Dust glow hints at wave of early star birth

For every star that glitters, many more lie behind a veil of dust.

Observing the universe at submillimeter wavelengths, where the glow from star-warmed dust is brightest, astronomers have found evidence that at early times in the universe, stars were born at a rate five times higher than visible-light studies have indicated. The images hint that the vast majority of stars in the cosmos were born well before the cosmos had reached half its current age.

"To some astronomers, it's a nightmare; to others, it's just what they expected," says James Dunlop of the University of Edinburgh in Scotland, a member of one of the two teams reporting their work in the July 16 NATURE. A rapid, early burst of star formation "could put a tight constraint on theories of galaxy formation," says Douglas Scott of the University of British Columbia in Vancouver in an accompanying commentary.

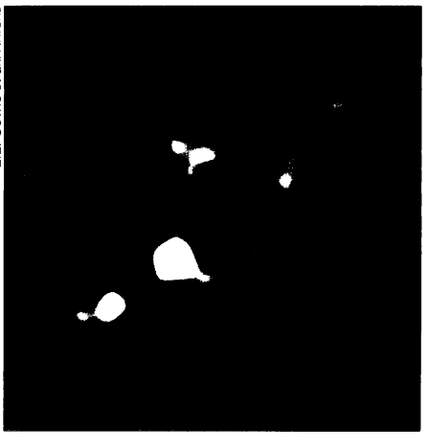
Early birth of stars and galaxies is consistent with certain theories of galaxy formation, says cosmologist Jeremiah P. Ostriker of Princeton University. These models assume that most of the matter in the cosmos is composed of invisible, slow-moving material known as cold dark matter and that this material has too low a density to keep the cosmos from expanding forever.

The two teams used a new high-resolution camera on the James Clerk Maxwell Telescope atop Hawaii's Mauna Kea. Amy J. Barger of the University of Hawaii in Honolulu and her colleagues pointed the telescope at two small, seemingly blank regions of the sky, while Dunlop and his collaborators examined the Hubble Deep Field, a patch of sky that has been studied extensively in visible light.

A submillimeter telescope is well suited for detecting dust in distant galaxies. The dust tends to quench visible and ultraviolet light emitted by stars and reradiate it in the far infrared. The expansion of the universe shifts the far-infrared radiation from distant galaxies to longer, submillimeter wavelengths.

Maps of the far-infrared background glow had already demonstrated that visible-light images drastically underestimate the amount of star formation (SN: 1/10/98, p. 20). The new observations reveal the dust in individual galaxies that creates some of the glow.

The researchers strongly suspect, based on the emissions recorded at two submillimeter wavelengths, that at least four of the seven galaxies they collectively identified are extremely distant, hailing from a time when the universe was no older than one-third its current age. Mark Dickinson of the Space Telescope Science Institute and the Johns Hopkins University, both in Baltimore, notes that to establish that the cosmos formed most of its stars when it was just a few billion years old, astronomers must directly measure how far away the galaxies reside. —*R. Cowen*



L.L. Cowie *et al.*/NATURE

The five brightest objects in this submillimeter map are dusty galaxies.

Possible Alzheimer's gene stirs conflict

It's naive to think that scientists aren't as combative as athletes or businesspeople. And nothing gets the competitive juices flowing as quickly as the hunt for a gene responsible for a feared human disease.

Since last year, investigators have been racing to find a gene on chromosome 12 that they suspect influences a person's chance of getting Alzheimer's disease late in life. At a meeting in Amsterdam this week, and in the August NATURE GENETICS, one group of researchers has fingered as the culprit an altered form of a gene called *A2M*.

"If you have the *A2M* mutation, this puts you at risk for Alzheimer's," says Rudolph E. Tanzi of the Massachusetts General Hospital in Boston, who led the study.

At the same meeting, however, two rival research teams rejected that claim. "We can't support it," says Margaret A. Pericak-Vance of Duke University Medical Center in Durham, N.C.

"Our own data don't show the same association, so the most parsimonious explanation is that it's not *A2M* but something nearby," agrees Peter H. St. George-Hyslop of the University of Toronto.

Tanzi counters that the two groups haven't adequately replicated his study. Moreover, he believes that some of the other scientists' results actually lend support to his group's conclusions. "I'm really surprised at how they're interpreting their data," Tanzi told SCIENCE NEWS. "What you have here is the strategic use of positive and negative data, taken out of context, to knock this result."

Everyone agrees that *A2M* is an appealing candidate for a gene involved in Alzheimer's disease. It encodes alpha-2 macroglobulin, a protein that deactivates proteases, enzymes that carve up other proteins.

Alpha-2 macroglobulin attaches to the same cell surface protein as APP and APOE, the products of two other genes involved in Alzheimer's disease. Moreover, studies have indicated that the unmutated form of the protein helps get rid of beta-amyloid, the APP fragment that accumulates excessively in the brains of Alzheimer's patients.

Initial genetic analyses, in which scientists compared the DNA of elderly people with Alzheimer's disease to unrelated, age-matched, healthy individuals, didn't confirm *A2M*'s link to the disease, admits Tanzi. His group then conducted sib-pair analyses, which compare siblings with and without a disease. The strategy, says Tanzi, reduces confounding factors of traditional linkage studies, such as the varying genetic background of those examined.

Using such analyses, Tanzi and his colleagues found that about 17 percent of people who got Alzheimer's after age 85 had a particular mutation in *A2M*. That same mutation appeared in only 4 percent of the siblings who were 85 or older and didn't have the disease.

Tanzi suggests that the mutant form of *A2M* makes the aging brain more vulnerable to Alzheimer's disease, although it doesn't guarantee that the illness will strike. It's unclear how the identified mutation, a small deletion, affects alpha-2 macroglobulin. It may hamper the protein's ability to bind other proteins.

As they tussle over *A2M*, researchers are also reassessing the gene that encodes APOE. This gene comes in three versions: E2, E3, and E4. Having the E4 version was thought to increase a person's odds of getting late-onset Alzheimer's, but a new study, reported in Amsterdam and also in NATURE GENETICS, suggests that the various *APOE* forms influence when, rather than if, a person gets the disease.

John C.S. Breitner of Johns Hopkins University in Baltimore and his colleagues found that about 40 percent of people, regardless of which *APOE* variant they had, got Alzheimer's disease if they lived long enough. This observation, notes Breitner, was likely obscured in earlier studies because they failed to include enough very old people, he says. —*J. Travis*