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REVIEW

Wildlife diseases: from individuals to ecosystems

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Summary

1. We review our ecological understanding of wildlife infectious diseases from the individual host to the ecosystem scale, highlighting where conceptual thinking lacks verification, discussing difficulties and challenges, and offering potential future research directions.
2. New molecular approaches hold potential to increase our understanding of parasite interactions within hosts. Also, advances in our knowledge of immune systems makes immunological parameters viable measures of parasite exposure, and useful tools for improving our understanding of causal mechanisms.
3. Studies of transmission dynamics have revealed the importance of heterogeneity in host behaviour and physiology, and of contact processes operating at different spatial and temporal scales. An important future challenge is to determine the key transmission mechanisms maintaining the persistence of different types of diseases in the wild.
4. Regulation of host populations is too complex to consider parasite effects in isolation from other factors. One solution is to seek a unified understanding of the conditions under which (and the ecological rules determining when) population scale impacts of parasites can occur.
5. Good evidence now shows that both direct effects of parasites, and trait mediated indirect effects, frequently mediate the success of invasive species and their impacts on recipient communities. A wider exploration of these effects is now needed.
6. At the ecosystem scale, research is needed to characterize the circumstances and conditions under which both fluxes in parasite biomass, and trait mediated effects, are significant in ecosystem processes, and to demonstrate that parasites do indeed increase 'ecosystem health'.
7. There is a general need for more empirical testing of predictions and subsequent development of theory in the classic research cycle. Experimental field studies, meta-analyses, the collection and analysis of long-term data sets, and data constrained modelling, will all be key to advancing our understanding.
8. Finally, we are only now beginning to understand the importance of cross-scale interactions associated with parasitism. Such interactions may offer key insights into bigger picture questions such as when and how different regulatory factors are important, when disease can cause species extinctions, and what characteristics are indicative of functionally resilient ecosystems.

Key-words: apparent competition, co-infection, contact network, ecosystem health, host regulation, immunodynamics, parasite loss, spillback, spillover, tradeoffs, trait mediated indirect effects, transmission

Introduction

A considerable amount of research into the ecology of wildlife infectious diseases in recent decades has greatly

expanded our understanding of the roles they play in the natural world. Scientific insights have been made at all scales of the classical biological hierarchy; from individuals to ecosystems. At the level of individual hosts, there is now good evidence demonstrating how both macroparasites (those where reproduction usually occurs via the transmission of free-living stages, including the parasitic helminths

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and arthropods) and microparasites (those that tend to have rapid reproduction within a host, including the bacteria, viruses, protozoa and fungi) significantly influence birth and death rates (Hudson *et al.* 2002), even when infections induce no obvious clinical signs (Telfer *et al.* 2002; Burthe *et al.* 2008). However, although different parasites often infect and even co-exist within the same host (Petney & Andrews 1998), the effects of multiple and concomitant infections are only beginning to be understood (Cox 2001; Pedersen & Fenton 2007).

At the level of host populations, it has only been in more recent years that a broad characterization of the influences of parasites and disease on population and community dynamics has been realized, even though theoretical support for such effects has been established for some time (Anderson & May 1979; May & Anderson 1979; Holt & Lawton 1994). It is now also clear that the effects of parasitism are likely to extend beyond their host populations. Theoretical and empirical studies demonstrate that parasites can change the outcome of interspecific interactions including competition and predation, and can thus play keystone roles in determining species coexistence and biodiversity (Holt & Dobson 2006; Hatcher, Dick & Dunn 2006). In addition, examples of parasite effects on their hosts influencing entire ecosystems are now accumulating (e.g. Thomas *et al.* 1998; Wood *et al.* 2007).

Close associations between host and parasite dynamics are inevitable because of the functional dependency of parasites on their hosts. Mechanisms operating at a range of biological scales may influence these interactions, and the interactions themselves may have multi-scale effects on ecological processes. Thus, the critical parasite fitness parameter, transmission potential, may be influenced by processes occurring within individual hosts and by factors operating at host population and community scales that influence between-host transmission. Similarly, from the host's perspective, infection may affect life history strategies, regulate populations or determine the community composition. It therefore seems likely that parasite-mediated mechanisms may be responsible for positive and negative dynamical feedbacks at and between multiple scales in ecosystems. Indeed, recent insights from a variety of different scales are now feeding back to refine our basic understanding of such multi-scale dynamical connections in disease ecology. For example, the influence of animal behaviour and social structure on disease transmission between individuals is increasingly recognized, as are the non-independence of infectious agents within a host's 'parasite community', and the context dependence of parasite regulation of hosts at the population scale.

Here we review recent developments in our ecological understanding of wildlife infectious diseases from the individual host to the ecosystem scale. A host focus is taken to facilitate a multi-scale view (with 'parasite' being used as a blanket term for infectious disease causing agents), and we use studies from natural populations where possible. Our aims are to highlight where conceptual thinking is currently

lacking verification, discuss challenges to further progress, and offer potential future research directions that will fundamentally advance our scientific understanding of disease ecology.

Within host effects and processes

INFECTION AND HOST FITNESS

Experimental demonstration of the costs of immune responses aimed at fighting infections clearly indicate that trade-offs are likely to exist between immune defence and other physiological processes such as reproduction (Martin *et al.* 2004; Hanssen 2006). From the host's perspective the optimal allocation of resources will depend on the long-term implications of infection. Theory predicts that for self-limiting infections the optimal strategy may be to invest in immune defence and maximize survival probability whilst, for chronic infections, fitness may be maximized by investment in current reproduction (Perrin, Christe & Richner 1996), the so-called terminal investment strategy (Clutton-Brock 1994). Studies of natural populations have provided evidence for both of these mechanisms. Bank voles *Myodes glareolus* and wood mice *Apodemus sylvaticus* infected with cowpox virus, an acute infection, are more likely than uninfected animals to delay breeding until the following year (Telfer *et al.* 2005a), possibly indicating that resources are preferentially allocated to immune defence. In contrast, deer mice *Peromyscus maniculatus* infected with the trematode *Schistosomatium douthitti*, causing chronic disease, may give priority to current reproduction as their litters are of heavier mass than uninfected animals (Schwanz 2008). Experimental studies that trigger immune responses using vaccines or antigens also show evidence of terminal investment (Hanssen 2006; Bonneaud *et al.* 2004) and demonstrate inter-individual variation in optimal strategies. For example, whilst immune challenge of male blue-footed boobies *Sula nebouxii* caused reductions in the numbers of hatchlings and fledglings in mature individuals, senescent birds showed increases in these same measures. These contrasts presumably reflect age-dependent differences in trade-offs (Velando, Drummond & Torres 2006).

The impact of an infection on host fitness may also depend on factors influencing condition. Survival of African armyworms *Spodoptera exempta* infected with the bacterium *Bacillus subtilis* increased when they were fed diets with higher protein to carbohydrate ratios (Povey *et al.* 2009). Reductions in host condition may also be caused by parasites. Thus, the direct and immune-associated costs of infection may influence host vulnerability to other infections, triggering a vicious cycle of infection and reduced condition, with implications for individual fitness and parasite transmission potential and, ultimately, host-parasite dynamics at the population level (Beldomenico & Begon 2010). One consequence of such a cycle is likely to be increased probability of simultaneous co-infection with more than one parasite species.

WITHIN-HOST PARASITE COMMUNITIES

Whilst there are an increasing number of co-infection studies in laboratory models and human medicine (often exploring pathological consequences for the patient), co-infection studies in natural populations are still at an early stage. Most studies have focussed on impacts on the parasite, primarily using helminth infection intensity data, and demonstrating both positive and negative effects (Lello *et al.* 2004; Behnke 2008; Behnke *et al.* 2009). Such associations may be mediated by direct competition for resources or attachment sites, or indirect competition via host immune responses (see reviews in Cox 2001; Graham *et al.* 2007; Pedersen & Fenton 2007). However, as most co-infection studies are observational and cross-sectional (infection data from a single time point per host), there remains debate about the extent to which parasite interactions structure parasite communities or whether confounding variables may explain some patterns (Behnke *et al.* 2005; Poulin 2007).

One key drawback of cross-sectional data is that the timing of establishment for different parasites is unknown, limiting our ability to determine the directionality of any interactions and which elements of the host–parasite interaction are affected. Moreover, infection order can be crucial in determining the outcome of both facilitative and competitive inter-specific interactions (Ishii *et al.* 2002; Thomas, Watson & Valverde-Garcia 2003; Jackson *et al.* 2006). In a study of two fish species, Karvonen, Seppala & Valtonen (2009) demonstrated that positive associations between trematode parasites could be diminished by prior exposure to one of the parasites. However, longitudinal data on individual hosts offer considerable potential to surmount these issues. For example, a study of the length of *Bartonella taylorii* infections in individually tagged field voles *Microtus agrestis* demonstrated that co-infection with *Babesia microti* decreased infection length, whilst infection with cowpox virus during the early phase (but not the late phase) of the *B. taylorii* infection increased infection length (Telfer *et al.* 2008). Factors other than infection sequence may pose further challenges for interpreting data from natural populations: intrinsic and extrinsic factors such as genetic variability in host and parasite populations (Jager & Schjorring 2006; Jackson *et al.* 2006; Thomas, Watson & Valverde-Garcia 2003) and host food type (Hodgson *et al.* 2004) can also be critical in determining outcomes.

