Biodiversity, Ecosystem Functioning, and Human Wellbeing

An Ecological and Economic Perspective

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OXFORD
UNIVERSITY PRESS

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Great Clarendon Street, Oxford OX2 6DP

Oxford University Press is a department of the University of Oxford.
It furthers the University's objective of excellence in research, scholarship,
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Oxford New York

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Kuala Lumpur Madrid Melbourne Mexico City Nairobi
New Delhi Shanghai Taipei Toronto

With offices in

Argentina Austria Brazil Chile Czech Republic France Greece
Guatemala Hungary Italy Japan Poland Portugal Singapore
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Published in the United States
by Oxford University Press Inc., New York

First published line 2009

British Library Cataloguing in Publication Data
Data available

Library of Congress Cataloging in Publication Data
Data available

Typeset by Newgen Imaging Systems (P) Ltd., Chennai, India
Printed in Great Britain
on acid-free paper by

CPI Antony Rowe, Chippenham, Wiltshire

ISBN 978–0–19–954795–1 (Hbk.)
ISBN 978–0–19–954796–8 (Pbk.)

10 9 8 7 6 5 4 3 2 1

CHAPTER 15

Biodiversity and ecosystem function: perspectives on disease

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15.1 Introduction

Experimental and comparative studies increasingly reveal that certain ecosystem functions are maximized in highly diverse ecological communities. Key among the ecosystem functions correlated with high biodiversity are rates of nutrient cycling, primary production, and resistance to disturbances such as drought (see Chapters 1 and 2). Despite considerable interest among ecologists in this dynamic area of research, we suspect that non-ecologists are unaware of or unimpressed by these functions served by high biodiversity. A 2007 survey shows that considerably more respondents in the European Union consider moral reasons stronger than utilitarian or economic reasons for protecting biodiversity (http://www.ec.europa.eu/public_opinion/archives/flash_arch_en.htm). In a 2002 survey in the USA (http://www.biodiversityproject.org/resourcespublicopinion.htm), utilitarian and economic functions of biodiversity, with the exception of human health, are not even mentioned. It would appear that the ecosystem functions typically addressed by biodiversity scientists are not among the principal concerns of many citizens when considering the consequences of biodiversity loss, even though there are increasing arguments for why we should be concerned (the Millennium Ecosystem Assessment (2005a) see Chapters 17–19). Globally, the Convention on Biological Diversity has been signed or ratified by the vast majority of the world's nations, committing them to preserve biodiversity in order to achieve sustainable development, but it has had little traction. Thus, there is a disconnect between public perception and governmental and scientific understandings.

The public, however, is interested in disease, both newly emerging and resurgent diseases, and it is here that the role of biodiversity may resonate with citizens. Recent studies have shown that high biodiversity can strongly reduce rates of disease transmission, and consequently that biodiversity loss can exacerbate disease risk. These studies themselves are diverse in the aspects of biodiversity under study, in the taxa of pathogens, hosts, and vectors involved, and in the mechanisms postulated. For example, high genotypic diversity within a species reduces transmission of bacteriophages that attack bacteria (Dennehy *et al.* 2007), microsporidians that attack *Daphnia* water fleas (Pulkkinen 2007), and fungi that attack rice plants (Zhu *et al.* 2000). On the other hand, high *species richness* reduces transmission of fungal pathogens of herbaceous plants (Roscher *et al.* 2007), viral pathogens of birds and humans (Ezenwa *et al.* 2006), helminth parasites of snails and vertebrates (Kopp and Jokela 2007), and bacterial pathogens of humans and other mammals (Ostfeld and Keesing 2000a, LoGiudice *et al.* 2003). Reduced structural diversity in tropical forests as a result of deforestation has been associated with higher risk of exposure to malaria (Vittor *et al.* 2006). High diversity can reduce disease transmission by reducing encounter rates between infected and susceptible hosts, and by regulating the abundance of species that are important for pathogen persistence, among other mechanisms (Keesing *et al.* 2006). Knowledge that biodiversity influences human health and that of valued wildlife and plants is likely to heighten interest among both scientists and non-scientists in the importance of biodiversity.

