Signs of a 'something' circling a star

It is apparently too low in mass to be a star, yet too warm to be what we think of as a planet—if, that is, it is there at all. But observations made less than three months ago of a star known as Giclas 29-38 have provided perhaps the most tantalizing evidence yet for the existence of some kind of "substellar object" circling a star other than our own sun.

"I'm not really ready to say, 'I think we got one,'" said Benjamin Zuckerman of the University of California at Los Angeles last week at the annual meeting of the American Astronomical Society's Division for Planetary Sciences in Pasadena, Calif. In fact, according to Zuckerman and Eric E. Becklin of the University of Hawaii's Institute for Astronomy in Honolulu, "there is at the moment not a single confirmed example of an extrasolar object, either isolated or in orbit around a star, that is unambiguously substellar."

What the two scientists do have is measurements, taken on Aug. 23 and 24 from the NASA Infrared Telescope Facility at Mauna Kea Observatory in Hawaii, indicating that Giclas 29-38 is unusually bright at one particular wavelength (3.5 microns) for a star of its type. Located about 50 light-years away, or a bit less than 300 trillion miles, it is a "white dwarf" star, most of whose infrared spectrum is just about what one would expect from such a star having a temperature of 11,500 kelvins. But there is a little excess—something left over.

A likely source of such infrared excess is energy given off by nearby solid material that has been heated by the star. The material could be in the form of tiny dust grains, perhaps flowing out from the star itself or else in a disk shape that some researchers believe could be a planetary system in the early stages of its formation. Such a disk was discovered in 1984 around the star Beta Pictoris (SN: 10/20/84, p.244). For various reasons, however, Zuckerman and Becklin note in the Nov. 12 NATURE (whose publication of their report was timed to match the Pasadena meeting), both sources of particles seem unlikely in the case of Giclas 29-38.

Another possibility, they suggest, could be "a true planet, such as Jupiter," orbiting close to the star. Such a planet, however, would probably have the same side always facing the star, producing one hot and one cold hemisphere that would show up as a pattern in some of the infrared emissions. No pattern of this kind has been observed, according to the researchers.

"The most natural explanation," the scientists maintain, is a "substellar brown dwarf" in orbit around the star. A so-called brown dwarf, so far only a hypothesis, would be an object whose mass is too low for the compression caused by its own self-gravity to trigger thermonuclear fusion at its heart, but high enough, in the view of Zuckerman and Becklin, for it to be capable of burning deuterium into helium. This would presumably give it something between 1 and 8 percent of the mass of the sun. In 1984, a brown dwarf was reported to be orbiting the star Van Biesbroeck 8 (SN: 12/15/84, p.373), but the spectral measurements that led to its discovery could not be repeated.

If a brown dwarf indeed circles Giclas 29-38, it should have between 4 and 8 percent of the sun's mass (or roughly 40 to 80 times the mass of Jupiter), about 15 percent of the sun's diameter, a faint 0.005 percent of its luminosity and a temperature of about 1,200 kelvins, the researchers estimate. "And unlike some previous claims," Zuckerman said at the meeting, "I'm confident that this signal is not going to go away."

Also during the gathering, Bradford A. Smith of the University of Arizona in Tucson reported that the disk of particles around Beta Pictoris is both larger and less symmetrical than previously thought, spanning about 900 astronomical units in one direction and about 1,100 in another. This asymmetry, suggested Daniel P. Whitmire of the University of Southwestern Louisiana in Lafayette, might be due to the presence of a brown dwarf moving around it in a noncircular orbit. *—J. Eberhart*

Better animal models for genetic defects

If researchers want animal models with one of 2,000 known genetic diseases affecting humans, they can expose laboratory animals to chemicals or radiation and have a million-to-one chance of getting the desired mutation in any given animal.

However, a new technique developed by University of Utah biologists may improve the odds to even money, perhaps making it easier to understand why mutated genes cause such diseases as cystic fibrosis and muscular dystrophy. Researchers want to know, for instance, whether mutated genes underproduce or overproduce certain substances, such as enzymes.

Eventually, the technique, which makes good and bad genes interchangeable, may allow researchers to reduce the occurrence of genetic diseases in humans. They also may be able to place mutated human genes in mice to see which of the 50,000 genetic defects in humans they cause, according to Mario R. Capecchi, whose report appears in the Nov. 6 CELL.

"With this method, we can change the gene the way we want it," he told SCIENCE NEWS.

Using a variation of gene therapy that researchers first used in 1980 to produce a black-and-white-haired mouse by injecting a black-hair gene into an albino mouse embryo, Capecchi and postdoctoral fellow Kirk R. Thomas mutated the human hypoxanthine phosphoribosyl transferase (HPRT) gene and successfully injected it into mouse stem cells. Stem cells are embryo-derived cells that have not yet decided what they want to be.

The next step will be to inject the altered cells into mouse embryos, which would then express the mutated gene. The cell insertion step, Thomas says, is difficult, but it has been done in several other laboratories.

In humans, the mutated HPRT gene causes Lesch-Nyhan syndrome, characterized by mental retardation and self-mutilation, including finger biting, eye gouging and head banging. The normal HPRT gene produces an enzyme that converts a nucleic acid, guanine, into precursors for RNA and DNA. It is not known why reductions in the enzyme cause the syndrome.

To accomplish the change, Capecchi and Thomas went against a common perception among scientists: When DNA strands are injected into stem cells, they will randomly exchange information with other genes, but only 1 of 1 million interchanges will be correct. The researchers showed that they could increase the number to 1 of 1,000 by using larger strands and that they could increase the odds to even money by setting up a selection system that allows only the cells undergoing the preferred recombination to live.

The two researchers placed modified guanine into a dish of stem cells. Those cells undergoing the unwanted recombination died because the HPRT gene manufactured an enzyme that tried to convert the modified guanine into RNA and DNA precursors. The few remaining cells survived because they made the precursors from smaller building blocks. It is not known why a similar process is not sufficient in humans with mutated HPRT genes.

While this procedure will allow interchangeability between good and bad genes in mouse stem cells for experimental purposes, the same techniques may be used in human bone marrow, which would make the change only in that individual. Exchanging good genes for bad in human stem cells, however, poses technical and ethical problems, Thomas says. *—S. Eisenberg*