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SPECIAL ISSUE: AMPHIBIAN DECLINES

Infectious disease and amphibian population declines

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Abstract. A series of recent papers have implicated pathogens and parasites in amphibian population declines. Here, we review evidence on the link between infectious disease and amphibian population declines. We conclude that available data provide the clearest link for the fungal disease amphibian chytridiomycosis, although other pathogens are also implicated. We suggest additional experimental and observational data that need to be collected to provide further support that these other pathogens are associated with declines. We suggest that, in common with many emerging infectious diseases (EIDs) of humans, domestic animals and other wildlife species, emergence of chytridiomycosis may be driven by anthropogenic introduction (pathogen pollution). Finally, we review a number of recent advances in the host–parasite ecology of chytridiomycosis that help explain its emergence and impact.

Key words. amphibian declines, conservation medicine, emerging diseases, global change, pathogens.

DISEASE AND DECLINES: DEMONSTRATING THE LINK

Recently, there has been growing interest in infectious diseases and their role in global amphibian declines (Daszak *et al*., 1999). In particular, chytridiomycosis, ranavirus disease, saprolegniosis and *Ribeiroia* sp. infection have been implicated tentatively as the cause of heightened mortality leading to amphibian population declines (Carey, 1993; Berger *et al*., 1998; Daszak *et al*., 1999; Johnson *et al*., 1999; Kiesecker *et al*., 2001). Demonstration of a link between declines and these infectious diseases is a challenging task that requires:

1. Fulfilment of Koch's postulates for pathogens hypothesized to cause disease in amphibians.
2. Identification of the causative pathogen in carcasses from mortality events that constitute part of a decline in population (mortality events may consist of mass die-offs or subtle increases in mortality rates).
3. Pathological evidence that the disease caused death in a significant number of cases within these mortality events (i.e. demonstration of gross, cellular or tissue damage concomitant with infection and with a valid mechanism for a cause of death proposed).
4. Clear evidence that the mortalities are the cause of declines.

These criteria are difficult to fulfil because they require collaborative research over long periods between scientists from different disciplines. For example, criterion 1 requires isolation of the pathogen in culture, rigorously planned infection experiments under biosecurity conditions and veterinary pathological analysis. Criterion 2 requires collection of a significant number of carcasses at the time of mass mortality events, or collection of carcasses over prolonged periods for declines where enzootic diseases may be slowly decreasing population size. Criterion 3 usually

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involves collaboration between microbiologists that understand the taxonomic and life histories of proposed pathogens and veterinary pathologists that can identify them as the cause of death. This in itself is a complex process that involves attributing the pathological changes seen in the tissues to the proposed pathogen and then elucidating a link between the lesions and death. Criterion 4 requires collection of ecological and microbiological data from declining populations over a temporal gradient. Finally, demonstrating the link between any pathogen and declines requires evidence (normally collected by ecologists and population biologists) that population reductions are not simply part of a normal cyclical of population fluctuations – itself a common life history trait of amphibians (Pechmann *et al*., 1991; Alford & Richards, 1999).

Analysis of published information on the pathogens listed above reveals gaps in these four criteria for almost all pathogens hypothesized to cause declines. The evidence for chytridiomycosis is discussed in the next section. Of the other pathogens, saprolegniosis is probably the most promising candidate as a cause of amphibian declines. *Saprolegnia* water moulds have been reported as a cause of amphibian egg mortality, and proposed as a cause of decline of the boreal toad (*Bufo boreas*) and the Cascades frog (*Rana cascadae*) in the Pacific North-west (U.S.A.) as part of a complex interaction involving climate change, ozone depletion and increased UV-B irradiation (Kiesecker & Blaustein, 1995; Kiesecker *et al*., 2001). This process is described further in a separate paper in this volume (Blaustein, 2003). Experimental and observational data implicate *Saprolegnia ferax* in egg mortality, and the link between such mortality and declines is suggested (Blaustein *et al*., 1994). Demonstrating this link more clearly will require a temporal analysis of saprolegniosis incidence (not *Saprolegnia* sp. prevalence) in declining populations, coupled with evidence that loss of egg masses is responsible for declines.

