Firefighter Findings
Biomonitoring Rescue Workers after WTC Attacks

As soon as it was evident that the World Trade Center (WTC) was being attacked on 11 September 2001, New York City firefighters responded to save lives. In the weeks of rescue and recovery efforts that followed the disaster, the firefighters continued to brave an unprecedented plume of fire-related chemicals and pulverized debris released by the collapse of the WTC and the fires that ensued. This month, researchers led by Philip Edelman of the Centers for Disease Control and Prevention report their analysis of chemicals found in the blood and urine of firefighters who worked at the WTC site [EHP111:1906–1911].

Three weeks after the disaster, scientists from the Centers for Disease Control and Prevention, in collaboration with the New York City Fire Department Bureau of Health Services, collected blood and urine samples from 370 firefighters working at the WTC site. The samples were categorized into exposure groups based upon when the firefighter arrived at the site, length of time spent at the site, and specific job assignments. The samples were then tested for traces of 110 potentially fire-related chemicals and compared to control samples obtained from 47 firefighters who had been previously assigned to desk duties and were not present at the WTC during the fires.

Edelman and colleagues found that 9 of the 110 chemicals were elevated in samples from firefighters who responded to the WTC as compared to the control firefighters. Special Operations Command firefighters—rescue, squad, and marine workers, whose training and assignments render them likely to receive more intense exposures—were exposed to a larger number of chemicals, with elevated concentrations of 14 potential toxicants.

The chemicals most commonly detected in exposed firefighters were metabolites of polycyclic aromatic hydrocarbons, which are known products of combustion. The most commonly found of these compounds was 1-hydroxypyrene, which was found to be twice as high in Special Operations Command firefighters as in other exposed or control firefighters. However, it is not known from this study whether Special Operations Command firefighters are continually exposed to higher levels of these chemicals or if these measurements reflect exposures that occurred principally during the WTC disaster.

Other chemicals that were detected to be higher in exposed firefighters were antimony, lead, heptachlorodibenzodioxin, and heptaehlorodibenzoofuran. Firefighters who arrived early to the disaster, especially those who witnessed the collapse of the towers, were more likely to have higher levels of these chemicals.

Although the levels of several of the chemicals measured were higher in the exposed firefighters as compared to the control firefighters, it is important to note that the levels were still relatively low compared to safety standards set for workplaces and the general population. None of the levels found are known to relate to clinical symptoms. However, limitations in the study make it impossible to predict whether firefighters who responded to the WTC disaster may have had exposure to other chemicals for which biomonitoring methods do not exist as of yet, such as asbestos and fiberglass.

This study is the only biomonitoring investigation to be conducted while rescue operations were ongoing, and it provides an exposure profile of a large group of firefighters. The authors state that biomonitoring methods such as those described in this report can be effective tools for determining exposure to toxic chemicals in firefighters. Biomonitoring of blood and urine can also serve to determine whether worker protection strategies are effective. —Luz Claudio

Insult to Newborn Immunity
Organochlorines in Mother’s Diet

In remote Atlantic coast villages of Canada, residents of small fishing communities who rely on marine food for subsistence often absorb unusually high doses of chemicals including organochlorines, a group of persistent industrial and agricultural chemicals that bioaccumulate in the aquatic food chain. Previous studies have shown that prenatal exposure to some organochlorines can result in suppression of children’s immune systems. Now a team of researchers led by Houda Bilrha of the Centre Hospitalier Universitaire de Québec–Laval University Medical Center in Quebec City has located a biomarker of immune system function—a chemical imbalance—that could indicate damage caused by organochlorines [EHP 111:1952–1957].

The organochlorine family includes polychlorinated biphenyls (PCBs), dichlorodiphenyltrichloroethane (DDT), dichlorodiphenylchloroethylene (DDE), hexachlorobenzene (HCB), polychlorodibenzofuran (PCDDs), and polychlorodibenzofurans (PCDFs). Once released, organochlorines circulate with atmospheric currents, traveling northward (in the Northern Hemisphere) from
southern latitudes into the globe’s colder regions. The chillier temperatures there cause the chemicals to be deposited on the sea surface by rain or snow, or they adhere to the sea surface through a process called vapor phase adsorption. Once thus trapped there, organochlorines accumulate in fatty tissues of organisms and biomagnify in the food chain. In colder regions, relatively high levels of these compounds have been found in the bodies of animals and people, as well as their offspring.