The last decade has seen an increasing emphasis on using a community ecology framework to understand the relative importance of top down (immune mediated) and bottom up (resource driven) mechanisms in driving interactions between co-infecting parasites, and to improve a priori predictions of likely interactions (Pedersen & Fenton 2007; Lello & Huxsell 2008; Graham 2008). These studies advocate consideration of the shared niche of different parasite species – defined based on the resources used, the location within the host and the immune response stimulated – and use examples from human medicine. This highlights the challenge of using this approach for disease ecology studies in natural populations:

the understanding of interactions between parasite species and the immune system of wild host species is often rudimentary at best. However, for interactions between microparasites and macroparasites infecting mammalian vertebrates, at least, the approach is likely to be informative due to well-known trade-offs between the T-helper cell type 1 (Th₁; typically induced by microparasites) and the T-helper cell type 2 (Th₂; associated with macroparasites such as worms) branches of the immune system. This has already been demonstrated by attempts to understand disparate results obtained in laboratory studies. Using a meta-analysis of 54 studies, Graham (2008) found that for red-blood cell dependent microparasites, co-infection with a helminth species that causes anaemia results in decreased microparasite density (bottom-up control), whilst a host's ability to control microparasite infections was impaired by helminths that suppressed the cytokine interferon (IFN)- γ (a cytokine typically produced by Th₁ cells). Interestingly, this facilitative immune-mediated interaction (i.e. release from top down control) was only apparent when there were no negative resource-mediated interactions. This indicates that, whilst there has perhaps been an emphasis on immune-mediated interactions (e.g. Lello & Huxsell 2008), we should not underestimate the potential of resource competition in driving observed patterns.

CO-INFECTION AND HOST FITNESS

Costs associated with trying to fight two simultaneous infections may result in pathological effects greater than the additive effects of each parasite in single infections. For a given level of malaria parasitemia, mice co-infected with the helminth *Litomosoides sigmodontis* showed greater reductions in body-mass and red blood cell density than mice only infected with malaria (Graham *et al.* 2005). Similarly, in a study of buffalo *Syncerus caffer*, Jolles *et al.* (2008) found that whilst infection with gastrointestinal worms was associated with poor body condition in hosts infected with bovine tuberculosis (Tb), there was no such effect in animals without Tb. Other data collected as part of the study are consistent with the hypothesis that co-infected individuals are unable to successfully control both infections due to trade-offs between Th₁ and Th₂ responses.

EFFECTS OF PARASITE INTERACTIONS AT THE HOST POPULATION SCALE

For competitive interactions, immune action against one parasite species may wholly or partially remove hosts from the population available to a second parasite (Telfer *et al.* 2007). Consequently, competition may drive temporal or spatial patterns of infection. A study that modelled immune mediated interactions within hosts found that, even for parasites with seasonal patterns that were out of phase, immune memory could potentially maintain the effects of interactions, causing temporal shifts in infection patterns (Lello *et al.* 2008). The predictions were compatible with population-level

data on *Graphidium strigosum* abundance in rabbits: peak abundance was 2 weeks later in rabbits co-infected with *Trichostrongylus retortaeformis* than in the uninfected rabbit subpopulation (*T. retortaeformis* is thought to reduce *G. strigosum* infection intensity within individuals via cross-immunity; Lello *et al.* 2004).

FUTURE DIRECTIONS

Our understanding of within-host processes in natural populations is currently hampered by a lack of appropriately detailed data. Whilst longitudinal studies can help, the potential to examine interaction networks will always be limited in studies that focus on a small group of parasites. However, recent advances in immunological and molecular approaches hold potential. Metagenomic techniques can be used to explore whole communities of pathogens. For example, using pyrosequencing of 16S rRNA sequences, Jones, Knight & Martin (2010) studied the bacterial community of 230 fleas, revealing non-random assemblages and negative interactions between dominant community members. As with studies targeted at specific parasite species, there was temporal and spatial variation in communities. However, this study also emphasized that a major challenge of such an approach will be in data analysis and interpretation, detecting 9986 unique phylotypes of which > 99% had average relative abundances < 0.1%. Other techniques that rely on amplification of target sequences by PCR, followed by one of several genetic profiling methods [e.g. denaturing gradient gel electrophoresis (DGGE); Nocker, Burr & Camper 2007] are less costly and less data intensive. In all such studies though, accounting for confounders and elucidating mechanisms of interaction for unknown species will be difficult.

An alternative host-orientated approach is to use immunological assays, either as a measure of exposure to specific types of pathogen or to improve understanding of causal mechanisms (Bradley & Jackson 2008). Although crude global measures of immunological investment have proved useful in some cases (e.g. high intensity trypanosome infections were more likely in animals with previously low lymphocyte counts; Beldomenico *et al.* 2009), they may not reflect important differences in immunological phenotype due to within host trade-offs. However, advances in our knowledge of immune systems make more sophisticated approaches involving cytokine and transcription factors now tractable, at least for species such as rodents that are closely related to laboratory species. For example, an individual's commitment to different arms of the adaptive immune system may be assessed by measuring the production of different cytokines [e.g. (IFN)- γ for Th₁ responses and IL-4 for Th₂ responses] (Graham *et al.* 2007; Bradley & Jackson 2008). Similarly, for innate immunity, assessing responsiveness of receptors such as Toll-like receptors (TLR), which are important in triggering subsequent adaptive responses, may be informative. In wood mice *Apodemus sylvaticus*, infection intensities of the nematode *Heligmosomoides polygyrus* are negatively corre-

lated with innate TLR-mediated immune responsiveness (Jackson *et al.* 2009). Thus, immunomodulation resulting in reduced initiation of immune responses may explain the positive associations, observed in a number of data sets, between *H. polygyrus* infection and other helminth infections (Behnke *et al.* 2009). Such approaches hold great potential for co-infection studies and an improved understanding of within-host interactions in general.

The transmission of parasites between hosts

Studies of parasite transmission in animal populations have tended to adopt one of two different approaches (Fenton *et al.* 2002). There are those that focus primarily on the association between population level phenomena, such as host density (reviewed in McCallum, Barlow & Hone 2001) or seasonal migration rates (reviewed in Altizer *et al.* 2006), and the rate of disease transmission throughout the population. One major benefit of this approach is that the population level patterns revealed can be indicative of the individual-level phenomena generating them. Such patterns can also be compared to the predictions and assumptions made in simple epidemiological models (e.g. Dwyer *et al.* 2000). However, this approach generally has limitations in indicating which mechanisms are operating at an individual level to result in the observed patterns at the population level (Fenton *et al.* 2002). The other general approach is to study how individual level properties, such as the probability that individuals make potentially transmissible contacts (e.g. Perkins *et al.* 2009) or degree of susceptibility or infectiousness (e.g. Galvani & May 2005), combine to affect the infection dynamics at the population level. Such studies provide good information on the importance of different factors to the transmission dynamics of particular parasites, but are usually technically more difficult to carry out and generally require a larger investment of time and money.

POPULATION SCALE APPROACHES TO UNDERSTANDING PARASITE TRANSMISSION

The mathematical representations of transmission commonly utilized in theoretical models make very simple assumptions about the relationship between the abundance of a parasite or its host in a given environment and the parasite transmission rate (reviewed in McCallum, Barlow & Hone 2001). A number of experimental studies have investigated whether such assumptions are valid in real host-parasite systems (reviewed in Fenton *et al.* 2002; Hudson *et al.* 2002). These have generally been successful in identifying details of the transmission process that do not conform to the simple assumptions made in theoretical models. For example, Knell, Begon & Thompson (1996) found, for the bacterial pathogen *Bacillus thuringiensis* infecting the Indian meal moth *Plodia interpunctella* in laboratory experiments, that the per capita transmission rate increased with host density for certain life history stages. Their experimental results implied that these changes were caused by changes in host

behaviour, particularly the rate of cannibalism of infected cadavers (Knell 1998a,b). A number of studies have also investigated factors influencing the transmission dynamics of baculoviruses infecting various Lepidoptera taxa (Dwyer & Hails 2002). These also strongly implied that changes in host behaviour in relation to life history stage and population density strongly influence the probability of hosts becoming infected (e.g. Dwyer 1991; Goulson *et al.* 1995). Fenton *et al.* (2002) conducted a meta-analysis (across experimental studies) of the relationship between host and pathogen abundance and the rate of transmission of infectious diseases. They showed that transmission rates were consistently a saturating function of parasite density, hypothesized as being due to host exposure to parasites becoming saturated at high density. However they also found that the relationship between transmission rate and host density could be either positive or negative, which they believed was associated with differences in the effects of density on host social interactions important to infection.

