Interactive comment on “Comparative assessment of ecotoxicity of urban aerosol” by B. Turóczki et al.

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Response to the Anonymous Referee #1

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.

As requested by the reviewer, we added a recent reference to this statement. (Pang et al, 2011).

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).
The sentence on p. 2 (19-21) has been rephrased as follows:

Extensive public health studies that establish the link between mass concentrations of PM2.5/PM10 and health problems within the population can be found in the literature (Pope and Dockery, 2006 and references therein). However, little is known about the relative importance of PM from different sources and the effect of seasonality on the toxicity.

We agree with the reviewer that many studies dealt with the toxicity of PM, but as Pope and Dockery put it: ‘One of the biggest gaps in our knowledge relates to what specific air pollutants, combination of pollutants, sources of pollutants, and characteristics of pollutants are most responsible for the observed health effects’. In addition, as we pointed out in the manuscript most studies were related to the assessments of ecotoxicity of organic extracts of aerosol samples. We stated the importance of direct (whole aerosol) testing with additional references as follows:

Due to the complexity of the PM2.5/PM10 chemical composition and the very low quantities available, direct measurements of the hazard posed by the particles are only sparsely available (Steenhof et al., 2011, Soto et al., 2008).

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

Each cigarette smoke sample was collected in a smokers’ room for only 30 minutes. Similarly, sampling time of biomass burning was also 30 minutes. We added to the following sentence to the end of this sub-section:

In the case of emission samples both the conditions of sampling and the short sampling times ensure that the contribution of ambient PM is practically negligible.

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?

As requested by the reviewer we added the following paragraph to the manuscript:

Resuspended road dust samples were collected with a special PM10 sampling unit including a PARTISOL FRM-2000 aerosol sampler operating at a flow rate of 16.7 l min⁻¹ (Turóczi et al., 2012; Gelencsér et al., 2011). A rectangular stainless-steel hood was fixed to the front of a laboratory cart 0.5 cm above the road surface. A leaf blower was connected to the hood through two facing nozzles via a split flexible hose to induce turbulence inside the hood. The hood was connected to an alumina housing containing the R&P PM10 inlet. A PARTISOL-FRM MODEL 2000 sampler collected resuspended PM1–10 samples in a cyclone separator and PM1 samples on filters. The sampling unit was powered with a portable electrical power generator, also mounted on the platform. The sampling times were 2 hours.

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.

In the cited paper (Kováts et al., 2012) filter spots of known surface area were used. They were processed as solid samples to obtain an aqueous suspension. The Ascent Software calculates EC50 as % of the suspension, but as the mass of aerosol on the filter is available from measurements, EC50 can be expressed as absolute mass of aerosol for the given (standardized) volume of the suspension.

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?
The sampling site in Budapest was equipped with a beta gauge monitor preceded by a PM10 inlet operated by the Hungarian Air Quality Monitoring Service. We added the following sentence to the manuscript.

According to the regulatory standard measurements the difference between the PM10 measured by $\beta$-gauge dust monitor and those measured gravimetrically at RH 50 % is less than 5 %.

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?

We now provided a more detailed description of the possible causes of the observed differences as follows:

As the highly ecotoxic fresh biomass burning aerosol of winter PM10/PM2.5 is substituted with SOA in summer, a potential reduction of ecotoxicity may be expected. Due to the higher temperatures in summer there is significantly less condensation of semi-volatile organic compounds many of which are known to be highly ecotoxic. Furthermore, the significantly higher degree of atmospheric mixing in summer reduces the relative share of primary emission particulates and increases the contribution of atmospheric transport. Bioaerosol particles of probably negligible ecotoxicity are also more abundant in summer than in winter. Under dry conditions in summer the contribution of resuspended dust (which is largely inert and has very low specific ecotoxicity) to PM10 can be quite significant. Overall, the combination of these reinforcing factors may account for the significantly higher EC50 values (lower ecotoxicities) found in summer PM10 relative to those in winter.
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?

We agree with the reviewer and modified the objected sentence as follows:

On the contrary, in summer the major source contributor to carbonaceous fraction of the PM2.5 in the region is secondary organic aerosol (SOA) from predominantly biogenic precursors (Gelencsér et al., 2007).

