

Rice Is a Significant Source of Methylmercury

Research in China Assesses Exposures

Human activities such as mining, smelting, and coal combustion disperse mercury that can be methylated by bacteria to produce methylmercury, a potent neurotoxicant. Methylating bacteria thrive in aquatic sediments rich in organic matter, and methylmercury biomagnification eventually leads to heavy contamination of top predators, including fish consumed by humans. Although fish and seafood are the most common dietary sources of methylmercury worldwide, new research from China demonstrates that rice, a staple food for billions, can be a primary source of methylmercury in areas where there is substantial inorganic mercury pollution, with calculated exposure exceeding current tolerable daily intakes [*EHP* 118(9):1183–1188; Zhang et al.].

The research was conducted in four regions in Guizhou province, an area of inland China with rich deposits of cinnabar (a mercury ore). Mercury mining and smelting have led to heavy pollution in Wanshan, while zinc smelting and coal combustion, which also release mercury, are the main contributors in Weining and Qingzhen, respectively. The fourth region, Leigong, is a remote nature reserve selected to represent an area with no sources of direct mercury contamination.

Methylmercury and total mercury exposure through drinking water, diet, and respiration were assessed for adults in the four regions. Previous sampling provided data for air, water, fish, meat, and poultry, while agricultural products (rice, corn, and vegetables), drinking water from Wanshan and Leigong, and total gaseous mercury in Wanshan were newly evaluated in this study. These data were collectively used to calculate probable daily intakes for the general adult population.

In all regions rice, vegetables, and meat (not including poultry and fish) accounted for 89–97% of total mercury exposure, whereas rice consumption accounted for 94–96% of methylmercury exposure. Fish contributed little; most of the fish consumed here are farmed species that grow rapidly and eat a diet that precludes significant methylmercury bioaccumulation.

Picture of Better Health

Prioritizing Air Pollution Control in China

China's Yangtze River Delta is one of the most heavily polluted and densely populated areas of the world, with some districts of Shanghai exceeding a population density of 40,000 people/km². Annual average concentrations of fine particulate matter (PM_{2.5}), microscopic airborne particles that cause heart and lung damage, are estimated to vary from 14 µg/m³ in the cleanest areas to 133 µg/m³ in the dirtiest. By contrast, most major cities in the United States have annual average PM_{2.5} levels between 7 and 20 µg/m³. Yet a new study suggests regions as highly polluted as the Yangtze River Delta can clear the air and in the process save thousands of lives by adopting common pollution-control technologies [*EHP* 118(9):1204–1210; Zhou et al.].

The study is believed to be one of the first risk assessments of a developing country using the Community Multiscale Air Quality (CMAQ) modeling system. Although CMAQ is widely used in the United States, most developing countries lack the exhaustive data on emissions, pollutant monitoring, and weather conditions needed to make accurate predictions. The research team capitalized on emissions data from China that NASA estimated using satellite measurements during a 2006 study of intercontinental pollution drift.

The researchers examined emissions of sulfur dioxide (SO₂), nitrogen oxides (NO_x), volatile organic compounds (VOCs), and PM_{2.5}, focusing on the effect of these pollutants on PM_{2.5} and ozone



concentrations. Ozone is a gas formed when NO_x and VOCs in the atmosphere react in sunlight. PM_{2.5} is linked to heart and respiratory disease, with resulting effects on premature death, and ozone has been associated with respiratory outcomes and premature death. They considered 10 control scenarios among major pollution sources—industry, power plants, motor vehicles, and domestic life—and focused on the health benefits per ton-of-emission reduction across scenarios.

Computer modeling predicted the greatest decline in mortality would come from reducing SO₂ emissions from power plants and PM_{2.5} emissions from industry, with each technology preventing approximately 1,200–24,000 deaths per year among the more than 80 million people living in the delta. This is based on the significant reduction in SO₂ emissions from coal-powered plants with the use of fluidized gas desulfurization, as well as the large health benefit per ton of avoided emissions of primary PM_{2.5} from industry. Measures to reduce NO_x and VOCs, including tighter vehicle-emissions standards, would have less impact on public health.

The authors point out that understanding sectoral differences in the ways emission-control strategies affect exposures and health risks can help guide strategies that are both economically and environmentally optimal. They conclude that their findings provide the basis for prioritizing pollution-control strategies in the Yangtze River Delta and provide a template for comparable analyses elsewhere.

