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Health Effects of Combined Exposure to Diesel Particles and Traffic Noise - a Methodology for Controlled Chamber Study

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1 Introduction
Exposure to traffic related particulate air pollution has been associated with cardiopulmonary disease and mortality (Dockery, 2009). Exposure to traffic noise has been recognised as an important risk factor for cardiovascular effects (Babisch, 2008). A few studies combine particles and noise exposure when studying observed health effects (Beelen et al. 2009). This is important to address as the similarity of the cardiovascular effects reported by the two distant disciplines, i.e. air pollution and noise, are striking. Mechanisms behind the observed health effects are still not fully understood.

The overall aim of this study was to develop and apply a methodology for controlled laboratory studies to examine the effects on humans in response to combined exposure to particles and noise. This methodology is based on controlled, well characterized chamber exposure and medical examination together with determination of physiological responses, cognitive performance, and self-ratings of sensory symptoms, fatigue, stress and annoyance.

2 Materials/Methods
Eighteen healthy volunteers (9 men and 9 women) were exposed to four different conditions: 1) Reference exposure (R): low diesel particle concentration (< 3 µg/m³) and low traffic noise (46 dB(A)), 2) Diesel exposure (D): high diesel particle concentration (~300 µg/m³) and low traffic noise, 3) Noise exposure (N): low diesel particle concentration and high traffic noise (75 dB(A)), 4) Diesel and noise exposure (DN): high diesel particle concentration and high traffic noise.

Outside the laboratory a light duty diesel vehicle (Volkswagen, 1998) running in idle mode was used for the generation of the particles. Swedish MK1 diesel fuel with sulphur content less than 10 ppm was used. Diesel exhaust was diluted in a specifically designed two stage system. Dilution was controlled to supply desired concentration of particles to a 22 m³ stainless steel chamber for different exposure scenarios. Detailed characterisations of both particle and gas phase were carried out. Particles were characterised by means of mass concentration (TEOM, model 1400a, R&P Inc.), number concentration and size distribution (SMPS 3934 TSI Inc. USA), effective density (DMA-APM system), particulate PAH and organic and elemental carbon analysis, elemental analysis (with Particle Induced X-ray Emission), and electron microscopy images. Concentrations of the following gases were monitored on-line: CO, CO₂, NO, NO₂. Gas phase concentrations of VOC, PAH, benzene, 1,3-butadiene, formaldehyde and acetaldehyde were determined via off lines methods.

The chamber acoustics were characterized by means of the reverberation time. Traffic noise recorded at a street crossing was played from
two loudspeakers reproducing each channel in a stereo recording. The noise had a continuous, natural change of the noise level.

Test subjects were exposed for three hours to each exposure scenario with at least one week interval between each scenario. At each session three test subjects stayed in the chamber. Prior to participation in the study test subjects underwent audiometry, spirometry, heart and lung auscultation, and skin prick test for atopy. Medical and work history was also registered according to a specific protocol.

For each exposure scenario the following measurements/tests were conducted five times (1 measurement/test before, 3 during and 1 after the exposure): a) Time series of ECG for frequency analysis of heart rate variability; b) Beat to beat blood pressure variations (Finometer®PRO, FMS, Finapres Medical Systems BV); c) Peak expiratory flow measurement for lung function assessment; d) Salivary cortisol; e) Self-ratings of sensory symptoms, fatigue, stress and annoyance; f) d2 test of attention for assessing cognitive performance.

Before and after each exposure, samples of venous blood, urine, breath condensate and nasal lavage were taken for analysis of markers of oxidative stress and inflammation. Lung function and nasal patency were measured by spirometry and acoustic rhinometry, respectively.

After each exposure endothelial dysfunction was assessed non-invasively using a pulse amplitude tonometry; EndoPAT (Itamar Medical Ltd).

The generalized estimating equations model (GEE) procedure in SPSS, version 18.0, was used to specify a repeated measures model. All outcomes were compared with R exposure.

3 Results
The following exposure characteristics have been measured. The average concentrations for all D and DN exposures: PM1 (particulate matter with the aerodynamic diameter below 1µm) mass concentration 276 (± 46) µg/m³, particle number concentration (size range 15 to 600 nm) 388000 (± 69000) particles/cm³, geometric mean diameter 89 (± 8) nm, GSD 1.98 (± 0.08); NO 9.8 (± 1.8) ppm, NO₂ 1.4 (± 0.5) ppm, CO 7 (± 1.5) ppm, CO₂ 1945 (± 218) ppm, and temperature 22.7 (± 0.3) deg. C. The average concentrations for all N and R exposures: PM1 mass concentration 2 (± 2) µg/m³, particle number concentration (size range 15 to 600 nm) 15 (± 15) particles/cm³, NO and NO₂ below detection limit, CO 0.3 (± 0.3) ppm, CO₂ 950 (± 83) ppm, and temperature 22.7 (± 0.3) deg. C.

On the basis of lung function assessment and self-reported symptoms the following effects have been seen. Decreased peak expiratory flow during exposure to diesel particles was observed i.e. during exposure D (group change) -0.7 l/min (p=0.04) and during exposure DN -0.9 l/min (p=0.02). Increased eye irritation was observed during exposure D and DN. During exposure D, in comparison to reference exposure, additional 13.2 units of irritation on a scale from 1 to 100 (p=0.03) and during exposure DN additional 12 units (p=0.05). Fatigue increased during exposure N (2.3 units, p=0.05) but during the combined exposure DN the increase in fatigue was not statistically significant (2.5 units, p=0.16). No effects have been seen in spirometry and rhinometry. Effects on cardiovascular system, analysis of biomarkers for oxidative stress and inflammation, salivary cortisol and cognitive performance are being evaluated.

4 Conclusions
Developed methodology enables quantitative determination of physiological responses and identification of relevant biomarkers in trial to understand underlying mechanisms for observed health effects during combined exposure to diesel particles and traffic noise.

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5 References
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