shuttle electrons to insoluble mineral oxides.

“I have had a long-running program to develop microbial fuel cells (MFCs) as power sources for underwater sensors,” Girguis says. “I wanted to extend this to vents, and use the sharp redox gradients at vents to generate electricity, both biologically and abiotically, for sensors. While some vents are being cabled to set up a long-term, high electrical power, broadband observatory, many vents are too remote for such developments. MFCs at vents will definitely provide power, and over time they harness the energy equivalent of 100 or more D-cell batteries per year. You can power a decent number of sensors with that amount of power. If you deploy this at cabled observatories at vents, you might be able to set up a ‘wireless’ network of sorts, using these MFC-powered sensors as wireless nodes.”

Girguis and Nielsen retrieved samples from the hydrothermal vent for lab tests. “We . . . chose not to use pure cultures as we believe the intact community best represented the capacities of those assemblages,” Girguis says. The researchers constructed a two-chamber, flow-through reactor with a pyrite electrode in one chamber subject to simulated hydrothermal conditions. Electroactive biofilms formed on pyrite in electrical but not physical contact with oxygenated water. Their phylogenetic and metagenomic analyses indicate that the microorganisms are Proteobacteria, mainly from the delta, gamma, and epsilon groups.

“Hydrothermal vents are, in my opinion, an exciting place to conduct such studies since vents are extremely heterogeneous habitats, harbor some of the highest-temperature habitats in our biosphere, and, depending on where you’re at in a vent field, offer microbes to varying amounts of reductant and oxidant for energy generation,” Girguis says.

“It would be difficult to assess the credibility of the suggestions that the electrical currents generated would be sufficient to power sea bed sensors without seeing a lot more data,” says David Richardson of the University of East Anglia in Norwich Research Park, Norwich, United Kingdom. “However, I think it is an imaginative idea and one worth pursuing with further studies of this nature.”

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Amphotericin B Binds Ergosterol Instead of Forming Ion Channels

Amphotericin B (AmB) kills fungi by binding ergosterol, a critical lipid in their cell membranes, according to chemist Martin Burke at the University of Illinois (UI) in Urbana-Champaign, and his collaborators. Thus, they debunk the long-held notion that this important antifungal agent kills such pathogens by forming ion channels in their membranes.

More than half a century after its discovery, AmB remains a widely used, sometimes lifesaving treatment for systemic fungal infections. However, its high toxicity prompted Burke and his collaborators to synthesize analogues, looking for a less-toxic version of AmB. While testing them, the chemists learned that AmB binds directly with ergosterol before ion channels form. Details of that research appear in the April 26, 2011 Proceedings of the National Academy of Sciences (doi:10.1073/pnas.1015023108).

This finding raises the question whether AmB acts against fungi by ergosterol binding or forming ion channels. To address that question, the UI chemists made and tested the antifungal activity of the derivative, C35deO-AmB, which binds ergosterol but does not form ion channels. When tested against Saccharomyces cerevisiae and Candida albicans separately in culture, C35deO-AmB kills both types of yeast nearly as well as does the parent compound AmB. Meanwhile, several other derivatives that do not bind ergosterol do not kill these types of yeast in culture. Thus, ergosterol binding appears to be the primary killing action. These more recent findings appear in the January 17, 2012 Proceedings of the National Academy of Sciences as an Epub [doi: 10.1073/pnas.1117280109].

The modifications built into C35deO-AmB and the other derivatives were crucial for identifying the

Engineered E. coli Cells Produce Ethanol from Brown Seaweed

After substantial genetic engineering, Escherichia coli cells provide a platform for producing ethanol from brown seaweed, according to Yasuo Yoshikuni of the Bio Architecture Lab in Berkeley, Calif., and his collaborators. A key part of the engineering task depended on moving a suite of genes encoding alginate transport and metabolism from Vibrio splendidus into E. coli, enabling it to make use of this major ingredient of seaweed. Other auxiliary genes improve the overall conversion of alginate to ethanol, which the cells release into the medium, yielding a titer of about 4.7% on a volume/volume basis. The yield is about 0.41 ethanol per total sugars on a weight basis, more than 80% of the theoretical yield, the researchers report. Details appear in the January 20, 2012 Science [doi: 10.1126/science.1214547].
principal mechanism underlying the antifungal effects of AmB, according to chemist Dale L. Boger of the Scripps Research Institute in La Jolla, Calif. This research “is a wonderful example of the power of organic synthesis to interrogate the structural features of complex natural products,” he says.

“Now that we know that ergosterol binding is the key mechanism, we can design drugs that target membrane lipids,” Burke says, referring to candidate antifungal drugs taking shape on his drawing board. “Ergosterol in yeast could serve as a model for lipids in other pathogens.” Although ergosterol appears to be unique to yeast and leishmania parasites, bacterial pathogens also make lipids for their membranes that differ from those found in humans, suggesting to Burke that those bacterial lipids also could be specifically targeted.

The ergosterol binding property of AmB also helps to explain why, after 50 years of clinical use, fungal pathogens rarely develop resistance to AmB. Ergosterol controls many cellular functions, including endocytosis, signaling pathways, and other cell membrane functions in yeast. “Ergosterol is a molecular node needed for proper functioning,” says Burke, “and if a cell tries to alter ergosterol, many processes suffer.” Findings from older scientific papers describe mutations in genes encoding ergosterol synthetic pathways that render yeast cells much less capable of launching infections. “Now those studies make sense,” he says.

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