Doctoral Dissertation Final Oral Defense

Maiko Arashiro

Understanding the Biological Effects of Isoprene-derived Secondary Organic Aerosol

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Isoprene (2-methyl-1,3-butadiene), a reactive volatile organic compound released primarily by terrestrial vegetation, is an important precursor to the formation of secondary organic aerosol (SOA), which has impacts on climate change and public health. Isoprene-derived SOA, which comprises a large mass fraction of global fine particulate matter ($PM_{2.5}$), results from the atmospheric chemical transformations of isoprene with controllable anthropogenic emissions such as oxides of nitrogen (NO_x) and sulfur dioxide. Because $PM_{2.5}$ from isoprene is a relatively new discovery, little is known about the toxicity of its resultant SOA. Through a series of *in vitro* exposure studies, we explored the effects of isoprene-derived SOA on oxidative stress-related gene expression levels in human bronchial epithelial cells (BEAS-2B). We began by generating atmospherically-relevant compositions of isoprene-derived SOA in an outdoor smog chamber facility, starting from isoprene as a precursor in the presence of NO_x and acidic sulfate aerosol, to expose BEAS-2B cells to the total isoprene SOA air mixture. We then systematically explored the effects of known composition types of isoprene-derived SOA on BEAS-2B cells by generating SOA through dark reactive uptake experiments in an indoor smog chamber facility by starting with key gaseous intermediates, including *trans*-β-isoprene epoxydiol (*trans*-IEPOX), methacrylic acid epoxide (MAE), or isoprene hydroxyhydroperoxides (ISOPOOH).

Chemical characterization coupled with biological analyses show that atmospherically-relevant compositions of isoprene-derived SOA alter the levels of oxidative stress-related gene expression within BEAS-2B cells, with MAE-derived SOA having a stronger influence on gene expression than IEPOX- or ISOPOOH-derived SOA. Importantly, this *in vitro* work reveals that there was an enrichment for altered expression of genes that are transcriptionally controlled by Nuclear factor (erythroid-derived 2)-like 2 (Nrf2). Additional studies are needed to understand the role of the Nrf2 pathway in controlling oxidative stress and other potential downstream biological effects resulting from isoprene SOA exposure. The findings from this initial exploration into the biological effects of isoprene-SOA emphasize the importance of future *in-vitro* and *in-vivo* work to inform policy not only because of the atmospheric abundance of isoprene-derived SOA, but also because the anthropogenic contribution is the only component amenable to control.

Committee:
Jason Surratt (Advisor)
Daniel Costa (Environmental Protection Agency)
Rebecca Fry
Ilona Jaspers
David Leith