Is there any synergy in the observed health effects due to exposure to particles and noise? Results from DINO study - controlled chamber exposure

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In urban environment exposure to airborne particles and noise coincide due to the same source – traffic. Epidemiological evidence shows that particulate air pollution as well as noise are important risk factors for cardiovascular diseases. A few studies combine particles and noise exposure when studying observed health effects. Mechanisms behind the observed health effects and possible synergistic effects are not fully understood.

The overall aims of the DINO study ("Health effects of combined exposure to **di**esel and **no**ise") was to determine influence of combined exposure to diesel exhaust (DE) particles and traffic noise on measurable human physiological response in order to understand the mechanisms behind observed health effects and to determine if combined exposure to DE and traffic noise can have synergistic effect on human health.

Eighteen test subjects were exposed to four different conditions: 1) Reference exposure (R): filtered air with low particle concentration (~2 μg m^{-3}) and low traffic noise (46 dB(A)), 2) Diesel exposure (D): high particle concentration (~300 μg m^{-3}) and low traffic noise, 3) Noise exposure (N): low particle concentration and high traffic noise (75 dB(A)), 4) Diesel and noise exposure (DN): high particle concentration and high traffic noise. Exposure took place in a 22 m^3 stainless steel chamber, three test subjects stayed in the chamber at each session (Wierzbicka et al., 2015)

The diesel exhaust was generated by an idling Volkswagen Passat (1998) placed outside the laboratory. Diesel exhaust was diluted in a specifically designed two stage system. 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 and electron microscopy images. Concentrations of the following gases were monitored on-line: CO, CO_2, NO, NO_2. Gas phase concentrations of VOC, PAH, benzene, 1.3-butadiene, formaldehyde and acetaldehyde were determined via off lines methods (Wierzbicka et al, 2015).

Peak expiratory flow measurement for lung function assessment were conducted before, during and after the exposure. Before and after each exposure as well as in the morning the day after exposure samples of venous blood were taken for analysis of markers of oxidative stress, inflammation and DNA damage.

Short-term exposure to DE at ~300 μg/m^3 caused temporary decline in peak expiratory flow in healthy subjects and the increase in leukocyte cell counts in peripheral blood indicated a systemic inflammatory responses (Xu et al., 2013). After DE exposure gene expression markers of inflammation, (interleukin-8 and tumor necrosis factor), oxidative stress (heme oxygenase (decycling-1)) and DNA repair (8-oxoguanine DNA glycosylase (OGG1)) were unaltered in peripheral blood mononuclear cells (PBMCs). No significant differences in DNA damage levels, measured by the comet assay, were observed after DE exposure, whereas exposure to high noise levels was associated with significantly increased levels of hOGG1-sensitive sites in PBMCs (Hemmingsen et al, 2015).

In this study, among the analyzed results and presented above, a synergistic effect due to combined exposure to particles and noise was not observed.

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