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CHAPTER FOURTEEN

Research Frontiers in Ecological Systems: Evaluating the Impacts of Infectious Disease on Ecosystems

Sharon L. Deem, Vanessa O. Ezenwa, Jessica R. Ward, and Bruce A. Wilcox

SUMMARY

AMONG BIOLOGISTS, THE AWARENESS OF INFECTIOUS DISEASES AND their population-, community-, and ecosystem-level impacts has increased dramatically over the past decade. This growth in interest seems to have paralleled an unprecedented global rise in the appearance of new pathogens, the spread of old pathogens, and changes in the pathology of many infectious agents in both humans and wildlife. However, the paucity of historical data makes it difficult to assess whether these current patterns represent a new or unique threat to ecosystem health. To evaluate the level of threat infectious diseases may pose to ecosystems, it is important to examine if, how, and why the impacts of diseases are changing in the modern world. Records of disease outbreaks in humans and livestock go back thousands of years, but relatively little historical information exists about infectious disease dynamics in natural ecosystems. As such, a fundamental question for disease ecologists is whether the ecosystem-level impacts of wildlife diseases are increasing on a global scale. In addition, given the mounting evidence implicating anthropogenic environmental changes in the emergence of various human infectious diseases, a second critical question is whether humans also contribute to the increased emergence and impact of infectious diseases in natural ecosystems. In this chapter we explore both questions by evaluating existing evidence from the literature and outline a research agenda for moving the field of disease ecology toward more definitive answers to these pressing questions. The research agenda we propose includes retrospective analyses of existing data on plant, animal, and human diseases to explore historical patterns of pathogen distribution, diversity, and prevalence; prospective long-term research projects to monitor infectious diseases in natural populations and communities; and experimental testing of probable drivers of disease emergence. For this research agenda to meet the ultimate objective of an increased capability to forecast disease outbreaks and to mitigate the negative impacts

of these diseases, it must have multiple, integrated foci, each of which contributes to the understanding of wildlife management, ecosystem conservation, and human health in our changing world.

Introduction

Among biologists, the awareness of infectious diseases and their population-, community- and ecosystem-level impacts has increased dramatically over the past decade (Daszak et al. 2000; Deem et al. 2001; Harvell et al. 1999; Woodroffe 1999). This growth in interest is vividly reflected in an almost exponential increase in the number of publications referring to "emerging infectious diseases" over the past fifteen years (figure 14.1), and seems to have paralleled an unprecedented global rise in the appearance of new pathogens, the spread of old pathogens, and changes in the pathology of many infectious agents in both humans and wildlife. Although recent attention to high-profile disease outbreaks in wildlife such as West Nile virus (birds in North America), Ebola hemorrhagic fever (gorillas and chimpanzees in Central Africa), canine and phocine distemper (African lions and seals in northern Europe), and chytridiomycosis (amphibians worldwide) might imply that diseases are

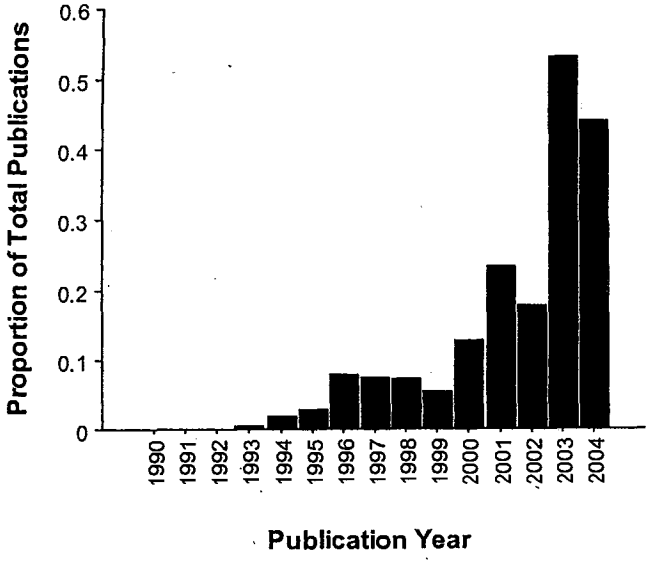

Figure 14.1. Proportion of publications (×1,000) cited in PubMed using the term "emerging infectious diseases" (1990–2004).

also having a larger impact on ecological systems, the paucity of historical data makes it difficult to assess whether these current patterns represent a new or unique threat to ecosystem health.

