Abstractions

FIRST AUTHOR

Many viral pathogens and their hosts are locked in a never-ending molecular arms race. Some viruses interfere with host immuity by encoding mimics of key host proteins. Poxvirus, for example, encodes a protein that competes with the substrate acted on by protein kinase R (PKR) — a mammalian protein that blocks viral replication. On page 485, Nels Elde, a postdoc at the Fred Hutchinson Cancer Research Center in Seattle, Washington, and his colleagues detail how primate PKR hits back, evolving to counter poxvirus mimicry. Elde tells *Nature* that molecular mimicry is a widespread evolutionary strategy.

When did you realize that mimicry is important for poxvirus evolution?

Our lab focuses on the evolutionary strategies that organisms use to defeat one another. Mimicry was largely uncharted territory, and was intriguing because it changes how we think about conflict. Turning the host against itself adds another layer of biological complexity. When we found that PKR is one of the fastest evolving primate genes, we knew it was responding to assaults and could be a target of mimicry. The poxvirus genome we studied comprises roughly 200 genes, and at least 10 of these are mimics.

How does mimicry evolve?

In a number of cases, mimicry starts when a pathogen 'steals' its host's genetic material through horizontal gene transfer, a process often used by viruses to take up foreign genetic material and incorporate it into their genomes. Over time, if the stolen gene proves to be a viable mimic, it can evolve to disrupt the host's normal protein interactions.

How can a host outfox a pathogen mimic?

This was our main question because most viruses evolve more rapidly than their hosts. One of the big surprises for us was the finding that primate hosts can make multiple, simultaneous changes to its proteins. So although the host evolves more slowly, it evolves in ways that level the playing field.

Do all hosts win out?

We looked at 20 different close and distant primate relatives. We found that humans, chimpanzees and orangutans are resistant to the poxvirus mimic, whereas gibbons, as well as most Old and New World monkeys, are quite susceptible. But our close relatives are resistant for different reasons, which indicates that species can adapt differently even when faced with similar arms races.

What's next for you?

This work focused on the host evolution side of things, but I also want to investigate how pathogens evolve. ■

MAKING THE PAPER

Eric Steig

Plugging data gaps shows warming across West Antarctica.

In recent decades, rapid warming of the western edge of the Antarctic Peninsula has seen plants moving in to colonize the area and Adélie penguins moving out. Although alarming, scientists thought that the warming — which has been disproportionate to that seen in most parts of the world — was a localized phenomenon, and that the bulk of the continent's interior was actually cooling. But a team led by Eric Steig of the University of Washington in Seattle, has shown that Antarctic warming extends beyond the peninsula and covers most of West Antarctica.

Earlier studies of Antarctic warming were based largely on discontinuous, ground-level observations from sparsely distributed weather stations. These included little data from West Antarctica, from which the penisula — a mountainous, icy arm of land that swirls in a northerly direction towards South America — extends. Steig's team has reconstructed Antarctic surface-temperature trends from 1957 to 2006 using satellite data and air-temperature records from weather stations.

Since the late the 1990s, Steig has been part of the International Trans Antarctic Scientific Expedition, an effort aiming to study hundreds of years of past climate and environmental change. As part of this project, Steig and researchers from other universities collected ice cores in West Antarctica, an area of relatively rapid snow accumulation, ideal for ice-core research. "But there was virtually no weather-station data there with which to compare the ice core records," he says. "I needed to extract more information than was available in the literature to make sense of the ice-core records."

In 1999, Steig realized he could use the surface-temperature measurements gathered by satellites. But at that point these had collected only 18 years' worth of data, so he faced a wait of almost a decade before he would have enough to produce meaningful results. In 2001, David Schneider, then a PhD student in Steig's lab, began to dig into the satellite data that was available and analyse it more thoroughly. Satellites do not make direct measurements of surface temperatures, instead calculating them from measurements of the radiation emitted from snow, so the data come with uncertainties. "David's work showed us the limitations of the satellite data, but also that it was more reliable than most people had assumed," says Steig.

By 2007, Steig finally had the 25 years' worth of data he needed, and teamed up with Michael Mann at Pennsylvania State University, University Park. They applied an algorithm that allowed them to fill in missing data across the Antarctic continent as far back as 1957, revealing the warming in West Antarctica. Careful examination of climate modelling results suggested that this warming relates to the regional loss of sea ice over at least the past 25 years (see page 459). Their results were initially met with scepticism. "Everyone I talked to said 'There's no way that's right.'" says Steig. But, with detailed explanations, he managed to change opinions. "Some then realized that they had data that supported us pretty strongly," he says.

Now Steig is heading back out into the field as a member of a team aiming to drill an ice core to the bottom of the West Antarctic ice sheet. Other researchers will take temperature measurements in the borehole, providing a definitive test of Steig's results — the cooler temperatures of the 1950s should still be detectable at depth. ■

FROM THE BLOGOSPHERE

"What science is worth shouting about?" asks Martin Fenner on his Gobbledygook blog on Nature Network. Fenner, a clinical fellow in oncology at Hannover Medical School in Germany, puts cancer research newsworthiness under the magnifying glass (http://tinyurl.com/a4fqvz).

He breaks down the stakeholders for cancer research news into five categories — basic researchers, drug companies, insurance companies, the media and patients — and discusses the motives each has when producing or reading results. For example, Fenner notes, the drug erlotinib won approval from regulatory agencies because it prolonged survival in patients with pancreatic cancer. But insurance companies might balk at the cost: in trials the median survival increase was less than two weeks, meaning the drug costs about US$410,000 per year of life saved.

Fenner concludes that the media needs to better distinguish between basic, early clinical and translational findings; scientists should take care when reporting cancer findings outside of specialist journals; and readers should be supremely sceptical. ■

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