclic autumnal moth, *Epirrita autumnata*, in relation to birch mast seeding. *Oecologia*, **135**, 354–361.
Korpimäki, E., Norrdahl, K., Klemola, T., Pettersen, T. & Stenseth, N.C. (2002) Dynamic effects of predators on cyclic voles: field experimentation and model extrapolation. *Proceedings of the Royal Society B-Biological Sciences*, **269**, 991–997.
Krebs, C.J. (2002) Two complementary paradigms for analysing population dynamics. *Philosophical Transactions of the Royal Society of London Series B-Biological Sciences*, **357**, 1211–1219.
Krebs, C.J. (2010) Case studies and ecological understanding. *The Ecology of Place: Contributions of Place-Based Research to Ecological and Evolutionary Understanding* (eds I. Billick & M.V. Proce). University of Chicago Press, Chicago, USA.
Krebs, C.J. (2011) Of lemmings and snowshoe hares: the ecology of northern Canada. *Proceedings of the Royal Society B-Biological Sciences*, **278**, 481–489.
Krebs, C.J., Boutin, S., Boonstra, R., Sinclair, A.R.E., Smith, J.N.M., Dale, M.R.T., Martin, K. & Turkington, R. (1995) Impact of food and predation on the snowshoe hare cycle. *Science*, **265**, 1112–1115.
Lambin, X. & Graham, I.M. (2003) Testing the specialist predator hypothesis for vole cycles. *Trends in Ecology and Evolution*, **18**, 493.
Lambin, X., Petty, S.J. & Mackinnon, J.L. (2000) Cyclic dynamics in field vole populations and generalist predation. *Journal of Animal Ecology*, **69**, 106–118.
Lambin, X., Elston, D.A., Petty, S.J. & MacKinnon, J.L. (1998) Spatial asynchrony and periodic travelling waves in cyclic populations of field voles. *Proceedings of the Royal Society B-Biological Sciences*, **265**, 1491–1496.
Lambin, X., Krebs, C.J., Moss, R., Stenseth, N.C. & Yoccoz, N.G. (1999) Population cycles and parasitism. *Science*, **286**, 2425.
Laurenson, M.K., Norman, R.A., Gilbert, L., Reid, H.W. & Hudson, P.J. (2003) Identifying disease reservoirs in complex systems: mountain hares as reservoirs of ticks and louping-ill virus, pathogens of red grouse. *Journal of Animal Ecology*, **72**, 177–185.
Laurenson, M.K., Norman, R.A., Gilbert, L., Reid, H.W. & Hudson, P.J. (2004) Mountain hares, louping-ill, red grouse and harvesting: complex interactions but few data. *Journal of Animal Ecology*, **73**, 811–813.
Liebhold, A. & Kamata, N. (2000) Introduction – Are population cycles and spatial synchrony a universal characteristic of forest insect populations? *Population Ecology*, **42**, 205–209.
Lochmiller, R.L. (1996) Immunocompetence and animal population regulation. *Oikos*, **76**, 594–602.
Lovat, L. (1911) *The Grouse in Health and in Disease*. Smith & Elder, London.
MacColl, A.D.C., Piertney, S.B., Moss, R. & Lambin, X. (2008) Spatial arrangement of kin affects recruitment success in young male red grouse. *Oikos*, **90**, 261–270.
Mackenzie, J.M.D. (1952) Fluctuations in the number of British Tetraonids. *Journal of Animal Ecology*, **21**, 128–153.
Martinez-Padilla, J., Mougeot, F., Perez-Rodriguez, L. & Bortolotti, G.R. (2007) Nematode parasites reduce carotenoid-based signalling in male red grouse. *Biology Letters*, **3**, 161–164.
Martinez-Padilla, J., Vergara, P., Mougeot, F. & Redpath, S.M. (2012) Parasitized mates increase infection risk for partners. *The American Naturalist*, **179**, 811–820.
Matthiopoulos, J., Halley, J.M. & Moss, R. (2005) Socially induced red grouse population cycles need abrupt transitions between tolerance and aggression. *Ecology*, **86**, 1883–1893.
Matthiopoulos, J., Moss, R. & Lambin, X. (1998) Models of red grouse cycles. A family affair? *Oikos*, **82**, 574–590.
Matthiopoulos, J., Moss, R. & Lambin, X. (2000) The kin-facilitation hypothesis for red grouse population cycles: territory sharing between relatives. *Ecological Modeling*, **127**, 53–63.
Matthiopoulos, J., Moss, R. & Lambin, X. (2002) The kin facilitation hypothesis for red grouse population cycles: territorial dynamics of the family cluster. *Ecological Modeling*, **147**, 291–307.
Matthiopoulos, J., Moss, R., Mougeot, F., Redpath, S.M. & Lambin, X. (2003) Territorial behaviour and population dynamics in red grouse *Lagopus lagopus scoticus*. II. Population models. *Journal of Animal Ecology*, **72**, 1083–1096.
May, R.M. (1999) Population biology: crash test for real. *Nature*, **398**, 371–372.
May, R.M. & Anderson, R.M. (1978) Regulation and stability of host-parasite population interactions. 2. Destabilizing processes. *Journal of Animal Ecology*, **47**, 249–267.
Mazuc, J., Bonneaud, C., Chastel, O. & Sorci, G. (2003) Social environment affects female and egg testosterone levels in the house sparrow (Passer domesticus). *Ecology Letters*, **6**, 1084–1090.
Moseley, M.H., Marriott, L., Nettleton, P., Dukes, J., Irvine, R.J. & Mougeot, F. (2007) Use of real-time RT-PCR to determine the prevalence of louping ill virus in live red grouse chicks. *Veterinary Record*, **161**, 660–661.
Moss, R., Parr, R. & Lambin, X. (1994) Effects of testosterone on breeding density, breeding success and survival of red grouse. *Proceedings of the Royal Society B-Biological Sciences*, **258**, 175–180.

