

# Emerging Infectious Diseases of Wildlife and Species Conservation

G. MEDINA-VOGEL

Facultad de Ecología y Recursos Naturales, Universidad Andrés Bello, República 440, Santiago, Chile

ABSTRACT There has been an increase in the emergence and reemergence of human infectious diseases on a global scale, and zoonotic diseases in which wildlife serves as the reservoir are a large contributing factor. Faced with this situation, there is a necessity to create integrated prevention strategies and predictive models to determine the sites most vulnerable to the emergence of new zoonoses. Cases have been documented in which pathogens responsible for infectious diseases in wild species have been readily transmitted between hosts and have threatened vulnerable declining populations. Habitat destruction and man-made changes in the landscape together with the introduction of alien species are significant environmental variables that affect the ecology of infectious diseases. Thus, the loss of biodiversity is illustrated to be related to both the emergence of new or the exacerbation of existing vectorborne zoonotic diseases through mechanisms such as the loss of the dilution effect and ecological release and simplification. Hence, it is important to consider this factor when assessing disease risk and disease prevention in domestic animals and humans. Diseases like leptospirosis in which water plays an important role are ecosystem health diseases; in fact, the reported higher prevalence of Leptospira spp. in river otters in southern Chile compared with species less adapted to aquatic environments and with terrestrial domestic carnivores provides evidence that man-made landscape alterations, including the introduction of alien species, has exacerbated the transmission and prevalence of leptospirosis in wildlife and thus the risk of human infection.

### INTRODUCTION

Humans are rapidly transforming whole ecosystems in a number of well-documented but often poorly understood ways (1). Growing human populations and changes in land use patterns have increased contact among humans, domestic animals, and wildlife, raising the risks of transmission of numerous pathogens from

animals to humans and vice versa (2, 3). Diseases are often transmitted between wild and domestic species, as well as from invasive species into resident populations (4, 5). Emergence of new infectious diseases frequently results from a change in ecology of host or pathogen (6), and when these relationships are disrupted, ecological effects may extend to many other parts of the ecosystem (Z). The increase in human activities has had tremendous environmental impacts on biodiversity, including habitat loss, introduction of alien species, eradication of native species, pollution, urbanization, and anthropogenic climate change. Each of these environmental disturbances affects the ecology of infectious diseases (3).

Biodiversity is defined as the variety of life on Earth at all levels of biological organization, from genes within populations of species to species composing communities that are the biological components of ecosystems ( $\underline{8}$ ). Biodiversity may be related to infectious diseases at any of the following levels: the genetic variation of pathogens, vectors, and hosts; the number of species within each of these groups; the competition between species; the diversity of habitats in an ecosystem; or changes in animal behavior ( $\underline{9}$ ). Emerging in-

Received: 9 December 2012, Accepted: 15 December 2012, Published: 13 December 2013. Editors: Ronald M. Atlas, University of Louisville, Louisville, KY, and Stanley Maloy, San Diego State University, San Diego, CA Citation: Medina-Vogel G. 2013. Emerging infectious diseases of wildlife and species conservation. *Microbiol Spectrum* 1(2):OH-0004-2012. doi:10.1128/microbiolspec.OH-0004-2012. Correspondence: G. Medina-Vogel, gmedina@unab.cl

© 2013 American Society for Microbiology. All rights reserved.

fectious diseases of wildlife are generally related to habitat loss and fragmentation, overexploitation, introduction of invasive alien species, environmental pollution, and anthropogenic climate change (3, 10– 13). There are many examples of emerging infectious diseases that have been clearly driven by direct human interventions that have altered exposure to pathogens and facilitated the transmission of disease. Moreover, the globalization of agriculture, commerce, and human travel has rapidly disseminated emerging diseases around the globe (14).

## EMERGING INFECTIOUS DISEASES OF WILDLIFE

Emerging infectious diseases have been increasingly reported as a cause of death and population declines of free-living wild animals (10). In 1988, an outbreak of phocine distemper virus in the European harbor seal (Phoca vitulina) was stimulated by the forced southern migration of infected harp seals (Phoca groenlandica) due to human depletion of their food stocks by overfishing, coupled with compromised immunity caused by pollution. This outbreak killed 18,000 harbor seals throughout the North Atlantic European coasts (15-17). There are a number of examples of emerging infectious diseases of wild terrestrial and marine fauna occurring in Antarctica, as recent evidence indicates that some microorganisms may have been introduced to Antarctic wildlife as a consequence of human activity (18, 19). Disease has been recorded or suspected in several unusual mortality events of Antarctic birds, such as avian cholera caused by infection with Pasteurella *multocida* (20). The disease has also been observed on more than one occasion on sub-Antarctic Campbell Island, where P. multocida has been isolated from dead rockhopper penguins (Eudyptes chrysocome) (21), and several hundred gentoo penguin chicks (Pygoscelis papua) were found dead on Signy Island, Antarctica (22). In addition, bursal disease virus, a pathogen of domestic chickens, has been identified in Adélie penguins (*Pygoscelis adeliae*) (23). These data highlight the threats to penguins posed by introduced pathogens. Weimerskirch (24) demonstrated that the worldwide spread of avian cholera is probably the major cause of the decline of the large yellow-nosed albatross (Diomedea chlororhynchos) on Amsterdam Island as well. Another pathogenic bacterium, Erysipelothrix, was also implicated. Infectious diseases in Antarctica have also been recorded among other taxa. At least 1,500 crabeater seals (*Lobodon carcinophagus*) were found dead in the Crown Price Gustav Channel, Antarctic Peninsula, in 1955 (25). All affected seals had swollen necks and blood running from their mouths; on dissection their intestines were empty, their livers were pale, and pus oozed from the neck glands when incised (26). The cause was suspected to be a highly contagious virus possibly exacerbated by stress from crowding and partial starvation as a result of being trapped by ice. Abiotic factors also affect the presence, distribution, and transmission of pathogens in Antarctica, including the recent increases in temperature (27). These consequences of climate change can play an important role in disease expansion toward higher latitudes (3).

