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Atmospheric Chemistry and Physics Discussions

Interactive comment on "Comparative assessment of ecotoxicity of urban aerosol" *by* B. Turóczi et al.

Anonymous Referee #1

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This paper applies a luminescence bioassay directly to ambient and source PM2.5 and PM10 filter samples to evaluate their toxicity. This is a unique approach and I found the results quite interesting. However, the implication that this approach delivers more specificity with respect to linking PM to health effects is exaggerated. While the method seems straightforward, the description in the methods section should be expanded. Please address the following comments.

1) The introductory material, especially on p.2, is not referenced. Please provide some background references for lines 13-15. While it is true that regulations treat PM as a single attribute, the state of the science of source attribution and chemical speciation helps to inform regulators on which emissions sources to control.

2) I don't believe the statement on p.2, lines 19-21 is true. There is a wealth of literature on epidemiological studies whose results are far more specific than implied here, for

example, see the review by Pope and Dockery (JAWMA, 56, 709-742, 2006).

3) What was the background (upwind) contribution, if any, to PM and toxicity to cigarette smoke and biomass samples?

4) Please describe the road dust sampling procedure in more detail. Apparently, a leaf blower was used to resuspend surface dust, which was then collected from the ambient air. Is this correct? Again, what was the contribution of background aerosol to the dust samples?

5) I don't understand how the assay was calibrated or applied. In Kovats et al. (2011), E50 is defined as the percent concentration which causes a 50% reduction in fluorescence. Here, it is defined as an absolute mass. Is the response linear with mass? How is it calibrated? Did the authors expose a fixed level of the bacteria to varying PM masses? Please explain this in greater detail.

6) Why wasn't mass determined gravimetrically in all cases? Was the beta gauge preceded by a PM10 inlet? How much confidence is there that the beta gauge and gravimetric mass is equivalent? Is the relationship constant with variation in chemical composition?

7) Figure 1 demonstrates a good qualitative picture of toxicity by source. However, I don't see how the nearly threefold smaller toxicity of summer versus winter PM can be explained by the difference between diesel and biomass burning toxicity. Since the results are given on a unit mass basis, are we to believe that winter vehicle emissions are more toxic than summer vehicle emissions? Or that the addition of wood smoke, even if it was as concentrated in PM as diesel emissions, made the winter PM three times more toxic?

8) On p.6, the authors attribute lower toxicity at higher mixing heights with admixture with less toxic aerosols. What evidence is there for this? Assuming that the surface emissions were mainly carbonaceous, I would expect aged aerosols to contain more

sulfate and perhaps, in winter, nitrate. Yet the authors state on the bottom of p. 6 that the largest contributor to PM2.5 in summer is SOA and cites Gelencser et al. (2007). However that study only concluded that SOA was a large contributor to total carbon, not PM2.5. Again, how about sulfate in Gelencser et al. (2007)? Why wasn't it measured in this study?

9) The conclusions place a great deal of weight on the relevance of this bioassay to human health effects, which has not yet been demonstrated. While the results are suggestive, I don't yet accept as fact that wood smoke is more toxic than diesel emissions.

Interactive comment on Atmos. Chem. Phys. Discuss., 12, 8533, 2012.