EcoHealth (© 2007)
DOI: 10.1007/s10393-007-0093-5

ECOHEALTH

Spread of Chytridiomycosis Has Caused the Rapid Global Decline and Extinction of Frogs

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Abstract: The global emergence and spread of the pathogenic, virulent, and highly transmissible fungus *Batrachochytrium dendrobatidis*, resulting in the disease chytridiomycosis, has caused the decline or extinction of up to about 200 species of frogs. Key postulates for this theory have been completely or partially fulfilled. In the absence of supportive evidence for alternative theories despite decades of research, it is important for the scientific community and conservation agencies to recognize and manage the threat of chytridiomycosis to remaining species of frogs, especially those that are naive to the pathogen. The impact of chytridiomycosis on frogs is the most spectacular loss of vertebrate biodiversity due to disease in recorded history.

Keywords: chytridiomycosis, *Batrachochytrium dendrobatidis*, decline, extinction, frogs, amphibians, postulates, global

INTRODUCTION

Amphibian populations have recently undergone declines and extinctions on a global scale (Stuart et al., 2004). Nine species have become extinct since 1980, and 113 more are possibly extinct. Four hundred and thirty-five species have demonstrated rapid declines since 1980. The declines of 233 species are attributed to overexploitation or habitat loss (Stuart et al., 2004). Of the remaining 202 rapidly declining species, and some populations of 5 species experiencing habitat loss, no cause has been attributed—these declines are labeled "enigmatic" by Stuart et al. (2004). Species affected by enigmatic declines are mostly stream-associated frogs at medium to high altitude, in protected forested sites in the tropics of Central and South America and northern Australia. Stuart et al. (2004) list the amphibian chytrid fungus *Batrachochytrium dendrobatidis (Bd)* as a commonly cited cause of declines of frogs but do not consider whether it is the primary cause of "enigmatic" declines. Here we present the evidence that the emergence and spread of this virulent skin pathogen, *Bd*, which causes chytridiomycosis, is by far the most likely primary cause of most of these "enigmatic" declines of frogs.

Because dead frogs are hard to find and are scavenged or decompose quickly, mass mortality events have been

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insidious and have largely gone undetected. Rapid declines (i.e., <1 year) can only be caused by the death of adults, and the only plausible causes of this in undisturbed habitats are a toxin, abnormal environmental change, or an infectious pathogen (Laurance et al., 1996). Discriminating among these possible causes has been difficult because investigations of the causes of declines usually start after the event, and no dead or dying frogs are observed or collected for necropsy. No evidence of a globally disseminated toxin has been presented and the current environmental changes in tropical areas (where most declines have occurred) have occurred previously and have not caused rapid declines (Alexander and Eischeid, 2001). By extrapolating what is known from a few well-studied sites during declines in Australia, Central America, North America, and Spain (Berger et al., 1998; Bosch et al., 2001; Muths et al., 2003; Lips et al., 2006; Schloegel et al., 2006) to sites with similar patterns of decline (Bell et al., 2004; La Marca et al., 2005; Scherer et al., 2005) and using the increasing knowledge of the biology of *Bd*, we show that chytridiomycosis is the only explanation, for which supporting evidence is available, for these global "enigmatic" declines and disappearances of frog populations and species. We also show that the most parsimonious explanation with supporting evidence for the emergence of chytridiomycosis is the introduction and spread of *Bd* among naive populations of frogs (Laurance et al., 1996; Berger et al., 1999; Daszak et al., 1999; Weldon et al., 2004).

Koch's postulates have been used in determining whether infectious agents cause disease in individuals (Evans, 1976). These traditional postulates have been conclusively satisfied for *Bd*, and studies have shown that it is highly pathogenic and virulent in individuals of declining species; experimental work shows that pure cultures isolated from sick and dying frogs cause fatal disease in otherwise healthy frogs (Lamirande and Nichols, 2002; Woodhams et al., 2003; Berger et al., 2004; Carey et al., 2006). Here we propose two mutually exclusive theoretical scenarios by which an infectious pathogen could cause global population declines, and propose criteria for differentiating between them. This framework can be applied to populations that have suddenly declined, even if mass mortality has not been detected. This is an important advance for conservation medicine, as traditional veterinary epidemiological approaches do not take into account the difficulties associated with the investigation of wildlife diseases (Wobeser, 1994).

