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Amphibian Declines and Chytridiomycosis: Pathogen Biology, Impacts and Conservation Responses

Anjaly Jose

Assistant professor, Department of Zoology, Nirmalagiri College (Autonomous), Kerala, India

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Abstract

Amphibians are declining faster than any other class of vertebrates, and a substantial part of that loss is attributable to a single group of fungal pathogens. This review covers the biology of the amphibian chytrids, the scale of the damage they have caused, and what conservation practice can and cannot do about it. Batrachochytrium dendrobatidis was described in 1999 after mass mortality events in Australia and Central America, and a second species specific to salamanders was described in 2013. Both infect keratinised skin, and the fungus kills by disrupting the transport of electrolytes across an organ that amphibians use for respiration and osmoregulation, leading to cardiac arrest. A global assessment associated the pathogen with declines in more than five hundred species, of which roughly ninety are presumed extinct, the largest documented loss of biodiversity attributable to a disease. Genomic evidence traces the hypervirulent lineage to east Asia and its global spread to the trade in live amphibians during the twentieth century. Outcomes vary with temperature, host skin peptides and skin bacterial communities, and a few populations have recovered without any decline in pathogen virulence. Effective measures remain limited to biosecurity, trade regulation and captive assurance colonies.

Keywords: Chytridiomycosis, Batrachochytrium, Amphibian Decline, Emerging Infectious Disease, Conservation

1. INTRODUCTION

Population losses among frogs and salamanders were first discussed as a general phenomenon in the late 1980s, and the pattern that puzzled field biologists was the disappearance of species from protected areas with intact habitat. The first global assessment concluded that nearly a third of amphibian species were threatened and that a large fraction of declines could not be explained by habitat loss or overexploitation .[1] Reassessment two decades later found the situation had not improved, with disease and climate change accounting for a growing share of deteriorations in status .[2]

The explanation for the unexplained cases emerged from work on mass mortality events in Queensland and Panama. Histological examination of dead and dying frogs revealed a chytrid fungus in the superficial layers of the skin, and the same organism was present at both sites on different continents .[3] It was formally described as Batrachochytrium dendrobatidis, the first chytrid known to parasitise a vertebrate .[4] A related species that attacks salamanders and newts, Batrachochytrium salamandrivorans, was described after mass mortality in Dutch fire salamander populations , and subsequent surveys traced its origin to Asian salamanders that carry it without severe disease .[5,6]

This paper reviews the biology of these pathogens, the evidence linking them to declines, the factors that determine whether an infected population persists, and the state of management.

2. PATHOGEN BIOLOGY AND LIFE CYCLE

The life cycle has two stages and is completed within days. A motile, uniflagellate zoospore swims in water, locates host skin, encysts and resorbs its flagellum. The resulting thallus develops within the epidermis into a zoosporangium, which matures and releases a new generation of zoospores through a discharge tube opening at the skin surface. Fig. 1 shows the sequence. In culture the cycle takes about four to five days at temperatures between 17 and 25 degrees Celsius .[7]

Thermal biology explains much of the epidemiology. Growth is optimal in the range just described, slows markedly above 28 degrees and stops near 30, so infected animals can clear the fungus by raising body temperature behaviourally, and outbreaks in the tropics concentrate at higher elevations and in cooler seasons. The salamander pathogen is adapted to cooler conditions still, with an optimum between 10 and 15 degrees, which shifts its risk zone towards temperate Europe and northern North America. Table 1 compares the two species.

Zoospores survive for a limited period in water without a host, and the fungus can persist on non-amphibian substrates and in tolerant host species. This reservoir capacity matters because it allows the pathogen to remain in a site after its most susceptible hosts have disappeared, which is why local extirpation of a species rarely leads to local elimination of the fungus.

