Disease emergence and biodiversity loss are among the most pervasive environmental problems confronting science and society. Widespread and dramatic losses of amphibian populations and species represent the deadly intersection between these issues (1), as illustrated by the work of Lips et al. (2) in this issue of PNAS. Amphibians are one of the most imperiled groups of vertebrates, with nearly half of all species in decline and >100 species extinct or nearly so (3). Although a number of these declines can be linked to habitat loss and land-use changes (4), many of the most rapidly declining species (nearly 50%) occur in areas without obvious environmental changes. Even more disturbing is that affected populations show few signs of recovery, and analyses across species and time confirm the unusual and unidirectional downward trend (5).

Since discovery of a chytrid fungus (*Batrachochytrium dendrobatidis*) pathogenic to amphibians in the mid-1990s (6), increased attention has focused on the pathogen’s role in the death and disappearance of amphibians. *Batrachochytrium* has been recovered from the skin of dead or dying frogs associated with numerous population declines, particularly from Central America, Australia, and New Zealand (7, 8). Experimental exposure of healthy frogs to chytrid zoospores causes rapid deterioration and death in certain species (6, 9). Although *Batrachochytrium* cannot explain all enigmatic amphibian population declines, the evidence linking the pathogen to amphibian losses has grown considerably in the last decade, and >60 studies have detailed its origin, pathology, physiology, life cycle, genetic variation, and infection dynamics.

Despite enormous progress, the link between *Batrachochytrium* infection and amphibian population declines has been hindered by data limitations (see ref. 10). During declines in Central America and Australia, for example, little information was available on the changes in chytrid infection prevalence before, during, and after the suspected epidemics or on how prevalence differed between morbund and healthy individuals, which is critical data for evaluating the causal role of infection (10). Collectively, these limitations precluded differentiation between two hypotheses: (i) the infection is entirely novel, spreading among immunologically naïve amphibian populations vs. (ii) it is an endemic infection that has increased in virulence or pathogenicity, likely as the result of environmental changes (11).

Without such information, it is impossible to determine whether efforts should focus on slowing the spread of *Batrachochytrium* or on controlling the environmental cofactors responsible for elevated infection. Finally, pathogens are not supposed to cause host population- or species-level losses because of reduced transmission rates at low host densities, casting doubt as to whether chytrid infection was the cause or a correlate of observed declines.

**Eyewitness to an Epidemic**

In this issue of PNAS, Lips et al. (2) present the most thorough and comprehensive documentation of an outbreak of chytridiomycosis and the resulting decline of an amphibian community. By combining long-term data (1998–2005), large-scale monitoring, and intensive sampling (nearly 30,000 amphibians representing 67 species), Lips et al. offer compelling evidence that *Batrachochytrium* is the proximate cause for observed declines in El Copé, Panama. Between May 1998 and July 2004, *Batrachochytrium* was not detected among the >1,500 sampled amphibians. Within 4 months of detecting the first infected frog in September 2004, however, the infection spread rapidly throughout the amphibian community.

After 6 years of relative stability, overall amphibian abundance and species richness exhibited a catastrophic decline. More than 300 dead or dying amphibians were collected by the authors (Fig. 1), and overall amphibian density and species richness declined by >50%. Importantly, infection prevalence and the subsequent decline were most severe around aquatic habitats during the breeding season, consistent with a largely waterborne pathogen.