Moss, R. & Watson, A. (1980) Inherent changes in the aggressive behaviour of a fluctuating red grouse *Lagopus lagopus scoticus* population. *Ardea*, **68**, 113–119.

Moss, R. & Watson, A. (1991) Population cycles and kin selection in red grouse *Lagopus lagopus scoticus*. *Ibis*, **133**, 113–120.

Moss, R. & Watson, A. (2001) *Population Cycles in Birds of the Grouse Family (Tetraonidae)*. pp. 53–111. Academic Press, London, UK.

Moss, R., Watson, A. & Parr, R. (1996) Experimental prevention of a population cycle in red grouse. *Ecology*, **77**, 1512–1530.

Moss, R., Watson, A. & Rothery, P. (1984) Inherent changes in body size, viability and behaviour of a fluctuating red grouse (*Lagopus lagopus scoticus*) population. *Journal of Animal Ecology*, **53**, 171–189.

Moss, R., Kolb, H.H., Marquiss, M., Watson, A., Treca, B., Watt, D. & Glennie, W. (1979) Aggressiveness and dominance in captive cock red grouse. *Aggressive Behaviour*, **5**, 58–84.

Moss, R., Trenholm, I.B., Watson, A. & Parr, R. (1990) Parasitism, predation and survival of hen red grouse *Lagopus lagopus scoticus* in spring. *Journal of Animal Ecology*, **59**, 631–642.

Moss, R., Watson, A., Trenholm, I.B. & Parr, R. (1993) Cecal threadworms *Trichostrongylus tenuis* in Red grouse *Lagopus lagopus scoticus* – Effects of weather and host density upon estimated worm burdens. *Parasitology*, **107**, 199–209.

Mougeot, F., Evans, S.A. & Redpath, S.M. (2005) Interactions between population processes in a cyclic species: parasites reduce autumn territorial behaviour of male red grouse. *Oecologia*, **144**, 289–298.

Mougeot, F., Redpath, S.M. & Piertney, S. (2006) Elevated spring testosterone increases parasite intensity in male red grouse. *Behavioral Ecology*, **17**, 117–125.

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

Mougeot, F., Redpath, S.M., Moss, R., Matthiopoulos, J. & Hudson, P.J. (2003b) Territorial behaviour and population dynamics in red grouse *Lagopus lagopus scoticus*. I. Population experiments. *Journal of Animal Ecology*, **72**, 1073–1082.

Mougeot, F., Piertney, S.B., Leckie, F., Evans, S., Moss, R., Redpath, S.M. & Hudson, P.J. (2005a) Experimentally increased aggressiveness reduces population kin structure and subsequent recruitment in red grouse *Lagopus lagopus scoticus*. *Journal of Animal Ecology*, **74**, 488–497.

Mougeot, F., Redpath, S.M., Piertney, S. & Hudson, P.J. (2005b) Separating behavioral and physiological mechanisms in testosterone-mediated trade-offs. *The American Naturalist*, **166**, 158–168.

