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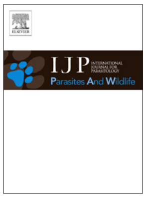

Invited Review

Exploiting parallels between livestock and wildlife: Predicting the impact of climate change on gastrointestinal nematodes in ruminants

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ABSTRACT

Global change, including climate, policy, land use and other associated environmental changes, is likely to have a major impact on parasitic disease in wildlife, altering the spatio-temporal patterns of transmission, with wide-ranging implications for wildlife, domestic animals, humans and ecosystem health. Predicting the potential impact of climate change on parasites infecting wildlife will become increasingly important in the management of species of conservation concern and control of disease at the wildlife–livestock and wildlife–human interface, but is confounded by incomplete knowledge of host–parasite interactions, logistical difficulties, small sample sizes and limited opportunities to manipulate the system. By exploiting parallels between livestock and wildlife, existing theoretical frameworks and research on livestock and their gastrointestinal nematodes can be adapted to wildlife systems. Similarities in the gastrointestinal nematodes and the life-histories of wild and domestic ruminants, coupled with a detailed knowledge of the ecology and life-cycle of the parasites, render the ruminant-GIN host–parasite system particularly amenable to a cross-disciplinary approach.

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1. Introduction

Parasites are ubiquitous in wildlife and livestock and are an important component of ecological communities (Dobson and Hudson, 1986). Far from being "benign symbionts living in equilibrium with their hosts", parasites have a profound effect on host survival, fecundity and behaviour (Hudson and Dobson, 1995). There is mounting theoretical and empirical evidence that parasites play an important role in influencing host populations through impacts on survival and reproduction (Holmes, 1995; Hudson et al., 1998; Tompkins and Begon, 1999; Watson, 2013) and trophic equilibria (Grenfell, 1992). Parasitic infection and disease in wildlife and at the livestock–wildlife interface, therefore, has the potential to impede conservation efforts by restricting the ranges of host species (Dobson and Hudson, 1986) and threatening the persistence of species of conservation concern (Laurenson et al., 1998; Morgan et al., 2005; Page, 2013).

The Intergovernmental Panel on Climate Change (IPCC) concluded that "Warming of the climate system is unequivocal, and since the 1950s, many of the observed changes are unprecedented over decades to millennia. The atmosphere and ocean have warmed, the amounts of snow and ice have diminished, sea level has risen, and the concentrations of greenhouse gases have increased" (IPCC, 2013). Global average surface temperatures increased by 0.85 °C between 1880 and 2012. However, the pattern of global warming is not spatially homogeneous. Analysis of observed surface temperatures estimates historic increases of up to 2.5 °C in parts of Canada, Brazil and Russia between 1901 and 2012 (IPCC, 2013). Changes in observed precipitation are more complex. Analysis of observed precipitation between 1951 and 2010 estimates historic decreases of up to 100 mm/year/decade in regions such as West Africa and contrasting increases of up to 100 mm/year/decade in regions such as Northern Europe (IPCC, 2013). Further temperature increases and changes in precipitation are predicted. Average surface temperatures could rise by more than 9 °C in the Arctic by 2081–2100 compared with the baseline period of 1986–2005 (IPCC, 2013).

Since many parasites have free-living stages and ectothermic intermediate hosts, their development and survival, and therefore, transmission dynamics, are inextricably linked with the environment (e.g. O'Connor et al., 2006). As a result, environmental perturbations caused by climate and associated anthropogenic and environmental change could have a profound impact on parasite phenology, host–parasite dynamics and host population dynamics (Kutz et al., 2005; van Dijk et al., 2010; Hoar, 2012; Altizer et al., 2013; Molnár et al., 2013). Declining numbers in a population of moose (*Alces alces andersoni*) in northwest Minnesota coincided with an increase in temperatures and lengthening of the annual growing season between 1960 and 2001. Pathogens, climate change and nutritionally deficient habitat were implicated as causative factors in the decline in numbers of moose (Murray et al., 2006). The authors concluded that this moose population is currently not viable and they emphasised the need to understand parasite dynamics in altered environments to predict and potentially mitigate negative changes in host–pathogen population dynamics.

Less is known about the effect of globalisation, policy and indirect effects of climate change on disease dynamics. It is possible to draw on observations of responses to recent environmental change and variability (e.g. McNeil et al., 2005; Moyes et al., 2011) to predict the impact of future changes. However, predicting the direction and magnitude of global change, and subsequently the impact on disease dynamics, is inherently difficult where drivers of change such as climate, anthropogenic pressures on land use, and policy interact. For example in the European Union agricultural land use, yield and on-farm provision for conservation and the environment are heavily influenced by the payment of subsidies under the Common Agricultural Policy and environmental constraints (Olesen and Bindi, 2002; Renwick et al., 2013). As a result, predictions are often centered on the impacts of climate change. A thorough understanding of the drivers of global change, host and parasite biology, ecology and distribution, and host–parasite dynamics, will be key to generating useful predictions of the likely impact of global change on wildlife and their parasite fauna.

2. Predicting the impact of climate change on parasite and host

The impact of climate change on parasites, hosts and parasitic disease is likely to be complex, particularly in multiple host/vector systems, at the edge of species' ranges, where species exhibit variability in key life-history traits that may act as a target for adaptation to climate change, and where there are non-linear interactions between climate and host/parasite response (van Dijk and Morgan, 2010; Rohr et al., 2011; Altizer et al., 2013). For example, species distribution models suggest that although increasing temperatures will result in the earlier spring emergence and later onset of diapause in blowfly (*Lucilia sericata*) in Great Britain, there may be a trade-off between increased development rates and temperature- and moisture-dependent mortality. This led to a decrease in the predicted probability of blowfly strike in sheep in regions where hot, dry summers are expected, resulting in two distinct periods of risk (Rose and Wall, 2011). A split transmission season under warming conditions is also predicted for *Ostertagia gruehneri,* an abomasal nematode of caribou, due to interactions between development and mortality rates at higher temperatures (Molnár et al., 2013). Predictions for climate change impacts on parasites are further complicated by concomitant changes such as: drug resistance in parasites of both livestock and wildlife (Chintoan-Uta et al., 2014); land use and habitat loss (Lafferty, 2009; Pascual and Bouma, 2009; Festa-Bianchet et al., 2011); host behaviour (Moyes et al., 2011), and; policy.

