Climate and amphibian declines

J. Alan Pounds

Various reasons have been proposed for the falling numbers of amphibians in many parts of the world. Changing climate is likely to be a key factor — but with complicated links to the immediate causes of these population declines.

The shallow lakes and ponds of western North America provide plenty of sites where western toads (*Bufo boreas*) can lay their strings of eggs. But all is not well with these amphibians. In the crystal clear waters surrounded by snow-capped peaks in the Cascade range, the jet-black embryos are suffering devastating mortality. They develop normally for a few days but then turn white and die by the hundreds of thousands. On page 681 of this issue, Kiesecker, Blaustein and Belden[1] present evidence that climate change may be the underlying cause. Reductions in water depth due to altered precipitation patterns expose the embryos to damaging ultraviolet-B (UV-B) radiation, thereby opening the door to lethal infection by a fungus, *Saprolegnia ferax* (Fig. 1).

A sense of urgency surrounds the study of amphibian mortality. In the Cascades, western toads and various frog species have been decreasing in abundance since the mid-1980s. Similar declines, some leading to the extinction of entire species, have been reported from upland areas around the world[2]. Are the patterns a warning of environmental changes that may have increasingly unfavourable consequences for plant and animal communities, and for humankind?

Kiesecker *et al.*[1] show how important it is to consider various potential agents when addressing this question. Previous studies in the Cascades, which examined the effects of UV-B exposure on embryos and its role in stimulating *Saprolegnia* outbreaks, pointed to stratospheric ozone depletion as a possible factor[3,4]. The new study, however, reveals that unusual weather, not ozone depletion, is the principal agent that increases exposure to UV-B. In extremely dry years, a large proportion of the western toad's egg-laying sites are in very shallow water, which provides little protection against UV-B. In water less than 20 cm deep, the white filaments of *Saprolegnia* invade and kill about 80% of the embryos, on average, compared to 12% in water deeper than 50 cm. Kiesecker *et al.* link the dry conditions to sea-surface warming in the Pacific, and so identify a complete chain of events whereby large-scale climate change causes wholesale mortality in an amphibian population.

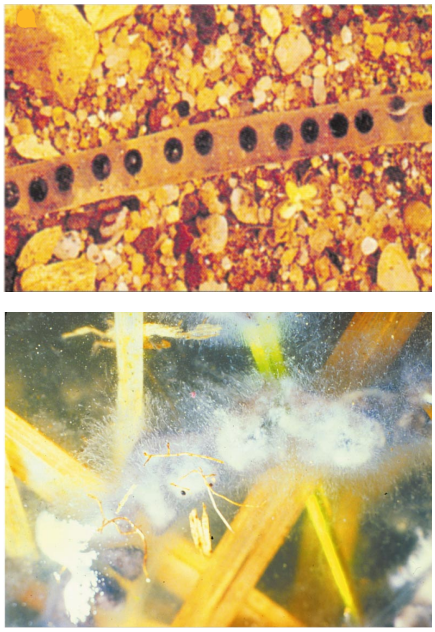

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Figure 1 **The end for embryonic western toads. Top, healthy eggs; below, the eggs infected with *Saprolegnia ferax*.**

Over the past 30 years, globally averaged air and sea-surface temperatures have risen sharply, and there is growing certainty that human activities are largely responsible[5]. A key region is the tropical Pacific Ocean, which has tended strongly towards warmer than average conditions since the mid-1970s[6]. Natural populations may be especially vulnerable where local climate is heavily influenced by the tropical Pacific. Together with long-term warming trends and changes in precipitation patterns, intense warm episodes of the El Niño/Southern Oscillation have conspired to produce extreme climatic events that have severely affected biological communities, from coral reefs to cloud forests[7,8].

Western toads may likewise be victims of these events. Tropical Pacific climate influences winter precipitation (snowfall) in the Cascades, which largely determines water levels in lakes and ponds the following spring[1]. Precipitation totals are correlated with the Southern Oscillation Index for the preceding summer. The mass mortality of western toad embryos parallels the wholesale mortality of adult golden toads (*Bufo periglenes*) and other amphibians in the mountains of Central America[8]. In both cases, alarming shifts in population patterns are linked to dry conditions associated with climate trends and fluctuations in the tropical Pacific.

Another recurring theme is epidemic disease. According to one hypothesis[9], declines of highland amphibians are due solely to a skin disease caused by a chytrid fungus. This pathogen may indeed be a serious threat, but the single-disease model is likely to be an oversimplification. Some of the mysterious

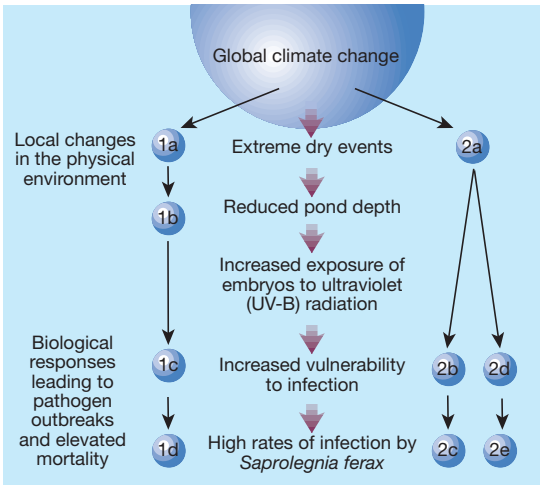

Figure 2 **Links between climate change and amphibian declines and extinctions. The central pathway outlines events leading to mass mortality of western toad embryos in the Cascade range of western North America[1]. The left- and right-hand pathways show other possible sequences. 1, Local increases in the day-to-day variability in precipitation** (1a)**, together with atmospheric contamination, cause increased concentrations of toxic substances in microenvironments frequented by adult amphibians** (1b)[11]**. A resultant weakening of immune responses** (1c) **leads to high infection rates by a pathogen** (1d) **and high adult mortality. 2, Reductions in mist frequency in a tropical cloud forest** (2a)[8] **lead to high adult mortality through one of two biological sequences. Sequence** 2b–2c **is like that of** 1c–1d**, involving an increase in host susceptibility to infection. Alternatively, a pathogen's reproductive rates increase** (2d) **while host susceptibility remains unchanged, leading to increased rates of pathogen transmission** (2e).

