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**ACPD** 14, C511–C513, 2014

> Interactive Comment

## Interactive comment on "Technical Note: Particulate reactive oxygen species concentrations and their association with environmental conditions in an urban, subtropical climate" by S. S. Khurshid et al.

## Anonymous Referee #1

Received and published: 16 March 2014

The paper presents data on ROS measured by the DCFH assay on fine particles collected from filters. Ambient data over a longer time period are presented, which the authors suggest make the paper unique. The use of integrated filters to assess ROS (reactive oxygen species), reactive being the key word, is somewhat suspect and never really addressed in the paper. Many very general statements are made without sufficient explanation or validation. This includes why the authors believe oxidants on the particle are much worse than gas species, or aerosol components that generate ROS in vivo (example transition metals, etc). It appears at times the authors equate what



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they refer to as exogenous and endogenous ROS, when discussing ROS modes of action or discussions referencing published work. Overall, a number of points should be clarified prior to publication.

Pg 5063 line28. Why are water-soluble ROS species more likely to cause oxidative stress and cell damage deep in the lung, as stated, versus if they were deposited in the upper airways (ie, by what mechanism)? What is special about deposition deep in the lung in terms of ROS?

Along these lines, the concentrations of gas phase oxidants appear to be much higher than the oxidants found on the particles measured by this assay. For example, the level or oxidants calculated by this assay is of the order of 0.01 to 4 nMolH2O2/m3 (see Abstract). O3 concentrations in this study are between 20 and 60 ppb (Fig 2), which is roughly 800 to 2500 nMoles O3/m3, orders of magnitude larger than the ROS measured. A similar calculation could be done for H2O2, say ambient levels are 1 ppb, or about 40 nMoles H2O2/m3, over an order of magnitude of the ROS measured. The authors seem to argue that transport of oxidants by particles deep in the lung make them much more toxic than gas phase oxidants, which are much more abundant, but may be deposited higher up in the respiratory system. Why is this?

Page 5064 Line 3. What is the difference between the ROS being measured and what is referred to as endogenous ROS? Can the two be readily compared? Also, the meaning of line 6 is not clear, how does exogenous ROS influence production of endogenous ROS (by what mechanism)? (Really, the authors should define what exactly is meant by endogenous and exogenous ROS).

Page 5065. The sampling method is:  $\sim$ 3hr sampling time, analysis 1hr after end of sampling. By definition, what's being measured is highly reactive. Are there any studies tested the viability of filter sampling of this type of ROS, or have the authors tested sensitivity of the measured ROS to sample integration time and delay between sampling and analysis?

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It is possible that many of the chemical components making up ROS that are being measured in this study on filters are in the gas phase given the low levels of particle water (except under high RH conditions), H2O2 being an example. There did not appear to be any gas denuders used in this study. How do the authors know if much of the ROS is actually a positive artifact and not really associated with the particle?

Page 5065 line 18. It is stated that the method used here was modified, but not explicitly stated what that modification was.

Page 5069, paragraph starting on line 6. The discussion here is on correlations between gas phase H2O2 and particle phase ROS? Is gas phase H2O2 correlated with particle phase H2O2, which in turn could be correlated with particle ROS. The logic on which this discussion is based is not clear.

Page 5070 line 16. The regression results, as far as I can tell, do not prove that ROS is a function of O3, T, and solar radiation since O3, T and solar radiation are correlated with each other (Table 1), they are not independent variables in the regression model.

First line of conclusions. Does this assay really measure the oxidative capacity of PM? It appears to measure the concentration of oxidants associated with the particle, which is not the same thing.

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