2.1. Modelling parasite and host dynamics under climate change scenarios

Empirical models such as species distribution models can be useful in identifying potential drivers of change (Pickles et al., 2013), particularly where detailed data and knowledge of the system are unavailable. For example, models can be constructed using distal (indirect) variables, such as precipitation, where proximal (direct) variables, such as soil moisture, are unavailable (Franklin, 2009). However, extrapolating beyond observed conditions to predict the impact of climate change relies on a number of assumptions, not least that correlations between variables remain constant under future conditions (Rose and Wall, 2011). Moreover, the response of host and parasite to change is often non-linear or threshold-dependent (Rohr et al., 2011). For example, there are species-specific optimal temperature and moisture requirements for the development and survival of the free-living stages of common gastrointestinal nematodes of ruminants. Above and below these optima the development success decreases (Rossanigo and Gruner, 1995).

Lafferty (2009) notes that factors other than climate, such as land use, play an important role in determining disease dynamics, and *"seasonality in disease does not necessarily indicate an effect of climate on disease"*. This is an especially pertinent point when considering the impact of climate change on parasites and infection dynamics, a system that exists and interacts on multiple scales and in multiple dimensions. In these systems, apparent correlations between climate, host and parasite life-history do not equate to causation (e.g. the seasonal arrest rate of nematode larvae and the peri-parturient rise in faecal egg counts in ewes; Lafferty, 2009). Compared with empirical models, mechanistic models require a more detailed understanding of the system and the underlying processes driving observed patterns, thus distinguishing between correlation and causation. In doing so they make fewer assumptions regarding the relationships and interactions between variables and are, therefore, better suited for projecting outside of the spatial and temporal range of observed data.

2.2. Modelling wildlife–parasite systems: challenges and opportunities

The development and application of mechanistic models are, however, limited by available data. Lack of suitable data for model parameterisation is a problem in wildlife–parasite systems due to incomplete knowledge of host–parasite dynamics, logistical difficulties such as access to remote areas, small sample sizes, conservation considerations and limited opportunities to manipulate the system experimentally (Kutz et al., 2009). Some valuable insights into parasite biology and host–parasite interactions have been gained by experimental infection of captive animals including reindeer (*Rangifer tarandus*; Hoar et al., 2012a) and thinhorn sheep (*Ovis dalli* ssp.; Kutz et al., 2004; Jenkins et al., 2005*)*. In addition, several notable longitudinal studies exist that have allowed field manipulations of gastrointestinal nematodes (GINs) in free-ranging hosts, including Soay sheep (*Ovis aries*; e.g. Gulland, 1992), Svalbard reindeer (*Rangifer tarandus platyrhynchus*; e.g. Albon et al., 2002; Carlsson et al., 2012a,b) and red grouse (*Lagopus lagopus scoticus*; e.g. Hudson et al., 1998). However, such opportunities are rare due to the difficulty in disentangling the effect of parasitism from numerous confounders in long term-correlational studies (e.g. seasonal availability of forage, inter- and intra-specific competition, anthropogenic disturbance and climatic factors; Murray et al., 2006).

In contrast, studies on parasites in livestock and model species are much more tractable with potential to manipulate and control for a range of confounders. Virtually every component of livestock systems can be tightly regulated, from nutrition, to host density and population structure, to host genetics and history of exposure to parasites and pathogens. In these systems, parasites are primarily an economic burden, affecting productivity and demanding significant resources for their effective control (Nieuwhof and Bishop, 2005). As a result, and by necessity, the biology of livestock parasites, the epidemiology of parasitic disease in livestock, and the impact of global change on disease dynamics, have been the focus of decades of detailed research.

This review focuses on gastrointestinal nematodes (GINs) infecting ruminants, the challenges in predicting the impact of global change on transmission potential and nematode population dynamics in free-ranging ruminants, and how theoretical and empirical research on GINs in livestock can be used to this end. The life-cycle and ecology of gastrointestinal nematodes in livestock ruminants is a well-studied system, with approaching a century of available field and laboratory data (e.g. Veglia, 1916), and 35 years of mathematical model development (reviewed by Smith and Grenfell, 1994). Roberts (1995) suggested that models for the dynamics of parasitic helminths could be divided into those concerning wildlife and those concerning livestock. However, there are many similarities between livestock, wildlife, and their GINs that can be exploited to use the wealth of research on GINs in livestock to provide new insights into wildlife systems (Table 1).

Table 1
Despite their divergent evolutionary histories and management of livestock by the farmer, there are many parallels between livestock such as sheep (*Ovis aries*) and wildlife such as Saiga antelope (*Saiga tatarica)* that can be exploited to predict the impact of climate change on gastrointestinal nematode infections. (Photos: Morgan, E. R. (saiga) & Rose, H. (sheep)).

Host species	Gastrointestinal nematodes (GINs)	Acquired immunity to GINs	Reproduction	Population age-structure	Spatial distribution
Saiga tatarica 	25/26 species shared with livestock	Some evidence	Seasonal	Seasonal variation	Annual migration between winter and summer ranges
Ovis aries 	All species shared with wild ruminants	Strong	Seasonal (some aseasonal)	Seasonal variation	Separate summer and winter grazing areas are common

3. Exploiting parallels between livestock and wildlife

Trichostrongyloid nematodes are an extremely diverse superfamily, with an equally diverse host range (Anderson, 2000). Primarily GINs, trichostrongyloids affect production in livestock (Nieuwhof and Bishop, 2005) and infect free-ranging ruminants worldwide (McKenna, 1997; Ferté et al., 2000; Hoberg et al., 2001; Morgan et al., 2003; Kutz et al., 2012).

