Competition mediated by parasites: biological and theoretical progress

Peter Hudson and Jon Greenman

Competition mediated through a shared natural enemy is known as 'apparent competition' and could be an important force determining community structure and biodiversity. Recently, experimental and theoretical studies have provided a new insight into the mechanisms and conditions that could influence coexistence or exclusion. Parasite-mediated competition is particularly significant, given the rise in emerging diseases and the opportunity that pathogens have to reduce host abundance.

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With his usual foresight, J.B.S. Haldane argued nearly 50 years ago that 'a non specific parasite is a powerful competitive weapon'[1]. One species can be a superior competitor simply by harbouring and transmitting a pathogen to a more vulnerable species. This is 'apparent' competition, which is mediated via a shared pathogen, and contrasts with 'resource' competition, in which there can be rivalry over resources, such as food or nesting sites. Competition is generally accepted as a major force influencing biodiversity, so it is likely that apparent competition could play a significant role in shaping community structure.

Interest in apparent competition has blossomed over the past ten years[2], but our understanding has recently advanced with some clear experimental studies and theoretical developments. On the biological side, there has been a wealth of examples[2] that have provided evidence of competition through a shared pathogen and highlighted the asymmetry in the competition with clear winners and losers. However, the majority of these examples have been descriptive and have often failed to disentangle the effects of resource competition from those of apparent competition. A recent experimental study[4] has separated these two forms of competition and shown apparent competition in action. The theory of parasite-mediated competition has centred on the deterministic 'susceptible–infectious' (SI) models that have been applied so successfully to the dynamics of single host–pathogen systems, such as measles[3]. However, transferring this theory from one host species to multiple host species has been hampered by problems in identifying the equilibria (caused by the intractability of the algebra). Further theoretical developments have only been resolved through an indirect route.

Parasitoids and moths: apparent competition in action

A recent laboratory study provides an excellent illustrative example of how the presence of a second host species can destabilize a parasite–host relationship and result in the elimination of one species[4] (Fig. 1). The system used was a simple ichneumonid parasitoid (*Venturia canescens*) and two caterpillar hosts (*Plodia interpunctella* and *Ephestia kuehniella*); the parasitoid lays her eggs inside the caterpillar host and the larval, parasitic stage of the parasitoid kills the caterpillar and emerges as an adult. When experimental chambers contained just a single host species plus the parasitoid then both persisted and exhibited damped oscillations tending towards a stable equilibrium. However, when the system was run with the parasitoid and two host species, the parasitoid had a greater impact on the species with the lower intrinsic growth rate – this species exhibited increasing oscillations in abundance and was eventually eliminated. The experimental design was particularly elegant because the two-host species system allowed free passage of the parasitoid between both host species but kept the hosts apart, thus avoiding interspecific resource competition.

These findings (also supported by theory[2,5]) imply that biological control will be more successful when a second host species with a higher intrinsic growth rate than the target species is added to the system. In addition, the effects of apparent competition will increase when the parasitoid shows a strong numerical response to its prey density. Within certain constraints, the same is true of parasite-mediated competition; the pathogen will usually have a greater impact when it has a greater basic reproductive number R_0 (the number of new cases arising from a primary case introduced into a population of susceptibles) in the reservoir species than the target species. Indeed, we can expect pathogens, which have short generation times (compared with predators) and are not limited by resources other than susceptible hosts, to play an important role in apparent competition.

Parasite-mediated competition and emerging diseases

Several parasites have become increasingly important to mankind, domestic animals and wildlife as a range of 'emerging diseases' has increased in prevalence. Many are derived from wildlife species and have been passed from one host species to another, causing significant mortality in those hosts with little resistance. These can be viewed as parasitic invasions of a naive host, an asymmetry brought about through a difference in relative susceptibility, which frequently occur as a consequence of environmental rather than evolutionary change[6]. For example, the conversion of grassland to maize agriculture in Argentina resulted in a rise in the rat (*Calomys musculinus*) population, increasing transmission of the junin virus to humans and causing many fatalities.