To evaluate the level of threat infectious diseases may pose to ecosystems, it is important to examine if, how, and why the impacts of diseases are changing in our modern world. Records of disease outbreaks in humans and livestock go back thousands of years (Fleming 1871, 1882; McNeill 1976), but relatively little historical information exists about infectious disease dynamics in natural ecosystems. As such, a fundamental question for disease ecologists is whether the ecosystem-level impacts of wildlife diseases are increasing on a global scale. In addition, given the mounting evidence implicating anthropogenic environmental changes in the emergence of various human infectious diseases (Institute of Medicine 2003; McMichael 2001; Patz et al. 2000, 2004), a second critical question is whether humans also contribute to the increased emergence and impact of infectious diseases in natural ecosystems. In this chapter we explore both questions by evaluating existing evidence from the literature and outline a research agenda for moving the field of disease ecology toward more definitive answers to these pressing questions. Throughout this chapter we use the term infectious disease broadly to encompass a variety of parasites and pathogens, including viruses, bacteria, protozoa, fungi, helminths, arthropods, prions, and the diseases that they cause.

Are the Impacts of Infectious Diseases Increasing in Natural Ecosystems?

Ecosystem properties are a function of the composition, distribution, and abundance of organisms in the system. The functional characteristics of species influence ecosystem properties through their roles as keystone species and ecological engineers, and through their interactions with other species, including infectious agents (Hooper et al. 2005). Disease impacts can occur on multiple scales, affecting individuals (fitness costs, mortality), populations (population size, gene flow), communities (shifts in dominant or abundant species, changes in species composition), and ecosystems (changes in ecosystem structure, function, and resilience). Disease can change the face of landscapes by removing keystone, abundant, and endangered species, or ecosystem engineers (Mouritsen and Poulin 2002; Collinge et al., chapter 6, this volume). The impacts of disease may also be less obvious if functional redundancy within an ecosystem allows one species to compensate for the loss of another, making it difficult to assess disease impacts at the ecosystem level. Thus, to deter-

mine whether disease impacts are increasing, a preliminary question that needs to be addressed is whether the prevalence of infectious diseases is increasing in natural systems.

The perception that disease has increased in recent years is difficult to confirm, owing to a lack of historical disease data for most systems. However, one example of historical data is a recent analysis of infectious diseases in marine organisms. Focusing on infectious disease reports in the literature between 1970 and 2001, this analysis showed that reports of disease, normalized by publication rates, increased in five of nine marine taxonomic groups, supporting the perceived increase in disease in those groups (Ward and Lafferty 2004). The analysis did not address the impact disease had on the affected populations and ecosystems, in part because disease has multiple impacts that are difficult to quantify and compare among different systems. However, several case studies suggest that when the prevalence of disease increases, the ecological impacts of disease are more severe. For example, wasting disease, caused by the slime mold *Labyrinthula zosterae* (Muehlstein et al. 1991), decimated *Zostera marina* eelgrass populations in the 1930s (Muehlstein et al. 1988). Loss of *Z. marina* precipitated the only known historical extinction of a marine invertebrate, the limpet *Lottia alveus* (Carlton et al. 1991). A more recent example is the shift from coral- to algal-dominated reefs in some areas of the Caribbean. Overfishing of herbivorous fishes, combined with disease-mediated mortality of the remaining abundant herbivore, the long-spined sea urchin, *Diadema antillarum*, released macroalgae from grazing pressure (Hughes 1994; Jackson et al. 2001; Lessios 1988). In the absence of herbivory, macroalgae increased at sites once dominated by coral (Hughes 1994).

The most noticeable impacts of disease are usually associated with epizootics (diseases that increase in incidence and prevalence suddenly and quickly) because they often have rapid, large, and unprecedented effects. The relative impact of epizootics on individuals, populations, communities, and ecosystems depends on the organisms affected (whether a consumer, a resource, or both) and on the preepizootic state of the system (i.e., how perturbed or resilient the system was). In contrast, enzootic or endemic pathogens—those with relatively constant prevalence within a population—often occur in a stable state, with less obvious effects on ecosystems. However, enzootic pathogens can act as important regulators of populations (Hudson et al. 1998; Kohler and Hoiland 2001; Tompkins and Begon 1999), and shifts in prevalence may lead to important ecosystem-level changes.