## THE GEOGRAPHIC ORIGIN OF PATHOGENS

Native pathogens are those that have coexisted with their native host populations, while alien pathogens originate from different geographic regions or different populations and provide unique challenges for new hosts (28). However, distinguishing native pathogens from alien pathogens is sometimes difficult when considering disease emergence and wildlife population declines on a global scale. For example, the amphibian pathogen Batrachochytrium dendrobatidis is responsible for the amphibian disease chytridiomycosis (29–31). This highly pathogenic, readily transmissible emerging disease with low host specificity across an entire animal class has no precedent in modern times (32). Since the discovery of chytridiomycosis associated with declines in the amphibian populations in Australia and Central America in 1988 (29), the pathogen that causes this disease has been described in several hundred different amphibian species and has caused pandemic disease that has decimated amphibian populations (33). Nevertheless, B. dendrobatidis has been detected in many regions with different histories of human exposure, raising questions about whether it is native to those environments and recent changes increased its virulence or it has recently been introduced and rapidly spread around the globe.

Because of the density-dependent nature of transmission, infectious diseases had been believed to be unlikely agents of extinction (<u>34</u>). However, infection with the microsporidian *Steinhausia* was clearly the cause of extinction of the Polynesian tree snail, *Partula turgida* (<u>35</u>). Likewise, *B. dendrobatidis* has been implicated in the extinction of the golden toad (*Incilius periglenes*) in Costa Rica, as well as the sharp-snouted day frog (*Taudactylus acutirostris*) and two species of gastricbrooding frogs (*Rheobatrachus* spp.) from Australia (36, 37).

# THE ROLE OF BIODIVERSITY

In addition to the direct impact of infectious disease on species diversity, the biodiversity of a habitat can influence the sensitivity of a population to infectious disease in many ways. Human alteration of the environment contributes to the loss of biodiversity and the subsequent impact of infectious disease. Several of these ecological impacts are described below.

1. Ecological release by loss of regulation by predators and competitors. Species extinction and the consequent reduction in biodiversity is not a random process (38, 39). In general, the "losers" are species with long life spans, large body masses, resource specialization, low reproductive rates, and other characteristics that make them much more susceptible to human activities (40). Thus, the abundance and diversity of carnivore predators may be greatly affected because they are particularly susceptible to habitat loss and fragmentation owing to their generally low population densities (38, 41, 42). This is not without consequences, as food chains contain a complex order of energy pathways that act as shock absorbers for dramatic population explosions (43). Therefore, in addition to the important role of predators in removing animals in poor health from communities  $(\underline{13})$ , the reduction in top predator populations can lead to the phenomenon of ecological release of prey species that are often reservoirs of disease.

Ecological release can also occur due to the loss of a competitor species utilizing the same food or space resource as the reservoir species (44, 45). Long-term studies have revealed the importance of interspecific competition in structuring communities of rodents in deserts; for example, an increase in the density of granivores was noted after experimentally removing larger competitors, such as the kangaroo rat, *Dipodomys* spp. (46).

2. Ecological simplification. A repercussion of ecological release is an increase in the abundance and geographic dispersion of generalist species, considered as the small group of "winners" in the global loss of biodiversity (39, 47). This process,

called ecological simplification, is the common denominator of current global anthropogenic change (40, 48). Generalist species have wide geographic distribution and a highly resilient ecology; further, they tend to be more competent as reservoirs or vectors compared with species with specialized niches (9, 13, 49, 50).

3. Loss of dilution effect. Decline in biodiversity may lead to loss of the dilution effect, considered as an ecosystem service that minimizes disease risk (11). This effect accounts for the decreased transmission of disease to a target species (e.g., Lyme disease in humans) when there is a greater number of species in the community (12). This is due to the decline in population density of reservoirs or vectors (51). In turn, there is a reduction in the frequency of encounters with these disease reservoirs or vectors (12). The result of the dilution effect is, therefore, a decrease in the prevalence of the pathogen resulting from the increase in species richness in a community (52).

# THE EFFECT OF LANDSCAPE STRUCTURE

A landscape is composed of multiple habitats. The mosaic of physical and biotic conditions that define each habitat and the interfaces between habitats play an important role in the biodiversity of lakes, rivers, swamps, grasslands, forests, riparian vegetation, marine seashore, and the successional regions between them. Within this landscape there are areas with human-mediated alterations like agricultural fields, grasslands used for farm animals, controlled forests, highways, recreation areas, cities, railways, and other human contrivances that constitute a matrix surrounding the remnants of wildlife habitats. Animals living in close proximity to this manmade matrix may experience altered habitats. Some animal populations may not be able to adapt to these changes, while others effectively meld into the new landscape. The dispersal of wildlife within this landscape is restricted by the hostility of the surroundings, habitat fragmentation, and availability of animal corridors within the matrix. This results in a mosaic of habitats within the landscape, where the population abundance of a species in one habitat patch is the result of both the quality of that habitat and the hostility of the surrounding matrix (53). Wildlife populations within this landscape of fragmented habitats can have spatial structures called metapopulations, which persist as a result of the combined dynamics of extinction within a