Fig 1: Life cycle of the amphibian chytrid fungus, completed within days at temperatures favourable for growth .[4,7]

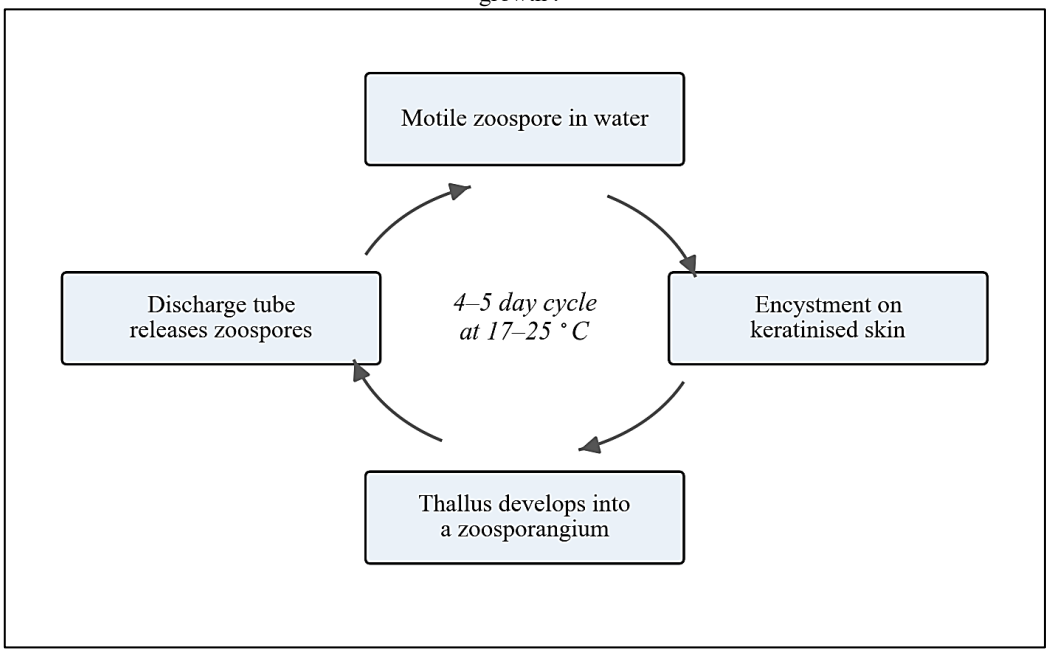


Table 1. Comparison of the two amphibian chytrid pathogens

Feature	B. dendrobatidis	B. salamandrivorans
First described	1999 [4]	2013 [5]
Principal hosts	Frogs, toads, some salamanders	Salamanders and newts
Growth optimum	17 to 25 °C	10 to 15 °C
Upper thermal limit	Near 30 °C	Near 25 °C
Inferred origin	East Asia [8]	East Asia [6]
Documented range	Global	Europe, with Asian reservoir

3. PATHOGENESIS

Amphibian skin performs functions that in other vertebrates are distributed among several organs. It contributes to gas exchange, regulates water balance and transports ions actively. Infection thickens and disorganises the epidermis, and the mechanism of death follows from that damage. Measurements in infected frogs showed reduced transport of sodium, potassium and chloride across the skin, progressive depletion of plasma electrolytes, and terminal cardiac arrest, with electrolyte supplementation delaying death .[9] Death is therefore a consequence of the loss of an osmoregulatory organ rather than of invasion of internal tissues, since the fungus does not penetrate beyond the epidermis.

The immune response is limited and in some respects suppressed. Susceptible species mount weak lymphocyte responses to infection, and fungal metabolites inhibit lymphocyte proliferation in culture, which may explain why heavy infections develop without effective clearance. Innate defences are more important:

antimicrobial peptides secreted by granular glands inhibit zoospores, and species whose peptide repertoires are more effective in vitro tend to persist better in the field.[10]

