

Smoking Gain?

Secondhand Smoke Exposure Influences Body Weight, Lipid Profiles in Offspring

An estimated 780,000 U.S. women continue smoking throughout pregnancy each year despite warnings about the elevated risk of birth defects in the children of female smokers. A new mouse study provides experimental evidence that exposure to secondhand cigarette smoke during pregnancy may lead to weight gain in offspring as well as changes in lipid profiles that may increase the chances of cardiovascular disease later in life [*EHP* 117:1042–1049; Ng et al.].

Oxidants in cigarette smoke have previously been shown to promote local and systemic inflammation and increase the risk of cardiovascular disease in both smokers and those exposed to secondhand smoke. Lipid oxidation in particular has been associated with cardiovascular diseases. Women have a higher risk of dying from cardiovascular disease than men and are more likely to die following a heart attack.

The current study may shed light on this observed sex-specific difference. Female pups of mice that were exposed to cigarette smoke for 4 hours a day, 5 days a week, throughout pregnancy grew up to have a higher body weight than their unexposed peers and had significant

increases in plasma high-density lipoprotein (HDL), low-density lipoprotein (LDL), and total protein. However, these differences were not observed when the adult female offspring were fed a high-fat diet instead of a regular diet. On the other hand, smoke-exposed male pups gained more weight and displayed altered lipid profiles compared with their sex-matched, unexposed counterparts when they were fed a high-fat diet but showed little evidence of an effect of smoke exposure when fed a normal diet.

Although maternal exposure to cigarette smoke appeared to influence weight gain and lipid profiles in their offspring, lipid parameters in the dams themselves showed little change in response to smoke exposure. Additional work is necessary to understand why lipoprotein levels—which reflect cholesterol metabolism—are altered in the offspring in response to cigarette smoke exposure during pregnancy.

Abnormal body weight and dyslipidemia (abnormal plasma lipoprotein levels) are among the best-established risk factors for cardiovascular diseases. Although the new study does not investigate the mechanisms of the observed changes, it does suggest that prevention programs that emphasize avoidance of cigarette smoke during pregnancy could reap long-range health benefits for the newborn.

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Mother Load

Arsenic May Contribute to Gestational Diabetes

Chronic exposure to arsenic—usually via drinking water contaminated with inorganic arsenic—has been associated with an increased risk of type 2 diabetes mellitus in countries around the world. New research shows that arsenic exposure may be an environmental risk factor for gestational diabetes as well [*EHP* 117:1059–1064; Ettinger et al.].

Arsenic may promote type 2 diabetes by increasing insulin resistance (inability to utilize insulin at the cellular level) and impairing insulin production. Insulin resistance is also a central feature of gestational diabetes, a potential complication during pregnancy that can lead to a 30–60% increased risk for the mother of developing lifelong diabetes, as well as impaired glucose tolerance, adverse birth outcomes, and obesity in her child.

The study was conducted near the Tar Creek Superfund site in Ottawa County, Oklahoma, whose residents include many Native Americans, a population already at elevated risk for type 2 diabetes. The area, once active in lead and zinc mining, has an above-average poverty rate compared with the rest of Oklahoma and the nation. Mine waste contaminated with assorted metals is still present and has been used to build roads, playgrounds, driveways, and house foundations. Moreover, 25% of drinking water samples tested in the area have naturally occurring arsenic levels exceeding the Environmental Protection Agency maximum contaminant level of 10 µg/L.



Millions of people worldwide may be exposed to naturally occurring arsenic in drinking water.

Total arsenic concentrations were measured in blood and hair samples collected at delivery from 532 women; blood was available from all women and hair from a subset of 179. Routine prenatal glucose tolerance tests conducted between weeks 24 and 28 of pregnancy yielded plasma glucose measurements, and questionnaires and medical record review provided data on sociodemographic characteristics, potential sources of arsenic exposure, and pregnancy history.

Blood arsenic concentrations, a measure of biologically active arsenic, were between 0.2 and 24.1 µg/L, whereas hair arsenic concentrations, an indicator of cumulative exposure, were 1.1–724.4 ng/g. Blood glucose levels ranged from 40 to 284 mg/dL. At a cut-off value of > 140 mg/dL, 12% of the women were identified as having impaired glucose tolerance; a cut-off value of 130 mg/dL yielded a prevalence of more than 20%. A statistically significant relationship existed between each increasing quartile of blood arsenic exposure and impaired glucose tolerance after controlling for health and demographic factors. Depending on the glucose test cut-off value, women in the highest quartile of arsenic exposure were 2.4–2.8 times more likely to have impaired glucose tolerance than women in the lowest quartile of exposure.