Ranaviruses (genus, *Ranavirus*, family Irodoviridae) comprise another important group of amphibian pathogens. These large, linear, double-stranded DNA viruses are known to infect amphibians, reptiles and fish, in which they are often highly virulent (Ahne *et al*., 1997; Mao *et al*., 1997; Hyatt *et al*., 1998; Daszak *et al*., 1999; Mao *et al*., 1999; Hyatt *et al*., 2001). Transmission studies (e.g. Hyatt *et al*., 1998; Langdon, 1989; Jancovich *et al*., 2001; Cullen & Owens, 2002) demonstrate that, within individual host species, some life stages are more susceptible than others and that some ranaviruses can cross animal orders, or even classes, while others appear to be more species-specific.

There has been a large increase in the number of new ranaviruses reported in the scientific literature in recent years (Hyatt *et al*., 2001), but there have been only two situations where these pathogens have been associated with significant levels of mortality in populations of wild amphibians. These circumstances involve deaths of (i) tiger salamanders (*Ambystoma tigrinum*) in Arizona, U.S.A. and in Saskatchewan, Canada (Jancovich *et al*., 1997; Bollinger *et al*., 1999; Jancovich *et al*., 2001) and (ii) common frogs (*Rana temporaria*) in the United Kingdom (Cunningham *et al*., 1996; Cunningham, 2001).

In North America, ranaviruses have been reported as aetiological agents associated with mortalities of tiger salamanders: an isolate called ATV, isolated first in Arizona (U.S.A.) (Jancovich *et al*., 1997, 2001) and an isolate named RRV, first isolated in Sakatchewan (Canada) (Bollinger *et al*., 1999). To date, these ranaviruses have been isolated from diseased tiger salamanders from six U.S. states and two Canadian provinces (Jancovich *et al*., 2001). While studies in Arizona and Saskatchewan have shown that infected populations undergo dramatic fluctuations, to date population declines (as opposed to mass mortalities) have not been reported. It has been proposed that viral mortality drives a normal, cyclical variation in population density at most sites, despite mortality events that often result in many thousands of individuals killed, loss of around 95% of a year class or complete removal of a year class (Carey *et al*., 2003). Similarly, in the United Kingdom, annual epidemics of ranavirus disease result in the deaths of large numbers (tens of thousands) of common frogs each year, but there is little evidence of associated population declines (Cunningham, 2001). It may be that an interplay between differential susceptibility of age classes, coupled with cycles of viral virulence and attenuation underlie this phenomenon.

Although ranaviruses are associated currently with amphibian mortalities, but not declines,

the potential for ranaviruses to impact amphibian populations significantly should not be dismissed. Ranaviruses are highly infective to a range of animal orders and species and have the potential to persist in the environment, and further evidence may implicate ranaviruses in population declines at some sites. For example, population depression of tiger salamanders does seem to have occurred at one Saskatchewan site following an outbreak of RRV disease in 1998 (Carey *et al*., 2003). The unregulated trade and movement of animals that are potentially infected with ranaviruses will undoubtedly result in the potential exposure of naive species and populations to these virulent pathogens, with possible outcomes of conservation concern (Cunningham *et al*., 2003).

The trematode parasite, *Ribeiroia ondatrae*, has been shown to cause deformities of some amphibian metamorphs (Johnson *et al*., 1999). At a small number of sites studied deformities occur at high incidence (e.g. 90% in some species at some sites — Johnson *et al*., 2002). However, no data have shown so far that parasite-caused deformities have caused mortality rates that are higher than normal in nature, or that the incidence of deformities seen has increased recently, concomitant with declines. Furthermore, data have not been published demonstrating that the populations with high incidence of deformities are undergoing declines.