Mountford, M.D., Watson, A., Moss, R., Parr, R. & Rothery, P. (1990) Land inheritance and population cycles of red grouse. *Red Grouse Population Processes* (eds A.N. Lance & J.H. Lawton), pp. 78–83. Royal Society for the Protection of Birds, Sandy, Bedfordshire.

New, L.F., Matthiopoulos, J., Redpath, S.M. & Buckland, S.T. (2009) Fitting models of multiple hypotheses to partial population data: investigating the causes of cycles in red grouse. *The American Naturalist*, **174**, 399–412.

Newborn, D. & Foster, R. (2002) Control of parasite burdens in wild red grouse *Lagopus lagopus scoticus* through the indirect application of anthelmintics. *Journal of Applied Ecology*, **39**, 909–914.

Newey, S. & Thirgood, S. (2004) Parasite-mediated reduction in fecundity of mountain hares. *Proceedings of the Royal Society of London Series B-Biological Sciences*, **271**, S413–S415.

Nicholson, A.J. (1933) The balance of animal populations. *Journal of Animal Ecology*, **2**, 132–178.

Piertney, S.B., Lambin, X., MacColl, A.D.C., Lock, K., Bacon, P.J., Dallas, J.F., Leckie, F., Mougeot, F., Racey, P.A., Redpath, S.M. & Moss, R. (2008) Temporal changes in kin structure through a population cycle in a territorial bird, the red grouse *Lagopus lagopus scoticus*. *Molecular Ecology*, **17**, 2544–2551.

Pilot, M., Dabrowski, M.J., Jancewicz, E., Schtickzelle, N. & Gliwicz, J. (2010) Temporally stable genetic variability and dynamic kinship structure in a fluctuating population of the root vole Microtus oeconomus. *Molecular Ecology*, **19**, 2800–2812.

Potts, G.R., Tapper, S.C. & Hudson, P.J. (1984) Population fluctuations in red grouse – analysis of bag records and a simulation-model. *Journal of Animal Ecology*, **53**, 21–36.

Ranta, E., Lindstrom, J. & Linden, H. (1995) Synchrony in tetraonid population-dynamics. *Journal of Animal Ecology*, **64**, 767–776.

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.

Reid, H.W. (1975) Experimental infection of the red grouse with louping-ill virus (*Flavivirus group*). I. The viraemia and antibody response. *Journal of Comparative Pathology*, **85**, 231–235.

Seivwright, L.J., Redpath, S.M., Mougeot, F., Leckie, F. & Hudson, P.J. (2005) Interactions between intrinsic and extrinsic mechanisms in a cyclic species: testosterone increases parasite infection in red grouse. *Proceedings of the Royal Society B-Biological Sciences*, **272**, 2299–2304.

Shaw, J.L. (1988) Arrested development of *Trichostrongylus tenuis* as 3rd stage larvae in Red grouse. *Research in Veterinary Science*, **45**, 256–258.

Shaw, J.L. & Moss, R. (1989a) Factors affecting the establishment of the cecal threadworm *Trichostrongylus tenuis* in Red grouse (*Lagopus lagopus scoticus*). *Parasitology*, **99**, 259–264.

Shaw, J.L. & Moss, R. (1989b) The role of parasite fecundity and longevity in the success of *Trichostrongylus tenuis* in low-density Red grouse populations. *Parasitology*, **99**, 253–258.

Shaw, J.L. & Moss, R. (1990) Effects of the cecal nematode *Trichostrongylus tenuis* on egg-Laying by captive Red grouse. *Research in Veterinary Science*, **48**, 59–63.

Shaw, J.L., Moss, R. & Pike, A.W. (1989) Development and survival of the free-living stages of *Trichostrongylus tenuis*, a cecal parasite of Red grouse *Lagopus lagopus scoticus*. *Parasitology*, **99**, 105–113.

Shaw, D.J., Haydon, D.T., Cattadori, I.M., Hudson, P.J. & Thirgood, S.J. (2004) The shape of red grouse cycles. *Journal of Animal Ecology*, **73**, 767–776.

Sheriff, M.J., Krebs, C.J. & Boonstra, R. (2010) Assessing stress in animal populations: Do fecal and plasma glucocorticoids tell the same story? *General and Comparative Endocrinology*, **166**, 614–619.

Sibly, R.M. & Hone, J. (2002) Population growth rate and its determinants: an overview. *Philosophical Transactions of the Royal Society of London Series B-Biological Sciences*, **357**, 1153–1170.

Sinclair, A.R.E. (1989) Population regulation of animals. *Ecological Concepts* (ed. J.M. Cherrett), pp. 197–241. Blackwell Scientific, Oxford, UK.

