Another slinky candidate for galaxy seeds

The uneven sprinkling of galaxy clusters in the universe today raises a question still unanswered after decades of study: How did the uniform soup of the newborn universe develop the initial, tiny density ripples that ultimately led to present-day lumpiness?

The theoretical field is thick with candidates such as magnetic monopoles—particles with a single north or south pole—and endless wires of tightly bunched energy called cosmic strings (SN: 12/7/96, p. 364). Such objects, if indeed they existed, might have attracted enough matter to start the clustering of larger clumps.

Now, another type of energy strand, called the semilocal string, has wriggled into the limelight. Researchers carrying out the first three-dimensional supercomputer simulations of these filaments of magnetic energy have found that they may have been abundant and durable in the first blaze of the Big Bang.

"What we've demonstrated so far is that semilocal strings are interesting and viable," says Julian Borrill of Lawrence Berkeley (Calif.) National Laboratory (LBNL), a member of the research team.

According to the simulations, which will be described in a future issue of PHYSICA B, semilocal strings first appeared at 10^{-35} of a second after the Big Bang. Both cosmic and semilocal strings could have formed as the newborn universe cooled, theorists say. As water changes from steam to liquid to ice, so cosmic phase changes cause the fundamental forces of nature—gravity and the strong, weak, and electromagnetic forces—to condense progressively out of the unified force with which the universe began (SN: 10/15/94, p. 248). This fracturing of forces would have led to energy concentrations such as strings.

Theory forbids cosmic strings from having ends, so they must appear as loops or infinite strands. Semilocal strings, however, can have ends, each of which is studded with a magnetic monopole. Semilocal strings can also curl back on themselves into loops, but then they quickly wink out of existence.

Only recently have supercomputers become capable of simulating semilocal strings in three dimensions. Borrill, Ana Achúcarro of the University of the Basque Country in Bilbao, Spain, and Andrew R. Liddle of the University of Sussex in Brighton, England, modeled them on a computer at LBNL. It performed simultaneous calculations on 3 billion points in fields of quantum force and matter.

The researchers are planning additional simulations to test whether semilocal strings could have been seeds for the universe's transition from smooth to lumpy, Borrill says. Cosmic strings have recently run into trouble in that arena because their expected pattern matches neither the distribution of galactic clusters nor the patchiness of background radiation left over from the Big Bang (SN: 1/27/96, p. 63).

It's too soon to say whether semilocal strings can overcome these difficulties, but the new findings "extend the range of possibilities substantially," says Tom Kibble of the Imperial College in London, who pioneered cosmic-string theory in the late 1970s. Semilocal strings might also help explain the universe's overwhelming preference for matter over antimatter, Borrill adds.


Borrill/LBNL

Semilocal strings of magnetic energy, as modeled by a supercomputer, writhe in space just after the Big Bang.

Aside from potentially seeding galaxies, semilocal strings promise to link cosmology and particle physics in tantalizing ways, says Tanmay Vachaspati of Case Western Reserve University in Cleveland, who invented semilocal strings with Achúcarro 7 years ago. Particle physicists have postulated a nearly identical string, called an electroweak string, composed of known and proposed elementary particles. Accelerators only about 10 times more powerful than the strongest available today could produce low-energy forms of these strings, he predicts.

—P. Weiss

Brain cell death remains unsolved mystery

If detectives found several corpses with bullet holes through the heart, they'd be surprised if autopsies showed that the deaths were actually from poisoning.

Similarly, when scientists last year found that people with neurodegenerative disorders such as Huntington's disease have brain cells stuffed with unusual clumps of mutant proteins, many simply assumed that the abnormal buildup inside the cells' nuclei caused the cell death characteristic of the illnesses (SN: 8/16/97, p. 102). That clue may have been misleading: Two studies in the Oct. 2 CELL suggest that the clumping is a largely irrelevant, perhaps even protective, cellular phenomenon.

In the first study, scientists genetically engineered mice to have the mutant gene responsible for spinocerebellar ataxia type 1 (SCA1), one of the diseases in which mutant proteins clump inside cells' nuclei. In some cases, the researchers had modified the gene so that the protein it encodes no longer sticks so readily to other copies of itself. Indeed, the mutant proteins didn't form discernible clumps inside nuclei. Nevertheless, the mice came down with typical symptoms of SCA1.

The proteins must still get into the nucleus to wreak havoc, the investigators found. They disabled the part of the mutant SCA1 gene that encodes the signal for its protein to move into the nucleus. Mice with this altered gene developed no disease symptoms. "If you block the protein from getting into the nucleus, you have a cure," says Harry T. Orr of the University of Minnesota in Minneapolis who headed the team that created the mice.

In the second study, Frédéric Saudou of Children's Hospital in Boston and his colleagues added the mutant gene responsible for Huntington's disease to rat brain cells grown in the laboratory. The investigators found that the protein encoded by the mutant gene triggers suicide in the same types of brain cells that die in patients with the disease.

Curiously, more cells committed suicide when Saudou added the gene for an enzyme that inhibits the aggregation of the mutant proteins. Saudou's colleague Michael E. Greenberg suggests that the clumps may protect nuclei from toxic effects of the unbound mutant proteins. "The clumps could be an effort to dispose of the protein," he says.

When the team modified the disease gene so that the protein had a signal that prevented it from staying in the nucleus, the brain cells didn't develop aggregates inside their nuclei and didn't die as often.

This cellular study is difficult to relate to the illness, suggests Michael R. Hayden of the University of British Columbia in Vancouver, because the protein encoded by the Huntington's disease gene can also clump inside the cell but outside the nucleus and may damage the brain cell without killing it.

Researchers caution that small protein clumps that went undetected in the experiments could still play a role. "It's a little too early to sort out what the answer is," says Christopher A. Ross of Johns Hopkins Medical Institutions in Baltimore.

"The aggregates clearly are not the whole story," adds Hayden. *—J. Travis*

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