given habitat fragment and recolonization among fragments by dispersal (54). Thus, the landscape experienced by a population represents a mosaic of good and bad places for the species. Therefore, species distribution is restricted in time and space because of natural habitat discontinuity and landscape heterogeneity, and because individuals are incapable of moving through or around major barriers (55). Anthropogenic factors, consequently, can be responsible for species extinction by eliminating connecting patches or turning a surrounding habitat into a barrier (3). As a result, human-mediated habitat fragmentation may also create local populations that are completely isolated from one another (56). This concept also applies to river shores and seashores. Medina-Vogel et al. (57) and Vianna et al. (58)demonstrated that natural discontinuity of the rocky seashore patches along the coast of Chile, the main habitat of the marine otter (Lontra felina), is becoming fragmented as a result of the intense human activities along intervening sandy seashores and the collateral abundance of stray dogs. Within this context, domesticwild interspecies interaction is unavoidable, with dogs acting both as predators and as vectors of diseases to marine otters (59). Thus, in terms of the importance for the ecology of infectious diseases, the habitat fragmentation process has four components: (i) reduction of the total amount of habitat in a landscape, (ii) increased distance between remaining habitat fragments, (iii) increased impact of outside factors on the remnant habitat fragments (edge effects), and (iv) changes in the species diversity within each habitat fragment (Fig. 1). The result is a significant change in the animal and plant community structure (1). For the ecology of infectious diseases, the first alteration may reduce the rate of contact between susceptible and infected hosts by modifying density- or frequency-dependent interactions; the second may reduce "herd immunity" of vulnerable populations by limiting exposure to pathogens; the third may facilitate the introduction of new pathogens or vectors; and the fourth may promote new species or interspecific interactions that could facilitate the transmission of disease (Fig. 1). A species which at the beginning of the process is less competitive, but acts as a host of a virulent pathogen to a second species, later can displace the second species as a result of apparent competition  $(\underline{60})$ (Fig. 1).

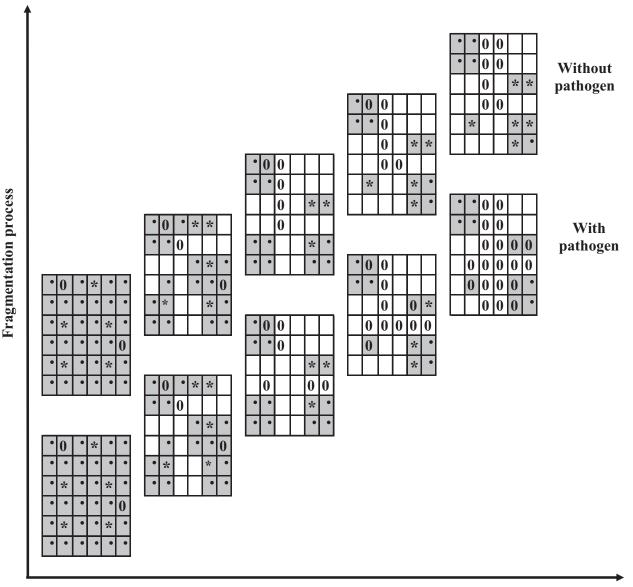
In western North America, prairie dog (*Cynomys* spp.) colonies vary in size and extension. Sylvatic plague caused by *Yersinia pestis* is positively correlated with the colony size (<u>61</u>, <u>62</u>). Prairie dogs are sympatric (species found in the same area) with populations of deer mice,

which are reservoir hosts for *Y. pestis* and their flea vectors. Dispersal of prairie dogs may occur more frequently in large colonies due to greater habitat suitability, thereby increasing the probability that a large colony will attract an immigrating prairie dog that is either infected or infested with infected fleas. Conversely, colony isolation, measured as the distance to the nearest plague-positive prairie dog colony, has been found to be negatively correlated with plague occurrence; even roads serve as barriers to sylvatic plague among black-tailed prairie dog colonies by affecting movement of or habitat quality for plague hosts or fleas that serve as vector for *Y. pestis* (<u>63</u>).

Gillespie and Chapman (64) studied parasite infection dynamics in red colobus (*Piliocolobus tephrosceles*) metapopulations inhabiting forest fragments in western Uganda. Their results demonstrate that an index of habitat degradation like stump density, as an indicator of forest extractive endeavors, significantly explained the prevalence of red colobus strongyle and rhabditoid nematode infection levels in forest fragments. In fact, they found a greater risk of infection with nematodes in the fragment with highest stump density than in the fragment with the lowest stump density. Colobus inhabiting fragments with high stump density are likely to experience a higher probability of contact with humans together with accompanying pathogens (64). This relationship between risk of disease exposure and proximity to urban areas or contact with humans has also been recorded for foxes (Urocyon cinereoargenteus) and bobcats (Lynx rufus) in urban and rural areas of California (65).

# THE EFFECT OF ALIEN SPECIES

Within the context of habitat fragmentation, alien species become particularly important. Alien species are those introduced by humans deliberately or by accident into new regions. Introduction or migration of infected wild and domestic animals has been an important factor in the emergence of many epizootics. Alien species are linked to the emergence of diseases such as West Nile virus in the Americas (<u>66</u>), squirrel poxvirus in the United Kingdom (<u>67</u>), and avian malaria in Hawaii (<u>68</u>), among others. Furthermore, alien species can participate as vectors and reservoirs of pathogens, posing a significant threat to global biodiversity when disease is introduced into native populations (<u>10</u>, <u>69</u>) that have not undergone selection for resistance to them (<u>31</u>).



