Long-Term Personal Exposure to Traffic and Coronary Calcification

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Background Epidemiological evidence suggests that long-term exposure to particulate matter (PM) air pollution accelerates atherogenesis and increases cardiopulmonary mortality [1;2]. We examined the relationship between long-term personal exposure to traffic, a major source of urban air pollution, and the degree of coronary artery calcification (CAC), controlling for major risk factors for arteriosclerosis and background PM air pollution.
Methods We used baseline data from the German Heinz Nixdorf Recall study, an ongoing population-based, prospective cohort study of 4814 inhabitants of the Rhein-Ruhr area [3]. Participants´ addresses were geocoded with a geographic information system (GIS). To assess personal long-term traffic exposure, the major source of ambient ultrafine particles (UFP, < 100 nm), distances between residence and major roads, namely autobahn (mean daily vehicle count >30,000) and federal highways (mean daily vehicle count >10,000 to ≤30,000) were calculated with the GIS. Distance to major roads is a useful surrogate for UFP-exposure, because UFP exhibit a steep decline in number concentration with increasing distance from major motorways [4]. Annual mean values for $PM_{2.5}$ mass concentration on a spatial scale of 1 km were estimated with the EURAD-model for the year 2002 (midpoint of baseline examination) and assigned to each address [5]. The main outcome was the degree of coronary artery calcification (CAC), assessed quantitatively by electron-beam computer tomography [6]. CAC is a highly reproducible and accurate measure of clinical and subclinical coronary atherosclerosis that correlates well with cardiovascular risk factors and clinical events. We evaluated the association between the logarithm of proximity to major roads and log(CAC+1) with linear regression, controlling for background $PM_{2.5}$ and individual level risk factors for coronary arteriosclerosis (age, sex, educational attainment, smoking status, number of cigarettes per day, environmental tobacco smoke exposure, hip-to-waist ratio, hypertension, systolic blood pressure, diabetes mellitus, HBA_{1c}, LDL, HDL).
Result We performed the analysis on 4464 out of 4814 participants for which information on all explanatory variables were available. 677 of 4464 participants (15.2%) lived within 200 m of a major road. Exposure values for background $PM_{2.5}$ ranged from 19.8 to 26.8 µg/m^3 (mean 22.8). Mean CAC-values were strongly dependent on age, sex and smoking status. Reducing the distance to a major road by half is associated with an increase in CAC by 1.13 (95% CI 1.01-1.26) in women and 1.09 (95% CI 0.97-1.23) in men in the crude model and by 1.06 (95% CI 0.96-1.17) in women and 1.07 (95% CI 0.96-1.20) in men in the adjusted model. In a subgroup analysis of 4167 participants without clinically manifest coronary artery disease (no history of myocardial infarction, bypass surgery, angioplasty or implantation of a coronary stent), the observed effects were similar (women: 1.09; 0.99-1.20 and men: 1.05; 0.94-1.17). The effect is comparable in size to the estimated increase in CAC due to six months of aging (1.06; 95% CI 1.05-1.07).
Discussion We could observe a substantial effect of long-term residential traffic exposure on CAC. This finding is in agreement with the results from a Dutch study, where cardiopulmonary mortality was shown to be associated with an indicator variable for residential traffic exposure [2]. Pathogenetic factors within the complex mixture of traffic emissions, including UFP, chemical composition of particles and noise, could be responsible for this effect.
Conclusions This study provides epidemiological evidence that long-term exposure to traffic-related emissions is associated with coronary artery calcification. More research is needed to identify the factors responsible for the adverse health effects of urban air pollution.

References

[1] Künzli N, Jerrett M, Mack WJ, Beckerman B, LaBree L, Gilliland F, Thomas D, Peters J, Hodis HN. Ambient air pollution and atherosclerosis in Los Angeles. Environ Health Perspect 2005;113(2):201-206.
[2] Hoek G, Brunekreef B, Goldbohm S, Fischer P, van den Brandt PA. Association between mortality and indicators of traffic-related air pollution in the Netherlands: a cohort study. Lancet 2002;360(9341):1203-1209.
[3] Schmermund A, Möhlenkamp S, Stang A, Grönemeyer D, Seibel R, Hirche H, Mann K, Siffert W, Lauterbach K, Siegrist S, Jöckel KH, Erbel R. Assessment of clinically silent atherosclerotic disease and established and novel risk factors for predicting myocardial infarction and cardiac death in healthy middle-aged subjects: Rationale and design of the Heinz Nixdorf RECALL Study. American Heart Journal 2002;144(2):212-218.
[4] Zhu Y, Hinds WC, Kim S, Sioutas C. Concentration and size distribution of ultrafine particles near a major highway. J Air Waste Manag Assoc 2002;52(9):1032-1042.
[5] Memmesheimer M, Friese E, Ebel A, Jakobs HJ, Feldmann H, Kessler C, Piekorz G. Long-term simulations of particulate matter in Europe on different scales using sequential nesting of a regional model. International Journal of Environment and Pollution 2004;22(1-2):108-132.
[6] Schmermund A, Möhlenkamp S, Berenbein S, Pump H, Moebus S, Roggenbuck U, Stang A, Seibel R, Grönemeyer D, Jöckel KH, Erbel R. Population-based assessment of subclinical coronary atherosclerosis using electron-beam computed tomography. Atherosclerosis 2006;185(1):177-182.