declines of frog populations in Central America and Australia have been accompanied by equally mysterious lizard declines[8,10]. A fungus that attacks moist-skinned amphibians is unlikely to attack reptiles, so the common denominator is probably broader than a single disease. Moreover, as Kiesecker *et al.* show, chytrids are not the only fungi killing amphibians in unprecedented numbers.

An alternative to the single-disease model is the hypothesis that extreme climatic events are causing amphibian declines by encouraging outbreaks of certain pathogens[11]. This idea is in line with the findings of Kiesecker *et al.* and with the growing recognition of the connections between climate and epidemics[12]. Pathogens may respond directly to weather, to physical factors influenced by weather, or to biological intermediaries, including changes in host susceptibility or in the vectors that transmit pathogens from one host to another.

Climate only loads the dice for disease outbreaks; it does not dictate when and where they will occur, and whether or not they will spread. It exerts its influence within the constraints of history, geography, natural history and potential interacting agents. Many amphibian populations are likely to suffer increased mortality in a warming world. There is no reason, however, to expect a close correspondence between climatic conditions and the occurrence of population declines.

Nor is there reason to expect a single chain of events whereby climate change leads to population declines (Fig. 2). The sequence of cause and effect that has resulted in the mass mortality of western toad embryos may apply to other shallow-water breeders with aquatic embryos. It might also apply where these species have suffered parasite-induced deformities[13]. But not all links in this particular chain apply to amphibian losses in general. In Costa Rica, Tilarán rain frogs (*Eleutherodactylus angelicus*) lay eggs in subterranean nests shielded from UV-B, but their numbers have nevertheless decreased. Many population declines, including those ascribed to chytrid outbreaks, have involved high mortality of adults rather than embryos. The association of chytrids with deaths of adult western toads in Colorado[14] illustrates a key point: a species or population might come under assault through several pathways.

Future studies of these sequences of cause and effect will help gauge the extent to which global climate change is driving amphibian population declines. During the 1990s, there was controversy over whether the declines were real, or simply a consequence of natural population fluctuations and direct human disturbances such as habitat destruction. Meanwhile, a separate debate focused on climate change and its relationship to greenhouse-gas emissions. Today, there is little doubt that both phenomena — amphibian declines and global warming — are real. If there is indeed a link between the two, as the work of Kiesecker *et al.* suggests, there is clearly a need for a rapid transition to cleaner energy sources if we are to avoid staggering losses of biodiversity. ■

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Cardiovascular biology

Hearts and bones

Mark Sussman

The idea of repairing damaged heart tissue with donated cells is an old one, but finding cells that can do the job has been frustrating. The solution may come from a select group of bone marrow cells.

Heart attacks kill cardiac cells. In the aftermath, the damaged area of the heart forms scar tissue, which impairs cardiac performance. For millions of people every year, this progression from heart attack to heart failure serves as a grim reminder that current drug treatments are no substitute for the loss of living, beating heart cells. Ultimately, doctors resort to heart transplantation as a treatment for heart failure. Replacing the dead heart cells with living ones would be an attractive alternative, but has been stymied by biological, technical and ethical issues. Many of these problems are now circumvented by the demonstration by Orlic and colleagues[1] (page 701 of this issue) that a specific population of cells derived from bone marrow might hold the key to producing functional cardiac cells in damaged hearts.

Damaged hearts in animal models have been loaded with a staggering array of donor cell populations that are thought to be potentially useful for repair or remodelling[2,3]. Examples of donor populations include cells that form adult skeletal muscle, immortalized cells from the atrial region of the heart, smooth muscle cells, bone marrow cells and muscle cells of the heart (called cardiomyocytes), from embryonic, fetal and adult stages of development. Successful transfer depends on the donor cells surviving, maturing, electromechanically coupling with existing heart cells, and having a beneficial effect on the function of the recipient heart. This is asking a lot of any cell population, so it is not surprising that experiments so far have had varying results, depending in large part upon exactly which donor cells are selected.

The idea of using bone marrow cells to repair the heart holds special allure. First, some marrow cells are multipotent — they are able to differentiate into several distinct cell types. In the context of the heart, such multipotent cells might be able to form heart muscle, as well as blood vessels to nourish the damaged area and promote its repopulation by muscle cells. Second, marrow cells are easily and routinely collected from adults, and so do not pose the ethical concerns inherent in using embryonic or fetal tissue. Third, treating a patient with cells derived from their own marrow eliminates the worry that the tissue will be rejected (a concern when using cells from another person). Fourth, transferring marrow cells into the scar tissue of a damaged heart is known to improve heart function, if the cells are first cultured for a week and then treated to induce the expression of muscle proteins[4].

This combination of characteristics makes bone-marrow-derived cells uniquely suited to the task of restoring structure and function in the wake of a heart attack. The problem is that the main function of bone marrow is to generate blood cells. Multipotent cells in adults represent only a fraction of bone marrow cells, most of which are at various maturational stages on the way to forming the blood. Cells already committed to becoming blood cells will not become part of a reconstituted heart-muscle wall. Ideally, the multipotent cells would first be sorted out from the mixture, and then transferred to their new home in the heart.

Orlic *et al.*[1] describe the consequences of