

Smoking mothers may endanger children

In another study on the hotly contested issue of passive smoking (SN: 1/24/81, p. 53; 7/4/81, p. 6), scientists from Beth Israel Hospital and Harvard Medical School in Boston have found that the lungs of children of smoking mothers grow at a slower rate than the lungs of children of non-smoking mothers. They previously reported that children of smokers had overall poorer lung function than did children of non-smokers (SN: 6/3/78, p. 361).

The researchers followed the change in FEV₁—the amount of air that can be forcefully expelled from the lungs in one second—in 1,156 children who spent more time around their mothers than their fathers. "After five years," they report, "the lungs of non-smoking children with mothers who smoke grow at only 93 percent of the rate of growth in non-smoking children with mothers who do not smoke."

Their conclusion? "Passive exposure to maternal cigarette smoke may have important effects on the development of pulmonary function in children," they report in the Sept. 22 *NEW ENGLAND JOURNAL OF MEDICINE*.

Scott Weiss, a member of the research team, says there are several possible causes of the diminished lung function. "It may be all an in utero effect, and not environmentally determined," he says. "It may be environmental—there may be a direct toxic effect. Or the effects may be mediated through respiratory infections or allergy," which he says occur more frequently in children of smokers.

A spokesperson for the industry-funded Tobacco Institute in Washington, D.C., criticized the treatment of data in the study as well as the failure to measure directly the children's exposure to cigarette smoke. Replies Weiss, "The fact that we found an effect may mean the effect would be more obvious and quantifiable if we used a more precise method of exposure."

Back to drawing board for fat cause?

Several years ago three Harvard Medical School researchers reported a possible factor in obesity (SN: 11/8/80, p. 295)—fewer sodium-potassium pumps in the cells of the body. The pumps, which they measured by determining the levels of a pump-associated enzyme (sodium-potassium ATPase), move sodium and potassium in and out of cells, using up energy in the process. Fewer pumps use less energy, they said, and less energy means fewer calories burned. So obese people with fewer pumps might eat the same amount of food as svelte people, yet gain weight.

But don't blame it on your pumps—yet. The number of pumps depends on ethnic origin and does not cause obesity, three researchers from the Scripps Clinic in La Jolla, Calif., report in the Sept. 29 *NEW ENGLAND JOURNAL OF MEDICINE*. They found Scandinavians tend to have more pumps than black, Asian and Jewish subjects, but no pump-obesity correlation within each group.

The California researchers had initially found the pump-obesity relationship too, but when they took a closer look at more people they found no difference between non-obese and obese people, as well as recent weight losers. Ethnic differences "can readily explain most of the earlier results," they report.

"Our conclusion is that if you look at a group of obese people and happen to have an increased representation of groups where ATPase activity is low [compared to the control group], you'll see a difference," says Ernest Beutler, who headed the California study. "Ethnic group by ethnic group, you see no relationship," he says.

But the Harvard group is sticking by its study for now. "It's an interesting piece of work, but what their observation means in respect to ours, I'm not sure yet," says Jeffrey Flier. Since their initial report, Flier says, his group has also found that ethnicity is a factor, but that the numbers of pumps do make a difference. In a study of Jewish Caucasian women between the ages of 30 and 40, "we found the same thing we did the first time," he says.

Looking for cleaner water softeners

A lot of salt is used to soften water. And unless downstream water users enjoy drinking brine, there's a big problem brewing, notes Purdue University engineer James Etzel. This West Lafayette, Ind., researcher also thinks he's onto a solution.

Conventional water softeners use polymer beads in an ion-exchange process. Initially, sodium ions cling to the polymer. But when calcium and magnesium ions—responsible for water hardness—wash through, they preferentially bind to the polymer. In the process, two sodium ions are kicked out for each calcium or magnesium ion bound. When the sodium is gone, concentrated brine is flushed through to regenerate the system. However, since only about 40 percent of the salt adheres to the column—the rest washes right through—a heavy load of salt ends up being pumped into community water supplies.

Household water softeners may add 3.5 pounds of salt for each pound of magnesium and calcium removed. And water used by the electronics industry, which must be devoid of impurities, may require 6 pounds of salt for each pound of contaminants removed. Etzel says many municipalities that now routinely soften their water have been told that "in the foreseeable future, Environmental Protection Agency regulations will prohibit them from discharging their regenerated brine."

Etzel's alternatives use organic chemicals that are either biodegradable and nontoxic, or are recyclable (even within an individual household) in place of the inorganic salt. Choline chloride is being tested in the degradable system. As this compound is already used as an animal-feed additive, Etzel says it is environmentally safe. A high-molecular-weight organic salt—hexadecylpyridinium gluconate—is used as the recoverable regenerant in the other system. Although such systems "would likely cost twice what you pay now," Etzel says they may prove the only ones society will tolerate. Commercial versions could be available within a few years, he says.

Love Canal—the vole toll

A four year survey of meadow voles, or field mice, living next to the Love Canal toxic-waste dump in Niagara Falls, N.Y., indicates notable wildlife poisoning. "The voles of Love Canal are dying prematurely, and the closer they live to the canal itself the younger they are dying," says John J. Christian of the State University of New York at Binghamton. Analyses confirmed the presence of several toxic halogenated hydrocarbons—including the pesticide lindane—in the animals' fatty tissue. Even voles living in an Emergency Declaration Area near the dump showed biological contamination and a reduced lifespan, notes Christian in the October *NATURAL HISTORY*. While this region was largely evacuated in 1980, roughly 150 families are still there.

Christian and colleagues trapped voles in three areas: along a fence (defining the inner-ring area evacuated in 1978) that surrounds the dump, along a road in the surrounding Emergency Declaration Area and in fields a mile from the dump. While average vole lifespan in the control area a mile from the dump was roughly 154 days, in the Emergency Declaration Area it was 105 days, and at the inner-ring fence an average of 84 days. Female voles had higher death rates, which Christian says might be because females metabolize toxic chemicals more quickly.

Though the more contaminated animals developed liver abnormalities and other organ changes, a precise cause of premature death has not been established. However, because levels of glycogen (stored energy) in the livers of inner-ring dwelling voles were low, Christian speculates the animals may lack sufficient energy to find food, escape predators, even to keep warm.

Among the more serious concerns raised by his findings, Christian notes that the most highly poisoned animals were captured in an area accessible to children and pets still living in the Emergency Declaration Area.