

### Topic Rooms

Within this Subject (24)

- Basic (13)
- Intermediate (5)
- Advanced (6)

### Other Topic Rooms

- Ecology**
  - Evolution
  - Ecosystem Ecology
  - Physiological Ecology
  - Population Ecology
  - Community Ecology
  - Global and Regional Ecology
  - Conservation and Restoration
  - Animal Behavior
  - Teach Ecology
- Earth Systems**
  - Earth's Climate: Past, Present, and Future
  - Terrestrial Geosystems
  - Marine Geosystems
- Soil, Agriculture, and Agricultural Biotechnology**
- Environmental Ethics**
- Biological Anthropology**
  - Scientific Underpinnings
  - Paleontology and Primate Evolution
  - Human Fossil Record
  - The Living Primates

### Connect



### Blogs

### ADVANCED

► **ANIMAL BEHAVIOR** | Lead Editor: Sara Tenney



## Disease Ecology

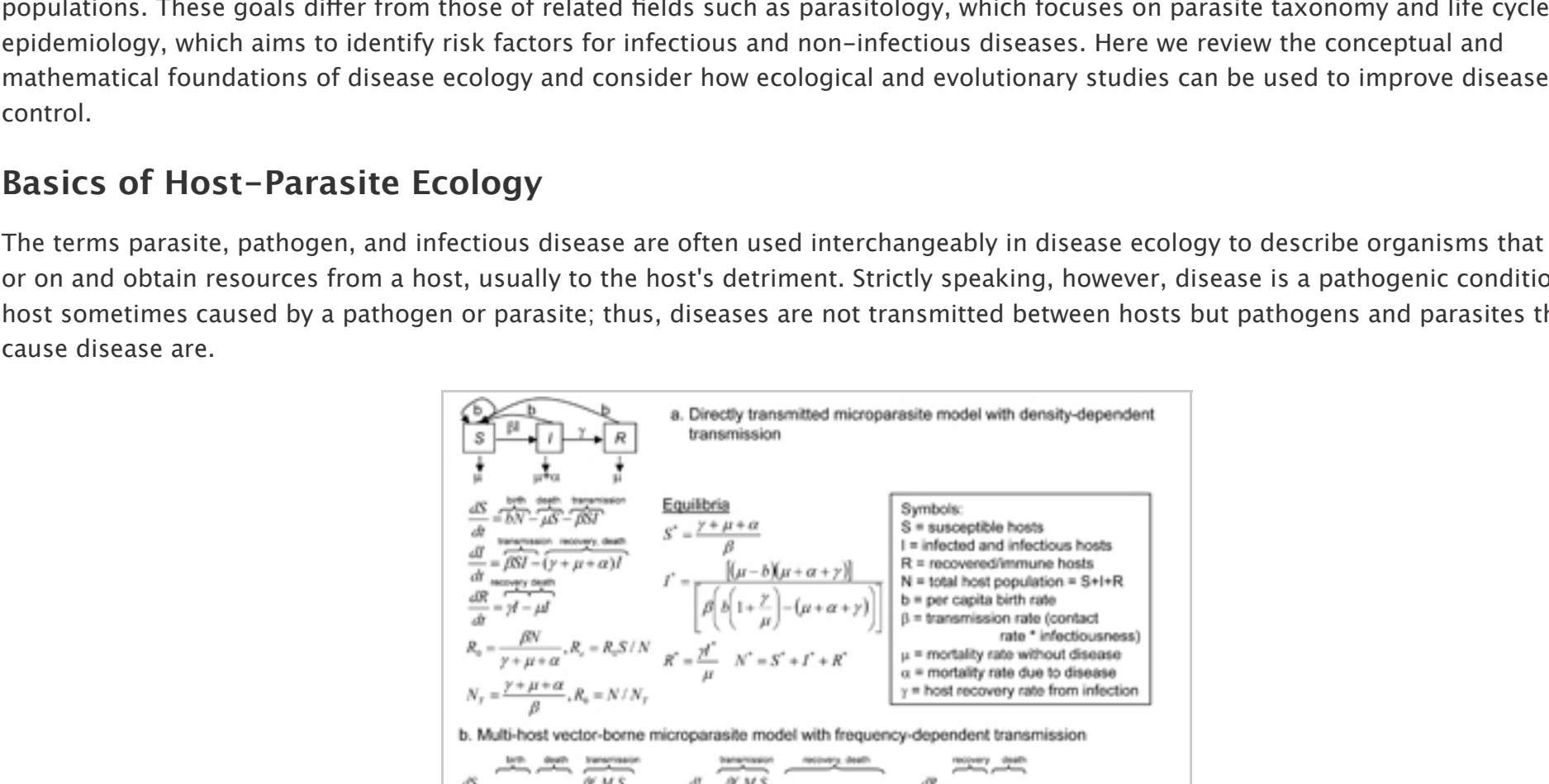
By: A. Marm Kilpatrick (*Ecology & Evolutionary Biology, University of California at Santa Cruz*) & Sonia Altizer (*Odum School of Ecology, University of Georgia*) © 2010 Nature Education