Parasite-mediated competition can act when an invading species introduces a parasite to a vulnerable, resident species. This might have been the scenario when grey squirrels (*Sciurus carolensis*) were introduced into Britain, bringing with them a parapox virus that reduced the competitive ability of the native reds (*S. vulgaris*)[7]. In conservation, rabies is one of the most threatening diseases imported by invading

  0169-5347/98/$19.00 PII: S0169-5347(98)01475-X

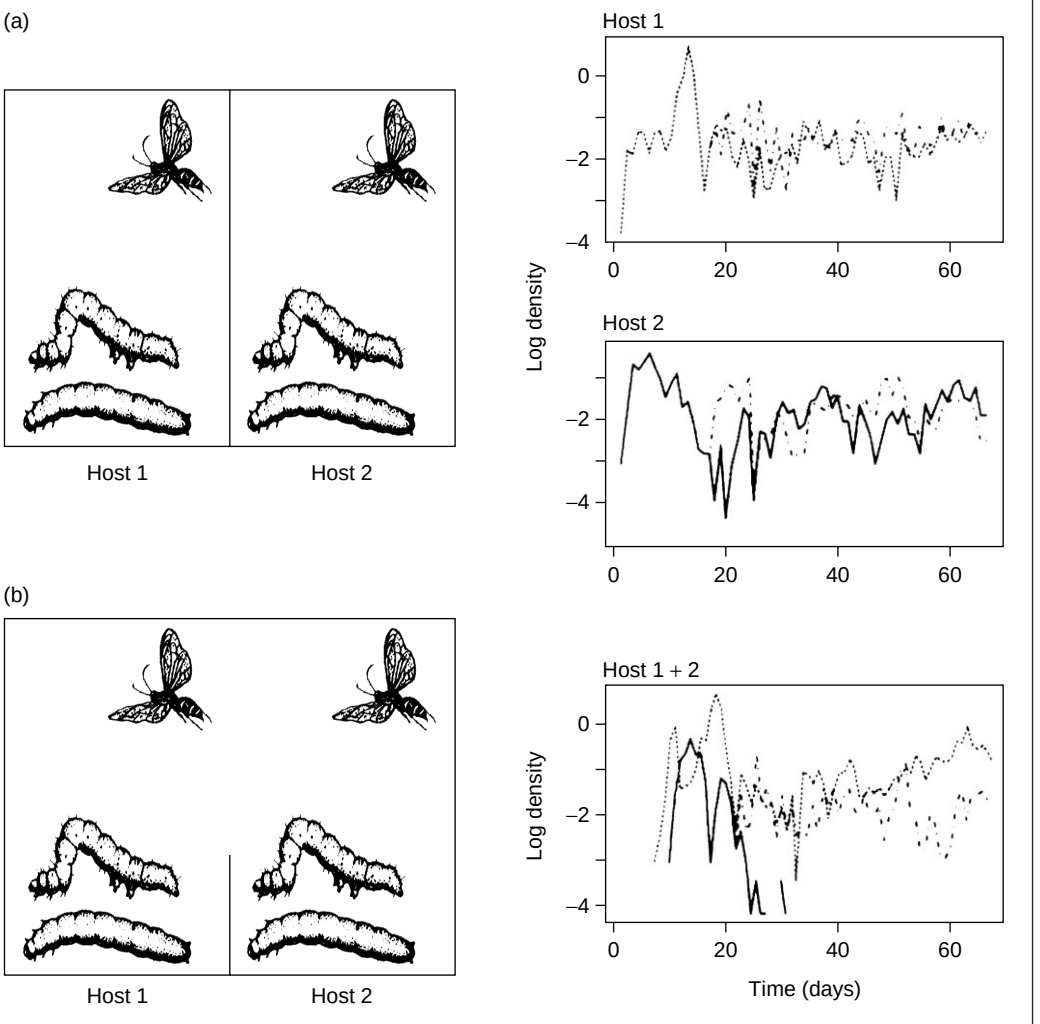


Fig. 1. Parasite-mediated competition via a parasitoid wasp (*Venturia canescens)* that lays her eggs in two caterpillar host species (*Plodia interpunctella* and *Ephestia kuehniella*). The experimental set-ups are illustrated on the left and the densities of hosts (host 1, dotted line; host 2, unbroken line) and parasitoid (dashed line) on the right. (a) When parasitoids were kept within single-host systems the parasitoid and each host coexisted with stable dynamics. (b) When the parasitoid has access to both host species, then host species 2 showed diverging oscillations in abundance and invariably went extinct. *Reproduced, with permission, from Ref. 4.*

species (usually by domestic dogs) that has exerted a significant form of competition on many of the rarer and endangered canids, such as Ethiopian wolves (*Canis simensis*) and wild dogs (*Lycaon pictus*)[8,9].