#### Time

**FIGURE 1** Effect of habitat fragmentation on three different species populations (•, \*, and 0). Species 0 is less competitive against species • and \*, but became a reservoir of a pathogen highly virulent for species \* and less virulent for species •. By apparent competition, species 0 displaced species \* and began competing for resources with species •, which was highly specialized to the disappearing habitat conditions. Empty boxes represent areas with loss of habitat. doi:10.1128/microbiolspec.OH-0004-2012.fl

Within a landscape, some animals move and live within distinctive areas, defined as their home range, and some animals territorially defend this space, thereby influencing the rate of contact with other animals as well as their own population size and density. Other animals migrate every year in a certain season following reproductive and food availability. Avoidance of competition is common in sympatric species and between native and alien species. Although competition may be avoided by sympatric species, their close proximity can provide the opportunity for transmission of pathogens ( $\underline{3}$ ). Infectious diseases are often maintained in a dynamic equilibrium in a population that is influenced by the landscape ( $\underline{70}$ ). Therefore, any

environmental factor with the capability to alter the dynamic, such as the introduction of a new reservoir or hosts, may have the capacity to modify the epidemiology of pathogens  $(\underline{71})$ .

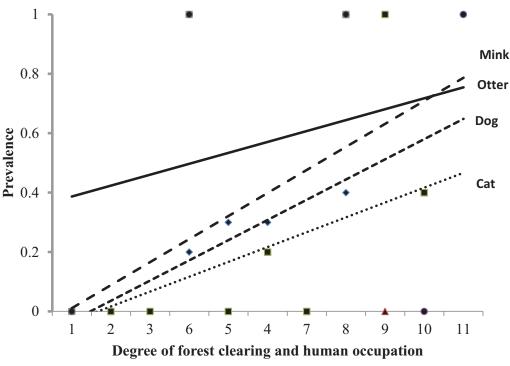
## THE COMBINED EFFECT—THE CASE OF LEPTOSPIRA

Leptospirosis is a zoonosis of global distribution. It is caused by the spirochete Leptospira, a pathogen with about 200 distinct serotypes (72). The severity of illness can range from an asymptomatic infection to a fatal illness involving the kidneys, liver, and other vital organs. However, there are no serotype-specific presentations of infection-each serotype may cause mild or severe disease depending on the host. Although in tropical areas incidence rates are particularly high, human cases are sporadic in Chile. Nevertheless, cases of leptospirosis in humans must be reported to the Chilean Ministry of Health. The diversity of serotypes in the population may be maintained by the reservoir hosts (73). A variety of wild and domestic animals can act as reservoir hosts for one or more serotypes and can shed the organism in their urine for months or years after being infected. This includes dogs, rats, swine, cattle, and raccoons in North America (74). Domestic as well as wild animals may come into contact with Leptospira by interspecies contact or by urine from farm animal reservoirs during activities such as swimming, drinking, or walking through contaminated water, soil, or mud. Humans become infected through contact of mucosal surfaces or abraded skin with contaminated soil or water or with animal urine or tissues. For example, participating in recreational activities in contaminated water increases the risk of human infection (75).

The prevalence of leptospirosis in animal populations indicates that transmission of *Leptospira* is influenced by human activities. Of 35 river otters tested for antibodies to Leptospira interrogans, 50% from Washington state were seropositive, but none of the 15 tested animals from Alaska were positive  $(\underline{76})$ . These results correlate with levels of exposure of otters to Toxoplasma gondii: otters from Washington state showed high levels of exposure to T. gondii while otters from Alaska did not. The southern sea otter (Enhydra lutris nereis) population in California and the Alaskan sea otter (E. lutris kenyoni) population in the Aleutian Islands have also shown serological evidence of exposure to *Lepstospira* spp. (77). These results suggest that living in regions close to higher human density and its associated agricultural activities, domestic animals, and accompanying rodent populations enhance exposure of river otters to these pathogens.

An ongoing research project in southern Chile is focused on identifying viral and bacterial agents in wild and domestic animals and assessing environmental variables associated with their prevalence in different species. More than 200 samples from domestic and wild animals have been tested for *Leptospira* spp. in southern Chile. The results demonstrate a high incidence of infection: 37% in dogs, 88 to 92% in cattle, 25% in sheep, 7% in horses, 70% in swine, and 47% in wild rodents (<u>78</u>).

The deliberate introduction of an alien species had a major impact on transmission of Leptospira in wild otters in Chile. The North American mink was brought to Chile in the 1930s for the pelt industry. By the 1970s a feral population had developed from animals that had escaped from mink farms (79). The North American mink is now widely distributed throughout the Andean lacustrine and riverine habitats in Argentina and the south of Chile from 38°S latitude to Tierra del Fuego Island and adjacent archipelagos at 55°S (80-83). In rivers, lakes, and the seashore of southern Chile, the alien North American mink now coexists with the native population of southern river otters (Lontra provocax). This otter is one of the species that is under major conservation threats globally. It has suffered a continual reduction in habitat as a result of riparian vegetation removal, river dredging, and pollution (84-86). In contrast, mink are less sensitive to human activities and can be found near human settlements, commonly poaching poultry. Hence, populations of mink are sympatric with domestic dogs and cats and have acquired Leptospira infections from these interactions. In addition, mink populations are also sympatric with populations of southern river otters  $(\underline{80}, \underline{81}, \underline{87})$ . Mink are semiaquatic, while otters are a more aquatic-adapted mustelid, so otters have significantly less habitat use overlap with domestic species than do mink. More than 50% of the diet of mink is wild rodents, while the diet of otters is nearly 100% aquatic macroinvertebrates and fish  $(\underline{80})$ . Nevertheless, otters show the highest prevalence of Leptospira infections among domestic and wild species (Fig. 2). Wildlife reservoirs are those more epidemiologically tied to a population in which the pathogen can be permanently maintained and from which infestation is transmitted to the defined target population (88). The higher prevalence of *Leptospira* in river otters compared with terrestrial species provides evidence that landscape alterations by humans, invasion of domestic species, and invasion of the alien mink species exacerbated the transmission and prevalence of



**FIGURE 2** Relationship between the degree of landscape transformation and human presence and the prevalence of *Leptospira* spp. in animals from the lake and river districts in southern Chile. Scale representing degree of forest clearing and human occupation: 1 (essentially no alterations) to 10 (high alteration and human presence). <u>doi:10.1128/microbiolspec.OH-0004-2012.f2</u>

*Leptospira* infections in the threatened, vulnerable otter population.

