Lack of Evidence for the Drought-linked Chytridiomycosis Hypothesis

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ABSTRACT: A significant amount of recent research has focused on the potentially synergistic roles of climate change and disease in causing amphibian declines and extinctions. Herein I discuss the drought-linked chytridiomycosis hypothesis (DLCH), which states that prolonged or intensified dry seasons trigger or exacerbate epidemics of chytridiomycosis, a potentially lethal skin disease of amphibians caused by the chytrid fungus *Batrachochytrium dendrobatidis*. I demonstrate that the DLCH runs contrary to our knowledge of *B. dendrobatidis* physiology, biogeography, and host–parasite ecology and conclude that abnormally dry weather should actually favor amphibians by decreasing the prevalence, severity, and spread of chytridiomycosis.

Key words: Amphibian declines, *Batrachochytrium dendrobatidis*, climate change, extinction, global warming, wildlife disease.

Numerous hypotheses have been put forth to explain the rapid population declines that have affected amphibian species worldwide in recent decades. One explanation, the climate-linked epidemic hypothesis, has received increased attention in recent years (Pounds and Crump, 1994; Pounds et al., 1999; Kiesecker et al., 2001; Pounds, 2001; Harvell et al., 2002; Ron et al., 2003; Pounds and Puschendorf, 2004; Lampo et al., 2006; Santiago-Paredes and La Marca, 2006), but for which no empirical data exist. To distinguish this hypothesis from other climate-linked epidemic hypotheses, I will hereafter refer to it as the drought-linked chytridiomycosis hypothesis.

The drought-linked chytridiomycosis hypothesis proposes that abnormally dry conditions can exacerbate chytridiomycosis outbreaks by 1) increasing amphibian stress levels and subsequently decreasing immune function in infected or susceptible individuals (Lampo et al., 2006); 2) hindering the ability of infected amphibians to rehydrate sufficiently: *Batrachochytrium dendrobatidis* (the fungus that causes chytridiomycosis) affects the skin of the pelvic patch (Berger et al., 1998) and thus may significantly decrease an amphibian’s water uptake during dry periods when water cannot be obtained directly from rainfall (Burrowes et al., 2004); and/or 3) forcing amphibians to spend more time in a diminishing number of cool, moist hibernacula or breeding ponds, resulting in high densities of amphibians and thus optimal disease-transfer conditions (Pounds et al., 1999; Burrowes et al., 2004; Pounds and Puschendorf, 2004; Lampo et al., 2006). Although any of these mechanisms could conceivably result in the increased prevalence or severity of chytridiomycosis, the drought-linked chytridiomycosis hypothesis is inconsistent with our current knowledge of *B. dendrobatidis* physiology, biogeography, and host–parasite ecology. Below I discuss several reasons why abnormally dry periods should actually favor amphibians by
decreasing the prevalence, severity, and spread of chytridiomycosis.

_Batrachochytrium dendrobatidis_ has waterborne zoospores (Longcore et al., 1999; Piotrowski et al., 2004), and cannot survive desiccation (Johnson et al., 2003), so it is unlikely that that dry weather would benefit the fungus. Indeed, bioclimatic modeling predicts that the fungus is more likely to inhabit wetter regions (Ron, 2005), and recent field studies confirm this prediction. Kriger et al. (2007) found that both the prevalence and intensity of _B. dendrobatidis_ infections in eastern Australia increase significantly in regions with high rainfall. Whereas chytrid infections were detected at all 17 sites that had received over 33 mm of rainfall in the 30 days prior to sampling, infections were detected at only half of the 14 sites that received less than 33 mm of rainfall. Kriger and Hero (2007a) demonstrated that _B. dendrobatidis_ is largely restricted to wet microhabitats. Whereas 38.8% of 402 frogs breeding in permanent water bodies were infected with _B. dendrobatidis_, only one of 117 frogs breeding in ephemeral water bodies or in leaf litter was infected. Similarly, Longcore et al. (2007) found significantly higher infection prevalence (22.9%, _n_ = 594) in amphibians utilizing aquatic hibernacula as compared to those utilizing terrestrial hibernacula (8.3%, _n_ = 157). Infection prevalence in their study was significantly lower in 2001 than in 2000, and though daily mean temperatures did not differ significantly between the 2 yr, 11 of the 12 mo in 2001 were drier than their corresponding month in 2000. Finally, droughts often coincide with higher-than-average temperatures, and it is widely recognized that high temperatures are unfavorable to the growth and development of _B. dendrobatidis_ (Piotrowski et al., 2004; Kriger and Hero, 2007b; Kriger et al., 2007).