Numerous models of GIN infections in livestock ruminants have been developed over the past 35 years, many aimed at forecasting seasonal variation in the risk of GIN infection and evaluating control strategies (reviewed by Smith and Grenfell, 1994; Cornell, 2005; Smith, 2011). These models draw on a wealth of intricate laboratory observations, experimental infections and field studies on GINs infecting livestock, for parameterisation and to incorporate climatic and management (host dynamics) influences on GIN development and survival (Smith et al., 1987; Smith and Galligan, 1988; Leathwick et al., 1992; Kao et al., 2000; Learmount et al., 2006; Dobson et al., 2011). These models provide a foundation for modelling the impact of climate change on GIN infection dynamics. However, the breadth of application of these models is not restricted to livestock-GIN systems. Similarities in GIN species infecting livestock and wildlife, host immune response, the life-history and ranging behaviour of the ruminant hosts, and the potential for adaptation to climate change can be exploited to extend livestock GIN models to wildlife systems.

3.1. Gastrointestinal nematode fauna of wild and domestic ruminants

Smith and Grenfell (1985) were among the first to develop a mechanistic modelling framework for GINs in ruminants, describing the population biology of *Ostertagia ostertagi* in cattle. The model has since been refined and extended to address specific questions of GIN epidemiology and control in cattle and applied to other GIN species by taking advantage of similarities in the life-cycle and life-history of trichostrongylid nematodes infecting cattle and sheep (Smith and Grenfell, 1994). To what extent can these models be applied to GIN species infecting free-ranging ruminants?

Table 2
Gastrointestinal nematodes of Saiga antelope in Kazakhstan, and presence (+) or absence (–) of the species in livestock ruminants. Table adapted from Morgan et al. (2005).

Gastrointestinal nematode species	Cattle	Goat	Sheep
Chabertia ovina	+	+	+
Haemonchus contortus	+	+	+
Marshallagia marshalli	+	+	+
M. mongolica	+	+	+
Nematodirella cameli	–	–	+
N. gazelle	–	–	–
N. longissimespiculata	+	+	+
Nematodirus abnormalis	+	+	+
N. andreevi	–	+	–
N. dogieli	–	+	+
N. gazellae	–	–	+
N. mauritanicus	–	+	+
N. oiratianus	+	+	+
N. spathinger	+	+	+
Oesophagostomum venulosum	+	+	+
Ostertagia orloffi	+	+	+
O. ostertagi	+	+	+
Parabronema skrjabini	+	+	+
Skrjabinema ovis	–	+	+
Stongyloides papillosus	–	–	+
Teladorsagia circumcincta	+	+	+
Trichostrongylus axei	+	+	+
T. colubriformis	+	+	+
T. probolorus	+	+	+
Trichuris ovis	+	+	+
T. skrjabini	+	+	+

3.1.1. Exploiting the conserved life cycle and broad host range of GINs

The life-cycle of trichostrongyloid GINs is well-known and broadly conserved. Eggs are deposited on pasture in faeces, develop to third-stage infective larvae, and migrate onto the herbage where they are ingested by the host. Once ingested the infective larvae exsheath and migrate into the gut mucosa. The infective larvae develop to fourth-stage larvae in the gut mucosa before re-entering the gut lumen and maturing to the adult stage (Anderson, 2000). Family-specific differences exist in the minutiae of this basic life-cycle. First, the majority of trichostrongylids (e.g. *Cooperia* spp., *Haemonchus* spp. and *Ostertagia* spp.) hatch as first-stage larvae, whereas *Marshallagia marshalli* hatch as second-stage larvae (Carlsson et al., 2013) and molineids in the subfamily Nematodirinae (e.g. *Nematodirus battus*) hatch as third-stage larvae (Thomas, 1959). Second, the site of infection is species specific. For example, *Haemonchus contortus* inhabits the abomasum and *Cooperia oncophora* inhabits the small intestine. Finally, developmental arrest (hypobiosis) of larvae in the gut mucosa has been observed in some species, but not all, and the stage at which hypobiosis occurs varies. For example, *Ostertagia* spp. arrest as fourth stage larvae in the host's abomasal mucosa (Anderson, 2000; Hoar et al., 2012a) whereas *Trichostrongylus colubriformis* arrests as third stage larvae in sheep (Eysker, 1978).

Despite these minor differences, the similarities in the life-cycle of GINs, their broad host range and the considerable overlap in species infecting livestock and wildlife (Morgan et al., 2003; Chintoan-Uta et al., 2014) enable us to apply existing nematode model frameworks to answer questions other than those for which the models were intended.

The majority of helminths found in Saiga antelope (*Saiga tatarica*) in Kazakhstan are shared with livestock, including 25 out of 26 species of GINs (Morgan et al., 2005; Table 2). These species include several of the major economically important GINs infecting livestock, such as *H. contortus* and *Teladorsagia circumcincta*. Building on the basic model framework of Coyne and Smith (1994) and Smith and Grenfell (1994), Morgan et al. (2006) simulated the seasonal transmission dynamics of GINs between Saiga antelope and domestic sheep in Kazakhstan. The model, parameterised using empirical data from studies on livestock, offered valuable new insights into the periods of peak transmission of GINs in Saiga,