Fewer studies have been conducted that investigate the relationship between host abundance and disease transmission rates under non-experimental conditions (Davis, Calvet & Leirs 2005). The longitudinal studies of cowpox virus in field vole *Microtus agrestis* populations revealed clear relationships between host abundance and the rate of change of the parasite (Begon *et al.* 2009a). The existence of replicated high resolution data for this system allowed Smith *et al.* (2009a) to infer a saturating relationship between host abundance and the rates of transmission of cowpox virus in the population and, moreover, find strong support for seasonal variation in both the transmission rate and the degree of density dependence in the relationship between host abundance and infection rate. Studies such as this highlight that the population scale can be very effective for indicating the factors likely to be important in determining transmission rates in particular systems; however they can only imply how mechanisms operating at the individual level result in the observed associations.

MECHANISTIC APPROACHES TO UNDERSTANDING PARASITE TRANSMISSION

Whether an individual will become infected is a function of both the probability that a potentially transmissible contact has been made with an infectious agent and the probability that such a contact will lead to infection (Begon *et al.* 2002). Within these two categories are a number of inter-related factors and clearly a full mechanistic understanding of the transmission process in any population is likely to take significant research effort. However, progress in our understanding of host-parasite dynamics in human and livestock populations has highlighted a few key mechanisms that are likely to also be important in determining transmission rates in wildlife populations.

Heterogeneous distributions of both contact rates and infectious potential amongst individuals can have important consequences for the rate of disease spread through human

and livestock populations (Woolhouse *et al.* 1997; Lloyd-Smith *et al.* 2005; Kao *et al.* 2007). This is particularly thought to be true of sexually transmitted diseases (STDs). In wildlife, a number of studies have looked at the sexual transmission of mites between their coleopteran hosts in relation to host reproductive activity [see Knell & Webberley (2004) for a review of STDs in insects]. High promiscuity in beetle breeding, with limited variation in individual contact rates (Webberley *et al.* 2006), can lead to infection levels becoming very high (Knell & Webberley 2004), and imply that skewed distributions of infectious potential may not occur in such systems. However, Nahrung & Clarke (2007) found evidence for different infection rates between males and females in populations of the eucalyptus leaf beetle *Chrysophtharta cloelia* and suggested that skewed mating patterns were responsible. Identifying breeding systems that lead to skewed sexual contact rates would thus be a valuable area for future research of STDs in wildlife (Knell & Webberley 2004).

For non-STDs, recent studies have investigated in detail the relationships between individual contact dynamics and the transmission of infectious diseases (Table 1). Some of these studies have made findings analogous to those for human diseases: that distributions of contacts within populations can be highly heterogeneous, and that highly connected individuals can be responsible for the majority of transmission. For example, Clay *et al.* (2009) found that a large majority (80%) of the contacts by individuals of the deer mouse *Peromyscus maniculatus* transmitting Sin Nombre virus (SNV) are made by a minority (20%) of the population (the '20/80 rule'; Woolhouse *et al.* 1997, 2005), and that the combination of the number and duration of contacts per individual is positively associated with the probability of that individual becoming infected. In contrast, however, Hamede *et al.* (2009) found that contact rates of Tasmanian devils *Sarcophilus harrisii* were more evenly distributed among individuals. This implies that targeting highly connected individuals to control the spread of devil facial tumour disease (DFTD; a transmissible cancer) would not be successful. One of the challenges in interpreting data on contact dynamics, as highlighted by this study, is distinguishing the contacts that are potentially important to transmission from those that are not (Perkins *et al.* 2009). For example, Drewe (2010) found that it was the particular type (e.g. grooming versus aggression) and direction of the interaction between infected and uninfected individual meerkats *Suricata suricatta* that could be used to predict whether Tb transmission occurred. In addition, the derived structure of a contact network can differ significantly depending on the method used to detect contacts (Perkins *et al.* 2009).

Heterogeneities in transmission potential can also arise because of differences in both susceptibility to infection, and the infectiousness of infected individuals. The causes and consequences of such heterogeneities have already been discussed in detail elsewhere (Hudson *et al.* 2002). In addition, as noted above, infections of different parasite taxa can

Table 1. Summary of recent studies relating host contact network structure and dynamics to parasite transmission dynamics

Host	Parasite	Behaviour	Field method	Insights into transmission	Ref.
<i>Peromyscus maniculatus</i>	Sin Nombre virus	Contact rates	Powder marking, PIT tags and antennae	Larger body size associated with higher transmission probability	1
<i>Meles meles</i>	<i>Mycobacterium bovis</i>	Inter-group movement	Trapping and bait marking	Positive association between inter-group movement and transmission	2
<i>Trichosurus vulpecula</i>	<i>M. bovis</i>	Contact rates	Radio telemetry	Contact rates related to population density	3
<i>Trichosurus vulpecula</i>	<i>M. bovis</i>	Contact rates	Proximity data loggers	Transmission may not be just via direct contact	4
<i>Syncerus caffer</i>	<i>M. bovis</i>	Frequency in the same herd	Radio-tracking	Disease spreads slower than mean field predictions	5
<i>Trichosurus vulpecula</i>	<i>M. bovis</i>	Direct and indirect contact	Capture–mark–recapture	Effects of contact heterogeneity	6
<i>Sarcophilus harrisi</i>	Devil facial tumour disease	Contact rates	Proximity sensing radio collars	Very little contact structure; well mixed	7
<i>Meles meles</i>	<i>M. bovis</i>	Contact rates	Proximity data loggers	Highly skewed contact structure	8
<i>Orcinus orca</i>	Hypothetical, directly transmitted	Social interactions	Visual observation	Contact structure made population more vulnerable to disease outbreaks	9
<i>Panthera leo</i>	Canine distemper virus	Contact network	Data on behaviour and movement	Network can sustain endemic infection provided multiple host species involved	10
<i>Suricata suricatta</i>	<i>M. bovis</i>	Intra and inter-group contacts	Visual observation	Most socially active not at highest risk of becoming infected	11
<i>Apis mellifera</i>	<i>Paenibacillus larvae</i>	Promiscuity	Direct manipulation	Number of matings per individual positively related to disease resistance	12
<i>Zootermopsis angusticollis</i>	<i>Metarhizium anisopliae</i>	Degree of inbreeding or outbreeding	Direct manipulation	Increased outbreeding led to decreased disease susceptibility	13
<i>Apodemus flavicollis</i>	Hypothetical disease	Frequency and duration of contacts	Capture–mark–recapture and radio-tracking	Network differs depending on method used to infer contacts; seasonally varying network structure	14
<i>Trichosurus vulpecula</i>	<i>M. bovis</i>	Den sharing	Observation of den choice and experimental manipulation of the possum : den ratio	More connected individuals more likely to become infected	15
<i>Egernia stokesii</i>	Three blood parasites and an ectoparasitic tick	Spatial proximity	Capture–mark–recapture	Either association between connectance and infection probability and/or ticks alter host behaviour	16
<i>Litoria nannotis</i> , <i>L. genimaculata</i> and <i>L. lesueuri</i>	<i>Batrachochytrium dendrobatidis</i>	Contact rates	Radio telemetry and harmonic direction finding	Plausibly related to species specific contact rates	17

References are 1. Clay *et al.* (2009); 2. Vicente *et al.* (2007); 3. Ramsey *et al.* (2002); 4. Ji, White & Clout (2005); 5. Cross *et al.* (2004); 6. Porphyre *et al.* (2008); 7. Hamede *et al.* (2009); 8. Böhm, Hutchings & White (2009); 9. Guimarães *et al.* (2007); 10. Craft *et al.* (2009); 11. Drewe (2010); 12. Seeley & Tarpay (2007); 13. Calleri *et al.* (2006); 14. Perkins *et al.* (2009); 15. Corner, Pfeiffer & Morris (2003); 16. Godfrey *et al.* (2009); 17. Rowley & Alford (2007).

interact to make hosts more or less susceptible to disease, highlighting the possibility that the probability of transmission can be affected by the immune status and infection status of the host (e.g. [Jolles *et al.* 2008](#); [Beldomenico *et al.* 2009](#)).

RECENT ADVANCES

Studies are now also using contact networks to explore the transmission dynamics of diseases among wildlife species, partly reflecting the recent broad adoption of the 'network

paradigm' for understanding complex dynamical systems in general (Newman 2002; May 2006). For example, Craft *et al.* (2008, 2009) provide compelling evidence that outbreaks of canine distemper virus in African lion *Panthera leo* populations are largely a result of inter-specific interactions (among lions, spotted hyenas *Crocuta crocuta* and jackals *Canis spp.*), and not simply due to interactions between lions. Similarly, the persistence of rabies in the Serengeti ecosystem is believed to be strongly influenced by interactions between domestic dogs and the other wild carnivore populations (Lembo *et al.* 2008). Studies are also beginning to reveal the extent to which the persistence of certain diseases in the wild depends on localized transmission events within individual host populations, and the extent to which it relies upon, or is influenced by, longer range transmission events (Girard *et al.* 2004; Davis *et al.* 2008; Snäll *et al.* 2008). This was highlighted recently in the study of the dramatic outbreaks of plague *Yersinia pestis* in populations of great gerbils *Rhombomys opimus* in Kazakhstan (Davis *et al.* 2008). Here, a contact network model showed that long range transmission events led to the widespread outbreak of the disease only when a sufficient number of burrows were occupied to sustain transmission across the landscape.

