

## Publication

## Ambient air pollution and the progression of atherosclerosis in adults

**JournalArticle (Originalarbeit in einer wissenschaftlichen Zeitschrift)****ID** 1195023**Author(s)** Künzli, N.; Jerrett, M.; Garcia-Esteban R.; Basagana, X.; Beckermann, B.; Gilliland, F.; Medina, M.; Peters, J.; Hodis, H. N.; Mack, W. J.**Author(s) at UniBasel** Künzli, Nino ;**Year** 2010**Title** Ambient air pollution and the progression of atherosclerosis in adults**Journal** PLoS ONE**Volume** 5**Number** 2**Pages / Article-Number** e9096**Mesh terms** Adult; Air Pollutants, poisoning; Air Pollution, analysis; Air Pollution, Indoor, analysis; Animals; Atherosclerosis, etiology; Carotid Arteries, pathology; Disease Progression; Environmental Exposure, analysis; Humans; Los Angeles; Particle Size; Particulate Matter, poisoning; Randomized Controlled Trials as Topic; Risk Assessment; Tunica Intima, pathology; Tunica Media, pathology

BACKGROUND: Cross-sectional studies suggest an association between exposure to ambient air pollution and atherosclerosis. We investigated the association between outdoor air quality and progression of subclinical atherosclerosis (common carotid artery intima-media thickness, CIMT). METHODOLOGY/PRINCIPAL FINDINGS: We examined data from five double-blind randomized trials that assessed effects of various treatments on the change in CIMT. The trials were conducted in the Los Angeles area. Spatial models and land-use data were used to estimate the home outdoor mean concentration of particulate matter up to 2.5 micrometer in diameter (PM<sub>2.5</sub>), and to classify residence by proximity to traffic-related pollution (within 100 m of highways). PM<sub>2.5</sub> and traffic proximity were positively associated with CIMT progression. Adjusted coefficients were larger than crude associations, not sensitive to modelling specifications, and statistically significant for highway proximity while of borderline significance for PM<sub>2.5</sub> ( $P = 0.08$ ). Annual CIMT progression among those living within 100 m of a highway was accelerated (5.5 micrometers/yr [95%CI: 0.13-10.79;  $p = 0.04$ ]) or more than twice the population mean progression. For PM<sub>2.5</sub>, coefficients were positive as well, reaching statistical significance in the socially disadvantaged; in subjects reporting lipid lowering treatment at baseline; among participants receiving on-trial treatments; and among the pool of four out of the five trials. CONCLUSION: Consistent with cross-sectional findings and animal studies, this is the first study to report an association between exposure to air pollution and the progression of atherosclerosis—indicated with CIMT change—in humans. Ostensibly, our results suggest that air pollution may contribute to the acceleration of cardiovascular disease development—the main causes of morbidity and mortality in many countries. However, the heterogeneity of the volunteering populations across the five trials the limited sample size within trials and other relevant subgroups, and the fact that some key findings reached statistical significance in subgroups rather than the sample precludes generalizations to the general population

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