Framework

We propose two alternative theoretical scenarios for how an infectious pathogen can cause severe population declines globally and drive species to extinction, based on current understanding of host–pathogen interactions. These explanations were first proposed for chytridiomycosis by Berger et al. (1998).

1. Distributional spread of a highly pathogenic, virulent, transmissible pathogen, leading to high mortality and failure of recruitment of the next generation in naive populations of susceptible species (definition of pathogenic: capable of causing disease; definition of virulent: causes severe disease). This has been named the "novel pathogen hypothesis" (Rachowicz et al., 2005). This is perhaps a misleading name to some, as while the pathogen *Bd* was unknown to science and is new to host populations, it is likely that it has existed for a long time in obscurity. A better name would be the "spreading pathogen hypothesis." This is the main reason for disease emergence and the only reason for pandemics to date (Morse, 1995). Some of the numerous examples of pandemics due to disease spread are the human influenza pandemics of the 20th century, the AIDS pandemic, the SARS pandemic, the canine parvoviral enteritis pandemic, crayfish plague, and recently the H5N1 avian influenza pandemic.
2. Abnormal environmental change (e.g., climate change, increased UV-B radiation, pollution) that results in an already widespread pathogen becoming highly pathogenic, virulent, and transmissible, resulting in high mortality and failure of recruitment of the next generation (the pathogen may already have some of these characteristics but not all prior to the environmental change). This has been named the "emerging endemic hypothesis" (Rachowicz et al., 2005). This scenario has not occurred on a global scale previously (Morse, 1995) and is a theoretical hypothesis based on extrapolation from the reasons for localized emergence of endemic pathogens. It is also a less parsimonious hypothesis.

There have been several papers discussing the strengths and weaknesses of the evidence for these scenarios (Daszak et al., 2003; McCallum, 2005; Rachowicz et al., 2005). Daszak et al. (2003) review the evidence and conclude that chytridiomycosis has caused amphibian population declines and that anthropogenic introduction appears to be a factor in its emergence. McCallum (2005)

states that the evidence for chytridiomycosis causing widespread amphibian declines is not conclusive and argues for the collection of more data on the biology of *Bd* which affects host population dynamics. He states that there is insufficient evidence to reject either of the above scenarios but does not discuss the evidence. Rachowicz et al. (2005) does not conclude that *Bd* is a novel pathogen and states that conclusive genetic evidence is needed before accepting this hypothesis. The scientific philosophy used in these latter two papers is called "refutationism" and is based on trying to disprove a hypothesis based on a particular line of evidence that tests the hypothesis. While we agree that it is important to test hypotheses, it is more important that action is taken to conserve species before the outcome of these rigorous tests are known.

Conservation has similar goals to medicine in that it tries to understand natural phenomenon quickly and manage them. Therefore, we have adopted a medical approach to determine causation (that includes inductive and deductive reasoning), and it is used successfully to determine likely causes of outbreaks leading to life-saving interventions (Dohoo et al., 2003). For example, John Snow determined the origin and method of transmission of cholera and suggested management techniques long before the discovery of the causal pathogen (Snow, 1860). This approach makes decisions and takes action based on the combined weight of all available evidence (a mental Bayesian approach) rather than waiting for proof of a hypothesis by a particular method. This example also highlights the importance of understanding the natural history of disease in order to determine causation, and the danger in relying on one line of evidence such as ecological correlations to test a hypothesis (Eyler, 2001). Therefore, we considered the approaches of multiple disciplines rather than relying solely on one, to provide a set of empirically testable postulates, which must be adequately satisfied before either scenario can be confidently accepted. These postulates are derived from current understanding of the factors which drive emergence of infectious disease, and they determine the effect that a disease has on host population dynamics, and traditional postulates that show causation of disease (Evans, 1976; Morse, 1995; Hudson et al., 2002). We conclude that the data for some evidence is not conclusive. Exceptions are likely and further research is crucial; however, there is currently enough combined evidence available to at least either strongly support or accept one hypothesis so that appropriate conservation management strategies can be undertaken (Australian Government Department of the Environment and Heritage, 2004).

