The Supernova 1987A Shows a Mind of Its Own—and a Burst of Neutrinos

The first nearby supernova in 400 years continues to baffle and delight since its discovery on the night of 23 February; it has also provided the first clear-cut result from neutrino astronomy and forces theory to face reality

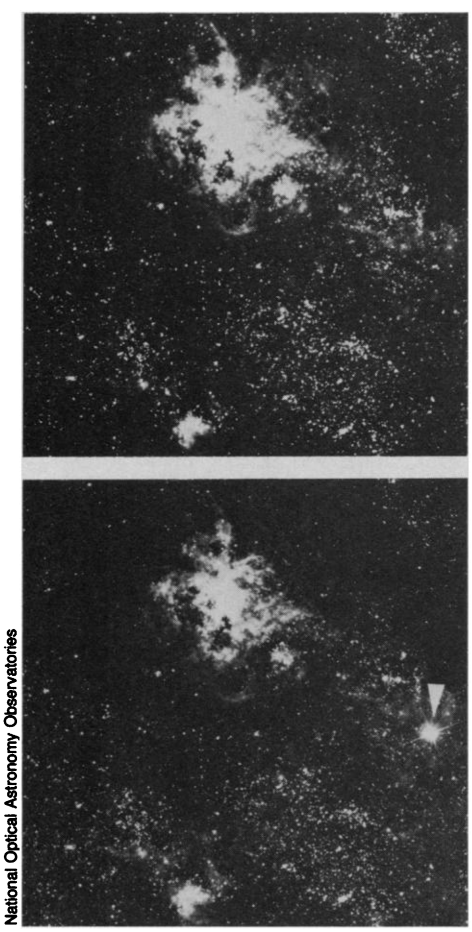
In the few short weeks since it first appeared, Supernova 1987A in the Large Magellanic Cloud has proved to be even more revealing than astronomers had hoped. Not only is it the brightest such eruption since an 1885 event in the Andromeda galaxy, and the closest since Kepler's supernova of 1604, but it continues to evolve in defiance of all the standard models of stellar explosions. The flood of new data has therefore left theorists and observers alike in a state of happy bewilderment.

The supernova had been discovered serendipitously by two amateur astronomers at the end of February and was the subject last week of a hurriedly called meeting at the Goddard Space Flight Center in Maryland.

Perhaps the most intriguing of the latest results comes not from the telescopes but from astronomy's newest arena: neutrino astronomy. On 9 March, after 2 weeks of data analysis, members of a Japanese–University of Pennsylvania collaboration announced that the Kamiokande II proton-decay detector, located deep underground in a mine near Kamioka, Japan, experienced a burst of neutrino events roughly 1 day before the supernova flared in visible light. Such a neutrino burst accords well with spectroscopic indications that 1987A is a so-called Type II supernova, one that is thought to result from the internal collapse of a very hot and very massive star. Indeed, says Princeton University theorist John Bahcall, who has been deeply involved in calculating how the various proton decay detectors would respond to supernova neutrinos, "If this result is true, it changes what has been a computer game into real science."

When the mass of a star is greater than about eight times the mass of the sun, goes the theory, the temperatures and densities at its center are able to sustain fusion reactions among heavy nuclei such as oxygen, nitrogen, and sulfur; eventually, in fact, the star develops a core of iron-56, which is the most tightly bound nucleus of all. Unfortunately for the star, however, this nuclear stability is fatal. The iron-56 is unable to provide any further fusion energy to support the core against gravity. Once the iron accumulates past a critical threshold—about 1.4 solar masses—the core collapses.

Within 1 second, according to computer calculations, the collapsing core becomes so dense that the electrons in the plasma are forced to merge with the protons in the nuclei, forming neutrons. Indeed, the nuclei themselves are forced to merge into a single giant mass of neutrons: a neutron star. On the other hand, this ball of nuclear matter is far stiffer and more resistant to compression than the original plasma. So it "bounces," sending a shock wave back through the upper layers of the star and blasting them into space. The result: a superheated shell of expanding matter that will, for a brief time, shine as brightly as a whole galaxy of normal stars.



