

Abstracts



FIRST AUTHOR

Ecological theory predicts that predator-prey interactions cause large fluctuations in population size. In reality, however, ecologists often find natural populations to

be more stable. On page 1240, Edward McCauley, an ecologist at the University of Calgary in Alberta, Canada, and his colleagues show that the availability of resources affects both maturation and mortality in juveniles of a tiny crustacean called *Daphnia*. This creates a developmental delay that enables small and large fluctuations in population to coexist. McCauley tells *Nature* that this work opens up new avenues of research.

Had this inconsistency between theory and observation been a long-standing conundrum?

Yes. Less variation than expected has been documented in a number of parasite-host, plant-herbivore and predator-prey systems. This has led ecologists to suggest various mechanisms — such as the diverse evolutionary strategies seen in different organisms — to account for the stability of many natural systems.

How did you realize that something was missing from predator-prey models?

Twenty-five years ago, we showed that *Daphnia* — freshwater herbivores — and their algal prey have an incredible range of population dynamics, exhibiting different types of population cycle that weren't predicted by theory. We joined forces with a group of theoreticians and explored how time delays caused by food availability and energy requirements might affect population dynamics. When we stripped this complex biological system down to its essential ingredients — by removing competing algae and other predators that might obscure large-scale fluctuations — we found that predator-prey cycles can exhibit both small and large oscillations. In our new work, we wanted to determine how these cycles could coexist. The key was testing whether food availability affected the length of *Daphnia*'s juvenile stage, as predicted by our models. When food is abundant, juveniles mature too quickly for a stable population to be maintained, whereas scarce food leads to longer maturation rates and smaller fluctuations in population.

What next?

Now we need to explain the prevalence of cycle types and how different patterns of population fluctuations coexist in the same environment. We think that combining models with field and laboratory studies will allow us to show how future changes in temperature and climate might affect the dynamics of communities. ■

MAKING THE PAPER

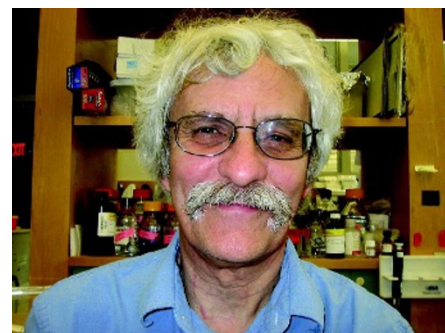
Peter Cresswell

A bacterium takes advantage of a facet of its host's immune response.

Peter Cresswell's work has generally focused on how the immune system responds to microbes, rather than how microbes infect their hosts. But these two processes are inextricably linked, as he learned from witnessing the food-borne pathogen *Listeria monocytogenes* co-opting its opponent for its own good. He and his colleagues now report on *Listeria*'s trickery in subverting its host's immune response.

Listeria produces a protein called LLO that injects pores into membranes, allowing the bacterium to escape from intracellular compartments charged with its destruction, and to replicate and spread with abandon. To become active, LLO must be chemically reduced, which the bacterium cannot achieve alone. Cresswell, an immunobiologist at Yale University School of Medicine, and his colleagues report that the host provides the pathogen with a reduction service actioned by the enzyme GILT. In mice in which the gene encoding GILT has been knocked out, *Listeria* clearance is speedier and the bacterium's growth in cells is drastically impaired (see page 1244).

Cresswell has worked in the field of antigen presentation for decades. Certain immune cells unfold bacterial proteins by exposing them to increasingly acidic environments in intracellular compartments called phagosomes that form when the cell engulfs a bacterium, and through reduction by GILT, which breaks disulphide bonds. This unfolding reveals antigenic 'epitopes' — portions of the protein that are then 'presented' on the cell surface and used as recognition sites by the immune system. In earlier work, Cresswell's group had developed a mouse strain lacking the GILT gene to investigate the enzyme's role in antigen processing and presentation. In a 2001 paper published in *Science*, the team found that antigen processing was significantly



diminished in animals lacking GILT.

Cresswell and his colleagues wondered what else they might learn from the *Gilt*-knockout mice. "We thought we could look at bacterial infections in the knockout mice and ask if we could see any differences," says Cresswell. "It was really as simple as that, a real 'look-see' experiment." If anything, he expected that GILT, which is normally secreted in response to infection, would be missed in the knockout mice, and that their immune responses would be weakened. But the presence or absence of GILT didn't seem to make much difference to infection by several pathogens, including viruses, that the researchers tested — until they came to *Listeria*.

Cresswell was initially surprised that the *Gilt*-knockout mice fared better than their normal counterparts against *Listeria* infection. But he quickly realized what the likely mechanism was — that the reducing enzyme GILT was activating the bacterial toxin LLO in phagosomes. This conclusion relied on two connections. First, Cresswell had worked with SLO, another member of the lysin protein family to which LLO belongs. "We knew that in order to activate SLO, you had to reduce it," Cresswell explains. Second, because of the work of a former colleague at Yale, he also knew that *Listeria* used a similar protein to "pop its way out" of phagosomes and into the cytosol where it replicates.

Thus, without GILT, LLO cannot readily escape the phagosome. Instead, it gets killed, digested and processed for priming subsequent immune responses, says Cresswell. As a result, GILT may represent a new therapeutic target for developing a *Listeria*-specific antibiotic. ■

FROM THE BLOGOSPHERE

Is your science ready for total transparency? Jean-Claude Bradley, a chemist at Drexel University in Philadelphia, Pennsylvania, works on the synthesis of new antimalarial compounds using Open Notebook Science — a practice that makes all aspects of experiments and lab notebooks publicly available online.

During a question-and-answer session on the Sceptical

Chymist blog, *Nature Chemistry* associate editor Neil Withers asked him when he last did an experiment in the lab.

"September 3, 2008. While I was in Southampton I spent the day with Cameron Neylon and we measured the solubility of a few compounds in organic solvents." The results are available online (see <http://tinyurl.com/5j4wwh>). Bradley continues: "We used this as

an example of how people can perform simple experiments and report measurements publicly that are difficult to find, even in expensive databases. We aim to collect a completely public dataset of solubilities of common compounds in organic solvents and create a predictive model." Chemists can best contribute to the world at large by sharing more data more quickly, says Bradley. ■

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