

## Air pollution linked to cardiovascular risk indices in healthy young adults

August 15 2007

Researchers in Taiwan have demonstrated for the first time that urban air pollution simultaneously affects key indicators of cardiovascular risk in young adults: inflammation, oxidative stress, coagulation and autonomic dysfunction.

The study, which appeared in the second issue for August of the American Journal of Respiratory and Critical Care Medicine, published by the American Thoracic Society, investigated the effect of common urban air pollutants on biological markers for inflammation, oxidative stress, coagulation and autonomic dysfunction in 76 healthy Taiwanese college students.

The researchers collected blood samples and performed electrocardiograms on each subject approximately every 30 days for the months of April, May and June in either 2004 or 2005. They then correlated the sample dates and time with monitoring data from a fixedsite air monitoring station on the students' campus. The concentrations of common urban air pollutants were averaged over 24, 48 and 72 hours.

They found significant increases in all indices of cardiovascular risk were associated with increased exposure to common pollutants. "This study provides evidence that urban air pollution is associated with systemic inflammation/oxidative stress, impairment of the fibrinogenic system, activation of blood coagulation and alterations in the autonomic nervous system in young, healthy humans," wrote the study's lead author Chang-Chuan Chan, Sc.D., of National Taiwan University's College of



Public Health.

"Most pollution literature has shown affects in elderly people, and although there have been experiments in young subjects, epidemiological research has not found such significant effects in young people as with these students in Taiwan," said Benoit Nemery, M.D., Ph.D., professor in the division of pneumonology at the Catholic University of Leuvin in Belgium, who was not involved in the research.

Specifically, the investigators found that increases in high-sensitivity C-reactive protein (hs-CRP) (an indicator of risk for a cardiovascular event), 8-hydroxy-2'-deoxyguanosine (8-OHdG) (a marker of oxidative stress), fibrinogen (a coagulation factor), plasminogen activator fibrinogen inhibitor-1 (PAI-1) (a marker of inflammation) and decreases in heart rate variability (HRV) (a predictor of increased cardiovascular risk) were associated with increases in particulate matter, sulfate, nitrate, and ozone when they analyzed pollutants singly, and that increases in 8-OHdG, fibrinogen, and PAI-1 and decreases HRV were correlated to increased ozone and sulfate levels over three day averages in multipollutant models.

The precise biological mechanisms involved were not able to be determined with this study's design. "Further studies with more detailed measurements of cardiovascular endpoints over time are still needed to elucidate the time sequence of pollution effects on cardiovascular endpoints in humans," wrote Dr. Chang-Chuan Chan.

"Many questions remain to be answered," wrote Joel Kaufman, M.D., M.P. H., in the accompanying editorial, "[But] these questions should by no means slow the important efforts to reduce exposures and benefit global public health."

Source: American Thoracic Society



Citation: Air pollution linked to cardiovascular risk indices in healthy young adults (2007, August 15) retrieved 4 May 2024 from <u>https://medicalxpress.com/news/2007-08-air-pollution-linked-cardiovascular-indices.html</u>

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