CHYTRIDIOMYCOSIS: AN EMERGING DISEASE RESPONSIBLE FOR SOME AMPHIBIAN DECLINES

Chytridiomycosis provides the clearest link between disease and declines (Berger *et al*., 1998; Daszak *et al*., 1999), albeit that this is demonstrated in a very small number of cases, usually montane amphibian species, and more data are needed urgently to assess its impact at other sites. Two publications have assessed the link between chytridiomycosis and declines by analysing pathological samples collected in time-series before and during declines. Firstly Green & Kagarise-Sherman (2001) demonstrated pathological findings consistent with fatal chytridiomycosis in two archived Yosemite toad (*Bufo canorus*) individuals collected during a large-scale decline in the U.S.A. These mortalities were attributed previously to bacterial infection (Carey, 1993), but this conclusion was reached without pathological analysis, and prior to the discovery of chytridiomycosis. Green & Kagarise-Sherman (2001) examined museum specimens collected during declines and demonstrated presence of the fungal parasite that causes chytridiomycosis, *Batrachochytrium dendrobatidis*. However, severe lesions caused by *B. dendrobatidis* were observed in only two of twelve adult amphibians collected. Secondly, amphibian populations in Australia and Panama appear to have undergone significant and long-term declines at the time that severe chytridiomycosis was found by veterinary pathological analysis of carcasses (Berger *et al*., 1998; Lips, 1998, 1999). Here, chytridiomycosis was demonstrated as the cause of death in more than 90% of carcasses. To support a link between declines and pathogens, it would be desirable to demonstrate that this heightened mortality and the causative pathogen were absent beforehand. For the second study samples were analysed from museum specimens archived prior to the onset of declines, and *B. dendrobatidis* was not observed (Berger *et al*., 1998).

Thus only one pathological study that includes museum specimens collected prior to declines has provided clear evidence for a link between chytridiomycosis and declines, albeit that this study covered two continents. Further studies of extant populations may allow a clearer understanding of impact. For example, McCallum & Dobson (1995) show how simple prevalence surveys can usefully demonstrate a parasites impact on a population if they include moribund animals. If the parasite prevalence is much higher in moribund animals than in the general population, it can be concluded that the parasite is causing substantial mortality. They also point out that definitive demonstration of impact will require manipulation experiments (e.g. treatment and eradication of the pathogen from one population).

Daszak *et al*. (1999, 2000, 2001) proposed that chytridiomycosis is a key representative of a growing cohort of wildlife emerging infectious diseases (EIDs). The Institute of Medicine (Lederberg *et al*., 1992), Morse (1993) and others define EIDs as diseases that are, or have recently, increased in incidence, impact, pathogenicity, geographical or host range. Also included are

pathogens that have recently evolved such as new strains of influenza, or the pandemic strain of canine parvovirus. However, these are a rarity, and most EIDs are caused by ancient microbes that jump host or expand range following environmental changes (Daszak *et al*., 2001). What characters of chytridiomycosis define it as an 'emerging' disease? First, it has recently increased in range. Discovery of this disease has led to increased attention to amphibian pathology and an increase in searches for the pathogen. However, the appearance of this disease associated with mass mortality events in south-western Australia and New Zealand (for example) was not due to increased surveillance, and the pathogen was not located in studies of tissues archived prior to the emergence event (Alpin & Kirkpatrick, 2000; Waldman *et al*., 2001). In New Zealand, chytridiomycosis was first diagnosed in the endemic Archey's frog (*Leiopelma archeyi*) in 2001, which has been declining for the past decade (Waldman *et al*., 2001). Importantly, New Zealand's four endemic anuran species include two highly vulnerable taxa: Maud Island frog (*Leiopelma pakeka*), which has a global population of about 20 000 individuals, and Hamilton's frog (*Leiopelma hamiltoni*), which has fewer than 300 left and is the world's rarest frog (Bell & Bell, 1994; Bell *et al*., 1998). Both are confined to small islands within New Zealand and both are at risk from an ongoing epizootic of chytridiomycosis, should the disease be introduced onto these islands. Secondly, chytridiomycosis appears to have recently (over the past few decades) increased in impact. In particular, the chytridiomycosis-related mass mortalities in Panama and the Tablelands (Australia) are associated with severe, long-term declines and local extinctions (Mahony, 1996; Lips, 1998; Williams & Hero, 1998; Lips, 1999). It appears unlikely that such declines could have been sustained over prolonged historical periods without previous significant species extinctions, suggesting that they are a recent phenomenon. Thirdly, phylogeographical data (Daszak *et al.* unpublished data) suggests that there has been recent movement of *B. dendrobatidis* between disparate populations of amphibians (i.e. range expansion of isolates). Sequences from the internally transcribed spacer region of *B. dendrobatidis* isolates from Australia and North, Central and South America show around 10% sequence divergence. However, a number of isolates from different continents have identical sequence or poor correlation between origin and position on the phylogenetic tree, suggesting recent transport of the pathogen between regions.