Enzootic and epizootic diseases have impacts on scales from individuals to ecosystems. The relevant question is how to assess these impacts to determine whether they are changing. This task is complicated by a

lack of historical baseline data and the complexity of quantifying and comparing impacts across ecosystems. Because humans increasingly dominate the world's ecosystems (Vitousek et al. 1997) and are a major driver of changes in ecosystem processes and resilience through declines in species diversity and alteration of community composition (Chapin et al. 2000), basic ecological principles suggest that the impacts of many infectious diseases will likely increase with human alteration of natural ecosystems. However, at this point it is still unclear if and how much humans are currently influencing infectious disease dynamics in natural systems.

Are Humans Contributing to Changes in the Impacts of Infectious Diseases on Natural Ecosystems?

Many human diseases classified as emerging infectious diseases originate from human-modified ecosystems (Despommier et al. 2006). For example, the reemergence of malaria and yellow fever is associated with fragmented forests, increases in the prevalence of schistosomiasis, cholera, and leptospirosis are linked with agricultural irrigation and dam building schemes (Gubler 1998; Patz et al. 2000), and spillover of Nipah virus from its reservoir host to humans may be associated with deforestation and intensified pig farming (Chua 2002). Modified ecosystems can provide new habitat for disease vectors or reservoirs, as in the case of mosquito-borne diseases, or facilitate vector dispersal or mechanisms of pathogen transport via terrestrial or aquatic habitat corridors. Furthermore, ecotonal processes, which involve the concentration and intensification of activities and processes associated with pathogen host switching, spread, and adaptation, can also drive infectious disease emergence (see Chapin et al., chapter 13, this volume). Since the current global phenomenon of increasing emerging infectious diseases can be explained largely as a consequence of anthropogenic changes affecting ecosystems at regional and landscape scales (figure 14.2), anthropogenically driven pathogen emergence events that result in human disease are likely only a subset of similar events affecting wild species.

Human activities that have been hypothesized as drivers of disease emergence in humans and wildlife generally fall into one of four categories: (1) environmental (e.g., climate change, deforestation, habitat fragmentation) and ecological change (e.g., biodiversity loss); (2) shifts in human demography (e.g., population growth and movement, urbanization); (3) increased global travel and trade (e.g., air travel, movement of livestock, pet trade, introduced species or pathogens, ballast water); and (4) technological and agricultural practices (e.g., changes in food