National Optical Astronomy Observatories

The Large Magellanic Cloud: before and after. *On top, an image taken in 1969. On the bottom, an image taken on 26 February 1987, 2 days after the supernova was discovered.*

The neutrinos, in this picture, are mostly produced as the electrons are merging with the protons in the core; each merger produces one neutrino, which then escapes. Indeed, calculations suggest that neutrinos can actually carry off most of a supernova's energy. One estimate puts the flux of neutrinos reaching Earth from Supernova 1987A at 100 billion per square centimeter.

Very few of those neutrinos will actually interact. Nonetheless, the theorists' prediction for the signal at Kamioka is remarkably similar to what was actually seen: 5 events in the first ½ second, three more events in the next 2 seconds, and 5 more events in the next 11 seconds. Moreover, the timing is right. Supernova models suggest that the shock wave will take about a day to propagate from the core of the star to its surface, which, paradoxically enough, will be cool, diffuse, and several billion kilometers in radius. (Before the explosion the core is already so hot that it warms the outer layers and causes the star to swell up into a "red supergiant" phase.) Only when the shock wave erupts will the explosion be apparent from the outside. The Kamioka events came at 7:35 a.m. universal time on 23 February, not quite 22 hours before the supernova was first seen in visible light.

As it happens, the Kamioka findings are in contradiction to earlier reports of a neutrino burst in the Soviet-Italian proton-decay detector under Mt. Blanc—the most glaring contradiction being that the Mt. Blanc events preceded the Kamioka events by some 4.6 hours. However, the European result has so many difficulties that many researchers now believe that it, not Kamioka's, is wrong.

For example, Kamiokande II is by far the larger detector—it is a Cherenkov counter with an effective interaction region of 1000 metric tons of water—which means that its results have a lower level of statistical uncertainty. Furthermore, while it is true that the five events in the Mt. Blanc burst are about what theory would predict for that detector, the events are also scattered over 7 seconds instead of being tightly bunched in the first fraction of a second. This spread could be explained if neutrinos are assumed to have a mass of about 10 electron volts—an intriguing result in itself—but such a large neutrino mass would make it hard to understand why the universe has not collapsed already. (At 10 eV apiece, primordial neutrinos left over from the Big Bang would have a ferocious gravitational effect on the cosmos.)

Finally, and perhaps most troublesome of all, the Mt. Blanc detector has experienced similar neutrino bursts in the past for no apparent reason. Indeed, the mystery bursts seem to come every two months or so. As Bahcall points out, it is an outrageous coincidence that a spurious burst should come just before Supernova 1987A—but coincidences do happen.

Whatever the fate of the Mt. Blanc events, the Kamioka result would seem to rule out any possibility that Supernova 1987A could be a Type I supernova, which is thought to arise from a very different mechanism than Type IIs. (A white dwarf star pulls in matter from a normal companion star until the mounting density and pressure trigger a runaway thermonuclear explosion; among other things, a Type I supernova produces no neutrinos.) However, this latest finding only adds to a deepening mystery: where is the star that blew up?

Immediately after the discovery, the deceased was identified as a previously cataloged 12th magnitude star known as Sanduleak −69 202, which lay only a few one-hundredths of an arc second from the detonation point. While this particular star was a bit of a puzzle—according to theory, the precursor should have been a red supergiant, while Sanduleak −69 202 was a much hotter *blue* supergiant—no other obvious candidate presented itself.

Now, however, that argument has gone by the boards: Sanduleak −69 202 is still there. On 6 March, in a meeting hurriedly convened at the Goddard Space Flight Center in Greenbelt, Maryland, to plan space, rocket, and balloon observations of the supernova, Robert Kirshner of the Harvard-Smithsonian Center for Astrophysics presented his early observations from the International Ultraviolet Explorer (IUE) satellite. The supernova's ultraviolet emissions are already fading rapidly, he said. At the shorter wavelengths, in fact, nothing is left but a low, steady background—which shows exactly the kind of spectral features one would expect from a blue supergiant like Sanduleak −69 202. Furthermore, said Kirshner, the pre-supernova images show that Sanduleak has a faint companion star some 3 arc seconds to one side; according to IUE, the companion is still there also.