The detailed chemical characterization of PM10 was out of the scope of our study. We agree with the reviewer that the inorganic constituents of the aerosol, which are likely much less ecotoxic than organic compounds, may still be important by affecting the total mass of the PM10. However, it does not seem to a major factor in our case, since a comprehensive recent study on PM10 chemistry in Europe showed that the mass contribution of inorganic salts to total PM10 vary quite little between the different seasons (Putaud et al., 2010).

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.

We agree with the reviewer that this finding is surprising and we also indicated it in our manuscript. We added the following sentence to this section including a newly found relevant reference.

While this finding warrants further studies it is worthy of note that by using a differ-
ent toxicological approach Klippel and Nussbaumer (2007) also found that particles emitted by incomplete biomass burning showed higher toxicity than diesel soot.

Response to the Anonymous Referee #2

There has been alot of research published on both the chronic and acute health effects of PM pollution. This prior research has utilized both epidemiological and toxicological approaches. This manuscript does not cite this literature and the authors make statements (e.g. in abstract “the potential acute effects of PM2.5/PM10 have never been assessed for lack of adequate methodology”) that are factually incorrect.

As requested by the reviewer, the abstract was rephrased as follows and more references were included in the introduction.

Extensive public health studies established the link between mass concentrations of PM2.5/PM10 and health problems within the population. However, little is known about the relative importance of PM from different sources and the effect of seasonality on the toxicity.

What is the relationship between the proposed assay and human health and other toxicology measures?

The Vibrio fischeri bioluminescence inhibition bioassay is relevant only to assess the hazard for ecosystems and not for human recipients. That is why throughout the manuscript the emphasis was put on ecotoxicology and on ecological risk assessment and all implications on human health issues were removed (See also answers to Reviewer 4).

The paper often seems to take the view that their assay is the ultimate indicator (e.g. page 8539 line 25). In reality there are many toxicology tests and my read of the literature is that they provide different answers about what components of PM (diesel versus biomass smoke, fresh versus aged, etc.) is the problem.

We agree with the reviewer that there are numerous indicators for ecotoxicity and hu...
man toxicity, which sometimes yield contradictory results. By no means we wanted to imply that our direct assay would be the ultimate method. Therefore we deleted the following sentence from the text: Any other chronic health effects of urban PM10 aside, this finding is very astonishing and reflects a dim view on urban air quality with all its potential consequences!

Furthermore, we rephrased another sentence as follows:

This finding is perhaps unexpected but definitely should have an impact on future air quality legislation.

This finding is unexpected and warrants further studies which might have an impact on future air quality legislation.

Page 8540 line 18 – “this highly unfavourable effect has never been considered” In the US there is a daily standard that targets the high pollution episodes raised by this concern.

As requested by the reviewer, we changed the objected sentence to:

This highly unfavourable effect is implicitly reflected in air quality standards on PM10/PM2.5 mass concentrations.

Response to the Anonymous Referee #4

1) The choice of the test This bioassay based on V. fisheri bioluminescence inhibition is a normalized test (Microtox) used in ecotoxicology, and more precisely for the impact of pollutants on natural ecosystems (rivers, soils, sea: : :). It is often used to assess pesticides or heavy metals for instance. It could be thus useful to test the ecotoxicity of chemicals present on aerosols towards natural ecosystems as they can be deposited in the environment (wet or dry deposition). However this test is not relevant for assessing the impact on human heath which is the main goal of this paper. Many tests exist which are currently used in pharmacology to assess the toxicity of drugs on human heath, they are based on enzymatic assays, human cells in culture or animal models. I
am convinced that these types of test should be used for aerosols instead of Microtox. The authors should comment on this point and change the objectives and conclusions of the manuscript.

The Vibrio fischeri bioluminescence inhibition bioassay is relevant only to assess the hazard for ecosystems and not for human recipients. The focus of the manuscript is on ecotoxicology and ecological risk assessment. We agree with the reviewer that using the results of a single ecotoxicity test is inadequate to predict human health risks. We rephrased the objected parts to avoid human health implications.

