384. Outdoor air pollution studies

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Late-breaking abstract: Do emissions from animal farms affect the airways of neighboring residents?

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Concerns about public health risks of intensive animal production in The Netherlands continue to rise, in particular related to outbreaks of infectious diseases such as Q-fever. An increased risk of asthma among neighboring residents is a specific concern raised by general practitioners (GPs) in areas with high animal densities. The aim was to study respiratory health effects among individuals living in the vicinity of animal farms.

Electronic medical record data for the year 2009 of all patients of 27 GPs in a region with a high density of animal farms were used. Density of animal farms around the home address was calculated using a Geographic Information System. Associations between farm exposure variables and respiratory diseases were analyzed in 22.406 children (0-17 y) and 70.142 adults (18-70 y), adjusting for age, sex, and household income. During the study period, a Q-fever outbreak occurred

in this region. "Possible Q-fever" was defined as a diagnosis of pneumonia or other infectious disease – the diagnosis code used by GPs for registration of suspected Q-fever.
Proximity to farms and modeled farm-related particulate matter (PM10) exposure were inversely associated with asthma, COPD, upper respiratory tract infections, and hay fever, whereas the same farm exposure variables were positively associated with possible Q-fever (all P<0.05). The presence of goat and sheep within 1 km of the home address was associated with possible Q-fever in adults (goat: OR 1.3; 95%CI 1.1-1.4, sheep: OR 1.3; 95%CI 1.1-1.5).
Both protective and adverse health effects among individuals living near animal farms were observed. These findings should be confirmed with diagnostic measures before firm conclusions can be drawn.

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Late-breaking abstract: Pneumonia hospitalizations and long-term exposure to air pollution: A cohort study

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Background: Long-term exposure to air pollution has been associated with cardiovascular and chronic lung disease, but limited evidence exist on the risk for pneumonia.
Aim: To investigate the effect of long-term exposure to traffic-related air pollution (up to 38 years) in Copenhagen and Aarhus on hospital admissions for pneumonia.
Methods: We followed 57053 participants of Danish Cancer, Diet, and Health cohort, aged 50-65 years at baseline (1993-1997) in Danish hospital discharge register for first hospital admissions for pneumonia between baseline and 2010. The annual nitrogen dioxide (NO_2) levels were estimated at residential address since 1971 as a proxy of exposure to traffic-related air pollution. We modelled the association between mean NO_2 levels and hospitalizations for pneumonia using Cox regression.
Results: During 12.7 years' mean follow-up, 3024 (5.7%) out of 53239 eligible people were admitted to hospital for pneumonia. Mean NO_2 levels were significantly positively associated with risk for first pneumonia hospitalization in the full cohort (hazard ratio and 95% confidence interval per double mean exposure: 1.25; 1.13-1.35); with similar effect for first-ever pneumonia admission in 46462 people without earlier hospitalizations for pneumonia or co-morbid conditions defined by Charlson (1.23; 1.11-1.37), and in 6292 people with history of co-morbid conditions (1.22; 1.02-1.46), wheras enahnced association was observed in 485 people with a history of pneumonia hospitalizations (1.45; 0.85-2.47).
Conclusions: Living in areas with high traffic-related air pollution increases the risk of hospitalization for pneumonia.

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Impact of climate change on ozone induced mortality in Europe

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Background: Ozone is a highly oxidative pollutant, associated with respiratory morbidity and mortality. All else being equal, ground-level ozone will increase as temperatures increase with climate change.
Aim: As a part of the Climate-TRAP project we used emission scenarios, models and epidemiological data to assess ozone-related health impacts under a changing climate.
Methods: European ozone concentrations were modelled at a grid size of 50x50 km using MATCH-RCA3. Projections from two climate models, ECHAM4 and HADLEY, were used, assuming greenhouse gas emission scenarios A2 and A1B. Four periods were compared: the baseline period was defined as 1961–1990, the current situation as 1990–2009, nearer future as 2021–2050 and further future as 2041–2060. The impact on mortality (short-term effect) was calculated for exposures above a daily maximum 8-hour concentrations of 70 μgm-3. We use a European-wide exposure-response function with country-specific baseline mortality.
Results: Comparing the current situation with the baseline period, the largest increase in ozone-associated mortality due to climate change (~4%) occurred in Belgium, Ireland, Netherlands and UK. Comparing the baseline period and the further future, the increase is projected to be biggest in Belgium, France, Spain and Portugal (10–14%) and the effect will be stronger for the A2 scenario. However, in Nordic and Baltic countries there will be a decrease in ozone-related mortality of the same magnitude.
Discussion: The current study suggests that projected effects of climate change on ozone levels could differentially influence mortality and morbidity across Europe.

