# Crib death: A genetic factor?

Despite the scores of theories advanced about the cause and nature of Sudden Infant Death Syndrome, SIDS continues to claim up to 10,000 infant lives each year and remains a medical enigma. But there are signs that the disorder may be growing somewhat less mysterious.

It is widely accepted by most medical researchers, for instance, that the syndrome involves some abnormal response to a temporary breathing blockage — a normal baby overcomes a blockage during sleep by momentarily choking or gasping for air, but the SIDS victim apparently is unable to compensate and dies during sleep. Results of one major research project indicate that this abnormal response may result from an inborn "learning disability" programed into a youngster's nervous sytem (SN: 4/15/78, p. 234).

Now, a report in the Feb. 28 NEW ENGLAND JOURNAL OF MEDICINE suggests that this type of SIDS response may not only be inborn but inherited. Researchers from Rutgers University Medical School in Piscataway, N.J., report that parents of SIDS victims respond significantly more poorly than do a matched group of control parents of normal children to two tests: breathing response to a high carbon dioxide mixture and an unconscious, compensatory response to a split-second blockage during inhalation.

While the results, obtained from 12 sets of SIDS parents and 12 sets of non-SIDS parents, "could not be expected to identify unequivocally the cause of SIDS," they do indicate that such deficient responses by the adults "may increase a potential parent's risk of having a child susceptible to SIDS," report Philip L. Schiffman, Robert E. Westlake, Teodoro V. Santiago and Norman H. Edelman.

The scientists note that apnea (momentary loss of breath) and other breathing problems tend to "cluster" in families. One interpretation of the study suggests that "the chances of an infant manifesting an extremely low response [to blockage and/or high carbon dioxide] would, of course, be greatest if both parents were low responders," the researchers say. "However,... an alternative explanation for the familial clustering of low ventilatory responses to carbon dioxide would be that the low response is determined by a separate autosomal-recessive gene.

"... it seems reasonable to us to suggest that an infant with both low ventilatory responsiveness to carbon dioxide and low responsiveness to flow resistive loads [temporary blockage] will be substantially more likely than the average infant to hypoventilate in the presence of an acute increase in resistance to airflow. This could occur at a susceptible age [SIDS usually strikes from 2 to 4 months of age], during an upper respiratory infection ... due to inappropriate upper-airway muscular relaxation or constriction during sleep."

In a separate investigation of three earlier crib death studies, University of Washington epidemiologist Philip Spiers reports that the syndrome appears to strike more often in the western United States, with a steadily increasing cross-country trend from East to West. In an interview with SCIENCE NEWS, Spiers allows that due to a lack of uniform diagnostic criteria and because of other crib death unknowns, his observations may be less than definitive. Nevertheless, he says the available data used in his study—published in the March INTERNATIONAL JOURNAL OF EPIDEMIOLOGY — do point to a significantly higher western frequency of SIDS.

Spiers speculates the trend may involve a greater "population mix" of different gene types in the West, which he suggests might in some way alter some offsprings' immune or other systems. □

# Prize-winning sperm: Raiding the icebox

While it may elicit chilling flashbacks of Hitler's warped visions of a "master race," it is just a "moderately expensive hobby" to 74-year-old California businessman Robert K. Graham. Graham's brainchild is a sperm bank with a rather exclusive list of donors: Nobel-prizewinning scientists. Five laureates—all anonymous except for Stanford University's William Shockley — have already contributed, says Graham, whose venture was publicized last week in newspapers and on national television.

Graham first revealed the project last summer in a bulletin published by Mensa, an organization to which he belongs and which is composed of persons with IQ scores in the top 2 percent of the nation. At the time, he said he was seeking to place the frozen sperm with bright, healthy women under 35 years of age who were married to sterile men. So far, seven women — all on the East Coast — have reportedly received the sperm.

The project, obviously aimed at producing a strain of extremely intelligent children, has predictably drawn sharp criticism from other scientists, humanists and the media; a New York Times editorial suggests that as far as human beings are concerned, "best" is in the eye of the beholder and offers that many women might choose Elvis Presley's genes to Shockley's.

Shockley, who won his Nobel in physics in 1956 but is currently better known for his belief that intelligence is primarily inherited, has said publicly he endorses Graham's effort at "increasing the people at the top of the population." It must also be noted, however, that scientists are unsure how much damage or other alteration genes have undergone by age 70—Shockley's current age. □

# Better assay for congenital CMV

Of the three million babies born in the United States each year approximately 30,000 are infected with a virus called cytomegalovirus. Although most of these infants appear perfectly normal at birth, many will go on to develop deafness, growth retardation, learning disabilities and other infection-caused disorders. And to date, there has been no accurate, rapid, convenient and inexpensive test for screening newborns for CMV.

Now an assay that comes somewhat closer to meeting the above criteria is reported in the February PEDIATRICS by Sergio Stagno, assistant professor of pediatrics and microbiology at the University of Alabama in Birmingham, and his colleagues. The assay, surprisingly, is for rheumatoid factor, the antibody that works against antibodies and is known to play a role in rheumatoid arthritis.

Until now it has been possible to diagnose congenital CMV by taking urine from the newborn, placing it in tissue culture and then isolating a virus from the culture. The problems with this test, though, are that there are not many centers in the United States that do it, it is expensive and it takes more than a week to get results. It also has been possible to use an electron microscope to look for CMV in the urine of newborns, but not every hospital has an electron microscope. Some other tests have been available as well, such as the IgM immunofluorescent test, which looks for antibodies of the IgM class directed against CMV. But one of the drawbacks of this assay is that it gives a lot of false positive results (that is, says that CMV is present in a blood sample when it really isn't).

When Stagno and his colleagues read in the medical literature that one of the reasons the fluorescent test gives so many false positive results is that fetuses and newborns with CMV infection produce rheumatoid factor for some reason, and IgM antibodies are reacting against rheumatoid factor rather than against CMV, they checked for themselves and, sure enough, found the factor in the blood of CMV-infected newborns. They reasoned that if the blood of every CMV newborn contained rheumatoid factor, but the blood of non CMV-infected newborns did not, a test for rheumatoid factor might turn out to be an accurate assay for congenital CMV. What's more, the rheumatoid factor test is especially desirable because it can be done in only a minute and is already commercially available at a modest price. Stagno and his co-workers then decided to see how accurate the rheumatoid factor test was for CMV by giving it, as well as existing CMV assays, to a large number of newborns.

The rheumatoid factor assay, they re-