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Air pollution from polycyclic aromatic hydrocarbons generated by human activities and their health effects in China

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ABSTRACT

The key factors that influence the health effects of polycyclic aromatic hydrocarbons (PAH) air emissions generated by human activities in China were identified in this study by considering the emission amounts, environmental fates, exposure, and damage at the national level. Results showed that the most significant substances affecting overall human health included benzo(a)pyrene (BaP), dibenz(a,h) anthracene (DahA), and fluoranthene (Flu), which are mainly generated from aluminum production, biomass indoor burning, and bituminous coal consumption for domestic use. Approximately 10% of the BaP, DahA, and Flu emissions corresponded to over 80% of all recorded human health effects and showed significant positive linear relationships with mortality from malignant tumors, as well as with the nervous system, heart, and cerebral-vascular diseases. Increasing aluminum recycling, biomass utilization, national pipeline gas penetration rate, and national heating coverage rate are effective approaches for reducing the overall health impact generated by PAH pollution.

1. Introduction

Polycyclic aromatic hydrocarbons (PAHs) are believed to cause human cancers (Liu et al., 2008). These compounds are frequently detected in surface water, sediments, soils, and plants. PAHs are mainly emitted from automobile exhaust, tobacco smoke, and industrial emissions (Rodgman et al., 2000; Chang et al., 2006; Hansen et al., 2008) because of the incomplete combustion of hydrocarbons (e.g., coal, oil, wood, and fuels). Human exposure to PAHs mainly occurs through polluted air, water, and food (Al-Saleh et al., 2013). Eighteen PAHs have been listed by the US Environmental Protection Agency (EPA) as priority pollutants to protect public health (Yan et al., 2004) because they are carcinogenic and mutagenic (Vineis and Husgafvel-Pursiainen, 2005; Singh et al., 2007). Several recent studies regarding PAHs have focused on atmospheric emissions (Xu et al., 2006; Zhang and Tao, 2009), epidemiology (Clapp et al., 2008; Olsson et al., 2010), and DNA damage analysis (Gunter et al., 2007; John et al., 2009; Yuan et al., 2013). Xu et al. (2006) established an air emission inventory of PAHs in China on the basis of a literature review and obtained significant uncertainties. Zhang and Tao (2009) developed a global PAH emission inventory on the basis of a literature review and Monte Carlo uncertainty analysis. However, no human toxicity potential (HTP) impact has been analyzed from these inventories. Clapp et al. (2008) and Olsson et al. (2010) investigated the epidemiological evidence linking PAH exposure to various cancers but did not study the emission inventory and environmental fate of PAHs. Gunter et al. (2007), John et al. (2009), and Yuan et al. (2013) examined PAHs that bind to human DNA but did not assess the emission inventories, environmental fates, intake routes, or risks. The aforementioned studies focused only on the individual level and failed to consider emission amounts, environmental fates, exposure pathways, intake routes, HTP risk, and damage at the national level. Therefore, the key factors necessary to improve the effectiveness of PAH pollutant control and disease treatment are difficult to determine because of the complicated relationship between PAH pollutants and their health effects. PAHs as a group of stable chemicals are difficult to predict, particularly in terms of their effect on human health because these effects depend on the intrinsic toxicity, fate, and exposure levels of PAHs (Jolliet et al., 2008). Jolliet et al. (2008) reported that the most abundant PAHs (e.g., Phenanthene, Fluoranthene, and Pyrene) comprise over 50% of the total PAH emissions. However, these PAHs are not necessarily the main contributors to human health. To determine the

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relationship between PAH air emissions and their population health effects, national PAH air emissions, environmental fates, human health effects, and the key factors affecting public health burden in China were investigated. Furthermore, the deoxyribonucleic acid (DNA) damage observed from these key factors was studied.

2. Methods and materials

2.1. Approach for estimating PAH air emissions in China

The PAHs emitted from coal consumption, coke and aluminum production, transportation, crop straw, and firewood burning, which are the main PAH emission sources in China (Xu et al., 2006; Zhang and Tao, 2009), were studied to evaluate the national PAH air pollutants from human activities in China. The inventory of national PAH air emissions from human activities was calculated by using Eq. (1).

$$Q_T = \sum_{R=1}^{n} Q_R = \sum_{R=1}^{n} \sum_{i=1}^{n} A_{i,R} \times E_i \quad (1)$$

where Q_T, Q_R, A, E, R, and i respectively represent the amount of national and provincial PAH air emissions, amount of material consumption from various sources, PAH emission factor, region, and source type.