Postulated Requirements for Scenario 1: Spreading Pathogen Hypothesis

a. The pathogen is capable of causing population decline and extinction. It is therefore highly pathogenic, virulent, and transmissible. (Disease modeling demonstrates that these are key factors for pathogens causing population decline and extinction [Anderson and May, 1979]. Infectious organisms may experience trade-offs between virulence and transmission that prevents them from causing extinction [McCallum and Dobson, 1995].)
b. The pathogen is spreading. Therefore, the declines and the pathogen show temporal–spatial patterns of spread, and the pathogen is highly conserved genetically due to its recent rapid spread. The pathogen also has at least one source from which it has emerged. (The spread of pathogens is the main reason for disease emergence [Morse, 1995]. Introduced pathogens may also be highly pathogenic and virulent because the host population is naive and has not undergone selection for resistance to them [Fenner and Ratcliffe, 1965].)
c. The pathogen is present during declines and mass mortality events and appears to be causing the mortality based on its epidemiology and pathogenesis. (This is the key factor that links the pathogen to the decline event [Wobeser, 1994].)

Evidence Bearing on Postulates for Scenario 1

a. *Bd* is highly pathogenic and virulent in many frog species experimentally; even initial low doses (e.g., 100 zoospores) have caused the rapid onset of 100% mortality (Berger et al., 1999, 2004; Woodhams et al., 2003; Carey et al., 2006). However, some species are resistant or developing resistance, and some populations of susceptible species, which live in adverse environmental conditions for *Bd*, are protected (Ardipradja, 2001; Daszak et al., 2004; Australian Government Department of the Environment and Heritage, 2004; Retallick et al., 2004; Weldon et al., 2004; Garner et al., 2005; McDonald et al., 2005; Pounds et al., 2006).
   Bd is highly transmissible given that it has a life-cycle stage that survives in water (Longcore et al., 1999). It can also survive on a wide range of larval and adult amphibian species at low intensities of infection without

causing mortality (McDonald et al., 2005; Hanselmann et al., 2005; Woodhams and Alford, 2005). These animals can act as reservoirs for transmission when host densities of vulnerable species are low, enabling *Bd* to drive species to extinction. In addition, the following combined evidence suggests *Bd* survives in the environment, which would further facilitate transmission when host densities are low by providing additional reservoirs of infection. *Bd* may survive in sterile environments for periods of up to 2 months, and it has been detected by PCR in the environment during mass mortality events (Johnson and Speare, 2003; Lips et al., 2006).

b. Studies of archived museum specimens and/or of recent mortality events suggest that *Bd* first appeared in amphibian populations immediately prior to their declines in USA (1974), Australia (1978), Costa Rica (1986), Panama (1996), and Venezuela (1986) (Berger et al., 1998; Lips, 1999; Speare et al., 2001; Bonaccorso et al., 2003; Puschendorf, 2003; Lips et al., 2006). Although data from most areas is not conclusive, the large study at El Copé, Panama, shows declines occurred when *Bd* appeared (Lips et al., 2006). Samples from 1566 amphibians collected from 2000 until July 2004 were negative when tested by PCR or histology. From September to December 2004, prevalence averaged 50% and the population declined abruptly (Lips et al., 2006). In Australia, the sudden appearance of Bd has been demonstrated by retrospective examination of museum specimens. The Australian epidemic appears to have commenced in southeast Queensland in the late 1970s (110 samples tested negative before 1978) before extending north and south along the coast (Australian Government Department of the Environment and Heritage, 2004). Chytridiomycosis in Western Australia was detected south of Perth in mid-1985 (612 earlier samples tested negative) and subsequently appeared to spread in all directions (Australian Government Department of the Environment and Heritage, 2004). After causing declines, *Bd* appears to settle into relatively stable endemic relationships in surviving frog populations, where it can always be detected in subsequent years in relatively small random samples of clinically healthy individuals, often at high prevalences (Berger et al., 2004; Retallick et al., 2004; McDonald et al., 2005). This is a similar sampling frame to museum specimens and gives confidence to the expectation that *Bd* will be present in museum specimens if it was present in the frog population at the time of collection. More work on archived collections of amphibians is needed to establish the historical distribution of *Bd*.

