Rubella threatens unborn in vaccine gap

An aggressive vaccination program has been so successful in curbing German measles that the disease may disappear from the United States within the next 15 years, a federal health official said last week. But in the meantime, the rubella virus, which produces a mild three-day rash in schoolchildren, still poses the threat of severe birth defects to unborn children of the roughly four to five million women aged 20 to 40 who have never been vaccinated or had rubella.

"If we continue what we're doing now, rubella will be eliminated from the United States in the next 15 to 30 years," Alan R. Hinman, director for immunization at the Centers for Disease Control (CDC) told an international conference on congenital rubella in Washington, D.C. U.S. doctors reported an all-time low of 954 cases of rubella last year, Hinman says, and probably fewer than 100 infants suffered virus-induced damage in the womb. He credits the curtailment to a blanket immunization program begun in the early 1970s requiring that all children be vaccinated against rubella before they enter kindergarten or the first grade. While the program has prevented epidemics that used to sweep the United States every six to nine years, it has left a "gap" of unprotected women, currently in their 20s and 30s, who were too old to have been included in the initial school immunization programs.

"That gap is one of the real problems," Hinman says. "Ten to 15 percent of all women in this age group are susceptible and don't know it." Many local health departments offer the vaccine free of charge, he says, and the CDC is attempting to reach the vulnerable women through immunization programs in the military, colleges and family planning clinics.

Though the chances of a pregnant woman contracting rubella in the United States have been greatly reduced, the few cases reported represent not only tragedy to the individuals, but cost to society that far outweighs the cost of an immunization program, Hinman told SCIENCE NEWS. When a woman contracts rubella during the first trimester of pregnancy, her fetus risks a 90 percent chance of infection. About 20 percent of those infected will develop a full-blown syndrome involving defects of the heart and eyes, deafness, mental retardation and a host of maladies that can affect virtually any organ in the body. Many more infants will also be affected to a lesser degree. Most of the children survive, and often incur high medical and educational costs, he says.

The danger of German measles to a developing fetus was first reported in 1941, when an Australian physician linked an unusually large number of children born with cataracts to their mothers' recollections of rashes in the first weeks of pregnancy. The rash was traced to rubella, and historical sleuthing uncovered a similar swelling in the ranks of children born with visual and hearing deficits after each rubella epidemic. The last such epidemic hit the United States in 1964-65, and resulted in 20,000 children with congenital defects and 11,000 pregnancies aborted to avoid the risk of such defects.

Recent follow-up studies of children born with defects associated with the syndrome show that some abnormalities do not surface until the second or third decade of life. An Australian study in 1970 found that 20 percent of the rubella-deafened population afflicted in that country's 1940 rubella epidemic developed either insulin-dependent diabetes or a pre-diabetic condition by the age of 30 — an incidence more than four times higher than the diabetes prevalence in the general population. Since then, researchers around the world have confirmed the Australian study and have detected an increased prevalence of other hormonal abnormalities, including growth hormone deficiency, and an over- or under-production of thyroid hormones in children with congenital rubella.

Several research teams are keeping a close check on the deafened survivors of the 1964 U.S. epidemic for clues to the ways diabetes develops. Fredda Ginsberg-Fellner, Mary E. Witt and co-workers at Mount Sinai School of Medicine in New York have studied 241 patients with congenital rubella, and found immune system irregularities in many that mirror the defects found in insulin-dependent diabetics (SN: 1/3/81, p. 5).

Researchers at the Medical College of Virginia in Richmond and at Gallaudet College in Washington, D.C., are currently collaborating in a similar study of rubella-deafened young adults. They hope the study will lead to better diagnosis and treatment of diabetes. Kathy Shaver, a geneticist at Gallaudet, says the research is based on the theory that the tendency to develop insulin-dependent (also called Type I) diabetes is inherited, and an environmental insult such as rubella virus might activate such a tendency to trigger disease.

—D. Franklin

Phosphorous and the earliest skeletons

About 570 million years ago, the organisms living in the world ocean took a momentous step when they began to relegate calcium carbonate or calcium phosphate to the outsides of their bodies. These coatings became the first preservable hard parts, and their development marks the boundary between the Precambrian and Cambrian geologic periods. In rocks from the earliest Cambrian, shells of calcium phosphate dominate the fossil record, later to be outnumbered by remains of organisms that formed calcium carbonate shells. But some aspects of the events remain a mystery. Two Australian scientists suggest that the early predominance of calcium phosphate shells, and the widespread distribution at that time of phosphorites, or phosphorous-rich sedimentary rocks, were due to a powerful overturning of ocean waters. This upwelling of deep water would have caused a major rise in phosphorous levels in the light-infused portions of the seas, providing the essential nutrients to fuel the rampant evolutionary events at the Precambrian-Cambrian boundary.

This argument is presented in the March 15 NATURE by Peter J. Cook and John H. Shergold, both of the Bureau of Mineral Resources in Canberra, Australia. They hypothesize that the increase in phosphorous, following a period of decreased oceanic overturn, would have caused biomass — particularly microscopic plants in the shallow marine waters — to increase sharply. They argue that the higher level of photosynthesis would have led to increased free oxygen in the atmosphere, but stress that the nutrient concentration, rather than the oxygen, was the "dominant control on evolution." The abundant food supply would have fostered the wide variety of structures observed in Cambrian fossils, and would have encouraged competition for the available environmental niches.

Eventually, the authors write, the supply of phosphorous was depleted, and organisms turned to calcium carbonate as the main material from which to construct their skeletons.

Cook and Shergold base their views both on the known, widespread formation of phosphorites at that time, and on the "high proportion of organisms with calcium phosphate rather than calcium carbonate skeletal structures." By the latter part of the early Cambrian period, calcium carbonate and chitin were the mainstays of skeleton construction, a pattern that persists today. They further argue that phosphorus inhibits calcification. Thus, they contend, until the phosphorus content of the water declined, and the mineral was concentrated into the phosphorite deposits, development of calcareous skeletal structures did not proceed.

As usual with studies of early life, many questions persist. For instance, the authors ask, what triggered the overturn of stagnant ocean water, and the consequent spread of phosphorus into the shallow surface waters? They suggest several possible causes, including glacial episodes that may have affected the density and salinity of seawater, and movement of the earth's crustal plates, which may have opened seaways, facilitating upwelling.

—C. Simon