In this chapter we explore some of the major issues that have shaped recent explorations of the biodiversity-ecosystem function relationship and

apply these concepts to infectious diseases. These issues include: (1) the shape of the association between biodiversity and ecosystem function; (2) the relative importance of species biodiversity *per se* (e.g. species richness or evenness) vs species composition; (3) the relative importance of species biodiversity *per se* versus diversity of functional groups or relevant life history traits; (4) whether functions are performed better by the most diverse communities or by monocultures of the species with highest performance of that specific function; (5) how natural sequences of species loss under environmental change (community disassembly) vs. random sequences imposed experimentally influence ecosystem function; and (6) the importance of diversity at organizational levels other than (host) species in influencing ecosystem function. Because the empirical basis for exploring these particular issues is limited, we focus on describing each issue as it pertains to infectious disease and speculating on what might be found in future studies.

15.2 Shape of diversity–disease curves

When the performance of an ecosystem function increases linearly with increases in biodiversity, three related conclusions can be drawn. First, each species contributes approximately equally to the ecosystem function. Second, functional redundancy among species is weak. Third, the loss of biodiversity will result in reduced functioning, irrespective of initial diversity. In contrast, the performance of an ecosystem function can increase asymptotically with increasing biodiversity, implying that individual species contribute unequally, that some redundancy occurs (particularly in highly diverse communities), and that loss of biodiversity has a stronger impact on ecosystem functioning when the initial community is species-poor than when it is species-rich (Schwartz *et al.* 2000 and Chapter 1).

The shapes of curves relating some measure of disease dynamics (e.g. risk or incidence) to species richness have only rarely been measured or predicted, but the scientific basis for the relationships between biodiversity and these properties of ecosystems are the same for functions such as energy flow and nutrient cycling; each species contributes in some way, uniquely or redundantly, to risk or incidence. For West Nile virus incidence rates in humans (Ezenwa *et al.* 2006), and both prevalence and severity of rust fungal infection in perennial ryegrass (Roscher *et al.* 2007), biodiversity–disease curves were negative and apparently linear. In contrast, for Lyme disease risk (measured both as density of infected ticks and as tick infection prevalence), increases in biodiversity resulted in curvilinear decreases to an asymptote (Schmidt and Ostfeld 2001, Ostfeld and LoGiudice 2003). Dizney and Ruedas (in press) show a strongly asymptotic, negative relationship between mammalian diversity and prevalence of infection of deer mice with hantavirus. Other studies linking biodiversity and disease dynamics cannot address this issue because of insufficient variation in diversity; when comparisons are restricted to communities with one versus two or three species, curve shape cannot be estimated.

We suspect that, in some cases, the shape of the curve relating biodiversity to disease risk might be more complex than simple linear or asymptotic functions. In particular, for arthropod vectors or parasites with complex life cycles, some lower threshold level of diversity might be necessary for the disease to exist. This would occur if the vector or parasite requires several host species to fulfil its life cycle. Beyond this lower richness threshold, however, one might expect a negative relationship between diversity and disease transmission, leading to a unimodal distribution. We are not aware of specific tests of this hypothesis.

15.3 Diversity (richness) versus species composition

When individual species have strongly disparate effects on an ecosystem function, then the species composition (identity and relative abundance of all species) of a community is likely to be more important than the number of species or the quantitative value of a diversity metric such as the Shannon index or Simpson index. Much debate has surrounded the issue of how to contend with this issue (see Chapter 4). For researchers interested specifically in effects of species richness, the potential for species to have unequal effects is sometimes considered a complication to be minimized by carefully

designed experiments. Often, these researchers will generate experimental communities by randomly selecting species and creating replicate communities at each level of diversity, in order to account for sampling artifacts. Such a design is intended to reduce the probability that the random inclusion of species with particularly strong effects is responsible for the relationship between diversity and function (see Chapters 1 and 2). However, these randomly assembled communities might not represent real communities of interacting species. To contend with this possibility, researchers concerned with how natural variation in diversity causes natural variation in ecosystem functioning must attempt to constrain species composition to mimic natural patterns and determine the relative importance of richness versus composition.

We are aware of only one study that explicitly contrasted species richness and community composition in affecting disease dynamics. In this study, LoGiudice *et al.* (2008) assessed the causes of variation in the proportion of nymphal blacklegged ticks infected with the Lyme disease spirochete among 49 forest fragments in three northeastern USA states. They found that species richness was significantly, but weakly, negatively correlated with nymphal infection prevalence. However, when they predicted nymphal infection prevalence using a model that specifically incorporated species identity, their results were significant and strong. Qualitatively similar results were obtained in studies of foliar fungal pathogens on grassland plants (Knops *et al.* 1999, Mitchell *et al.* 2002, 2003). Although severity of fungal disease was negatively correlated with species richness, the effect of diversity on disease severity was indirect; plots with higher diversity had lower density of the most susceptible grass hosts, and disease severity was positively correlated with host density (Mitchell *et al.* 2002). As a consequence, it appears that community composition had a more direct impact on disease dynamics than did species diversity *per se*.