Bd is very highly conserved genetically and may be a clone given its extremely low genetic variation (Morehouse et al., 2003). Multilocus sequence typing was used to examine genetic diversity among fungal strains from North America, Panama, and Australia, and from frogs imported from Africa, and only five variable nucleotide positions were detected among 10 loci (5918 base pairs) (Morehouse et al., 2003). This is more consistent with *Bd* emerging recently and spreading globally than it is with long separate evolutionary histories of *Bd* in different continents. The genome of *Bd* has been sequenced, and examination of other parts of the genome of different geographical isolates should be undertaken to confirm its genetic homogeneity (Broad Institute of Harvard and MIT, 2006).

Declines associated with chytridiomycosis in Australia and the Americas have appeared to spread following relatively clear geographic and temporal patterns (Laurance et al., 1996; Berger et al., 1998; Lips, 1999). The emergence of chytridiomycosis in different continents and countries at different times is also consistent with geographic spread of the disease (Berger et al., 1998; Lips, 1999; Speare et al., 2001; Weldon et al., 2004; Lips et al., 2006). Therefore, priority should be given to the prevention of further spread of *Bd*.

Bd may have spread from southern African frogs. It has been found in samples collected in 1938 in South Africa, and the prevalence in African clawed frogs *(Xenopus laevis)* has remained stable since then, suggesting a stable endemic relationship between pathogen and host (Weldon et al., 2004). At present, this date precedes the earliest known dates for *Bd* in other continents by at least 23 years (Weldon et al., 2004; Ouellet et al., 2005). This suggests that *Bd* may have spread from Africa to most other continents in the 1960s and 1970s. This coincides with the general rapid growth in transportation of goods by air. Demonstration of higher levels of genetic diversity among isolates from southern Africa would support the hypothesis that *Bd* originated in that region.

c. Severe chytridiomycosis causing rapid death of frogs has been found associated with mass mortality of frog populations at the times of their declines in Australia, Panama, Costa Rica, and Spain (Berger et al., 1998; Lips, 1998, 1999; Bosch et al., 2001; Lips et al., 2006). The histopathological appearance of severe chytridiomycosis

causing death during declines is identical to that seen in frogs that die from experimental infections (Berger et al., 1998). This pathological evidence is traditionally used in medicine to link the pathogen to the cause of mortality in individuals. Furthermore, severe chytridiomycosis has not been found in healthy wild frogs demonstrating high odds of death due to the severe form of the disease (Berger et al., 2004, unpublished observations).

The epidemiology of chytridiomycosis is consistent with the pattern of mortalities and enigmatic frog declines. *Bd* is much more virulent in adults compared with tadpoles, and it is adults that have been affected in mass mortality events. *Bd* is a waterborne pathogen mostly infecting frogs associated with permanent water, particularly streams, and it is these species of frogs that have experienced the most severe declines. *Bd* is pathogenic and virulent over a broad range of temperatures (12°–27°C) but has its greatest virulence at temperatures from 12°–23°C (Berger et al., 1998, 2004; Longcore et al., 1999; Woodhams et al., 2003; Carey et al., 2006). Pathogenicity and virulence decreases significantly as temperatures are raised above 27°C. It is frog species that occur at temperatures consistently below this upper threshold for *Bd*, which have been most affected (Berger et al., 1998, 2004; McDonald et al., 2005; Pounds et al., 2006). The lower temperature threshold for the virulence of *Bd* is not known, but temperate species have experienced less extinction compared with sub-tropical and tropical species of frogs. However, several determinants of disease and extinction other than temperature, such as size and distribution of populations, could account for these differences. In North America and Spain, *Bd* has its greatest effect on alpine species that experience freezing temperatures during winter (Bosch et al., 2001; Muths et al., 2003; Scherer et al., 2005).

Postulated Requirements for Scenario 2: Endemic Pathogen Hypothesis

a. The pathogen becomes highly pathogenic, virulent, and transmissible when abnormal environmental changes occur. (The pathogen may already have some of these characteristics but not all prior to the environmental change [Morse, 1995].)
b. The pathogen is endemic. Therefore, the declines and the pathogen do not show temporal–spatial patterns of spread unless the environmental change has a pattern of spread and the pathogen has geographical genetic variation consistent with genetic drift due to isolation and differences in selection pressure. The pathogen does not have a recent origin. (Typically, endemic pathogens may cause cyclical epidemics when immunity wanes or when particularly favorable environmental conditions for the pathogen occur [Thrushfield, 2005].)
c. Abnormal environmental changes occur immediately prior to and during pathogen outbreaks.
d. The pathogen continues to be present during declines and mass mortality events, and appears to be causing the mortality based on its epidemiology.