Citation: Kilpatrick, A. M. & Altizer, S. (2010) Disease Ecology. *Nature Education Knowledge* 3(10):55



## How do climate, evolution, and free-living hosts interact to determine the dynamics of pathogens and the burden of disease?

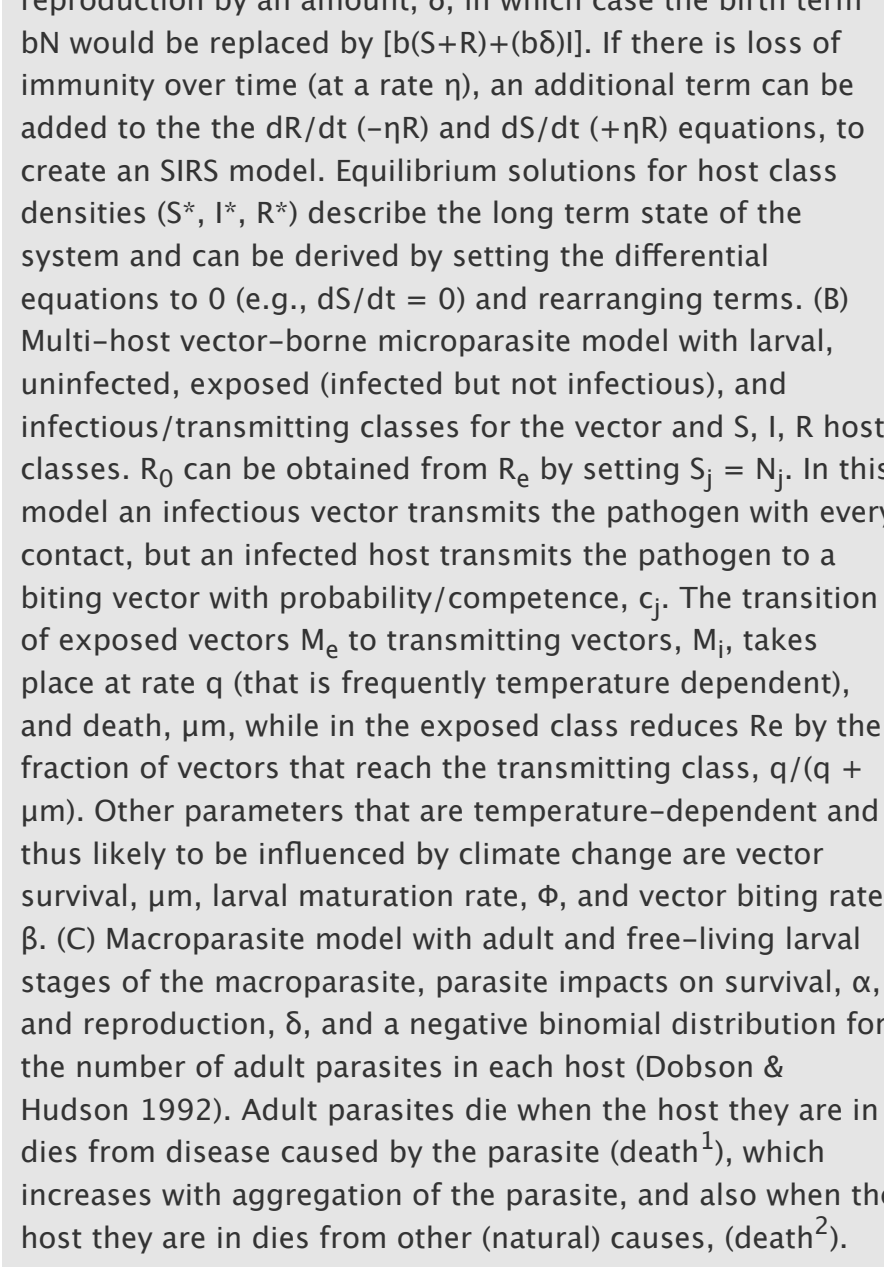
Aa Aa Aa



Infectious diseases have long been known to cause devastating illnesses in humans, crops, and livestock, but until recently pathogens were assumed to have little impact on wild plant and animal populations, except in rare and sometimes spectacular die-off events. During the past two decades, it has become increasingly apparent that parasitic organisms are not only a common and integral part of ecosystems, but they also influence the abundance of wild populations, can cause extinctions of their hosts, and serve as drivers of evolution (Hudson *et al.* 2002). The field of disease ecology, defined as the ecological study of host–pathogen interactions within the context of their environment and evolution, has grown out of this awareness of the pervasive role of pathogens in ecosystems. At the foundation of disease ecology are efforts to understand pathogen transmission and spread over space and time and impacts on host populations. These goals differ from those of related fields such as parasitology, which focuses on parasite taxonomy and life cycles, and epidemiology, which aims to identify risk factors for infectious and non-infectious diseases. Here we review the conceptual and mathematical foundations of disease ecology and consider how ecological and evolutionary studies can be used to improve disease control.

### Basics of Host–Parasite Ecology

The terms parasite, pathogen, and infectious disease are often used interchangeably in disease ecology to describe organisms that live in or on and obtain resources from a host, usually to the host's detriment. Strictly speaking, however, disease is a pathogenic condition of a host sometimes caused by a pathogen or parasite; thus, diseases are not transmitted between hosts but pathogens and parasites that cause disease are.



© 2011 Nature Education All rights reserved.