The reverse can also take place. If the parasite weakens the competitive ability of the resident species, invasion can occur[10,11]. The native mussel found along the coast of South Africa (*Perna perna*) is heavily infested with two trematode species – bucephalid sporocysts castrate the host whereas *Proctoeces* species reduce the growth and the competitive ability of the host. In recent years, the European mussel (*Mytilus galloprovincialis*), which is not infected by the trematodes, has been introduced and is now slowly and relentlessly replacing the native species. This might, of course, have occurred without the parasite, but it seems reasonable to suppose that the trematodes are impinging on the competitive ability of the native species.

Interactions between the pathogens of rabbits (*Oryctolagus cuniculus*) and hares (*Lepus europaeus*) provide some field evidence that pathogens can influence a competitive outcome. Studies in the Netherlands found a stomach worm (*Graphidium strigosum*) to be more pathogenic in hares than rabbits. Development and transmission of the worm are favoured in shady woodlands where the presence of rabbits increases the pool of infective stages that can infect and exclude the hares[12]. In contrast, the myxoma virus has a dramatic effect on the population size of rabbits but not hares, and so the demise of rabbits was generally followed by a rise in some of the local hare population[13,14]. At first glance, this would imply that the myxoma virus had reduced competition by the rabbit on the hare. However, this could also have come about through other forms of apparent competition. For example, the loss of rabbits might have reduced the fox population and this could have led to reduced predation on hares and a subsequent increase in the hare population.

One of the clearest demonstrations of apparent competition is when an invading pathogen reverses the outcome of resource competition between two species. The classic example is the experimental study undertaken by Park[15] and reanalysed by Anderson and May[16]. This study looked at competition between two flour beetles (*Tribolium confusum* and *T. castaneum*) and the influence of a sporozoan parasite (*Adelina tribolii*). When mixed cultures were run without the pathogen, *T. confusum* invariably dominated, but when cultures included the parasite the outcome was reversed. The sporozoan is more pathogenic in the dominant competitor. Consequently, when the two competitors are present, the pathogen reduces the competitive ability of the dominant species and reverses the outcome of the contest, permitting the inferior competitor to persist (Fig. 2).

Theoretical developments

The theoretical underpinning for parasite-mediated competition is based on the susceptible–infectious (S–I) model[17], which describes the impact of a directly transmitted microparasite on the dynamics of two host species where neither develop resistance. This has been extended to include other affects, such as density dependence in host-population growth and resource competition between species[18–20]. Nevertheless, the general relevance of the model has been constrained by a range of mathematical difficulties and biological assumptions.

Mathematical difficulties

Ideally, in the analysis of a dynamic model, we classify the system behaviour (as either stable or unstable) and identify the conditions under which the model exhibits these

behaviours. For the two-dimensional Lotka-Volterra models, which describe simple trophic interactions, the analysis is straightforward. However, for higher order systems, such as multihost systems and multipathogen systems, the stability analysis is more complex. Some progress can be made by using simplifying assumptions[21,22]. This has proved successful with multipathogen systems by presenting the findings graphically within the parameter space determined by the respective basic reproductive numbers (R_0) of each pathogen[23]. However, in parasite-mediated competition we are particularly interested in pathogens that are shared between host species, and here the algebraic analysis comes up against intractable problems. Initially, this problem was avoided by undertaking extensive numerical simulations and drawing generalized conjectures about the number and stability of the various system equilibria[17–19]. Such an approach is not foolproof and has been shown by counterexample not to be universally valid[24]. Indeed, the dynamics of the system are more varied and interesting than were originally thought. The challenge has been to find a way round the algebraic problems in order to gain some qualitative understanding of the range of possible behaviour patterns within the system.

Recent advances have been made towards this aim through the application of bifurcation theory. A bifurcation point defines a set of parameters that identifies a qualitative change in the dynamical behaviour of the system – for example, from stability to instability, or convergent into sustained oscillations. The bifurcation points define surfaces that separate the different behavioural modes. An example is given in Box 1 where a cross section of parameter space is taken, defined by the host reproductive numbers. A key property of the dominant (transcritical) type of bifurcation is the 'duality' of its surface. It forms the stability boundary for one equilibrium and the relevance boundary for a second equilibrium (negative equilibria are not biologically relevant). Also, close to this boundary, the two equilibria typically have opposite stability states (i.e. stable and unstable) and, consequently, one can infer properties of the second equilibria from those of the first. One way of looking at the duality relationship is that the first equilibrium acts as a 'gate' into the relevant region for the second, hence this type of analysis is now referred to as 'gateway analysis'[25].

