Do-It-Yourself Universes

An analysis of localized inflation suggests that empty space may be spawning universes by the billions, without us ever knowing; was our own universe created this way?

"It shows what a radical change there has been in the climate of science from a few decades ago," says Alan H. Guth of the Massachusetts Institute of Technology (MIT). "Now we have the mathematical tools that allow us to seriously discuss the prospects of creating a universe in your basement."

It should be said at the outset that Guth makes this statement with a smile. Nonetheless, he and a number of other physicists have recently begun to investigate the process of universe formation with some mathematical rigor. And while their results suggest that creating a universe in the basement may be impossible, even in principle, they also suggest that spontaneous quantum dynamics on a microscopic scale may be doing what technology cannot: creating new universes all around us.

The analysis of universe formation starts from the idea of cosmic inflation, which was first proposed by Guth in 1980. According to modern grand unified theories of particle physics, he explains, the dynamics of certain quantum fields may have forced the universe to undergo a period of exponentially fast expansion within the first microsecond after the Big Bang. This hypothesis turns out to be an elegant way of explaining a number of cosmological observations, such as the remarkable homogeneity of the universe, and as a general outline, it is now widely accepted among cosmologists.

However, the very success of the model has led Guth and others to ask what would happen if we could somehow reproduce the conditions of the Big Bang in the modern universe. More precisely, what would happen if a sample of matter were somehow compressed into a tiny region of ultrahigh density and temperature—say 10^{24} K?

At first glance, says Guth, the question seems to pose a formidable paradox. On the one hand, Einstein's equations of relativity predict that an observer outside the compressed region would see it as an ordinary black hole. And since black holes do not inflate, one would expect it to just sit there.

On the other hand, says Guth, if one imagines an observer located inside the region of compression—leaving aside practical matters of survival—the surroundings would seem identical to the conditions that prevailed just after the Big Bang. So one would expect the observer to see a new era of inflation. Indeed, the experiment sounds dangerous: what is to keep this superhot region from expanding outward exponentially and overwhelming us all?

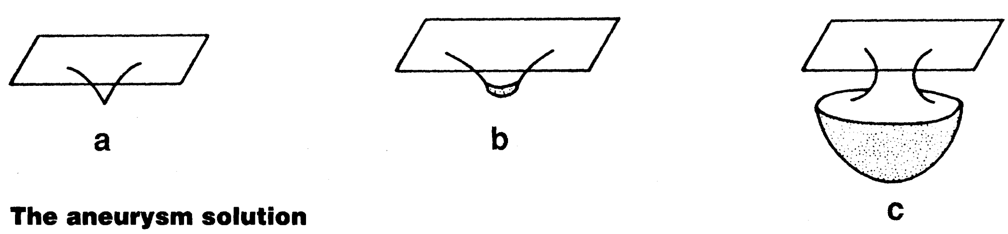
But in reality, says Guth, there is no paradox. He and his MIT colleagues Steven K. Blau and Eduardo I. Guendelman have recently done some calculations showing that both arguments can be correct, depending upon the precise initial conditions. In one solution, for example, the outside universe simply crushes the hot region into a standard black hole. However, there is a much more interesting solution in which the hot region does indeed inflate—but in a totally new direction that is perpendicular to ordinary space and time. It becomes a kind of aneurysm bulging outward from the side of our familiar universe. In fact, it quickly pinches off and becomes a separate universe of its own. To the hypothetical observer, the inflation process goes on from there exactly as it once did for us; ultimately, he may well see this newborn cosmos expand to a scale of billions of light-years, producing galaxies, stars, planets, and even life.

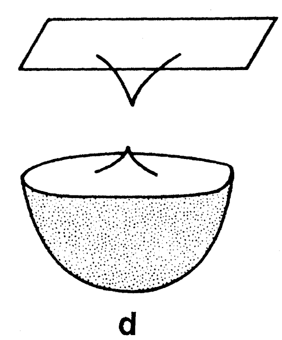
Meanwhile, says Guth, our own universe is left with a real black hole, rather like a bit of scar tissue where the inflating region pinched off. A similar scar—a "navel"—is left in the newborn universe. However, these scars quickly heal themselves, since small black holes spontaneously evaporate by a quantum process known as Hawking radiation. In the end, neither our universe nor the newborn universe is left with any sign of what has happened.

Now, on the face of it, says Guth, we seem to have replaced one paradox by another. The mathematical solution has given us something for nothing: namely, a whole new universe. But is that really such a paradox? Look at our own universe, says Guth. Its total electric charge, its total kinetic and potential energy, its total angular momentum—all seem to be precisely equal to zero. In fact, according to our current understanding of cosmology, the total amount of every conserved quantum number in the universe appears to be zero. All the nonzero quantities—for example, the preponderance of matter over antimatter—can in principle be understood in terms of conservation laws that were broken by quantum dynamics in the Big Bang. In other words, modern particle theory tells us that there is really no barrier to creating a universe out of nothing. "The universe," Guth likes to say, "is the ultimate free lunch."

But does this mean that it really is possible (in principle) to make a universe in the basement? More realistically, does it mean that every star that collapses to form a black hole is actually creating a new universe in the process? Or better still, does it mean that *our* universe was born this way, as a gravitational catastrophe in some larger cosmos?