“The previously overlooked acute effects of urban PM10 may add to the established effects of gaseous primary pollutants aggravating health problems during severe air pollution episodes.“

was replaced with

These effects of urban PM10 may be useful supplementary indicators besides the mass concentrations of PM2.5/PM10 in cities.

In the Conclusion the following statement was deleted:

“This effect potentially aggravates the health risks posed by the high ambient concentrations of urban particulate matter.”

The last sentence of the Conclusion was rephrased as follows:

These results refer to the ecotoxicity of the particulates only and definitely not to their potential acute or chronic (carcinogenic, mutagenic, teratogenic etc.) effects on humans.

2) Discussion on the method The test used is usually performed on homogenous liquid phase. Here the authors are using a heterogeneous liquid/solid phase and this might induce some problems concerning the interpretation of the data. Toxic compounds which are at the surface of the particles may have various solubilities, and thus can...
dissolve more or less in the aqueous phase. Only solubilized compounds will enter in contact with the bacteria and will contribute to the bioluminescence inhibition. This could explain some “surprising results” obtained in this paper. P5 line 22: Diesel engine emission samples have higher EC50 Values than biomass some samples. This could be easily explained by the difference of solubilities of very hydrophobic diesel compounds compared to more soluble compounds such as sugars (levoglucosan). This test could reflect the solubility of the compounds in water and not their real toxicity; it could thus give false results. In addition, if human health is considered, the small particules (PM2.5-10) reach the lung cells and can be directly in contact with the cells, hydrophobic molecules can directly penetrate the human cell membrane. In that case the toxicity results could be very different. The authors should comment on these remarks. The authors should also check (at least for some samples) the content of the aqueous phase. They should measure a quick MS fingerprint or measure the Kow value that gives the lipophiliy of the solution. Kow values are indicators of the solubility in tissues. These data could help them to give a more accurate interpretation of their results.

We agree with the reviewer that the exact mechanism of biological exposure in the test system may not be fully understood. Aqueous samples are used during the conventional protocol of the Vibrio fischeri bioluminescence inhibition bioassay (in compliance with ISO 11348-3). For testing the toxicity of solid samples via direct contact between V. fischeri bacteria and particles, Lappalainen et al. (1999, 2001) presented a novel protocol. In their work luminescence intensity is evaluated in kinetic mode. As the bacterial suspension is injected to the sample, the luminous intensity increases to a peak (maximum) within 30 s (that is why the system is called Flash). The results are expressed as the ratio of luminescence at 30s normalized to the peak value.

However, the discussion of solubilities and other potential issues are clearly out of the scope of our manuscript. We rely on the fact that the protocol has been standardised, the ISO standard (ISO 21338:2010: Water quality - Kinetic determination of the in-
hibitory effects of sediment, other solids and coloured samples on the light emission of Vibrio fischeri /kinetic luminescent bacteria test/) was issued in 2010. The Flash system, in fact, uses a suspension of the solid sample without prior extraction. In the suspension bacteria are in direct contact with toxic particles. Previously, we have adapted this protocol for developing a ‘whole-aerosol’ testing procedure (Kováts et al., 2012). In order to treat particulate matter on filters as ‘whole-aerosol’ samples exposed filters were first ground and homogenized then applied as solid samples in the protocol, making direct contact possible for the bacteria. (The term ‘whole-aerosol’ was created after the terms ‘whole-sediment’ ecotoxicity testing or ‘whole-sediment’ sample, referring to the fact that the sample is tested directly without any prior separation or extraction and test organisms are in direct contact with particles).

3) Comparison Summer/Winter samples P6 line 3-16: Winter samples proved to be more ecotoxic than summer ones. The authors interpret the data mainly by the result of photooxidation which is more intense in summertime. However, many other factors could be responsible for these differences. To prove that photooxidation is a main factor, the authors should compare samples collected during the days and the nights of the same period. Alternatively they could perform laboratory experiments where they could expose the collected aerosols to light. Then they could perform the biotests on the photooxidized and non photooxidized particles.

We fully agree with the reviewer that the effect of photooxidation cannot be proven without further experiments. Therefore we deleted the following sentence from the manuscript: ’It might also be that photooxidation reduces the acute effects (ecotoxicity) of emission particulates (and volatile organic compounds) though it cannot be proven by the results of the present study.’


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