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Interactive comment on "Enhanced toxicity of aerosol in fog conditions in the Po Valley, Italy" *by* Stefano Decesari et al.

Anonymous Referee #1

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This paper uses a rat macrophage assay to assess the toxicity of aerosols measured in the Po Valley during the cold season when fogs were often present and emissions from wood burning prevalent. The main finding is that under these conditions, fogs lead to SOA that is toxic (as per this specific assay used). The results of this paper are interesting in that they add to an existing body of literature showing the toxicity of aerosols increase with oxidation processes, along with the fact that wood smoke aerosol components have a high oxidative potential.

These authors have reported associations between their ROS measurement and WSOC in a number of past studies and asserted that the WSOC was secondary (for example, [Daher et al., 2012; Saffari et al., 2013; 2014]). The authors should note this and clearly state what is new about this work, ie, that the processing may be heterogeneous?

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There is a substantial body of published literature on oxidative potential, albeit with different assays, that discusses the effect of oxidation on increased toxicity. Examples include chamber studies and analysis of ambient data (discussed more below). None of these, which are very pertinent to this paper, are cited in this work.

No evidence or reference is provided establishing that this assay (that is, this specific ROS measurement) is linked to adverse human health effects, although a health connection is implicitly assumed throughout. It would appear the implicit assumption is that because this is a cellular assay it can be directly connected to adverse human health responses, but there are many components to a cellular assay that can lead to various responses, so the connection is not established until empirically proven. This could be done by citing comparisons of their assay responses to other assays that have established links to health outcomes or oxidative stress markers or cite specific associations between this assay and health effects. As the paper stands, there really is no basis for asserting that these results specifically apply to human health, instead the author need to qualify this assertion throughout the paper.

Finally, there is the question of importance on a broader scale and associated assertions by the authors of wide ranging impacts. The authors suggest that populations are commonly exposed to aerosol that has been fog-processed, but is this true, what is the evidence for this? Quantitative support for this assumption should be provided to demonstrate that this mechanism is truly of broad importance, as stated. Overly expansive statements of the importance of this work should also be avoided throughout.

There are a number of other issues that also need to be addressed, which are discussed in more detail below.

Detailed comments.

Could not find any data on the various sample sizes (N).

The authors measured and report ROS of collected fog water and claim this is poten-

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tially linked to adverse health. How does this happen? Is the exposure route through inhaling fog drops? Likely not. Instead the argument is that the fog serves mainly as a chemical reactor that produces the toxic species. The drops evaporate and the fine PM is now more toxic. This assumes that all species in the fog contributing to ROS remain during evaporation, but it is stated that much of it is small molecular weight organic acids, which are likely very volatile and lost. If the fog ROS is equivalent to the ambient PM ROS, than these volatile species play no role. There seems to be some inconsistency in the author's arguments. Maybe this can be clarified.

A number of studies, such as chamber studies, have shown that if you take primary emissions, (say from a combustion source, like an automobile) and oxidize them, the oxidative potential substantially increases [Li et al., 2009] [McWhinney et al., 2011]. Likewise, chamber experiments in which SOA is produced from various VOCs show that some compounds, such as those found in biomass burning emissions, when oxidized have high intrinsic oxidative potentials [McWhinney et al., 2013]. It has even been shown that fresh soot that is subsequently oxidized has substantially increased oxidative potential (eg, [Antinolo et al., 2015; Shiraiwa et al., 2012]). All of these results are extremely pertinent to this work, but never cited nor discussed.

A variety of elemental concentrations of transition metals were measured, which are claimed to be redox active. Take Fe, for example. In the soluble form is redox active, but the insoluble form is not. Most measured elemental Fe is not soluble (many references show this) so no association to the water-soluble form, and hence redox activity, can be assumed a priori. The point here is that the use of elemental metal concentrations to infer toxicity through an oxidative stress response is not correct. This must be rectified in the manuscript.

The authors assert there are policy implications, but is it really a novel finding that aged biomass burning smoke is toxic? There are many publications on the toxicity of smoke to humans (some should have been cited). The main finding here is that cloud process increases the ROS produced by rat macrophage. This specific finding should be stated

in the context of overall known toxicity of smoke. (Ie, the authors could state something along the lines of, smoke is known to be toxic, here we show that fog processing of the smoke, increases the toxicity. . .).

Finally, the last line of the main text states: The enhanced toxicity of fog droplets observed in this study suggests that the historical reduction of fog frequency may result in an unintended improvement of air quality in many continental areas, overlapping also with the deliberate reduction of PM emissions put into practice since the early 90's in many developed countries This assumes that fogs are more effective than other atmospheric processes (eg, aqueous reactions in haze or gas phase oxidation followed by partitioning) in converting wood smoke emissions to species toxic to humans. Is there any evidence for this? The point is what proof do the authors have that if the fogs were not present the aerosol would not still chemically evolve over time to a similar toxicity as fog-processed smoke?

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