Biological limitations

There are several biological limitations with these models. These include a lack of lasting immunity, a random pattern of mixing and a need for frequent and direct transmission between species. Indeed, for a species to be excluded usually requires frequent interspecific contact and high rates of interspecific transmission. Such contacts are rare and not as common as contact between conspecifics. For example, in the transmission of the viral rinderpest between ungulates in the Ngorogoro Crater (Tanzania), the probability of between-species transmission is far lower than within-species transmission[26]. Nevertheless, when individuals within a species are evenly spread in territories, but territories are independent between species, then interspecific contact and transmission may be greater than intraspecific transmission. In any case, between-species transmission is far more likely when the pathogen has long-lived free-living stages, particularly when they cause a chronic infection in the reservoir host, or where there is a vector that increases between-species exposure.

Vectors and parasite-mediated competition via one host

Ticks and other invertebrate vectors take their blood meal from a range of vertebrate hosts and provide an opportune route for between-species transmission. The bacterium *Borrelia burgdorferi*, which causes Lyme borreliosis in humans, has increased dramatically in North America as tick numbers have multiplied following the expansion of white-tailed deer in rough scrub. The pathogen normally circulates in populations of the white-footed mouse (*Peromyscus leucopus*), but changes in the acorn crop have altered the movement of mice and, together with other ecological factors, resulted in the increased passage of the pathogen from mouse to tick to humans[27,28].

Interestingly, parasite-mediated competition can occur through the presence of a second species that need not be a host for the pathogen. For example, in tick-borne pathogens, not all hosts are amplifiers; some produce a strong immune response, so there is insignificant multiplication of the pathogen within the host and no transmission from host to vector. However, by providing a blood meal for the vector, they maintain the vector population and help the pathogen to persist. For example, the mountain hare (*Lepus timidus*)

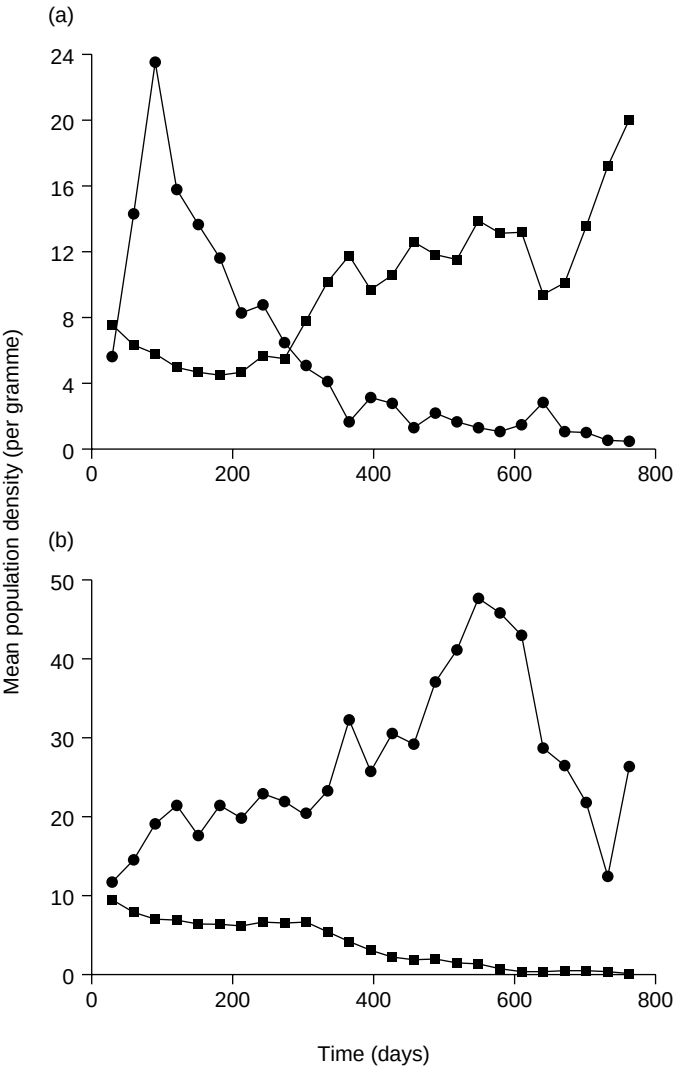
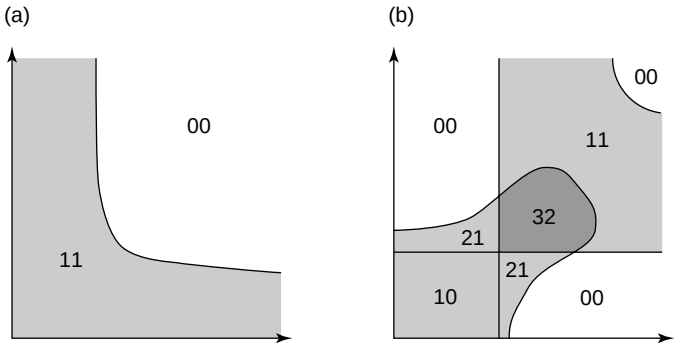