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Long-term exposure to air pollution and asthma hospitalizations in elderly adults: A cohort study

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Background: Exposure to air pollution in early life contributes to the burden of childhood asthma, but it is not clear whether lifetime exposure to air pollution can lead to asthma onset or progression in adulthood. We studied the effect of exposure to traffic-related air pollution over 35 years on the risk for hospitalization for asthma in elderly.
Methods: We followed 57 053 participants in the Danish Diet, Cancer and Health cohort, aged 50-65 years at baseline (1993-1997), for first hospital admission for asthma until 2006. Annual levels of nitrogen dioxide (NO_2) were estimated at all residential addresses since 1971. We modelled the association between NO_2 and hospitalization for asthma using Cox regression in people with and without previous hospitalizations for asthma, and assessed effect modification by co-morbid conditions.
Findings: During 9.9 years' mean follow-up, 977 of 53 695 eligible people (1.9%) were admitted to hospital for asthma: 821 (1.5%) admissions were among 53 143 people who had not and 176 (31.9%) among 552 people who had been hospitalized for asthma before baseline. NO_2 levels were positively associated with risk for asthma hospitalization in the full cohort (hazard ratio and 95% confidence interval per inter-quartile range, 5.8 $\mu g/m^3$: 1.12; 1.04-1.22), and in people without previous asthma hospitalization (1.10; 1.01-1.20), with the strongest effects for people with a history of asthma hospitalization (1.41; 1.15-2.07). Enhanced associations between NO_2 and admissions for asthma were observed for people with COPD (1.30; 1.07-1.52).
Interpretation: Air pollution is a risk factor for progression and/or onset of asthma in late adulthood.

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Increased plasmatic levels of soluble HLA-G molecules are associated to short-term exposure to fine urban particulate matter

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Diesel particles have been shown to enhance allergic inflammation and immune responses. HLA-G is an atypical HLA class I molecule with immuno-modulatory properties. It is detectable in plasma in a soluble form (sHLA-G) and its production is triggered by several stimuli, including IL-10.
The aim of this study is to investigate the effect of short-term exposure to traffic pollution on plasmatic sHLA-G and IL-10 levels in healthy subjects. We recruited 27 healthy non-smoking policemen exposed to traffic and 17 office workers as controls. Before and after the first weekly shift, plasmatic sHLA-G and IL-10, and lung volumes were measured in each subject. sHLA-G and IL-10 levels were analyzed by ELISA. Individual pollution exposure was estimated by airborne PM2.5 and PM10 concentrations.
Pre-shift sHLA-G levels were similar in both groups and increased after shift in the subjects exposed to traffic (before 10.5±1.6 vs after 29.7±4.5 ng/ml, p<0.0001), but not in controls. After shift IL-10 exhibited an opposite trend in the two groups: no changes were observed in the exposed subjects, whereas IL-10 levels decreased in office workers (before 0.7±0.5 vs after 0.5±0.4 pg/ml, p=0.03). sHLA-G cross-shift changes in exposed policemen were positively correlated with PM2.5 levels (rho=0.51, p<0.05). Lung volumes did not exhibit any change after shift in either groups.
In conclusion, short-term exposure to traffic pollution affects the HLA-G system, irrespective of IL-10 levels. The results suggest that fine PM had a systemic immuno-modulatory effect that occurs without changes in lung function.

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Antioxidant supplementation attenuates changes in innate immunity associated with diesel exhaust (DE) in the lung: A controlled crossover exposure study

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Rationale: Oxidative stress is thought to induce negative health effects associated with exposure to DE and may result from altered innate immunity. Therefore, anti-oxidant supplementation may modify DE-related changes in phagocytosis in airways.

