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Interactive comment on "Reactive oxygen species (ROS) emissions and formation pathways in residential wood smoke under different combustion and aging conditions" *by* Jun Zhou et al.

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This paper presents a very detailed analysis of reactive oxygen species (ROS) on aerosol particles produced from wood burning. The work systematically investigates differences in wood burners and burning conditions. Primary and secondary ROS is also studied. For what it is investigating, the paper is very clearly written and the methods and results are well supported. The paper was a pleasure to read. I have no major comments with the overall work, however, first it should be clarified exactly what species are thought to be measured with this assay (e.g., list them) and how, or what

evidence, or what is the basis for claiming throughout this paper that these species are associated with adverse health.

For example, a major overall issue that must be resolved is the conflation of different assays for measuring ROS and their links to adverse health. This paper uses the DCFH assay on collected particles to measure reactive oxygen species on or within the particles (or more specifically, components of the aerosol that will react with DCF, see comment above on specifying these). As far as I know of, there is no published study linking this measurement of ROS to adverse health effects, so it is not clear to me how these authors can relate this work to health effects, other than in a general way. In contrast there are a range of other assays that attempt to measure the ability of particles to consume reductants or generate ROS in vivo. Unfortunately these assays have also been referred to as measurements of ROS, but more recently are referred to as measurements of aerosol oxidative potential. These assays have been linked to adverse health responses, but are not what is being measured in this work. This paper tends to add to the confusion in the aerosol community on ROS by not precisely distinguishing the measurements presented here from other oxidative potential measurements. This needs to be rectified. The issue is discussed further below.

Specific comments

The abstract should be modified to clarify the type of ROS being investigated as the term ROS has many meanings. As an example, could change the first sentence to. . .

Wood combustion emissions can induce oxidative stress in the human respiratory tract caused by reactive oxygen species (ROS) on aerosol particles, that are emitted either directly or formed after oxidation in the atmosphere.

It would also be helpful to state somewhere in the abstract how ROS was measured, ie the DCFH assay. This would add much need clarity on what this paper means by the term ROS

Line 53-54, These adverse effects may be related to oxidative stress caused by free radicals induced by inhaled PM, which overwhelms the antioxidants in the body (Lobo et al., 2010). It seems this is the justification in this paper; that particle bound ROS measured with the DCFH assay is potentially associated with adverse health. But there is a very significant logical jump to assert particle bound ROS results in the physiological oxidative stress response (ie, as discussed in Lobo et al). For example, maybe all particle bound ROS is eliminated in the highly reducing environment of the lung lining fluid? Further more, this assay does not measure ROS induced by PM, it measures ROS on the particle as it exits in the ambient air.

Line 55 states: In turn, free radical formation may be due to reactive oxygen species (ROS) present in atmospheric aerosol, transition metals undergoing Fenton reactions, or redox cycling organic compounds like quinones.

Clarify where the free radical formation is occurring; within the particle in ambient air or once the particle is deposited and interacts with physiological species. This distinction is critical as they are both referred to as ROS, but are different processes and measured with different assays. To be clear, most health effects studies on ROS are not linked to the ROS being measured in this paper, they are linked to assays that measure ROS generation by physiological species interacting with aerosol species. This can be quantified through measuring ROS generated by cells exposed to particles and measuring loss of reductants or generation of oxidants in a simulated physiological environment. Example assays in the first case include measurements of cellular responses generating ROS measured with the DCFH probe, and in the second case acellular test assays such as DTT, GSH, Ascorbic Acid, where the loss of the reductant is measured (ie, DTT, GSH, AA) or various ways are used for detecting oxidants generated in the simulated environments. This is very different from the ROS measured in this paper.

If the authors know of references linking particle-bound DCFH-measured ROS (ie, what is reported in this paper) with adverse health endpoints, they should be included. If

there are none, maybe this should be noted as a motivation for needed research.

The authors could include references that specifically tested ROS formed in vivo (ie, not what is measured here) with health endpoints instead of providing only generic references on this subject (see ref. list at end).

Regarding metal ions, fenton reactions and quinones, does this assay detect these species or is this sentence saying that these species make ROS in the particle in the ambient atmosphere, which this assay measures. Please clarify. This is confusing because these species are mainly what drive the oxidative potential of aerosols measured with the assays noted above.

Ling 67 and throughout. It is really incorrect to call the measurements reported in this paper as oxidative potential. The measurements reported here are of the ROS associated with particles (i.e., H2O2, organic peroxides). Again, one must be very clear to distinguish this measurement from other assays, which largely measure a different property of the aerosol (ie, which should only be called oxidative potential), but unfortunately is also referred to as ROS.

It would be helpful if the authors included a discussion/summary of specifically what species the DCFH assay detects. (There are papers published on this).

Use of a PAM and smog chamber for aging experiments. In both cases please give specific OH concentrations the particles are exposed to so the reader has a sense of differences between these experiments and actual atmospheric aging (eg, ambient OH concentration is noted to be approx. 1E6 1/cm3). A brief discussion justifying this method for simulating actual aging processes should be included since this method comprises a major aspect of this paper and OH concentrations are likely very high and unrealistic. There will be chemical processes that do not occur in the actual ambient atmosphere that will occur in these experiments (ie, any radical-radical reactions pathways).

Line 207, this method is not measuring oxidative potential.

Ling 473-474 it states: This indicates that applying automatic combustion devices operated at optimum conditions, to achieve near-complete combustion, is most effective to minimize ROS emissions. This is a significant finding, but achieving near complete combustion also likely significantly reduces emissions of OA and BC. BC is known to be toxic. It might be important to also note this here.

Final line of paper: The comparison and evolution of ROS with different combustion and aging conditions in this study might be used for a speedy assessment of potential health risks of wood combustion emissions from different combustion and aging conditions. This assumes that the ROS associated with the particle is a health risk. Again, what evidence is there to support this?

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