The Mid and Lower North Shore of the St. Lawrence River, a remote Atlantic coast region of Québec, comprises 21 communities spread over a shoreline of several hundred kilometers extending from Sheldrake to Blanc-Sablon. A large proportion of the residents rely on marine food for subsistence. Several surveys conducted since 1990 have revealed that communities on the Lower North Shore, in particular, are overexposed to PCBs, mercury, and dioxin-like compounds, compared to other populations in southern Québec.

Bilhā and colleagues examined a study population of 47 pregnant women who had resided for at least five years in subsistence fishing communities of the Mid and Lower North Shore. The reference group comprised 65 women who were residents of two nearby non–subsistence fishing communities and who therefore could be expected to have lower exposure to food chain contaminants.

Cord blood samples were collected when each woman gave birth, and were analyzed for concentrations of mercury, 14 PCB congeners, and 11 chlorinated pesticides and metabolites. The researchers also isolated immune cells from cord blood samples to test their responsiveness to immune insult by exposing them to phytohemagglutinin, a mitogen used to assess potential for T cell responses. Once stimulated, immune cells normally respond by producing cytokines, small proteins that mediate and regulate immune system function. The researchers tested whether cytokine production by immune cells collected from cord blood was impaired in newborns that had been exposed to PCBs and other organochlorines in utero.

As expected, mean concentrations of p,p’-DDE, HCB, and PCBs were significantly higher in the subsistence fishing group than in the reference group. DDT is not as persistent at its main metabolite DDE and was not detected in most participants. More importantly, the researchers found that lymphocyte cells of newborns exposed to higher concentrations of PCBs and DDE secreted fewer cytokines than those of control newborns.

These results suggest that DDE and PCBs subtly affect the immune system of young children, which could potentially make them more susceptible to infectious diseases. The authors are planning an epidemiological study to investigate whether these subtle biological effects translate into a higher risk of infectious disease in infants from these coastal populations. –John Tibbetts

Flavonoids in Food
How Potent Is Their Protection?

Telling a child “eat your chemoprotective phytochemicals” is just one way of saying “eat your vegetables—they’re good for you!” Now science can back up this parental wisdom with molecular studies that demonstrate plants’ disease-fighting potential. In vitro studies have shown that certain phytochemicals called flavonoids interact with the aryl hydrocarbon receptor (AhR), the same receptor through which 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and other halogenated aromatic compounds exert their toxic and mutagenic effects. In some types of cancer cells, flavonoids may inhibit TCDD from activating the AhR, exerting a chemoprotective effect, report Shu Zhang, Chunhua Qin, and Stephen H. Safe of Texas A&M University [EH1 111:1877–1882].

Zhang and colleagues assert that the traditional method for measuring TCDD toxicity fails to address the increasing evidence that the AhR binds a host of endogenous chemicals considered to be chemoprotective, such as phytochemicals. In other words, TCDD may not be as dangerous to human health as expected, given that we eat foods that may protect against its effects. The team therefore assessed the interaction of TCDD and flavonoids with an eye toward determining whether the effect of flavonoids needs to be taken into consideration when assessing the overall toxicity of TCDD to humans.

The researchers used an AhR-responsive assay to test the action of 13 dietary flavonoids in human breast and liver cancer cells and mouse liver cancer cells. The assay used a luciferase gene that was activated only when the compounds interacted with the AhR in the cell lines.

Some flavonoids turned out to be AhR agonists—that is, they mimicked TCDD’s action—and others were antagonists—they blocked stimulation of the AhR by TCDD. Their potency varied, usually by cell type (mouse versus human). The maximum level of agonist activity reached by a flavonoid was just 25% of that reached by TCDD. In terms of antagonist effects, one flavonoid (luteolin) acted as an AhR antagonist, and two (baicalein and cantharidin) triggered production of a protein that helps metabolize carcinogens. “These data suggest that dietary phytochemicals exhibit substantial cell context–dependent AhR agonist as well as antagonist activities,” the team reports.

The results of this study complement previous reports showing the AhR agonist and antagonist activities of numerous chemoprotective phytochemicals. Because food has relatively high concentrations of compounds that interact with the AhR, the authors write that “risk assessment of dietary toxic equivalents of TCDD and related compounds should also take into account AhR agonist/antagonist activities of phytochemicals.” –Tina Adler