# National study finds strong link between diabetes and air pollution

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A national epidemiologic study finds a strong, consistent correlation between adult diabetes and particulate air pollution that persists after adjustment for other risk factors like obesity and ethnicity, report researchers from Children's Hospital Boston. The relationship was seen even at exposure levels below the current EPA safety limit.

The report, published in the October issue of Diabetes *Care*, is among the first large-scale population-based studies to link diabetes prevalence with air pollution. It is consistent with prior laboratory studies finding an increase in insulin resistance, a precursor to diabetes, in obese mice exposed to particulates, and an increase in markers of inflammation (which may contribute to insulin resistance) in both the mice and obese diabetic patients after particulate exposure.

Like the laboratory studies, the current study focused on fine particulates of 0.1-2.5 nanometers in size (known as PM2.5), a main component of haze, smoke and motor vehicle exhaust. The investigators, led by John Pearson and John Brownstein, PhD, of the Children's Hospital Informatics Program, obtained county-by-county data on PM2.5 pollution from the Environmental Protection Agency (EPA), covering every county in the contiguous United States for 2004 and 2005.

They then combined the EPA data with data from the Centers for Disease Control (CDC) and the U.S. Census to ascertain the prevalence of adult diabetes and to adjust for known diabetes risk factors, including obesity, exercise, geographic latitude, ethnicity and population density (a

measure of urbanization).

"We wanted to do everything possible to reduce confounding and ensure the validity of our findings," says Pearson, the study's first author.

In all analyses, there was a strong and consistent association between diabetes prevalence and PM2.5 concentrations. For every 10 μg/m3 increase in PM2.5 exposure, there was a 1 percent increase in diabetes prevalence. This finding was seen in both 2004 and 2005, and remained consistent and significant when differing estimates of PM2.5 exposure were used.

"We didn't have data on individual exposure, so we can't prove causality, and we can't know exactly the mechanism of these peoples' diabetes," acknowledges Brownstein. "But pollution came across as a significant predictor in all our models."

Even among counties falling within EPA limits for exposure, those with highest versus the lowest levels of PM2.5 pollution had a more than 20 percent increase in diabetes prevalence, which remained after controlling for diabetes risk factors.

"From a policy perspective, the findings suggest that the current EPA limits on exposure may not be adequate to prevent negative public health outcomes from particulate matter exposure," Brownstein says.

"Many environmental factors may contribute to the epidemic of diabetes in the United States and worldwide," notes Allison Goldfine, MD, head of clinical research at the Joslin Diabetes Center and a coauthor on the study. "While a lot of attention has correctly been attributed to caloric excess and sedentary behaviors, additional factors may provide novel approaches to diabetes prevention."

Based on their findings, the researchers call for more study of environmental factors in diabetes, including basic research on the inflammatory mechanisms in diabetes and the role of PM2.5.

"We would like to access better individual-level data on diabetes and exposure," adds Brownstein. "We also have an interest in investigating this finding internationally where standards may be less stringent."

Provided by Children's Hospital Boston