15.4 Species diversity versus functional or trait diversity

In many ecological communities, some species are strongly similar in their performance of particular functions (e.g. rate of resource depletion or of net primary production). As a consequence, researchers have begun to classify species into functional groups (e.g. grouping herbaceous plant communities into legumes, non-leguminous forbs, and grasses) and ask whether diversity of functional groups better explains ecosystem functions than does species diversity (e.g. Naeem and Wright 2003, Reich *et al.* 2004). Functional group classifications often are based on taxonomy or on physiology directly relevant to the ecosystem function of interest (i.e. they represent 'complementarity' of functions). In most cases, functional group diversity performs better than species diversity in explaining ecosystem functioning (Diaz and Cabido 2001, Hooper *et al.* 2005, Reich *et al.* 2004). However, the predictive precision of standard classifications of species into functional groups rarely is significantly higher than that of random classifications (Wright *et al.* 2006 and Chapter 4), and the most appropriate algorithms for classifying species into functional groups are not yet clear.

Comparisons of species diversity versus diversity in functional traits have not, to our knowledge, been made for studies of the effects of biodiversity on disease dynamics. Potentially, one could categorize hosts of a pathogen according to their abilities to support pathogen population growth and transmission (i.e. reservoir competence). Alternatively, one could categorize species in a community by trophic level relative to the predominant hosts for the pathogen. Such a categorization would allow researchers to ask whether diversity of natural enemies of the host, or of resources for the host, reduces or enhances disease transmission (Ostfeld and Holt 2004, Holt 2008).

Increasingly, there is a move to abandon functional groups and focus on species traits, or the characters of species that represent what they do, but disease ecologists are only now evaluating this approach. For example, host quality, reservoir competence, body size, longevity, reproductive rate, and other traits would cleanly identify species into their roles in particular processes, which seems far more useful than attempting to group species into discrete categories when most species overlap in what they do (see Chapter 4). Studies of plant traits abound, but the study of traits of heterotrophs

lags behind, and of pathogens and their hosts, even further behind (see Chapter 20).

15.5 Most diverse versus single highest functioning

Niche theory predicts that natural communities will consist of species that do not overlap completely in their suite of functions. One consequence of this expectation is that a diverse assemblage of species will tend to perform ecosystem functions more efficiently than will a monoculture, even if the species comprising the monoculture is more efficient than other single species. In other words, the functional complementarity arising from polycultures can cause them to out-perform all monocultures. Selection effects, however, in which one or a few species have disproportionate impacts on ecosystem functioning, may dominate and minimize the influence of complementarity. While there is strong evidence for complementarity in some studies, other experimental studies comparing performance of diverse communities to monocultures suggest that selection effects are stronger than complementarity (reviewed in Cardinale *et al.* 2006a). On the other hand, this pattern may also be due to limitations in the existing empirical studies and their interpretation. Cardinale *et al.* (2007) demonstrated that most of the reported plant-based studies actually do exhibit complementarity, or at least do not support the sampling effect alone. The resolution of this issue is important because the consequences for both the biodiversity-ecosystem function debate and the conservation of biodiversity are so profound.

