ORIGINAL ARTICLE

Acute nasal pro-inflammatory response to air pollution depends on characteristics other than particle mass concentration or oxidative potential: the RAPTES project

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ABSTRACT

Objectives To investigate which air pollution characteristics are associated with biomarkers for acute nasal airway inflammation in healthy subjects. We hypothesised that associations would be strongest for oxidative potential (OP) of particles.

Methods 31 volunteers were exposed to ambient air pollution at five sites in The Netherlands: two traffic sites, an underground train station, a farm and an urban background site. Each subject visited at least three sites between March and October 2009 and was exposed for 5 h per visit including exercise for 20 min every hour (h). Air pollution measurements during this 5-h-period included particulate matter (PM) mass concentration, elemental composition, elemental and organic carbon (OC), particle number concentration, OP, endotoxins, O_3 and NO_2. Pro-inflammatory biomarkers were measured before, 2 and 18 h postexposure, including cytokine IL-6 and IL-8, protein and lactoferrin in nasal lavage (NAL) as well as IL-6 in blood. One- and two-pollutant mixed models were used to analyse associations between exposure and changes in biomarkers.

Results In two-pollutant models, cytokines in NAL were positively associated with OC, endotoxin and NO_2; protein was associated with NO_2; and lactoferrin was associated with all PM characteristics that were high at the underground site. In blood, associations with OC and endotoxin were negative.

Conclusions We observed no consistent effects in two-pollutant models for PM mass concentration and OP. Instead, we found consistent associations with nasal inflammatory markers for other PM characteristics, specifically OC, endotoxin and NO_2.

What this paper adds

- It is not well known which air pollution characteristics are responsible for adverse health effects.
- This paper shows that acute air pollution exposure is associated with increased expression of biomarkers of nasal inflammation in healthy subjects.
- In two-pollutant models we found consistent associations for organic carbon, endotoxin and NO_2, but not for particulate matter (PM) mass concentration or oxidative potential of particles.
- Our results suggest that air quality management should focus on specific air pollution compounds rather than on PM mass concentration which is currently used for air quality legislation.

INTRODUCTION

It is not well known which particulate matter (PM) characteristics are responsible for adverse health effects.[1 2] Numerous characteristics, like particle size, number concentration, transition metals, organics, sulfates and nitrates, and biological components such as endotoxins have been proposed. Thus far, the available in vitro, in vivo and epidemiological data provide insufficient information to enable further quantification on the importance of individual PM characteristics.[1 3]

Currently, attention is being given to the capacity of particles to exert oxidative stress as it is thought to be an important mechanism underlying PM-induced adverse health effects.[4] Oxidative stress results when the balance between the generation of reactive oxygen species, or free radicals, overrides the cells' antioxidant defences. High levels of oxidative stress induce inflammatory responses via a cascade of events including activation of various transcription factors and stimulation of cytokine production.[4 5] Since the oxidative potential (OP) of particles reflects several of the physical and chemical properties that contribute to PM toxicity, it may be a biologically meaningful, integrative measure to predict PM-related health effects.[6 7]

Being the entry port for inhaled PM, the nasal cavity is a primary target for effects from ambient particles. In the nasopharyngeal cavity, deposition rates are the highest for the coarse size fraction (~70%) and those particles in the ultrafine size fraction that are about 1 nm in diameter (~90%).[8] Deposited particles can trigger nasal cells to release pro-inflammatory mediators (eg, cytokines, reactive oxygen species) or cause cell damage to the

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