Abstractions

FIRST AUTHOR

Roughly 2.4 billion years ago, enough molecular oxygen began being produced to support the evolution of oxygen-dependent 'aerobic' organisms. This period has been dubbed the Great Oxidation Event. But how the oxygen was produced remains a mystery. Traces of hydrocarbons in 2.7-billion-year-old shales in Australia are widely accepted as evidence of microorganisms including oxygen-producing cyanobacteria. However, that evidence leaves a 300-million-year gap. On page 1101, Birger Rasmussen, a geologist at Curtin University of Technology in Bentley, Australia, and his colleagues say that their measurements refute the evidence for oxygen-producing microbes existing 2.7 billion years ago. Rasmussen tells *Nature* that there may not have been a gap at all.

Did you set out to debunk this evidence?

No. We collected black shale samples for fun in 2000 during a separate project. The shales turned out be a goldmine of information: we found a 1-millimetre-thick layer that provided unambiguous evidence for a major asteroid impact. Then, a few years ago, I met co-author Jochen Brocks from the Australian National University in Canberra and mentioned that we had also found organic residues in the shales. When he told me about the controversy over the biomarkers, we came up with a test to authenticate them.

What did you find?

Brocks found that the carbon isotope signatures differed between the fossilized hydrocarbons used as biomarkers and the shale rock. The subsequent discovery of solidified oil droplets, which we knew formed in the shale, provided an additional comparison with rock-originated compounds. We found that the isotopic composition of this oil was the same as that of the rock, but distinctly different from that of the hydrocarbons — making it impossible to explain that the biomarkers came from those rocks.

So was there a time gap?

We can't be sure. Our data eliminate the only evidence for organisms existing 300 million years before the Great Oxidation Event. So the best hypothesis for the emergence of cyanobacteria is the increased oxygen production seen roughly 2.4 billion years ago.

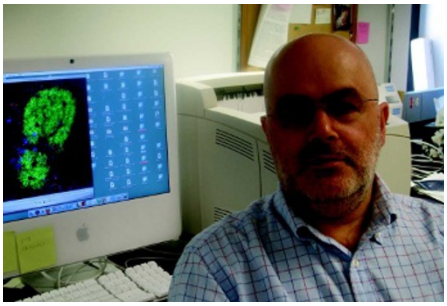
How might the biomarkers that caused the confusion have entered the rocks?

There are many potential sources of contamination — for example, in drilling or sample preparation. Drilling fluid, such as grease, could have been introduced. And, as Brocks recently showed, organic molecules from plastic sample bags can enter rocks. ■

MAKING THE PAPER

Alexander Chervonsky

Gut bacteria may safeguard against a form of diabetes.



Autoimmunity, which occurs when the immune system attacks the body's own cells, is responsible for many human diseases, including type 1 diabetes. What triggers the autoimmune response that causes diabetes is not known. By building on the ideas of a former colleague, Alexander Chervonsky has made a specific connection between gut bacteria and the immune systems of diabetic mice.

As a postdoc, Chervonsky, now at the University of Chicago in Illinois, trained with the late Charlie Janeway, a champion of the idea that the immune system's tailored responses to specific invaders must be preceded by general — or innate — responses to 'non-self' invaders. Chervonsky wondered whether a similar general response might precede the specific attack on pancreatic cells that causes type 1 diabetes.

A strain of mice dubbed NOD — non-obese diabetic — is genetically predisposed to develop diabetes similar to human type 1 diabetes. To test Chervonsky's idea, his team engineered NOD mice that were missing the gene for MyD88, a protein with an important role in transmitting innate immune signals. These mice were protected against developing diabetes. But how?

Since the early 1990s, researchers have recognized that the microbes present in the animals' environment can influence whether or not NOD mice become diabetic. Chervonsky wondered what might happen if the mice lacking *Myd88* were completely free of microbes — including the 'friendly' gut bacteria that colonize animals soon after birth and aid digestion.

Germ-free mouse pups were born and raised in a sterile environment with other germ-free mice. Germ-free mice lacking *Myd88* exhibited a high incidence of diabetes just as normal NOD mice do. "I thought, great, that probably means that gut bacteria can be protective against diabetes," says Chervonsky. But he needed more evidence. When the team added back a cocktail of a few known gut microorganisms to the germ-free animals, the incidence of diabetes decreased. The same thing happened when germ-free NOD mice were 'transplanted' with gut bacteria from NOD mice lacking *Myd88*.

Finally, led by co-author Jeffrey Gordon at Washington University School of Medicine in St Louis, Missouri, the team showed that there were specific differences in the types and amounts of gut bacteria present in normal NOD mice compared with NOD mice lacking *Myd88*.

Together, the results indicate that specific types of gut bacteria are protective against diabetes in NOD mice (see page 1109). "We think that, normally, the innate immune system controls the amount of 'friendly' bacteria living in the gut to keep it in balance," says Chervonsky. "So the signalling that goes through MyD88 is controlling the proliferation of certain types of microorganism." When MyD88 signalling is lost, the microbes that begin thriving somehow block the autoimmune signals that trigger diabetes. If those microbes are lost altogether (as in the germ-free mice), autoimmunity returns.

Exactly how the microbes protect the mouse against autoimmunity remains a mystery, but Chervonsky proposes a possible scenario. The pancreatic lymph node in which the autoimmune cells are activated is also the site of drainage for gut microbes. "It is likely that the microbes are trying to protect themselves and want to block immune responses for their own survival," explains Chervonsky. ■

FROM THE BLOGOSPHERE

The online world often feels like a foreign land, writes Timo Hannay, Nature.com's publishing director, at Nascent, *Nature*'s blog on web technology (http://tinyurl.com/4jz8sc).

Unfamiliar 'languages' such as patches in open-source software, links, online comments, votes and Facebook 'pokes' are the social currencies of the web. Unfamiliar things force us to reassess our own assumptions and prejudices.

Unfortunately, many scientific publishers have responded to the "foreign land called the Internet" with ignorance and denial, notes Hannay: for example, the PRISM initiative, a campaign criticizing open-access publishing. Scientists also seem reluctant. Hannay quotes Jim Hendler, one of the founders of the Semantic Web: "While scientists have gloried in the disruptive effect that the Web is having on publishers and libraries ... we are much more resistant to letting it be a disruptive force in the practice of our disciplines." Take scientists' lack of enthusiasm to do things that are of minimal personal effort but of potential shared benefit, such as depositing manuscripts and notes into repositories. Hannay's message for publishers and scientists alike: "It's not enough to merely accept change, you have to promote it." ■

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