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Mini Review

In An Elderly Panel Cohort Associations of Primary and Secondary Organic Aerosols with Airway and Systemic Inflammation

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Abstract

To safeguard vulnerable populations, exposure-response data on the constituents of particulate air pollution are required. Particulate matter with a diameter of less than 2.5 millimetres (PM2.5) can produce reactive oxygen species which can lead to oxidative stress. Primary organics from combustion sources and secondary organics from volatile organic compounds that have been photo chemically oxidized are two examples. We looked at how primary and secondary organic particle components, size fractions, and the potential of particles to induce cellular production of reactive oxygen species differed in airway and systemic inflammatory responses.

INTRODUCTION

Reactive oxygen species derived from particle components may induce oxidative stress, according to experimental evidence. When antioxidant defences are overwhelmed, this oxidative stress may be followed by airway and systemic inflammation (Shinyashiki M, 2009). However, there is a lack of supporting evidence in human populations. Responses of oxidative stress to PM2 Direct effects on the surface of the particle or soluble compounds like ultrafine-particle-rich reactive organic chemicals can lead to exposures. Organic PM2.5 components like oxygenated polycyclic aromatic hydrocarbons are examples of reactive chemicals (Mills NL, 2009). The ability of PM to stimulate cellular production of reactive oxygen species and the potential of PM to directly produce reactive oxygen species are two potential mechanisms of PM-induced oxidative stress. Because a significant portion of the mass of PM2.5 and PM10 is biologically inactive, while a variable and frequently insignificant portion has the potential to cause oxidative stress, the oxidant potential of PM can be independent of mass (Robinson AL, 2007). As a result, epidemiological analyses of exposure-response relations based solely on mass may obscure underlying effects. Inorganic substances make up a significant portion of the mass of PM2.5. On the other hand, on the basis of a growing body of research4,5, we hypothesized that the organic portion of PM2.5 is to blame for the cardiovascular effects. There are many organic components in aerosols that have the potential to be oxidants (Delfino RJ, 2009). These organic components vary a lot by location, time of day, and season. Primarily, the effects of semi volatile organic compounds and combustion-related primary organic aerosols may differ from those of photochemical related secondary organic aerosols. When combustion emissions are cooled to ambient temperature, primary organic aerosols already exist in the particle phase. When volatile reactive organic precursors are oxidized to form low-volatility products that condense to produce aerosols, secondary organic aerosols typically result (Polidori A, 2007). Precursors come from both natural and man-made sources. Older primary semi volatile organic compounds are also used in part to make secondary organic aerosols. Few studies have examined the relationship between variations in this multipollutant characteristic of PM2.5 and inflammatory-mediated respiratory or cardiovascular diseases. The cardiovascular effects of PM have been proposed to be explained by airway deposition of proinflammatory particles leading to a progression from airway to systemic inflammation (Arhami M, 2010).

METHODS

Population and Design

Non-smokers' over the age of 65 were required to be eligible, as was no indoor exposure to tobacco smoke. We only recruited people with a confirmed history of coronary artery disease, a group that may be at risk for myocardial infarction from exposure to air pollution1. Out of 105 volunteers, 21 were ineligible, 24 dropped out, or the biomarker data was insufficient or invalid, leaving 60 subjects (Stone EA, 2006). In 2005–2006, two retirement communities were examined, and in 2006–2007, two more. Weekly measurements of plasma IL-6 and exhaled NO were taken on each subject for up to 12 weeks. To increase the variability of primary and secondary organic aerosols, each community was studied over two distinct 6-week seasonal periods (Rogge WF, 1993). Primary organic aerosols primarily originate from automobile traffic in the Los Angeles basin.

Measurements of Exposure

We determined the concentrations of the following particulate air pollutants over the week preceding each Friday when biomarkers were measured: total number of particles, as well as PM2.5 black carbon, organic carbon, and elemental carbon. Black carbon and elemental carbon are markers of primary organic aerosols that are similar but not identical (Verma V, 2009). As previously mentioned, we estimated secondary organic carbon as a marker of secondary organic aerosols and primary organic carbon as a marker of primary organic aerosols from total organic carbon. In addition, we used the Sioutas Personal Cascade Impactors12 to collect daily size-segregated particle samples on filters for five days prior to biomarker measurements12.13. Three size fractions were sampled: accumulation mode particles with diameters between 0.25 and 2.5 micrometres (PM0.25–2.5); and particles in the coarse mode, 2.5–10 m in size. Due to the fact that the upper cut point for the ultrafine mode is considered to be 0.1–0.2 m, PM0.25 is referred to here as "quasi-ultrafine." Standard gravimetric techniques were used to determine the daily particle mass. After combining the five filters, we looked at the 5-day average organic component concentrations in PM0.25. As previously described13,14, we used Gas Chromatography/Mass Spectrometry to examine the composites for 92 distinct organic compounds (Lane KB, 1998). We categorized representative organic components as follows: organic acids, n-alkanes, and polycyclic aromatic hydrocarbons with low, medium, and high molecular weights are all examples. Primary organic aerosols are thought to contain the majority of polycyclic aromatic hydrocarbons. Using a Total Organic Carbon Analyser, composites were also analysed for water-soluble organic carbon in aqueous extracts. Hopanes are tracers of primary vehicular aerosols because they are present in the lubricant oils of diesel and gasoline vehicles.15,16 Although a portion of water-soluble organic carbon comes from biomass burning, both organic acids and water-soluble organic carbon are tracers of secondary organic aerosols.19 We also measured pollutant gases that are regulated by the US EPA using standard federal reference methods. These included hourly NOx, CO, and O3, which are non-PM markers for fossil fuel combustion and photochemistry, respectively.

DISCUSSION

IL-6 had a positive correlation with primary organic aerosol markers, particularly carbonaceous markers, as previously mentioned9,10. However, the presence of specific organic chemicals in IL-6 did not have a positive correlation with secondary organic aerosol markers. Exhaled NO showed the opposite pattern: it was linked to the secondary organic aerosol marker in PM2.5 secondary organic carbon, to the secondary organic aerosol marker in PM0.25 water-soluble organic carbon, and to the secondary organic aerosol marker in organic acids, but not to the primary organic aerosol marker. However, primary organics still have an impact on airway inflammation; In order to confirm this with greater certainty, a larger sample size would be required. However, the different effects of organics on pulmonary and extra pulmonary target sites may be determined by different chemical reactions, solubility, and interstitial transport of aerosol components. The associations of IL-6 with NOx and CO, which are indicators of combustion sources in the Los Angeles air basin, back up the findings regarding the particles. On the other hand, the associations of exhaled NO with O3, which is a clear indicator of the photochemical activity of the pollutant, contradict the findings. The hypothesized progression from airway to systemic inflammation by particle deposition through the airway is not supported by our divergent findings for particle components.4,5 Furthermore, we were unable to explain the negative association between exhaled NO and IL-6. We also discovered that both outcomes are related to the potential of soluble components in particle extracts to induce generation of cellular reactive oxygen species in vitro. A cohort study involving 1000 adults found no association between exhaled NO and C-reactive protein or fibrinogen.25 Based on these findings, we hypothesize that air pollutants may have an impact on both exhaled NO and IL-6 through oxidative stress or redox signalling. However, experimental models would be required to test the hypothesis of shared mechanisms. For instance, the production of reactive oxygen species, which is indicative of the extract's particle concentration and chemical toxicity, may simply have been a nonspecific reflection of cell injury and stress in response to the in vitro challenge.

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