Since *B. dendrobatidis* was described from Eastern Australia and Costa Rica and from captive dendrobatids at the U.S. National Zoo in 1998 (Berger *et al*., 1998; Pessier *et al*., 1999), increased surveillance has expanded the known geographical range and number of species known to be infected with this parasite (Carey *et al*. 2003; www.jcu.edu.au/Department/PHTM/frogs/ampdis.htm). However, at sites where monitoring of amphibian populations has occurred historically and recent chytridiomycosis-induced declines have been reported (e.g. Central America, Queensland Australia and Spain) it is likely that the disease has emerged recently (i.e. increased its host or geographical range, or impact). *Batrachochytrium dendrobatidis* has recently been reported from Spain (Bosch *et al*., 2001), south-western Australia (Alpin & Kirkpatrick, 2000) and New Zealand (Waldman *et al*., 2001), and has apparently caused mass mortality and declines of the midwife toad (*Alytes obstetricans*) (Spain) and endemic frogs from New Zealand. Retrospective studies using museum specimens have found *B. dendrobatidis* infection in *Bufo canorus* in North America (see also above), and in African clawed frogs (*Xenopus laevis*) in Southern Africa (Carey *et al*., 2003). In the latter case, an association between infection and clinical signs or mortality has not yet been found and it may be that *X. laevis* is relatively resistant to this parasite. In summary, since the first reports of chytridiomycosis in 1998, wild amphibians have been found with chytrid infection in at least five continents, namely North America, South America, Australia, Europe and Africa, with infection associated with mass mortalities and declines in the first four.

What factors have driven, and continue to drive the spread of chytridiomycosis? This question is central to the study of emerging diseases, because all EIDs are linked to anthropogenic environmental changes that foster increased transmission within or between hosts (Daszak & Cunningham, 2003). In the case of chytridiomycosis, only one factor has been implicated so far

in forcing emergence — the anthropogenic introduction of this disease to new regions and host species (Daszak *et al*., 2001; Dobson & Foufopoulos, 2001; Cunningham *et al*., 2003; Daszak & Cunningham, 2003). This process, termed 'pathogen pollution' (Daszak *et al*., 2000), underlies emergence of a suite of human EIDs, and the majority of wildlife EIDs for which driving factors have been identified (Dobson & Foufopoulos, 2001; Daszak & Cunningham, 2002). Pathogen pollution is an under-reported form of anthropogenic environmental change that can have severe consequences for ecosystems, similar to those caused by introduction of other exotics (Cunningham *et al*., 2003). In particular, it is recognized increasingly as a cause of biodiversity loss through outbreaks causing mass mortality and declines in naive species (Cunningham *et al*., 2003).

What evidence is there for pathogen pollution driving chytridiomycosis? First, chytridiomycosis has been identified in five forms of amphibian trade or anthropogenic movement: pet trade; movement of zoo animals; food trade; laboratory animal trade (e.g. *Xenopus* in the U.S.A. Reed *et al.*, 2000) and inadvertent or deliberate introduction of amphibians to new regions via releases of pet species or laboratory animals into the wild, or use as biocontrol agents (e.g. Cane toads (*Bufo marinus*) in Hawai'i and Australia; Daszak *et al*., 2001). These trades have expanded during the last 20–40 years and have globalized, with central collection points and more efficient transport of large numbers of live specimens due to increased use of air travel. The volume of trade is large, with 1 million bullfrogs imported into the U.S.A. every year for the food trade, for example (Cunningham *et al*., 2003). Secondly, unpublished data on the DNA sequence phylogeny of *B. dendrobatidis* suggests strongly that recent mixing between populations has occurred (Daszak *et al.* unpublished data). Finally, the pattern of declines associated with chytridiomycosis (catastrophic population declines that occurred rapidly over large areas, with disjunct temporal and spatial progression to reported outbreaks) was remarkably consistent with the introduction of virulent pathogens into naive populations (Daszak *et al*., 1999). These data support the introduction hypothesis, but it is likely that the situation is more complex, with introduction being only one of a range of environmental factors ('cofactors') that promote emergence (see Fig. 1). Although data are still lacking in many cases, hypothetical links between increased UV-B, stress, immunosuppression, pollution and climate change have been proposed and are being tested. For example, Pounds *et al*. (1999) reported evidence that amphibian, reptilian and avian population declines at Monteverde, Costa Rica, were a response to climate change. Here, evidence suggests that increased numbers of dry days and longer dry periods were caused by a climate change-induced rise in the altitude at which cloud formed and resulted in the extirpation of a number of species. Pounds *et al*. (1999) hypothesized that, as the environment dried, amphibian population density increased in moist areas and led to a heightened impact from waterborne diseases such as chytridiomycosis.

