Source Apportionment of Fine Particulate Matter in the U.S. and Associations with Lung Inflammatory Markers IL-8, COX-2, and HO-1

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INTRODUCTION AND BACKGROUND

Associations are well established between particulate matter (PM) mass concentrations and increased human mortality and morbidity. Fine particulate matter (particle diameter < 2.5 mm) is most strongly linked to adverse health impacts. The toxicity of PM may depend on the PM source (e.g. vehicle exhaust, coal combustion, etc.) and composition of PM which will vary by location. While a number of epidemiological studies have shown that certain PM sources are associated with specific health outcomes, the mechanisms are still unclear.

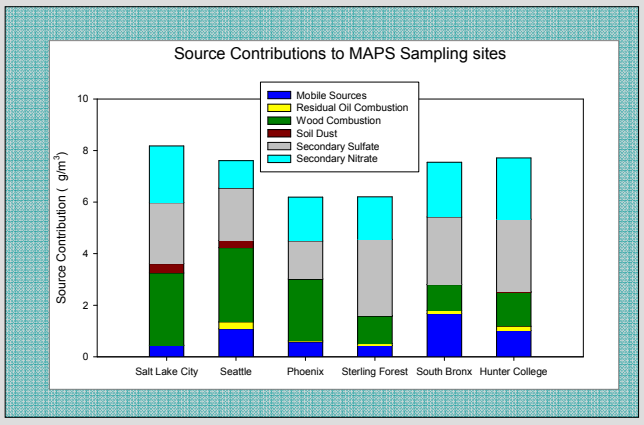
In this study, the association between fine PM sources and three lung inflammatory markers (interleukin-8, cyclooxygenase-2, and heme oxygenase-1) was evaluated in multiple cities with contrasting PM sources as part of the Multiple Pollutant Study (MAPS).

SOURCE APPORTIONMENT

The EPA Chemical Mass Balance Model (EPA CMB8.2) was used to quantify the sources impacting each site.

Operational scheme of the CMB Model:

Speciated data from each sampling site + ***Source Profiles*** → ***Estimated source contributions to each site***

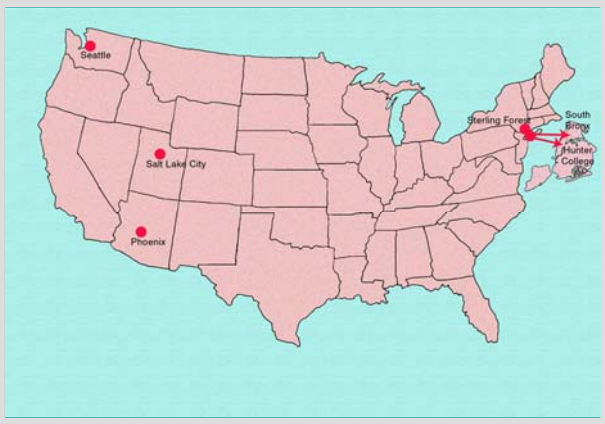


SAMPLING AND ANALYSIS

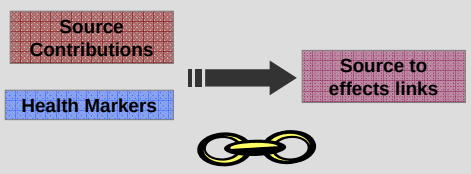
Fine particles were collected in six sites in the U.S. from December 2003 to May 2004 [See map below]. One-week samples (24-hrs a day) were collected at each site over a 4-week period using a ChemVol high-volume cascade impactor operating at a flowrate of 800 L/min. Samples were analyzed for the following:

Chemical Speciation
- Trace metals [Inductively Coupled Plasma – Optical Emission Spectroscopy]
- Ions [Ion Chromatography]
- Elemental Carbon [Thermal Evolution and Combustion]

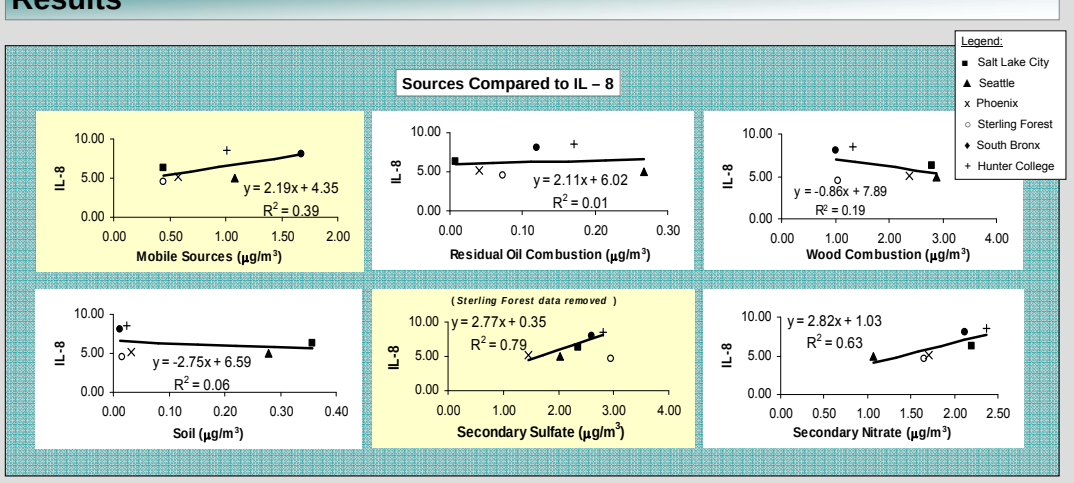
Lung Inflammatory Markers
- Interleukin (IL) – 8 [Enzyme – Linked Immunosorbent Assay]
- Heme oxygenase (HO)-1 and cyclooxygenase (COX)-2 [Reverse Transcriptase – Polymerase Chain Reaction]

