

Better than Eating Worms? Children's Dietary Exposure to OP Pesticides

Widespread agricultural use of organophosphate (OP) pesticides frequently leads to low-level exposures in adults and children who eat conventionally grown foods. The frequently used one-time measurement of OP metabolites reveals only short-term exposures, thereby providing little evidence on long-term low-level exposures. A new article presents longitudinal evidence that foods grown in conventional fashion—that is, with the use of pesticides—may be a predominant source of exposure in children [*EHP* 116:537–542; Lu et al.].

The article sprang from the Children's Pesticide Exposure Study, which focused on two groups of children (3–11 years of age) in the Seattle and Atlanta areas from July 2003 to May 2004. For the present study, the researchers examined 23 children in Seattle (19 of whom completed the study) who normally consumed conventional diets. Parents tracked food consumption during the study and collected urine samples twice daily.

During interventions over the course of the study, the children's conventional diets were replaced with organic diets and the differences in urinary metabolites of OP pesticides were measured. The organic diets substituted conventionally produced grain, fruit, juice, and vegetables with those produced without pesticides; meat and dairy products rarely contain OP pesticides and were not substituted. During the summer and fall 2003 intervention periods (15 and 12 days, respectively),



For some families, food is likely the primary source of pesticide exposure

the children consumed their regular diets from days 1 to 3 and organic diets from days 4 to 8. After day 8, the children resumed their regular diets. There was no organic diet intervention during the winter and spring 2004 sampling periods.

Urine samples were analyzed for metabolites of malathion, chlorpyrifos, diazinon, coumaphos, and methyl pirimiphos; only chlorpyrifos and malathion metabolites were detected frequently enough for statistical analysis. These metabolite levels fell to nearly or fully nondetectable levels within days of the children beginning an organic diet intervention and rose when the children returned to conventional diets.

Given that OP pesticides were reported by parents not to have been used in the home and that urinary metabolites were clearly affected by diet, the researchers conclude that conventionally produced foods were

the primary source of OP pesticide exposure for the children in this study. They also attribute higher dietary exposures to imported produce eaten in the winter and spring when domestic produce is not available. This finding is supported by a 2006 Environmental Protection Agency report showing that OP residues on imported produce have risen since 1996 even as residues on domestically grown produce have fallen.

The authors caution, however, that their findings do not promote limiting fresh produce or eating only organic items, as it is unknown whether the observed exposures are harmful. Additionally, the study group did not represent the general population. However, the findings do provide a basis for more accurate assessment of exposure and associated efforts to determine the effects of OP pesticides on children's health.

—Julia R. Barrett

Arsenic and Erectile Dysfunction Drinking Contaminated Well Water Increases Risk

Age is the most common risk factor for erectile dysfunction (ED), the consistent or recurrent inability to attain and/or maintain a penile erection sufficient for sexual performance. The correlation between age and ED is attributed to declines in testosterone levels; growing evidence links the condition to cardiovascular disease (CVD) as well. Now researchers from Taiwan have found a direct correlation between ED, the decline of testosterone, and exposure to arsenic via well water—a connection of potential concern for the millions of men worldwide who drink groundwater contaminated with naturally occurring arsenic [*EHP* 116:532–536; Hsieh et al.].

Besides its association with ED, CVD has also been linked to chronic arsenic exposure, perhaps by reducing the synthesis of nitric oxide (NO), which is involved in the control of smooth muscle in blood vessels. In the penis, NO activates cyclic guanosine monophosphate, which dilates blood vessels, allowing the penis to become engorged with blood. Testosterone can regulate activity of the enzyme nitric oxide synthase, which creates NO.

The researchers measured free testosterone levels in the blood of 129 men with ED and 48 without. The average age of the study

participants was about 67 years. Sixty-six of the participants were from an arsenic-endemic area in northeast Taiwan where residents have used contaminated artesian well water for more than 50 years. Arsenic exposure was determined by analysis of participants' well water.

As arsenic exposure of participants increased, so did the risk of ED. The prevalence of ED was 83.3% among men from the arsenic-endemic area compared with 66.7% among men outside this area. Moreover, as the arsenic exposure of the participants increased, their testosterone levels decreased.

The risk of carotid atherosclerosis increased with increasing levels of exposure, but only in men who drank well water containing arsenic concentrations higher than 50 ppb. These men also had a significantly higher risk of ED than men who drank water with arsenic concentrations below 50 ppb, even after adjustments for testosterone levels. Other risk factors for ED did not affect the associations.

According to the authors, arsenic exposure appears to increase ED risk by decreasing testosterone levels. However, they speculate that other factors are at work, as decreases in testosterone did not account for all the ED found in men with high arsenic exposures. The presence of oxygen free radicals can inhibit the synthesis of NO and impair blood vessel function. Therefore, the researchers suggest that oxidative stress from high arsenic exposure may also increase ED risk. —Kris Freeman

Cooking with Wood May Fuel Low Birth Weight

Kitchen Smoke Puts Babies at Risk

The etiology of low birth weight (LBW; defined as weighing less than 2.5 kg at birth) is complex, with demographic, nutritional, reproductive, and socioeconomic factors each potentially playing a role. Inhaled tobacco smoke is the leading cause of LBW in industrialized countries, and inhaled smoke from the world's most widely used cooking fuel, wood, can impair fetal growth much the same way. A team of researchers therefore launched a population-based study to examine the risk of LBW specifically in relation to use of wood fuel during pregnancy [EHP 116:543–549; Siddiqui et al.]. They found that maternal exposure to pollutants from wood smoke increases the risk of LBW, which is linked with myriad health problems including nutritional deficiencies, impaired psychomotor development, and chronic disease.

