

Air Pollution and Health; Are Particulates the Answer?
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Many recent studies clearly indicate that there are health effects of contemporary air pollution in the US. The most common indicators of the pollution associated with these effects have been measures of particulate matter. In many multi-pollutant studies, measures of particulate matter appear to be the most strongly associated with health endpoints, but this is not true for all studies. This heterogeneity needs to be explored to help understand what is happening. It is complicated even further because all of the measured pollutants in these studies could be acting as surrogates for some other pollution component that is not measured. Toxicology studies have been of limited help; cardiovascular responses have been found in dogs exposed to concentrated particles, but the responses have not been related to PM mass *per se*. Differences have been found in the responses of various inflammatory markers in rats and human volunteers exposed to different particle extracts. These results suggest clearly that to the extent that particulate matter is involved in the air pollution/health equation, all particulate matter is not equally toxic. The only way to resolve this question is to understand air quality in considerable detail and to learn which measures are the best indicators of health responses, realizing that these measures themselves can even be surrogates.

The ARIES (Aerosol Research Inhalation Epidemiology Study) was designed to help precise those components were most associated with health responses. This study combined an intensive air quality monitoring program (which measured well over 100 air quality variables per day) in Atlanta with several epidemiological studies. The several studies allowed investigators to study different endpoints, but also to determine if there was consistency in response for similar endpoints. The endpoints measured were all acute responses, which have largely been associated with air pollution, including particulate matter, in earlier studies. (The study designs which consider longer term responses do not lend themselves as readily to studies of detailed pollution components because these studies usually involve the comparison of several geographic entities; hence detailed monitoring would be required for each entity in the study.) The monitoring program began in the summer of 1998. Analyses have recently been completed using 2 years or 25 months of air quality data for several of the epidemiological endpoints. Manuscripts are now under preparation, but a review of the major results achieved to date is most illuminating.

Real-time mortality data were collected for the two counties in which Atlanta is located. For total mortality there is a statistically-significant association only for CO. If the analysis is restricted to those 65 older, a panoply of pollutants are associated with excess mortality in single pollutant models. These pollutants include: PM-10; PM-2.5; the "coarse" fraction of PM-10; CO; organic carbon; and elemental carbon. In another epidemiological study, emergency hospital admissions were related to air quality. Responses differed by health endpoint. For cardiovascular diseases, the following were significantly associated with emergency room admissions: NO₂; CO; Polar VOCs; PM-2.5; elemental carbon; and organic carbon. For total respiratory diseases, CO, SO₂, and

PM-10 were significantly associated with emergency room admissions. Unscheduled physician visits focused upon respiratory endpoints. The only really short-term (0-2 day) response to air pollution was an association between upper respiratory disease and PM-10, the "coarse" fraction of PM-10, and NO₂. When longer lag times were considered between pollution exposure and physician visits, several more pollutants were indicted. For asthma, PM-10, the "coarse" fraction, ozone, NO₂, and elemental carbon were all significantly associated with increased physician visits. For upper and lower disease infections, there was some variability in response, but PM-10, the "coarse" fraction, and NO₂ were all associated with increased visits. Another element of the epidemiology studies examined whether there was any association between air quality levels and events reflecting the discharge of therapeutic shock or pacing among patients wearing defibrillators. Preliminary analyses indicate significant associations between these events and levels of CO, the "coarse" fraction of PM-10, and the organic carbon fraction of PM-2.5. Results for other endpoints and from multi-pollutant models are not yet available.

Is Atlanta typical and can these results be extrapolated elsewhere? As with all studies, we have more comfort in their results when they are replicated in additional settings. Additional settings must be considered, but Atlanta is an appropriate study area. It is out of compliance for the ozone standard and will be out of compliance for the current fine particulate standard; hence pollution levels are high enough to detect impacts of standard exceedances. Secondly Atlanta air quality is influenced by a variety of sources: traffic, power plants, pulp and paper plants, agriculture, light industry of several types. Hence analyses are not limited to a small subset of sources. Finally there is no reason to suspect that there is a unique source of air pollution in Atlanta, which would not likely be found elsewhere. Hence these results are not likely due to some obscure component(s) of pollution found only in Atlanta.

There are several conclusions that can be derived from this study. First of all, it supports the results from other studies that find links between air pollution and health at current US levels of pollution. We have not yet investigated the shapes of dose-response curves; hence no statements can be made about the existence of thresholds, below which health effects are not seen. Secondly, the results suggest that different pollutants are associated with different endpoints. The contrast between the pollutants associated with cardiovascular and respiratory endpoints is striking. CO, PM-2.5, and the carbon-containing components of PM-2.5 are associated with cardiovascular responses. The "coarse" fraction of PM-10, PM-10, and the gaseous pollutants are associated with respiratory responses. Finally, the results clearly indicate that all components of PM are not equally toxic. It looks as if the "coarse" fraction of PM cannot be dismissed. Its presence in the results could indicate that it is a good surrogate for another culprit or it could indicate that this fraction itself is of health concern. Clearly more consideration is warranted. With respect to PM-2.5, chemistry appears to be very important. Whenever PM-2.5 is significantly associated with a health endpoint, carbon-containing particles are also associated with that endpoint; sulfates and nitrates are never significantly associated with a health endpoint. To date metals have not yet shown up in any analyses; however, the only metal variable considered to date was total soluble metals. Analyses examining specific metals are planned.

To return to the title question, are particulates the answer? The associations are clearly there. It is unclear to what extent some of these variables may be surrogates for some other pollutant, but particulates (both the fine and coarse fractions) cannot be ignored. But if it is particulates, it isn't all particulates. Intense efforts are required to make sure that we target the correct ones; otherwise specific pollution measures may provide little health benefit. Finally we cannot forget the gases.