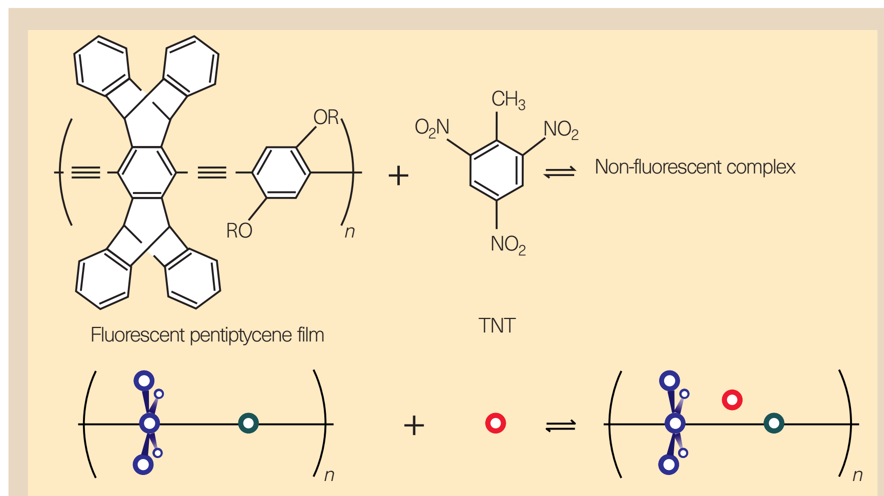

Figure 2 **Chemistry of the prototype TNT chemosensor devised by Yang and Swager[1] (top), with the geometrical relationships being depicted below. The scheme uses a fluorescent polymer film, and because the pentiptycene molecules are not all flat, the film contains pores into which TNT molecules can enter. Binding of a TNT molecule to the polymer results in quenching of fluorescence — the non-fluorescent complex shown here. The consequent change in intensity can be easily measured, and serves as a signal indicating the presence of TNT. As discussed in the text, this approach has yet to be 'reduced to practice'.**

and reversibility (through rinsing) are all encouraging. The sensitivity to TNT is not yet described, however. And although the discrimination against several planar aromatic compounds is good news, field use may reveal interference by other, as-yet-unknown, compounds; one emerging approach to such challenges of selectivity is the sensor-array format[5–7], by which large collections of nonspecific chemosensors all interact with a sample, and the analyte concentration is extracted by network analysis.

None of these drawbacks is cause for pessimism. Research on chemosensors is just starting. Each new discovery adds to the toolbox that will help all of us smell better — and in this case we can hope, in due course, for a viable field detector for TNT. □

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1. Yang, J.-S. & Swager, T. M. *J. Am. Chem. Soc.* **120,** 5321–5322 (1998).
2. Yoon, J.-Y. & Czarnik, A. W. *J. Am. Chem. Soc.* **114,** 5874–5875 (1992).
3. Czarnik, A. W. *Accts Chem. Res.* **27,** 302–308 (1994).
4. Zhou, Q. & Swager, T. M. *J. Am. Chem. Soc.* **117,** 7017–7018; 12593–12602 (1995).
5. Michael, K. L., Taylor, L. C., Schultz, S. L. & Walt, D. R. *Analyt. Chem.* **70,** 1242–1248 (1998).
6. Lavigne, J. J. *et al. J. Am. Chem. Soc.* **120,** 6429–6430 (1998).
7. Doleman, B. J., Sanner, R. D., Severin, E. J., Grubbs, R. H. & Lewis, N. S. *Analyt. Chem.* **70,** 2560–2564 (1998).

Ecology

A declining amphibian conundrum

Tim Halliday

Over the past decade there have been dramatic declines among some amphibian populations in many parts of the world, including a number of apparent species extinctions[1]. These events have caused particular concern because many have occurred in protected areas such as nature reserves and national parks[2,3]. Moreover, until recently they have been detected only after they have happened. The Declining Amphibian Populations Task Force — a worldwide network of scientists, of which I am an international director — has been investigating the declines since 1991, and we have reached several conclusions. First, amphibian declines are occurring throughout the world, although some regions are not affected. Second, at most affected sites some species are declining whereas others are not. Finally, there is no single cause for these declines.