The construction of detailed contact networks requires a considerable research investment. There is thus still a need for more simplistic representations of the transmission process that incorporate sensible representations of what are believed to be important mechanisms influencing transmission. For example, Hosseini, Dhondt & Dobson (2004) recognized strong seasonal aggregation in the flock sizes of the house finch *Carpodacus mexicanus* from empirical data, and incorporated that into the transmission function of a simple susceptible-infected-recovered disease model for house finch conjunctivitis *Mycoplasma gallisepticum*. Another approach is to use Bayesian model fitting methods to infer the most likely transmission dynamics from empirical data (e.g. Snäll *et al.* 2008; Smith *et al.* 2009a).

FUTURE DIRECTIONS

The acquisition of sufficient data has led to the transmission process now being understood in terms of the relative strengths of, and interactions between, various biotic and abiotic mechanisms (Table 1). However, while studies of human and livestock populations have provided important insights into plausible determinants of disease dynamics in wild populations, their application is potentially limited because most wild animal populations are more variable in time and space. A key challenge for future research will be to identify the most important mechanisms determining the dynamics of parasites, and then to hopefully identify useful generalities across different systems. The benefits of combining both the population scale and the mechanistic approaches to advance our understanding of transmission are exemplified by past research into the transmission of baculoviruses in the larvae of various species of Lepidoptera, where studies have indicated the roles of host behaviour, the spatial distri-

bution of the virus or infected hosts, host susceptibility, host dispersal and even potentially unrecognized transmission mechanisms in the transmission of the virus (see Cotter *et al.* 2004; D'Amico *et al.* 2005; Parker, Elder & Dwyer 2010; and references therein).

The surge of progress in characterizing contact network dynamics in real populations raises the possibility of identifying generalities in the effects of animal social dynamics on the transmission dynamics of different diseases. The ongoing development of remote sensing devices (Guilford *et al.* 2008; Handcock *et al.* 2009) is making it increasingly feasible to acquire data on details relevant to the transmission process. Partly as a result of this, the increasing availability of high resolution data on both host and parasite dynamics will obviously allow more insights to be made into the details of the transmission process. However, such detailed studies will never be conducted for the majority of host/parasite systems. Therefore, a potentially valuable future research direction is to investigate how much data is needed to infer particular details about the transmission process, through the analysis of existing data for well-studied systems. Such 'retrospective' studies could also facilitate research and development into statistical methods to infer information, such as behavioural dynamics (Guilford *et al.* 2009) and even contact network structure (Clauset, Moore & Newman 2008), based on differing types and amounts of data. Whilst such studies are unlikely to reveal new insights into transmission mechanisms, they will provide practical guidance on the amount of research effort required to improve our knowledge about key details of the transmission process.

Further research is also required to improve our understanding of the relative roles of intra- and inter-host population transmission in determining host-pathogen dynamics and disease persistence (Hudson *et al.* 2002). This can be separated into understanding the relative roles of transmission processes that operate at different spatial scales (e.g. local social contact versus rare long distance migration) and further understanding the roles of cross-species transmission in multi-host systems. Not only will such research help explain the persistence of diseases in wild populations but also it is likely to be highly informative for controlling diseases of detriment to humans (e.g. Stenseth *et al.* 2008). From a theoretical perspective, there are numerous formal relationships and analogues between metapopulation theory and theory of disease persistence in host populations (summarized in Hudson *et al.* 2002). Metapopulation theory has already been extended to represent the transmission process in diseases of humans (e.g. Rohani, Earn & Grenfell 1999; Keeling & Gilligan 2000; Xia, Björnstad & Grenfell 2004). Studies that reveal how generally applicable and useful such theory is to understanding the dynamics of diseases in wildlife would be a helpful contribution (e.g. Snäll *et al.* 2008). For example, what types of diseases actually rely on relatively rare transmission events between host populations to persist in the wild? The practical benefits of understanding the wider transmission network are highlighted by detailed studies of Tb

transmission in European badgers *Meles meles* (Tuytens *et al.* 2000; Vicente *et al.* 2007; Böhm, Hutchings & White 2009). Here, detailed characterization of the contact networks and the host social dynamics in perturbed and unperturbed populations has highlighted that culling badgers in a naive way to control the spread of the disease may actually lead to the increased transmission of Tb amongst badgers.

Population scale effects

Over the last decade there have been seen several high profile cases of emerging diseases significantly impacting native biodiversity (Smith, Acevedo-Whitehouse & Pedersen 2009b). One example is the ongoing epidemic of chytridiomycosis, which has been implicated in population declines and extinctions of frog species globally (Skerratt *et al.* 2007; Rohr *et al.* 2008a). Another is the facial tumour disease (DFTD) of Tasmanian devils, which is believed to have the potential to drive the species to extinction (McCallum *et al.* 2007; McCallum 2008). However, whether or not infectious diseases, and the parasitic agents which cause them, commonly regulate wild populations under normal circumstances is still open to debate. Regulation refers to the tendency of a population to decrease in size when it is above a particular level, but to increase when below that level. Thus any process that influences birth and death rates (and also immigration and emigration rates) in a density-dependent fashion has the potential to regulate a population (Turchin 2003). However, conclusively demonstrating regulation in the wild is hindered by the need for often logistically difficult population scale experiments that perturb the system being studied away from equilibrium to both (i) separate factors causing population impacts from those that are simply correlated or associated with impacts (Scott & Dobson 1989), and (ii) show that such factors are indeed causes of regulation and do not just act in a compensatory fashion (i.e. only affecting individuals that would have lowered fitness anyway due to the effects of other regulatory forces such as competition and predation; Holmes 1982).

PARASITE REGULATION OF HOST POPULATIONS

Although experimental perturbations have been carried out successfully with many captive host-parasite systems (see examples in Hudson *et al.* 2002), with the addition or removal of parasites clearly demonstrating regulation, it was not until the study of Hudson, Dobson & Newborn (1998) that the first demonstration that parasites can reduce the numerical abundance of wild hosts in a regulatory manner was provided. Their population-scale manipulation of infection levels of the gastrointestinal parasite *Trichostrongylus tenuis*, through anthelmintic treatment, dramatically reduced the extent of population crashes in their red grouse *Lagopus lagopus scoticus* host. Motivated by this study, subsequent research on other systems has investigated whether parasites can act as dominant regulatory forces in other wild host populations. Indeed, 10 years ago the field of wildlife disease

seemed poised to demonstrate a common dominant role of parasites in the natural regulation of vertebrate host populations (Hudson *et al.* 2002). However, little in the way of further critical support has been forthcoming. More often than not, long-term research programmes on wild populations have instead reinforced how regulation by parasitism and disease cannot be assumed based on individual-scale experiments and correlational population-scale data. For example, studies had initially suggested a role of the gastrointestinal parasite *Trichostrongylus retortaeformis* in the regulation of mountain hare *Lepus timidus* populations in the UK. The parasite induces reductions in host fecundity and there is also evidence of a negative relationship with host body condition (Newey & Thirgood 2004; Newey, Thirgood & Hudson 2004). However, recent modelling work based on long-term data indicates that such effects are unlikely to be sufficient to drive the demographic patterns observed in this system (Townsend *et al.* 2009). This highlights that without properly designed and interpreted population scale manipulations there remains significant uncertainty about whether potential regulatory effects of parasites are actually realized in the wild (Irvine 2006). This applies to well studied wild vertebrate populations such as Soay sheep *Ovis aries* and Svalbard reindeer *Rangifer tarandus platyrhynchus* where, as was the case in the mountain hare system, strongly suggestive evidence for potential regulatory roles of parasites has also been collated (Wilson *et al.* 2004; Albon *et al.* 2002).

For invertebrate hosts, captive experimental studies have clearly demonstrated the regulatory impact that parasites can have. For example, Sait, Begon & Thompson (1994) showed that the granulosis virus of the Indian meal moth *Plodia interpunctella* causes its host populations to cycle around significantly lower abundance levels (with a significantly longer cycle period) than uninfected populations. For wild invertebrate hosts, some of the best evidence for similarly regulatory effects of parasites comes from biocontrol releases, such as the release of a nuclear polyhedrosis virus several decades ago to control spruce sawfly *Gilpinia hercyniae* in Canada. In this case, the virus remains endemic, while the pest insect populations remain relatively low (Bonsall 2004). However, as for vertebrates, although disease impacts on individuals and correlations between infection burdens and population declines are frequently observed (e.g. Decaestecker *et al.* 2005), whether and under what conditions parasites and disease play a significant role in the natural regulation of insect host populations is not at all clear. Again, there are several long-term studies demonstrating the potential for such effects in natural systems. For example, in an analysis of a 15-year data set of the caddisfly *Brachycentrus americanus* and its microsporidian disease, Kohler & Hoiland (2001) present compelling observational evidence that the pathogen is largely responsible for driving observed *Brachycentrus* dynamics in North American coldwater streams. However, a definitive experimental demonstration of the regulation of insect populations by parasites and disease in natural systems is still lacking. As for vertebrate populations, when such manipulations are carried out the

reality is generally more complex, as recently exemplified by [Laws *et al.* \(2009\)](#). In that study, experimental manipulations of grasshopper populations in North American grassland indicated that the effects of predators and pathogens were highly inter-related.