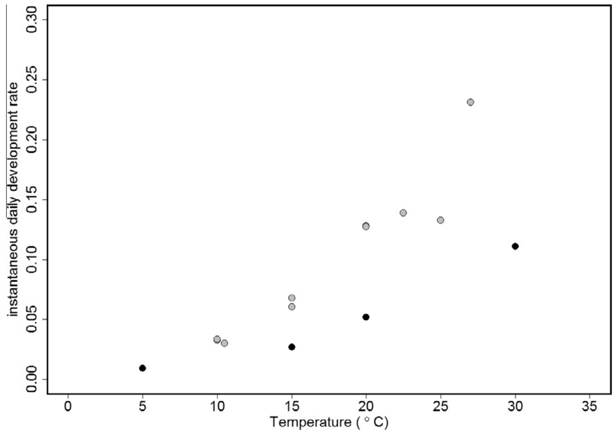

Fig. 1. Comparison of the instantaneous daily development rate of *Ostertagia ostertagi* (grey) and *O. gruehneri* (black) at a range of constant temperatures. Instantaneous daily development rates were estimated from the time to 50% development of L3, derived from data published in the literature (*O. ostertagi*: Rose, 1961; Pandey, 1972; Young et al., 1980) and original data (*O. gruehneri*: Hoar, 2012) as described by Azam et al. (2012).

and the contribution of both host species to transmission dynamics. Such applications and adaptations of theoretical frameworks developed for livestock systems can have important implications for conservation and livestock production where there is transmission between livestock and wildlife. Furthermore, these models could be easily extended to incorporate weather data based on climate scenario projections to predict the likely impact of climate change on transmission dynamics.

3.1.2. Bridging the gap between livestock and wildlife GINs

There is an abundance of data for estimation of key life-cycle parameters of economically important GIN species, dating back several decades. However, there are a number of GINs that infect host species of conservation concern but are not economically important to the livestock sector. Recent studies are generating valuable data for parameterisation of wildlife-GIN models (Hoar, 2012; Hoar et al., 2012a,b; Carlsson et al., 2013) but significant gaps remain. Generating meaningful predictions in the absence of sufficient data for model parameterisation and validation will be difficult. In these cases, alternative methods of parameterisation must be sought. Using a Metabolic Theory of Ecology, Molnár et al. (2013) were able to generate estimates for the temperature-dependent development and mortality rates of the free-living stages of *O. gruehneri*, an Arctic nematode species that infects caribou and reindeer (*Rangifer tarandus* spp.). The parameter estimates were consistent with observations of development and mortality obtained under field and laboratory conditions, demonstrating the potential for further application of this method to other species. It may also be possible to substitute missing parameter estimates with estimates derived from closely related species if validation data are available. However, care should be taken to ensure key parameters such as temperature-dependent development rates are accurately estimated, as divergent evolutionary histories can result in significant differences between closely related species (Hoberg et al., 1999; Fig. 1).

3.2. Immunoepidemiology

Acquired immunity is an important regulatory process in gastrointestinal nematode infection, reducing nematode establishment, survival, fecundity and development (Barger et al., 1985; Woolhouse, 1998; Roberts, 1999). Therefore, an understanding of acquired immunity and its impact on host–parasite dynamics is essential to predicting the impact of global change on infection dynamics.

Much of our knowledge of the immune response and development of acquired immunity of ruminants to GIN infection is based on studies on livestock. These studies demonstrate the potential for ruminants to mount a strong regulatory immune response to GIN infection and the presence of acquired immunity (Barger et al., 1985; Armour, 1989; Winter et al., 1996; Vercruysse and Claerebout, 1997). However, assessing levels of acquired immunity is complex (Claerebout and Vercruysse, 2000). Consequently, acquired immunity in GIN models (*cf.* immune response e.g. Singleton et al., 2011) is often represented implicitly by decreasing establishment rate as a function of the duration of infection (Grenfell et al., 1987), or explicitly as a single measure of resistance that increases and decays as a function of the duration of, or magnitude of exposure to, infective stages (Anderson and May, 1985; Roberts and Grenfell, 1991; Grenfell et al., 1995; Roberts and Heesterbeek, 1995). However, it is unclear to what extent the potential application of acquired immunity functions based on livestock ruminants can be extended to free-ranging ruminants.

3.2.1. Evidence for acquired immunity to GINs in wild ruminants

Evidence for acquired immunity to GINs in free-ranging ruminants is mixed. Acquired immunity from repeated exposure to helminth infection should result in a convex age-intensity curve (Anderson and May, 1985), i.e. the intensity of infection is lowest in the youngest individuals that have not had sufficient opportunity to accumulate a significant worm burden and the oldest individuals that have developed acquired immunity to infection. Irvine et al. (2000) found no decrease in the intensity of infection of *O. gruehneri, M. marshalli* or *Teladorsagia* spp. with increasing age in Svalbard reindeer (*R. tarandus platyrhynchus*) and therefore no evidence of acquired immunity. No significant differences were observed in the age-intensity profile of *O. gruehneri* and *T. boreoarcticus* worm burdens in a herd of barrenground caribou (*R. tarandus groenlandicus*; Hoar, 2012). A similar pattern was found for *M. marshalli* in a survey of culled Saiga antelope in Kazakhstan (Morgan et al., 2005). However, in the same study a decrease in the intensity of infection with *N. gazellae* was observed in Saiga over 3.5 years old, independent of sample size, suggestive of either acquired immunity or parasite-induced mortality. Finally, Davidson et al. (1980) observed lower *H. contortus* burdens in adult white-tailed deer (*Odocoileus virginianus*) than in fawns, indicating potential acquired immunity, which was later confirmed experimentally. Deer previously exposed to *H. contortus* infection (“immunised”) had lower worm burdens than naïve deer (“nonimmunised”) following a challenge infection. Furthermore, although one of the three immunised deer had worm burdens comparable to the nonimmunized deer, the faecal egg count was substantially lowered. Although the sample size in this study is too small to draw conclusions, the study indicates the potential for white-tailed deer to develop effective acquired immunity against *H. contortus* resulting in immune exclusion of ingested L3 and immune regulation of nematode fecundity.

