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Climate disruption and parasite–host dynamics: patterns and processes associated with warming and the frequency of extreme climatic events

P.J. Hudson[1]*, I.M. Cattadori[1], B. Boag[1] and A.P. Dobson[2]

[1]Center for Infectious Disease Dynamics, Penn State University, PA 16802, USA: [2]Department of Ecological and Evolutionary Biology, Princeton University, Princeton, NJ 08544, USA

Abstract

Levels of parasitism and the dynamics of helminth systems is subject to the impact of environmental conditions such that we may expect long term increases in temperature will increase the force of infection and the parasite's basic reproduction number, R_0. We postulate that an increase in the force of infection will only lead to an increase in mean intensity of adults when adult parasite mortality is not determined by acquired immunity. Preliminary examination of long term trends of parasites of rabbits and grouse confirm these predictions. Parasite development rate increases with temperature and while laboratory studies indicate this is linear some recent studies indicate that this may be non-linear and would have an important impact on R_0. Warming would also reduce the selective pressure for the development of arrestment and this would increase R_0 so that in systems like the grouse and *Trichostrongylus tenuis* this would increase the instability and lead to larger disease outbreaks. Extreme climatic events that act across populations appear important in synchronizing transmission and disease outbreaks, so it is speculated that climate disruption will lead to increased frequency and intensity of disease outbreaks in parasite populations not regulated by acquired immunity.

Introduction

The evidence that average global temperature anomalies have increased over the past 80 years with a consistent and faster rise over the past 25 years is now irrefutable (Stott, 2000). The earth' surface is a warmer place with higher maximum summer temperatures, longer summer seasons and fewer cold days. These seasonal climate changes affect a wide range of biological systems, increasing the duration of the seasonal breeding season so many organisms now breed earlier and longer than previously at the same location (e.g. Fitter & Fitter, 2002). For example studies on the phenology of breeding have shown that temperate invertebrates, birds and amphibians breed on average 5 to 7 days earlier than they did a decade ago while trees flower 3 days earlier and shrubs 5 days earlier (e.g. Cotton, 2003; Parmesan & Yohe, 2003). Warmer summer seasons are equivalent to a shift in average climatic conditions to higher latitudes and higher altitudes and so we may expect to see organisms expand their range accordingly with perhaps a parallel retreat from lower latitudes and altitudes. Meta-analyses of existing data confirm a consistent temperature-related shift in range of species as diverse as molluscs to mammals and from grasses to trees, although such data are invariably correlational and confounded with other anthropogenic changes in the environment (Root *et al.*, 2003). Undertaking controlled comparisons or experiments at large scales over long time periods is expensive and difficult and replication is almost impossible since climate change is a global phenomenon and events are not truly independent. Nevertheless, undertaking long term studies of abundance, coupled with field and

*Fax: (814) 865 9131
E-mail:pjh18@psu.edu

laboratory experiments and model fitting, should provide insights into the parsimonious mechanisms that explain how climate disruption has influenced natural systems, including parasite–host systems.

How will an increase in seasonality affect host–parasite systems? In general, if hosts breed earlier and for longer this will result in the production of more susceptible young individuals and this will lead to an increase in transmission. From the parasite perspective, an increase in the duration and average temperature of the summer season will also increase the window of transmission and development rate of infective stages resulting in increased transmission and effectively increase in the parasite's basic reproduction number, R_0 (Dobson & Carper, 1992). These arguments lead us to predict that global warming will lead to increased levels of parasitism. However, the observed level of parasitism in the host population will be a consequence of how the parasite is regulated. For example, in parasite–host systems with little acquired immunity a rise in the average level of parasitism would be expected as long as this is not balanced by increased parasite–induced mortality. In contrast, parasite–host systems where the parasite is regulated by acquired immunity may not have increased levels of parasitism simply because the faster infection level leads to faster response of the host's immune system and faster regulation of parasite abundance within each host. The best type of parasite–host system for investigating how climate change may affect parasite intensity should be one from high latitudes or across variable altitudes where we can expect the effects of climate change on seasonality to be greatest and where we can examine how infection patterns vary. In the present paper we seek to add insights to the broad question: What are the effects of climate disruption on parasite-host systems? We examine long term data on gastro-intestinal nematodes in wild red grouse and rabbits sampled in the uplands of Scotland and England with respect to their patterns of mortality in adult hosts. We go on and examine how climate disruption could influence the life history of the parasitic stages and the effect this would have on temporal and spatial dynamics.

