land to be destroyed in the mantle, as happens at deep-sea trenches on Earth.

Over in eastern Ishtar, the Brown group finds a situation reminiscent of the northern Pacific Ocean and Alaska. On Venus, several high-riding blocks of crust marked by a distinctive cross hatched pattern called tessera seem to have converged and docked against preexisting highlands, just as the Pacific plate has swept up bits and pieces of crust to form the geologic patchwork of southern Alaska.

In the Head and Crumpler model of Ishtar, the accreted blocks may have formed when plumes rising from the mantle beneath a spreading center like Aphrodite spewed extra lava to thicken the growing crust, the way Iceland has formed perched on the Mid-Atlantic Ridge. Head and Crumpler added such mantle plumes to their model of Aphrodite in order to explain some thicker-than-average crust there.

Despite all the effort, the Brown workers have yet to win many converts outside their own ranks. Elsewhere in geology, some still wonder whether Head has pushed the data too far. And interpretations of the same observations can vary immensely. When Soviet geologist A. A. Pronin of the Vernadsky Institute looks at the data from Ishtar, he sees a mantle plume rising beneath it that is dragging the crust outward to form mountain belts as the plume spreads away. The Brown model, however, has crust converging on Ishtar to make the mountains.

Among geophysicists, other types of data loom large. Geophysicist Walter Kiefer of NASA's Goddard Space Flight Center in Greenbelt, Maryland, calculates that Head's proposed spreading center in western Aphrodite could account for only 10 to 20% of its topography and gravity variations. "I don't think we can rule out spreading centers," he says, but from a geophysics point of view, "it must play a minor role."

Whether a Venusian version of plate tectonics, a terrain dominated by the motions of the underlying mantle, or something between the two turns out to be the case, both geologists and geophysicists are eager to settle the matter with Magellan data. "All the blind men are going to have to talk to each other just to describe this elephant," says Sean Solomon of Massachusetts Institute of Technology, "much less to understand how to make one."

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ADDITIONAL READING

J. W. Head and L. S. Crumpler, "Venus geology and tectonics," *Nature* **346**, 525 (1990).

W. M. Kaula, "Venus: A contrast in evolution to Earth," *Science* **247**, 1191 (1990).

"Magellan arrives at Venus," special issue of *Geophys. Res. Letts.* **17**, (no. 9) (1990).

NF's Cancer Connection

Less than a month after the neurofibromatosis (NF) gene was isolated, investigators have their first solid clues about how the gene brings on this disease, which affects 100,000 people in the United States alone. And that discovery lands NF, which was virtually ignored by researchers until 6 years ago, in the middle of one of the hottest areas of cancer biology.

Ray White of the University of Utah has found that the protein coded for by the NF gene is a close cousin of a recently identified protein, GTPase-activating protein or GAP. Discovered just 3 years ago by Frank McCormick at Cetus, GAP is being furiously investigated in a number of labs, as it seems to play a pivotal role in many human cancers. NF, like cancer, is characterized by runaway cell growth, which gives rise to numerous benign tumors known as neurofibromas. White reported the discovery in the 10 August issue of *Cell*.

Not only does White's latest finding tell researchers how the gene might be working but it also makes it possible to at least envision therapeutic strategies. "This catapults us forward," says Peter Bellermann, executive director of the National Neurofibromatosis Foundation. "Do we have a cure? No. But we do know what avenues to pursue."

Perhaps the biggest payoff from this discovery is that some of the top guns in cancer research will now turn their sights to NF, says White, who adds, "and these guys are very good." Already, McCormick at Cetus and Michael Wigler at Cold Spring Harbor Laboratory, who calls White's discovery "riveting," are gearing up to study the new NF gene and what it might mean for cancer in general.

White discovered the family resemblance between the NF gene and the GAP gene when he compared the NF gene's sequence to that of nearly 20,000 proteins in computer databases. Such a comparison would normally be done as soon as a new gene is identified, but when White and another team led by Francis Collins of the University of Michigan rushed to publication last month, both groups had sequenced just a small chunk of this mammoth gene. Once White had more sequence in hand, he scanned the databases, looking for any similarity that might give some hint as to what the gene does.

The match he found was better than he could have hoped for: a stretch of 360 amino acids in the NF protein was remarkably similar to—in fact, it was 25% identical to—the catalytic region of GAP. What's more, the NF protein is even more closely related to the yeast equivalent of GAP, IRA1, not just in the catalytic region but extending 860–amino acids downstream and 350 in the other direction. Fascinating, says Wigler, who points out that, like GAP, the NF gene must be crucial to normal cellular functioning since it has been conserved throughout evolution.

But it will take some time to figure out what the NF gene is doing because, despite several years of intensive study, little is known about GAP except that it interacts with *ras*, a gene that, when mutated, is involved in perhaps 25% of human cancers. Exactly how GAP and the *ras* protein interact, however, is controversial, though one thing is clear: *ras* signals cells to grow, and when things are working normally, GAP down regulates or turns off the *ras* protein. In short, GAP keeps *ras* from running away with the cell. But GAP may have another role as well; in addition to acting upon the *ras* protein, GAP also seems to be a target for it, receiving and then transmitting signals down a still mysterious pathway.

This understanding, rudimentary as it is, suggests a couple of hypotheses for what the NF gene might be doing, both of which fit handily with White and Collins' initial speculation that NF is one of a small class of tumor suppressor genes. One possibility is that the NF gene ordinarily holds a *ras*-like protein in check, and when both copies of the NF gene are inactivated by a mutation, cell growth runs amok. The other model posits that the NF gene ordinarily plays an essential role in Schwann cells, the cells that give rise to neurofibromas, signaling them to differentiate. If the gene is knocked out, then the cells keep dividing relentlessly, massing into tumors. "Both models are simplistic and probably wrong," says White, but they are a place to start.

The next step is to figure out whether the NF protein interacts directly with *ras* or with a protein similar to it, which White is already setting out to do in collaboration with McCormick. McCormick predicts an answer in a couple of months.

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