It is important to note that the absence of evidence for acquired immunity does not equate to the absence of acquired immunity. In Svalbard reindeer the age-intensity curves for *M. marshalli, O. gruehneri,* and *Teladorsagia* spp. reached an asymptote with increasing age (Irvine et al. 2000). This may indicate the presence of some degree of protective immunity or other underlying density-dependent processes regulating worm burdens. At the same time, artefactual convexity in age-intensity curves can arise from overdispersion along with lower sample sizes in older age classes (Pacala and Dobson 1988). Insights into acquired immunity in livestock may shed light on the development of acquired immunity in free-ranging ruminants.

3.2.2. Differences in acquired immunity between livestock and wild ruminants

There may be a number of reasons for the mixed evidence for acquired immunity to GINs in free-ranging ruminants. Consistent with the evidence presented for acquired immunity in free-ranging ruminants above, a strong regulatory immune response is mounted in livestock against highly pathogenic species such as *H. contortus* (Barger et al., 1985) and *N. battus* (Taylor and Thomas, 1986). Therefore, models describing the acquisition of immunity against *H. contortus* and *N. battus* may be applicable to free-ranging ruminants. However, the development of acquired immunity is not simply a function of pathogenicity. Cattle rapidly develop protective immunity against *C. oncophora* within 1 year, but acquired immunity against a more pathogenic species, *O. ostertagi,* is slower to develop (Vercruysse and Claerebout, 1997).

The observed differences in the response of livestock and free-ranging ruminants to GINs in the subfamilies Osertagiinae (which includes *Marshallagia* spp.) and Cooperiinae could be a question of scale. Studies on GIN infections in cattle suggest that the development of acquired immunity is dependent on the level of exposure to infection (Ploeger et al., 1995). Irvine et al. (2000) observed mean total worm burdens of 6675 in reindeer calves in late winter, which is an order of magnitude lower than observed mean worm burdens in cattle calves naturally infected by *O. ostertagi* and *C. oncophora* (Armour, 1989). Therefore, the apparent absence of an acquired immune response may simply be a consequence of lower exposure to infection in free-ranging ruminants.

Alternatively, Irvine et al. (2000) suggest that the lack of evidence for acquired immunity to GINs in Svalbard reindeer may reflect the high cost associated with mounting an immune response. Coop and Kyriazakis (1999) propose a nutrient partitioning framework whereby growth (including repair and replacement of lost protein and damaged tissue as a result of GIN infection) and reproduction are prioritised over immunity, potentially explaining differences in acquired immunity observed in domestic and free-ranging ruminants, as well as the peri-parturient relaxation of immunity. While livestock ruminants are usually maintained on a high plane of nutrition, free-ranging ruminants often exist under conditions of nutrient deficit (Murray et al., 2006). Under these conditions, the limited nutritional resources may be allocated to reproductive effort and body maintenance at the expense of the immune response. Studies using the mouse-*Heligmosomoides polygyrus* model suggest that energy and protein deficits result in down-regulation of Th2 cytokines essential to the immune response against GINs, ultimately increasing the longevity of the nematodes (Koski and Scott, 2001).

Where the benefits of mounting an immune response outweigh the energetic costs, an increase in immune function may be expected. Migratory species, in particular, may be exposed to a wider range of pathogens and parasites during migratory periods than their resident counterparts. Pairwise comparisons of spleen and bursa of Fabricius size in migratory and non-migratory birds of the same genus revealed that, relative to body mass, migratory birds' immune defence organs were larger than non-migratory birds' (Møller and Erritzøe, 1998). Assuming that spleen and bursa size are both heritable, and that bursa size is also related to recent exposure to parasites, Møller and Erritzøe suggest both an increased exposure to parasites during migration and the evolution of larger immune defence organs in migratory birds. This concept remains to be tested in migratory ruminants.

3.2.3. The impact of global change on the immunoepidemiology of GIN infections in ruminants

The differences observed in acquired immunity in livestock and free-ranging ruminants could be (and are likely to be) due to interactions between parasite species (pathogenicity), exposure to infection and allocation of limited resources (ecological immunology; Buehler et al., 2010). Understanding the immunoepidemiology of GIN infections in free-ranging ruminants will become increasingly important when predicting the impact of climate and global change on infection dynamics, particularly where changes in the intensity of infection and infection pressure are expected and when considering migratory species.

Taylor and Thomas (1986) subjected 8 week old lambs to primary and challenge *N. battus* infections. Following challenge, lambs were divided into "responders" and "non-responders" based on worm burden, worm size and worm fecundity. Non-responders' mean worm burdens were almost 80 times greater than responders. The significant variation in observed immune response in livestock demonstrates the potential for substantial uncertainty in predictions based on inaccurate assumptions of presence or absence of acquired immunity. Assuming a weak immune response will result in higher predicted worm burdens, increased pasture contamination by free-living stages and thus increased transmission to susceptible hosts, whereas assuming a strong immune response will result in lower predicted worm burdens, lower pasture contamination and reduced transmission to susceptible hosts. This highlights the potentially key role that host immunity may play in predicting GIN transmission dynamics.

It is important to also consider the potential for altered host immunocompetence (and hence the altered epidemiology of GIN infection and parasitic disease) due to indirect effects of climate change. Studies on migratory birds suggest that the demands of migration result in changes in the immune response to parasites and pathogens (reviewed by Altizer et al., 2011). Climate-driven changes in host distribution, behaviour and population dynamics, such as changes in the timing and route of migration that result in increased energetic demands, and access to less favoured, nutritionally deficient habitat, could compromise the immune response to GIN infection. Thus, simulations considering the impact of global change or management strategies on GIN infections in free-ranging ruminants, need to consider the effect of changes on host ecology (such as migration) on immune response.