Climate disruption has two components, first an increase in average temperatures and second, an increase in extreme environmental events including an increase in the frequency of storms and extreme temperature and precipitation events (Easterling *et al.*, 2000) While we start by looking at the effects of increased temperature and the force of infection on mean intensities and temporal dynamics, we also examine how the variability in the weather conditions could influence the development rate of parasite stages and how these effects could synchronize transmission across host populations. We stress that these data were never collected specifically to examine hypotheses of the effects of climate disruption. However, when considered together they lead us to postulate that climate disruption could lead to increased rates of disease outbreaks when host immunity does not play a major role in regulating parasites. These findings indicate that more rigorous analysis of parasite–host systems would provide better insights into the effects of climate disruption on the frequency of disease outbreaks.

Temporal patterns in parasites associated with climate disruption

Since most parasitic worms do not replicate within their definitive host, the host-age parasite-intensity relationship is essentially a birth death process where increases are a function of transmission and final equilibrium levels determined by the point at which transmission and parasite mortality rate are balanced (fig. 1., Hudson & Dobson, 1995, Hudson *et al.*, 2002). In some parasite species, the mortality rate of the parasite is negligible so that infection levels simply increase with the age of the host, referred to by Hudson & Dobson (1995) as a type I age intensity relationship (fig. 1a). One example of a type I is the rabbit stomach nematode *Graphidium strigosum* where the mean intensity increases linearly with age indicating that parasite mortality rates must be trivial (fig. 2a). Indeed, comparative data show that even in immunosuppressed individuals there is no change in the slope of this relationship, indicating that immunity is not affecting mortality (I.M. Cattadori, B. Boag & P.J. Hudson, unpublished). With increased warming and an increased force of infection we would predict the slope of this age intensity relationship to increase and a positive relationship between the annual mean level of infection and average temperature would be observed. As expected, a rise in the mean intensity of *G. strigosum* is observed over time, in association with increased summer temperatures (fig. 2a).

In nematode species with a constant number of worms recruited and a constant mortality rate the age intensity curve will rise to an asymptote determined by the balance between birth and death rates (type II, Hudson & Dobson 1995), as illustrated by the caecal nematode of red grouse, *Trichostrongylus tenuis* (fig. 2b). In this system, if warming increases the force of infection but does not result in an increase in parasite mortality then the curve is expected to rise more sharply to a higher asymptote (fig. 1b) although this level will be influenced by any changes in the parasite-induced mortality rate and host birth rate. In general, over a period of warming a steady increase in the mean level of infection in mature hosts is expected. From our long term study of *T. tenuis* in red grouse an increase in the mean levels is observed but these are highly variable between years and determined in part by the relative abundance of the host (fig. 2b).

When parasitic worms stimulate acquired immunity then the shape of the age intensity curve will rise to a peak, turn over and decline in older hosts (type III, Hudson & Dobson, 1995) as the worm mortality rate increases with the immune response (fig. 1c). If we assume that immunity develops as a consequence of the cumulative exposure of the host to infections then any increase in the force of infection brought about by warming would change the shape of this relationship, leading to faster rates of infection and an earlier peak in infection of larger magnitude (fig. 1c); a pattern known as 'peak shift' (Woolhouse, 1998). As such, during a period of warming we would expect no change in the mean