Parasites are frequently grouped into microparasites and macroparasites to reflect differences in their population biology (Anderson & May 1979). Microparasites (which include viruses, bacteria, fungi, and most protozoa, including malaria) reproduce inside their hosts on rapid timescales, much shorter than their hosts' lifespans. This often, but not always, causes short-term infections that can result in host death or the development of immunity. For microparasites, disease ecologists group hosts into classes that reflect their stage of exposure, including susceptible (S), exposed (E; infected but not yet infectious), infectious (I; infected and able to transmit the pathogen), and, for some pathogens, recovered (R; or immune). Compartment models with accompanying equations are used to track changes in the numbers of hosts in each of these classes (but not the number of parasites in each host's body) and are described as, for example, SIR or SI models depending on the host categories that best fit the biology of the pathogen (Figure 1a, b). Models can and should be simplified to eliminate classes that aren't necessary to capture transmission dynamics. For instance, the exposed class could be eliminated if the latent period is very short relative to other stages or processes (as in Figure 1a), and the recovered class could be eliminated if few hosts recover from infection. In addition, models can be adapted to take into account a free-living stage of the pathogen if the pathogen is transmitted through water, soil, air, etc.

In contrast, macroparasites (mostly parasitic worms called helminths and parasitic arthropods such as lice) are larger, longer-lived, and rarely complete their life cycle within a single host. Instead, adult macroparasites usually shed infective stages such as eggs or larvae into the environment, and these may or may not infect the same host that the adult macroparasites live in (Figure 1c). For these pathogens, the host's immune response is often incomplete or short-lived, resulting in persistent infections and continuous re-infection. Because the impacts of macroparasites on their hosts, and often parasite survival and fecundity, depend strongly on the number of adult parasites in each host, disease ecologists keep track of the number of macroparasites in each host and mathematically quantify their distribution across hosts (Figure 1c). The distribution of parasites among hosts almost always shows evidence for aggregation or clumping which means that most hosts have only a few or no parasites, and a few host individuals have many. This clumping tendency can determine the population-level impacts of macroparasites because negative effects of parasites intensify for hosts with the highest parasite numbers and can also determine the level of competition between individual parasites within a host.