Evidence Bearing on Postulates for Scenario 2

a. There is strong evidence that the prevalence and virulence of *Bd* is determined by environmental conditions and is favored by moderate temperatures, such as those between 12° and 27°C, and environments with permanent water, particularly streams (Berger et al., 1998, 2004, in press; Longcore et al., 1999; Woodhams et al., 2003; Carey et al., 2006; Lips et al., 2006). These factors may determine the precise timing of increases in incidence of chytridiomycosis when it is endemic, such as the dramatic increase in prevalence in winter at low elevations in northern Queensland associated with temperatures dropping well below the upper threshold of tolerance for *Bd* (Berger et al., 1998, 2004; Woodhams et al., 2003; Retallick et al., 2004; McDonald et al., 2005; Woodhams and Alford, 2005). The environment will also affect the severity of an epidemic when *Bd* is introduced into a naive population. However, it will be a less important determinant compared with when the disease is endemic and selection for resistance has occurred (Thrusfield, 2005).

   The above evidence shows that abnormal climatic conditions are not necessary for *Bd* to become highly pathogenic, virulent, and transmissible. In stark contrast, there is evidence to accept the opposing hypothesis, that environmental change is having a protective effect for frog populations threatened by chytridiomycosis. The survival of remnant populations of the green and golden bell frog *(Litoria aurea)* has occurred at some contaminated sites (e.g., a gold mine, copper smelter), suggesting that some pollutants may have antifungal effects on *Bd* and is currently being studied (Department of Environment and Conservation NSW, 2005; Berger et al., in press).

b. There is evidence that *Bd* has not been endemic for substantial periods before the onset of declines. Retrospective and contemporary surveys of specimens from areas where declines have occurred in Australia and Panama provide evidence to reject the hypothesis that chytridiomycosis was endemic prior to declines (Australian Government Department of the Environment and Heritage, 2004; Lips et al., 2006). There is also evidence of a temporal–spatial spread of declines and chytridiomycosis but no environmental change such as climate change or increased UV-B radiation associated with these patterns (Laurance et al., 1996; Berger et al., 1998; McDonald and Alford, 1999; Lips et al., 2006). As discussed for the first hypothesis, there is genetic evidence to suggest that geographically separated isolates share a very recent ancestor and therefore are recently introduced (Morehouse et al., 2003). As stated above, there is evidence that the pathogen may have originated recently from Africa.

c. There have been no consistent abnormal environmental changes immediately prior to and during enigmatic declines globally, which could have precipitated an unprecedented increase in pathogenicity, virulence, and transmissibility of *Bd* (Laurance et al., 1996; Alexander and Eischeid, 2001; Stuart et al., 2004). Pounds et al. (2006) present a correlation between warmer years associated with global warming and the last year a species of *Atelopus* was seen. There are alternate hypotheses regarding mechanisms for global warming as a key factor in driving extinctions due to chytridiomycosis: 1) that it promotes the spread of *Bd;* or 2) that it significantly increases its virulence.

The plausible methods of spread of *Bd* are movement of infected hosts or contaminated fomites such as water either naturally or via trade. However, if one could show that *Bd* appeared in populations for the first time during unusually warm years, then it suggests a role for global warming in spreading *Bd*. Pounds et al. (2006) provide an explanation for the other hypothesis—how global warming could increase the virulence of *Bd*. They suggest that chytridiomycosis outbreaks are facilitated in warmer years that are due to global warming by higher minimum and lower maximum temperatures, which may be optimal for the disease. A way to provide evidence for this relationship would be to demonstrate that *Bd* had been present within a population for a short period previously and that outbreaks did not occur until warmer conditions due to global warming were present. One would still need to show that outbreaks were more severe due to the conditions created by global warming. This hypothesis is still dependent on the pathogen being introduced, as the annual climatic conditions associated with climate change, that have been suggested as being associated with outbreaks of chytridiomycosis, have occurred previously. The only change has been a long-term trend of more warmer-than-average years. If the pathogen was endemic and warmer years precipitated outbreaks, then one would expect an increased frequency of outbreaks, not novel epidemics.