Whether, or under what circumstances, monocultures can perform better than polycultures in influencing disease dynamics has not, to our knowledge, been assessed. Numerous examples of disease outbreaks in crop and livestock monocultures provide evidence that monocultures can have extremely high rates of disease transmission (reviewed in Keesing *et al.* 2006). But for certain disease systems, one might expect low diversity to be beneficial. For example, in the Lyme disease system in eastern North America, a small number of small mammals – white-footed mice, eastern chipmunks, short-tailed shrews, and masked shrews – are responsible for feeding and infecting a large percentage of the tick vector of the bacterial pathogen (Brisson *et al.* 2008). A large suite of other mammals and birds feed many ticks, but infect very few of them, and are considered 'dilution hosts' (Ostfeld and Keesing 2000). The dilution hosts differ in the degree to which they reduce the proportion of ticks infected, with grey squirrels being the most potent of the group (Ostfeld and LoGiudice 2003, Ostfeld *et al.* 2006). Simulation models indicate that a 'community' of dilution hosts consisting solely of grey squirrels would reduce disease risk to lower levels than would a naturally diverse community of dilution hosts (R. S. Ostfeld, unpublished). One caveat to generalizing from this result is that the performance of an ecosystem function, such as diluting disease risk, by a particular species is often contingent on the composition of the remaining community. For instance, shrews can act as a dilution host in some (species-poor) communities, but as an amplification host in other (species-rich) communities (Ostfeld *et al.* 2006). This is an example of context dependency that is well described in other studies of biodiversity-ecosystem functioning (Chapter 4). Therefore comparisons of performance in monoculture vs. polyculture must be made with caution. Even if monocultures of high-performing species outperform polycultures in diluting disease risk, applying this knowledge to real-world situations seems difficult or impossible. Returning to the Lyme disease example, replacing a diverse assemblage of vertebrates with a monoculture of squirrels seems neither practical nor desirable, even though it would reduce disease risk. Of course, these issues highlight the importance of defining what we mean by function. If the only function of interest is reducing risk of Lyme disease, then one non-permissive species might suffice, but if we are concerned with other diseases (i.e. other functional roles), then it is likely that greater diversity would be required.

15.6 Natural versus random sequences of community assembly or disassembly

A common approach to assessing the consequences of variation in biodiversity for ecosystem functioning is to assemble ecological communities by

drawing species at random from a species pool and correlate functioning with species richness. A less common approach is to remove species from intact communities, which is more appropriate in mimicking local extinction, but is far less tractable (Symstad *et al.* 2001, Diaz *et al.* 2003). The main purpose of such experimental designs is to specifically assess the importance of species diversity *per se*, while avoiding the potential for species diversity, species composition, and other factors to co-vary. Of course, such experiments can also focus on functional diversity or both, and often the two are correlated (Naeem 2002). Natural communities, however, appear not to be assembled or disassembled randomly (Zavaleta and Hulvey 2004, Bracken *et al.* 2008). More commonly, some species are widespread and highly resilient to disturbances, while others are more restricted in occurrence and sensitive to disturbances. As a consequence, some species will tend to occur in most or all communities from rich to poor, whereas others will tend to exist only in species-rich communities. Studies in which species are added or removed in random order cannot provide insight into the importance of natural sequences of species addition (community assembly) or loss (community disassembly) (e.g. Fukami and Morin 2002). Moreover, given that many directly transmitted diseases exhibit density-dependent transmission, the nature of the methodologies commonly used to assemble experimental diversity treatments is likely to confound simple richness effects. Additive designs, which maintain similar abundance of individual species in single and multiple species assemblages, act to increase overall density as richness increases, whereas substitutive designs, which maintain the same overall abundance in single and mixed assemblages, act to reduce density of individual species as richness increases. However, it is important to note that sequences of species assembly or disassembly are often unpredictable. For example, extinction debt theory argues that dominant species are sometimes highly sensitive to forces of extinction. Tests of theory will require approaches where communities are assembled in both random and orderly fashions.

In a recent study, Ostfeld and LoGiudice (2003) used a simulation model to disassemble virtual vertebrate communities either in random sequence or in sequences suggested by natural disassembly rules. The roles of each species in contributing to Lyme disease risk had been empirically parameterized (LoGiudice *et al.* 2003), allowing the aggregate contribution of each community to be estimated. Random selection of species for removal resulted in a strong, positive correlation between species diversity and disease risk. This result occurred because one species – the white-footed mouse – has a particularly large positive impact on Lyme disease risk, and more diverse communities had a higher probability of including white-footed mice. But when communities were disassembled under rules with some empirical support, the opposite pattern was obtained – lower diversity caused increased disease risk. This result occurred because white-footed mice exist in all communities irrespective of diversity (LoGiudice *et al.* 2008, Nupp and Swihart 1996), and so less diverse communities had mice but few other hosts capable of diluting the impact of mice (Ostfeld and LoGiudice 2003).