This last point is now addressed by Berger *et al.*[4], reporting in *Proceedings of the National Academy of Sciences*. They have identified a pathogen that links declines among frog populations in two such geographically distinct parts of the world — Panama and Queensland, Australia. Population declines have been well documented at both of these localities[5,6] and, in an exemplary example of international scientific collaboration, biologists from Australia, the United Kingdom and the United States have come together to examine the material collected from the two areas.

Berger and colleagues studied many dead and dying frogs; this allowed them to identify the cause of population declines as they occurred rather than after the event. They found that the same pathogen, a chytrid fungus, is the cause of mortality at the two localities. The fungus invades the skin of adult frogs and probably causes death by interfering with their cutaneous respiration and water uptake. It particularly infects the pelvic patch, an important site of water absorption in many frogs.

There are a number of remarkable features about this discovery. First, the chytrid fungus is a newly discovered genus. Rather than having a typical branched, filamentous structure, it resembles a protozoan and was originally misidentified as such. Second, although chytrids are widespread in soil and are known to attack plants and insects, this is the first to be identified as a vertebrate pathogen. Third, this fungus has now been found in two distant natural localities, as well as among captive amphibians in zoos and aquaria in Australia and the United States. Finally, the chytrid seems to attack just adult amphibians (Fig. 1), having no harmful effects on their tadpoles. This is probably because it only attacks skin that contains the protein keratin, which does not occur in amphibians until metamorphosis. This feature differentiates Berger and colleagues' study from many previous reports of amphibian population declines, a characteristic of which has been reproductive failure owing to mortality among eggs and larvae.

Although this is a very exciting discovery, claims in the popular media[7] that the answer to the declining amphibian conundrum has been found are premature. It does not solve the puzzle, but it does raise further questions. For example, is the chytrid fungus ubiquitous, or has it only recently found its way to Australia and Panama? If it is widespread, why have amphibians only just become susceptible to it? If it has been introduced, where has it come from and how is it being spread? Until we can answer these questions, we will not be able to assess the significance of the new findings in the context of amphibian declines. Of immediate concern is the alarming possibility that herpetologists, seeking evidence for causes of declines in natural populations, may be unwittingly helping to spread disease on footwear or collecting gear[7].

This is not the first time that disease has been implicated in amphibian population declines. The ubiquitous bacterium *Aeromonas hydrophila*, which causes the condition known as 'red leg', has been a contributory factor in some cases[8,9], and iridoviruses have been implicated in mass frog mortalities in the United Kingdom[10]. All of these events may be linked not by the specific pathogen, but by the possibility that the immune systems of many amphibian species are being compromised by environmental factors such as climate change, chemical contamination or increased levels of ultra-

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Figure 1 **Decline and fall — this tree frog (*Litoria caerulea*) is one of the amphibian species that Berger *et al.*[4] have found to be infected with the chytrid fungus.**

violet radiation[8]. Amphibian declines seem to have many causes, and it is vital that scientists do not relax their attempts to investigate these other factors — for which there is mounting evidence — in the mistaken belief that this new study has solved the puzzle.

The significance of Berger and colleagues' discovery[4] may go beyond the mystery of amphibian declines — this is the first time that an infectious disease pandemic has been implicated in the decline and possible extinction of animals. Moreover, if the chytrid pathogen was accidentally introduced into previously naive populations, as measles was to South America, we may be seeing a new kind of anthropogenic insult to the environment. This threat may be more insidious, and difficult to control, than existing environmental problems such as chemical pollution or habitat destruction. □

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1. Wake, D. B. *Science* **253,** 860 (1991).
2. Pounds, J. A., Fogden, M. P., Savage, J. M. & Gorman, G. C. *Conserv. Biol.* **11,** 1307–1322 (1997).
3. Drost, C. A. & Fellers, G. M. *Conserv. Biol.* **10,** 414–425 (1996).
4. Berger, L. *et al. Proc. Natl Acad. Sci. USA* **95,** 9031–9036 (1998).
5. Lips, K. R. *Conserv. Biol.* **12,** 106–117 (1998).
6. Richards, S. R., McDonald, K. R. & Alford, R. A. *Pacific Conserv. Biol.* **1,** 66–77 (1993).
7. Anderson, I. *New Sci.* No. 2140, 4–5 (1998).
8. Carey, C. *Conserv. Biol.* **7,** 355–362 (1993).
9. Bradford, D. F. *J. Herpetol.* **25,** 174–177 (1991).
10. Cunningham, A. A. *et al. Phil. Trans. R. Soc. Lond. B* **351,** 1539–1557 (1996).