Sinclair, A.R.E. & Arcese, P. (1995) Population consequences of predation-sensitive foraging – the Serengeti Wildebeest. *Ecology*, **76**, 882–891.

Sinervo, B., Svensson, E. & Comendant, T. (2000) Density cycles and an offspring quantity and quality game driven by natural selection. *Nature*, **406**, 985–988.

Stenseth, N.C. (1999) Population cycles in voles and lemmings: density dependence and phase dependence in a stochastic world. *Oikos*, **87**, 427–461.

Stenseth, N.C., Bjornstad, O.N. & Falck, W. (1996) Is spacing behaviour coupled with predation causing the microtine density cycle? A synthesis of current process-oriented and pattern-oriented studies. *Proceedings of the Royal Society of London Series B-Biological Sciences*, **263**, 1423–1435.

Stien, A., Irvine, R.J., Ropstad, E., Halvorsen, O., Langvatn, R. & Albon, S.D. (2002) The impact of gastrointestinal nematodes on wild reindeer: experimental and cross-sectional studies. *Journal of Animal Ecology*, **71**, 937–945.

Thirgood, S.J., Redpath, S.M., Haydon, D.T., Rothery, P., Newton, I. & Hudson, P.J. (2000) Habitat loss and raptor predation: disentangling long- and short-term causes of red grouse declines. *Proceedings of the Royal Society of London. Series B-Biological Sciences*, **267**, 651–656.

Tompkins, D.M. & Begon, M. (1999) Parasites can regulate wildlife populations. *Parasitology Today*, **15**, 311–313.

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

Vergara, P. & Martinez-Padilla, J. (2012) Social context decouples the relationship between a sexual ornament and testosterone levels in a male wild bird. *Hormones and Behavior*, **62**, 407–412.

Vergara, P., Redpath, S.M., Martínez-Padilla, J. & Mougeot, F. (2012) Environmental conditions influence red grouse ornamentation at a population level. *Biological Journal of the Linnean Society*, **107**, 788–798.

Verhulst, S., Dieleman, S.J. & Parmentier, H.K. (1999) A tradeoff between immunocompetence and sexual ornamentation. *Proceedings of the National Academy of Sciences of the United States of America*, **96**, 4478–4481.

Watson, A. (1985) Social class, socially-induced loss, recruitment and breeding of red grouse. *Oecologia*, **67**, 493–498.

Watson, A. & Jenkins, D. (1968) Experiments on population control by territorial behaviour in red grouse. *Journal of Animal Ecology*, **37**, 595–614.

Watson, A. & Moss, R. (2008) *Grouse*. Collins, London.

Watson, A., Moss, R. & Parr, R. (1984) Effects of food enrichment on numbers and spacing behaviour of red grouse. *Journal of Animal Ecology*, **53**, 663–678.

Watson, A., Moss, R. & Rae, S. (1998) Population dynamics of Scottish Rock Ptarmigan cycles. *Ecology*, **79**, 1174–1192.

Watson, A., Moss, R. & Rothery, P. (2000) Weather and synchrony in 10-year population cycles of Rock ptarmigan and Red grouse in Scotland. *Ecology*, **81**, 2126–2136.

Watson, A., Moss, R., Parr, R., Mountford, M.D. & Rothery, P. (1994) Kin landownership, differential aggression between kin and nonkin, and population fluctuations in Red grouse. *Journal of Animal Ecology*, **63**, 39–50.

Webster, L.M.I., Mello, L.V., Mougeot, F., Martinez-Padilla, J., Paterson, S. & Piertney, S.B. (2011a) Identification of genes responding to nematode infection in red grouse. *Molecular Ecology Resources*, **11**, 305–313.

Webster, L.M.I., Paterson, S., Mougeot, F., Martinez-Padilla, J. & Piertney, S.B. (2011b) Transcriptomic response of red grouse to gastrointestinal nematode parasites and testosterone: implications for population dynamics. *Molecular Ecology*, **20**, 920–931.

Williams, J.P. (1985) Statistical analysis of fluctuations in red grouse bag data. *Oecologia*, **65**, 269–272.

Wilson, G.R. & Wilson, L.P. (1978) Haematology weight and condition of red grouse (*Lagopus lagopus scoticus*) infected with caecal threadworms (*Trichostrongylus tenuis*). *Research in Veterinary Science*, **25**, 331–336.

Received 17 January 2013; accepted 30 April 2013
Handling Editor: Ken Wilson