3.3. Seasonal variation in host distribution and population dynamics

Several interacting factors drive the transmission dynamics and epidemiology of nematode infections in both livestock and wildlife ruminants including reproduction, parasite life-history, forage availability and host movements. Species distribution models have revealed the potential for ecological mismatch between the free-living stages of the protostrongylid nematode *Parelaphostrongylus tenuis*, its definitive host (white-tailed deer) and several gastropod intermediate hosts (Pickles et al., 2013). Therefore, simply predicting changes in the potential developmental success or geographic range of GINs under scenarios of global change is insufficient to predict changes in infection dynamics. The seasonal availability of susceptible hosts and host adaptation to climate change must also be considered.

3.3.1. Current patterns of host availability

Despite their divergent evolutionary histories and the almost complete control farmers exert over the life-history and ranging

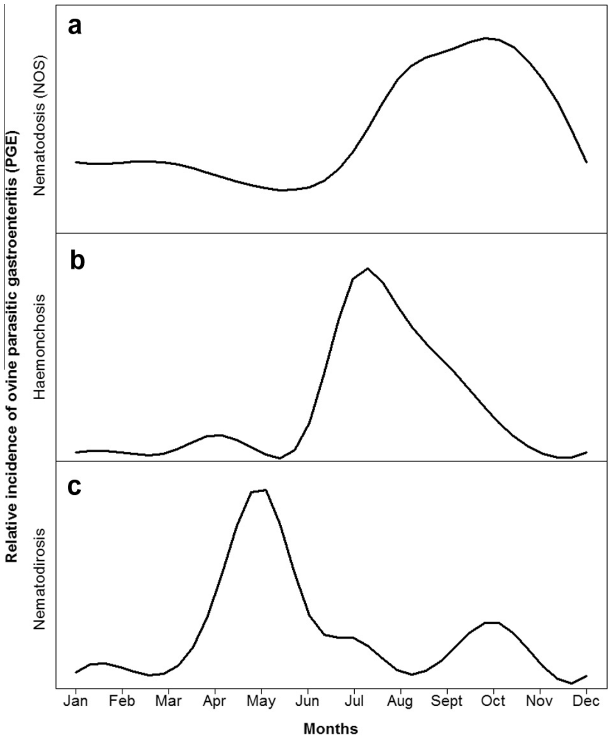

Fig. 2. The relative seasonal incidence of ovine parasitic gastroenteritis (PGE) in the Southwest of England, UK, based on monthly diagnoses of (a) Nematodosis (NOS = species not otherwise specified), (b) Haemonchosis and (c) Nematodirosis (van Dijk et al., 2008).

behaviour of livestock, there remain some surprising similarities in the annual life-cycle and movements of livestock and free-ranging ruminants. While the separation of hosts of different sexes and age class, and highly managed seasonal breeding patterns, are typical of many livestock systems such as dairy cattle herds, in other livestock systems and in free-ranging ruminants considerable temporal variation exists in the age-structure and spatial distribution of the host populations. Such temporal variation in contact between hosts of different ages, sex, and susceptibility to infection could have a significant impact on patterns of GIN transmission and should therefore be considered when predicting the impact of global change on GIN-ruminant systems. Since some GIN transmission models include livestock movement and reproduction (Roberts and Heesterbeek, 1995; Kao et al., 2000; Guthrie et al., 2010) and others can be easily adapted to simulate host movements (Morgan et al., 2007), existing GIN transmission models can be applied to free-ranging ruminant systems, where long-distance migrations and seasonal variation in host densities and group structure (e.g. during rutting) are common.

Model simulations indicate that acquired immunity to GIN infection, periodic removal of infected animals (i.e. livestock sales) and replacement with new susceptible hosts (i.e. lambing and calving) are necessary to produce the qualitative patterns in the availability of infective larvae on pasture observed in livestock systems (Roberts and Grenfell, 1991), whereas seasonal variation in the development and mortality of the free-living stages is responsible for the magnitude of the worm burdens (Roberts and Grenfell, 1992). In sheep production systems in temperate regions lambing provides a fresh cohort of susceptible hosts in the spring. Interactions between the timing of lambing, host immunity, pasture management and a favourable climate for development of eggs to infective larvae, gives rise to peaks in *Nematodirus* spp. infections in spring, *H. contortus* infections in summer and *Teladorsagia* and *Trichostrongylus* spp. infections in late summer/early autumn (Fig. 2).

The relative contribution of management, acquired immunity and seasonal variations in the development and mortality of free-living stages to the observed seasonal patterns of the availability of infective stages on pasture will vary dependent on GIN species, host species (acquired immune response) and management system. For example, in livestock systems the initial source of pasture contamination in the spring is heavily dependent on climatic conditions and species-specific variation in the ecology of the free-living stages of the GINs. Most GIN species survive well on pasture over winter in temperate regions such as the UK, resulting in the potential for year-round availability of infective stages on pasture (*Teladorsagia and Trichostrongylus* spp.; Fig. 2a) and infection of young lambs in spring (*Nematodirus* spp.; Fig. 2c). However, over winter survival of *H. contortus* infective stages on pasture is low in temperate regions (Rose, 1963) and the primary source of *H. contortus* pasture contamination for spring lambs is ewes that have developed patent infections as a result of the peri-parturient relaxation of immunity and maturation of hypobiotic larvae (fourth-stage larvae that have undergone arrested development in the gut mucosa). This results in an initial peak in infection in ewes around the time of lambing followed by a peak in infection in lambs later in the summer (Fig. 2b). Since many free-ranging ruminants exhibit similar seasonal reproductive cycles to those seen in livestock systems, we can draw on these analogies and our understanding of the impact of the reproductive cycle in livestock on infection dynamics, to fill gaps in our understanding of the epidemiology of GIN infection in wildlife.

