SCIENCE NEWS of the week

Dioxin's 'Fingerprint' Lingers for Decades

Over time, the human body rids itself of some of its dioxin contamination. Only a few years ago, scientists thought that telltale residues even of high exposures would fade beneath the level of detection, erasing evidence of exposure, long before any manifestation of disease. But several new studies recently presented at an international meeting in Fukuoka, Japan, show that a characteristic, latent "fingerprint" of dioxin contamination remains in body fat more than 20 to 30 years after exposure. Two of the studies were even able to find that indelible fingerprint in the blood.

These studies, reported at the Sixth International Symposium on Chlorinated Dioxins and Related Compounds, hold open the prospect of at last identifying individuals who had heavy dioxin exposure in the past. It is by comparing their health against that of individuals with almost no exposure that dioxin's elusive toxicology is likely to be resolved.

Recently, several research teams have reported using sophisticated analytical techniques to identify an apparent low-level background human contamination with dioxins in industrialized societies (SN:7/13/85,p.26). But the exposures they identified were not necessarily old, nor did most show high elevations of dioxins in those individuals who had — according to questionnaires — received potentially heavy exposure. At the Japan meeting, several research groups reported finding fingerprints that appeared not only to be decades old but also to earmark signs of heavy exposure.

For example, traces of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) were identified in the fat of six workers contaminated during a 1953 chlorophenol explosion in West Germany. Arnold Schecter of the State University of New York in Binghamton and John J. Ryan of Health and Welfare Canada in Ottawa report that one of these workers still carries TCDD levels of 141 parts per trillion (ppt) in his fat — 10 to 20 times the general population's background level. "If we calculate back, using a five-and-a-half-year half-life, he must have had 12,000 ppt TCDD contamination at the time of the initial exposure," Schecter says. Ironically, he adds, this man was denied workmen's compensation for health problems because he's been unable to prove heavy contamination.

Inability to demonstrate contamination has also plagued U.S. Vietnam veterans with health problems that they believe may be the result of exposure to Agent Orange (SN:5/19/86,p.314), a defoliant contaminated with trace levels of TCDD. But at the meeting, a researcher with the New Jersey Agent Orange Commission in Trenton reported finding an average 10-fold excess of TCDD — roughly 45 ppt in fat — among 9 of 10 veterans who had been Agent Orange sprayers in Vietnam roughly 20 years ago. They compared this group with two sets of matched controls: veterans who never served in Southeast Asia and Vietnam veterans whose jobs should not have exposed them to Agent Orange. Until now, says Peter Kahn, a biochemist from Rutgers University in New Brunswick, N.J., and one of the study's authors, "nobody had established that heavily exposed Vietnam veterans had more [TCDD in them] than unexposed veterans."

Because the body stores dioxin in fat, human dioxin analysis has been done on fat-rich breast milk or on surgically extracted fat. Most researchers would prefer a simple blood test. At the Japan meeting, both Kahn's group and chemists from the Centers for Disease Control (CDC) in Atlanta independently reported for the first time being able not only to detect dioxin in the fat of blood from people who had fasted, but also to demonstrate that the concentration of dioxins in the blood's fat matches their concentration in the body's fat deposits. (Fasting draws stored fat into the blood.)

Both Kahn and Larry Needham at CDC say their technique needs some refinement before dioxin analysis can be reliably conducted using blood. Even then, Needham says, this type of analysis — because it's complicated, expensive and requires a lot of blood — is not likely to become "routine." *— J. Raloff*

AIDS: Treatment and transmission

"It's been a tumultuous month in the AIDS field," Martin S. Hirsch of Massachusetts General Hospital in Boston said at the Interscience Conference on Antimicrobial Agents and Chemotherapy this week. And several presentations at the New Orleans conference relating to drug treatments and heterosexual transmission may add to the rapidly accumulating data bank, if not to the tumult.

Hirsch's comment was in response to the announcement of the expanded availability of azidothymidine (trade name AZT), an anti-AIDS drug (SN: 9/27/86,p.196). Samuel Broder of the National Cancer Institute released further data on the AZT trials at the meeting, including a description of what happens to disease-fighting T4 cells during drug treatment. Viral killing of these white blood cells is blamed for the deadly immunosuppression of AIDS.

While AZT did initially boost the already-low T4 levels — as those of placebo-treated patients continued to fall — the average in the AZT-treated group eventually dropped to pretreatment levels, presumably through AZT's suppression of bone marrow production of T4 precursors. But Broder says the fall isn't an overwhelming concern. Only *some* patients showed the decrease, he says, "and even with some patients' fall, [AZT] does seem to be translating to a clinical benefit."

Broder says he's just gotten a go-ahead from the Food and Drug Administration to try a similar drug, dideoxycytidine, in AIDS patients. Other drugs under development were discussed at the meeting, including D-penicillamine, a compound marketed for rheumatoid arthritis and Wilson's disease, a rare metabolic disease. George Washington University researchers in Washington, D.C., tried that drug in 10 patients who had the AIDS virus and perpetually swollen glands. It suppressed the virus but also temporarily depressed T cell function.

Raymond Schinazi and colleagues at Emory University in Atlanta are working on a drug similar in structure and function to AZT. Called CS-85, it is at least 7 to 10 times less toxic than AZT in bone marrow cell culture, he says.

On the transmission side, several studies reported at the meeting added to the accumulating evidence of female-to-male transmission (SN:10/5/85, p.213). While AIDS incidence in Africa and Haiti indicates that such transmission occurs, it has been difficult to document in the United States because of the relative rarity of female cases.

Brian Salzman and his colleagues at Montefiore Medical Center in New York City studied 12 male partners of female AIDS patients. The men had no other risk factors for AIDS, yet six of them had antibodies to the virus, indicating that they had been exposed.

Heterosexual transmission apparently doesn't require multiple exposures to the virus: A study by the Centers for Disease Control turned up the case of a woman who developed blood signs of viral exposure after only one sexual encounter with her husband, who had picked up the virus via transfusion. *— J. Silberner*