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Atmospheric Chemistry and Physics Discussions

Interactive comment on "Oxidative potential of ambient water-soluble $PM_{2.5}$ measured by Dithiothreitol (DTT) and Ascorbic Acid (AA) assays in the southeastern United States: contrasts in sources and health associations" *by* T. Fang et al.

Anonymous Referee #1

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In this study, the authors assessed the water-soluble oxidative potential of PM2.5 collected in Southeastern U.S. based on both DTT and AA assays, and compared the results of two assays in the view of their association with chemical components, sources and emergency department (ED) visits. In my opinion, this is an important and careful study with large database, providing essential information on the origin and potential health outcome of the water-soluble oxidative potentials of PM2.5. In addition, the result could help future studies to better interpret the data based on those assays.

However, there are several issues that should be addressed in the manuscript.

A major concern is on the oxidative potential of PM2.5. Actually I'm afraid that oxidative potential from water-soluble components is far from enough for the evaluation of PM2.5's toxicity or health effects, given that a series of studies have suggested that some hydrophobic components (e.g. represented by PAHs) from vehicles are the major toxic components on human health (Delfino et al., Environ Health Perspect 2010, 118: 756-762; etc.). Why not measure the oxidative potential of hydrophobic components if the authors wanted to link the oxidative potential of PM2.5 with some health outcomes?

The following are some specific comments: 1. Page 30615, section 2.2.1: Could sonication for half an hour in the water phase generate OH radical, which could result in great oxidative potential? 2. Page 30625, line 15: The spatio-temporal analysis could not draw the conclusion that the oxidative potential is influenced by different components from different sources, because there are no evidences in this part showed the similar trend for chemical components and sources. 3. Page 30626, line1: r2 or r? In case of r2, it's better to convert it into r, since r is used throughout the manuscript. 4. Page 30626, line 25: Since Pearson's r is used. Please provide information on the normality of the data and on whether data was log-transformed. 5. Page 30630, section 3.2.2: It is better to provide detailed data of the risk ratio (together with 95% CI) in the text instead of in Fig. 4 only. 6. Page 30631, line 3: The authors claimed that the epidemiology analysis "support aerosol particle oxidative potential as a mechanism contributing to these PM-induced adverse health effects", which I think might not be true. A more robust association doesn't necessarily mean a possible mechanism. For example, sulfate is considered as "benign" (page 30612, line 13), but actually sulfate is strongly associated with adverse health effects in epidemiology studies, probably because sulfate was co-emitted with toxic pollutants (Grahame, EHP, 2012). Personally, I think the association of DTT could also possibly be attributed to co-emission with toxic pollutants, especially given that (1). DTT is sensitive to organic species; (2). There is a lack of toxicology studies showing the health effects of oxidative potential; (3). No significant association was observed for AA. Although the authors attributed it to the

different uncertainties (page 30630, line 19), it is not convincing since r values of 0.60 and 0.68 are not that different.

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