PCBs' legacy can affect next generation

Seventeen years ago, 2,000 residents of central Taiwan fell prey to a range of debilitating and disfiguring symptoms, including stomach pain, chemically induced acne, and nerve inflammation. This poisoning, known as the Yu Cheng incident, traced to a machine for commercially clarifying rice oil that accidentally leaked polychlorinated biphenyls (PCBs) into the edible product.

Though Yu Cheng has been largely forgotten throughout most of the world, its legacy endures—in a new generation and a different form. Children born to women poisoned during the incident now exhibit a host of physical, neurological, and developmental abnormalities, researchers reported last week at the International Neurotoxicology Conference in Hot Springs, Ark.

Once widely used as an electrical insulator in transformers and other equipment, PCBs today contaminate most humans and wildlife. A mother will pass some PCBs on to her baby during pregnancy and breast-feeding. Though no one knows exactly how much the Yu Cheng mothers transferred to their babies, measurements were made of PCBs in the blood of these women in 1992, notes Yue-Liang Leon Guo of the National Cheng Kung University Medical Center in Tainan, Taiwan.

Those assays showed that the women had about 50 times more PCBs in their blood than unexposed adults did. Their children, however, possessed only about 6 times the concentrations seen in other youngsters.

Walter J. Rogan of the National Institute of Environmental Health Sciences, in Research Triangle Park, N.C., served on a team that studied the health of 117 Yu Cheng children. He notes that many possess unusual dark pigmentation on parts of their bodies, bronze-colored nails, recurrent conjunctivitis, and teeth that fall out easily.

More troubling is "a global cognitive delay [among these children] in every measure we have tested," he says. Compared to neighboring children born with normal, background concentrations of PCBs, most Yu Cheng children exhibit small, but significant, delays in attaining normal developmental milestones, such as stacking blocks, speaking, and walking.

Seen as early as 6 weeks, when an infant should display its first "social smiles," he says, these delays remain in evidence through at least age 11. So "these kids don't seem to get better" over time, Rogan concludes.

Not all Yu Cheng children exhibit all the symptoms, nor do reasoning and problem-solving delays necessarily correlate with physical symptoms, such as pigmented nails.

Yu Cheng children have also proved twice as likely as age-matched, unexposed Taiwanese youngsters to suffer from middle ear disease, reported Guo. But if one looks just at the incidence of severe problems, such as recurrent ear infections, the 100 Yu Cheng children he's following experience about six times more, he points out.

At age 8 to 13, Yu Cheng girls tend to be not only 2 to 3 centimeters shorter than unexposed girls, but also leaner and less muscular, Guo finds. Exposed boys show no comparable growth deficits at that age, but they do produce unusually high concentrations of estrogen, the primary female sex hormone.

To explore the cognitive differences picked up by Rogan's team, Guo's group has administered a series of psychological tests to roughly 90 children born to Yu Cheng victims between 1985 and 1991 and to a similar number of age-matched children whose parents had not been exposed. Though boys ordinarily outperform girls on matching and reasoning tests that rely on spatial or visual cues, Yu Cheng boys lost that advantage. They performed less well than the other boys—and equivalent to both groups of girls.

Yu Cheng girls, Guo found, preferred the same toys and playtime activities—such as sports and video games—selected by both groups of boys. These girls generally expressed a dislike for making friends, caring for animals, and playing with Barbie dolls, all favored by the other group of girls.

Both Rogan and Guo expressed hope that by probing Yu Cheng's legacy in this new generation of children, an understanding may emerge about what signs of toxicity to look for in less exposed populations. However, Guo points out, because the PCBs in the Taiwanese accident were themselves laced with dibenzofurans—a family of compounds closely related to dioxins—some share of the youngsters' toxicity may also trace to those substances. *— J. Raloff*

Hallucinating brains pose for first scans

A 23-year-old man suffering from schizophrenia sits in a laboratory and hallucinates. He sees disembodied human heads rolling across a vivid backdrop, uttering curses and insults at him.

As this bizarre foray into psychosis begins, the man presses a button that activates brain-imaging equipment. A halo of sensors measures the blood surging and ebbing throughout his brain, enabling scientists to enter the anatomical realm of hallucinations.

Brain scans of this man and five people who frequently hear voices as a result of their schizophrenia have yielded the first direct look at the biological underpinnings of hallucinations, according to a report in the Nov. 9 NATURE.

"We now have a map of brain areas involved in the production of hallucinations," contends David A. Silbersweig, a psychiatrist at New York Hospital–Cornell University Medical Center in New York City. "The network of structures that we found is larger than was previously suspected."

Silbersweig and New York Hospital radiologist Emily Stern collaborated with researchers at Hammersmith Hospital in London. Silbersweig and Stern developed several technical innovations that allowed volunteers to activate PET scanners during hallucinations by pressing a button.

A total of 22 to 25 blood flow scans were obtained for each participant, about half during hallucinations and the rest when no voices or visions intruded on their consciousness. The researchers were then able to tag brain areas uniquely linked to hallucinations.

In the five people who heard imaginary voices, blood flow rose markedly in several areas—the hippocampus, parahippocampal gyrus, cingulate gyrus, thalamus, and striatum. These regions, which lie below the brain's outer cortical layer, help to integrate knowledge about one's personal history and emotions with current and past sensations, the scientists assert.

Tissue at the brain's surface involved in hearing also showed blood flow gains during auditory hallucinations.

The man who saw acid-tongued rolling heads displayed comparable, but more widespread, blood flow jumps, notably in cortical areas that integrate sights with sounds, the investigators hold.

Hallucinators also experienced blood flow drops in regions at the front of the brain and in an area that may determine whether voices originate from the self or others. But a structure considered crucial for talking silently to oneself was unaffected by the presence of imaginary voices, Silbersweig notes.

These findings expand on PET data obtained from hallucinators at times when they did not hear voices, he adds (SN: 9/9/95, p.166).

Overactive inner-brain areas that help regulate the chemical messenger dopamine, combined with sluggish frontal-brain structures, may set the stage for hallucinations, Silbersweig theorizes. Inappropriate neural activity in acoustic or visual regions near the back of the brain may then get misinterpreted as a genuine experience, he suggests.

— B. Bower