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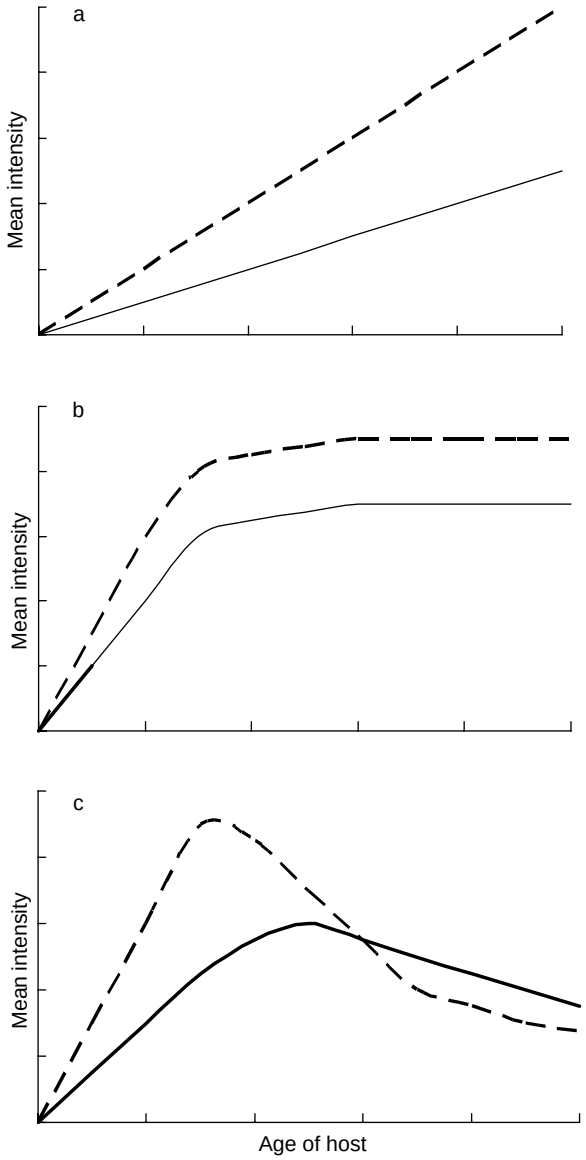

Fig. 1. Age intensity curves for parasite species with a moderate (solid line) and high (dashed line) force of infection with respect to adult worm mortality: (a) no worm mortality (type I), (b) constant worm mortality (type II) and (c) increasing worm mortality introduced thorough acquired immunity (type III). Note that increasing the force of infection through such processes as climate warming would increase intensity in old hosts in both of the first two situations but not with acquired immunity.

intensity in adults or even a decrease as the immune response in adults becomes stronger. *Trichostrongylus retortaeformis* is a nematode infection of the small intestines of rabbits that exhibits a characteristic turnover in its age-intensity curve (fig. 2c) which is consistent with the principle of acquired immunity and not other explanations (Cattadori *et al.*, 2005a). As expected, long term data show no increase in the mean level of infection which contrasts well with the increase observed in the *G. strigosum* collected from the same individuals over the same time period (fig. 2c). In summary, this cursory examination of two systems leads us to suppose that climate disruption will have a greater impact on species not regulated by acquired immunity.

Environmental variation and parasite development rate

In general, there is a minimum temperature below which free-living stages of parasites do not develop and as temperature increases so parasite development rate increases linearly. This is a pattern well documented in a wide range of nematode species where workers have incubated free-living stages at set temperatures and recorded development rates (Anderson, 2000). Development rate in natural conditions can then be modelled using a day-degree approach, based on the assumption that a fixed amount of thermal energy is required by a parasite to develop, and this accumulates daily according to the local temperature (e.g. Gettinby & Patton, 1981; Grenfell & Smith, 1983; Smith, 1990). Implicit in this assumption is that variation in temperature is not important but development is achieved after the thermal energy is accumulated; an assumption that has been confirmed in some systems (Gibson, 1981; Salih & Grainger, 1982).

In a study of the development rate of eggs of the caecal nematode, *Heterakis gallinarum*, Saunders *et al.* (2000) found that an increase in temperature conditions resulted in a linear increase in the development rate which could be described by a simple day degree model. However, when the parasite was placed in a daily temperature cycle, development started significantly earlier than that expected from the linear model (fig. 3a). Furthermore, the development time of eggs placed in stochastic fluctuations in temperature with the same thermal energy as the cycle is, again, significantly earlier (fig. 3b) indicating that fluctuations in temperature and particularly increased variation, could accelerate parasite development rates (Saunders, *et al.*, 2002).