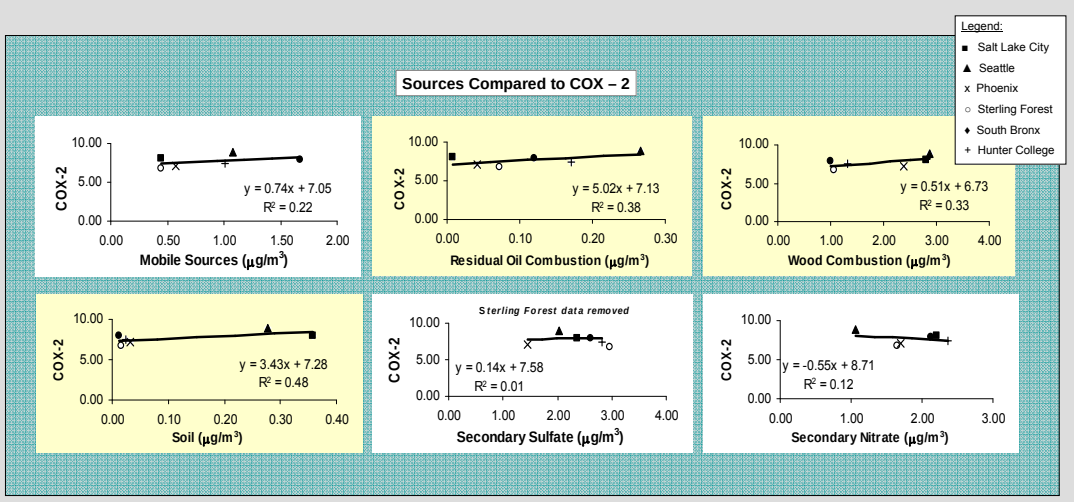


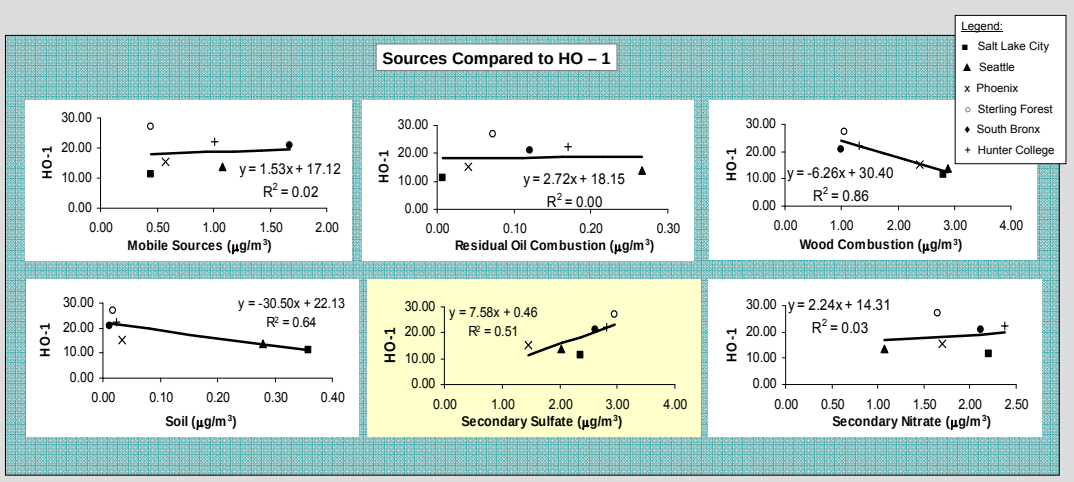
A linear regression was used to determine the relationship between PM sources and health effects



Results







Conclusions

Different source contributions across the sampling sites allowed for evaluation of the relationship between sources contributions and the inflammatory markers. Each inflammatory marker showed varying relationships with the sources. IL-8 and HO-1 were both correlated with secondary sulfate from coal combustion, although at differing levels. Weak associations were observed with COX-2 to wood combustion, residual oil combustion, and soil. Combustion and industrial sources appear to elicit an inflammatory response which is consistent with previous health studies.

Disclaimer: Although this work was reviewed by EPA and approved for publication, it may not necessarily reflect official Agency policy.