Pathogens can be transmitted in many different ways, including: by direct contact between hosts; through air, water, soil or other surfaces; or via biting arthropods, which themselves can be hosts in which the pathogen reproduces (Figure 1b). Environmental factors like temperature and humidity can determine pathogen survival outside of a host, with significant impacts on transmission. Environmental conditions can also affect pathogen replication inside arthropod vectors, which in turn determines whether the vector will transmit the pathogen subsequently (Figure 1b).

Disease ecologists utilize a quantitative approach developed from population ecology and frequently use mathematical models to develop and test hypotheses, integrate data, and guide disease control efforts. A cornerstone of this approach is the basic reproductive ratio ( $R_0$ ) of the pathogen, which describes the initial growth of a pathogen in a previously unexposed host population (Figure 1). In a deterministic setting,  $R_0$  determines whether the pathogen can invade and spread (e.g., if  $R_0$  exceeds 1). In the real stochastic world, the number of transmission chains is longer and pathogen invasion is more likely (but not certain) for values of  $R_0 > 1$ . In many cases  $R_0$  can be derived intuitively as the ratio of new infections (the  $\beta SI$ ) caused by one infectious individual ( $I = 1$ ) when the population is wholly susceptible ( $S = N$ ), divided by the rate that infectious individuals are lost (the sum of the three loss terms: natural death, disease-caused death, and recovery  $-\mu + \alpha + \gamma$ ).

A more general quantity ( $R_0$ ) is the effective reproductive ratio in a host population in which some individuals may be resistant to infection due to previous exposure, vaccination, or other causes (e.g., inheritance of maternal antibodies). In many cases,  $R_0$  is simply  $R_0$  multiplied by the fraction of the host population that is susceptible,  $R_0 = R_0S_1/N$ . For microparasites,  $R_0$  is the number of hosts an individual infects over its infectious lifespan whereas for macroparasites,  $R_0$  is the number of adult offspring that a single adult parasite gives rise to over its lifespan.

A key aspect of pathogen transmission is whether and how this depends on host population density (Lloyd-Smith *et al.* 2005a). Transmission of a pathogen can increase with host density (linearly or non-linearly), which is termed density-dependent transmission (Figure 1a — the chance that each susceptible becomes infected is  $\beta I$  which is proportional to the density of infected hosts), the rate that the total number of susceptibles are infected per unit time is  $\beta SI$ ). A consequence is that for these density-dependent pathogens there will exist a threshold density ( $N_T$ ) of hosts below which transmission is inefficient and the pathogen cannot persist ( $R_0 < 1$ ) in that host population. This has important implications for control as discussed below.

Alternatively, transmission of a pathogen may be relatively unaffected by host density. Transmission of these pathogens is termed frequency-dependent (Figure 1b) because the force of infection — the per capita rate at which a susceptible individual becomes infected ( $\beta I/N$  for frequency dependent transmission or  $\beta I$  for density dependent transmission) — increases with the fraction of the host population that is infectious but does not increase with overall host density. As a result, there is no threshold density for pathogen invasion if transmission is frequency dependent. In theory, such pathogens can persist at very low host densities. Empirical work on mice, voles, ladybird beetles, frogs, and plants has shown that the transmission of most pathogens probably falls between these two extremes (Hudson *et al.* 2002).

The mode of transmission of a pathogen plays an important role in whether transmission is frequency or density dependent. Pathogens can be spread by direct contact (kissing can spread herpes viruses), aerosol (sneezing can spread influenza viruses), indirect contact (ingesting water contaminated with fecal material can cause infection with cholera bacteria), or by vectors (ticks and mosquitoes can spread viruses and bacteria between hosts). Transmission of pathogens spread by aerosol and water frequently increases with host density. In contrast, transmission of sexually transmitted pathogens (assuming rates of sexual contact don't necessarily increase with host density) and some vector-borne diseases is thought to be frequency dependent (Anderson & May 1991).

