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Atmospheric Chemistry and Physics Discussions

Interactive comment on "Atmospheric conditions and composition that influence $PM_{2.5}$ oxidative potential in Beijing, China" *by* Steven J. Campbell et al.

Anonymous Referee #1

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Based on a large suite of ambient data, this paper presents an analysis of four different chemical assays that quantify, what the authors refer to as, oxidative potential (OP). It is based on a data set from a multi-investigator field campaign in Beijing involving summer and winter sampling periods. Overall, the topic of particle OP is of current interest as a new measure that potentially better links aerosols to adverse health. The paper mainly repeats various analysis approaches (with a few tweaks) done in many other studies and seems to largely support earlier findings, from what I can tell, since what the actual new findings are is not really clear. The paper should be substantially edited before considering publication. The following are major issues:

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Discussion paper

What are the major findings of this paper? The Abstract provides very little insight on results.

The data interpretation is often sloppy and statements are made that are either speculation (see below) or illogical. Tightening up the paper would also lead to a much more concise and readable paper.

This paper is about assays, but few details on the assay (ie, how specifically the measurement was made) are given. Most details are in the supplement, yet this information is critical to the data interpretation. This is a major lapse since how the assay was conducted largely determines everything else in the paper. How can comparisons be made between these results and others if one doesn't even know if the assays are comparable? The most obvious is the AA assay; the authors seem to have developed their own protocol, different from previous methods, but go on anyway to compare their AA results to other published work. Specifically, for the AA assay they do not extract in synthetic lung fluid (this is not even mentioned in the paper), yet they compare their data throughout with previous AA assay results that do. What type of aerosol species are included in the assays, are these assays measuring water soluble or all aerosol species?

The reason for using these assays is to better link aerosols to adverse health, but there is no discussion in the Introduction/Background of the current knowledge on this matter. A number of the assays have been tested in health studies, less is known about some of the others. For example, what is the logic of detailed investigation of measurement that shows no evidence of being linked to health? Is the argument here that we don't know which assays are linked to health so these four were simply chosen? State exactly why these assays were investigated.

The term OP in this paper is used in a very broad sense. Assays that measure very different physiological processes related to ROS are all grouped as simply OP. More precise terminology would allow more detailed conclusion. It would seem better to

separate out the assays that measure exogenous ROS (DCFH) and the assays that measure species that can form ROS in vivo (DTT, AA). As a guide maybe refer to the figure in Lakey et al, Sci Reports 2016. As an example why this may matter, maybe the lung lining fluid has sufficient antioxidants present to suppress all the ROS on the particles, (one might want to ponder the difference in concentrations of ROS on the particle, ng/m3 based on the DCFH assay, and typical O3 concentrations, ug/m3, aren't both are exogenous ROS). But the ROS generated from aerosol components through interactions with physiological species is a different mechanism to produce ROS that may involve catalytic reactions (eg, Fenton reaction). Furthermore, can exogenous ROS species be translocated to other organs in the body such as is known for species than form ROS in vivo (eg, metal nanoparticles)? In my view, greater insights would be possible if the authors separated out these different processes (and hence assays) that can lead to oxidative stress.

Specific Comments.

The abstract is not informative since it contains little actual results. Most of the discussion is on what was done, whereas more emphasis could be placed on findings. For example, what exactly is the new results from this extensive research?

Line 64 defines OP: The capability of PM to produce ROS with subsequent depletion of anti-oxidants upon inhalation is defined as oxidative potential (OP) (Bates et al., 2019). By this definition is DCFH assay a measure OP since it does not produce ROS, as far as I know?

Line 174, typo analyze?

No detail is provided within the paper on the assay methods, instead it is given in the supplement, yet this is critical information needed in the interpretation of the results.

Please discuss limitations in measuring ROS with the DCFH assay using a filter that measures ROS on the particle (note the key word reactive).

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For what reason was just the AA picked to be shown in Fig 1 and the other assays shown in the supplement? (Same for later on in the paper).

If the assays are so highly correlated with mass (eg, AAv and DCFH), does that mean that the assays are not that useful? Why not just use mass to link to adverse health or particle toxicity, it is much easier to measure?

Line 289, given the high correlation between AAv and PM2.5 mass, why is it surprising or meaningful for AAm to have an inverse relation with mass since AAm= AAv/PM2.5 mass? This is totally expected and not really informative.

Lines 294-307 on possible reasons a large fraction of mass may be OP inactive on high mass days. What about the effects of particle age (oxidation, or other chemical aging processes). High PM2.5 mass could be fresher emissions? An example is PAHs to quinones or nitro-PAHs.

Lines 311-312. Not sure of the relevance of PM10 discussion since it was a PM2.5 measurement of DCFH.

Line 329, are there any quinones that are semi-volatile, please list them. There are semi volatile PAHs, but when they are oxidized does the volatility change?

Line 332, how specifically does boundary layer height affect the assay results?

Line 415-430 and on. The metal ions were not measured so how can the statement be made that the list of metals correlated to AA and DTT are related to redox reactions or on the role of Fe in various reactions. The logic does not follow. The authors are equating all chemical forms of these metals in the particles to the just the ion forms.

Line 51-545. If one does a more complete aerosol chemical analysis what is the point of the assay. Why not just use the chemical species in the health/toxicity studies?

Line 671 states in the conclusions: At present no single assay is completely representative of the totality of OP effects present in atmospheric PM. What is the basis for this

statement? How do the findings of this paper support this statement?

The last line of the conclusions; again, what is the basis for this bold statement, how do the findings of this paper support this statement?

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