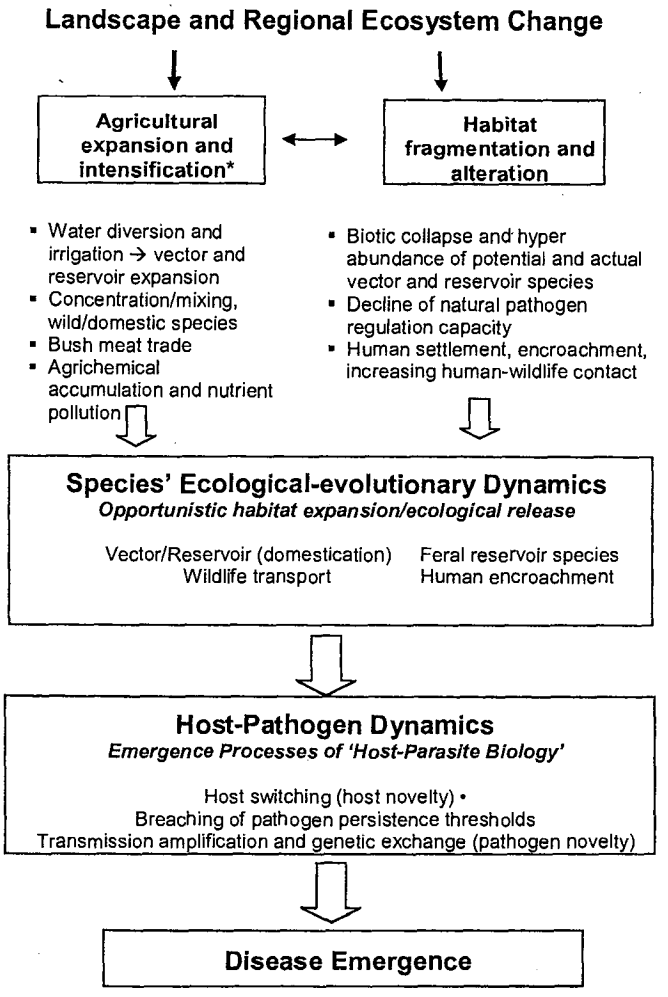

Figure 14.2. Landscape and regional ecosystem alteration and pathogen emergence. Undisturbed natural ecosystems maintain relatively complete ecological assemblages in which component species and ecological processes are regulated and the effects of extreme environmental events are buffered by the system's inherent resilience. Human encroachment on natural habitat and wildlife encroachment on domestic habitats, along with the associated exposures, facilitates spillover and spillback of pathogens. The coalescing of human and animal hosts and of reservoir and vector species within altered ecosystems, and the movement, shifting, and mixing across the ecosystem continuum, both affect host-pathogen dynamics in a manner that facilitates disease emergence. The result is increased opportunities for host switching (pathogen novelty), the breaching of pathogen persistence thresholds (owing to unprecedented host

natural systems, how can these less obvious disease impacts be identified and assessed?

Since ecosystems were not historically monitored to detect pathogen emergence, one approach to addressing this question is to ask whether anthropogenic changes affect the conditions known to underlie or favor altered host-pathogen relationships. For example, ecological theory suggests a number of mechanisms by which human disturbances can contribute to pathogen emergence. Preeminent among these is the effect of landscape modification on the spatial distribution, composition, and abundance of species in a community. Human activities such as deforestation, the use of pesticides, and omission of various forms of pollution often result in the loss of predators. In fact, carnivorous mammals typically are the first species to disappear following forest fragmentation (Harris 1984; Laurance and Bierregaard 1997). Local extinction represents the loss of top-down natural control in ecological communities that can result in an increase in abundance of other species (Laurance and Bierregard 1997; Terborgh et al. 2001). Reduction of species diversity by whatever means (pollution, pesticides, habitat loss, nonsustainable harvesting) can contribute to the phenomenon of ecological release in remaining species, whose natural predators, competitors, or parasites are reduced in number or eliminated. Some of the released species may be reservoirs or vectors of zoonotic pathogens. If so, ecological release may result in the proliferation of released species and an increased prevalence of the pathogens they carry (Summers et al. 2003).

Climate change is another human-induced environmental change with the capacity to alter interactions between native hosts and their endemic pathogens. A major prediction regarding the effects of climate change on infectious disease is that climate warming can accelerate parasite development times and transmission rates, changing the abundance or prevalence of many endemic pathogens (Kovats et al. 2001; Harvell et al. 2002). As an example, warming temperatures in the Canadian Arctic over the last twenty years may account for the increased prevalence and intensity of the lungworm *Umingmakstrongylus pallikuukensis* in native musk oxen (Kutz et al. 2004). While *U. pallikuukensis* is thought to be an endemic parasite of musk oxen in the region, increasing parasite development rates within intermediate gastropod hosts un-

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population densities), and transmission amplification and increased rates of pathogen adaptation and evolution of immune system detection avoidance, pathogenicity, and infectiveness (owing to increased opportunities for the interaction of endemic infection cycles and of pathogen strains, and the greater density and genetic variability of pathogen populations). (Adapted from Wilcox and Gubler 2005.)

der warmer summer conditions could be driving the recent emergence of this parasite. Selective declines in musk oxen populations infected with the parasite suggest that these changes in host-parasite dynamics may be having important population-level impacts, with as yet unknown ecosystem-level ramifications.

Although it seems likely, based on the circumstantial evidence, that humans are contributing to the effects of diseases on natural systems, we still lack direct evidence relating specific human activities to increasing prevalence, pathogenicity, or ecosystem-wide impacts of disease. In the same way, our sense that infectious diseases are increasing in prevalence and impact broadly remains largely speculative, since detection and surveillance also have increased substantially. To begin addressing these uncertainties, in the last section of this chapter we lay out a research agenda that can help guide the field of disease ecology toward answering the fundamental questions raised in this chapter.

An Integrated Research Agenda for the Future of Disease Ecology

To advance the current state of knowledge in infectious disease ecology, an interdisciplinary approach is needed in which the causes and consequences of disease emergence and responses to disease outbreaks are viewed from multiple perspectives. Key issues to be addressed include ascertaining whether infectious diseases are increasing globally, determining what the main drivers and mechanisms are for disease emergence, understanding the role of parasite and pathogen dynamics in population, community, and ecosystem ecology, and identifying associations between and among wildlife, domestic animal, ecosystem, and human health. Collaboration among professionals with unique skills is critical. Ecologists, epidemiologists, microbiologists, immunologists, and others are needed to study hosts, pathogens, and their interactions at various scales. Veterinarians, medical doctors, and public health officials are needed to explore links among wildlife, human, and domestic animal health. In addition, sociologists, economists, and politicians are required for translating scientific findings into tenable policies that mitigate the impacts of infectious diseases on natural ecosystems and human societies.