ECOLOGICAL EXPLANATIONS OF THE IMPACT OF CHYTRIDIOMYCOSIS

Chytridiomycosis is an extreme example of the impact of wildlife EIDs on biodiversity. It is implicated in epizootic mass mortality on a global scale, has a wide host range comprising a class of vertebrates, and is linked to species declines, local population extinctions and species extinctions. In temperate and tropical montane amphibian species, it has been linked to mass mortalities, declines and hypothesized as the cause of species extinctions. For example, Pounds *et al*. (1999) suggest that chytridiomycosis may have been the proximal cause of a climate change-driven extinction of the golden toad (*Bufo periglenes*) in Costa Rica. Despite earlier reports (Daszak & Cunningham, 1999), extensive searches have failed to locate the sharp-snouted frog (*Taudactylus acutirostris*) (J.M. Hero, personal communication) and this species is almost certainly extinct. The last few specimens seen by humans were larvae, collected from a site at which the adults had died of chytridiomycosis. These larvae were brought into captivity, where they metamorphosed and also died of chytridiomycosis.

Why is this disease so devastating to amphibian populations? Daszak *et al*. (1999) proposed a simple population pyramid model to explain the impact of chytridiomycosis. In this scenario (Fig. 1), host ecological traits (naive populations

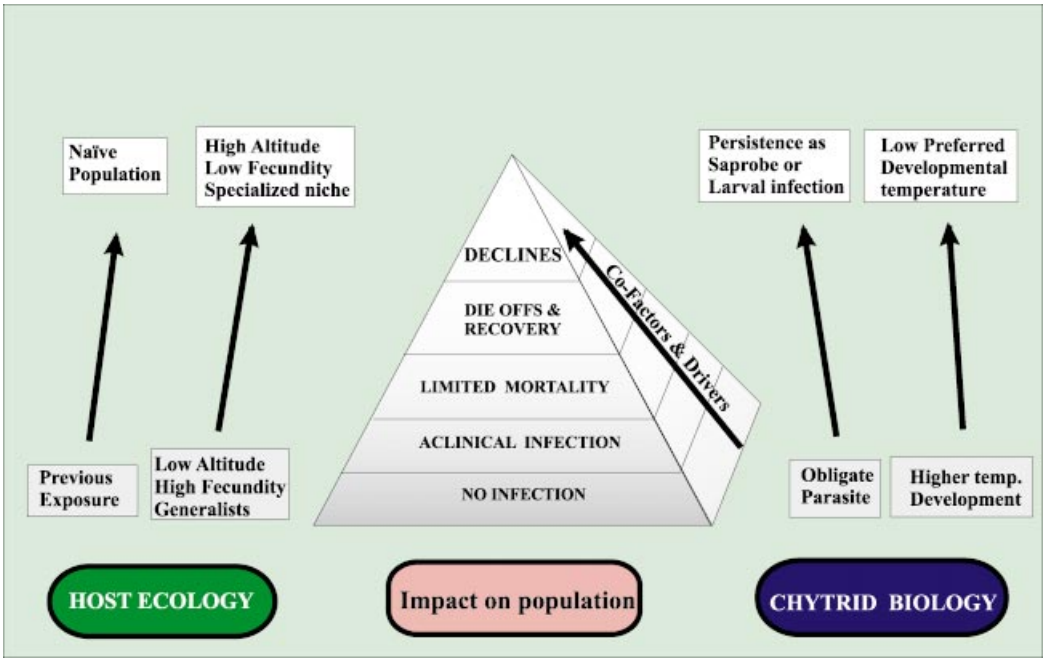

Fig. 1 A diagrammatic model describing hypotheses on the impact of chytridiomycosis on amphibian populations. We propose that host ecological or life history traits (left) and biological characters of the pathogen (right) are likely to produce a range of effects on different amphibian species and populations (centre), some of which have been reported recently (see text). The range of outcomes progresses from: no infection in the presence of the pathogen (i.e. a resistant species); through to presence of infected animals, but no disease (aclinical infection); disease presence resulting in limited mortality; populations in which infection causes mass mortality events but with subsequent recovery; and populations in which the pathogen causes declines, local extinctions and species extinctions. In this model chytridiomycosis appears to cause declines and extinctions in a select group of amphibians predisposed to heightened impact by certain life history traits. Note that disease outbreaks may be driven by a range of cofactors and drivers. These include disease introduction (pathogen pollution) and climate change, both of which are supported by some data. Hypothesized cofactors include stress, pollution, increased levels of UV-B light and others. This diagram is adapted from Daszak *et al*. (1999), following the ecological terminology and concepts in Williams & Hero (1998).

of high altitude, stream breeding, low fecundity, habitat specialist amphibians) and the biological traits of *B. dendrobatidis* (more rapid growth in cool temperatures, wide host range, high virulence, aclinical infection in larvae, potential ability to survive outside the host in the wild) combine to produce local extinctions in a small proportion of amphibian species that are susceptible. This hypothetical scenario was based partly on the few studies of *B. dendrobatidis* biology (e.g. Longcore *et al*., 1999) and on ecological studies of sympatric, declining and non-declining species (Williams & Hero, 1998). Data collected in the last 2 years have begun to provide support to the following aspects of the pyramid model of Daszak *et al*. (1999):

1. Decline population tend to be naive: The recent emergence of the disease in Western Australia, Spain and New Zealand (see above), associated with high mortality rates and rapid population declines suggest introduction into naïve populations. This is still a hypothetical point, since serological surveys for evidence that amphibians had previously been infected (and developed some immunity)

have not yet been undertaken in wild populations. However, only the retrospective studies described by Berger *et al*. (1998) suggest that the pathogen was not present previously.