## **CONCLUDING REMARKS**

Despite evidence demonstrating the role of biodiversity depletion in the increase in zoonotic diseases, it has been suggested that the risk of emergence of infectious diseases will be higher where the biodiversity of mammals is greater, due to the assumption that each species carries an unknown number of potential pathogens ( $\underline{89}$ ,  $\underline{90}$ ).

Three consequences of global biodiversity loss related to the emergence and increased incidence of infectious diseases are (i) ecological release, (ii) ecological simplification, and (iii) loss of the dilution effect. Thus, it is proposed that biodiversity plays a crucial role in the animals' risk of infection, and is therefore a determining factor in the emergence of new zoonotic diseases. Habitat alterations such as fragmentation, overexploitation of hosts and reservoirs, new interspecific interactions with alien species, new interspecific interactions with domestic species, pollution, and new distributions as a result of climate change are affecting biodiversity globally with unprecedented magnitude and speed. However, it seems that the factor that is of particular concern is the increase in interspecific interactions between domestic and alien species and wild species, which is bringing together hosts never before in contact. This is of special concern in natural regions including islands, national parks, and protected areas. Climate change is forcing many species into new geographic distributions, altering the animal communities in certain regions. As a result, some species will become extinct and others will expand, setting up new host-parasite, parasite-vectorhost, and host-host interactions.

Biodiversity and the landscape structure play important roles in wildlife health through various mechanisms. Therefore, the "hot spots" on which to focus in monitoring emerging infectious diseases in wildlife should be located in areas with habitat loss, such as through deforestation (with special emphasis on the edges between habitat remnants and the matrix generated, and on the movement of domestic species into habitats) and where hydrological, agricultural, or aquaculture development projects are taking place. In the short term, permanent disease monitoring is needed to prevent the increase of vectors or unwanted reservoir populations and the emergence of diseases associated with trends of habitat lost and land use changes. Moreover, when defining longterm goals of maintaining the health of endangered species, disease in nearby populations of domestic and sympatric alien species should be permanently monitored.

The inclusion of studies of infectious disease in biodiversity and interspecific interactions between wildlife and domestic animals should be an important and complementary aspect to understanding human health and beyond—the health of ecosystems including humans as part of nature. As emphasized throughout the volume One Health: People, Animals, and the Environment (91), most of the causative agents of emerging infectious diseases in humans are zoonotic. The advantage of focusing on biodiversity monitoring for the prevention of disease outbreaks or emergence is that even when all the necessary factors for the occurrence of illness are present (reservoir species, pathogens, intermediate or terminal hosts, and appropriate weather conditions), biodiverse communities possess the capacity to alleviate emergence events, making the biodiversity loss the ultimate determinant of the onset of illness.

#### ACKNOWLEDGMENTS

Funding was provided by the Chilean Fund for Science and Technology (Fondecyt) project 1100139: "Presence of infectious diseases in wild species: the effect of alien invasive North American mink (*Neovison vison*) and the coexistence with stray dogs and cats." I also want to thank Pamela Lepe, Claudio Soto, and Lucía Alladio, students in the Ph.D. program in Conservation Medicine who assisted with reference searches.

### REFERENCES

1. Pullin AS. 2002. Conservation Biology, p 345. Cambridge University Press, Cambridge, United Kingdom.

**2.** Daszak P, Cunningham AA. 2002. Emerging infectious diseases: a key role for conservation medicine, p 40–61. *In* Aguirre AA, Ostfeld RS, Tabor GM, House C, Pearl MC (ed), *Conservation Medicine: Ecological Health in Practice*. Oxford University Press, New York, NY.

3. Medina-Vogel G. 2010. Ecología de enfermedades infecciosas emergentes y conservación de especies silvestres. Arch Med Vet 42:11–24.

4. Garner TW, Perkins MW, Govindarajulu P, Seglie D, Walker S, Cunningham AA, Fisher MC. 2006. The emerging amphibian pathogen *Batrachochytrium dendrobatidis* globally infects introduced populations of the North American bullfrog, *Rana catesbeiana*. *Biol Lett* 3:455–459.

5. Mañas S, Ceña JC, Ruiz-Olmo J, Palazón S, Domingo M, Wolfinbarger, JB, Bloom ME. 2001. Aleutian mink disease parvovirus in wild riparian carnivores in Spain. J Wildl Dis 37:138–144.

6. Schrag SJ, Wiener P. 1995. Emerging infectious disease: what are the relative roles of ecology and evolution? *Trends Ecol Evol* 10:319–324.

7. Epstein PR. 2002. Biodiversity, climate change, and emerging infectious diseases, p 27–39. *In* Aguirre AA, Ostfeld RS, Tabor GM, House C, Pearl MC (ed), *Conservation Medicine: Ecological Health in Practice*. Oxford University Press, New York, NY.

Pimm SL, Alves MA, Chivian E, Bernstein A. 2008. What is biodiversity?, p 3–26. *In* Chivian E, Bernstein A (ed), *Sustaining Life: How Human Health Depends on Biodiversity*. Oxford University Press, New York, NY.
 Molyneux DH, Ostfeld RS, Bernstein A, Chivian E. 2008. Ecosystem

disturbance, biodiversity loss, and human infectious disease, p 287-323. In Chivian E, Bernstein A (ed), *Sustaining Life: How Human Health Depends on Biodiversity*. Oxford University Press, New York, NY.

10. Daszak P, Cunningham AA, Hyatt AD. 2000. Emerging infectious diseases of wildlife—threats to biodiversity and human health. *Science* 287:443–449.

