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