



Homeless? Whaling threatens the survival of creatures such as this worm that live off whale carcasses.

across a strange menagerie thriving on and near a submerged whale skeleton. Since then, he and other researchers have shown that dead whales, like hydrothermal vents and cold seeps, can for decades support their own deep-sea biological communities. After all, the carcass of a great whale deposits up to 160 tons of blubber, meat, and bone in one fell swoop.

To estimate how 2 centuries of commercial whaling has affected whale-fall communities, Smith and his colleagues have now combined whale-population estimates with two ecological models: one that links habitat loss to biodiversity and a second that estimates how abundant a species must be to avoid going extinct. The best published estimates indicate that 75% of whale populations—and therefore whale-fall habitats—have been lost in the North Atlantic since large-scale whaling began in the early 1800s, Smith says. Based on those numbers, both models predict that whaling has already caused about 40% of North Atlantic whale-fall species to go extinct. Even at the so-called sustainable levels of whaling being considered by the International Whaling Commission, in which whale populations would be maintained at 50% of historic, prewhaling levels, 15% of the whale-fall species will disappear forever, according to the models.

In an ambitious effort that may help identify whale-fall species before they do go extinct, Smith and his colleagues have recently towed out to sea the huge carcasses of five whales that had beached themselves and died, sunk them, and periodically returned to each carcass in a submersible. Sleeper sharks, crabs, and hundreds of hagfish munched away at the carcasses for months, and many thousands of pea-sized amphipods nibbled on the smaller pieces. A strange assortment of creatures then colonized the bones and nearby nutrient-rich sediments, including several new species of

the bone-eating zombie worm (*Science*, 30 July 2004, p. 668), which uses symbiotic bacteria to help digest the fatty marrow of whale bones. Over many decades, these microbes and other free-living bacteria break down oils trapped in whale bones, producing sulfide that fuels the growth of an average of 185 species per large whale skeleton. From these studies, the Hawaii team has upped the tally of species potentially unique to whale falls to 32.

“It’s absolutely fascinating,” says biological oceanographer Steven Palumbi of Stanford University in California. The work, he adds, shows that “there’s a whole community of organisms in the deep sea that specializes in being the undertaker of these whale carcasses.” And, says Palumbi, the modeling demonstrates that human activity can drive at least some deep-ocean creatures, besides whales, to extinction.

—DAN FERBER

DNA Tells Story of Heart Drug Failure

Physicians have long known that a drug commonly prescribed for heart failure helps only about half the patients who receive it.

Now, researchers are explaining that puzzle using genetics. A study reported at the AAAS meeting found that a difference of a single amino acid within the drug’s protein target may determine whether the drug works. The discovery could ultimately help physicians better juggle drugs in heart failure patients and possibly in those with high blood pressure as well.

In the late 1990s, pulmonologist **Stephen Liggett** of the University of Cincinnati, Ohio, along with his colleagues, found that people had a certain polymorphism, or genetic variation, in the gene encoding the beta-1 adrenergic receptor. That’s the receptor targeted by the heart drugs known as beta-blockers.

In the general population, there are two common forms of the receptor gene: One version makes the receptor with arginine at a particular site; the other, which varies by just a single nucleotide, places a glycine there instead. Because every person has two copies of the receptor gene, inheriting one from each parent, an individual can have two copies of the glycine variant, two of the arginine, or one of each. Mice endowed with the human arginine variant are both more susceptible to

heart failure and more responsive to beta-blockers, raising the possibility that the same holds true in people.

So, Liggett’s team recruited 1040 volunteers, all people with severe heart failure. Roughly 490 had two copies of arginine, 450 had one of arginine and one of glycine, and the rest had two copies of the glycine version. Patients were randomly assigned to receive either a placebo or the drug bucindolol, a beta-blocker.

The researchers found that the cohort with two copies of the arginine variant were helped most by the drug. Compared to the placebo group, the bucindolol users experienced fewer deaths and hospitalizations over about 2 years (and in some cases up to five). Over the course of the study, 82% of them survived compared to 65% of those on the placebo. But those with one copy of each receptor variant or two copies of the glycine version weren’t helped at all by the drug, faring about as poorly as the patients on placebo who had two copies of the arginine variant.

Liggett intends to put together a larger study to confirm the findings. This time, however, all patients would receive the bucindolol because it wouldn’t be ethical to give double-copy arginine patients a placebo, Liggett said during his presentation.

QUOTE

“I don’t see any evidence of a backlash by this Administration [against supporters of Democrat John Kerry]. If we were to withhold funding from every scientist who was a Democrat, there wouldn’t be much science.”

—Presidential science adviser John Marburger, discussing the politicization of science at a pre-AAAS meeting workshop sponsored by the National Association of Science Writers.



Liggett notes that, on its own, the variation in the beta-adrenergic receptor gene doesn’t seem to affect heart failure risk. But people with two copies of the gene for the arginine variant and two copies of another gene—a combination nearly unique to African Americans—have 10 times the risk of heart disease.

“He’s found a polymorphism that seems to predict response” to bucindolol, says molecular pharmacologist Kathy Giacomini of the University of California, San Francisco. The result, she adds, is “very exciting” and “very specific.” It’s not clear yet, says Liggett, whether the findings apply to other beta-blockers, which are also used to treat high blood pressure.

—JENNIFER COUZIN