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Atmospheric Chemistry and Physics Discussions

Interactive comment on "Relationship between the molecular composition, visible light absorption, and health-related properties of smoldering woodsmoke aerosols" *by* Lam Kam Chan et al.

Anonymous Referee #1

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This manuscript presents comprehensive molecular characterization of smoldering woodsmoke aerosols generated from controlled burning of different hardwood samples. Organic aerosol compositions were correlated with cell toxicity and visible light-absorption measurements to probe their potential health and climate effects. While this manuscript is overall well-written, there are several major concerns regarding the study design that need to be addressed before publication.

(1) The selection of cell type and biological endpoint for toxicity testing does not seem to be relevant to the major route of exposure for woodsmoke aerosol exposure. The authors stated that for the cell toxicity bioassay, human epidermal keratinocytes (i.e., skin

cells) were used to determine the toxicity of WSA, and the protein content from envelope formation was used as a quantitative biomarker from toxicant exposures. However, compared to dermal contact, inhalation should be more relevant. Use of pulmonary cell models to measure cytotoxicity (cell death or cell proliferation) would be more appropriate for this type of research. The authors should provide clear justifications for the usage of cell type and biological endpoint for toxicity testing.

(2) As discussed in section 3.1 (Line 188-201), the authors provided an exposure scenario, stating that a human adult would take ∼2175 breaths to reach the cell toxicity EC50 value of Red Oak smoldering woodsmoke aerosols. Again, since epidermal keratinocytes (i.e., skin cells) were used to determine cell toxicity by measuring the protein content from envelope formation, the EC50 values derived from this assay are not appropriate parameters for an exposure assessment through inhalation. The authors should limit the inhalation-related discussion based on their EC50 values.

(3) The toxicity bioassay was quantified colorimetrically at the wavelength of 495 nm. This wavelength overlaps with the visible brown carbon absorption. Did the author measure the absorption from aerosol sample extracts to account for the potential interference from brown carbon absorption at this wavelength?

(4) Line 158: What were the positive controls used in the cell toxicity bioassay to simulate the maximum activity?

(5) Line 245: The cell toxicity measured by the protein content from envelope formation was not a direct measure of cell death. How did the authors determine whether the WSA extracts kill cells?

(6) The rationale for measuring AhR and ER activity from woodsmoke aerosol exposure should be strengthen. What are the underlying hypotheses?

(7) Throughout the manuscript, a lot of discussions between molecular composition and biological endpoints (e.g., AhR activity) were based on correlations. The authors

should be careful about drawing conclusions from this relationship since correlation does not imply causation. Validation work should be carried out.

(8) For the brown carbon measurement, why did the author measure the visible light only (400-700 nm), but not the full range for tropospheric-relevant radiation (300-700 nm)? Note that most brown carbon constituents absorb light at near-UV wavelengths (300-400 nm) and to a lesser extent visible light.

(9) Line 380-385: What fractions of sinapaldehyde and coniferaldehyde converted to their enol forms in the extract solutions?

(10) Line 386: The authors could check on the pKa of these detected products and compare that to the pH of your solution.

(11) Line 423-425: Does the formation of phenolate occur only in the aqueous solution?

(12) Line 12 and 86: did the author mean to say “hardwood” here?

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