nected by a very long (several thousand mile) baseline, can already resolve structures milliseconds in size and drastic improvement may not be possible. But Valtonen thinks that the present radio techniques should be capable of giving data at least five times better for M 87 and Centaurus A. Field suggests that a new long-baseline infrared instrument would allow optical astronomers to approach the resolution achieved by radio astronomers and might permit "imaging the accretion disk itself—something mindblowing but possible."

At this point the alternative to black hole models are rather limited. One possibility is that the core of active galaxies has a dense cluster of stars providing that requisite energy through frequent supernova reactions, but a number of astronomers think the 100-million-year memory observed in the double radio galaxies is inconsistent with a source powered by numerous small flickering explosions. Another possibility is that the machine could be an object that has not quite collapsed to the characteristic diameter of a black hole. Such objects are usually called spinars or magnetoids, because it is postulated that rotating internal magnetic fields would stop their further contraction. But most astronomers believe that spinars would subsequently collapse into black holes, perhaps within only about 1 million years. Thus they might be more accurately understood as way stations leading to a black hole.

The other major alternative is that "new physics" will be required for the final explanation. Burbidge, Halton Arp at the Hale Observatories, and a few other American astronomers hold this view. which has been criticized as, in fact, a false alternative. "New physics," says Ed Spiegel, at Columbia, "is a nonidea." Burbidge, however, argues that if black holes had really been formed in the middle of large galaxies by the evolution of stars into superstars and then black holes, thermal energy should be produced that we do not see. Rees, for his part, agrees, but argues that black holes are capable of qualitatively and quantitatively accounting for the phenomena that we do see.

So the problem of ascertaining the validity of the black hole hypothesis is as much a problem in the sociology of science as anything else. In spite of the vocal opponents and sharp skeptics, as well as the phalanx of uncommitted voters, it must be considered the leading explanation for the most cataclysmic events we see in the universe.

-William D. Metz

## UPDATE

## Function of the src Gene Product

Avian sarcoma virus (ASV) is an RNA-containing virus that causes sarcomas in birds. The virus, which is also known as Rous sarcoma virus, carries a gene—the *src* (for sarcoma) gene—coding for a protein that must be produced in order for the virus to transform normal cells to malignant ones. Several months ago, Raymond Erikson and his colleagues at the University of Colorado Medical Center reported that they had identified the *src* gene product as a protein with a molecular weight of 60,000, an identification that was subsequently confirmed by two other groups of investigators (*Science*, 13 January, p. 161).

The researchers were excited by this development because they thought it might lead to the achievement of a long-sought goal—the identification, at least in the ASV system, of the specific biochemical event or events causing the malignant state. But at the time the 60,000-dalton protein was discovered no function could be ascribed to it. Now, newly acquired evidence shows it to be an enzyme, specifically a kinase that transfers the terminal phosphate group from adenosine triphosphate (ATP) to an acceptor protein. Other kinase enzymes are known to be involved in the regulation of a wide variety of cellular activities. Therefore, participation of a kinase in the events producing transformation, including as it does many changes in cell properties, is an attractive idea.

The experiments indicating that the *src* gene product is a kinase are basically of two types. One type was described in the April issue of the *Proceedings of the National Academy of Sciences* by Erikson and Marc Collett, also at Colorado, and more recently by Arthur Levinson, J. Michael Bishop, and Harold Varmus of the University of California at San Francisco. These investigators detected the kinase activity in protein precipitated from extracts of transformed cells by treatment of the extracts with antibody specifically directed against the *src* gene product.

## Kinase Activity Not a Contaminant

Although there was always the chance that the kinase activity in the precipitate belonged not to the *src* gene product, but rather to a contaminant that had precipitated with it, the results of the second type of experiment, performed in the laboratories mentioned above, militate against this possibility. Here, purified segments of the RNA genome of ASV containing the *src* gene were used to direct protein synthesis in the test tube. The synthetic protein also has kinase activity. Other investigators, including Karen Beemon and Tony Hunter of the Salk Institute, have obtained similar results with both types of experiments, and there now seems to be general agreement that the kinase is a *src* gene product.

The identity of the cell protein naturally phosphorylated by the kinase is unknown, however. The investigators doing the work think that locating this protein could be very difficult, but they also agree that the cell skeleton is a good place to start their search. The cell skeleton consists of a network of tubules and filaments thought by many investigators to be involved in the control of cell division. This network usually disperses in cells that have undergone transformation initiated by a variety of agents, including ASV. The hypothesis is that dispersion of the cell skeleton disrupts the normal signals controlling cell division and thus leads to uncontrolled proliferation and the other changes characteristic of transformation. Erikson has speculated that the kinase product of the *src* gene may distrupt the cell skeleton by phosphorylating one of the proteins forming it.

Bishop sounds a note of caution, however. He points out that there is now no evidence that the cell skeleton is the immediate target of the *src* gene product or even that dispersal of the cell skeleton is the initial event of transformation. Moreover, transformation involves a large number of changes, many of which may not be produced directly by the kinase. It is also possible that the *src* gene encodes additional, as yet undetected, functions. Thus, although investigators are enthused about the latest advance toward unraveling transformation, the problem is not yet solved.—J.L.M.

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