Brown dwarfs: Finding the lithium benchmark

Too massive to be planets, too tiny to be stars, brown dwarfs occupy a special niche in the celestial netherworld. These dim objects—long sought, but never definitively detected—are thought to form just as stars do, from the gravitational collapse of a cloud of gas and dust. But unlike bona fide stars, they lack the mass to ignite and sustain a nuclear fire at their cores.

Over the years, astronomers have found many faint objects that seemed to qualify as brown dwarfs. But faintness alone doesn't prove their existence. Scientists must also look for the signature of lithium.

Created along with hydrogen and helium in the Big Bang, lithium is destroyed in the nuclear furnace of low-mass stars. That's because the churning motion within these stars pulls lithium from the surface, mixing it into the hydrogen-burning core. Brown dwarfs, in contrast, never get hot enough to fuse hydrogen, so they should retain most of their lithium allotment.

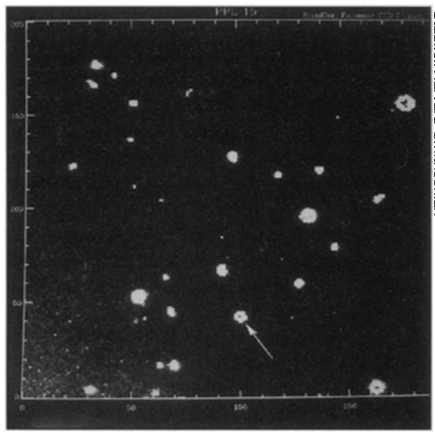
Now, using the world's largest optical telescope, astronomers have for the first time detected lithium in an object believed to be a brown dwarf. Even if the object, a member of the Pleiades star cluster known as PPL 15, doesn't quite make the grade as a true brown dwarf, the work provides a new benchmark in the study of low-mass objects, says study coauthor Gibor Basri of the University of California, Berkeley.

He and his colleagues, Geoffrey W. Marcy of San Francisco State University and James R. Graham of Berkeley, reported their work last week at a meeting of the American Astronomical Society in Pittsburgh.

Relying on work by John Stauffer of the Harvard-Smithsonian Center for Astrophysics in Cambridge, Mass., and his colleagues, who last year suggested that PPL 15 could qualify as a Pleiades brown dwarf, Basri's team recently trained the 10-meter W.M. Keck Telescope atop Hawaii's Mauna Kea on the faint body.

The team obtained visible light and near-infrared spectra of the dim object. A dark line in the spectra revealed the fingerprint of lithium absorption, the first time astronomers had seen such a signature in a brown dwarf candidate. Knowing that the Pleiades cluster, although relatively young, has existed long enough for all of its low-mass stars to have destroyed their lithium, the researchers took similar spectra of slightly brighter, presumably more massive members of the cluster. They found no evidence of lithium.

The team estimates the mass of PPL 15 at just under 8 percent that of the sun, or about 80 times the mass of Jupiter—nearly the maximum mass a brown dwarf can have. The findings suggest that PPL 15 "is right at the hairy edge—right at the transition between a brown dwarf and a star," says James W. Liebert of the University of Arizona in Tucson. Graham speculates that PPL 15 might begin to burn hydrogen but could not sustain fusion.



Stauffer/Harvard-Smithsonian

Near-infrared image of PPL 15 (arrow).

From estimates of the distance to the Pleiades and of the luminosity of PPL 15 and the other, slightly brighter objects that the team examined, Basri and his colleagues calculate the cluster's age at about 110 million years, rather than the usual estimate of 76 million years. The study suggests that other young star clusters may also be older than models of stellar evolution indicate, he adds.

"Either we don't understand lithium depletion in low-mass stellar configurations, or we don't understand models that give the ages of the Pleiades and other young clusters," says Liebert.

The team now plans to observe even fainter, lower-mass Pleiades members in the hope of recording their lithium signature and confirming their brown dwarf identity. *—R. Cowen*

Single gene causes ataxia, cancer risk

Some toddlers toddle a lot not because of their youth, but because they have inherited a fatal neurological disorder, ataxia-telangiectasia (A-T). These children also suffer from involuntary movements and slurred speech. Moreover, they and perhaps their parents, who are carriers of A-T, face a higher than normal risk of cancer.

Now, researchers announce that mutations in a key segment of a single gene cause the disease. Earlier studies had suggested that A-T results from mutations of several genes.

"The identification of a single gene responsible for A-T should enable clinical geneticists to offer reliable diagnostic tests, including prenatal diagnosis and carrier detection to all A-T families," report Yosef Shiloh of the Sackler School of Medicine at Tel Aviv University and his colleagues in the June 23 SCIENCE. In the past year, he and his colleagues have tested a prenatal screen that he expects will now find broad use.

When both members of a couple carry the A-T mutated (ATM) gene, their children have a one in four chance of having the disease. Between 1 in 40,000 and 1 in 100,000 people develop the disorder. Certain groups, including Italians and Turks, have the higher incidence. A-T carriers, who inherit the defective gene from only one parent and don't develop the disease, form about 1 percent of the U.S. population.

A-T patients suffer from weak immune systems and premature aging, among other ills, and usually die in their teens or early twenties. Patients, and to a lesser degree carriers, are sensitive to ionizing radiation. Other studies have suggested that carriers face roughly four times the general population's risk of getting cancer. In addition, women may have six times the normal risk of breast cancer (SN: 1/4/92, p.4).

Discovery of the ATM gene segment will enable scientists to test "whether or not female or male carriers of this disease are at an increased risk of developing cancer," says David L. Nelson of the National Cancer Institute in Bethesda, Md. Studies have yet to prove that carriers have a heightened chance of getting cancer, he argues.

Efforts to develop a treatment for A-T and to learn more about how cancer develops will also benefit from the gene discovery, scientists say.

The gene's protein resembles phosphatidylinositol-3 kinase (PI-3 kinase) and other well-known enzymes involved in immune function, cell death, and cell division, says Shiloh. Information about those enzymes should help researchers in their pursuit of a treatment for A-T, says Nathaniel Heintz, a Howard Hughes Medical Institute investigator at Rockefeller University in New York City.

Studying ATM will also shed light on the possible link—which researchers are just now making—between the body's control of both cell division and cell death, says Heintz. A-T patients suffer from rapid loss of brain cells as well as from tumors. *— T. Adler*