2. Increased impact on low fecundity species: in Australia, a differential impact appears to occur between high and low fecundity sympatric species. For example, populations of the highly fecund *Bufo marinus* are not declining in the wild, although individuals have died of chytridiomycosis following experimental infection (Daszak *et al*., 1999; Hyatt, unpublished data). This is a predictable outcome for a virulent pathogen moving into and through naive populations because fecund species have a greater potential to survive extreme depopulation events. Assuming that the pathogen kills susceptible animals in both populations at the same rate, re-invasion of the pathogen would reduce an increased proportion of the now smaller number of the low fecundity species' offspring. It may be that highly fecund species such as *B. marinus* resist chytridiomycosis-induced declines by very high recruitment rates. However, the lack of observed impact of chytridiomycosis on *B. marinus* populations may also be due to the largely lowland distribution of this species in Australia, where higher temperatures would likely suppress chytridiomycosis (see below).
3. Temperature: data collected using cultures of *B. dendrobatidis* suggest optimum pathogen growth at 23 °C, stasis at 28–29 °C and death at 30 °C *in vitro* (Longcore *et al*., 1999). Experimental infection of susceptible dendrobatid frogs show that temperatures in the upper 20 °Cs reversibly halt the progression of disease (Nichols, personal communication). In the laboratory, cultures kept at 4 °C prolong their productive life significantly (Daszak, unpublished observation). Boyle *et al*. (2003) have reported a cryopreservation protocol for *B. dendrobatidis*. It is unknown if the pathogen is able to survive freezing in nature; however, it occurs in the Rocky mountains, Maine and Ontario in North America, all of which are regions that are regularly snow-covered during winter months. The ability of *B. dendrobatidis* to survive cold temperatures and its increased growth under relatively cool environmental conditions may explain the late winter–spring mortality events characteristic of declines in the Colorado Rockies, the heightened impact on high altitude populations and the relative lack of declines noticed at low altitudes in the tropics (Daszak *et al*., 1999).
4. Wide host range: *B. dendrobatidis* has now been reported from 13 amphibian families including frogs, toads and salamanders in the wild and in captivity. Carey *et al*. (2003) provide a current list of host species and the Amphibian Disease website (www.jcu.edu.au/department/PHTM/frogs/ampdis.htm) updates regularly the geographical and host reports for this pathogen. Notably, this fungus has now been identified in salamanders collected from the wild (Davidson *et al*., 2003), whereas in the past it had been reported only in an imported captive species of salamander. The field salamander isolate was able to infect and kill anurans, demonstrating that the wide host range may not be isolate-specific.
5. Virulence: experimental infections have shown that chytridiomycosis is highly pathogenic to some Australian frogs (Berger *et al*., 1998) and dendrobatid species (Longcore *et al*., 1999; Pessier *et al*., 1999; Nichols *et al*., 2001). However, the pathogen appears not to be virulent in all species of amphibians. Notably, experimental infections of bullfrogs (Daszak *et al.* unpublished data) and tiger salamanders (Davidson *et al*., 2003) resulted in no mortality attributable to chytridiomycosis.
6. Persistence at low host densities: Daszak *et al*. (1999) hypothesized that aclinical infections of larvae and survival as a saprobe would enable *B. dendrobatidis* to circumvent the threshold density effect by persisting in affected areas once all adults had been removed. The first scenario is similar to that proposed for extinction of wildlife, for example African wild dogs in the Serengeti (Cleaveland & Dye, 1995), where reservoir hosts are present. Few studies have so far studied infection of larvae, but it has been shown that prevalence of oral chytridiomycosis in larvae can be very high, for example up to 67% in populations of the declining mountain yellow-legged frog (*Rana muscosa*) in the Sierra Nevada (Fellers *et al*., 2001). Waldman *et al*. (2001) also provide field observations that suggest infected larvae survive significantly longer than adults in ponds