A research agenda intended to meet these objectives should have multiple, integrated foci, each of which contributes to the understanding of wildlife management, ecosystem conservation, and human health in our changing world (figure 14.3). An understanding of historical disease dynamics is essential (figure 14.3, step 1). To do this, existing data on plant, animal, and human diseases must be compiled to explore histori-

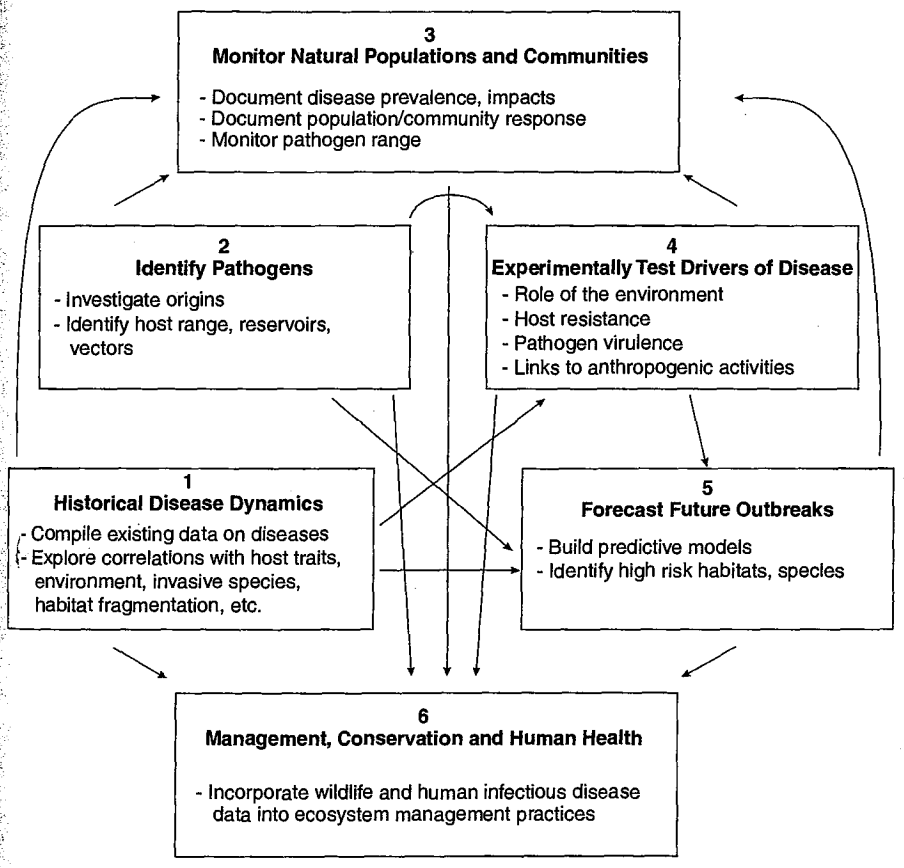

Figure 14.3. An integrated research agenda combining (1) retrospective studies of historical disease dynamics, (2) identification of pathogens and their hosts, (3) monitoring of natural populations and communities to document current disease incidence and prevalence, impacts, and range of pathogens, (4) experiments to test drivers of disease outbreaks (including the role of the environment in host resistance and pathogen virulence), and (5) theoretical ecology to build predictive models and forecast future outbreaks. This integrated research agenda allows implementation of preventive measures to mitigate the negative impacts of infectious diseases. Research clusters are interconnected, providing data and methods to further knowledge in other areas. All approaches feed into an overarching goal of management and conservation of natural resources.

processing, antibiotic use, air and water pollution, intensified agriculture and animal husbandry, overfishing) (Dobson and Foufopoulos 2001; Jackson et al. 2001; McMichael 2004; reviewed in Morse 1995; Patz et al. 2000). These anthropogenic processes can directly and indirectly affect disease emergence by introducing new pathogens into naïve ecosystems, by decreasing ecosystem resilience, making ecosystems more vulnerable to pathogen invasion, or by changing enzootic host-pathogen interactions (reviewed in Kim et al. 2005). In all these cases, the ecosystem-level effects of diseases are also likely to be changing. For this reason, it is important to develop a better understanding of the associations between and among anthropogenic drivers, disease emergence, and potential ecosystem-level consequences.