PARASITES AND HOST POPULATION CYCLES

In addition to simply suppressing host numerical abundance in a density-dependent manner, theoretical models have also suggested that parasites and disease can cause oscillations in host abundance ([Anderson & May 1979](#)). Hence, parasites have also been postulated as a potential cause of many of the population cycles, both vertebrate and invertebrate, observed in the wild ([Bonsall 2004](#); [Irvine 2006](#)). However, as for simple numerical suppression effects, experimental confirmation for such a role of parasites in the dynamics of their wild populations has yet to be obtained, despite observational evidence and modelling studies supporting this hypothesis for many systems. For example, while red grouse in the UK is one of the systems in which parasites have also been hypothesized to drive observed population cycles, the parasite removal manipulation of Hudson, [Dobson & Newborn \(1998\)](#) did not remove the cycles but only lessened the crashes. This left the possibility that the parasites were simply acting to amplify the cycles caused by other factors ([Lambin *et al.* 1999](#)). Further investigation of this system has indicated that it is indeed likely that parasite effects interact with host intrinsic factors to cause the observed dynamics ([Redpath *et al.* 2006](#); [New *et al.* 2009](#)). Such considerations, in conjunction with observations that parasitic nematodes of Soay sheep likewise increase the depth of population crashes initiated by winter food shortage ([Wilson *et al.* 2004](#)), and their own analytical findings on the mountain hare system, led [Townsend *et al.* \(2009\)](#) to re-affirm that parasites in wild populations appear more likely to play a role in amplifying underlying fluctuations that are driven by other causes, rather than driving the fluctuations themselves.

Evidence from other systems lends strong support to this hypothesis. In a key recent experimental study, [Pedersen & Greives \(2008\)](#) provided a second clear demonstration of parasites decreasing the numerical abundance of their hosts in a regulatory manner; anthelmintic treatment of fluctuating deer mice *Peromyscus maniculatus* populations in North America (to remove intestinal nematodes) lessened the rate and magnitude of population crashes. However, by conducting their manipulation in a factorial design in which food supplementation was also provided, the authors demonstrated that the host population impact of parasites was limited to the crash phase of seasonal population booms driven by food resource pulses. Similarly, [Forrester & Finley \(2006\)](#) demonstrated that infection by the copepod parasite *Pharodes tortuensis* appears to alter the strength of pre-existing density dependence of bridled goby *Coryphopterus glaucofraenum*. Through the use of mark-recapture analysis, they showed that bridled goby survival generally declines with increasing density, with the decline being steeper for

gobies with access to few refuges than for gobies in neighbourhoods where refuges were common, and the negative effects of high density and refuge shortage being more severe for parasitized gobies than for gobies free of parasites. Finally, examining the link between disease and host population dynamics from another angle in the long-studied field vole *Microtus agrestis*/cowpox virus system in the UK, [Begon *et al.* \(2009b\)](#) demonstrated that the timing of disease epidemics is driven by seasonal host dynamics, and not vice-versa. Hence, current evidence appears to generally support the hypothesis that parasites play a secondary rather than a driving role in cyclical host population dynamics.

A NOVEL SYNTHESIS

The limited evidence for parasite regulation of their hosts under normal circumstances contrasts with decade's worth of evidence demonstrating the significant impacts that emerging diseases and biocontrol agents can have on their host populations (e.g. [Plowright 1982](#)). Taken together these suggest that true regulation by diseases under *natural* circumstances may be rare, only occurring under relatively unusual circumstances. The two experimental studies to date that have provided strong evidence for such regulatory effects in the wild may offer an insight into what those circumstances might be – both Hudson, [Dobson & Newborn \(1998\)](#) and [Pedersen & Greives \(2008\)](#) demonstrated the suppressed numerical abundance of wild hosts by parasites in systems where population fluctuations were most likely already being driven by other factors. This suggests that the potential for population scale impacts of parasites may only be actually realized in the wild when host populations are perturbed by other factors.

A dependence on disturbance or instability of some form may also provide a common link between the regulatory effects of parasites considered here, and population scale effects of parasites and disease linked to disease emergence (e.g. chytridiomycosis and DFTD), species invasion (see below), pollution (e.g. [Rohr *et al.* 2008b](#); [Kelly *et al.* 2010a,b](#)), habitat ([Patz *et al.* 2004](#)) and even host population bottlenecks ([Tompkins 2007](#)). Support for this hypothesis can be seen in studies that have looked to experimentally demonstrate apparent competition via shared parasites. Here, the force of parasite infection from a reservoir species is recognized as having the potential to regulate sympatric populations of susceptible species ([Hudson & Greenman 1998](#); [Tompkins, Draycott & Hudson 2000a](#); [Tompkins *et al.* 2000b](#)). However, field experiments tend to find evidence for such effects only in systems where disturbance leads to traditional and newly-acquired hosts of the same parasite occurring in sympatry (e.g. [van Nouhuys & Hanski 2000](#); [LoGiudice 2003](#)), and not in more established systems (e.g. [Pope *et al.* 2002](#); [Laurenson *et al.* 2003](#)).

FUTURE DIRECTIONS

Based on this re-assessment of the available evidence, indicating that regulation of host populations is too complex to con-

sider parasite effects in isolation from other factors such as disturbance and other interspecific interactions, we suggest that research effort needs to shift to exploring whether we can achieve a unified understanding of the conditions under which (and the ecological rules determining when) population scale impacts of parasites and disease can occur. As noted above, one potentially fruitful line of enquiry would be to explore the apparent link between population scale effects and disturbance, consideration of which could potentially facilitate a common ecological understanding not only of regulation but also the disease impacts of emerging diseases, species invasions, pollution/disease interactions, habitat disturbance and host population bottlenecks. If the hypothesized relationship between perturbation by other factors and population scale disease impact is upheld, there are multiple mechanisms that could potentially (and non-exclusively) be causing it, including both coevolutionary and numerical mechanisms among others. Investigation of such mechanisms and their relative importance (and possible interactions) may also provide a much needed framework for understanding observed patterns in the field that are potentially driven by multiple factors, including the correlation between increasing community diversity and decreasing disease risk (Keesing, Holt & Ostfeld 2006; Johnson & Thieltges 2010) and the greater perceived threat that parasites pose to shrinking ecosystems (McCallum & Dobson 1995; Holmes 1996).

Community scale effects

In recent years there has been burgeoning interest in the role of parasitism in the success and impacts of biological invasions (Prenter *et al.* 2004; Dunn 2009). Invasions dramati-

cally affect biodiversity and community structure (Mack *et al.* 2000; Pimentel *et al.* 2001); hence there is a need to understand the underlying mechanisms. Furthermore, invasions provide us with 'experiments' in which we can study the effect of parasitism on host population dynamics and on the outcome of interspecific interactions (Fig. 1). Invaders may be released from the regulatory effects of parasitism, or may act as reservoirs or as sink hosts, thereby mediating parasite impact on native hosts (Hudson *et al.* 2002; Torchin, Lafferty & Kuris 2002; Telfer *et al.* 2005b). Also, in addition to the direct impacts of parasitism, parasite modification of host behaviour and life history [trait mediated indirect effects (TMIEs); Werner & Peacor 2003] can be a powerful influence on competitive and trophic interactions within the community (Hatcher, Dick & Dunn 2006).