Roberts and Grenfell's (1991, 1992) models demonstrate the importance of host removal, which could also include parasite-induced mortality in wildlife hosts, and annual reproduction cycles on the observed patterns of GIN transmission. However, these models consider a population of hosts in a fixed location, such as on a single pasture. Transhumance and movement of livestock between pastures is employed worldwide to maximise the use of seasonally and inter-annually variable resources, primarily water and forage (Boone et al., 2008). Throughout Europe it is common for sheep in marginal grazing systems to have distinct summer and winter grazing areas. In British upland areas, sheep are grazed on extensive areas of common land at low stocking densities between approximately April and November (Fig. 3). The majority of sheep are then "away grazed" during the winter months on improved grassland in lowland areas. On their return from away grazing in March/April, yearlings are turned back out onto the common land and pregnant ewes are maintained at higher stocking densities on limited land or housed near the farmhouse for lambing before

Fig. 3. In marginal grazing systems in Europe sheep often occupy separate summer and winter grazing areas, analogous to the summer and winter ranges of migratory ruminants. In the uplands of Wales, UK, (shown here) sheep are often grazed on extensive areas of land at low stocking densities over the summer period, and sent to lowland dairy farms for winter grazing at higher stocking densities. (Photo: Rose, H.).

returning to the common land in June/July (Rose, H. unpublished data).

Similar patterns are seen in livestock production systems globally (Morgan et al., 2007; Boone et al., 2008) and in the natural seasonal movements and variation in density of migratory ruminant species. Migratory caribou occupy winter feeding grounds south of the tree line and, following the spring green-up, migrate to calving and summer ranges on the arctic tundra hundreds of kilometres north of the winter grounds (McNeil et al., 2005; Festa-Bianchet et al., 2011; Hoar, 2012). Saiga antelope migrate between winter grounds in southern Kazakhstan and northern summer grazing grounds (Bekenov et al., 1998; Morgan et al., 2006) and bighorn sheep (*Ovis canadensis*) migrate to higher elevations for lambing and summer grazing to avoid predators and optimise nutritional intake (Festa-Bianchet, 1988).

In addition to seasonal host population dynamics, movements of livestock and wildlife are likely to lead to altered spatial patterns of pasture contamination and exposure to infective stages that are left behind when the hosts move. Seasonal movements of livestock and wildlife may also interact to increase GIN transmission risk between species (Morgan et al., 2007). The spatial distribution and the timing of reproduction of ruminants are to a great extent determined by forage availability (Post et al., 2003) and ecological barriers (see Kutz et al., this issue), as well as increasingly by anthropogenic influences (Singh et al., 2010). How free-ranging ruminants and farming strategies adapt to climate change and altered vegetation growth patterns and human influences will, therefore, play an important role in determining the future impact of GINs on the sustainability of the livestock industry and the conservation of wild ruminants.

3.3.2. Host adaptation to climate change

In both livestock and migratory wildlife systems, the return to the summer range or calving area is often determined by climatic conditions and the rate of herbage growth (McNeil et al., 2005). Any changes in annual patterns of herbage growth may lead to altered host distribution and GIN transmission. Recent shifts in plant phenology have been observed, coinciding with increases in temperature and a lengthening of the growing season (Murray et al., 2006; reviewed by Cleland et al., 2007). Continued climate change may result in complex changes in seasonal vegetation growth rates (Hunt et al., 1991; Duru et al., 2012), potentially leading to altered grazing patterns in livestock (Duru et al., 2012) and wildlife (McNeil et al., 2005). Furthermore, varying degrees of phenological and distributional changes at different trophic levels due to climate change can lead to a mismatch in the phenology and ecology of interacting species. Egg hatch date advanced less in four species of passerines than the advancement of the peak caterpillar biomass date, resulting in a mismatch between the timing of increased food demand and peak food availability (Both et al., 2009). Since the epidemiology of GIN infections in livestock is inextricably linked with farm management and host availability, how a farmer or ruminant host responds to changes in grass growth and parasite phenology will determine the overall impact of climate warming on the seasonal dynamics of infection. Analysis of phenological traits of wild plants, fruit trees, and agricultural plants in Germany revealed that traits influenced by farmer intervention, such as harvest time, advanced less than non-farmer driven traits (Menzel et al., 2006), indicating that some mismatch between future grass growth, parasite phenology and host factors (the latter being heavily influenced by farmer intervention) could be expected in agricultural systems.

In ruminant-GIN interactions, there is potential for a decoupling of host reproduction and hypobiosis, affecting the epidemiology of infection in lambs and calves. Hypobiosis enables the persistence of GINs during periods unfavourable for transmission (Gibbs, 1982; Hoar et al., 2012a), and may also stabilise host–parasite population dynamics in systems with a degree of aggregation similar to that observed for *O. gruehneri* in reindeer and *T. circumcincta* in Soay sheep (Gaba and Gourbière, 2008). The key factors determining when trichostrongyle nematodes enter and exit hypobiosis (inhibition and disinhibition) are poorly understood: study design is complicated by correlated and confounding variables such as host age, temperature and photoperiod; species ecotypes and isolates vary in their propensity to arrest; and factors associated with inhibition and disinhibition vary between and within species (reviewed by Michel, 1974; Gibbs, 1982; Eysker, 1993, 1997; Somerville and Davey, 2002). Understanding the factors driving patterns of hypobiosis will be important in predicting the potential epidemiological implications of global change and any alterations in the seasonal distribution and reproduction of ruminant hosts. If a minimum period of hypobiosis is required, as observed for *O. gruehneri* in experimental infections of reindeer (Hoar et al., 2012a), any advances or delays in host reproduction could result in an asynchrony between maturation of hypobiotic larvae and the arrival of susceptible hosts. A phenological mismatch between parasite and host may also occur if hypobiosis is dependent entirely on host physiological status or environmental factors, and if changes in seasonal reproductive behaviour occur at a different rate to environmental changes. A longitudinal study of farms practicing “out of season lambing” (lambing throughout the year) in Ontario, Canada, designed to elucidate whether a peri-parturient relaxation of immunity or environmental factors gave rise to the peri-parturient rise in *H. contortus* faecal egg counts (FEC) in ewes, concluded that seasonal variations in both environmental and physiological factors determined patterns of inhibition and disinhibition of *H. contortus* (Falzon et al., 2013). A peri-parturient egg rise was observed in ewes regardless of timing of lambing (autumn, winter or spring). A concurrent increase in FEC was not observed in ewes that were not pregnant or that were classed as in early gestation during the spring lambing season, suggesting that the peri-parturient egg rise was not associated with seasonal variability in environmental factors. However, FEC in ewes increased during late gestation in autumn lambing ewes but then decreased during lactation, indicative of an increase in arrest rate. These rather complex observations suggest that in *H. contortus* infecting sheep in Ontario, environmental factors are important in driving the inhibition of ingested larvae during autumn, whereas host physiology determines the disinhibition rate of hypobiotic larvae in existing infections.

