Swimming in Allergens?

Pool Use and Asthma

Atopic asthma (inflammation of the airways caused by exposure to airborne allergens) has become increasingly prevalent since the 1960s and is now the most common chronic childhood disease in the United States and many other industrialized countries. The cause for the rise is unclear, though many hypotheses have been put forth. Now researchers provide new findings that further support one proposed reason—increased use of indoor chlorinated swimming pools by children [***EHP* 114:1567–1573; Bernard et al.**].

The researchers studied 341 children aged 10–13 years who had attended, at varying rates, three indoor pools in Brussels, Belgium. Ambient levels of a highly reactive chlorine by-product, trichloramine, ranged from 0.25 to 0.54 mg/m^3 at these pools. Trichloramine is created when chlorine reacts with organic matter such as sweat or urine. The researchers administered various tests to the participants, including a questionnaire about health history and pool attendance, an exercise-induced bronchoconstriction test, and a measurement of total serum and aeroallergen-specific IgE (a mediator of atopic asthma).

Forty children had asthma, as indicated by previous doctor diagnosis or the exercise-induced bronchoconstriction test. Cumulative time spent at swimming pools emerged as one of the most consistent predictors of asthma, just after family history of asthma or hay fever and atopy (a genetic tendency toward developing IgE-mediated allergies).

Pooling data. Regular exposure to chlorine at indoor pools may contribute to atopic asthma.

Time spent at pools was associated with increased incidence of asthma only in children with elevated serum IgE. All the effects were dose-related and most strongly linked to pool attendance before the age of about 7 years, suggesting that attendance at indoor chlorinated pools, especially by young children, interacts with atopic status to promote the development of childhood atopic asthma.

The scientists suggest a possible mechanism—that chlorine by-products such as trichloramine disrupt the protective epithelial barriers of the respiratory tract, allowing allergens to enter the lungs. This idea is supported by earlier findings from the same team that children who used an indoor pool showed increased levels of blood markers that indicated damage to these protective membranes, indeed after as little as 1 hour of exposure.

The findings suggest that regular attendance at pools, especially during early life, can promote the development of atopic asthma. Given that atopic asthma is the form of the disease that appears largely responsible for the childhood asthma epidemic and is a chronic disease that greatly affects quality of life, the study points to the need for preventive measures, the authors state. One option is that children younger than 7, especially those with allergies, could avoid strongly chlorinated swimming pools, as identified by a strong smell of chlorine at the surface of the water (for outdoor pools) or inside the enclosure (for indoor pools). **–Angela Spivey**

This Little PBDE Went to Market

Estimating Intake from Grocery Store Foods

High concentrations of polybrominated diphenyl ethers (PBDEs) found in the U.S. population are a cause for concern because of these compounds' similarity to polychlorinated biphenyls. Unlike the latter, which have recently decreased in blood levels, PBDEs have increased substantially in the last two to three decades. A new U.S. "market basket" survey measuring values of PBDEs in grocery store foods shows which chemicals within this class are taken in by eating and adds to a growing body of evidence that food is only part of how humans are exposed to these chemicals [***EHP* 114:1515–1520; Schecter et al.**]. This article is also the first to estimate U.S. PBDE intake via food from infancy to old age.

Chemicals in the case. New findings show how PBDEs in dairy products, meats, and other foods contribute to levels in Americans' blood.

PBDEs are flame retardants applied to fabrics, incorporated into plastics and electronics, and mixed into the foam cushioning used in furniture. The behavior of PBDE congeners can differ due to variable physical, chemical, and biological properties. Though human health effects are not yet well understood, PBDEs' reach in animal studies includes reproductive and developmental toxicity, endocrine disruption, cancer, and central nervous system effects. High levels of PBDEs have been found in human milk, blood, and adipose tissue, as well as in food. U.S. blood and breast milk samples have shown levels 10 to 20 times higher than similar samples from Europeans.

The team used high-resolution mass spectrometry to measure 13 different PBDE congeners in samples of 62 basic foods including fresh and processed meats, fish, milk products, and eggs. The foods analyzed were purchased at three large national chain supermarkets in Dallas in 2003 and 2004.

Of the 13 congeners measured, only about half were found as major contaminants of the food sampled, a finding that parallels earlier observations of the relative prevalence of various congeners in human blood. Although levels of PBDEs varied greatly even within samples of the same type of food, some trends were clear: fish had the most PBDE contamination by weight, followed by meats and dairy foods. But when relative consumption of these foods by Americans was taken into account, meat contributed the most PBDEs to the diet of Americans beyond weaning. (Nursing infants' intake of PBDEs is primarily via breast milk.)

The analysis showed that U.S. foods are generally more contaminated by PBDEs than foods in Japan or Spain, as reported in earlier surveys. But these differences still are not enough to explain the much larger blood and milk burdens observed in Americans. The authors suggest, as others have earlier, that additional routes of exposure, such as house dust inhalation and ingestion, also play important roles in PBDE exposure among Americans. **–Victoria McGovern**

Faulty Folic Acid Assumptions

Prenatal Supplements Not Always a Good Idea

Dietary folic acid supplementation in women of childbearing age has been a major public health success story, reducing the incidence of neural tube defects (NTDs) by an estimated 50–70%. The CDC currently recommends that all women of childbearing age eat a diet high in folic acid or take a daily multivitamin with 0.4 mg of folic acid each day, with higher intake from before conception through the first three months of pregnancy. In light of a new analysis of NTDs and folate pathway genes, however, that blanket recommendation may need to be fine-tuned [***EHP* 114:1547–1552; Boyles et al.**].

