Pyrethroids in the Home

Nondietary Pesticide Exposure in Children

Because pyrethroid pesticides are often used in conventional agriculture, people are routinely exposed to trace amounts in foods. Similar exposure to organophosphorus (OP) pesticides has been described previously in results from the Children's Pesticide Exposure Study, an investigation of pesticide exposures among 23 Seattle children aged 3–11. Unlike OP pesticides, however, pyrethroids are also approved for residential use. The latest findings from this study show that residential use of pyrethroids appears to be a more significant source of exposure to this class of pesticides than diet [***EHP* 114:1419–1423; Lu et al.**].

With the phaseout of residential use of the commonly used OP pesticides chlorpyrifos and diazinon, home use of pyrethroids has increased. Depending on the compound and the dose, pyrethroids may affect neurological development, disrupt hormones, induce cancer, and suppress the immune system. However, little is known about the extent and effects of human exposure.

Close to the source. New data show that OP pesticides used in the home contribute more to children's exposure than diet.

Using samples collected during the summer of 2003, researchers at Emory University and the CDC determined urinary pyrethroid metabolite levels during 15 consecutive days for each child. During days 1–3 and 9–15, the children consumed foods prepared from conventionally grown crops. On days 4–8, organic items were substituted for plant-based foods such as fruits, vegetables, pasta, and cereal.

During the entire 15-day sampling period, the dominant metabolite seen was PBA, a nonspecific metabolite of permethrin, cypermethrin, and deltamethrin. PBA was detected in 82% of samples and had the highest median concentration, 0.45 µg/L. *trans*-DCCA and *cis*-DCCA, metabolites of permethrin, cypermethrin, and cyfluthrin, were also common, detected in 71% and 35% of all samples, respectively. Concentrations of *cis*-DCCA were too low to quantify; the median *trans*-DCCA concentration was 0.38 µg/L. The metabolites FPBA, derived from cyfluthrin, and DBCA, derived from deltamethrin, were each detected in only 2% of samples.

Comparing metabolites between dietary phases, the researchers saw no apparent trend. However, seven children in families that reported using pyrethroid pesticides had significantly higher levels of PBA and *trans*-DCCA than the other children and accounted for most of the FPBA-containing samples and all of the DBCA-containing samples. Interestingly, the older children experienced higher exposure than the younger ones. Typically younger children have higher exposure due to behaviors such as mouthing items and playing on floors, but the older children in this study spent time at sports facilities where pyrethroids may have been used.

The researchers conclude that an organic diet alone is unlikely to dramatically decrease a child's exposure to pyrethroids the way it does exposure to OP pesticides. Limiting residential use of pyrethroids and preventing children's contact with treated areas are very likely the best measures for decreasing their exposure to these pesticides. **–Julia R. Barrett**

Heavy Traffic Can Be a Pain in the . . . Ear?

Vehicle Emissions Linked to Otitis Media

Traffic is a major source of air pollutants, and more studies are looking at the role of traffic-related air pollution in children's health. Researchers report in this issue that young children exposed to higher levels of traffic pollution have a greater incidence of otitis media (middle ear infections) than those exposed to lower levels [***EHP* 114:1414–1418; Brauer et al.**]. In 2002, the same team found that such pollution increased the risk for asthma and upper respiratory tract infections in young children. Now they focus on otitis media because upper respiratory tract infections often progress to ear infections, which are one of the leading reasons for visits to doctors and the use of antibiotics in childhood.

The new study involved approximately 3,700 Dutch children and 650 German children surveyed from birth to age 2 years. Researchers in those countries monitored the concentrations of three common traffic-related pollutants (nitrogen dioxide, particulate matter less than 2.5 µm in diameter, and elemental carbon) at 40 different sites in each country, then used those data to estimate exposures at each child's residence. The levels of pollutants measured were similar in both countries and fell within a range commonly experienced by people living in industrialized nations. Information about doctor-diagnosed otitis media came from questionnaires answered by parents.

Aural pollution? Traffic pollution is linked to increases in cases of ear infection in children.

Both groups of children showed an increase in otitis media in association with greater traffic pollution exposure. By age 2, a third of the children had experienced otitis media at least once. The adjusted odds ratios of contracting otitis media associated with modest increases in exposure to the different air pollutants ranged from 1.09 to 1.24, and the risk of ear infections was similar for each of the three pollutants measured. Although environmental tobacco smoke has been linked to otitis media in studies by other researchers, exposure to this agent did not alter the associations between traffic pollution and otitis media observed in this study.

Otitis media has been estimated to cost the U.S. health care system $3–5 billion yearly. These findings, the first to link traffic pollution to otitis media, represent an additional consequence of air pollution. Protecting children from exposure to vehicular emissions—for example, by building major roadways away from residential zones, improving automobile emission standards, and driving less—may reduce the risk of otitis media. **–Carol Potera**

Top to bottom: Rebecca Ellis/iStockphoto; Photodisc

A Lethal Change in the Weather

Temperature Extremes and Premature Mortality

Extremely hot and cold days can be fatal to certain vulnerable populations, as the more than 160 deaths in two weeks during California's July 2006 heat wave clearly showed. The elderly and lower-income individuals are generally acknowledged to be most vulnerable to the effects of temperature extremes, but relatively little is known about how such extremes combine with underlying medical conditions to increase mortality risks. Now, a team from the Harvard School of Public Health has analyzed millions of death records from 50 U.S. cities to identify factors that increase the risk of dying on extremely hot or cold days [***EHP* 114:1331–1336; Medina-Ramón et al.**]. The study is the first of its size to identify specific diseases that produce the largest relative mortality increases on extreme temperature days.

