

DOCTORAL THESIS

Chemical and toxicological characterization of chemical contaminants in air pollution particulate matter

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**CHEMICAL AND TOXICOLOGICAL CHARACTERIZATION OF
CHEMICAL CONTAMINANTS IN AIR POLLUTION
PARTICULATE MATTER**

BILLAH MD. BAKI

Ph.D. Thesis

HONG KONG BAPTIST UNIVERSITY

2015

Declaration

I hereby declare that this thesis represents my own work which has been done after registration for the degree of Ph.D at Hong Kong Baptist University, and has not been previously included in a thesis or dissertation submitted to this or any other institution for a degree, diploma or other qualifications.

Signature:

Date: August 2015

Abstract

In wintertime hazy episodes, the air pollution in northern China has always reached to an alarming level. In the winter of 2012-13, the trans-boundary air pollution from China has attracted national and global attention. An elevated public awareness to the unprecedented pollution levels has prompted much investigation on the health effects of fine particulate matter (PM), in particular PM_{2.5}. Since PM-elicited harmful effects primarily depends on the composition of chemical contaminants adsorbed, in this study we characterized the chemical compositions of PM_{2.5} and determined its associated toxicity.

Samples of PM_{2.5} were collected using high-volume samplers installed in five northern and southern cities in China. One typical (polycyclic aromatic hydrocarbons, PAHs) and one emerging (perfluorinated compounds, PFCs) family of organic pollutants were analyzed using gas chromatography–mass spectrometry (GC-MS) or liquid chromatography-MS-MS (LC-MS-MS), followed by in-vitro or/and in vivo studies. In Chapters 2 and 3, sixteen PAH congeners in PM_{2.5} samples collected from five different cities (Hong Kong (HK), Guangzhou (GZ), Xiamen (XM), Xi'an (XA) and Beijing (BJ)) in the winter and summer time 2012-13 was analyzed. The biological effects of the sample extracts were determined using the human bronchial epithelial cells BEAS-2B. The total PAH concentrations ranged from 3.35 to 80.45 ng/m³ air, leading by BJ, followed by XA, XM, GZ and HK. In a comparison of the physical and chemical data of the samples obtained during the winter and summer sampling periods, the amount of PM collected per unit time and the concentrations of PAHs adsorbed were found to be remarkably greater in the winter time. In the cell culture study, the expression levels of the pro-inflammatory cytokine (interleukin-6, IL-6) and detoxifying enzymes (i.e. cytochrome p450 enzymes, CYP1A1 and CYP1B1) were found to be stimulated in the treatment. The cells exposed to sample extracts prepared from XA and BJ demonstrated significant migratory activities, indicating a sign of increase of tumorigenicity. These data highlighted the risk of getting lung cancer in local population.

In chapters 4 and 5, we focused on the emerging pollutants PFCs, in particular PFOS. Chemical characterization was implemented using the winter samples.

Biological effects of PFOS were conducted using omics approach in a maternal-fetal model. Therefore, in the first part of chapter 4, we measured the concentrations of nine PFC congeners in PM_{2.5} samples using LC-MS-MS. Generally, the eight PFCs, namely PFOS, PFDoA, PFUdA, PFDA, PFNA, PFOA, PFHxA and PFBA were detected in all the sampling cities, with the exception PFHxS which was below the limit of detection. The total PFC concentrations ranged from 121.2 to 192.2pg/m³, leading by GZ, followed by XA, BJ, XM and HK. The data denoted the risk of inhalation exposure to PFCs through PM_{2.5}, which enter into blood circulations via lung alveoli, presumably penetrates through placenta in affecting fetal health. Therefore, in the latter part of chapter 4, the potential adverse effect of prenatal exposure to the prototypical PFC congener PFOS was used in the maternal-fetal mouse model to determine its effects on fetal liver and pancreas. Transcriptomic analysis demonstrated that the in-utero exposure to PFOS affect the expression of genes associated with fatty acid metabolism, lipid transport, and steroid synthesis in fetal livers. KEGG pathway analysis showed these changes were primarily associated with modulations of arachidonic, linoleic acid, retinol metabolism and PPAR signaling pathways in fetal liver. To identify additional target fetal tissue of PFOS, in chapter 5, we investigated the effects of PFOS on the protein expression in fetal pancreas using the technique of “Isobaric tags for relative and absolute quantitation” (iTRAQ). We identified changes in the protein expressions involved in pancreatic secretion, protein digestion and absorption, protein processing in endoplasmic reticulum, fat digestion and absorption, glycerolipid metabolism and steroid biosynthesis. The perturbations to these targets may increase the risk of pancreatitis in mouse offspring. Collectively, this study provided a comprehensive chemical and biological analysis of PM_{2.5} collected in China and demonstrated the toxicities, *in vitro* and *in vivo* of the adsorbed chemical contaminants.

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