**11. Ostfeld RS, LoGiudice K.** 2003. Community disassembly, biodiversity loss, and the erosion of an ecosystem service. *Ecology* **84**:1421–1427.

**12.** Ostfeld RS. 2009. Biodiversity loss and the rise of zoonotic pathogens. *Clin Microbiol Infect* **15**(Suppl 1):40–43.

**13. Morand S.** 2011. Infectious diseases, biodiversity and global changes: how the biodiversity sciences may help, p 231–254. *In* López-Pujol J (ed), *The Importance of Biological Interactions in the Study of Biodiversity*. InTech, Rijeka, Croatia.

14. Daszak P, Cunningham AA, Hyatt AD. 2001. Anthropogenic environmental change and the emergence of infectious diseases in wildlife. *Acta Tropica* 78:103–116.

15. Dietz R, Ansen CT, Have P, Heide-Jørgensen MP. 1989. Clue to seal epizootic? *Nature* 338:627.

16. Hall AJ, Pomeroy PP, Harwood J. 1992. The descriptive epizootiology of phocine distemper in the UK during 1988/89. *Sci Total Environ* 115:31–44.

17. Heide-Jørgensen MP, Harkonen T, Dietz R, Thompson PM. 1992. Retrospective of the 1988 European seal epizootic. *Dis Aquat Organ* 13:37–62.

18. Broman T, Bergström S, On SL, Palmgren H, McCafferty DJ, Sellin M, Olsen B. 2000. Isolation and characterization of *Campylobacter jejuni* subsp. *jejuni* from macaroni penguins (*Eudyptes chrysolophus*) in the subantartic region. *Appl Environ Microbiol* 66:449–452.

19. Palmgren H, McCafferty D, Aspán A, Broman T, Sellin M, Wollin R, Bergström S, Olsen B. 2000. *Salmonella* in sub-Antarctica: low heterogeneity in *Salmonella* serotypes in South Georgian seals and birds. *Epidemiol Infect* 125:257–262.

**20.** Parmelee DF, Maxson SJ, Bernstein NP. 1979. Fowl cholera outbreak among brown skuas at Palmer Station. *Antarct J U S* **14**:168–169.

**21. de Lisle GW, Stanislawak WL, Moors PJ.** 1990. *Pasteurella multocida* infections in rockhopper penguins (*Eudyptes chrysocome*) from Campbell Island, New Zealand. *J Wildl Dis* **26:**283–285.

22. MacDonald JW, Conroy JW. 1971. Virus disease resembling puffinosis in the gentoo penguin *Pygoscelis papua* on Signy Island, South Orkney Islands. *Br Antarct Surv Bull* 26:80–83.

23. Gardner H, Kerry K, Riddle M, Brouwer S, Gleeson L. 1997. Poultry virus infection in Antarctic penguins. *Nature* 387:245.

**24. Weimerskirch H.** 2004. Diseases threaten Southern Ocean albatrosses. *Polar Biol* **27**:374–379.

25. Laws RM, Taylor RJ. 1957. A mass dying of crabeater seals, Lobodon carcinophagus (gray). Proc Zool Soc Lond 129:315-325.

26. Fuchs V. 1982. Of Ice and Men. Anthony Nelson, London, United Kingdom.

27. Wobeser AG. 2006. Essentials of Disease in Wild Animals. Blackwell Publishing, Ames, IA.

**28.** Dobson A, Foufopolus J. 2001. Emerging infectious pathogens of wildlife. *Philos Trans R Soc Lond B Biol Sci* **356**:1001–1012.

29. Berger L, Speare R, Daszak P, Green DE, Cunningham AA, Goggin CL, Slocombe R, Ragan MA, Hyatt AD, McDonald KR, Hines HB, Lips KR, Marantelli G, Parkes H. 1998. Chytridiomycosis causes amphibian mortality associated with population declines in the rain forests of Australia and Central America. *Proc Natl Acad Sci USA* **95**:9031–9036.

**30.** Longcore JE, Pessier AP, Nichols DK. 1999. *Batrachochytrium dendrobatidis* gen. et sp. nov., a chytrid pathogenic to amphibians. *Mycologia* **91**:219–227.

**31.** Skerratt LF, Berger L, Speare R, Cashins S, McDonald KR, Phillott AD, Hines HB, Kenyon N. 2007. Spread of chytridiomycosis has caused the rapid global decline and extinction of frogs. *Ecohealth* **4**:125–134.

32. Gascon C, Collins JP, Moore RD, Church DR, McKay JE, Mendelson JR III (ed). 2007. *Amphibian Conservation Action Plan*. World Conservation Union/Species Survival Commission Amphibian Specialist Group, Gland, Switzerland and Cambridge, United Kingdom. <u>http://www.amphibianark.org/pdf/ACAP.pdf</u> (last accessed June 10, 2013).

**33. Fisher MC, Garner TW.** 2007. The relationship between the emergence of *Batrachochytrium dendrobatidis*, the international trade in amphibians and introduced amphibian species. *Fungal Biol Rev* **21**:2–9.

34. Smith FS, Sax FS, Lafferty KD. 2006. Evidence for the role of infectious disease in species extinction and endangerment. *Conserv Biol* 20:1349–1357.

35. Daszak P, Cunningham AA. 1999. Extinction by infection. *Trends Ecol Evol* 14:279.

**36.** Daszak P, Berger L, Cunningham AA, Hyatt AD, Green DE, Speare R. 1999. Emerging infectious diseases and amphibian population declines. *Emerg Infect Dis* **5**:735–748.

**37. Schloegel LM, Hero JM, Berger L, Speare R, McDonald K, Daszak P.** 2006. The decline of the sharp-snouted day frog (*Taudactylus acutirostris*): the first documented case of extinction by infection in a free-ranging wildlife species? *Ecohealth* **3**:35–40.