The researchers examined approximately 7.8 million death records for the period 1989 through 2000. They defined extreme temperatures for each city as the coldest 1% of daytime highs and the warmest 1% of nighttime lows. These are the most physically challenging conditions, with people unable to warm up even in the daytime or cool off even at night. The data were analyzed using a case-only approach, a technique borrowed from genetic research that allows the identification of time-invariant factors (such as gender) that modify the effect of a time-variant risk factor (such as weather). This approach allowed the researchers to compare the individual characteristics of those dying under extreme weather conditions with those dying on other days.

The study's large sample size provided enough statistical power for researchers to see how a variety of individual characteristics, including presence of chronic conditions, affected vulnerability to weather extremes for a specific cause of death. For example, previous studies had already shown that blacks are more likely than whites to die on a hot day, but the authors found that susceptibility in this subgroup was more pronounced when death was due to cardiovascular disease. Conversely, the elderly and diabetics were more vulnerable to heat when the primary cause of death was *not* due to cardiovascular disease. The researchers also found a large increase in vulnerability to heat in individuals who suffered from atrial fibrillation, a finding that has not previously been reported. Cardiovascular deaths, especially cardiac arrest deaths, also showed a greater relative increase on extremely cold days.

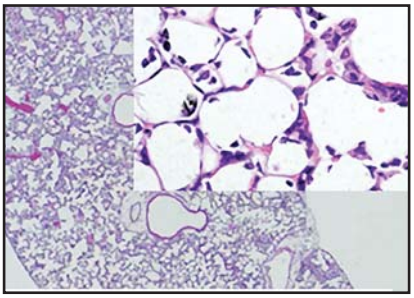
Hot science. Identifying disease risks related to extreme temps can help officials target services such as fan distribution to those most at risk.

The authors note that public health officials can target appropriate health services and infrastructure by knowing which subpopulations are particularly at risk from temperature extremes as well as the most common mortality causes that may affect them. This kind of information will be even more important as some subgroups (such as diabetics and the elderly) increase as a percentage of the population at large, and as global warming raises the probability of higher maximum temperatures. **–Nancy Bazilchuk**

Tiny Intensifiers

Nanoparticles Worsen Lung Effects of Bacterial Endotoxin

Exposure to particulate matter in the air, especially extremely fine particles, has been associated with increased morbidity and mortality from lung and cardiovascular disease. Effects grow more significant with decreasing particle size. However, the size-related effects of nanoparticles—particles less than 100 nm (0.1 µm) in diameter—on pulmonary inflammatory conditions have not been fully investigated. This month a team of Japanese investigators reports that nanoparticles can increase lung inflammation associated with bacterial endotoxin, or lipopolysaccharide (LPS) [***EHP* 114:1325–1330; Inoue et al.**].



Nanoparticles in inflamed lung tissue

Inhalation of particles with a mass median aerodynamic diameter of 10 µm or less is associated with increased hospitalization for asthma, bronchiolar irritation, and lower respiratory tract infections, while exposure to particles 2.5 µm and smaller, including common carbon-cored pollutants from diesel exhaust, exhibit a stronger epidemiological link to death from cardiopulmonary and respiratory effects. Particles even smaller, 0.1 µm or less, are thought to move beyond the respiratory system, perhaps reaching the blood stream. The tiniest particles are not just smaller than other pollutants; they have more surface area for a given weight—imagine the difference between the surface area of a solid glass cube compared to that of an equal weight of fine glass beads. Both the small size of nanoparticles and the high surface area that they present to cells may contribute to their effects.

For the current study the researchers used ultrafine carbon black, a form of elemental carbon used in the printing industry, to explore how exposure to nanoparticles impacted antigen-related inflammation of airways in mice. Using carbon black formulations with diameters of 14 nm and 56 nm, they looked at how LPS's effects changed in the context of nanoparticles in the airway.

The effects of the nanoparticles by themselves was slight, while exposure to LPS alone increased by 12-fold the number of cells harvested by bronchoalveolar lavage (a measure of airway inflammation). However, simultaneous exposure to LPS and to 14-nm particles amplified the effect to yield a 20-fold increase. Simultaneous exposure to LPS and to 56-nm particles resulted in a smaller, statistically insignificant boost in the effects of LPS.

It was not just cell infiltration that was affected. Histology showed that lung exposure to a mixture of 14-nm particles, which had only minor effects themselves, and LPS led to recruitment of neutrophils in the parenchyma, the actual respiratory surface of the lung. LPS-driven oxidative stress and expression of chemokines and cytokines also were amplified in the presence of these small particles, and changes in blood coagulatory factors were seen as well. The larger 56-nm particles increased the effects of LPS in some but not all assays.

Taken together, these observations suggest that ultrafine carbon-cored particles, perhaps including those present in vehicle exhaust, can make respiratory damage from commonly encountered bacterial endotoxins even worse. **–Victoria McGovern**

Top to bottom: Associated Press; Inoue et al.