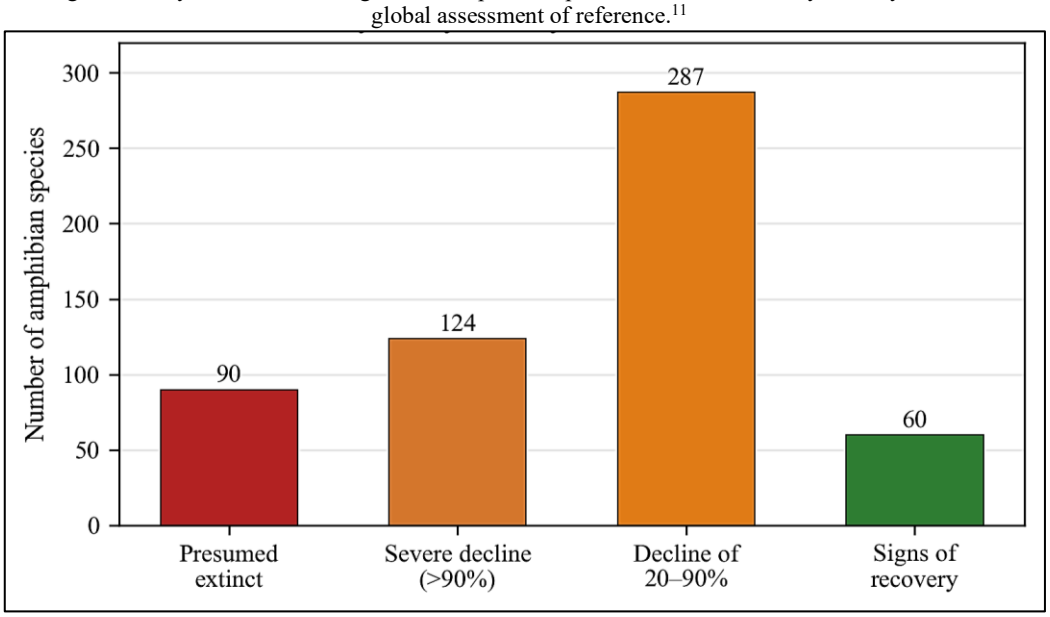
4. THE SCALE OF THE IMPACT

Quantifying the damage required assembling records that were scattered across regional literatures. A global synthesis identified 501 species with declines associated with chytridiomycosis. Of these, 90 are presumed extinct or extinct in the wild, a further 124 have declined by more than ninety percent, and about sixty show some evidence of recovery.[11] Fig. 2 shows the breakdown. The authors describe this as the greatest recorded loss of biodiversity attributable to a pathogen, and the concentration of losses in Central America, the Andes and eastern Australia reflects both the climate suitable for the fungus and the concentration of narrowly distributed montane species.

The pattern of arrival supports a causal reading rather than a coincidental one. In Panama, monitoring at a site before, during and after the arrival of the pathogen recorded a wave of mortality moving through the amphibian assemblage, with declines in the great majority of species over a single season.[12] Comparable wave-like spread was documented in Central America and Australia. Losses fell disproportionately on stream-associated and montane species, on those with narrow ranges and on those that were already rare.

These effects extend beyond the amphibians themselves. Loss of tadpoles changes algal communities and nutrient cycling in headwater streams, and snake assemblages that fed on frogs in affected Panamanian sites declined in abundance and diversity after their prey disappeared.

Fig 2: Severity of declines among the 50 amphibian species associated with chytridiomycosis in the global assessment of reference.[11]



5. ORIGINS AND SPREAD

The fungus was long suspected of being introduced rather than newly virulent, and genomic work settled the question. Sequencing of isolates worldwide identified several deeply divergent lineages, of which one, the global panzootic lineage, accounts for almost all outbreak-associated infections.[13] Expanded sampling located the greatest genetic diversity, and therefore the probable origin, in the Korean peninsula and adjacent parts of east Asia, and dated the emergence of the panzootic lineage to the early twentieth century, coinciding with the expansion of intercontinental trade in live amphibians.[8]

Two trade routes are implicated. The African clawed frog was exported in large numbers for pregnancy testing from the 1930s and carries the fungus with little disease. The American bullfrog is farmed for food and traded live across continents, and surveys of animals in trade regularly find infection. Both species tolerate infection well, which makes them effective carriers. The salamander pathogen followed the same pattern through the trade in Asian newts for the pet market.[6]

6. WHY SOME POPULATIONS PERSIST

Outcomes vary widely between species and between populations of the same species, and several factors contribute. Temperature is one, since populations in warm lowland sites experience conditions that limit fungal growth. Host defences are a second: differences in skin peptide composition correlate with survival.[10] A third is

the skin microbiome, since some resident bacteria produce antifungal metabolites, and experimental addition of such bacteria reduced mortality in laboratory trials.[14]

Evidence for recovery is genuine but modest. Populations of the mountain yellow-legged frog in California have increased at some sites despite continuing exposure, and the increase is associated with greater resistance among survivors rather than with the disappearance of the pathogen.[15] In Panama, an assemblage that had collapsed showed partial recovery, and experiments comparing archived and contemporary isolates found no reduction in virulence, which indicates that the change occurred in the hosts.[16] Recovery of this kind follows severe mortality and leaves populations smaller and less genetically diverse than before.