Out of season lambing may become more common under climate change and altered grass growth patterns. There is also considerable potential for farmers to use phenological mismatches between parasite and host availability to their advantage, adopting altered farm management strategies to avoid grazing and lambing at periods of peak transmission risk (Morgan and Wall, 2009). However, predicting farmer behaviour is complicated by socio-economic, psychological and technological considerations (Edwards-Jones, 2006). In this case, observations on the current distribution and behaviour of wild ruminants, and recent changes associated with climate change, can reveal potential directions of adaptation to climate change and the consequences of these changes (Kutz et al., 2009).

Variability in the current seasonal distribution and migratory behaviour of two caribou herds in Arctic Canada and Alaska in relation to environmental factors such as the speed of green-up and snow depth suggested that there may be complex changes in the density, migration routes, and spatial distribution of the caribou herds due to changes in the timing and depth of snow cover, faster green-up and greater insect harassment expected under future climate change (McNeil et al., 2005). Climate change associated with increased vegetation growth (growing degree days) at key periods in the annual breeding cycle of Scottish red deer (*Cervus elaphus*) on the Isle of Rum, UK, has already resulted in significant changes in the phenology of the deer (Moyes et al., 2011). Notably, an advance in parturition dates of 12 days over the 28 year study period was associated with a significant increase in growing degree days in the months preceding the rutting period in the previous year. In the Scottish red deer population, advances in parturition did not significantly impact calf birth weight and survival (Moyes et al., 2011). However, observations of caribou indicate that changes in the timing of reproduction that result in trophic mismatches may have negative consequences. Between 1993 and 2006, Post and Forchhammer (2008) observed a mismatch between the onset of the plant growing season, which advanced by 14.8 days, and the onset of calving in caribou, which advanced by only 3.82 days. The change coincided with an increase in calf mortality and a decrease in calf production. These studies suggest that advances in lambing dates and spring turnout of livestock onto pasture in line with advances in the onset of the grass growth season will enable farmers to maximise pasture utilisation and productivity, but failure to track changes in grass growth could result in productivity losses due to mismatches between pasture availability and demand.

There is also, of course, the possibility of parasite adaptation to climate change and changes in host availability. Significant variation in hatching behaviour (van Dijk and Morgan, 2010) and the propensity for developmental arrest (Troell et al., 2006) has been observed in trichostrongyloid nematodes. Such variation may enable transmission under fluctuating environmental conditions, potentially providing a pre-adaptation for global change (van Dijk and Morgan, 2010). Scenario-based model simulations will enable the evaluation of the likely impact of observed and predicted changes in ruminant migration, reproductive patterns and population dynamics, along with the potential for parasite adaptation, on the epidemiology of GIN infection in livestock and free-ranging ruminants.

4. Conclusion

Global change, including changes in climate, land use, vegetation growth, policy and host availability, is likely to have a major impact on the seasonal transmission of GINs in livestock and free-ranging ruminants. However, predicting the impact of global change is complex, and relies on a detailed understanding of parasite and host biology, ecology, and interactions. Since livestock GIN species and closely related species are present in free-living ruminants, the impact and potential application of research on patterns of transmission of GINs in sheep and cattle extends beyond livestock systems. Parallels between livestock and wildlife may offer significant opportunities to parameterise existing mechanistic models for GIN species infecting free-ranging ruminants.

Further development of GIN transmission models focussing on providing a single core framework integrating the parasite life-cycle with climate and host movements would enable closer integration of livestock- and wildlife-GIN systems to explore the impact of global change on the dynamics of GIN infections in ruminants. Where possible, variation in the key life-history and immunological parameters of GIN and host species should be compared to evaluate the feasibility of the application of livestock GIN models to free-ranging systems on a case-by-case basis. Variation in the host immune response to GIN infection may be of key importance under global change scenarios where dramatic changes are predicted in the timing and magnitude of exposure to infective stages and/or intensity of infection, or where predicted changes in forage availability/migratory behaviour are likely to result in significant changes in energy demands on the host.

Continued monitoring of parasite diversity and infection dynamics in free-ranging species will provide a baseline against which we can measure the impact of global change, but also provide valuable longitudinal datasets that can be used to validate the application of livestock-GIN models to free-ranging systems.

However, knowledge exchange is a two-way process. Wildlife host–parasite systems in rapidly changing environments, such as barrenground caribou and *O. gruehneri* in Arctic Canada, are offering unparalleled opportunities to observe the response of ruminants and their parasite fauna to climate change and potentially a rare chance to validate model predictions. A greater synthesis of parasitology research in wildlife and livestock is encouraged to further our understanding and improve predictions of the patterns of infection and disease under likely global climate change scenarios.

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