Climate disruption, R_0 and temporal dynamics

Findings from the previous section show that climate disruption will lead to increased rates of parasite development and consequently faster infection rates and may lead to an to increase in the basic reproduction number, R_0. While changes in R_0 can have important effects on the dynamics of the parasite–host system, the dynamical changes depend on which components of R_0 are affected since even the same value of R_0 can have very different dynamical outputs. In fact, while climate change may increase larval development rate this will only affect dynamics if it increases the transmission rate (β), but if increased temperature leads to reduced humidity and this reduces the availability of infective larvae then one response may cancel out the other so there is no effective increase in β. Interestingly, there may also be other climate-mediated effects on the parasite–host system that occur within the host, for example trichostrongylid nematodes, ingested during periods of harsh weather conditions, enter a period of arrested development

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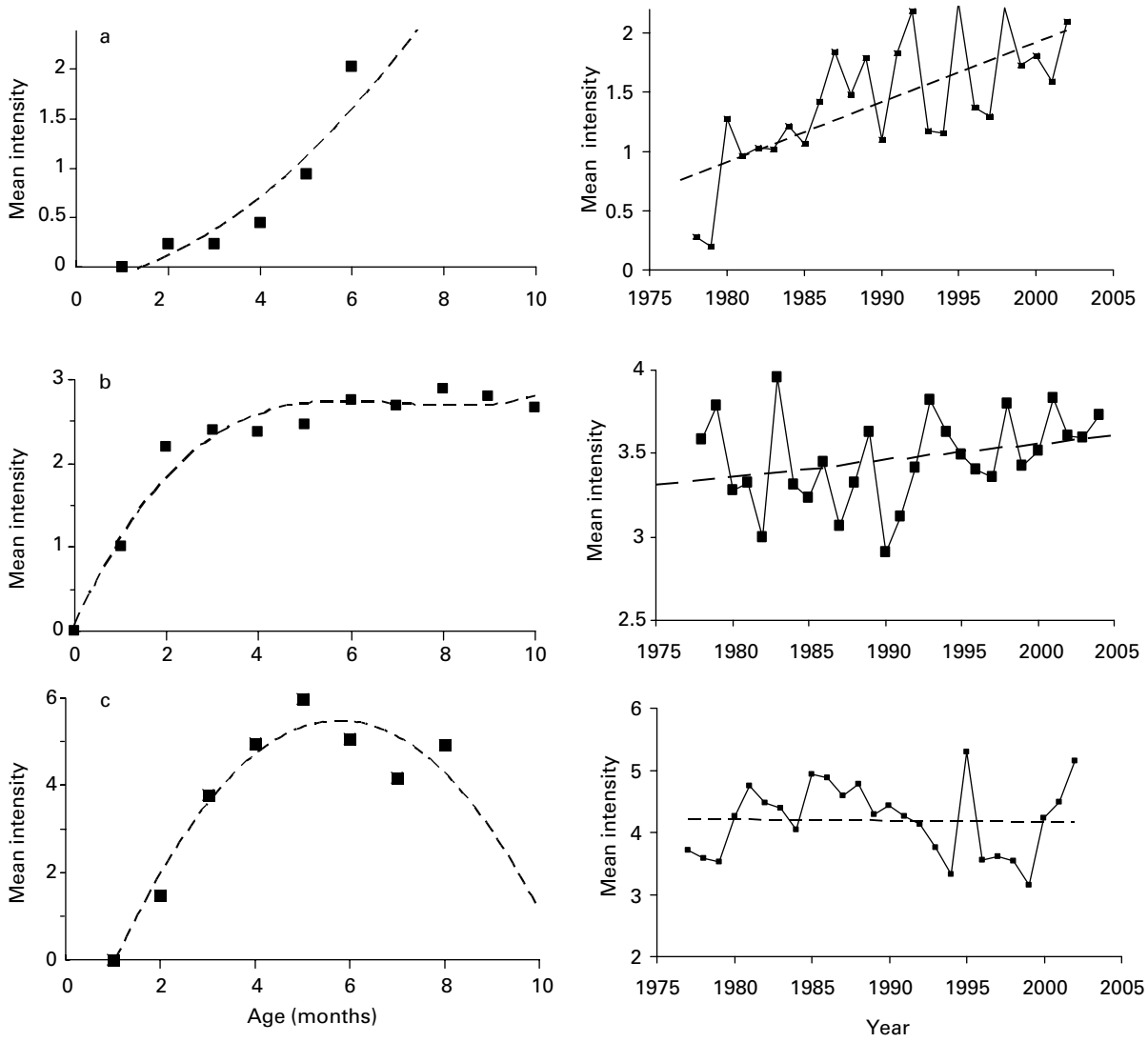

Fig. 2. Age intensity curves for three parasite species and long term trends in intensity: (a) *Graphidium strigosum*, stomach worm of rabbits, a type I age intensity curve, (b) *Trichostronglyus tenuis*, a caecal nematode of red grouse, a type II age intensity curve, (c) *Trichostronglyus retortaeformis*, a nematode of the rabbit small intestine with a type III age intensity curve generated by acquired immunity (Cattadori *et al.* 2005a).

known as hypobiosis and do not emerge until environmental conditions become favourable for transmission (Michel, 1974). This can result in a synchronized emergence of arrested stages at the time when hosts are collecting nutrients to breed and consequently have a major impact on host productivity. With warming we would expect selection against parasites entering an arrested stage and increased rates of parasite turnover which will increase R_0.