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BPA and Insulin Resistance

Evidence of Effects in Dams and Offspring

Recent population studies have associated increased exposure to persistent organic pollutants with an elevated risk of insulin resistance and thus a greater likelihood of developing type 2 diabetes and heart disease. Among these chemicals is bisphenol A (BPA), a pervasive endocrine-disrupting compound used to make polycarbonate plastic and epoxy resins. Now researchers report evidence that exposure to environmentally relevant doses of BPA during pregnancy may alter insulin sensitivity and glucose homeostasis in mice, with potential disease-related consequences for both the mother and her male offspring [*EHP* 118(9):1243–1250; Alonso-Magdalena et al.].

The study evaluated the effects of two different doses of BPA (10 or 100 µg/kg/d) administered to pregnant mice during days 9–16 of gestation. Glucose metabolism experiments were performed on the mice during pregnancy and subsequently on their offspring.

BPA exposure aggravated the insulin resistance that occurs during pregnancy, and four months postpartum, BPA-treated mice weighed more and had more severe insulin resistance than untreated females. The BPA-treated mice also showed elevated plasma levels of insulin,

leptin, triglycerides, and glycerol (a breakdown product of triglycerides), as well as molecular changes indicating reduced insulin sensitivity in skeletal muscle and liver.

Given that levels of the hormone leptin are normally increased during pregnancy, the authors propose that future research should seek to determine whether BPA directly regulates leptin release from fatty tissue or whether the observed hyperleptinemia is a consequence of the altered metabolic state of these animals.

Previously the same research team had shown a relationship between BPA exposure and glucose intolerance and insulin resistance in adult male mice. In the present study, they further observed that, at 6 months of age, male offspring exposed to BPA *in utero* had reduced glucose tolerance, increased insulin resistance, and altered blood parameters compared with offspring of untreated mothers. Moreover, studies of the male offspring's pancreases showed altered calcium signaling and insulin secretion.

The authors conclude that BPA exposure during pregnancy can alter the mother's glucose metabolism during pregnancy and later in life, and may contribute to metabolic disorders relevant to glucose homeostasis in the male offspring. The findings also suggest that BPA exposure should be further examined as a risk factor for diabetes.

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Traffic Trouble

Study Links Diabetes to Vehicular Pollution

There is a well-documented relationship between exposure to particulate matter (PM) in ambient air pollution and risk of developing cardiovascular disease. Subclinical or low-grade inflammation, believed to serve as an intermediary between air pollution and cardiovascular/metabolic health risks, is associated with impaired glucose metabolism, but few studies to date have examined the relationship between air pollution and diabetes. For the first time, a prospective study provides evidence linking exposure to traffic-related air pollution with an increase in the risk of developing type 2 diabetes in women [*EHP* 118(9):1273–1279; Krämer et al.].

In the current study, researchers investigated the relationship between air pollution exposure and new-onset incident type 2 diabetes using information from the prospective Study on the Influence of Air Pollution on Lung, Inflammation, and Aging (SALIA). The authors also assessed whether baseline inflammation was associated with pollution exposure.

The SALIA cohort is composed of 1,775 women aged 54–55 years without diabetes at enrollment. The women lived in the highly industrialized Ruhr district of Germany or in rural, nonindustrial towns nearby. Using data obtained from cross-sectional surveys administered in 1985–1994 and a follow-up interview in 2006, the investigators analyzed the incidence of type 2 diabetes over 1990–2006. They also collected information on symptoms and diagnoses of respiratory disease, home and occupational exposure to air pollution, smoking status, and socioeconomic status. They took initial height and weight measurements, and collected nonfasting blood serum samples to measure complement factor C3c, a blood protein that served as a marker for subclinical inflammation. They estimated exposure to nitrogen dioxide (NO₂) and PM, the major components of traffic emissions, by applying land-use regression models.

Between 1990 and 2006, 187 participants (10.5%) were diagnosed with type 2 diabetes. Exposure to traffic-related air pollution and higher levels of C3c in the blood at baseline were both associated with increased diabetes risk. Living within 100 m of a busy roadway was associated with more than double the risk of diabetes for women with a lower education level compared with women in the same group who did not live near a busy roadway; women with higher education who lived near busy roads had no altered risk.

Overall, the researchers observed significant associations with PM and NO₂ exposure. The slightly stronger associations of risk with NO₂ exposure than with PM exposure further support a link between traffic-related air pollution exposure and diabetes, since most sources of NO₂ are traffic-related.

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