The notable differences in PAH emission factors caused by variations in water content rate, burning efficiency, oxygen supply condition, fuel type, etc., have been reported (Zhang, 2010). The emission factor of individual PAHs from each source in the present study was measured by Zhang (2010), who developed a database of PAH emission factors through uncertainty analysis (Table S1). The total values of national and provincial coal consumption (i.e., domestic use, electricity generation, industry consumption), metallurgical and indigenous coking, straws (i.e., corn, straw, wheat) and firewood indoor burning, diesel and gasoline consumption for transportation, and electrolytic aluminum were obtained or calculated on the basis of statistical data (Table S2; NBSC, 2004–2012). The national and provincial yields of straw, the ratio of dry residue from crop production, the proportional value of straw burning within a region, and the burning efficiency of straws were obtained from relevant references (NBSC, 2004–2014; China Natural Resources Database; Wang and Zhang, 2008). Only rice, wheat, and corn straw burning were considered in this work because of the lack of information relevant to PAH emissions on other crop straw sources. Furthermore, other crops showed low contributions to overall crop production (NBSC, 2004–2014).

2.2. Methodology for determining human health effects

Human health characterization factors generally represent regional or local levels. A regionalized model for assessing human health effects (e.g., environmental fate, exposure, intake, and toxic information) that can represent the outcomes of pollutants has not yet been developed in China. Therefore, the USEtox method, a method frequently used in the comparative assessment of human toxicity, was used in this study to investigate the effects of environmental exposure. The USEtox model provides a rapid and transparent tool for assessing human health effects by adopting multi-media fate (i.e., home air, industry air, urban air, rural air, freshwater, sea, agricultural soil, and natural soil) and multi-pathway (i.e., air, drinking water, leaf and root crops, meat, dairy products, and fish) models to identify the environmental exposure and toxic effects of pollutants (USEtox, 2010). The USEtox model is currently recommended by the European Union and the US EPA to characterize human health impacts in comparative chemical toxicity assessments.

The characterization factors of 16 individual PAHs (i.e., naphthalene (Nap), acenaphthylene (Aay), acenaphthene (Ace), fluorene (Fluo), phenanthrene (Phe), anthracene (Ant), fluoranthene (Flu), pyrene (Pyr), benzo[a]anthracene (BaA), chrysene (Chr), benzo[b]fluoranthene (BbF), benzo[k]flouranthene (BkF), benzo[a]pyrene (BaP), dibenz(ah)anthracene (DahA), benzo[ghi]perylene (BghiP), and indeno(1,2,3-cd)pyrene (IcdP)) that are commonly detected in China were quantified on the basis of the environmental and population conditions in China by using thefollowing equation:

$$CF_i = \sum_{i=1}^{n} iF_i \times EF_i = \sum_{i=1}^{n} FF_i \times XF_i \times EF_i \quad (2)$$

where CF_i, iF_i, EF_i, XF_i, and FF_i are the human health characterization factors (case/kg-emission), intake fraction (kg-intake/kg-emission), effect factor(case/kg-intake), fate factor (days), and exposure factor ($days^{-1}$) of individual PAH_i, respectively. The intake fractions (iF_i, kg-intake/kg-emission) of identified key individual PAHs in each province of China were calculated on the basis of the USEtox model, which uses national and regional data for calculations. Regional information including national and provincial geographies and environmental conditions (i.e., temperature, land and sea area, soil type, wind speed, rain rate, soil erosion) were used to determine the environmental fate of PAHs (NBSC, 2004–2013). The number of humans and total food (i.e., meat, dairy, freshwater and marine fish) intake in each province of China were used to assess the total population intake of individual PAHs in China. The effect factors (EF_i, case/kg-intake) of identified key individual PAHs were determined by using the following experimental animal data-based equation:

$$EF_i = \frac{0.5}{ED_{50} \times BW \times LT_h \times N_{365}} \quad (3)$$

where ED_{50}, BW, LT_h, and N_{365} represent benchmark dose resulting in 50% human risk of an incidence above background (mg/kg-day), average body weight (70 kg/person), and life time of human (70 years), and number of days per year (365 days/year), respectively. The chemical database and detailed methodology for the USEtox model are available online (http://www.usetox.org). The toxicity database dressed from laboratory studies based on animal experiments for EF_i calculation is available online (http://www.epa.gov/iris). Furthermore, the characterization factor was determined to address the animal–human discrepancy by using the unit risks identified in epidemiology studies (WHO, 2010; 8.7×10^{-5} cases per ng/m^3):

$$CF_i = \sum_{i=1}^{n} EP_i \times UR_i \quad (4)$$

where EP_i, and UR_i is the air exposure concentration (ng/m^3 per kg-emission) and unit risk (cases per ng/m^3)of the individual PAH_i, respectively. Toxicity equivalence factors (TEFs) were employed in this study to determine the ED_{50} and unit risk of PAHs. BaP was used as the reference substance. The TEFs of 16 PAHs were obtained from relevant references (Nisbet and Lagoy, 1992; Van den Berg, 1998; Fang et al., 2002).

PAH air emission inventory was used to determine national and provincial HTP impacts:

$$HT = \sum_{i=1}^{n} HT_i = \sum_{i=1}^{n} CF_i \times E_i \quad (5)$$