Tobacco smoke and wood smoke work in two ways to thwart fetal development. One occurs when carbon monoxide combines with hemoglobin to cross the placenta. This causes hypoxia, or a decreased oxygen supply to tissue, which limits the ability of the placenta to transfer nutrients to the fetus. The other occurs when inhaled particulate matter from smoke impairs fetal growth by damaging cells through oxidative stress.

The team of U.S.- and Pakistan-based researchers studied births in the latter country, where more than half the population cooks with wood and the 19% LBW rate is among the world's highest. The researchers studied 634 women who gave birth from 2000 through 2002 in the poor, semirural community of Rehri Goth. Interviewers collected data on the mothers' cooking habits and family demographics. The researchers also obtained pregnancy and delivery data from the

mothers' and infants' medical records.

Women who used wood fuel during pregnancy had a significantly higher risk of delivering LBW babies than those who cooked with natural gas—23% versus 15%. More time spent cooking was linked with increased LBW risk in wood users but not natural gas users. Wood users were poorer than users of natural gas; more of them lived in crowded, run-down houses; and they were more likely to be illiterate. Wood users also tended to weigh less than natural gas users.

Although such socioeconomic factors may play a greater role in birth outcomes, cooking fuel is one factor that is relatively amenable to change. The authors now propose studies of the health impact of smoke-free stoves. The World Health Organization has predicted that if all Pakistani households cooking with wood converted to cleaner fuels, the incidence of LBW would fall from the current rate of 19% to just below the 15% target rate set by the organization. —Cynthia Washam



Wood is the cooking fuel of choice for 53% of Pakistani households

Bleached, But Not by the Sun

Sunscreen Linked to Coral Damage

Warm, shallow, sun-drenched seas sparkling with brilliantly colored fish and coral species—we've all seen dazzling images of tropical reefs. Coral reefs are among the most biologically productive and diverse ecosystems in the world, providing food protein for half a billion people. But tropical reefs have begun dying from bleaching, with the frequency and spatial extent of such bleaching increasing dramatically over the past 20 years. Now a study finds that chemical compounds in sunscreen products can cause abrupt and complete bleaching of hard corals, even at extremely low concentrations [EHP 116:441–447; Danovaro et al.].

Zooxanthellae, symbiotic algae that live in healthy coral tissue, provide nutrients to corals through photosynthesis. The algae also help make the spectacular colors for which corals are known. The corals lose their color when zooxanthellae die or leave the reef; the protective skeletons of the corals are thus exposed, and the corals die. Rising seawater temperatures, bacterial and viral diseases, ultraviolet light or other radiation, and pollution have been blamed for coral bleaching.

Scientists at the Polytechnic University of the Marche Region in Ancona,

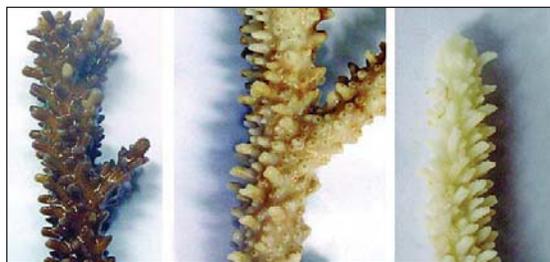
Italy, studied the effects of sunscreen exposure on samples from tropical reefs. The researchers collected branches of hard coral from sites in the Red Sea, the Caribbean Sea, the Indian Ocean off Thailand, and the Pacific Ocean near Indonesia.

Coral branches were immersed in bags of virus-free seawater supplemented with various quantities of sunscreen, then incubated *in situ*. These samples were compared with controls also incubated *in situ* in virus-free seawater.

The researchers found that among the several brands of sunscreen tested, four commonly found ingredients—paraben, cinnamate, benzophenone, and camphor derivatives—can stimulate dormant viral infections in zooxanthellae. The sunscreen chemicals caused viruses within zooxanthellae to replicate until their algal hosts exploded, spilling viruses into the surrounding seawater, which could then spread infection to nearby coral communities.

Coral bleaching occurred, often within a few hours, but always within 4 days, at sunscreen quantities as small as 10 $\mu\text{L/L}$. Controls remained healthy.

The researchers estimate that approximately 10% of the world's coral reefs are potentially threatened by sunscreen that washes off swimmers in reef waters. The study suggests that, as tourism continues to increase in tropical reef areas, the impact of sunscreens on coral bleaching could rise significantly in the future. —John Tibbetts



Higher temperatures worsen bleaching. (left to right) Control sample of *Acropora divaricata*; exposed sample incubated at 28°C; exposed sample incubated at 30°C.