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LUND UNIVERSITY

PO Box 117
221 00 Lund
+46 46-222 00 00

A methodology for assessment of combined effects of particles and noise on humans during controlled chamber exposure

Wierzbicka A.¹, Albin M.², Andersson U.B.K.², Assarsson E.², Axmon A.², Barregård L.⁵, Berglund M.³, Bohgard M.¹, Broberg K.², Brunskog J.⁴, Gudmundsson A.¹, Gunnskog A.², Hagerman I.³, Jönsson B.², Kåredal M.², Nilsson P.¹, Österberg K.², Pagels J.¹, Poulsen T.⁴, Rissler J.¹, Stockfelt L.⁵, and Sällsten G.⁵

¹Division of Ergonomics and Aerosol Technology, Lund University, Sweden

²Division of Occupational and Environmental Medicine, Lund University, Sweden

³Department of Cardiology, Karolinska University Hospital, Huddinge, Sweden

⁴Division of Acoustic Technology Technical University of Denmark, Denmark

⁵Division of Occupational and Environmental Medicine, University of Gothenburg, Sweden

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Epidemiological evidence shows that particulate air pollution as well as noise are important risk factors for cardiopulmonary disease and mortality. In urban environment these two stressors coincide due to the same source – traffic.

Thus the overall aim of this study was to develop and apply a methodology for controlled laboratory studies to examine 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.

Eighteen healthy volunteers (9 men and 9 women) were exposed to four different conditions: 1) Zero exposure: low particle concentration (less than 3 µg/m³ and 100 particles/cm³) and low traffic noise (46 dB(A)), 2) High particle concentration (300 µg/m³, 400 000 particles/cm³.) and low traffic noise, 3) Low particle concentration and high traffic noise (75 dB(A)), 4) High particle concentration and high traffic noise.

Outside the laboratory a diesel Volkswagen Passat (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 defined and traffic noise was played via a surround loudspeakers system.

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 reactive hyperemia procedure; EndoPAT (Itamar Medical Ltd).

Preliminary results show that quantitative measures of physiological effects can be obtained with this methodology and that frequency analysis of heart rate variability, blood pressure and endothelial dysfunction assessment can be used as non-invasive markers for cardiovascular effects in these settings.

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