INVASIONS AND PARASITE LOSS

Invaders are often larger, and reach higher population densities, both in comparison with populations in their native habitat and in comparison with similar indigenous species in the new habitat (Torchin, Lafferty & Kuris 2002; Grosholz & Ruiz 2003). One hypothesis which may explain such success is their release from the regulatory pressures of natural enemies including parasites (Torchin, Lafferty & Kuris 2002; Torchin *et al.* 2003). A number of community studies provide evidence for lower parasite diversity in invaders compared to their native counterparts. For example, in Northern Ireland, communities containing the native amphipod *Gammarus duebeni celticus* have been invaded by three exotic amphipod species. The native species hosts a higher diversity of parasites; five parasite species have been detected, of which three are shared by the native and invasive species, but two are

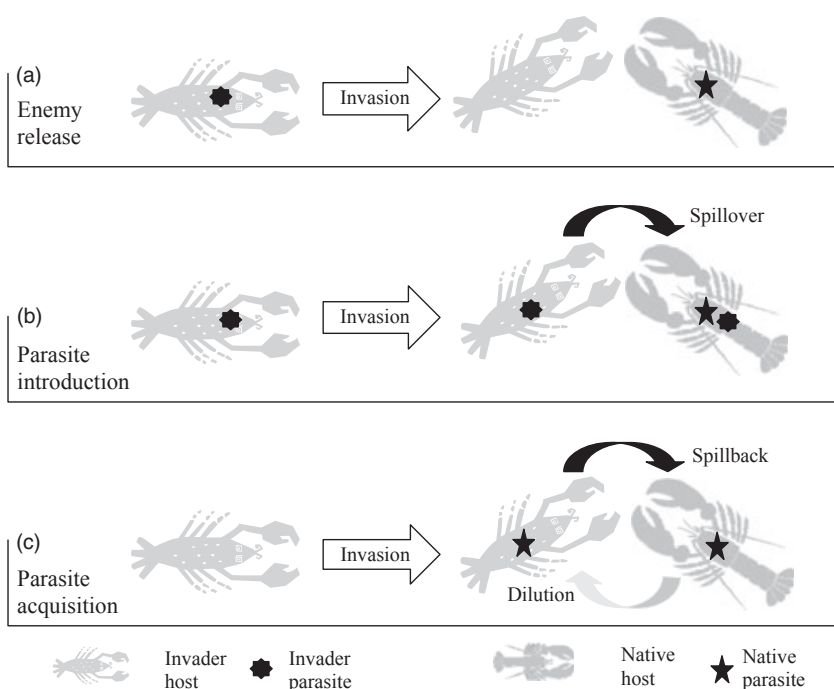


Fig. 1. The fate of parasites in the course of an invasion. Many invaders fail to establish. For invaders that succeed, the main outcomes for parasites are illustrated. (a) Enemy release; loss of parasites during the invasion process may benefit the invader. Parasites may be lost through the effects of sub-sampling or through selective pressures experienced during translocation or establishment. (b) Parasite introduction; parasites introduced with the invader may spillover to infect species in the invaded habitat. (c) Parasite acquisition; an invader may acquire parasites in the new habitat. If the invader is a less competent host, this may dilute the impact of the parasite (grey arrow). However, if the invader is a more competent host, spillover of parasites to native hosts may occur (black arrow).

restricted to the native host (Dunn & Dick 1998; MacNeil *et al.* 2003a,b). Torchin, Byers & Huspeni (2005) found a similar pattern in mudsnail communities in North America; whilst the native snail was host to 10 trematode species, the invader harboured only one. Biogeographical comparisons have also found reduced parasite diversity in an invader's range in comparison with their native range (Table 2). For example, Torchin *et al.* (2003) reviewed parasitism in the native and invasive ranges of 26 invertebrate and vertebrate species. For most species, parasite diversity was higher in their native range (average number of parasite species 16) than the invasive range (average seven species, of which three had invaded with the host, whilst four were acquired in the new habitat). Parasites may be lost as a result of sub-sampling of hosts from the native range, and through selective pressures encountered by parasites and their hosts during the translocation, establishment and invasion processes. These mechanisms were recently investigated for chewing lice on 36 species of bird introduced to New Zealand; MacLeod *et al.* (2010) found that few parasites were lost as a result of sampling effects, but that most parasites were lost because their hosts or the parasites themselves failed to establish. For hosts that became established, parasite establishment was positively associated with host body mass and the number of hosts introduced.

Although successful invaders may experience lower parasite diversity, however, it is interesting to note that those parasites infecting invaders in their new range often reach similar or higher prevalence's than in their native habitat (Torchin *et al.* 2003; Torchin, Byers & Huspeni 2005). Furthermore, Colautti *et al.* (2004) caution that many studies may over-estimate the extent of enemy release, because invading propagules originate from a subset of the native populations. For example, starlings *Sturnus vulgaris* have

lower parasite diversity in their invasive North American range than in the native European ranges (Colautti *et al.* 2005). However, a comparison of parasite diversity in the invasive and source (UK) populations found no enemy release; although 13/20 parasites had been lost from the source range, a further 17 species had been acquired in the new habitat (Colautti *et al.* 2005). Several other empirical studies that take into account the likely source population(s) for the invasion suggest that the role of enemy release may be less important than previously thought (Table 2). In addition, irrespective of whether parasite species loss upon invasion occurs or not, no studies have yet clearly demonstrated improved fitness as a result of such loss. Invasive species experience a range of effects, including population bottlenecks and the selection pressures imposed by a novel environment, and the potential for release from parasite regulation needs to be considered within this context.

INVASIONS AND PARASITE INTRODUCTION

Although some parasite species may be lost, invasive hosts may also introduce parasites into their new range, with potential disease transmission ('spillover') to native hosts. Even if the native species is not a competent host, the invasive species may act as a reservoir for disease transmission to indigenous host populations (Prenter *et al.* 2004). One well studied example is that of the squirrel pox virus which evidence indicates is mediating the displacement of the native red squirrel *Sciurus vulgaris* by the invasive American grey squirrel *S. carolinensis* in the UK. The virus causes high mortality and is therefore predicted to die out in red squirrel populations, but greys are asymptomatic and appear to act as a reservoir (Tompkins *et al.* 2002). Theoretical studies have concluded that, in many regions, the rate of replacement of

Table 2. Examples of studies comparing parasite diversity in the native versus the invasive range of the host

Study system	Pattern observed	Reference
Parasitism in the invasive vs. the native range		
Marine animals (meta-analysis of the literature)	Average parasite diversity higher in native populations	Torchin, Lafferty & Kuris (2002)
Range of species (meta-analysis of the literature)	Average parasite diversity higher in native populations	Torchin <i>et al.</i> (2003)
Tree frog <i>Eleutherodactylus coqui</i>	Parasite diversity higher in the native range	Marr, Mautz & Hara (2008)
Parasitism in invasive vs. source populations		
Helminth parasites of the starling <i>Sturnus vulgaris</i>	Parasite diversity higher in native range, but no evidence for enemy release once host population source taken into account	Colautti <i>et al.</i> (2005)
Microsporidian parasites of the amphipod <i>Dikerogammarus villosus</i>	Parasite diversity higher in the invaded range	Wattier <i>et al.</i> (2007)
Rabbit fish <i>Siganus rivulatus</i>	Parasite diversity higher in native range, but no evidence for enemy release once host population source taken into account	Pasternak, Diamant & Abelson (2007)
Microsporidian parasites of the amphipod <i>Crangonyx pseudogracilis</i>	Parasite diversity higher in native range, but no evidence for enemy release once the source of the invasion was taken into account	Slothouber Galbreath <i>et al.</i> (2010)
Chewing lice of birds introduced to New Zealand	Parasite diversity higher in the native range, even once likely host population sources taken into account	MacLeod <i>et al.</i> (2010)

the native species cannot be explained by competitive effects alone, but is mediated by the shared virus (Tompkins, White & Boots 2003). Field studies support these findings; the rate of invasions in England (where the virus is present) was up to 25 times higher than in virus free regions of Scotland and Italy (Rushton *et al.* 2006).

INVASIONS AND PARASITE ACQUISITION

As noted above, invaders frequently acquire parasites in their new range. The outcome of such acquisition depends upon the relative impact of the shared parasite on native and invading species. If the invader is a less competent host, parasite dilution may benefit the native species (Norman *et al.* 1999; Ostfeld & Keesing 2000). Conversely, the invader may act as a reservoir for the parasite, which could increase the impact of the parasite via 'spillback' to the native host population (Daszak, Cunningham & Hyatt 2000; Kelly *et al.* 2009). Although conclusive demonstrations for both of these mechanisms acting in wild populations are still lacking, evidence is mounting to support their occurrence. In the UK, the replacement of the white clawed crayfish *Austropotamobius pallipes* by the North American signal crayfish *Pacifastacus leniusculus* appears to be mediated by both spillover and spillback. Crayfish plague *Aphanomyces astaci* was introduced to the UK by the signal crayfish and has caused rapid extinction of native populations, which have been subsequently replaced by the invasive species (Holdich 2003). In addition, the invasive crayfish has acquired the native microsporidian parasite *Thelohania contejeani* (Dunn *et al.* 2009). *Thelohania contejeani* causes porcelain disease and eventual death in the native species, but infected signal crayfish were found to be asymptomatic. The invasive crayfish reaches much higher densities than its rival, and parasite prevalence is also high, suggesting that parasite spillback may be an important factor in species replacement. Evidence is also mounting for invasive species diluting the impact of parasitism on native hosts. For example, prevalence of *Bartonella* spp. (a flea-vectored bacterial infection) declined in wood mice *Apodemus sylvaticus* in Ireland as densities of the invading bank vole *Myodes glareolus* increased. The infection was detected in wood mice, but not in bank voles, suggesting that the less competent invader acted as a sink for the parasite (Telfer *et al.* 2005b).