Consider the *T. tenuis*–grouse system where there is good experimental evidence to show that the parasite reduces the breeding output of the female grouse and plays an important role in generating the instability observed in shooting records of grouse populations (Hudson, 1986; Hudson *et al.*, 1992, 1998). Dobson & Hudson (1992) have captured the dynamics of this system in an analytical model that examines not only the host and adult parasite population but also the free-living stages and arrested larval stages. Using this model we can explore how the dynamics of the system varies with the basic reproduction number R_0 of the parasite and the effect of reduced arrestment. Essentially the basic reproduction number is the ratio of the birth rate to the death rate of the parasite, equivalent to the product of the average number of new infections established by each female worm and the life expectancy of each parasite stage. In a nematode system such as this, with arrested development, parasite birth rate is the product of the rate of egg production by the

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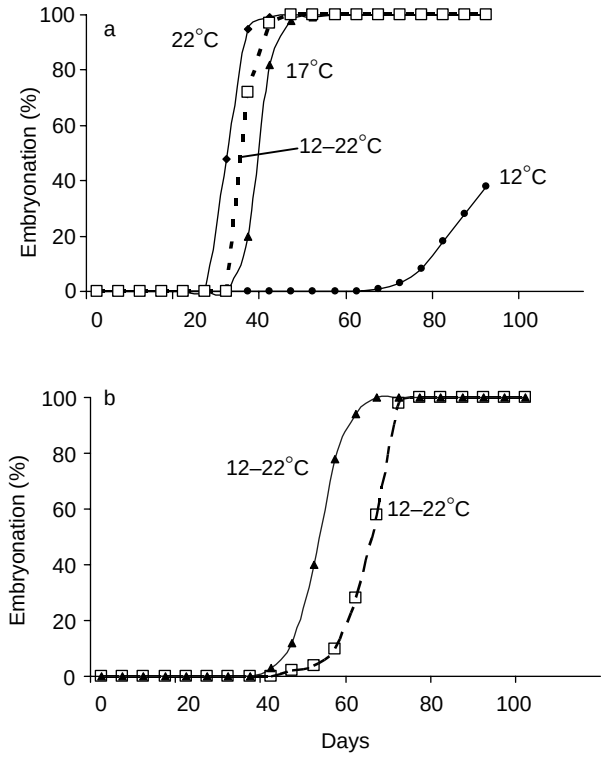

Fig. 3. Development rate of eggs of *Heterakis gallinarum*, a pheasant caecal nematode, in relation to constant and variable temperature conditions: (a) constant conditions (solid line) compared with a diel cycle (dashed), note that the diel cycle is faster than the mean of 17°C, (b) a diel cycle (dashed) compared with a stochastic cycle (solid) where the day degrees are equal, the eggs in the stochastic cycle developed faster (after Saunders *et al.*, 2000, 2002).

parasite (λ), the rate of ingestion of infective stages (β) and the size of the host population (H) and can be summarized in a single birth rate term: $T1 = \beta\lambda H$. Mortality rates include adult parasite mortality (μ_1), loss of parasites through the natural mortality of the host (b) plus the parasite induced mortality rate (α) and can be summarized as: $M1 = \mu_P + \alpha + b$. The mortality of the free-living stages is dependent on their own environmentally determined natural mortality rate (γ) and the rate at which infective stages are taken up by hosts and can be described as $M2 = \gamma + \beta H$. We assume the mortality rate of the arrested stages occurs through their own natural mortality rate (μ_A), host death rate (b) and the rate at which they develop into adults (θ) and can be expressed as $M3 = \mu_A + b + \theta$. By varying this development rate (θ) we effectively alter the period that larvae arrest and the proportion that arrest (σ) can also be considered. The basic reproduction number for this system with arrested development is then:

$$R_0 = T_1 M_1 M_2[(1 - \sigma) + \sigma\theta M_3]$$

The value of R_0 increases with the life expectancy of the free-living stages (γ) since this effectively increases rates of infection but R_0 will decrease with arrestment ($\theta + \sigma$) since the delay in development effectively reduces the rate of turnover in the parasite population. This leads us to suppose that if warming reduced arrestment and at the same time increased the development time this could have a dramatic effect on R_0 and may well alter the dynamics of the host population. This can be seen more clearly if the dynamics of the host population with respect to the host birth rate and the effective growth rate of the parasite population, R_0 (fig. 4) are considered. Increases in R_0, or indeed increases in β effectively increase the instability of the parasite–host system, increasing both the period of oscillations but also the amplitude, leading to outbreaks of parasitism followed by dramatic crashes in abundance (fig. 4a). Thus, in this system at least, an increase in R_0 would lead to increased an instability in the dynamics of the parasite–host system. Similarly, a reduction in the period of arrestment (θ) for any given host birth rate will tend to increase instability but in this instance shorten the cycle period since here the time-delayed feedback is being reduced.

Climate change, extreme events and synchrony in transmission

Climate could play an important role in synchronizing disease outbreaks over large spatial areas. More than 50 years ago, Moran (1953) proposed that independent populations which have the same underlying density-dependent structure will fluctuate in synchrony when their climatic conditions are correlated. The extent of this synchrony between populations will depend on the correlation of these climatic events between localities and this correlation would increase with increased frequency of large scale extreme events, as expected with climate change.

Cattadori *et al.* (2005b) examined the spatial synchrony of grouse populations in northern England and identified the years when grouse populations were unpredictably brought into synchrony. Analyses of climatic conditions together with levels of parasitism by *T. tenuis* in these years identified weather variables that were associated with these changes and subsequent model fitting provided evidence to suppose that climatic conditions acted on parasite transmission and either accelerated transmission, leading to a common, large-scale disease outbreak and leading to all host populations declining or acted to reduce transmission, leading to low parasite intensity and an increase in the host population. This is an important study since it provides good evidence to suppose that an increase in the frequency of extreme events could lead to spatially synchronized disease outbreaks.

Discussion

In the present paper we examined how climate disruption, in terms of warming, and an increase in temperature variability could influence parasite–host systems using data from a number of natural wild

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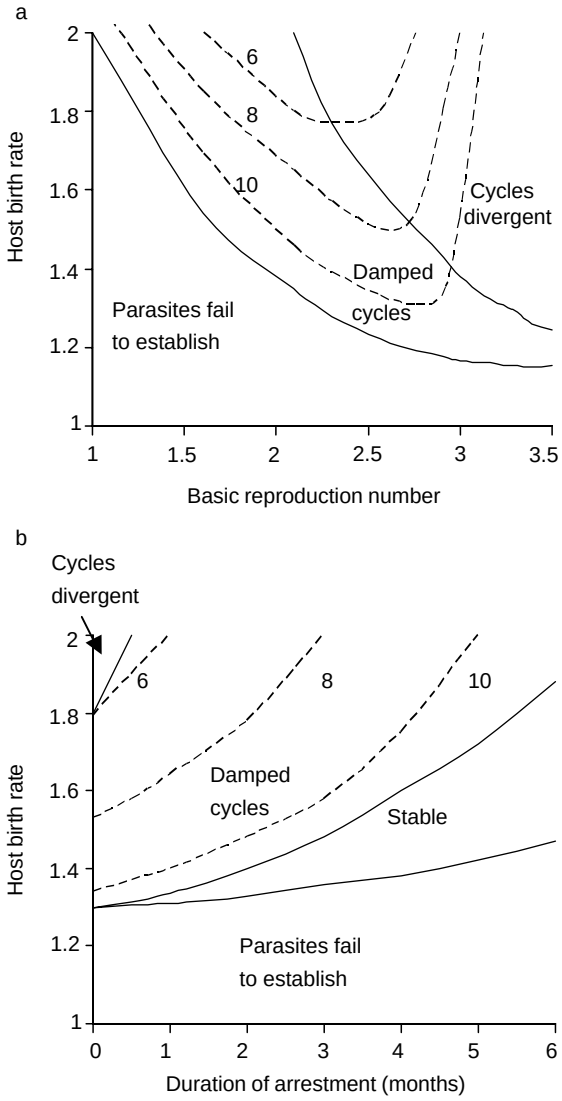

