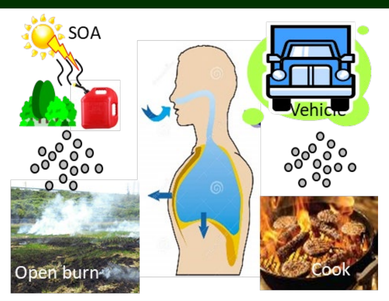
Novel Method for Source Apportionment of Toxicity of Atmospheric Organic Aerosols



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1. Introduction

Fine particulate matter (PM2.5) in the atmosphere is of high priority for air quality management efforts due to observed associations with adverse effects on human health. To examine countermeasures against PM emission, origins of PM2.5 should be elucidated. On the other hand, toxicity per PM2.5 mass can remarkably differ among emission sources or atmospheres in different places. Therefore, it is better to understand source contributions not only to PM2.5 mass but also to PM2.5 toxicity. In this study, we have proposed a new approach to estimate source contributions to various kinds of toxicities by atmospheric organic aerosols (OA), using chemical mass balance model and toxicity data (**Fig. 1**). To see how this approach works, we performed the following studies.

2. Methods

First, we chose four kinds of sources (i.e. secondary organic aerosols: SOA, automobiles, open burning of biomass, and cooking) as possible important OA sources for source apportionment. Then we conducted source testing and collected PM2.5 samples from these sources (**Fig. 2**). We also collected ambient PM2.5 samples in different places (e.g. Ryogoku, Tokyo as an urban site; northern foot of Mt. Fuji as a forest site; and Hedo, Okinawa as a remote site in summer) for evaluating our approach (**Fig. 3**). The chemical composition (i.e. elemental carbon, organic carbon: OC, ionic species, elements, and organic compounds) of the PM2.5 samples were measured. Cytotoxicity and cellular responses to the samples, including oxidative stress (measured with gene expression of heme oxygenase-1 and reporter gene assay for Nrf2), DNA-damage (measured with umu test), inflammation (measured with gene expression of interleukin-8), and aryl hydrocarbon receptor agonist activity, were also quantified (**Fig. 4**).