TRAIT MEDIATED INDIRECT EFFECTS

Strong evidence indicates that parasites can also change the outcome of competitive and trophic interactions between species through their effects on host behaviour; there is growing interest in the impact of these trait mediated indirect effects (TMIEs) on the outcome of biological invasions (Hatcher, Dick & Dunn 2006; Dunn 2009). Through their impacts on host behaviour, parasites may modify the outcome of competitive and predatory interactions between native and invasive species. For example, the invasive mosquito *Aedes albopictus* is a stronger competitor than the

native North American mosquito *Ochlerotatus triseriatus*. However, the gregarine parasite *Ascogregarina taiwanensis* was found to reduce the competitive strength of the invader (Aliabadi & Juliano 2002). Parasite prevalence was low in the years following the introduction of *A. albopictus* to North America, and this enemy release appeared to increase the competitive strength of the invader during the initial phase of invasion (Juliano & Lounibos 2005).

Modifications of the impact of predation through changes in host behaviour are of particular interest for parasites that are trophically transmitted from the intermediate to the definitive host, as these parasites often increase their likelihood of transmission by manipulating the host's antipredator responses. For example, the invasive American brine shrimp *Artemia franciscana* has acquired cestode parasites in its new Mediterranean range. These parasites cause colour change and reversed phototaxis in the native but not the invasive species; hence, the native is likely to experience higher rates of predation (Georgiev *et al.* 2007), which may in turn facilitate its replacement by the invader. Similarly, a native acanthocephalan parasite that affects both native and invasive amphipods in France increases predation risk for the native but not the invasive species (Tain, Perrot-Minnot & Cezilly 2006).

Parasites can also modify the predatory strength of the host, and hence the impact of the host on prey populations. Dick *et al.* (2010) recently explored the effect of parasitism on the predatory functional response of the invasive amphipod *Gammarus pulex* and found that individuals infected with the acanthocephalan *Echinorhynchus truttae* consumed 30% more prey than did unparasitized individuals. Although fecundity was lower in infected females, parasite prevalence was low during the peak reproductive period. Thus the parasite has little effect on population growth, but a large per capita effect on predatory rate and hence may enhance rather than reduce the impact of this invader. *Gammarus pulex* decreases the diversity and richness of communities it invades, particularly through predation on other macro-invertebrates (Kelly *et al.* 2006). The increase in predatory functional response of parasitized hosts may therefore have ramifications throughout the community.

In invertebrate communities, invading species and their native counterparts often engage in intra-guild predation (predation amongst species that are also competitors; Polis, Myers & Holt 1989; Holt & Polis 1997). It has recently been demonstrated that parasitism can theoretically play a keystone role in intra-guild predation, increasing the conditions under which intra-guild predators may coexist and even leading to dominance of the inferior competitor and intra-guild predator (Hatcher, Dick & Dunn 2008). For example, competition and intra-guild predation appear to be key mechanisms underpinning the replacement of the native amphipod *Gammarus duebeni celticus* by the invasive *Gammarus pulex* in Northern Ireland (Dick 2008). However, intra-guild predation is modified by the acanthocephalan parasite *Echinorhynchus truttae* (MacNeil *et al.* 2003c; Hatcher, Dick & Dunn

2008). Field and laboratory manipulations revealed that *E. truttae* did not directly affect the survival of the native or the invader in single species populations. However, the parasite reduced predation by the invader on the native *G. d. celticus*, thereby enhancing coexistence.

FUTURE DIRECTIONS

Evidence from a range of species highlights the potential role of parasites in mediating the success of invasive species and their impacts on the recipient communities. In addition to the well studied direct impacts of parasitism, recent work has highlighted the importance of trait mediated indirect effects of parasites. These interactions are of particular interest because of the relatively rapid timescale over which they operate. Traits such as growth and fecundity that are considered in classic studies of interspecific competition may, in fact, respond too slowly to react to processes such as intra-guild predation between natives and invaders (Hatcher, Dick & Dunn 2006). It is suggested here that a wider exploration of these effects will strengthen our understanding of the impact of parasitism in interspecific interactions. In particular, while the evidence presented here supporting community scale effects of parasites linked to biological invasions is compelling, more clear demonstrations of parasites playing structuring roles in communities are needed for their general importance to be properly assessed.

Ecosystem scale effects

Since the 1980s, multiple authors have highlighted that ecosystem studies have traditionally overlooked parasites, as exemplified by looking at most food-webs that have been constructed to date (Marcogliese & Cone 1997). Although their potential importance to key ecosystem characteristics such as connectance (a metric considered to affect food web stability) has been continually voiced by parasitologists (Marcogliese 2004; Lafferty *et al.* 2008), the difficulty of quantifying parasites and their interactions by standard ecological methods has led to their exclusion. It has also perhaps been generally assumed that parasites, being small, are insignificant contributors to the flow of energy through food webs (Marcogliese & Cone 1997). However, in recent years this view has dramatically changed.

PARASITES AND ECOSYSTEM RESILIENCE

Initial efforts to include parasites in food-webs indicated little effect beyond obvious increases in species richness, number of links, trophic levels and food chain length (e.g. Huxham, Raffaelli & Pike 1995; Thompson, Mouritsen & Poulin 2005). However, more suitable methods for incorporating parasites into food-webs (developed by Lafferty, Dobson & Kuris 2006) have revealed that traditional approaches generally underestimate the effects of parasites on food web metrics. In their re-analysis of four previously published food-webs, Lafferty, Dobson & Kuris (2006) demonstrated that

parasites consistently (and sometimes dramatically) increase connectance (the proportion of potential links between organisms in a food web that is realized), and can also increase other food web statistics such as nestedness (a measure of the asymmetry of interactions between organisms). More recent studies have replicated these results (e.g. Amundsen *et al.* 2009). Since both higher connectance and higher nestedness (resulting from more 'cohesive' food webs that are organized around a central core of interactions) are thought to make an ecosystem more resistant to species extinctions (ecosystem 'resilience'), these results indicate that parasites may potentially be having very important ecosystem scale influences (Hudson, Dobson & Lafferty 2006). However, we are only now starting to investigate whether such influences are realized or not; for example, does an increase in connectedness provided by parasites in a food-web actually translate into an increase in ecosystem resilience to perturbations? In what other ways does parasitism influence ecosystem properties?

Recent results of a long-term study by Kuris *et al.* (2008) show very clearly that parasites are not always 'bit players' in ecosystem energetics. Through an extensive 5-year quantification of the free-living and infectious biomass in three estuaries, they demonstrated that parasites can have substantial biomass in these ecosystems, exceeding that of top predators. This makes a strong case that parasites could potentially have major influences on ecosystem function simply through changes in their abundance; under such circumstances the traditional assumption that parasites are insignificant contributors to the flow of energy through food webs clearly does not hold. With no other studies of a similar magnitude yet available it is too soon to assess whether parasites are commonly represented in other systems to a similar degree, or whether the ecosystem type studied by Kuris *et al.* is particularly unusual. One other study to date has shown that the findings of Kuris *et al.* do apply elsewhere; Sukhdeo & Hernandez (2004) demonstrated that larval acanthocephalan populations in freshwater streams also have the expected biomass roughly equivalent to a population of top predators. However, further studies of a broad range of ecosystem types are still needed to understand whether or not these findings are general and, if not, under what circumstances and conditions fluxes in overall parasite abundance are significant in ecosystem energetics.

TROPHIC CONSEQUENCES OF PARASITE IMPACTS

Taking a step back from the larger scale considerations of ecosystem resilience, evidence is now available that shows clearly how, as for other trophic interactions, parasites and their disease impacts on host populations can have important ecosystem scale consequences (Hudson, Dobson & Lafferty 2006). This is particularly the case if keystone species are impacted. For example, when microsporidian epidemics cause population crashes of the caddisfly *Glossosoma nigrior*, the reduction in pressure from this dominant herbivore leads to increased abundance of algae and the filter feeders and

grazers of algae, in addition to predatory insects (Kohler & Wiley 1997). As another example, it has been predicted that reductions in Tasmanian devil abundance caused by the impact of the facial tumour disease DFTD will result in mesopredator release, prey release, and changes in ecosystem function through changes in community composition, productivity and stability (Jones *et al.* 2007). In addition, although recent genetic surveys indicate that some subpopulations of Tasmanian devils may be resistant to DFTD (Siddle *et al.* 2010), devil populations may be prevented from recovering even if the disease impact is removed, due to invasive fox populations in Tasmania increasing in abundance and potentially encroaching on the devils' ecological niche (Jones *et al.* 2007). Hence, reductions in host abundance due to parasite impacts may not only have important consequences for the competitors, predators or prey of that species, but when combined with other factors may even cause irreversible ecosystem changes. However as noted above, such impacts of parasites do appear to be context dependent, and hence the occurrence of ecosystem scale consequences resulting from them will be similarly limited.