A key anthropogenic driver of disease emergence is the direct introduction of pathogens into new geographic areas or hosts as a result of the movement of livestock, wildlife, plants, or pathogens (Anderson et al. 2004; Daszak et al. 2000). A classic example is the introduction of smallpox into the New World by the Spanish. The disease caused mass mortality events among Native Americans, effectively reshaping the trajectory of the human ecosystem in North America (Diamond 1997). Similarly, human activities introduced many nonhuman diseases into new areas, with important consequences for natural ecosystems. Rinderpest, introduced into Africa in the late 1800s as a result of the movement of cattle, caused massive declines of native ruminant species, with cascading effects on predator populations and woodland structure (Plowright 1982; Prins and Van der Jeugd 1993). More recently, the international trade in *Xenopus* frogs from Africa beginning in the 1930s was suggested as a mechanism for the global dissemination of chytrid fungus (Weldon et al. 2004), a pathogen implicated in amphibian population declines worldwide (Daszak et al. 1999; Retallick et al. 2004).

Large-scale disease-induced population declines and species's extinctions can have clear ecosystem-level effects ranging from the disruption of trophic interactions to the loss of functional redundancy and ecosystem resilience (Chapin et al., chapter 13, this volume). The rinderpest and chytrid fungus examples illustrate the extent to which humans can contribute to the ecosystem-level impacts of infectious disease. However, not all disease emergence events lead to massive die-offs, especially when emergence is not associated with the introduction of a novel pathogen into naïve host populations. In many cases the anthropogenic processes driving disease emergence in wildlife may simply modify existing host-pathogen relationships, leading to subtle changes in pathogen prevalence or pathogenicity that have less obvious effects at the individual, population, or community levels. Given the lack of information on historical distribution, diversity, and prevalence of endemic pathogens in

cal patterns of pathogen distribution, diversity, and prevalence. Such analyses can contribute important baseline data on disease distributions in natural populations, suggest underlying processes accounting for these baseline patterns, and help reveal when and why disease patterns deviate from the baseline. Existing efforts using historical data have shown that this is a useful approach that should be expanded. Sources such as the Global Mammal Parasite Database (http://www.mammalparasites.org), a free online resource for infectious disease records in wild mammals (Nunn and Altizer 2005), illustrates the range of disease data already available in the literature and the various questions that can be addressed with such data. Also, analyses such as the one described earlier in this chapter by Ward and Lafferty (2004) show that quantitative syntheses of current data can illuminate important patterns and focus attention on future priorities.

As we collect and collate data on patterns of infectious diseases among hosts, we must remember that parasites and pathogens themselves are an integral part of ecosystems (Horwitz and Wilcox 2005). As such, it is important to document the diversity and distribution of pathogens occurring in natural systems and understand their role in shaping ecosystem processes (figure 14.3, step 2). The diversity of currently described pathogens is probably a significant underestimation of true pathogen diversity. With recent advances in molecular techniques, opportunities for identifying new parasites, exploring the phylogenetic relationships among parasites, and understanding patterns of host specificity are increasing and will be critical for understanding factors that drive disease emergence. For example, although climate change and nutrient enrichment are often hypothesized as drivers of marine diseases, experimental tests of causation are rarely possible because of the rapid and ephemeral nature of some disease outbreaks (Cerrano et al. 2000; Harvell et al. 2001) and the difficulty in isolating, identifying, and culturing the causative pathogens (Richardson 1998). Advances in parasite identification techniques will help overcome these important challenges.

Another important step is to initiate long-term research projects to monitor infectious diseases in natural populations and communities (figure 14.3, step 3). Prospective studies focused on documenting disease prevalence and incidence across hosts and habitats and tracking routes of disease spread will provide ecological and epidemiological data to predict, and possibly prevent, future disease outbreaks. Such studies would also monitor species-, community-, and ecosystem-level responses to disease, providing information on the role pathogens play in ecosystem dynamics. These monitoring projects must involve multiple disciplines to build a complete understanding of the genetic, immunological, ecological, and social implications of infectious disease. Although long-term disease monitoring is rare, platforms such as the National Ecological Observatory

Network (www.neoninc.org) pave the way for such undertakings. In addition, many existing human disease surveillance and long-term land use and cover change projects are well poised to contribute to this effort by incorporating wildlife disease surveillance into existing project protocols.