### Pathogen Impacts on Populations and Communities

Pathogen impacts on host populations (Figure 2) depend on several factors, including pathogen virulence, the reduction in host fitness (survival or reproduction) caused by the pathogen. The size of the impact also depends on whether the pathogen reduces host survival (parameter  $\alpha$  in the models in Figure 1a–c), reproduction (parameter  $\delta$ ), or both (as modelled in Figure 1c). In general, for pathogens that lower host survival, those with intermediate virulence tend to have the largest negative impacts on host populations. This is because hosts infected with highly virulent pathogens tend to die quickly, thus cutting short the infectious period of parasite transmission (see below for additional details on the evolution of virulence). For similar reasons, mathematical models also predict that sterilizing pathogens (those that reduce fecundity,  $\delta$ ) can cause greater reductions in host population size than those that reduce survival (Anderson & May 1981).

Pathogens that have density-dependent transmission (Figure 1a) can regulate host populations (i.e., reduce and hold them to a lower density than without the pathogen present) in the absence of any other density-dependent factors influencing host abundance. This requires that the mortality rate from the pathogen be greater than a value that depends on a combination of host survival, reproduction, recovery, and loss of immunity (Anderson & May 1979). There is growing empirical support for parasite-mediated host regulation (or altered host population dynamics) from study systems ranging from conjunctivitis in house finches to parasitic nematodes in red grouse and feral Soay sheep (Gulland 1992, Hudson *et al.* 1998, Hochachka & Dhondt 2000) (Figure 2).

In some cases, diseases caused by novel pathogens have caused dramatic host population declines, including chytridiomycosis in amphibians, chestnut blight in American chestnuts, avian malaria in Hawaiian birds, devil facial tumour disease in Tasmanian devils, and sudden oak death in Californian trees. Theory suggests that density dependent specialist pathogens (i.e., those infecting a single host) alone will rarely be able to drive their hosts extinct, although host numbers could be reduced to the point that stochastic extinctions occur. In contrast, pathogens are more likely to cause host extinctions if they exhibit frequency-dependent transmission, have with long-lived infectious stages, or are multi-host pathogens that can be transmitted between common reservoir hosts and more vulnerable target species (de Castro & Bolker 2005). These three traits allow for persistent transmission (and decreased host fitness) when a host is at low abundance.

One consequence of the reduced fitness caused by pathogens is that host species that can escape infection of a pathogen by emigrating to a new region may have significantly higher population growth rates and invasion potential (Torchin *et al.* 2003). For example, the European green crabs that colonized North America, likely by hitchhiking on ships, had only a small subset of the pathogens that infect them in Europe and are larger and more fecund as a result. Species like green crabs that are moved to new regions by humans are called "introduced" or "exotic" species. Introduced host species can also bring and introduce pathogens with them to new areas, with sometimes devastating effects on native host species, as exemplified by parapoxvirus of gray squirrels and resulting negative impacts on native red squirrels across the United Kingdom. Pathogens can also impact host species interactions in other ways that increase host community diversity, including preventing competitive exclusion and altering predation pressure (Bradley *et al.* 2008).

In humans and domesticated plants and animals, substantial efforts have been made to reduce pathogen transmission or to eradicate pathogens from populations altogether. The most commonly adopted strategies include culling (for animals, plants, and disease vectors), behavioral modifications including quarantine and social distancing, and vaccination. Culling is used when transmission is believed to be density-dependent, and efforts are made to reduce densities below the threshold density,  $N_T$ . Quarantines and social distancing are efforts to decrease contact rates between infectious and susceptible individuals and have been applied successfully for human pathogens, including SARS and HIV.

The third main strategy, vaccination, is an attempt to increase herd immunity — the fraction of the population that is immune to infection, either from prior exposure or vaccination. A frequent goal of vaccination is to vaccinate a large enough fraction of the host population to prevent invasion by or locally eradicate the pathogen. This critical vaccination threshold in a homogeneous population is  $1-1/R_0$  because this reduces  $R_0 = R_0S_1/N$  below 1. A key benefit from vaccination is that susceptible hosts, even though they themselves aren't vaccinated, are less likely to be infected because they are surrounded by immune individuals. In addition, recent work on protecting Ethiopian wolves against rabies virus has shown that even relatively low-coverage vaccination can reduce pathogen transmission and may prevent local host extinction.

