

# ***Interactive comment on “Inflammatory responses to secondary organic aerosols (SOA) generated from biogenic and anthropogenic precursors” by Wing Y. Tuet et al.***

**Anonymous Referee #1**

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In this work, the authors examined the cellular response to laboratory-generated secondary organic aerosol (SOA). The health impacts of SOA have been an emerging topic lately, and this work focuses on characterizing the production of reactive oxygen/nitrogen species (ROS/RNS) in macrophages for different types of SOA, and the production of biomarkers of oxidative stress. This is an important topic because oxidative stress has been proposed to be the main mechanism by which particulate matter leads to cardiopulmonary outcomes.

The authors produced SOA in chamber experiments under various conditions, collected SOA onto filters, and exposed SOA extracts to a cell line for murine alveolar macrophages. The results were compared to measurements of chemical oxidative

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potential using the now commonly used dithiothreitol (DTT) assay. While there is a general agreement in oxidative response, there are subtle differences between the assays, suggesting different biological mechanisms that may inspire future work. The experiments are conducted in a careful manner and the results are well interpreted. This is a new area for ACPD to publish, but I believe it is a relevant topic. Otherwise the paper is technically strong and I have no major comments. It should be published after these minor comments:

- Page 4 Line 73: The authors state that there are many gaps. What are the gaps? What is the specific gap this work is attempting to address?
- Page 4 Line 76-81: the authors state that health studies focus on primary emissions rather than SOA, but then cited more SOA studies than primary studies. Seems contradictory. In fact, there is now a lot of attention on SOA. I suggest rephrasing.
- Page 5 line 95: Why were IL-6 and TNF-alpha chosen as the biomarkers? There are many other markers (such as HO-1, IL-17). Are these biomarkers better indicators of oxidative stress and better linked to health endpoints than others? Given that there is a nuanced response shown in Fig. 4, perhaps the choice of IL-6 and TNF-alpha was deliberate, but as a reader I am not sure why.
- Page 8 Line 149: 45% relative humidity is still quite dry. I would not label it as "humid".
- Page 8 lines 154-158: does an acidic seed affect the background ROS production? Or is there sufficient buffer that cells are exposed to the same pH?
- Page 8 line 161: What is zero air? Is this purified air? How is the air purified?
- Page 8 line 169: presumably this concentration of OH is yielded only upon irradiation for the specific set of chamber lights.
- Page 10 line 212: why is 24 hrs chosen? What happens if cytokine levels were measured earlier or later? Are there recovery effects of exposure?

- Page 12 line 247: H<sub>2</sub>O<sub>2</sub> is unlikely to be taken up by inorganic seeds particles on a Teflon filter (as shown by the authors' results), but may be taken up if there are organics coated on the filter. Is it possible there is further heterogeneous reactions of H<sub>2</sub>O<sub>2</sub> on the organics, given the H<sub>2</sub>O<sub>2</sub> concentrations are 3ppm?

- Page 12-13 lines 259-268: This is a central finding of this manuscript: the carbon backbone seems to play a bigger role than formation conditions. While I do not dispute the results, this finding is hard to rationalize. Formation conditions will affect mostly the functional groups that go onto the molecule (there may be small changes in the backbone with fragmentation pathways), while precursor identity will determine the size and shape of the backbone. ROS is likely produced through electron transfer to/from the functional groups interacting (or reacting) with O<sub>2</sub>, H<sub>2</sub>O, antioxidants and NADPH. It is therefore difficult to imagine that the functional group matters less than the backbone structure. Also, by that logic, reactions that change the molecular structure (such as oligomerization, fragmentation) would change the cellular ROS quite significantly. Is there any evidence of that?

- Page 16 line 326: this is an interesting explanation. If fatty acids are really changing cell functions that significantly, meat cooking organic aerosols, which are composed almost entirely of fatty acids, would elicit very strong responses.

- Page 16 Line 343: Naphthalene is not "completely" different. For example, IL-6 and TNF-alpha are still somewhat positively correlated at low levels. Perhaps it is just a more distinct pattern.

- Page 18 Line 395-396 and Fig. 3b: what does significant correlation mean? There is an asterisk in Fig. 3b. Does that mean the trend is statistically significant? If so, please provide statistical justification (e.g. 95% confidence interval?). Does it have to be a linear model? Does the correlation still stand if naphthalene SOA (which is the outlier) points are removed? It would seem reasonable to me to remove the naphthalene system if there is reason to believe it has a very different toxicological mechanism.

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- Page 19 lines 404-425: What is the relationship between ROS/RNS and cytokines for these SOA systems? It seems that plotting them against each other would help explain trends in each SOA system, or at least establish whether or not ROS/RNS are linked to upregulation of these cytokines.

Technical comments:

- Page 3 Line 52: “anti-oxidant” should be “antioxidant”
- Page 7 line 127: “form” should be “from”
- Page 21 line 464: “RNS/RNS” should be “ROS/RNS”

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