So what is left? Was the precursor one of the handful of very dim stars surrounding Sanduleak −69 202? Does a slight, fuzzy asymmetry in the old Sanduleak images indicate the presence of yet another companion, almost hidden in the glare of its big brother? Either way, the theorists have a problem: how could stars that dim have enough mass to go supernova?

Adding still more to the puzzle is 1987A's refusal to behave like the textbook supernovas. Its spectra, its evolution in luminosity, and, of course, the neutrino bursts, all point to its being a Type II supernova. And yet in Kirshner's ultraviolet spectra it looks very much like a Type I. Add in the speed with which it has evolved, plus its relative dimness—it rapidly reached a plateau about magnitude 4.5, far lower than earlier predictions of magnitude 2 or brighter—and one has to conclude that Supernova 1987A is very much of an individualist. Perhaps Stanford E. Woosley of the University of California at Santa Cruz summed it up best: "This is an event unique in our lifetimes," he said at the Goddard meeting, "and it's not a time to be taking the word of theoreticians too seriously." ∎

M. MITCHELL WALDROP

Briefing:

Human Cancer Gene Sequenced

Only a few months ago, a group of investigators isolated the gene for retinoblastoma, a rare eye tumor of children. This is the first human cancer gene ever isolated. Now another group has sequenced the entire gene and pinpointed the reasons why it fails to function in some patients.

The significance of this work, say cancer researchers, is that it may lead to an understanding of cancers in general. The retinoblastoma gene, which is a recessive cancer-causing gene, is thought to be involved in common cancers as well as retinoblastoma, which is relatively rare.

The retinoblastoma gene sequence is reported in this issue of *Science* (p. 1394) by Wen-Hwa Lee and his colleagues at the University of California at San Diego. Lee's group is an active competitor of the group, headed by Thaddeus Dryja of the Massachusetts Eye and Ear Infirmary, that first isolated the retinoblastoma gene, as reported in the 16 October issue of *Nature*.

About one in 20,000 children develop retinoblastoma, which makes it the most common eye tumor in children. It is treatable when caught early, but survivors have a higher than normal risk of developing other cancers later in life, particularly osteosarcoma, a bone cancer. Since the retinoblastoma gene is "highly expressed in essentially all tissues," according to Lee, it may be a gene that causes a variety of cancers.

Unlike other cancer genes, the retinoblastoma gene causes cancer by its absence rather than by its presence. A cell that has even one copy of the gene appears normal, but when both copies are absent or nonfunctional, the cell, apparently, is cancerous. The retinoblastoma gene is thought to code for a normal cellular protein that may be essential for keeping cell growth in check.

When Lee and his colleagues looked at the transcription of the retinoblastoma gene in tumor cells from children with this cancer, they found that the gene was not expressed at all in cells from two patients. In four other patients, transcription of the gene was abruptly and prematurely terminated. Now, says Lee, "we are testing to see what the gene does."

Lee and his associates find that the retinoblastoma gene codes for a protein that is 816 amino acids long. Their first thought was to search databases of protein sequences to see if the protein was already known or whether the sequence at least resembled that of a known protein. They had no luck, however, indicating that the retinoblastoma protein may be unlike any that have already been studied.

By analyzing the predicted amino acid sequence of the retinoblastoma protein, Lee found that the protein contains regions that should bind well to DNA. Now, he says, he is trying to isolate the protein and determine if it is a DNA-binding protein. If so, he says, "the retinoblastoma gene is probably a regulatory gene."

Lee and his colleagues also are looking for abnormalities in the retinoblastoma gene among patients with other cancers, particularly osteosarcoma. So far, he has evidence that some patients have abnormal retinoblastoma genes whereas others do not. "At this moment, my thinking is that abnormalities in the retinoblastoma gene probably account for a portion of osteosarcoma," Lee says.

Since several laboratories are now actively studying the retinoblastoma gene, everyone expects that it will not be long before they learn exactly what it does and how. And, if the gene is tied to other cancers as well, the findings may have enormous clinical applications. ∎ GINA KOLATA