

Dr. Erica Bell
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27.5.2009

Dear Dr. Bell,

Thank you for the Community Event reminder to be held on World Environment Day, June 5.

I was just browsing the internet and noticed your study on diabetes and mention of how, ***"...The current provision of health services in Tasmania needed to change in order to meet the requirements of a community where there is increasing demand for the prevention and management of chronic diseases..."***

As a former rural health worker in this state I could not agree more, especially chronic diseases like asthma and other respiratory diseases, cancer, heart attack, blood clotting, etc. known to be caused or aggravated by particle pollution on a grand scale in Tasmania from planned burning.

In regards to your Diabetes Risk Factor research, I am wondering if you were aware of the following research that was being undertaken at the time, and if not, do the results have any bearing on your findings or future outcomes please?

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Subject: [chescience] air pollution and diabetes

Abstract of study attached. Consider how these changes are linked not only to diabetes but also to cardiovascular disease, metabolic syndrome, Alzheimer's disease, dementia.

Ted Schettler

Ohio State-led research

Breathing dirty air may increase risk of diabetes

Monday, January 19, 2009 12:10 AM

By [Misti Crane](#)

THE COLUMBUS DISPATCH

A new study led by Ohio State University researchers suggests a connection between air pollution and diabetes.

The study, published online today in the journal *Circulation*, reports that dirty air makes fat mice more likely to get diabetes and raises questions about how closely pollution and Type 2 diabetes are linked in humans.

The study found that air pollution exaggerates insulin resistance and fat inflammation in overfed mice exposed to either filtered air or dirty air for six months.

All the mice were fed a fast-food diet before the experiment to make them obese, said lead researcher Dr. Sanjay Rajagopalan, director of vascular medicine at Ohio State University Medical Center. The National Institutes of Health paid for the research.

The amount of soot in the air breathed by the mice in the pollution group was comparable to the air you'd breathe sitting behind an idling truck, he said.

"We found a very, very strong exaggeration of diabetes" in the pollution group, Rajagopalan said.

Much remains unanswered, but the explanation could lie inside the walls of blood vessels, where inflammation can occur. And inflammation has been linked to environmental factors, including pollution.

"This inflammation in the vessel wall, it's not unique to atherosclerosis. It's in fact the molecular underpinning of obesity and diabetes," he said.

"The mechanisms that lead to heart disease and metabolic diseases such as diabetes are in fact very similar."

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Aruni Bhatnagar, a professor of medicine in the division of cardiology at the University of Louisville, said the study is thought-provoking and should prompt more research.

"The human story is quite complicated, and nothing like this has been done in humans," said Bhatnagar, who wrote an editorial to accompany the research.

But pollution has been linked to heart disease, and diabetes, obesity and heart disease have almost identical risk factors, making a connection seem plausible, he said.

"Almost everything that causes heart disease also causes diabetes. It is not altogether surprising that, given the exposure, there appears to be a link."

About 24 million Americans have diabetes.

Although the researchers caution that more study is needed, they say their work might add more weight to efforts to toughen clean-air standards.

"I think the evidence is surely coming to a point where these standards may need to be revised again," Rajagopalan said.

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Submitted on June 13, 2008

Accepted on October 24, 2008

Ambient Air Pollution Exaggerates Adipose Inflammation and Insulin Resistance in a Mouse Model of Diet-Induced Obesity

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From the Davis Heart and Lung Research Institute (Q.S., P.Y., J.A.D., T.K., M.B.M., Y.C., S.P., S.R.), Division of Environmental Health Sciences (Q.S.), Division of Biostatistics (B.L.), Department of Molecular and Cellular Biochemistry (M.C.O.), and Division of Cardiothoracic Surgery (S.D.M.-B.), Colleges of Medicine and Public Health, Ohio State University, Columbus; Life Sciences Institute (C.N.L.) and Department of Internal Medicine (R.D.B.), University of Michigan, Ann Arbor; and Department of Environmental Medicine (L.C.C.), New York University School of Medicine, New York.

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Background—There is a strong link between urbanization and type 2 diabetes mellitus. Although a multitude of mechanisms have been proposed, there are no studies evaluating the impact of ambient air pollutants and the propensity to develop type 2 diabetes mellitus. We hypothesized that exposure to ambient fine particulate matter (<2.5 μm ; $\text{PM}_{2.5}$) exaggerates diet-induced insulin resistance, adipose inflammation, and visceral adiposity.

Methods and Results—Male C57BL/6 mice were fed high-fat chow for 10 weeks and randomly assigned to concentrated ambient $\text{PM}_{2.5}$ or filtered air (n=14 per group) for 24 weeks. $\text{PM}_{2.5}$ -exposed C57BL/6 mice exhibited marked whole-body insulin resistance, systemic inflammation, and an increase in visceral adiposity. $\text{PM}_{2.5}$ exposure induced signaling abnormalities characteristic of insulin resistance, including decreased Akt and endothelial nitric oxide synthase phosphorylation in the endothelium and increased protein kinase C expression. These abnormalities were associated with abnormalities in vascular relaxation to insulin and acetylcholine. $\text{PM}_{2.5}$ increased adipose tissue macrophages (F4/80⁺ cells) in visceral fat expressing higher levels of tumor necrosis factor- α /interleukin-6 and lower interleukin-10/*N*-acetyl-galactosamine specific lectin 1. To test the impact of $\text{PM}_{2.5}$ in eliciting direct monocyte infiltration into fat, we rendered FVBN mice expressing yellow fluorescent protein (YFP) under control of a monocyte-specific promoter (*c-fms*, *c-fms*^{YFP}) diabetic over 10 weeks and then exposed these mice to $\text{PM}_{2.5}$ or saline intratracheally. $\text{PM}_{2.5}$ induced YFP cell accumulation in visceral fat and potentiated YFP cell adhesion in the microcirculation.

Conclusion— $\text{PM}_{2.5}$ exposure exaggerates insulin resistance and visceral inflammation/adiposity. These findings provide a new link between air pollution and type 2 diabetes mellitus.

Thank you and all the best with the policy and practices forum.

Yours faithfully,

Clive M. Stott

Clean air is one of our most precious resources, essential for our survival and quality of life.
<http://www.cleanairtas.com>