Unfortunately, the evidence of Pounds et al. (2006) is limited. They only provide a correlation of a warmer year with the last year a species is seen in the following year. This is not a correlation with the timing of declines due to chytridiomycosis, as the exact timing of declines cannot be determined from the data set. However, for the benefit of testing their hypothesis, let us assume that declines due to chytridiomycosis occur in a species in a warmer year. Data on the history of chytridiomycosis in these species is still needed to determine whether the timing of the declines is determined by increased virulence or spread of *Bd*. One could then try to determine the role of climate change. If it is not possible to obtain this data, then there are alternative methods for testing the role of climate change. One could test the effects of climate change on the virulence of *Bd* by studying frog populations where chytridiomycosis has become endemic. We are currently doing this within the tropics of Queensland. The prevalence of chytridiomycosis is largely dependent on seasonal temperature changes with consistently higher prevalence in winter compared with summer (range of odds ratios, 4.8–5.7) in the tropics (Retallick et al., 2004; McDonald et al., 2005; Woodhams and Alford, 2005) [Skerratt et al., unpublished observations]. The change in mean daily minimum and maximum temperatures between summer and winter in the Queensland tropics can be 6°–8°C. The important change is likely to be the drop in the mean daily maximum temperature from temperatures that are lethal to *Bd*, during summer, to temperatures that are within its preferred range in winter. This temperature change is a much greater change compared with the temperature changes seen with global warming, yet it only increases the prevalence of the disease and does not result in large declines of species susceptible to chytridiomycosis. Therefore, current data suggests that the changes seen with climate change do not explain the emergence of *Bd*.

In addition, the yearly average prevalence of chytridiomycosis has declined in populations where it is endemic in the Wet Tropics in Queensland over time despite drier than average years and global warming (McDonald et al., 2005). In cases where the timing of declines is known, they are not correlated with climate change (Laurance et al., 1996; McDonald and Alford, 1999; Bell et al., 2004; Scherer et al., 2005).

Lastly, given that the pathogenicity and virulence of *Bd* occurs over a broad experimental temperature range, then minor temperature changes associated with climate change are likely to have a minor effect unless they are strongly related to the spread of *Bd* (Berger et al., 1998, 2004; Carey et al., 2006). Because *Bd* is continuing to spread, it is possible to investigate the role of climate change. However, while it is important to consider the effects of climate change on chytridiomycosis, it is more important that the utmost is done to prevent the further spread of *Bd* into naive frog populations.

d. The evidence that the pathogen is present during declines and causes the mortality is accepted and is consistent with both hypotheses.

Discussion

We show that there is strong evidence to implicate chytridiomycosis caused by *Bd* as the major primary cause of extinctions and declines of anurans that have not been due to obvious causes, such as habitat loss described by Stuart et al. (2004). Further, we show that the evidence supports the hypothesis that these extinctions and declines are a result of the pathogenic, virulent, and highly transmissible amphibian chytrid fungus, *Bd*, spreading into naive populations. There is strong evidence against the theoretical and less parsimonious hypothesis that global environmental change has caused the emergence of *Bd* and that *Bd* was an endemic pathogen in declining populations. It is therefore important for conservation agencies to direct their major resources and energy towards research on the control and management of chytridiomycosis. In Australia, this has already occurred; *Bd* has been accepted by the federal government as a ''Key Threatening Process'' in 2002, and a draft Threat Abatement Plan was produced in 2004 and finalized in 2006 (Australian Government Department of the Environment and Heritage, 2004, 2006a, b). Recommendations in this plan cover quarantine issues, endangered species recovery projects, and future research. It has led to major funding (several million dollars) from the Australian Government Department of the Environment and Heritage, and the Australian Research Council for research and for the development of guidelines on determining the distribution, preventing the spread, and understanding the epidemiology and pathogenesis of chytridiomycosis. This work is ongoing but some of the research has been published in scientific journals, as government reports, and on the Amphibian Diseases Home Page (Speare et al., 2005; Speare, 2006; Berger et al., in press). Knowledge of the epidemiology and pathogenesis of *Bd* is essential in attempts to prevent further declines and in planning captive husbandry, selection for resistance, and reintroduction as part of species recovery projects. It is important for other nations to develop their own control plans.