Fig. 2. The classic experiments undertaken by Thomas Park[15] on the competition between two species of flour beetles (*Tribolium confusum* and *T. casteneum*) illustrating how the presence of a shared pathogen (*Adelina tribolii*) reverses the results of resource competition. (a) Population changes in *T. casteneum* (squares) and *T. confusum* (circles) in the presence of a shared infection, illustrating how *T. casteneum* dominates. (b) The two beetles in the absence of infection, illustrating how *T. confusum* dominates. *Reproduced, with permission, from Ref. 16.*

Box 1. Gateway analysis illustrated

The changes in the behaviour of multihost, pathogen models can be revealed through bifurcation analysis. This analysis identifies bifurcation points that define surfaces that separate different types of model behaviour, such as stability or instability. As an example, consider the case of a shared microparasite between two host species that we can describe with the basic susceptible–infectious model. The two figures illustrate the bifurcation surfaces in the cross section of parameter space; defined by the reciprocals of the reproductive numbers (the number of new cases arising from a primary case introduced into a population of susceptibles) of the pathogen in each host species i (R_{0i}). The reciprocal is used so that single host infections persist close to the origin. The shaded areas are the regions where the infected coexistence equilibria are relevant (i.e. positive) and the numbers represent p,q, where there are p relevant equilibria of which q are stable. In (a), where between-species transmission-coefficients are low, infected coexistence occurs for sufficiently high values of R_{0i}, but host exclusion is not possible. In (b), where between-species transmission-coefficients are high, then stable infected coexistence is not possible for high R_{0i}, in which case host exclusion takes place. Figure (b) also shows that when these transmission coefficients are high then complexities can arise, with multiple equilibria present.



is not an amplifier of the louping-ill virus but is an important host for the ticks that can carry it and allows both ticks and virus to persist in red grouse (*Lagopus l. scoticus*)[29]. Therefore, competition is being mediated through the effects of a pathogen even though the host might not play any role in amplifying the virus. However, this is still parasite-mediated competition because in the absence of the hares the impact on the grouse would be reduced. Haldane would be fascinated to see that even a highly specific pathogen can be a powerful competitive weapon.

Conclusion

Apparent competition, mediated through pathogens, can influence community structure and might account for a range of interesting dynamics in host populations. These dynamics can now be explored more rigorously and the theory developed further by applying gateway analysis. Systems for which transmission is via a vector or through long-lived infective stages are more likely to generate parasite-mediated competition because of increased transmission between species. Perhaps our failure to understand the dynamics of some infectious diseases is because the role of secondary, or even supplementary, hosts and vectors have not been considered in sufficient detail.

Acknowledgements

We would like to thank Bob Holt, Mike Bonsall, Mike Hassel, Dan Tompkins, Ken Wilson and Tim Benton for their helpful comments on the draft manuscript. Bob Holt kindly pointed out some interesting features of parasite mediated competition.