If these results are confirmed, it appears there may be a subgroup of women in whom folic acid supplementation is actually positively associated with the formation of NTDs in their offspring—a startling finding the authors acknowledge to be counterintuitive. Counterintuitive or not, the study may have uncovered individuals susceptible to adverse outcomes stemming from overactivity in the folate metabolism pathway during a critical stage of embryonic neurodevelopment.

The researchers analyzed the genomes of 304 families where at least one individual had an NTD. They focused their analysis on 28 single-nucleotide polymorphisms (SNPs) in 11 genes known to be involved in the folate metabolism pathway, and stratified the genomic results by potential gene–gene interactions and by whether the mothers had taken folate-containing nutritional supplements prior to conception.

The results showed that certain SNPs in the betaine-homocysteine methyltransferase (*BHMT*) gene were significantly associated with NTDs, and that the significance was strongest with mothers who took folate supplements before conception. The group also found an associative gene–gene interaction: significance increased in the *BHMT* rs3733890 SNP when the data were stratified by preferential transmission of one particular *MTHFR* allele from parent to offspring. However, *MTHFR*, the most widely studied gene implicated in NTD research, was not found to be a significant single risk factor.

The authors speculate that the stratification method they employed may have inadvertently grouped families by one or more unidentified cofactors correlated with folate supplementation, or that the *BHMT* polymorphism may create a genetic variant that promotes overactivity of the folate metabolic cycle, given the high folate levels achieved during supplementation. This overactivity may inappropriately silence growth factors necessary for proper neural tube closure. Whether this potentially important polymorphic anomaly is grounded in analytic methodology or in actual biology, further research is clearly indicated.

NTDs are known to be complex multifactorial disorders arising from an array of genetic and environmental interactions. In their various physiologic manifestations, they are either profoundly disabling or fatal. Although the preventive effect of folic acid supplementation is undeniable, its protective mechanism of action is still poorly understood. And if the results of this study are replicated and confirmed, the public health directives concerning folic acid supplementation may need to be revised. **–Ernie Hood**

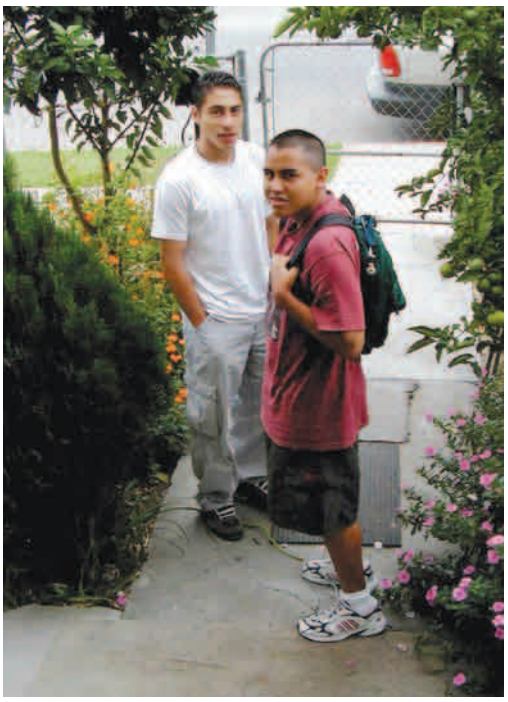
A Backpack's Worth of Data

Elevated Teen Cancer Risks Linked to Air Pollution

It is difficult to assess the cancer risks associated with exposures to air pollutants because much of the health focus has been on the major outdoor pollutants; far less is known about exposures inside homes and buildings, where pollutants may be far more concentrated. Better assessments would measure personal exposures to inhaled pollutants, but such measurements are both costly and challenging to collect. Now a research team has developed an effective method to monitor personal exposures to air pollution with the goal of estimating cancer risks for indoor and outdoor exposures in urban areas [***EHP* 114:1558–1566; Sax et al.**].

The team recruited 87 high school students from Los Angeles and New York City. Three types of measurements were obtained: indoor home samples, outdoor home samples, and personal exposure samples. To obtain personal exposure samples, each teenager wore a regular backpack modified to carry sampling equipment and various types of samplers for aldehydes, particles, and volatile organic compounds (VOCs). This enabled sampling of air wherever the teenager spent time over a 48-hour period, providing an integrated measurement of the air exposures from all indoor and outdoor environments. The personal, ambient, and modeled concentrations were used together with EPA data and other toxicological information to determine excess cancer risks associated with the exposure levels.

In both cities, median cancer risks from personal VOC exposures were much greater than from ambient exposures. Of the VOCs measured, formaldehyde carried the greatest cancer risk (more than 1 in 1 million, based on current EPA data), despite the decline of indoor levels since the banning of formaldehyde foam insulation. Levels of 1,4-dichlorobenzene also posed a substantial risk, though the carcinogenicity of the compound is uncertain, and the risk applied only to teenagers who had sources of this compound (e.g., toilet bowl deodorizers) in their homes. Benzene, the only known human carcinogen in the group of VOCs, posed the greatest risk from outdoor sources. Automobile exhaust and tobacco smoke are two major sources of this pollutant.



Sonja Sax

It goes where they go. A new backpack monitor records teens' personal exposures to pollutants.

This study is unique in its focus on teenage groups in two of the nation's largest and most polluted cities. The two cities have different climates and different housing and commuting options, all factors that can influence exposures to both outdoor and indoor pollutants. Surprisingly, however, the two cities differed little in terms of overall cancer risks. In both cities, indoor exposures were a large determinant of cancer risks.

It is unclear whether the results of this study can be generalized to teens living in other urban areas. Large cancer risks were associated with exposures to many of the VOCs studied, but the toxicological data used to determine these risks have substantial uncertainty. Still, the findings provide valuable assessments of the risks associated with inhaling a suite of pollutants, both gaseous and particle-bound, based on personal exposure measurements that can be used to improve modeled estimates. **–M. Nathaniel Mead**