7. MANAGEMENT OPTIONS

Treatment of individual animals is straightforward, since antifungal agents and heat treatment clear infection reliably in captivity. Treating populations is not. Attempts to disinfect habitats or to treat wild animals at scale have succeeded only in small closed systems such as isolated ponds, and reinfection from reservoir hosts usually follows.[17]

Three approaches have practical value. Biosecurity in the field, including disinfection of boots and equipment between sites, slows human-assisted spread and costs little. Regulation of trade in live amphibians addresses the route by which both pathogens reached new continents, and import restrictions adopted in North America and Europe in response to the salamander pathogen represent the first case in which such a measure was taken before the pathogen arrived.[18] Captive assurance colonies preserve species that cannot currently survive in the wild, at high cost per species and with the unresolved problem of where such animals could eventually be released.

Approaches under investigation include bioaugmentation with protective bacteria, selection for resistance in captive stock, and vaccination using killed fungus or fungal fractions. None has yet produced durable protection under field conditions, and reviews of mitigation conclude that expectations should remain modest.[17,19]

8. SURVEILLANCE, DIAGNOSIS AND RISK ASSESSMENT

Everything described above depends on being able to detect infection reliably, and diagnosis was a limiting factor in the first decade of work. Histology of toe clips detects only heavy infections and requires killing or injuring the animal. The quantitative polymerase chain reaction assay developed for this pathogen changed both constraints: it works on a swab taken from the skin of a released animal, detects infections equivalent to a single zoospore, and returns a load estimate rather than a presence or absence call.[20] Load matters because mortality in most species follows a threshold, so a population carrying light infections is in a different situation from one approaching that threshold.

Two cautions apply to the resulting data. Swab results depend on sampling technique, on the season and on the body region sampled, so prevalence figures from different studies are not directly comparable. Inhibitors carried over from skin secretions and from environmental samples cause false negatives unless controls are included. Environmental sampling of water, which avoids handling animals altogether, is now common but detects the pathogen without indicating which host carries it.

Risk assessment for the salamander pathogen has been conducted largely in advance of its arrival in the Americas, which hold the greatest diversity of salamanders in the world. Susceptibility trials on North American species, combined with climate matching and with records of the newt trade, identified regions of the Appalachian mountains and the Pacific north west as areas where an introduction would be most damaging [18]. This assessment is the basis for the import restrictions mentioned in Section 7 and for targeted surveillance at ports of entry, an approach that reviews of the field regard as the most defensible use of limited resources.[19]

Climate change complicates both surveillance and prediction. Warming reduces the area suitable for the frog pathogen at low elevations while extending the season at high ones, and increases the frequency of temperature extremes that stress hosts without necessarily excluding the fungus. Because the outcome depends on the difference between host and pathogen thermal responses rather than on mean temperature, projections require both to be measured for the species in question, and these data exist for very few of them.

Host genetics has begun to inform both surveillance and captive management. Variation at major histocompatibility loci is associated with survival of infection in several frog species, with particular alleles more frequent among survivors after outbreaks and in populations that persist.[21] Screening for such markers offers a way to identify populations likely to withstand exposure and to avoid breeding out resistance in assurance colonies, where small founder numbers and selection for ease of husbandry both work against it. The association is statistical rather than deterministic, and resistance in the field also depends on skin peptides, microbiome and temperature, so marker-based selection supplements rather than replaces the measurement of survival under challenge.

9. CONCLUSION

Chytridiomycosis is the clearest case in which a single pathogen has driven losses of biodiversity at a global scale, and the sequence from discovery to explanation is unusually complete: the organism was identified, its pathogenesis established at the level of skin physiology, its origin traced by genomics and its spread linked to a specific human activity. What remains incomplete is the response. Effective intervention in wild populations is not available, and the measures that work, namely biosecurity and trade regulation, address future introductions rather than existing infections. The partial recoveries observed in California and Panama show that host populations can adapt, which is encouraging for species that retain sufficient numbers and genetic variation, and offers little to those that do not. Preventing the next introduction is the part of the problem where action is most likely to succeed.

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