**38.** Duffy JE. 2002. Biodiversity and ecosystem function: the consumer connection. *Oikos* **99:**201–219.

**39.** McKinney ML, Lockwood, JL. 1999. Biotic homogenization: a few winners replacing many losers in the next mass extinction. *Trends Ecol Evol* **14**:450–453.

40. Díaz S, Fargione J, Chapin FS III, Tilman D. 2006. Biodiversity loss threatens human well-being. *PLoS Biol* 4:e277. <u>doi:10.1371/journal.</u> pbio.0040277.

41. Terborgh J, Lopez V, Nuñez P, Rao M, Shahabuddin G, Orihuela G, Riveros M, Ascanio R, Adler GH, Lambert TD, Balbas L. 2001. Ecological meltdown in predator-free forest fragments. *Science* 294:1923–1926.

**42.** McMichael AJ. 2004. Environmental and social influences on emerging infectious diseases: past, present and future. *Philos Trans R Soc Lond B Biol Sci* **359**:1049–1058.

43. McCann KS. 2000. The diversity-stability debate. *Nature* 405:228–233.

44. Begon M, Townsend CR, Harper JL. 2006. *Ecology: from Individuals to Ecosystems*, 4th ed. Blackwell Publishing, Oxford, United Kingdom.

**45.** Caut S, Casanovas JG, Virgos E, Lozano J, Witmer GW, Courchamp F. 2007. Rats dying for mice: modelling the competitor release effect. *Austral Ecol* **32**:858–868.

**46.** Heske EJ, Brown JH, Mistry S. 1994. Long-term experimental study of a Chihuahuan Desert rodent community: 13 years of competition. *Ecology* 75:438–445.

**47. Auffray JF, Renaud S, Claude J.** 2009. Rodent biodiversity in changing environments. *Kasetsart J (Nat Sci)* **43:**83–93.

48. Pongsiri MJ, Roman J, Ezenwa VO, Goldberg TL, Koren HS, Newbold SC, Ostfeld RS, Pattanayak SK, Salkeld DJ. 2009. Biodiversity loss affects global disease ecology. *BioScience* 59:945–954.

**49.** Chaisiri K, Chaeychomsri W, Siruntawineti J, Bordes F, Herbreteau V, Morand S. 2010. Human-dominated habitats and helminth parasitism in Southeast Asian murids. *Parasitol Res* **107**:931–937.

50. Mills JN. 2006. Biodiversity loss and emerging infectious diseases: an example from the rodent-borne hemorrhagic fevers. *Biodiversity* 7:9–17.
51. Schmidt KA, Ostfeld RS. 2001. Biodiversity and the dilution effect in disease ecology. *Ecology* 82:609–619.

**52.** Clay CA, Lehmer EM, Jeor SS, Dearing MD. 2009. Sin Nombre virus and rodent species diversity: a test of the dilution and amplification hypotheses. *PLoS One* 4:e6467. <u>doi:10.1371/journal.pone.0006467</u>.

**53.** Andrén H. 1994. Effects of habitat fragmentation on birds and mammals in landscapes with different proportions of suitable habitat: a review. *Oikos* **71**:355–366.

54. McCullough DR (ed). 1996. Metapopulations and Wildlife Conservation. Island Press, Washington, DC.

55. Wiens JA. 1996. Wildlife in patchy environments: metapopulations, mosaics and management, p 53–84. *In* McCullough DR (ed), *Metapopulations and Wildlife Conservation*. Island Press, Washington, DC.

**56.** Hastings A, Harrison S. 1994. Metapopulation dynamics and genetics. *Annu Rev Ecol Syst* **25**:167–188.

**57.** Medina-Vogel G, Merino LO, Monsalve Alarcón R, Vianna JA. 2008. Coastal-marine discontinuities, critical patch size and isolation: implications for marine otter conservation. *Anim Conserv* **11**:57–64.

58. Vianna JA, Ayerdi P, Medina-Vogel G, Mangel JC, Zeballos H, Apaza M, Faugeron S. 2010. Phylogrography of the marine otter (*Lontra felina*): historical and contemporary factors determining its distribution. *J Hered* 101:676–689.

**59.** Medina-Vogel G. Boher F, Flores G, Santibañez A, Soto-Azat C. 2007. Spacing behavior of marine otters (*Lontra felina*) in relation to land refuges and fishery wastes in central Chile. *J Mammal* 88:487–494.

**60. Lafferty KD.** 2008. Effect of disease on community interactions and food web structure, p 205–222. *In* Ostfeld RS, Keesing F, Eviner VT (ed), *Infectious Disease Ecology: Effects of Ecosystems on Disease and of Disease on Ecosystems.* Princeton University Press, Princeton, NJ.

**61.** Cully JF Jr, Williams ES. 2001. Interspecific comparisons of sylvatic plague in prairie dogs. J Mammal 82:894–905.

**62.** Lomolino MV, Smith GA. 2001. Dynamic biogeography of prairie dog (*Cynomys ludovicianus*) town near the edge of their range. *J Mammal* **82**:937–945.

63. Collinge SK, Johnson WC, Ray C, Matchett R, Grensten J, Cully JF, Gage, KL, Kosoy MY, Loye JE, Martin AP. 2005. Landscape structure and plague occurrence in black-tailed prairie dogs on grasslands of the western USA. *Landsc Ecol* 20:941–955.

**64.** Gillespie TR, Chapman CA. 2006. Prediction of parasite infection dynamics in primate metapopulations based on attributes of forest fragmentation. *Conserv Biol* **20:**441–448.

**65.** Riley SP, Foley J, Chomel B. 2004. Exposure to feline and canine pathogens in bobcat and gray foxes in urban and rural zones of a national park in California. *J Wildl Dis* **40:11–22**.

