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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.

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Reviewer 1

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

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what the actual new findings are is not really clear. The paper should be substantially edited before considering publication. The following are major issues:

We thank the reviewers for their comments. Please see below point-by-point answers, addressing the reviewer's comments. All of our answers are given in blue below and we indicate the lines changed in the revised manuscript.

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

We have now amended the abstract and conclusions section to further emphasise the key findings and conclusions of the paper.

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.

See below more specific comments.

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?

Due to the length of the manuscript, we felt it necessary to include the specific chem-

ical details of the assays in the supplementary material. The methods used are well documented in the previous literature and easily accessible in detail in the supplement. We have now specified in the main manuscript that we use an AA-only based assay (line 180-183) that does not contain additional synthetic lung fluid components, and that filter samples are extracted at pH 7 before reaction at pH 2, i.e. extraction is performed at physiological neutral pH conditions. Furthermore, we do not compare quantitative values as stated in lines 266-268, but qualitatively compare correlations of our AA method vs aerosol components with previous studies. In fact, we observe that this AA-based assay is in relatively good agreement with previous studies in terms of correlation between assay response and specific particle components, as discussed in Section 3.2

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 main aspect here is to identify how the response of four of the most widely-used OP and ROS assays are linked to other atmospheric components and processes using one of the most comprehensive atmospheric datasets acquired in recent years, during the APHH-Beijing campaign. Such comprehensive comparisons are sparse in the literature, and this campaign provided a particularly unique opportunity to correlate aerosol OP and particle-bound ROS with a uniquely comprehensive dataset. This novelty has now been highlighted further in both the abstract and conclusion in the main manuscript. Such studies constitute an essential step in terms of understanding assay response, as a well-constrained understanding of aerosol chemical influences on these assays allow better understanding of their response and thus a firm founda-

tion to determine the health-relevance of such measurements. We have now added more information regarding the links between aerosol OP and toxicity in air pollution epidemiology; please see Section S2 of the Electronic Supplementary Information.

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.

This distinction is made in the introduction (line 103-108), but has now also been elaborated on in Section 2.2.2, as well as in Section S2 of the ESI. We have further clarified definitions in lines 242-250.

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).

We now more clearly distinguish the different sensitivities of different assays lines (101-105). We refer to the assays specifically as DCFHv, AAv, EPRv and DTTv for e.g. volume-normalised data throughout the manuscript to distinguish the assay response, due to the differing sensitivities of each assay to different chemical components present in PM. We have now discussed these definitions in lines 242-350. We do in places refer to total OP of PM to summarise all of the assay responses (e.g. all show a stronger correlation in winter compared to summer). Additionally, we discuss the limited knowledge available in the literature about compound-specific reactivity of the different assays in lines 83-105, and some of the broader implications of antioxidant-oxidant

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balance in the biological context in Section S2 of the ESI.

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.

We would like to emphasise that this study is not a biomedical or epidemiological study, and thus we cannot address potential translocations of particles within the body; please see Section S2 of the ESI for further information. See comment above regarding the separation of assays in the manuscript. 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?

As discussed above, we have now further clarified that DCFH predominantly measures particle-bound ROS (lines 101-105).

Line 174, typo analyze?

We consistently use British English throughout the manuscript.

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.

As discussed above, we have now elaborated Section 2.2.2 (lines 164-175), and due to the length of the manuscript, detailed assay protocols are easily accessible in the supplementary material.

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

This is a valid point, and it is certainly a limitation of filter-based measurements for particle-bound ROS and OP. We cannot rule out underestimating both particle-bound ROS and OP using offline filter-based measurements, but this offline method allows unparalleled comparison with other aerosol composition measurements. Additionally, with reference to Figure 2, we observe substantial variability in the mass-normalised DCFHm values in both summer and winter, implying that we are capturing a variability of particle-bound ROS as measured by DCFH. These may well be longer-lived components, and at present it is difficult to estimate quantitatively the degradation of short-lived ROS species prior to analysis. We have added additional discussion in 187-190 in the revised manuscript.

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).

Figure 1 is only an example of the daily variation of the assays, and given the length of the manuscript and the inclusion of several other figures, we only show AA here as an example of the daily variability, and the other three assays are contained in the ESI. We have added all four assays to Figure 6, and added DTT data to Figure 7. Additionally, all tables in the manuscript and figures in the ESI contain data measured using all four assays where they are not already presented in the main text.

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?

AAv and EPRv are only significantly correlated with PM2.5 mass concentrations in winter, and the in summer show a poor correlation, as evidenced in Figure 2 and discussed at length in lines 279-290. Poorer correlations are also observed for DDTv and DCFHv vs. PM2.5 mass in summer compared to winter. As we also state in lines 295-297

the difference in the strength of correlations between volume-normalised assays and overall mass in the summer and winter indicates if anything that PM mass is not always a good indicator for predicting the oxidising properties of particles, and that either source related, composition related or atmospheric changes alter the oxidising properties of particles, and hence acellular methods and indeed relationship between assay response and composition changes are required to determine the oxidative potential of ambient particles in Beijing.

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.

We believe this is not the case. If indeed a linear relationship was observed between volume-normalised OP measurements and PM2.5 mass concentration (which is not always the case, see above) then normalising per unit mass we would see relatively constant mass normalised value on each day (as the OP would simply scale with PM2.5 mass concentration). However, as observed in Figure 3 and discussed in lines 303-323 in the revised manuscript, substantial variability of the mass normalised values is observed for each assay, and is the case with AA and DTT, days with higher PM concentrations tend to have lower intrinsic OPm values compared to lower PM mass days. If the above comment was the case, we would not observe this and would observe relatively constant OPm between low and high mass days.

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.

This is an interesting point but we do not have the required additional information to comment further on the ageing of the particles and to evaluate this argument, but certainly this should be considered in future studies as the reasons behind observations

displayed in Figure 3 were unclear.

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

This is a valid point and these lines have now been removed from the revised manuscript.

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?

There are a range of smaller quinones that have been detected in both the gas and particle phase, and have been found to have a temperature-dependent concentration in the particle phase (see e.g. Dalgado-Saborit el al., Atmospheric Environment 77 (2013) 974-982.) We have clarified this further in the revised manuscript and added this reference (lines 350-354).

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

Boundary layer data was unfortunately not available at the time of writing and we can thus not comment further on its effects.

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.

As discussed in lines 454-463, we do not comment on the speciation or redox-state of the metals we observe a correlation with as that data was not available at the time of writing. However, we observe correlations with a range of metals with AA and DTT and qualitatively compare these observations with previous studies. Future work should certainly explore the role of metal speciation and redox-state further and not only water-soluble vs. total metal content; we partially discuss this in Section S2 of the ESI.

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?

A complete chemical analysis of aerosol is challenging, and requires a breadth of expertise and instrumentation to perform, in addition to the large expense often associated with full chemical speciation. From an oxidative potential perspective, and demonstrated in this paper, a broad range of known chemical components contribute to the response of acellular assays, in particular AA and DTT. Thus, the assays encompass a wide range of chemical components, from the perspective of their oxidative potential, and thus provide a relatively simple metric to describe a chemically complex process. This point has now been emphasised further in the revised manuscript (lines 715-718, Section S2 of the ESI).

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?

This statement has now been amended and clarified in the revised manuscript (see lines 715-718).

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?

We do not think that our statement at the end of the conclusion section is particularly bold but we have reworded in the revised manuscript (see lines 715-718).

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