Fig. 4. Changes in the temporal dynamics of the red grouse–*Trichostrongylus tenuis* system, (a) in relation to changes in the basic reproduction number (R_0) assuming no arrestment and (b) with changes in the duration in arrestment but with constant larval survival of 4 weeks. The solid line denotes changes in the dynamics from parasites failing to establish, damped oscillations and divergent cycles (developed from Dobson & Hudson, 1992).

nematode–host systems. We showed some long term data on the parasites of rabbits and grouse to suppose that mean parasite intensities increase with climate warming in nematode species with no strong acquired immune response but had little effect on species with an acquired response. We examined evidence on how increased temperature increases the development rate of free-living stages and showed that the development rate of *Heterakis gallinarum* eggs increased with variability in temperature. We also examined the dynamics of the red grouse–*T. tenuis* system and showed that if climate change reduced arrestment of larval stages this would increase the basic reproduction number R_0 of the parasite and would increase instability and cycle period of the system leading to temporal disease outbreaks. An increased frequency of extreme climatic events would also synchronize populations across large scales leading to spatially synchronized outbreaks of infection. Thus, if these findings are indeed representative, we can postulate that increased global warming could result in synchronized large scale outbreaks of parasitic infections, particularly in species where parasite mortality rate in the host is not reduced through acquired immunity.

Data used in the present study were not collected specifically to examine long term changes in parasite intensity. The most common data available are in the form of long term counts of parasites in adult hosts. We postulated that the response of the parasite to climate warming would depend on the pattern of parasite mortality in the host as reflected by the shape of the age-intensity curve. In systems with little parasite mortality or mortality not associated with acquired immunity we should observe a consistent rise in intensity with age and a predicted rise in the mean intensity of infection with warming. Preliminary analysis for *Graphidum strigosum*, a rabbit stomach worm, confirmed a long term rise in mean levels associated with a rise in temperature and some further data from *T. tenuis* in grouse supported this. This contrasted with observations for *T. retortaeformis*, a parasite that stimulates a strong acquired immune response in systems with the development of acquired immunity, parasite mortality is initially low and so the age intensity curve rises, but with the accumulated exposure, the mortality rate of worms will increase so the age intensity curve will peak when infection and mortality are equal and then fall as the mortality rate becomes larger than the infection. If climate change leads to an increase in transmission this will result in the peak abundance being larger and in younger individuals while the mean levels in adults will not rise and may indeed fall. These findings are supportive of our general explanation of how climate disruption will influence parasite abundance but now require a more rigorous analysis that would examine changes in the shape of age-intensity curves for different species with respect to year to year variations in the length of the transmission season and the relative abundance of the host.

The finding that increased variation in climatic conditions can accelerate development leads us to suppose that care needs to be taken when applying day-degree models since the rate of parasite development in some species can increase non-linearly with temperature. Clearly, this is just a preliminary finding and since *H. gallinarum* is an egg-transmitted nematode this may not be observed in species where the infective stage is a larva. We need to examine in more detail if stochastic variation in temperature can accelerate development faster than small scale variations and then identify the mechanism involved. Interestingly, in their detailed studies of *Haemonchus contortus*, Crofton *et al.* (1965) showed there was geographical variation in the response of parasites to variation in temperature so there appears to be selection for a variable response between populations.

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General theory shows us that those parasites which have a greater impact on host fecundity than host mortality will tend to destabilize the parasite–host system leading to fluctuations in abundances, although the final outcome will be a tension with a series of features of the system including the pattern of parasite distribution within the host, time delays in development rate and seasonal forcing of infection (May & Anderson, 1978; Dobson & Hudson, 1992, 1994). In general, models of parasite–host systems indicate that nematode–host systems are often unstable and this instability increases R_0, with a greater infection rate (β), reduced arrested development and a reduction in the susceptibility of hosts to infection (more aggregated). In this respect we postulate that increased R_0 will increase the instability of nematode systems and lead to increased severity of outbreaks. With increased variation in climatic conditions and, in particular, extreme events we can expect that large scale climatic events will synchronize transmission across populations and lead to more large scale outbreaks. Of course, such patterns are preliminary but warrant further research.

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