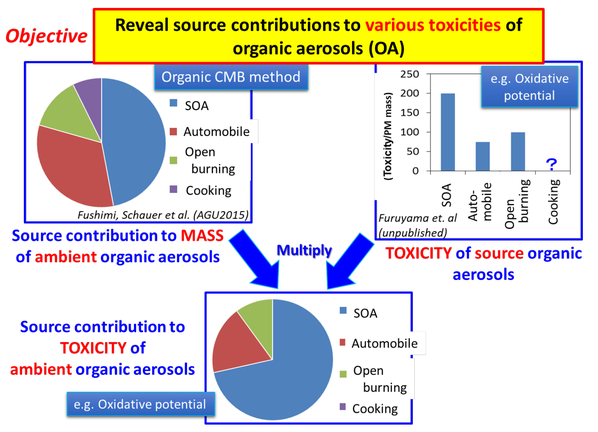

Fig. 1. Objective and approach

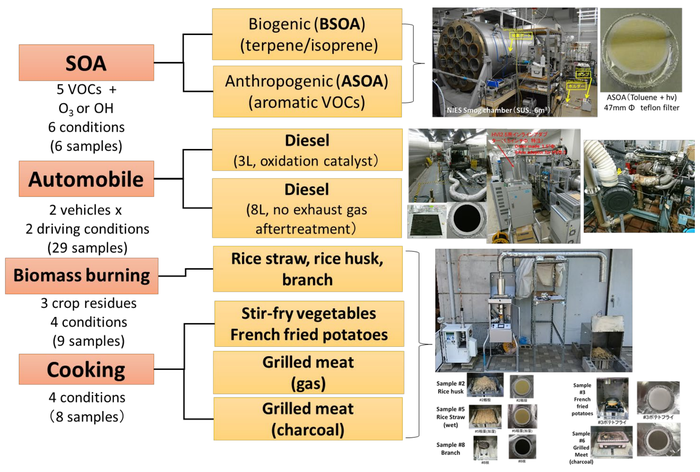

Fig. 2. Target $PM_{2.5}$ sources

Fig. 3. Target ambient $PM_{2.5}$ samples

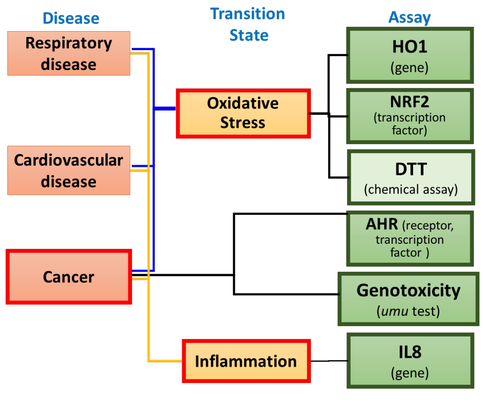

Fig. 4. Target toxicities and assays

3. Results and Discussion

【$PM_{2.5}$ compositions】

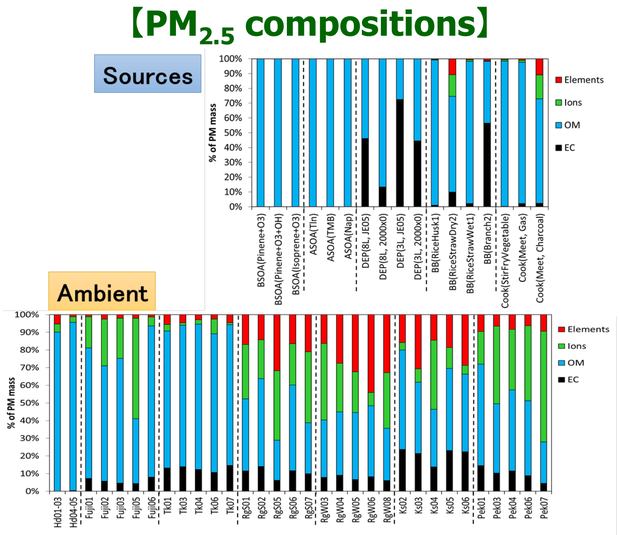

Fig. 5. PM mass and major components of the source and ambient PM2.5 samples

【Source profiles developed】

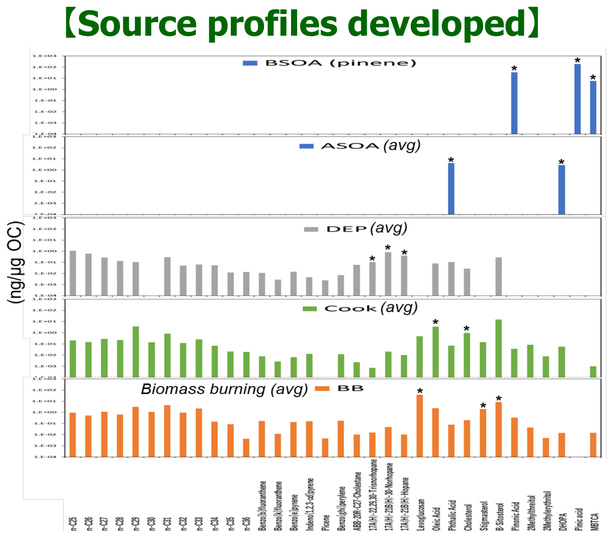

Fig. 6. Examples of new source profiles

【Cellular/acellular responses of source $PM_{2.5}$ samples】

- The cellular/acellular responses per OC were greatly different among atmospheric and source aerosol samples (**Fig. 7**).
- Trends of oxidative potential (HO-1, Nrf2, DTT) and inflammation (IL8) were similar with each other (**Fig. 7-9**).
- BSOA, Cook < Ambient aerosols < BB, DEP, ASOA (Fig. 7-9).
- Naphthalene-SOA showed strongest responses (Fig. 7-8).
- Trends of AHR and DNA-damage (umu) differed remarkably from those of oxidative potential and inflammation (Fig. 7).
- All kinds of cellular/acellular responses of cooking emissions were low consistently (Fig. 7).

【Cellular/acellular responses of ambient $PM_{2.5}$ samples】

- The cellular/acellular responses of remote site samples (Hedo, summer) and forest site samples (Fuji, summer) were consistently low (**Fig. 9**). These results may suggest that the cellular/acellular responses of aged particles and BPOA/BSOA are low.
- Urban and roadside site samples showed stronger cellular/acellular responses (Fig. 9).