Despite the various hypotheses regarding drivers of disease emergence, obtaining direct evidence on environmental and anthropogenic factors influencing disease remains a significant hurdle for the field of disease ecology. The three steps outlined above will facilitate experimental testing of drivers of disease emergence, the next critical step in an integrated research agenda (figure 14.3, step 4). As more detailed information on historical patterns of disease distributions, parasite characteristics, and population-, community-, and ecosystem-level responses to infectious diseases becomes available, we will be able to test explicit hypotheses on the role of climate change, biodiversity loss, pollution, habitat fragmentation, and other factors in disease emergence. Experimental approaches will also provide information on pathogen virulence across hosts and levels of host resistance across species and habitats. These data, combined with long-term monitoring, will strengthen our ability to make predictions about future disease outbreaks.

An increased capability to forecast disease outbreaks is a tangible benefit of the integrated research approach described (figure 14.3, step 5). Predictive models must be built using accurate data for parameters of interest. Both retrospective and prospective studies to gather these data will be instrumental in designing these models. We will have the ability to identify species and habitats most vulnerable to the impacts of infectious disease, and the types of pathogens and parasites that pose the greatest threat. In turn, these data inform management, conservation and public health strategies to minimize the impacts of infectious diseases on wildlife, ecosystem, and human health. Ultimately, our ability to mitigate negative impacts of infectious diseases depends on interdisciplinary collaboration and the development of creative and integrative solutions. For example, it is important to recognize that pathogens are natural components of ecosystems that help shape various ecological processes. Thus, an important management challenge will be to devise strategies whereby natural interactions between pathogens and their hosts are conserved, while minimizing the effects of emerging infectious diseases.

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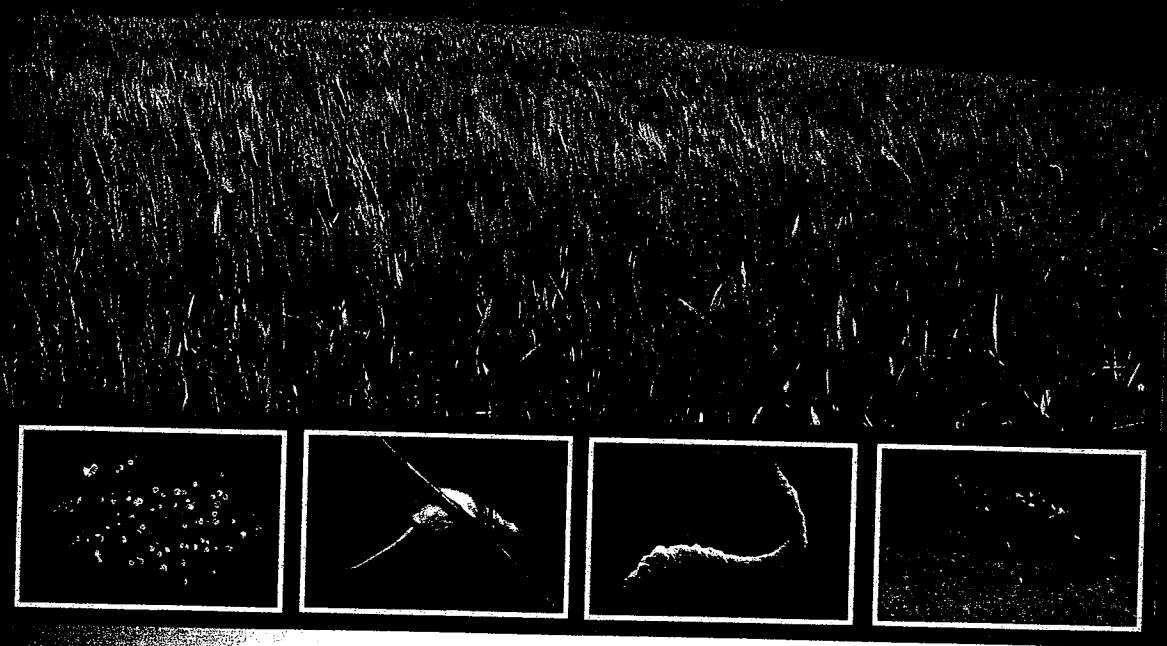
Infectious Disease Ecology

Effects of Ecosystems on Disease and of Disease on Ecosystems



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