increased risk of heart disease—in foods. He also suggested to OSHA that defibrillators be installed in workplaces. Paul Portney, president of Resources for the Future in Washington, D.C., says that suggesting regulations, not just approving them, is "a huge change" for OIRA. **–JOCELYN KAISER**

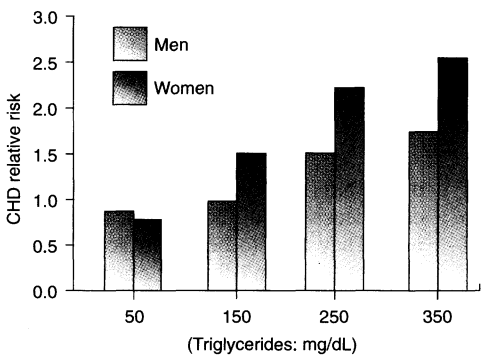
GENOME RESEARCH

Possible New Heart Disease Risk Factor

After embarking on a basic study of gene regulation, a research team has instead uncovered a new gene that may be an important risk factor in cardiovascular diseases. "It was a jewel that we pulled out—one that we weren't exactly looking for," says Edward Rubin of Lawrence Berkeley National Laboratory in California, who led the multidisciplinary team.

The new gene encodes a previously unknown member of the apolipoprotein (APO) family of proteins, which influence blood lipid levels. Researchers have been fascinated by this family since it was discovered decades ago, because the proteins play key roles in transporting cholesterol, triglycerides, and other blood lipids into and out of various tissues. They also found that mutations in several of the 15 or so *APO* genes increase susceptibility to heart disease, because they raise blood cholesterol or triglyceride concentrations. But it's been 10 years since the last new *APO* gene was reported, and researchers thought they'd all been found.

Not so, reports the Rubin team on page 169. They have also shown that mutations in the gene lead to increased blood triglyceride levels in both mice and humans. "This is a wake-up call" that there are surprises in the genome even for well-studied fields, says atherosclerosis researcher Alan Tall of Columbia University. More work will be needed to confirm that *APOAV* variations influence the risk of human cardiovascular diseases. But if they do, the protein might be a target for new lipid-lowering drugs.



Heart threat? Coronary heart disease (CHD) risk rises with triglyceride levels, which may be influenced by the new *APOAV* gene.

CREDITS: (TOP TO BOTTOM) BAS; ADAPTED FROM W. P. CASTELLI *ET AL.*, *THE AMERICAN JOURNAL OF CARDIOLOGY* 70(411) (1992)

The original goal of Rubin and his colleagues from the Stanford Human Genome Center, the University of Texas Southwestern Medical Center (UT Southwestern) in Dallas, and the University of Lille, France, was to use cross-species sequence comparisons to better understand the regulation of three *APO* genes that are shared by humans, mice, and rabbits, but controlled differently. But since stumbling on the new gene while doing the comparison, the researchers changed their focus.

To determine what the gene does, they knocked it out in some mice and created others that carried extra copies of the human gene. The results were dramatic: The mice without the gene had blood triglyceride levels four times those of normal mice, whereas the mice with the extra *APOAV* copies had levels that were only a third of normal. This showed that *APOAV* somehow reduces blood triglyceride concentrations. When the team realized that "not only was this gene missed, but it's important, then we really got excited about it," recalls postdoc Len Pennacchio, lead author of the paper.

Convinced they were on to something important, Rubin, Pennacchio, and their colleagues decided to see whether variations in the *APOAV* gene influence blood triglyceride concentrations in humans. The team identified "markers," changes in single bases, at four locations, three within the gene and one outside it. At each location, most Caucasians have the same base, but a minor subset has a different one. In genetic association studies, the researchers found that for each of the three markers within the gene, the less common base corresponded to increased triglyceride levels, independent of diet. The marker outside the gene showed no association with triglyceride levels. This suggests that the gene likely regulates triglyceride levels in humans as well as in mice and thus may influence their risk of developing a cardiovascular disease.

To try to pin down such a link, Rubin's lab plans to study the effects of a high-fat diet on the knockout mice and those that overexpress the gene. Finding a difference in the animals' susceptibility to atherosclerosis would provide evidence that the gene is a risk factor for cardiovascular disease. The team also hopes that others will pursue the question in humans. Even without those studies, cholesterol researcher and Nobel laureate Joseph Goldstein of UT Southwestern is impressed. This study "raises the bar for these functional genomic papers," he says. "Hopefully every paper that identifies a new gene for the first time will be as complete as this one and as informative." **–CAROLINE SEYDEL**

Caroline Seydel is a freelance science writer in Los Angeles.

POLAR RESEARCH

Fire Guts British Antarctic Lab

CAMBRIDGE, U.K.—Britain's polar biology program is reeling from a fire that has destroyed its main antarctic laboratory. No injuries were reported, but the fire—which broke out on 28 September—did about $3 million worth of damage and has jeopardized about one-fifth of all ongoing British antarctic research, officials say.

The cause of the fire that consumed the Bonner Laboratory at Rothera Research Station on the Antarctic Peninsula is still unknown, but the loss of the lab will set back investigations of climate change, Mars-like environments, and how organisms respond to extreme conditions. During peak research season, the Bonner Lab accommodates about 30 researchers. The blaze did not affect the station's living quarters, where 21 staff members remain safe.

Ablaze. In high winds, researchers had to let the lab burn itself out.

Three principal research projects were under way at the lab, comprising almost all of Britain's terrestrial and near-shore biological research in Antarctica. Two projects looked at how sea-floor and terrestrial communities tolerate harsh conditions and high levels of ultraviolet radiation. The third project used the Mars-like environment of some parts of the Antarctic to understand how life might have survived on Mars and how scientists can best look for signs of life on future Mars missions. The Bonner Lab also housed the continent's only year-round temperature, humidity, and ultraviolet radiation monitoring program. Restoring this program will be a top priority in rebuilding the lab.

"We could potentially lose the best research of its type that's being done in the Antarctic," says Lloyd Peck, the British Antarctic Survey's (BAS's) lead scientist for antarctic biology. But the lab will rise from the ashes soon, he predicts. Says Chris Rapley, director of BAS: "We are committed to rebuilding the Bonner Lab." **–BEN SHOUSE**