invaded by *B. dendrobatidis* and that this provides a delayed impact for outbreaks. Recent work also suggests that infected metamorphosed individuals of less susceptible species would be able to circumvent the threshold effect and drive more-susceptible populations to extinction. Data from experimental infections (see (5) above) and the repeated finding of low level infections in individual wild-caught ranid frogs (Daszak *et al.* unpublished observation) suggest that some species may be relatively resistant to chytridiomycosis, and therefore efficient reservoir hosts. If bullfrogs are less susceptible to infection than other species, their widespread introduction to the United Kingdom, Europe, South America and Western United States suggests they could be an efficient vector and reservoir for this disease.

Range of outcomes in different populations: the diagrammatic model of Daszak *et al*. (1999) proposes that in the majority of populations where *B. dendrobatidis* exists declines do not occur. Recent work has provided significant evidence in support. First, chytridiomycosis occurs in salamander populations that are not in decline, but rather undergo cyclical fluctuations and high mortality associated with iridovirus infection (Davidson *et al*., 2003). Secondly, our previously unpublished observations have shown that chytridiomycosis was present in two amphibian species (*R. catesbeiana* and *R. utricularia*) in the late 1970s and early 1980s at the Savannah River Site (SRS), South Carolina. Amphibian populations have been monitored intensively at SRS for over 25 years with little evidence of declines, particularly for these ranid species (Pechmann *et al*., 1991). Individuals were collected live and seemingly healthy, and chytrid-associated lesions observed in these specimens were inconsistent with the extensive changes seen in the skin of moribund amphibians (e.g. by Berger *et al*., 1998). Thus it appears that some populations can support the presence of chytridiomycosis without suffering long-term declines. Note that this is not an unusual situation, even with highly virulent pathogens. For example, influenza caused the death of between 20 and 40 million humans in 1918–19, but usually causes a febrile disease that leads to recovery. Some wildlife species, e.g. the black-footed ferret and African wild dogs, are extremely susceptible to canine distemper virus, whereas others become infected but usually survive. In these cases, the outcome of an introduced pathogen depends on host immunity, naiveté, the evolution of new strains and other factors. For chytridiomycosis these, as well as environmental factors (e.g. low temperature, ability of the pathogen to survive saprophytically, etc.), are likely to be important, as they would promote a heightened impact and persistence at low host density.

In summary, a number of pathogens are associated with declines, but the clearest link so far exists for amphibian chytridiomycosis. This disease has caused widespread amphibian population declines, particularly in montane amphibian species in tropical regions, and at least one species extinction (*Taudactylus acutirostris*). Chytridiomycosis appears to be locally increasing in impact or moving into new regions (although it is clearly widespread). Anthropogenic introduction (pathogen pollution) appears to be a factor in its emergence. Finally, evidence suggests that a series of unusual biological traits define the ability of this pathogen to cause declines and extinctions. We propose that declines and extinctions occur for an ecologically predisposed group of species, whereas most populations are probably not significantly affected by this pathogen.

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