A key question in understanding the effects of biodiversity loss on disease dynamics, or any other ecosystem function, is whether commonness, ubiquity, or resilience are correlated with the performance of the function of interest. For disease systems, if dilution hosts tend to be common and resilient and reservoir hosts rare and sensitive, then decreases in diversity will reduce disease risk because species-poor communities will be characterized by many dilution hosts and few reservoirs. However, if reservoir hosts are common and resilient and dilution hosts tend to occur only in more species-rich communities, then decreases in diversity will strongly increase disease risk. These correlations have not been adequately addressed in zoonotic disease systems, although it appears that common and ubiquitous species tend to be the most competent reservoirs for zoonotic pathogens (Ostfeld and Keesing 2000b). Potential evolutionary explanations for the correlation between ubiquity and high reservoir competence have not yet been explored. Two possibilities are that: (1) pathogens that are likely to experience a broad range of hosts (e.g. because they are transmitted by a generalist vector) might tend to specialize on hosts they are likely to encounter frequently; and (2) hosts whose life history traits promote commonness and

resilience (e.g. rapid reproductive rate and short life span) might tend to tolerate infections (Jolles, Keesing, and Ostfeld, in litt.).

15.7 Other levels of biodiversity

Researchers assessing relationships between biodiversity and ecosystem functioning tend to focus on species diversity (and especially species richness), but biodiversity at other levels – genotypic, habitat type, landscape – can also potentially affect ecosystem functioning. These levels of diversity are only beginning to be studied in disease systems. Zhu *et al.* (2000) found that the incidence and severity of rice blast (a fungal pathogen) was considerably lower in agricultural fields with higher diversity of rice cultivars. Dennehy *et al.* (2007) found that two natural mutants of *Pseudomonas* bacteria were able to reduce prevalence of infection with bacteriophages compared with monocultures of the wild type. Neither mutant genotype was a viable host for the phage, and genotypic polycultures strongly diluted phage infection, sometimes to extinction.

Diversity at the genotypic level may also influence disease ecology. For example, Pulkkinen (2007) determined that the presence of > 1 genotype of *Daphnia magna* strongly reduced infection prevalence of a focal genotype with a parasitic microsporidian, *Glugoides intestinalis*. Another example is that of aphids and their fungal pathogens. Studies on susceptibility of pea aphids to a fungal pathogen have indicated significant genetic (between-clone) variability in fungal resistance (Ferrari *et al.* 2001). This suggests that a community with low genetic diversity comprising just the most resistant clone would result in the greatest reduction in disease risk. However, follow-up studies revealed significant Genotype × Environment interactions such that resistance ranking of individual clones changed with temperature (Blanford *et al.* 2003; Stacey *et al.* 2003). In this case, resistance under just one set of environmental conditions could be achieved with low genetic diversity, but to maintain resistance under environmental variation would require multiple clones. Further studies have now suggested that resistance to fungus is at least partly conferred by the presence of certain secondary bacterial symbionts within the aphids (Scarborough *et al.* 2005). How the symbionts differ between clones and how function is affected by environment is not clear, but the example illustrates the potential for considerable complexity in diversity–function relationships, even with just one host–pathogen combination.

Biodiversity can also be a factor *within* hosts. Recent studies have shown the potential importance of host coinfection with multiple disease agents. These mixed infections are common and have the potential to dramatically alter population dynamics and evolution of a particular disease agent (Cox 2001).

At the other end of the biodiversity spectrum, Vittor *et al.* (2006) found that decreases in tropical forest structural diversity that accompany deforestation strongly increase abundance and human-biting rates of the mosquito *Anopheles darlingi*, which is the primary vector of malaria in tropical America. Similarly, increases in yellow fever (Brown 1977), leishmaniasis (Sutherst 1993), and Ebola virus (Walsh *et al.* 2003) have all been linked to gross changes in habitat diversity and accompanying human encroachment on tropical forests increasing contact with key disease organisms and/or vectors. The exact nature of the diversity changes most potent in affecting disease transmission in these systems remains to be determined.

15.8 Looking forward

Ecologists have developed strong empirical and theoretical foundations for understanding the relationship between biodiversity and ecosystem functioning. Key issues include the shape of the relationships between biodiversity and specific ecosystem functions, the mechanisms underlying these relationships (e.g. the importance of species diversity per se versus species composition, functional traits), and how different patterns of community assembly affect functioning. Debates about these issues provide important insights for advancing our understanding of the effects of biodiversity on infectious diseases.