These results suggest that chronic arsenic exposure may increase the risk of developing gestational diabetes. A better understanding of this and other factors through further research may identify modifiable risk factors this condition.

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A Lucrative Investment Controlling Lead Paint Yields Big Dividends

Fewer children today have the high blood lead levels seen in kids a few decades ago, when gasoline and paint still came fully leaded. But a surprising number of children still have blood lead levels that may place them at risk for a variety of cognitive, emotional, and behavioral problems. The good news is that boosting current efforts to protect U.S. children from one major source of lead—the house paint used prior to a 1978 ban, which still appears in many homes—may pay for itself many times over [EHP 117:1162–1167; Gould].

According to National Health and Nutritional Examination Survey data from 2003 to 2006, an estimated 25% of the 28 million U.S. children aged 6 years and younger have blood lead levels between 2 and 10 µg/dL, a range in which persistent cognitive damage is known to occur. Another 200,000 children are estimated to have levels over 10 µg/dL.

Using data from published studies, the author performed a cost–benefit analysis of the effects of controlling children’s exposure to lead paint. She calculated that controlling lead paint in the approximately 1 million worst-case housing units would cost between \$1.2 billion and \$11 billion, depending on many factors including local costs of lead abatement. But the benefits to be derived from controlling lead hazards could range from \$181 billion to \$269 billion. For example, abatement could save \$11–53 billion in immediate medical treatment and \$30–146 million in special education costs. Reducing the incidence of attention deficit/hyperactivity disorder (ADHD) related to lead paint exposure might save \$267 million; and because both ADHD and lead exposure have been associated with criminal behavior, crime-related costs could shrink by \$1.7 billion with efforts to eliminate and contain lead-laden paint.

The author concludes that every dollar spent to limit U.S. children’s exposure to lead paint—such as through paint stripping, replacement, and covering with a special encapsulant coating—could net \$17–221. By comparison, vaccination

against the most common childhood diseases is estimated to save \$5.30–16.50 for every dollar spent on immunizations.

The author noted that the cost savings from better lead mitigation could be even higher than estimated in the current study. For one thing, the calculations of potential benefit pertain only to children under age 6. Yet getting rid of lead paint would benefit other segments of the population as well. Also, the analysis excluded many potential costs of lead exposure, including future health care expenses and the indirect costs of criminal activity.

U.S. public health and housing policies have been slow to address the lingering problems related to lead paint, the author asserts. Given the huge projected savings and income in terms of health care, crime prevention, education, lifetime earnings, and tax revenues, she writes, the time for proactive and universal lead control has never been better.

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It can cost a homeowner more than \$200,000 to clean up lead contamination after uncontrolled power sanding, compared with about \$1,200 to incorporate lead-safe work practices into repainting.

Rainy Day Reaction Human West Nile Viruses Cases Respond to Weather Patterns

Piecemeal evidence suggests weather may have played a role in the rapid spread of West Nile virus (WNV) across the United States and into Canada and Central America following its detection in New York City in 1999. A team of U.S. and Canadian researchers has looked more comprehensively at the evidence by analyzing a spectrum of weather factors for 17 climatically diverse states, and found several significant links with the incidence of human WNV cases [EHP 117:1049–1052; Soverow et al.]. The analysis was based on 16,298 WNV cases reported to the Centers for Disease Control and Prevention from 2001 to 2005, as well as year-round temperature, precipitation, and dew point data from 351 weather stations in close proximity to the infected people.

A 12°F increase in maximum daily temperature was associated with a 45–72% increase in WNV case reports within a 1-month period. Precipitation was also associated with WNV, which increased

29–66% in association with a single-day rainfall of at least 50 mm within 3 weeks of diagnosis. Smaller amounts of precipitation were associated with smaller increases in WNV cases, consistent with a dose–response effect. Increases in cumulative weekly precipitation and mean weekly dew point temperature (a measure of relative humidity) were also associated with an increase in WNV cases.

The findings, which hold up across season and location, generally mesh with what is known about the biology of WNV, humans, mosquito vectors, and bird reservoir hosts. The authors write that additional research will be needed to address some limitations of their work—notably gaps in data from a number of geographic regions and the influence of localized interactions of factors such as bird populations, vegetation, mosquito control efforts, and acquired immunity in both humans and animals. If these weather–disease links are confirmed, and if climate changes in North America unfold as predicted with increases in temperature and precipitation, public health officials may be better able to prevent or mitigate outbreaks in the future.

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