**Host Heterogeneity**

Although most simple models (Figure 1a, b) assume all individuals are identical, substantial evidence has shown the existence of heterogeneity in host characteristics related to pathogen transmission. These include differences between hosts in susceptibility, contact rates, and infectiousness as well as spatiotemporal variability in host characteristics or the environment. This heterogeneity has large impacts on pathogen transmission and, as a result, on efforts to control disease. Studies of a number of parasites have shown that 20% of the host individuals are responsible for at least 80% of subsequent transmission. In the extreme, individuals that cause more secondary infections than expected (e.g., the 95th or 99th percentile of a Poisson distribution with mean  $R_0$ ) have been termed superspreaders (Lloyd-Smith *et al.* 2005b). Superspreaders have been identified for a variety of human diseases including SARS, HIV/AIDS, and measles. Clearly, the efficacy of control measures can be greatly enhanced if individuals that play dominant roles in transmission can be identified and targeted (e.g., for vaccination), as was shown in recent work on yellow-necked mice (Ferrari *et al.* 2004).

An important category of heterogeneity is that of multi-host pathogens that can be transmitted between several different host species. One group of these is zoonotic pathogens that are shared between humans and non-human animals. Many of the most important human-emerging infectious diseases, including HIV, influenza, and SARS arose (or continue to arise) from transmission from one or more animal species to humans. For these multi-host pathogens, some host species are amplifiers (their presence increases transmission of the pathogen) whereas others may dampen transmission or even prevent disease. This heterogeneity has been shown in the transmission of plant diseases (e.g., sudden oak death and Jarrah dieback, rooted in trees in Australia) as well as animal diseases (e.g., brucellosis, rinderpest, chytridiomycosis). One hypothesis that is actively being studied is the dilution effect, which posits that disease risk will decrease as the species diversity of hosts increases, and it may act through a range of possible mechanisms (Keesing *et al.* 2006). This dampening effect of host diversity can be especially important for vector-borne pathogens (Figure 1b) where vectors often take a limited number of bloodmeals. The best-studied examples are the Lyme disease bacterium *Borrelia burgdorferi* and West Nile virus (Figure 3), two pathogens that are transmitted between dozens of animal species by ticks and mosquitoes, respectively. If vectors feed more heavily ( $f_j$ ) on the most competent hosts (those with a high  $c_j$ ) in communities with fewer host species, this can cause a relationship consistent with the dilution effect. For example, in Lyme disease it is hypothesized that the presence of a diverse mammal and bird community reduces the fraction of ticks that feed on highly competent and permissive white-footed mice (Keesing *et al.* 2006).

During the past two decades, disease ecologists have demonstrated how spatial structure, dispersal patterns, and landscape-level heterogeneity can influence the spatial spread of pathogens. For example, researchers have examined how spatially localized movements can give rise to travelling waves of infection (a peak of infection followed by a trough) for pathogens such as rabies in raccoons, measles and influenza in humans, and baculoviruses in insects. In such cases, the rate of spatial spread depends strongly on the distribution of the dispersal kernel (i.e., the range and distribution of distances that infected individuals or pathogen particles spread). Other studies have examined host-parasite dynamics in the context of metapopulations, whereby hosts and pathogens can move among interconnected patches. Many of these studies have shown that a limited amount of host movement among patches can increase the long-term persistence of hosts and parasites, in part by allowing animals to escape from heavily parasitized areas.