Considerable evidence is accumulating that biodiversity can strongly reduce disease transmission and risk (Dobson 2005, Rudolf and Antonovics

2005, Keesing *et al.* 2006, Dobson *et al.* 2006, Begon 2008, Molyneux *et al.* 2008). However, as we describe above, the issues of shape, mechanisms, and assembly patterns are only beginning to be addressed. Although we have limited our discussion to the effects of biodiversity on disease risk, we should also point out that diseases can strongly influence biodiversity.

Diseases play a fundamental (but largely unappreciated) role in shaping communities and mediating interactions between and across trophic levels. We cannot hope to fully understand the consequences of biodiversity change for almost any function without increasing our understanding of the role of disease organisms in community-level processes. Diseases can play important roles in natural regulation of pests and pathogens, provide new tools controlling them (e.g. Thomas and Read 2007), and can strongly influence the extent to which exotic plants and animals become damaging invaders in the introduced environment (Torchin *et al.* 2003; Mitchell and Power 2003).

Understanding the full relationship between biodiversity and disease risk will require exploration of both ecological and socio-economic factors (Fig. 15.1). Such exploration is particularly important in understanding the relationship between biodiversity and disease emergence (as opposed to tranmission dynamics of extant diseases). Risk is defined as the product of 'hazard × likelihood'. For emerging diseases, which are often accompanied by 'spillover' from a few principal host to secondary hosts, we suggest that 'hazard' might be represented by the prevalence of a particular pathogen and 'likelihood' represented by the probability or frequency of exposure to the pathogen. We further suggest that ecological change, such as loss of biodiversity or change in host species composition, is likely to have the greatest influence on the nature and magnitude of the hazard. On the other hand, many socio-economic factors (such as urban encroachment, land-use change, hunting, travel, and trade) broadly determine the chances of exposure, given a particular hazard. If we are to understand and manage disease risk, we need improved understanding of not just the ecology of infectious diseases, but also the social and economic contexts.

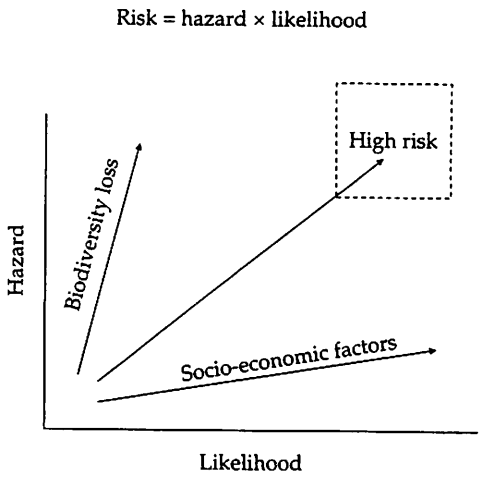

Figure 15.1 Conceptual model defining risk of an emerging infectious disease or disease spillover. Risk is the product of 'hazard × likelihood'. Ecological changes such as loss of biodiversity can affect the prevalence of a pathogen or parasite increasing disease hazard. However, transmission of the disease into a new population of hosts depends on frequency of exposure. For spillover of a zoonotic disease into a human population, likelihood of exposure is strongly determined by socio-economic factors.

Another research frontier is determining whether increases in vertebrate species diversity might increase the total burden of zoonotic disease. This might be expected if more vertebrate species simply add more zoonotic pathogens capable of infecting humans. Thus, even if risk of any particular disease declines with increasing vertebrate diversity, the total risk from all zoonotic pathogens might increase. Although such a relationship might exist, the evidence to support it is weak to non-existent. Interestingly, Jones *et al.* (2008) found that greater numbers of infectious diseases of humans have emerged in the temperate zone, where vertebrate diversity is lower, than in the tropics, where vertebrate diversity is higher. Although a detection bias partially accounts for this pattern, a simple correlation between vertebrate diversity and numbers of emerging diseases does not appear to exist.

Finally, one of the most important areas of biodiversity-ecosystem function research involves making it relevant to real-world problems, a main focus of this volume. As noted earlier, ecosystem functions like net primary productivity, nitrogen mineralization, and trace gas efflux do not

resonate with the public at large; nor do they readily translate into ecosystem services. Diseases, however, matter tremendously to humans, are well studied, and the economic costs and consequences of disease are well known. The role of biodiversity appears to matter in virtually all its levels, from genotypic to population to community to structural, and a variety of mechanisms are at play. In a world where both biodiversity is changing dramatically and diseases are emerging and resurging, understanding the role of biodiversity in the ecology of diseases is arguably one of the most important areas in biodiversity-ecosystem function research.