66. Anderson JF, Andreadis TG, Vossbrinck CR, Tirrell S, Wakem EM, French RA, Garmendia AE, Van Kruiningen HJ. 1999. Isolation of West Nile virus from mosquitoes, crows, and a Cooper's hawk in Connecticut. *Science* **286**:2331–2333.

**67.** Sainsbury AW, Nettleton P, Gilray J, Gurnell J. 2000. Grey squirrels have high seroprevalence to a parapoxvirus associated with deaths in red squirrels. *Anim Conserv* **3**:229–233.

**68.** van Riper C, van Riper SG, Goff ML, Laird M. 1986. The epizootiology and ecological significance of malaria in Hawaiian land birds. *Ecol Monogr* **56**:327–344.

**69.** Cunningham AA, Daszak P, Rodriguez JP. 2003. Pathogen pollution: defining a parasitological threat to biodiversity conservation. *J Parasitol* **89:**S78–S83.

**70. Cabello CC, Cabello CF.** 2008. [Zoonoses with wildlife reservoirs: a threat to public health and the economy]. *Rev Med Chil* **136**:385–393. (In Spanish.)

71. Tabor GM. 2002. Defining conservation medicine, p 8–16. *In* Aguirre AA, Ostfeld RS, Tabor GM, House C, Pearl MC (ed), *Conservation Medicine: Ecological Health in Practice*. Oxford University Press, New York, NY.

72. Farr RW. 1995. Leptospirosis. Clin Infect Dis 21:1-6; quiz 7-8.

73. Lagadec E, Gomard Y, Guernier V, Dietrich M, Pascalis H, Temmam S, Ramasindrazana B, Goodman SM, Tortosa P, Delagi K. 2012. Pathgogenic *Leptospira* spp. in bats, Madagascar and Union of the Comoros. *Emerg Infect Dis* 18:1696–1698.

74. Levett P. 2005. Leptospirosis, p 2789–2798. *In* Mandell GL, Bennett JE, Dolin R (ed), *Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases*, 5th ed. Churchill Livingstone, Philadelphia, PA.

75. Vinetz, JM, Wilcox BA, Aguirre A, Gollin LX, Katz AR, Fujioka RS, Maly K, Horwitz P, Chang H. 2005. Beyond disciplinary boundaries: leptospirosis as a model of incorporating transdisciplinary approaches to understand infectious disease emergence. *Ecohealth* 2:291–306.

76. Gaydos JK, Conrad PA, Gilardi KV, Blundell GM, Ben-David M. 2007. Does human proximity affect antibody prevalence in marine-foraging river otters (*Lontra canadensis*)? J Wildl Dis 43:116–123.

77. Hanni KD, Mazet JA, Gulland FM, Estes J, Staedler M, Murray MJ, Miller M, Jessup DA. 2003. Clinical pathology and assessment of pathogen exposure in southern and Alaskan sea otters. *J Wildl Dis* 39:837–850.

78. Zunino E, Pizarro R. 2007. [Leptospirosis: a literature review.] *Rev Chil Infectol* 24:220–226. (In Spanish.)

79. Jaksic FM, Iriarte JA, Jiménez JE, Martínez DR. 2002. Invaders without frontiers: cross-border invasions of exotic mammals. *Biol Invasions* 4:157–173.

**80. Medina G.** 1997. A comparison of the diet and distribution of the southern river otter (*Lutra provocax*) and mink (*Mustela vison*) in Southern Chile. J Zool 242:291–297.

**81.** Fasola L, Chehébar C, Macdonald DW, Porro G, Cassini M. 2009. Do alien North American mink compete for resources with native South American river otter in Argentinean Patagonia? *J Zool* 277:187–195.

82. Ibarra JT, Fasola L, Macdonald DW, Rozzi R, Bonacic C. 2009. Invasive American mink *Mustela vison* in wetlands of the Cape Horn Biosphere Reserve, southern Chile: what are they eating? *Oryx* 43:87–90.
83. Rozzi R, Sheriffs M. 2003. El visón (*Mustela vison* Schreber: Carnivora: Mustelidae), un nuevo mamífero exótico para la isla Navarino. *An Inst Patagon* 31:97–104.

84. Medina-Vogel G. 1996. Conservation status of *Lutra provocax* in Chile. *Pacific Conserv Biol* 2:414–419.

**85.** Medina-Vogel G, Kaufmann VS, Monsalve R, Gomez V. 2003. The relationship between riparian vegetation, woody debris, stream morphology, human activity and the use of rivers by southern river otter in Chile. *Oryx* **37:**422–430.

86. Medina-Vogel G, Gonzalez-Lagos C. 2008. Habitat use and diet of endangered southern river otter *Lontra provocax* in a predominantly palustrine wetland in Chile. *Wildl Biol* 14:211–220.

87. Aued MB, Chehébar C, Porro G, Macdonald DW, Cassini MH. 2003. Environmental correlates of the distribution of southern river otters *Lontra provocax*. Oryx 37:413–421.

**88. Haydon DT, Laurenson MK, Sillero-Zubiri C.** 2002. Integrating epidemiology into population viability analysis: managing the risk posed by rabies and canine distemper to the Ethiopian wolf. *Conserv Biol* **16**:1372–1385.

89. Atlas R, Rubin C, Maloy S, Daszak P, Colwell R, Hyde B. 2010. One Health—attaining optimal health for people, animals, and the environment. *Microbe* 5:383–389.

90. Dunn R. 2010. Global mapping of ecosystem disservices: the unspoken reality that nature sometimes kills us. *Biotropica* 42:555–557.

**91.** Atlas RM, Maloy S (ed). 2014. One Health: People, Animals, and the Environment. ASM Press, Washington, DC.