group didn't single out any particular material, however.

Pettengill didn't either until, just the week before he was due to give his AGU lecture, he was analyzing the results of one of the last experiments conducted with the Venus-orbiting Magellan spacecraft before its fiery demise in the planet's atmosphere (*Science*, 21 October 1994, p. 366). In that experiment, the orbiter aimed its radar beam so that instead of bouncing right back to the spacecraft, as in conventional radar imaging, it glanced off the planet to be picked up on Earth. The experiment was an effort to gather clues about the nature of the surface that are not present in high-angle reflections. And Pettengill and his colleagues Peter Ford of MIT and Richard Simpson of Stanford University reasoned that the strength and polarization of the low-angle reflections from Maxwell Montes, one of the highest regions on the planet, might tell them something about the mystery material.

The results, as Pettengill interprets them, support the general idea of a volcanic frost—but a frost that's very different from the metallic compounds that the Washington University group had in mind. "The fit [of the data] wasn't what you'd expect for a high-conductivity material like a metal," recalls Pettengill, "but rather it was perhaps 100,000 times less conductive, which tells you you've got a semiconductor there, not a good insulator, not a good conductor, but something in between. This was a real piece of luck, because there aren't very many materials that have that property; that led me immediately to the table of physical constants. I found tellurium and germanium had about the right amount of conductivity, and then my eyes fastened on this fascinating fact that tellurium has a freezing point exactly at the temperature corresponding to the altitude on Venus where we see the edges of this bright material." Pettengill figures that a minimum of a few micrometers of tellurium frosted onto the high-altitude surfaces would do the job.

Lunine isn't sure that the solution to Venus's bright highlands will turn out to be so simple. "There are so many other elements coming out of lavas," he says, that any tellurium would likely be deposited as a mixture, and "you wonder what the properties of the mixture would be." Fegley, though, says, "I believe it is plausible."

The problem, says Pettengill, is now "something for the chemists to work on." Venus specialists won't be the only ones eager for an answer. Given the extensive radar studies of Mercury, Mars, and the asteroids, planetary scientists of all stripes will want to know whether an element nearly as rare as gold can confound radar studies of the solar system.

–Richard A. Kerr

MOLECULAR BIOLOGY

Obesity: Leptin Receptor Weighs In

The biggest news in obesity research over the past year has been "the fat gene." Just 13 months ago, Jeffrey Friedman's team at Rockefeller University cloned the mouse gene for leptin, a protein that controls body weight. Within months, several labs had shown that leptin injections cause mice to lose weight, and found leptin in humans as well. Meanwhile, Amgen Pharmaceuticals had paid a hefty $20 million for an exclusive license to develop leptin-based products (*Science*, 28 July, p. 475). Now 1996 is starting off with more news about leptin—news that could be just as big.

Last week, a research team from Millennium Pharmaceuticals in Cambridge, Massachusetts, and Roche Research Gent, a division of Hoffmann-La Roche in Gent, Belgium, reported they may have found molecules that mediate leptin's effects in the body: two related receptors for leptin from humans and mice. There is still some uncertainty over the precise function of these molecules, but the report, in the 29 December 1995 issue of *Cell*, "is a major breakthrough," says obesity researcher José Caro, of Jefferson Medical College in Philadelphia. It could help scientists understand how leptin works to control weight, and why overweight humans, who produce large amounts of the protein, appear insensitive to it. It may also lead scientists to substances that might mimic or enhance leptin's effects and prove useful as anti-obesity drugs. "It opens a whole new opportunity for pharmacological research," says Rockefeller University obesity researcher Jules Hirsch.

Hunting in the head. The Millennium-Roche team, led by Millennium researchers Louis Tartaglia and Robert Tepper, started their receptor hunt in the mouse brain. Because the brain controls metabolic rates and appetite—functions that leptin affects—the researchers reasoned that the brain must contain leptin receptors. They narrowed their search to cells in the part of the brain that binds the most leptin: the choroid plexus, a tissue that lines brain cavities known as ventricles. The team took messenger RNA from the plexus, converted it to DNA, and put it into cultured cells, which then made proteins from the introduced DNA. From those cells, they selected the ones that bound to leptin, a sign that they were making a leptin receptor. They sequenced the DNA that had been introduced into those cells and found that it was very similar to DNA for a family of receptors for signaling molecules called cytokines. At that point, they knew they were on to something.

The precise identity of that something, however, was still unclear. "We weren't sure that the receptor we identified would be the functionally important receptor in the brain," Tepper says. It binds leptin, but it could be a simple transport receptor that moves leptin across the blood-brain barrier—the choroid plexus is a major site for such molecular transport—rather than a receptor that transmits leptin's signal to a cell. Indeed, the DNA sequence of the receptor clone showed that the receptor lacks most of a taillike segment, usually seen in cytokine receptors, that protrudes into the cell and is responsible for intracellular signaling.

So the group went fishing again, this time in human brain tissue. Using the mouse receptor DNA as bait, they pulled out a similar stretch of DNA. This one looked more like the code for a functional receptor, because it specified a longer receptor molecule, complete with signaling tail.

One gene, two tails? The researchers suggest that mice and humans each have one gene for the leptin receptor, which makes two forms of the protein, one of which acts in the choroid plexus to transport leptin across the blood-brain barrier, while the other functions in other parts of the brain and body to carry out leptin's physiological effects. To investigate this hypothesis, they are now looking for a short-tailed form in humans and a long-tailed form in mice.

Even if this receptor turns out to be a transport molecule, the finding is important, says Bruce Spiegelman, a cell biologist who studies obesity at the Dana-Farber Cancer Institute in Boston, because it will provide a "foot in the door" to lead researchers to the right receptor. And if it turns out that it is the right receptor—the one that transmits leptin's message inside cells—then researchers can begin right away to use the molecule to help unravel the biology of weight control and obesity. Among other things, they can now ask whether obese people are insensitive to leptin because of defects in the molecule itself or in signaling pathways that the receptor turns on inside cells. The discovery will also trigger a new race to find drugs that boost the activity of the receptor.

A number of researchers caution that there is no guarantee such drugs will be found. Small organic compounds that can mimic the effects of protein hormones such as leptin have been notoriously elusive. For instance, "nobody has ever found or developed an organic compound that can mimic insulin," says Spiegelman, "and we have had the insulin receptor in hand for years." Nevertheless a host of labs will undoubtedly be bucking those odds in the coming year, hoping for a fat payoff.

–Marcia Barinaga