In addition to direct impacts on host populations, trait mediated indirect effects of parasites (TMIEs; see above) may also have ecosystem scale consequences when they occur (Hudson, Dobson & Lafferty 2006; Lefevre *et al.* 2009). Particularly dramatic consequences can be observed when parasites play 'ecosystem engineering' roles, i.e. when their influences on their hosts result in habitat creation or change for other community members (Thomas *et al.* 1999). The influence that trematode infection of the New Zealand cockle *Austrovenus stutchburyi* has on intertidal flat community structure is a well studied example of this phenomenon. Here, infection of the cockle impairs its burrowing ability, resulting in more cockles remaining on the sediment surface where they provide the only hard substrate in this ecosystem (Thomas *et al.* 1998). Although the influence on behaviour appears not to be manipulative, but simply a side-effect of the infection process (Tompkins, Mouritsen & Poulin 2004), cockle parasitism is associated with intertidal community structure on a large spatial scale, influencing physical habitat characteristics with cascading effects on biodiversity and ecosystem productivity (Mouritsen & Poulin 2010). In another example, Wood *et al.* (2007) demonstrated that the behavioural reduction in grazing that trematode infection of the herbivorous marine gastropod *Littorina littorea* causes indirectly affects the composition of the macro-algal community and may in turn affect other species that depend on macro-algae for resources or habitat structure. Hence, irrespective of whether parasites are impacting the numerical abundance of their hosts, they may still be having larger scale effects.

FUTURE DIRECTIONS

As noted above, although we now know that parasite biomass can be relatively significant in ecosystems, further research is needed to characterize the circumstances and con-

ditions under which this occurs. In addition, it is yet to be demonstrated that increases in connectedness and other food web parameters due to parasites can actually translate into increased ecosystem resilience (i.e. while the absence of parasites can indicate the effects of stressors on ecosystems, is their presence indeed indicative of better 'ecosystem health'; Marcogliese 2005), even in cases where overall parasite biomass is relatively high.

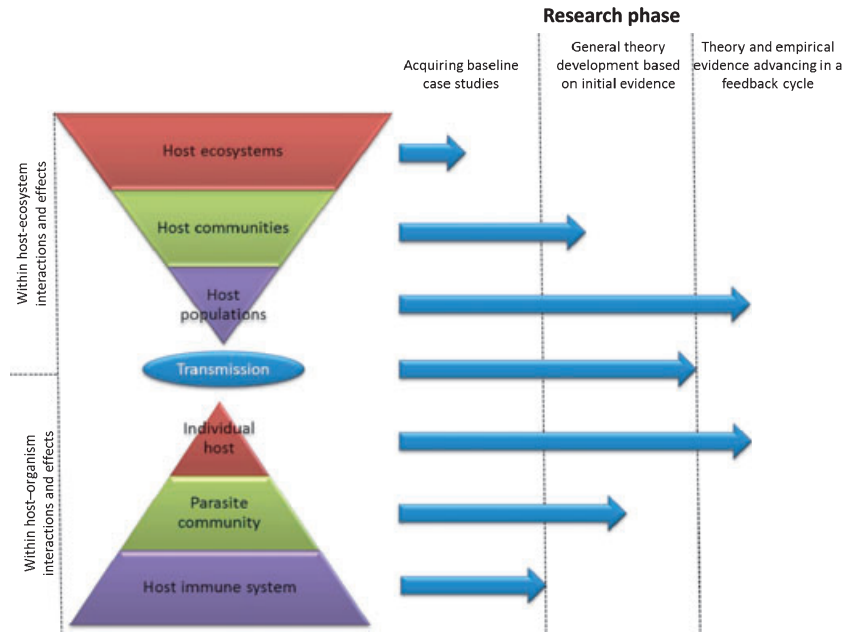
For the trophic consequences of parasites, influences via their direct impacts on host populations appear to be highly context dependent (see above). However, evidence for parasite driven TMIEs is becoming both common and highly varied; hence it is perhaps these more subtle effects that are far more important than direct impacts in shaping natural communities and ecosystems. However it appears that, to date, we are only just 'breaking the ice' in terms of determining when and how such influences tend to be important.

Conclusion

Like most fields of academic research, distinct phases can be identified in the development of wildlife disease ecology as a scientific discipline (Fig. 2). Prior to the 1990s most attention was given to the population scale impacts of parasites and disease. Considerations of both within-host processes and larger ecological scales was generally limited to direct and obvious impacts, such as the clinical effects of infection or the dramatic effects of emerging diseases such as those caused by Rinderpest in Africa (Plowright 1982). Over the past two decades, however, there has been a surge of interest at both these larger and smaller scales. For within-host processes this has been partly driven by progress and development of appropriate tools in related disciplines such as immunology, whilst focus on the potential effects of parasitism at community and ecosystem scales has been driven by increasing interest in global change processes such as invading species, emerging disease, and biodiversity loss. For parasite transmission between hosts, hurdles imposed by a lack of appropriate tools have recently been overcome, allowing us to start to discern the underlying mechanisms driving population scale patterns.

It is apparent in our review that there have been considerable advances in our knowledge of the potential ways in which parasites influence individual to ecosystem-level processes. At the population scale (and also for direct host/parasite interactions), research is now in a mature phase where our understanding is being refined through the established scientific process of theory and empirical evidence developing in a feedback cycle, identifying what effects are actually realized in the field and under what conditions they do so. However, at the greater scale of the host community, and the lesser scales of transmission between hosts and parasite interactions within hosts, it can be argued that research is still in an earlier phase with general theory still being developed based on initial evidence. The challenge for research at these scales is now to develop into a more mature phase where

Fig. 2. Our perspective on the current state of play of research into wildlife disease ecology across different ecological scales. Transmission connects within-organism disease dynamics (lower triangle) to larger ecological scales (upper triangle). These effects interact between multiple scales of organization (different sections of the triangles), however our current level of understanding at these different scales differs significantly (arrows).



stronger connections between general theory and empirical data are refined, clarifying what influences and interactions actually play important roles in wild populations, and under what conditions and circumstances they do so. For the relatively less developed areas of wildlife disease ecology, focusing at one end of the scale on the influence of parasites in ecosystems (and, to a certain degree, the role of trait mediated indirect effects), and at the other end of the scale on the interactions between parasites and host immunology, the challenge is still to complete developing sound initial theoretical frameworks.

Arguably, it is the empirical testing of theoretical predictions and subsequent development of theory in the classic research cycle that has led to significant progress in our understanding of host-parasite population dynamics in recent years. The recent emphasis on community ecology frameworks to help understand within host interactions (Pedersen & Fenton 2007; Graham 2008) should facilitate similar hypothesis-driven testing in this field, as should the call to synthesize theory across an understanding of disease transmission mechanisms (see above), and the need to understand what potential community scale influences of parasites (such as parasite-mediated apparent competition) are actually realized in the field and the context in which they do so. Rapid progress in empirical research is most likely to be achieved by a combination of approaches. Field-based experimental studies could provide a lot of the necessary ground-truthing for new theory; such research may be costly but proof of concept demonstrations of many hypothesized parasite driven mechanisms operating in the wild (including, among others, interspecific parasite interactions and their implications for host fitness, the causes and consequences of transmission heterogeneities, parasite spillback and dilution, and improved fitness due to parasite species loss) are still lacking. Both meta-analyses and the collection and analysis

of long-term data sets will be invaluable for characterizing the conditions and circumstances under which potential parasite interactions and influences are actually realized (e.g. Graham 2008). Improving our mechanistic understanding of parasite related ecological processes in such a manner will also enable the identification of the contexts in which different transmission mechanisms drive different population scale dynamics, TMIEs influence community structure, and ecosystem-scale influences occur.

In parallel, further empirical and theoretical research is needed to explain and predict how parasitism at the level of individuals and populations scales up into complex ecological processes. We are only now beginning to understand the importance of such cross-scale interactions associated with parasitism. For example, strong interactions between parasite species may translate into effects at the level of the host population: during unusually deadly canine distemper virus outbreaks in lions, within pride mortality rates were positively correlated with the proportion of individuals showing high intensity *Babesia* infections, suggesting that co-infection was a major contributing factor during fatal epidemics (Munson *et al.* 2008). In addition, Cattadori, Haydon & Hudson (2005) working on the red grouse/*T. tenuis* system, have illustrated clearly how environmental effects on parasite transmission rates might lead to synchronized dynamics among multiple host populations. Finally, TMIEs can have strong counter-intuitive effects on population dynamics and community structure (Abrams & Matsuda 1996), and the recent review of Lefevre *et al.* (2009) shows that parasites may influence populations, communities and whole ecosystems through such effects.

A key focus of ecology is the search for useful generalities. It is here that cross-scale, and indeed cross-disciplinary, research may most benefit our understanding of wildlife disease ecology. For example, joining together studies of par-

asites in invaders and residents may help unify our understanding of how the stability and complexity of parasite communities develop over time (e.g. MacLeod *et al.* 2010), drawing from established seminal ecological theory such as that developed for island biogeography. Along similar lines, both the confirmation that investment in resistance to parasites likely trades-off against other life-history investments, and the recognition that immuno-dynamics may place a role in determining the population scale impacts of parasites (Beldomenico & Begon 2010), means that an integration of principles from evolutionary biology and immunology will be necessary for our understanding of wildlife disease ecology. Such cross-disciplinary work may offer key insights into bigger picture questions such as what determines when and how different potential regulatory factors (e.g. competition, predation, parasitism, herbivory) are important for understanding abundance patterns (see Holmes 1995), when parasites and disease can actually cause species extinctions, and what levels and combinations of different factors are indicative of functionally resilient ecosystems.

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