References

1 Haldane, J.B.S. (1949) **Disease and evolution,** *Ric. Sci.* (Suppl.) 19, 68–76
2 Holt, R.D. and Lawton, J.H. (1994) **The ecological consequences of shared natural enemies,** *Annu. Rev. Ecol. Syst.* 25, 495–520
3 Grenfell, B.T. and Harwood, J. (1997) **(Meta)population dynamics of infectious diseases,** *Trends Ecol. Evol.* 12, 395–399
4 Bonsall, M.B. and Hassell, M.P. (1997) **Apparent competition structures ecological assemblages,** *Nature* 388, 371–372
5 Holt, R.D. (1997) **Community modules,** in *Multitrophic Interactions in Terrestrial Systems* (Gange, A.C. and Brown, V.K., eds), pp. 333–350, Blackwell
6 Schragg, S.J. and Wiener, P. (1995) **Emerging infectious disease: what are the relative roles of ecology and evolution?** *Trends Ecol. Evol.* 10, 319–324
7 Sainsbury, A. and Ward, L. (1996) **Parapox infection in red squirrels,** *Vet. Rec.* 138, 400
8 MacDonald, D.W. (1993) **Rabies and wildlife: a conservation problem?** *Onderstepoort J. Vet. Res.* 60, 351–355
9 Pain, S. (1997) **Plague dogs,** *New Sci.* 155, 32–37
10 Calvo-Ugarteburu, G. and McQuaid, C.D. (1997) **Parasitism and introduced species: epidemiology of trematodes in the intertidal mussels *Perna perna* and *Mytilus galloprovincialis*,** *J. Exp. Mar. Biol. Ecol.* 120, 47–65
11 Calvo-Ugarteburu, G. and McQuaid, C.D. **Parasitism and invasive species: effects of digenetic trematodes on mussels,** *Mar. Ecol. Prog. Ser.* (in press)
12 Broekhuizen, S. and Kememrs, R. (1976) **The stomach worm, *Graphidium strigosum* (Dujardin), in the European Hare, *Lepus europaeus*, Pallas,** in *Ecology and Management of European Hare Populations* (Pielowski, Z. and Pucek, Z., eds), pp. 157–171, Polish Hunt Society
13 Siriez, H. (1960) *Lapin et myxomatose. L'evolution de la maladie 1956–1960 et quelques compterments a une precedente etude*, Éditions Sep
14 Rothschild, M. (1958) **A further note on the increase of hares (*Lepus europaeus*) in France,** *Proc. Zool. Soc. London* 131, 328–344
15 Park, T. (1948) **Experimental studies of interspecific competition I. Competition between populations of the flour beetles, *Tribolium confusum* and *Tribolium castaneum*,** *Ecol. Monogr.* 18, 267–307
16 Anderson, R.M. and May, R.M. (1986) **The invasion, persistence and spread of infectious diseases within animal and plant communities,** *Philos. Trans. R. Soc. London Ser. B* 314, 533–570
17 Holt, R.D. and Pickering, J. (1985) **Infectious disease and species coexistence: a model of Lotka-Volterra form,** *Am. Nat.* 126, 196–211
18 Begon, M. *et al.* (1992) **Disease and community structure: the importance of host self-regulation in a host–host pathogen model,** *Am. Nat.* 139, 1131–1150
19 Begon, M. and Bowers, R.G. (1995) **Beyond host–pathogen dynamics,** in *Ecology of Infectious Diseases in Natural Populations* (Grenfell, B.T. and Dobson, A.P., eds), pp. 478–509, Cambridge University Press
20 Bowers, R.G. and Turner, J. (1997) **Community structure and the interplay between interspecific infection and competition,** *J. Theor. Biol.* 187, 95–109
21 Yan, G. (1996) **Parasite-mediated competition: a model of directly transmitted macroparasites,** *Am. Nat.* 148, 1089–1112
22 Hochberg, M.E. and Holt, R.D. (1990) **The coexistence of competing parasites I. The role of cross species infection,** *Am. Nat.* 136, 517–541
23 Gupta, A., Swinton, J. and Anderson, R.M. (1994) **Theoretical studies of the effects of heterogeneity in the parasite population of the transmission dynamics of malaria,** *Proc. R. Soc. London Ser. B* 256, 231–238
24 Greenman, J.V. and Hudson, P.J. (1997) **Infected coexistence: instability with and without density-dependent regulation,** *J. Theor Biol.* 185, 345–356
25 Greenman, J.V. and Hudson, P.J. (1998) **Exploring parasite-mediated competition using Gateway Analysis: complexity and dynamics in multi-host pathogen models,** *Stirling Mathematical Ecology Group* (Working Paper 13)
26 Dobson, A.P. (1995) **The ecology and epidemiology of rinderpest virus in Serengeti and Ngorogoro conservation area** in *Serengeti II. Dynamics, Management and Conservation of an Ecosystem* (Sinclair, A.R.E. and Arcese, P., eds), pp. 485–505, The University of Chicago Press
27 Ostfeld, R. (1997) **The ecology of Lyme disease risk,** *Sci. Am.* 85, 338–346
28 Jones, C.G. *et al.* (1998) **Chain reactions linking acorns to gypsy moth outbreaks and Lyme disease risk,** *Science* 279, 1023–1026
29 Hudson, P.J. *et al.* (1995) **Persistence and transmission of tick borne viruses: *Ixodes ricinus* and louping-ill virus in red grouse populations,** *Parasitology* 111, S49–S58