Alas, no, says Guth. The equations contain a Catch-22. He and his MIT colleague Edward Farhi have recently proved that the only way to produce a new universe inside an old one is to have a singularity to start with—a "white hole" that so drastically pinches the curvature of space and time that





The aneurysm solution

Four stages in the process of universe creation. In frame a, the rectangle represents our universe, which is assumed to be infinite and flat. The cusp represents the white hole that seems necessary to get the localized inflation started. In frame b the tip of the cusp has begun to inflate, and in frame c the neck joining the two universes has begun to pinch off. In frame d the universes have separated. The two cusps now represent black holes, which will quickly evaporate by Hawking radiation. The newborn universe will continue to inflate, possibly until it is billions of light-years in extent.

it can give the new universe a kind of jump start. Furthermore, since Einstein's equations seem to rule out any attempt to make a white hole by compressing ordinary matter—it would just form a black hole—the enterprise seems stymied.

However, says Guth, there may be a way out. Consider the vacuum: ordinary, empty space. According to quantum field theory it is not really empty. On a submicroscopic scale it is constantly undergoing quantum fluctuations in energy and density, rather like a boiling cauldron. Indeed, he says, these oscillations are so violent and so rapid that the vacuum might very easily be creating tiny inflationary regions all the time, even without a white hole. None of these regions would last for more than an instant. And yet, even that instant might be sufficient for the inflation/pinch-off mechanism to come into play. In other words, says Guth, new universes may be constantly coming into existence all around us, spontaneously and invisibly. And conversely, since quantum fluctuations are not restricted by Einstein's equations in the same way black holes are, it is entirely possible that our own universe came into existence this way.

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Of course, says Guth, one has to take these speculations with a liberal grain of salt. For example, the calculations that led to the aneurysm solution and the necessity of a white hole assume that our own space-time is perfectly flat, and that the high-density region is spherically symmetric. The assumptions are necessary to make the equations tractable, but they are oversimplified, to say the least. Nonetheless, Guth believes that these idealized solutions do reveal the essence of what would happen in more realistic situations. He and his colleagues are currently trying to formulate a more quantitative calculation of universe formation in the quantum vacuum, to see if the qualitative arguments really hold up. ■

M. MITCHELL WALDROP

ADDITIONAL READING

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Defect in Alzheimer's Is on Chromosome 21

The genetic defect in Alzheimer's disease is located on the same region of chromosome 21 that contains the gene for brain amyloid, a protein that accumulates abnormally in the brains of both Alzheimer's and Down's patients

SOMETIMES in scientific research, concepts and technologies mature in parallel to produce a major advance. This has just occurred in Alzheimer's research. New data from one group of investigators show that the genetic defect in familial Alzheimer's disease patients is located on chromosome 21—the same chromosome of which there is an extra copy in Down syndrome. Meanwhile, results from two other groups indicate that the gene coding for β amyloid protein, which accumulates in the walls of blood vessels and in the neuronal tissue of both Alzheimer's and aged Down's brains, also maps to chromosome 21. (See pp. 877, 880, and 885 of this issue.)

The new results directly support the notion that at least one form of Alzheimer's is inherited and that a similar genetic defect may occur in both familial Alzheimer's and Down syndrome. Results from a fourth research team show that abnormal amyloid plaques appear in the brains of a wide range of aged mammals as well as humans and suggest that aged nonhuman primates can serve as animal models for certain aspects of Alzheimer's disease. (See p. 873 of this issue.)

Patients with Alzheimer's disease progressively lose their ability to remember and reason. In later stages of the illness they become completely helpless and ultimately die. Physicians describe two forms of the disease—one that occurs fairly early in life at about the age of 50 and another that occurs much later. The strongest evidence that at least one form of Alzheimer's is inherited comes from studies of families in which the disease occurs early, although not all early onset Alzheimer's is clearly inherited and the so-called sporadic or later form may also turn out to have a genetic cause.

"We now have the first clear-cut indication that those families with an inherited form of Alzheimer's disease share an abnormal gene that is located on chromosome 21," says James Gusella of Massachusetts General Hospital (MGH) and Harvard Medical School. He and Peter St George-Hyslop, also of MGH and Harvard, head a large international group of scientists who traced Alzheimer's through several generations of four families in which an autosomal dominant gene causes the disease. They discovered two genetic markers that identify the region of chromosome 21 containing the defect, but Gusella says that it is not yet appropriate to use the markers to predict who might develop Alzheimer's because more than one gene locus may be involved.

The second team of investigators led by Rachel Neve and Rudolph Tanzi, of Harvard Medical School, cloned a complementary DNA (cDNA) probe for β amyloid protein and localized the gene to chromosome 21. "The fact that the gene for β protein is on human chromosome 21 may be a coincidence," says Neve. "The β protein gene may not cause Alzheimer's disease, but it's in the same region of chromosome 21 as the gene described by the Gusella group."

Tanzi and Neve also show that production of messenger RNA (mRNA) for β protein is increased in fetal Down's brain, which suggests that the extra dosage of chromosome 21 and the gene for β protein may lead to the abnormal deposition of amyloid protein in the brains of these individuals. They also find that the mRNA for amyloid exists in many human tissues in addition to brain.

A third research group has also cloned a cDNA for the brain amyloid protein gene and similarly finds it on chromosome 21. "The amyloid polypeptide that is found in plaques is a very short protein, about 42 amino acids long," says Dmitry Goldgaber of the National Institute of Neurological and Communicative Disorders and Stroke. "But the native protein is many times larger than the final protein." Goldgaber and Carlton Gajdusek, also of NINCDS, and their colleagues postulate in their paper that extracellular deposits of "amyloid in Alzheimer's disease and Down syndrome may well be formed from a precursor synthesized in neurons as well as in other cells, such as microglia and brain macrophages."

The fourth group of investigators, Dennis Selkoe of Harvard Medical School, Linda Cork and Donald Price of Johns Hopkins University School of Medicine, and their