Our knowledge of chytrid biology, detailed above, allows us to make important predictions regarding the influence of dry spells on chytridiomycosis host–parasite ecology. The first is that a significant proportion of a _B. dendrobatidis_ population would likely be killed or be unable to reproduce during dry periods. Although dry conditions may increase amphibian stress levels, _B. dendrobatidis_ would likely be unable to take advantage of the increased susceptibility of amphibians at these times, and thus we would expect decreases in the prevalence and intensity of chytrid infections during extended dry periods. The second prediction is that, as streams serve as likely vectors for the waterborne zoospores of _B. dendrobatidis_ (Kriger and Hero, 2007a), a reduction in the number of flowing streams due to low rainfall would hinder the spread of the fungus. Further, if amphibians are forced to remain in hibernacula during dry periods, few individuals would be moving long distances, resulting in diminished opportunities for the chytrid fungus to be transferred between catchments or metapopulations (an important consideration when declines are concurrent with the apparent introduction of the fungus to a region (e.g., Lampo et al., 2006).

There exist no published studies that describe chytridiomycosis-linked mortality events concurrent with a reduction in either the available number of breeding ponds or the number of flowing streams. On the contrary, the vast majority of studies (Lips, 1998, 1999; Bosch et al., 2001; Muths et al., 2003; Lips et al., 2006; Rachowicz et al., 2006) detail mortality events occurring at permanent water bodies, and thus there is no reason to believe that chytridiomycosis epidemics are caused by an increase in the density of breeding aggregations due to abnormally dry weather. Although the number of ephemeral water bodies certainly decreases during dry spells, amphibians that breed in these habitats are at low risk of acquiring chytrid infections (Kriger and Hero, 2007a).

_Batrachochytrium dendrobatidis_ has yet to be detected in amphibian retreat sites (Rowley et al., 2007). However, search
effort has been low and it is certainly conceivable that amphibians may aggregate at hibernacula during dry periods and thus increase disease transmission. If this were the case, we would expect low detectability of amphibians during dry periods, because the amphibians would be hidden and inactive. Though 1987 and 1988 were the two driest of the 26 yr examined by Lampo et al. (2006), these years had the third and fourth highest number of *Atelopus* collected, a result inconsistent with the hibernaculum hypothesis (it is also worth mentioning that neither chytrid nor population declines were detected in the 1987 dry year). Pounds and Crump (1994) concluded that *Atelopus varius* actually left their retreat sites en masse during the extreme dry periods of their study.

A strong association with aquatic habitats is a significant predictor of declining status in both Australian and Central American amphibians (Williams and Hero, 1998; Lips et al., 2003), with relatively few reports of declines in terrestrial breeding species, the ecologic guild most susceptible to desiccation, and least susceptible to chytrid infections (Lips, 1999; Lips et al., 2006; Kriger and Hero, 2007a). Thus, although it is plausible that *B. dendrobatidis* could act in conjunction with dry seasons to create a synergistic effect that results in high incidence of lethal desiccations and subsequent population decline, as hypothesized by Burrowes et al. (2004), this is unlikely to have contributed significantly to global amphibian declines. Interestingly, one of the few reported declines of a terrestrial breeding amphibian community occurred at La Selva, Costa Rica, where a significant increase in rainfall was recorded over the 35-yr study period (Whitfield et al., 2007). One of the few reports of declines in an ephemeral breeder (*Bufo periglenes*, also in Costa Rica) occurred during an abnormally dry year, yet had ecologic characteristics that the authors (Pounds and Crump, 1994) concluded were inconsistent with those of a decline caused by moisture stress.

Both climate anomalies (whether human induced or naturally occurring) and pathogens clearly contribute to global amphibian declines (Berger et al., 1998; Lips et al., 2006; Pounds et al., 2006; Rachowicz et al., 2006; Reading, 2007), and the interactions between the two remain largely unknown (Pounds et al., 2007). However, dry conditions are unlikely to favor *B. dendrobatidis*, and a great deal of empirically derived evidence will be required before researchers can add the drought-linked chytridiomycosis hypothesis to the list of valid mechanisms by which amphibian declines occur. Further, *B. dendrobatidis*, ranaviruses, and other amphibian pathogens are widespread (Green et al., 2002; Garner et al., 2005; Ouellet et al., 2005; Kriger et al., 2007). If enough climatic variables are examined and enough pathogens are sampled for, it is inevitable that researchers will occasionally detect some form of climate anomaly coincident with some form of infection at a site that has experienced amphibian decline. I suggest that in the absence of empirical data, researchers should be hesitant to conclude that a decline was caused or exacerbated by the synergism between a climate anomaly and pathogen unless the mechanism by which the decline likely occurred is in parallel with our current knowledge of the host–pathogen system in question.

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