Conservation agencies outside of Australia have been comparatively slow to recognize the threat of chytridiomycosis. It is now 10 years since an infectious disease was proposed as the cause of frog declines in Australia, and 8 years since the initial discovery of chytridiomycosis by Berger et al.(1998) as the cause of frog declines and extinction in Australia and Panama. We believe this slow response reflects a lack of recognition of the importance of disease in regulating wildlife populations by conservation science (Wilcox, 2006). While there has been a generally increasing theoretical interest in ecology of disease in free living populations over the past 30 years, there have been few empirical studies (Hudson et al., 2002).

There are old lessons that need to be re-learned from amphibian declines. One is that emerging diseases can cause rapid decline and extinction of species in pristine habitats. Diseases have caused population declines and have been associated with extinctions previously (Daszak et al., 2000). However, this is the first time that an emerging disease has been documented to cause the decline or extinction of hundreds of species not otherwise threatened. The threat of invasive plants and pests to conservation is well known. The fact that parasites, both macro- and micro-, could cause similar devastation is not a great leap of logic. Another lesson to be learned is that this pathogen has been able to emerge worldwide very quickly, causing widespread declines and extinctions. It appears very likely that one of the causes of this emergence is movement of the pathogen, probably aided by human transport. Globalization without adequate quarantine, surveillance, and parasite control programs will result in further outbreaks of disease in wildlife populations and loss of biodiversity. Finally, there is an obvious need for a global strategy to abate the threat of chytridiomycosis (Skerratt et al., 2006). At

present, evidence suggests that there should be substantial effort directed towards preventing the spread of *Bd* into new geographic regions. Therefore, there is an urgent need to map the present and past global distribution of *Bd*, targeting large unsurveyed areas such as Asia and improving the biosecurity of frog populations found to be free of *Bd*. This will involve actions such as implementing quarantine and health screening protocols for amphibian trade.

There are two international bodies, the Office International des Epizooties (OIE) and the World Conservation Union (IUCN), which have interests and expertise in controlling diseases and conserving species, respectively. These bodies have started to develop strategies but need to work together to coordinate a global plan to control chytridiomycosis. The Amphibian Conservation Summit in Washington in 2005 called for major funding for research on chytridiomycosis (Mendelson et al., 2006). It suggested that regional diagnostic centers and rapid response teams be set up to deal with outbreaks. Chytridiomycosis was added to the list of wildlife diseases of concern of the OIE in 2001 (OIE Working Group on Wildlife Diseases, 2001–2005), and Williams et al. (2002) proposed that it be listed as a notifiable disease with consequent testing requirements for import and export of amphibians. This has not happened, but the OIE Working Group on Wildlife Diseases has sent a questionnaire to member countries each year, since 2002, asking whether chytridiomycosis is present or absent and the number of animals affected each year (OIE Working Group on Wildlife Diseases, 2001–2005). Guidelines for screening amphibians for diseases in translocation programs have been formulated by the Veterinary Specialist Group within the IUCN (Cunningham et al., 2001). The Aquatic Animal Health Standards Commission of the OIE, to enable it to address the issue of the spread of amphibian diseases, produced a questionnaire which was sent to Member Countries, in order to get more information on amphibian trade and amphibian health in different parts of the world; they have formed an ad hoc Group on Amphibian Diseases (OIE Fish Diseases Commission, 2002; OIE Aquatic Animal Health Standards Commission 2003, 2006). A forum on chytridiomycosis involving government and non-government organizations and individuals concerned with controlling the effects of disease on biodiversity, led by the OIE and IUCN, would facilitate international and national strategies and enable development of a rapid communication system of research results to inform and update policy.

Acknowledgments

We thank R.A. Alford, R. Puschendorf, E. Molina, V. Hemingway, R. Campbell, L. Owens, Kris Murray, Jodi Rowley, Jamie Voyles, two anonymous referees, and the specialist editor for critical review of the paper. The financial support from the Australian Government Department of the Environment and Heritage, the Australian Research Council, and James Cook University made this review possible.

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