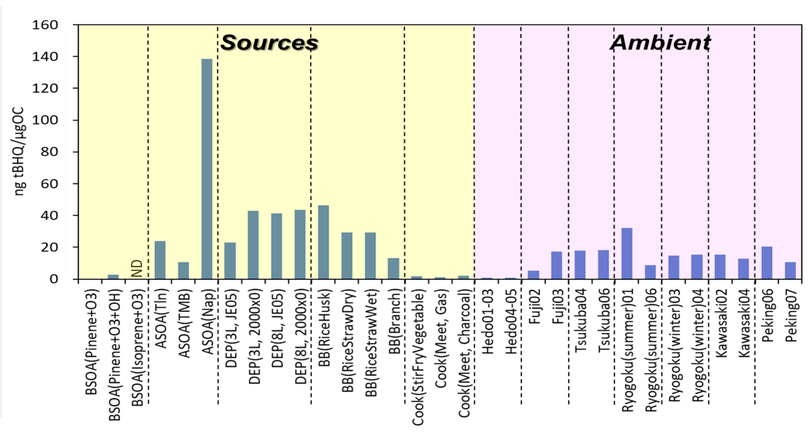

Fig. 8. Oxidative potentials (HO-1) of the source and ambient PM2.5 samples

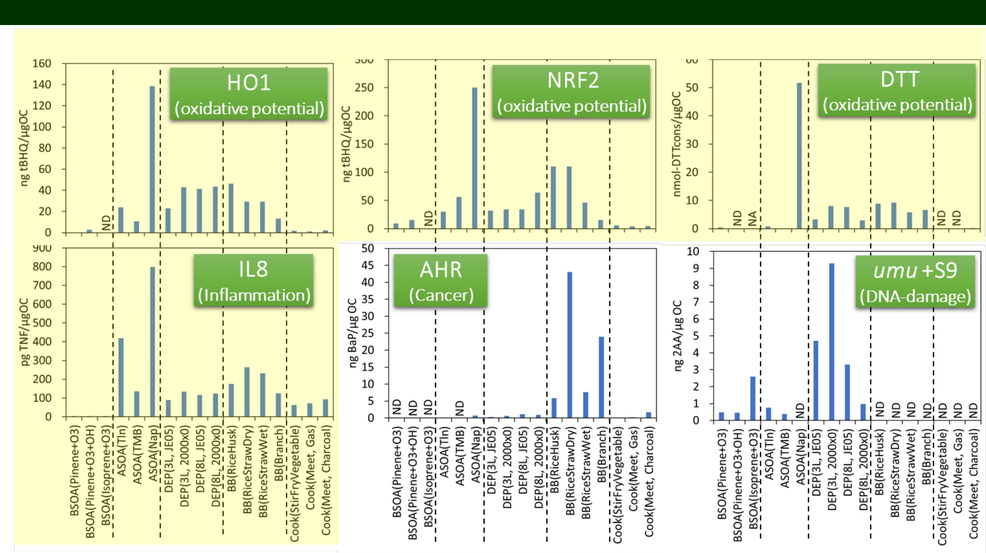

Fig. 7. Cellular/acellular responses of the source PM2.5 samples

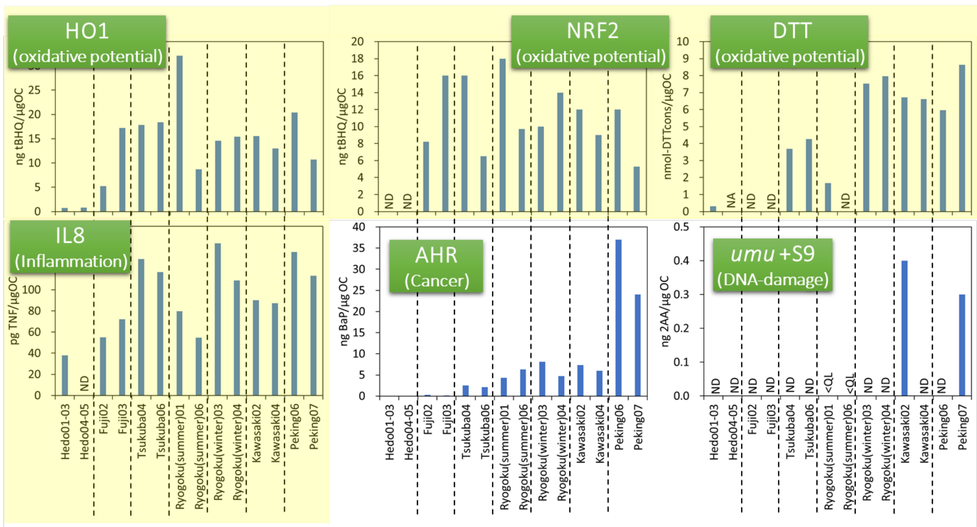

Fig. 9. Cellular/acellular responses of the ambient PM2.5 samples

4. Summary

- We have proposed a new approach to estimate source contributions to various kinds of toxicities by atmospheric organic aerosols (OA), using chemical mass balance model and toxicity data.
- The cellular/acellular responses per OC were greatly different among atmospheric and source aerosol samples.

Acknowledgements

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