The findings of Lips et al. (2) significantly advance our knowledge of disease-related amphibian declines in three important dimensions. First, their data demonstrate that *Batrachochytrium* was absent or at low prevalence (<0.3%) in amphibians before fall 2004. This finding suggests strongly that the pathogen was a new arrival to the area (or at least to amphibian hosts), consistent with the spread of an epidemic “wave.” Thus, although environmental cofactors are likely to influence the spread of *Batrachochytrium* (e.g., ref. 12), it is not necessary to invoke an additional proximal agent in explaining...
the El Cope´ decline. Second, the authors verified that infection levels among moribund and dead amphibians were substantially higher (> 100%) than those in healthy animals, suggesting that the presence of infection was not coincidental. Correspondingly, exposure of frogs collected from beyond the epidemic zone to Batrachochytrium caused infection and mortality. Third, the results illustrate the variability among species in their sensitivity to infection and the occurrence of infection but limited pathology in postdecline sites (see also ref. 13). Less-sensitive species may therefore function as “reservoir” hosts, allowing the persistence of infection after declines in sensitive amphibian species. Finally, the work of Lips et al. (2) illustrates how rapidly pathogen invasion and subsequent host declines can occur. In the course of only a few months, species richness and amphibian density declined by 60% and 90%, respectively. No other pathogens, climate anomalies, exotic predators, or land-use changes could be linked to the declines. Although the results of Lips et al. (2) substantiate the link between chytridiomycosis and amphibian population declines, significant questions remain. Even if Batrachochytrium is the proximate cause of amphibian mortality, it is likely that additional biotic and abiotic variables affect its spread and pathology. Typically, diseases “emerge” owing to environmental changes that enhance pathogen transmission, including species introductions, land-use shifts, climate change, or pollution (14, 15). Recent work by Pounds et al. (12), for example, suggests that temperature shifts promote Batrachochytrium infection at certain elevations and may therefore have indirectly driven the loss of numerous harlequin frog species in Central America and South America. However, considering that the chytrid appears to be on the move, particularly in Central America and eastern Australia, identifying the primary transport vectors is a fundamental research priority. Some amphibian species exhibit infection with little or no pathology [e.g., reservoir hosts (16, 17)], and it is likely that the natural and human-mediated movement of these animals plays an important role in pathogen redistribution. The importance of alternate vectors (e.g., birds, fish, invertebrates), extended free-living stages, and humans (including researchers) as dispersal agents for Batrachochytrium also should be considered (18).

A better understanding of the dispersal mechanisms, role of environmental cofactors, and host–parasite ecology is critical in formulating an effective response to this crisis: What can be done to prevent further losses associated with the epidemic? The results of Lips et al. (2) suggest that it is possible to predict the future trajectory of chytrid epidemics, at least at regional scales. Targeted removal or control of major transport vectors might help prevent further spread of the disease and its devastating consequences. If, after an epidemic, surviving amphibians exhibit resistance or tolerance to infection (19), is it possible to vaccinate populations in advance of the pathogen’s arrival? Captive breeding and reintroduction strategies for sensitive species also may hold promise but would only be viable if Batrachochytrium disappeared from the environment after an epidemic, which does not appear to be the case (19). Use of chemical or thermal treatments to eliminate infection in affected animals is limited by the same problem.

Ecosystem Consequences of Disease? Amphibians represent an important link between terrestrial and aquatic food webs, and the widespread loss of populations and even entire amphibian species is likely to have far-reaching consequences for affected ecosystems. Herbivorous larval anurans can be the dominant grazers on aquatic algae, particularly in small wetlands and streams (1, 20). Through metamorphosis, amphibians transport nutrients from aquatic environments into terrestrial systems. Adult amphibians are significant insect predators and important prey items for reptiles, birds, and mammals. Pest control by amphibians is considered a valuable ecosystem service (1, 20). The loss of so many individual amphibians, and especially the irreversible loss of entire species, is likely to alter aquatic and terrestrial food webs, nutrient cycling, algal biomass, and insect abundance. Unfortunately, despite their likely importance, the ecological consequences of amphibian declines have received little attention by the research community. By offering a more definitive link between chytrid infections and amphibian population losses, the results of Lips et al. (2) lay the groundwork to pursue this important research area.

Historically, wildlife diseases were assumed not to cause widespread mortality or drive the loss of host populations or species. In part this assumption stems from the difficulties inherent in witnessing an epidemic die-off. And in part this assumption stems from a population-based perspective on disease. Population models of host–parasite dynamics predict decreased transmission with reduced host density, thereby protecting the host from pathogen-induced extremation. However, as evidenced by chytrid epidemics in amphibian communities, catastrophic declines and even extinctions associated with disease are more than a theoretical possibility, and may be more widespread than previously suspected. The involvement of reservoir hosts, transport vectors, and extended free-living stages can decouple the dependency of a pathogen on the survival of any single host species (10). Given the widespread and expanding number of emerging diseases, of human as well as wildlife populations, a deeper understanding of these phenomena requires adopting a broader perspective on disease ecology: moving beyond populations to incorporate the reciprocal interactions among host–parasite dynamics, ecological communities, and ecosystem processes.