Methods: 12 participants (4 mild asthmatics and 8 healthy controls) completed a double-blinded, randomized, crossover, counter-balanced study of 3 exposure conditions, each separated by a 2-wk washout period: (1) DE (300ug $PM_{2.5}/m^3$ for 2 hours) with anti-oxidant (N-acetylcysteine 600mg 3x/day for 5 days preceding, and on the day of the exposure) ["DEN"], (2) DE with placebo ["DEP"], or (3) filtered air with placebo ["FAP"]. Induced sputum was collected 6 hours after each exposure. Sputum neutrophils and macrophages were analyzed by flow cytometry for changes in innate immune response (phagocytosis). Those with PC20≤8 were defined as "reactive" and those with PC20>8 as "non-reactive".
Results: Phagocytosis in sputum macrophages was greater after DEP than after FAP (mean phagocytosis units = 22.8 and 15.8, respectively) and this difference was attenuated by DEN (mean phagocytosis units = 17.8 units). 6 subjects were methacholine-reactive at baseline; reactivity status did not modify phagocytosis.
Conclusions: DE-associated increase in phagocytosis in human airways appears mitigated by anti-oxidant supplementation (N-acetylcysteine). The suggestion that DE upregulates the innate immune response in sputum macrophages, and that this is mediated by oxidative stress, parallels novel data from our lab regarding changes in airway reactivity to methacholine in response to FAP, DEP, and DEN.

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Outdoor exposure to formaldehyde (CH2O) is associated with an increased risk of hospitalization for respiratory diseases in children

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Background: Children living near wood industries have an increased risk of developing respiratory diseases.
Objectives: To assess if residential outdoor exposure to NO2 and CH2O was associated with the risk of hospitalization for respiratory diseases in children.
Methods: In 2006, all the children (3-14 years) living in the Viadana district (the largest wood manufacturing area in Northern Italy) were surveyed through a parental questionnaire (n=3854) and their home addresses were geocoded. Their history of hospitalization for respiratory diseases (ICD-IX: 460-519) was assessed from January 2007 to December 2009, using discharge records obtained from the local Health Unit. To assess the outdoor exposure to NO2 and CH2O, 63 passive samplers were installed in the area using a Partitioning Around Medoids (PAM) algorithm. Pollutants were monitored twice, both in winter and in summer 2010. Kriging interpolation was used to attribute the average annual concentration of pollutants to each child. Poisson regression models were used to assess the association between the hospitalization rates (HR) and the average concentration of pollutants.
Results: By December 2010, 3798 (98.5%) children had been traced. During the 3 years of follow-up, 121 hospital admissions, caused by respiratory diseases, occurred (annual HR = 10.8/1000/year). The HR for respiratory diseases slightly increased with increasing outdoor exposure to NO2 ($\mu g/m^3$) (RR:1.02; 95%CI: 0.95-1.10), while they were strongly associated to CH2O outdoor concentration ($\mu g/m^3$) (RR:2.41; 95%CI: 1.07-5.43).
Conclusions: Emissions from wood industries apparently have a serious impact on children's health.

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Altered haemodynamic response to norepinephrine following diesel exhaust inhalation

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Background: Exposure to particulate air pollution, of which diesel exhaust (DE) is a major component, increases cardiovascular morbidity and mortality. Although the underlying mechanisms of these effects are not fully understood, there are reports of increased blood pressure and reduced heart rate variability consistent with altered autonomic nervous function following exposure to particulate air pollution.
Objective: We tested the hypothesis that exposure to DE alters the haemodynamic response to systemic infusion of norepinephrine (NE).
Methods: In a randomized double-blind crossover study, 14 healthy non-smoking volunteers were exposed to dilute diesel exhaust or filtered air for one hour during intermittent exercise. Two hours after the exposure, the subjects received intravenous infusions of NE (50 ng/kg/min over 15 mins) and the haemodynamic response was assessed using invasive blood pressure, pulse wave velocity and thoracic bioimpedance.
Results: In comparison to filtered air, cardiac index was reduced (P=0.027) and systemic vascular resistance was increased (P<0.0001) following DE exposure. Despite this, the vasopressor response to infused NE was attenuated: increases in mean arterial pressure and pulse wave velocity were both reduced (P<0.0001 for both) whilst changes in heart rate were similar (P=0.28).
Conclusions: Our results show unexpected haemodynamic responses to NE after exposure to dilute DE, with both attenuated vasopressor responses and an inhibitory effect on cardiac index. These novel findings suggest that DE inhalation alters the cardiovascular response to adregenic stimulation, which could represent an effect on the autonomic nervous system.