

gen therapy — lung damage — was lessened.

These findings are of "great importance," attests Sumner J. Yaffe, director of the Center for Research for Mothers and Children within the National Institute of Child Health and Human Development in Bethesda, Md. He cautions, though, that the therapy will have to be studied in more patients before it can truly be said to be more effective and safer than conventional treatment alone. —*J. A. Treichel*

## Joggers' fever may be immune response

During the past few years, several researchers have noted that people who jog regularly also exhibit symptoms similar to the "acute phase" immune response — a physiological reaction to infection that occurs in humans and other vertebrates and is characterized by fever, an increase in white blood cells, lowered blood plasma levels of zinc and iron, and heightened plasma levels of certain proteins. The perpetrator of the immune response is believed to be an "endogenous pyrogen," which is released by certain white blood cells in response to invading microorganisms. Researchers at the University of Michigan in Ann Arbor have now induced an immune response in rats by injecting them with blood from humans who had just exercised, indicating that this response can be caused by a substance produced during exercise.

According to their report in the May 6 SCIENCE, physiologists Joseph G. Cannon and Matthew J. Kluger injected rats with human plasma taken from subjects before and after exercise. The rats that received post-exercise plasma developed fever, depressed blood levels of iron and zinc, and other signs of the acute-phase immune response. Those rats receiving pre-exercise plasma showed no physiological changes. More specifically, only a certain fraction of the plasma — that containing mononuclear leukocytes, a type of white blood cell — caused the physiological changes associated with the immune response.

Although scientists are fairly certain that endogenous pyrogen causes the response, Cannon says, "nobody knows exactly what endogenous pyrogen is." Cannon and Kluger believe it is a small protein produced by the mononuclear leukocytes. But attempts to purify the substance have failed because, as Cannon explained, it takes only a "very tiny amount to cause a biological response," and isolation of such small quantities from blood serum is extremely difficult.

The next step, Cannon told SCIENCE NEWS, is to investigate why the body would have this type of response to exercise. "I don't know if it is important to anybody who runs," he says, "at least not yet."

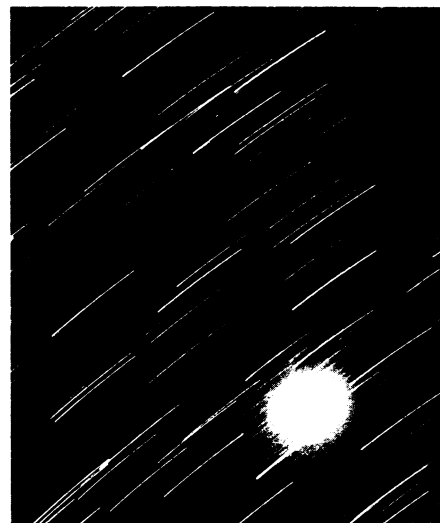
—*P. Taulbee*

## Satellite and earthlings discover a comet

The first observer to see it coming was not a human being at all, but a device—the U.S./Dutch/British Infrared Astronomy Satellite (IRAS), in orbit around the earth. At first, in fact, the satellite's human mentors did not even realize what their device had found. It was a comet, which on May 11 passed closer to the earth than all but one other comet ever known before.

On April 25, while scanning the sky, IRAS detected something that it was able to identify only as a fast-moving object, a category in which most entries are asteroids. Two days later, observers in Kvistaberg, Sweden, alerted to the find, photographed it through earth-based telescopes and concluded from its fuzzy appearance that it was indeed a comet, the fourth to be discovered this year. On May 3, two very human observers—Genichi Araki of Japan and G. E. D. Alcock of Great Britain, both amateur astronomers and both unaware of the IRAS finding—independently made the same discovery, and reported it to the Central Bureau for Astronomical Telegrams in Cambridge, Mass. (which the IRAS team had neglected to do). As a result, all three discoverers are commemorated in the comet's name: IRAS-Araki-Alcock.

On May 11, it passed within about 2.9



Newly discovered comet IRAS-Araki-Alcock shows tail, as photographed May 9 from Table Mountain Observatory in California.

million miles of earth, a proximity bettered only by comet Lexell in 1770. Researchers this week were trying to take advantage of the close visit by attempting what might be only the second successful observation by radar of a cometary nucleus. —*J. Eberhart*

## Genetic engineering yields prenatal test

Researchers at the Johns Hopkins University School of Medicine in Baltimore have developed a prenatal diagnostic test that utilizes genetic engineering techniques to detect hemoglobin abnormalities. The test, used to pinpoint diseases such as sickle cell anemia and beta thalassemia in developing fetuses, was 100 percent accurate and proved safer than most existing tests. It may someday be used to uncover other genetically transmitted diseases. The technique, developed by Corinne D. Boehm and colleagues, was reported in the May 5 NEW ENGLAND JOURNAL OF MEDICINE.

At present, the most useful application of this test is in the detection of beta-thalassemia — a recessive disease that causes severe anemia in 1,200 to 1,800 newborns each year. Using current methods, blood must be extracted directly from the fetus. This technique is risky, with complications leading to fetal death in three percent of those tested. And only a few medical centers perform the procedure. The new test, however, uses fetal cells taken through amniocentesis — a routine procedure in which the fluid that cushions the fetus in the womb is extracted. This procedure carries only a 0.5 percent risk to the fetus.

Restriction endonucleases, bacterial enzymes that cut DNA at specific sequences, are the probes used in this tech-

nique to locate certain genes—called DNA polymorphisms — that lie near the genes for hemoglobin. Except in extremely rare instances, the polymorphism and hemoglobin genes are inherited together. DNA polymorphisms occur when different, but normal, gene sequences are able to exist at a particular site on the chromosome. Fetuses from parents who possess a different gene sequence on each chromosome, and are thus heterozygous for the polymorphism, can be diagnosed in all cases.

The researchers use family studies to determine which polymorphisms are inherited along with the defective hemoglobin gene. By analyzing cells from the parents and comparing these to the fetal cells, they identify the fetal polymorphism type. If the fetal polymorphism type is the one inherited along with the defective hemoglobin gene, the child will be affected by the disease.

Although this indirect test can be used to detect sickle cell anemia, this disease can be detected directly by a similar method (SN: 7/10/82, p. 23). These methods hold promise for early detection of diseases such as phenylketonuria and Duchenne's muscular dystrophy. In order to develop these tests, Boehm says, "we'll need a probe that is specific for the defective gene, or located very close to the gene on that chromosome." —*P. Taulbee*