# Long-term exposure to fine particles of traffic pollution associated with increased risk of heart disease

Results from large cohort study presented at EuroPRevent 2013

**Topics:** Cardiovascular Disease Prevention - Risk Assessment and Management
**Date:** 18 Apr 2013

Fine particle matter and traffic noise are believed to act through similar biologic pathways, thereby increasing cardiovascular risk; they both cause an imbalance in the autonomic nervous system, which feeds into the complex mechanisms regulating blood pressure, blood lipids, glucose level, clotting and viscosity.

*Rome, 18 April 2013.* The association between road traffic and heart disease has been suggested in several studies. In 2012 a large prospective cohort study from Denmark showed that traffic noise was significantly associated with risk of heart attack - for every 10 decibel increase in noise exposure (either at the time of the attack or over the five years preceding it) there was a 12% increased risk.(1)

Now, a new study presented at the EuroPRevent 2013 congress in Rome shows that long-term exposure to fine particle matter (PM) air pollution in part derived from traffic pollution is also associated with atherosclerosis independent of traffic noise.(2)

Details of the study were described by Dr Hagen Kälsch from West-German Heart Center in Essen, Germany, who explained that the study was designed to establish where responsibility for the increased heart risks associated with traffic actually lay - with noise or particle pollution, or both.

The study was based on data from the German Heinz Nixdorf Recall Study, a population-based cohort of 4814 participants with a mean age of 60 years. Their proximity to roads with high traffic volume was calculated with official street maps, their long-term exposure to particle pollutants assessed with a chemistry transport model, and road traffic noise recorded by validated tests. The participants' level of atherosclerosis was evaluated by measurement of vascular vessel calcification in the thoracic aorta, a common marker of subclinical atherosclerosis (known as TAC), by computed tomography imaging.

Results showed that in the 4238 subjects included in the study small particulate matter (designated as PM2.5) and proximity to major roads were both associated with an increasing level of aortic calcification - for every increase in particle volume up to 2.4 micrometers (PM2.5) the degree of calcification increased by 20.7% and for every 100 metre proximity to heavy traffic by 10%. The study also found a borderline

increase in TAC for night time noise (of 3.2% per 5 decibels). The associations of PM2.5 and road traffic noise were not modified by each other.

Commenting on the results, Dr Kälsch confirms that long-term exposure to fine PM air pollution and to road traffic noise are both independently associated with TAC as a measure of subclinical atherosclerosis.

"These two major types of traffic emissions help explain the observed associations between living close to high traffic and subclinical atherosclerosis," he says. "The considerable size of the associations underscores the importance of long-term exposure to air pollution and road traffic noise as risk factors for atherosclerosis."

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TAC, alongside coronary artery calcification (CAC), is a reliable marker of subclinical atherosclerosis. While sharing cardiovascular risk factors with coronary atherosclerosis, TAC like TAC has been shown to be independently related to the incidence of cardiovascular events.

A further study reported at this congress from French investigators found that all the main air pollutants (carbon monoxide (CO), nitrogen dioxide ($NO_2$), sulphur dioxide ($SO_2$), and particulate matter measured as PM10 or PM2.5, but with the exception of ozone ($O_3$)) were significantly associated with an increased risk of myocardial infarction.(3)

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**Notes to editor**

**EuroPrevent**
EuroPRevent 2013, the world's leading congress in preventive cardiology, is organised by the European Association for Cardiovascular Prevention and Rehabilitation (EACPR), an association of the European Society of Cardiology, from 18-20 April 2013.

**About the European Society of Cardiology (ESC)**
The European Society of Cardiology (ESC) represents 80,000 cardiology professionals across Europe and the Mediterranean. Its mission is to reduce the burden of cardiovascular disease in Europe.

**References**

1. Sørensen M, Andersen ZJ, Nordsborg RB et al. Road traffic noise and incident myocardial infarction: a prospective cohort study. PLOS One 2012; doi: 10.1371/journal.pone.0039283.
2. Kaelsch H, Hennig F, Moebus S, et al. Is urban particulate air pollution or road traffic noise responsible for the association of traffic proximity with subclinical atherosclerosis? Results from the Heinz Nixdorf Recall Study. Presented at EuroPRevent 2013 Poster presentation P307.
3. Mustafic H, Jabre P, Caussin C, et al. Main air pollutants and myocardial infarction: a systematic review and meta-analysis, Presented at EuroPRevent 2013 Poster presentation P89.

**http://www.escardio.org/about/press/press-releases/pr-13/Pages/exposure-fine-particles-traffic-pollution-increased-risk-heart-disease.aspx**