### Host–pathogen Evolution: Virulence, Resistance, and Coevolution

Parasites and hosts frequently evolve in response to one another and to their changing environment. A key question in disease ecology concerns the evolution of virulence: Why do parasites harm their hosts (parameters  $\alpha$  and  $\delta$  in models in Figure 1), given that they depend on their hosts for their own transmission (Anderson & May 1982, Read 1994)? Conventional wisdom holds that parasites should evolve to become benign and therefore prolong the lives of the hosts they infect. However, many parasites cause substantial harm, partly because replication inevitably damages host tissues and consumes host resources. One explanation for the maintenance of virulence is that parasites too quickly too slowly will not produce sufficient transmission stages (which would reduce  $\beta$ ) whereas parasites that replicate too rapidly will kill their hosts before they can transmit. Thus, selection might favor parasites with intermediate levels of within-host replication (and hence, virulence) that balance the transmission benefits of higher replication with the costs of faster host death. In short, there is hypothesized to be a tradeoff between increasing  $\beta$ , which would increase transmission, and increasing  $\alpha$  which decreases transmission (Figure 1). Despite the popularity of this trade-off theory, other explanations for pathogen virulence exist, and only a handful of studies (including experimental work on rodent malaria, bacterial pathogens of *Daphnia*, and a protozoan parasite of monarch butterflies) have so far provided clear support for the trade-off theory.

Hosts can also evolve in response to infection in ways that influence virulence. The best observed examples of the host evolution in response to disease include studies of Australian rabbits and myxoma virus, crickets and parasitoid flies, and the bacteria and phages. Host strategies for combating infection can be grouped into two categories: host tolerance and host resistance (Boots *et al.* 2009). Host tolerance describes the ability of a host to tolerate infection with a pathogen by minimizing the damage done but without impeding replication or transmission of the pathogen. In contrast, host resistance strategies reduce the probability that a host is infected, reduce pathogen replication within the host, and/or increase the speed of pathogen clearance (recovery). Given that hosts would benefit from resisting infection, an outstanding question is, "Why aren't hosts more resistant to pathogens?" Potential explanations include: a trade-off between resistance traits other fitness-related traits, pathogen evolution to evade or counter host resistance traits, and trade-offs among defenses aimed at different parasite types or strains (Schmid–Hempel 2005). In contrast, traits that confer tolerance are frequently expected to evolve to fixation, assuming the benefits of these traits outweigh the costs.

Host–parasite interactions, thus, can lead to co-evolutionary dynamics that can increase the genetic diversity of both hosts and pathogens through co-speciation events and genetic arms races. One process that can lead to co-evolutionary change is negative frequency-dependent selection, whereby multiple host and parasite genotypes exist and only some host–parasite combinations result in infection. Over time, the frequency of resistant genotypes in a population can be affected by, and feed back to, local parasite genotype dynamics. This has been illustrated by long-term studies of trematode parasites infecting freshwater snails in New Zealand (Lively & Dybdahl 2000). This work also showed that high infection rates can ultimately favor host sexual reproduction as a strategy for generating novel host genotypes that may resist infection by common parasite clones.

### Future Directions

The growing frontier of disease ecology includes at least four important areas: interactions between pathogen species and strains (including the within-host dynamics of infection and interactions with the immune system), explicit consideration of the spatial context of transmission, dynamics and drivers of transmission of multi-host pathogens (Figure 1b), and evolution of hosts and parasites in the context of environmental change. The last three of these areas were touched on above.

Studies of interactions between different host species, both at the population-level and within individual hosts, have identified both immune-mediated and resource-competition-based mechanisms by which functionally distinct parasites can interact (Graham 2008). For example, immunological tradeoffs (energy devoted to one branch of the immune system that is used to fight off one kind of parasite cannot be used for another) have been shown to affect the outcome of infection by microbial pathogens for tubercle already infected by intestinal nematodes (Figure 1c). This tradeoff has been studied for bovine tuberculosis in African buffalo and tuberculosis and HIV infections in humans. In other cases, pathogens can dynamically interfere with each other's prevalence at the population-level even in the absence of co-infection or cross-immunity, as demonstrated for measles and whooping cough in some European cities.

A key challenge for disease ecology is to understand the transmission of pathogens between hosts while accounting for the many interacting factors discussed above, all within an environment that is warming, acidifying, urbanizing, and becoming far more connected.

