Gene appears to alter lead's toxicity

New data indicate that a mutant gene appears to modify the body's storage of lead — thus changing what organs face the earliest risk of toxicity.

Because this gene, first described 8 years ago (SN: 2/21/87, p.123), occurs in perhaps 15 percent of U.S. whites but "is essentially nonexistent in African Americans," its effects "could have implications for environmental equity," points out Karl T. Kelsey of the Harvard School of Public Health in Boston, who directed the new study.

In most people, ALAD is the second in a series of enzymes that the body employs to create porphyrins, ringshaped chemicals involved in the production of heme. Heme is the oxygencarrying component of red blood cells. But some people carry an ALAD-2 gene producing a variant form of the enzyme, one that binds lead tightly.

Kelsey and his coworkers decided to investigate how the gene might affect storage of the bone-seeking heavy metal in 691 construction workers, most of them white men. In the March Environmental Health Perspectives, they report that workers with and without the variant gene had similarly low concentrations of lead in their blood (about 7.75 micrograms per deciliter).

However, individuals with the variant gene shunted proportionately more of their stored lead into the spongier trabecular bone (of kneecaps and vertebrae, for instance) than did those with the normal gene. This bone tends to mineralize, taking up and releasing its calcium — and lead — more readily than the denser cortical tissue that makes up shins and other long bones.

In addition, the kidneys of people with ALAD-2 appeared to suffer more stress than those of people with the normal gene. Kidney damage can be a symptom of lead toxicity.

Taken together with earlier studies by others, Kelsey says, his group's findings appear to indicate that in people with the variant gene, lead binds better in the red cells, causing individuals with high exposures (unlike the construction workers) to develop higher concentrations of lead in their blood. But the data also suggest that this tightly bound lead "is less bioavailable to the long bones, which is where long-term lead deposition usually occurs."

Moreover, he notes, a study investigating attention deficits in children exposed to lead, published last year, hints that the ALAD-2 gene may help keep lead from the brain, where it can diminish IQ and neurological function.

Kelsey now plans to study the gene's effect on lead transfer from mothers to nursing babies. — J. Raloff

Vanishing zooplankton

Fish and some birds have become less abundant off the Southern California coast in recent decades. Now, some researchers think they know why: an 80 percent drop in the population of zooplankton — tiny, drifting creatures that form a crucial link in the ocean food chain.

The decline appears to stem from a warming sea surface. The result is "a dramatic change in the abundance of open-ocean sea life," says marine biologist John McGowan of Scripps Institution of Oceanography in La Jolla, Calif. Pacific mackerel, anchovies, and many other fish feed on zooplankton.

McGowan and oceanographer Dean Roemmich analyzed data on zooplankton density and ocean temperatures gathered between 1951 and 1993 during 222 cruises in a 130,000-square-kilometer area off the California coast from San Diego to Point Conception. They found a dramatic drop in zooplankton, along with a rise in ocean surface temperatures of 1.2° to 1.6°C, they report in the March 3 Science. Waters below 200 meters had not warmed.

The result, a steeper density gradient, has slowed the upwelling of nitrates, phosphates, and other nutrients used by phytoplankton to the surface, the researchers suggest. Zooplankton feed on phytoplankton.

The decline in zooplankton may help explain why California fishing tonnages have dropped 35 percent since the 1950s, McGowan says. In addition, the population of the sooty shearwater, a seabird that eats zooplankton, has plunged 90 percent over the last 8 years, he notes. But "there are mixed signals," he adds. For example, squid and whales, which eat fish, seem to be as abundant as ever.

The warming ocean off California may reflect a natural trend. But it could also result from the greenhouse effect, the trapping of heat by certain gases, the scientists say. If ocean surfaces warm globally over the next 40 years, they report, "the biological impacts could be devastating." — J. Kaiser

Infants' deaths become less mysterious

Getting a baby to sleep can require everything from lullables to car rides. And deciding the best sleeping position for infants has proved almost as difficult for parents — and scientists.

Studies suggest that sleeping position may be a life-or-death issue for some infants. Researchers in four countries have found that as a smaller proportion of children slept prone, the rate of sudden infant death syndrome (SIDS) fell. Defined as the unexplained death of healthy infants, SIDS is the leading cause of death of Western children 1 month to 1 year old. As a result of these studies, many physicians now contradict the advice of previous decades and say babies should sleep only on their sides or backs (SN: 7/2/94, p.13).

Nevertheless, "there is by no means a consensus" that changing how children sleep reduces the incidence of SIDS, assert Terence Dwyer and his colleagues from the University of Tasmania in Australia in the March 8 JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION (JAMA). But their new data may convince more doctors to warn of the dangers of infants sleeping prone.

Dwyer and his colleagues have analyzed data on 1,330 infants born each year

in Tasmania since 1988. They are seeing fewer cases of SIDS, largely because fewer infants sleep prone, they say. The rate of SIDS decreased from 7.6 to 4.1 per 1,000 live births between 1988 and 1992. During that time, the percentage of 1-month-old babies sleeping prone dropped from almost 30 percent to 4 percent.

In a separate study in JAMA, Hillary Sandra Klonoff-Cohen and her colleagues at the University of California, San Diego find that sleeping position is less important than another possible cause of SIDS — breathing cigarette smoke.

The scientists interviewed 200 parents of children under age 1 who died of SIDS and 200 parents of healthy babies who were similar to the SIDS victims in age, race, and other characteristics. Most children who die of SIDS are 2 to 4 months old.

The team found that about two-thirds of both the youngsters who died of SIDS and the healthy babies routinely slept on their abdomens. However, 80 percent of the SIDS victims were prone when they died.

Exposure to smoke also puts children at risk of SIDS, Klonoff-Cohen and her group report. Infants breathing the smoke of others in the same house were 3 1/2 times more likely to die of SIDS than the youngsters not exposed to smoke in their homes, they say. Smoke may impair the control of children's breathing, scientists suggest.

"In view of these [studies'] results, how should health care professionals advise parents?" Marian Willinger of the National Institutes of Health in Bethesda, Md., asks in an accompanying editorial. She recommends keeping babies away from smoke as much as possible and off their stomachs when sleeping.

— T. Adler

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