Superconductivity

Strain yourself

Ivan K. Schuller

On page 453 of this issue[1], J.-P. Locquet and colleagues show that the superconducting transition, or critical, temperature (T_c) of a particular copper oxide can be doubled by the clever use of epitaxial strain. Increases in transition temperature using pressure have been accomplished before, but the increase obtained here is much larger than any achieved by using standard pressure techniques.

Thin solid films have been used for many years to study materials under physical conditions that cannot be achieved in the bulk. Very large pressure can be applied, and the intrinsically different behaviour of lower-dimensional systems can be observed, for example. Films have shown us many new phenomena, including giant magnetoresistance, dimensional crossover in superconducting multilayers, competition between superconductivity and magnetism, and the high reflectivity of X-ray mirrors[2–4]. The properties of thin films are different from those in the bulk largely because films are grown artificially, under conditions far from equilibrium (usually using a variety of sophisticated, high-vacuum techniques).

But quantitative structural and chemical characterization of a thin film is difficult, and many studies fail because defects such as roughness or interdiffusion of atoms invalidate the conclusions obtained. Locquet and collaborators have avoided these problems in a systematic and thorough fashion. Their films produce clear, sharp peaks in X-ray diffraction, and atomic-resolution electron microscopy shows a low defect density. (Generally, defects tend to depress the superconducting transition temperature, especially in the high-T_c oxides.)

Locquet and colleagues have grown films of $La_{1.9}Sr_{0.1}CuO_4$ on substrates carefully chosen to be of slightly smaller lattice constant (atomic spacing) than the natural value for $La_{1.9}Sr_{0.1}CuO_4$. Because the layers are very thin, the $La_{1.9}Sr_{0.1}CuO_4$ adopts this lattice constant, putting itself under considerable compressive strain to do so. It is not surprising that strain should affect T_c, because pressure has a measurable effect on ceramic superconductors. But the size of the increase in $La_{1.9}Sr_{0.1}CuO_4$ is striking. The authors measure a factor-of-two change in the superconducting T_c of their films, compared with that of the bulk alloy — an increase from 25 to 49 K.

The strain-dependence of T_c has opposite signs along different crystallographic orientations, so applying hydrostatic pressure (which is necessarily isotropic) may not increase T_c, and could even decrease it. But by an appropriate choice of the substrate material and growth temperature, Locquet and co-workers could apply uniaxial strain only in the directions that increase T_c.

In addition, this work may help to determine the critical structural parameters that control superconductivity, and perhaps even to clarify the mechanism that gives rise to superconductivity. Unfortunately, the results of these experiments alone cannot make a definitive statement in this direction. But they do have one intriguing and controversial implication: if the compression in the plane of the film gives rise to an expansion perpendicular to it (the 'Poisson effect') (Fig. 1), that would imply that the superconducting T_c increases with increasing distance between CuO_2 planes. This is contrary to several experimental and theoretical claims[5,6]. But some caution should be exercised with this conclusion, as the Poisson effect does not always occur in thin films. Furthermore, experiments generally find that separating two CuO_2 planes from one another decreases

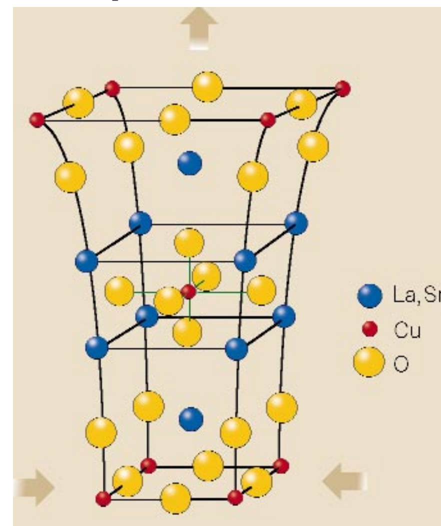

Figure 1 **$La_{1.9}Sr_{0.1}CuO_4$, on a substrate that forces it to distort its usual crystal structure (the actual strain is exaggerated here). This distortion can increase the substance's superconducting transition temperature by a factor of two.**

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