### References and Recommended Reading

- Anderson, R. M. & May, R. M. Population Biology of Infectious Diseases I. *Nature* **280**, 361–367 (1979).
- . The population dynamics of microparasites and their invertebrate hosts. *Philosophical Transactions of the Royal Society of London Series B* **291**, 451–524 (1981).
- . Coevolution of hosts and parasites. *Parasitology* **85**, 411–426 (1982).
- . *Infectious diseases of humans: Dynamics and control*. Oxford University Press, London (1991).
- Boots, M., Best, A., *et al.* The role of ecological feedbacks in the evolution of host defence: what does theory tell us? *Philosophical Transactions of the Royal Society B* **364**, 27–36 (2009).
- Bradley, D. J., Gilbert, G. S., *et al.* Pathogens promote plant diversity through a compensatory response. *Ecology Letters* **11**, 461–469 (2008).
- de Castro, F. J., Bullock, B. Mechanisms of disease-induced extinction. *Ecology Letters* **8**, 117–126 (2005).
- Dobson, A. P. & Hudson, P. J. Regulation and stability of a free living host–parasite system — *Trichostrongylus tenuis* in red grouse. 2. Population models. *Journal of Animal Ecology* **61**, 487–498 (1992).
- Ferrari, N., Cattadori, I. M., *et al.* The role of host sex in parasite dynamics: field experiments on the yellow-necked mouse Apodemus flavicollis. *Ecology Letters* **7**, 88–94 (2004).
- Graham, A. L. Ecological rules governing helminth–microparasite coinfection. *Proceedings of the National Academy of Sciences USA* **105**, 566–570 (2008).
- Gulland, F. M. D. The Role of Nematode Parasites in Soay Sheep (Ovis–Aries L) Mortality During a Population Crash. *Parasitology* **105**, 493–503 (1992).
- Hochachka, W. M. & Dhondt, A. A. Density-dependent decline of host abundance resulting from a new infectious disease. *Proceedings of the National Academy of Sciences USA* **97**, 5303–5306 (2000).
- Hudson, P. J., Dobson, A. P., *et al.* Prevention of population cycles by parasite removal. *Science* **282**, 2256–2258 (1998).
- Hudson, P. J., Rizzoli, A., *et al.* *The Ecology of Wildlife Diseases*. Oxford, UK, Oxford Press (2005).
- Keesing, J., Holt, R. D., *et al.* Effects of species diversity on disease risk. *Ecology Letters* **9**, 482–498 (2006).
- Kilpatrick, A. M., Daszak, P., *et al.* Host heterogeneity dominates West Nile virus transmission. *Proceedings of the Royal Society B* **273**, 2327–2333 (2006).
- Lively, C. M. & Dybdahl, M. F. Parasite adaptation to locally common host genotypes. *Nature* **405**, 679–681 (2000).
- Lloyd-Smith, J. O., Cross, P. C., *et al.* Should we expect population thresholds for wildlife disease? *Trends in Ecology & Evolution* **20**, 511–519 (2005a).
- Lloyd-Smith, J. O., Schreiber, S. J., *et al.* Superspreading and the impact of individual variation on disease emergence. *Nature* **438**, 355–359 (2005b).
- Read, A. F. The evolution of virulence. *Trends in Microbiology* **2**, 73–76 (1994).
- Schmid–Hempel, P. Evolutionary ecology of insect immune defenses. *Annual Review of Entomology* **50**, 529–551 (2005).
- Torchin, M. E., LaFerty, K. D., *et al.* Introduced species and their missing parasites. *Nature* **421**, 628–630 (2003).

### BASIC

- The Diversity of Behavior
- What Functions of Living Systems Underlie Behavior?
- Both Environment and Genetic Makeup Influence Behavior
- How Does Social Behavior Evolve?
- An Introduction to Animal Communication
- Animal Behavior Introduction
- Mating Systems in Sexual Animals
- Measuring Animal Preferences and Choice Behavior
- Perceptual Worlds and Sensory Ecology
- Satellite Telemetry and its Impact on the Study of Animal Migration
- The Use of Biotelemetry in the Study of Animal Migration
- The Use of Radar in the Study of Animal Migration
- The Use of Stable Isotopes in the Study of Animal Migration

### ADVANCED

- An Introduction to Eusociality
- The Ecology of Avian Brood Parasitism
- Rapid Effects of Steroid Hormones on Animal Behavior
- Social Parasitism in Ants
- Causes and Consequences of Biodiversity Declines
- Disease Ecology

### INTERMEDIATE

- Animal Migration
- Cooperation, Conflict, and the Evolution of Complex Animal Societies
- Sexual Selection
- Territoriality and Aggression
- The Development of Birdsong