Environment International 84 (2015) 7–16

Contents lists available at ScienceDirect

Environment International

journal homepage: www.elsevier.com/locate/envint

Review article

Spatiotemporal analysis and human exposure assessment on polycyclic aromatic hydrocarbons in indoor air, settled house dust, and diet: A review

Yuning Ma, Stuart Harrad *

School of Geography, Earth, and Environmental Sciences, University of Birmingham, Birmingham, B15 2TT, UK

ARTICLE INFO

Article history:
Received 21 August 2014
Received in revised form 1 July 2015
Accepted 2 July 2015
Available online 18 July 2015

Keywords:
Polycyclic aromatic hydrocarbons
Indoor air
Indoor dust
Dietary intake
Temporal and geographical trends

ABSTRACT

This review summarizes the published literature on the presence of polycyclic aromatic hydrocarbons (PAH) in indoor air, settled house dust, and food, and highlights geographical and temporal trends in indoor PAH contamination. In both indoor air and dust, ΣPAH concentrations in North America have decreased over the past 30 years with a halving time of 6.7 ± 1.9 years in indoor air and 5.0 ± 2.3 years in indoor dust. In contrast, indoor PAH concentrations in Asia have remained steady. Concentrations of ΣPAH in indoor air are significantly ($p < 0.01$) higher in Asia than North America. In studies recording both vapor and particulate phases, the global average concentration in indoor air of ΣPAH excluding naphthalene is between 7 and 14,300 ng/m^3. Over a similar period, the average ΣPAH concentration in house dust ranges between 127 to 115,817 ng/g. Indoor/outdoor ratios of atmospheric concentrations of ΣPAH have declined globally with a half-life of 6.3 ± 2.3 years. While indoor/outdoor ratios for benzo[*a*]pyrene toxicity equivalents (BaP_{eq}) declined in North America with a half-life of 12.2 ± 3.2 years, no significant decline was observed when data from all regions were considered. Comparison of the global database, revealed that I/O ratios for ΣPAH (average = 4.3 ± 1.3), exceeded significantly those of BaP_{eq} (average = 1.7 ± 0.4) in the same samples. The significant decline in global I/O ratios suggests that indoor sources of PAH have been controlled more effectively than outdoor sources. Moreover, the significantly higher I/O ratios for ΣPAH compared to BaP_{eq}, imply that indoor sources of PAH emit proportionally more of the less carcinogenic PAH than outdoor sources. Dietary exposure to PAH ranges from 137 to 55,000 ng/day. Definitive spatiotemporal trends in dietary exposure were precluded due to relatively small number of relevant studies. However, although reported in only one study, PAH concentrations in Chinese diets exceeded those in diet from other parts of the world, a pattern consistent with the spatial trends observed for concentrations of PAH in indoor air. Evaluation of human exposure to ΣPAH via inhalation, dust and diet ingestion, suggests that while intake via diet and inhalation exceeds that via dust ingestion; all three pathways contribute and merit continued assessment.

Contents

* Corresponding author.
E-mail address: s.j.harrad@bham.ac.uk (S. Harrad).

http://dx.doi.org/10.1016/j.envint.2015.07.006
0160-4120/

1. Introduction

People spend an average of approximately 90% of their time indoors (Diffey, 2011; Health Canada, 1989; U.S. Environmental Protection Agency, 1989). Indoor occupants potentially have contact with pollutants generated from building materials, electronics, toys, furniture, carpets, paints, household chemicals (e.g. glues, detergents, insecticides), and domestic combustion activities (e.g. cooking, heating, smoking) (CDC, 2013). Outdoor pollutants can also enter through infiltration and ventilation depending on home insulation conditions and ventilation frequencies (CDC, 2013).

Prominent amongst the wide variety of contaminants reported as present in the indoor environment (WHO, 2010; Butte and Heinzow, 2002; Farré and Barceló, 2013), polycyclic aromatic hydrocarbons (PAH) are a group of organic pollutants with two or more aromatic rings fused in linear (e.g. anthracene), angular (e.g. phenanthrene), cluster (e.g. triphenylene), and cyclic (e.g. coronene) arrays. Some PAH cause cancer, birth defects, mutations, and immune system disruption (Srogi, 2007) and have even been reported to correlate negatively with gross domestic product (GDP) and gross domestic income (GDI) (Zhang and Tao, 2009). Special attention has fallen recently on pregnant women and children, who spend more time indoors than average, showing adverse fetal growth, miscarriage and prematurity due to the PAH and other indoor contaminations (Patelarou and Kelly, 2014).

While natural sources (e.g. forest fires) of PAH exist, anthropogenic sources predominate, comprising activities such as incomplete fuel combustion for space heating and traffic, waste incineration, and other high temperature industrial/chemical processes (coke ovens, aluminium production, anode baking, mineral oils, tars, and creosote) (U. S. Environmental Protection Agency, 2008). All of those anthropogenic PAH may transport and ingress to the indoor environment. Also the heating, burning, and cooking activities indoors may directly elevate the PAH contaminations indoors (U. S. Environmental Protection Agency, 2008).

Depending on factors such as the vapor pressure, a proportion of the indoor burden of airborne contaminants, such as PAH, may exist in the vapor phase, with the remainder partitioning to suspended or resuspended particulates as well as dust deposited to room surfaces (settled dust) (Harrad et al., 2010). Thus, inhalation of air (both gas and particle phases) and ingestion of settled house dust (SHD) are two prominent pathways of human exposure to such indoor contaminants as PAHs (Harrad et al., 2010). Additional exposure may occur via contact with outdoor air and soil, and also the ingestion of diet. Contamination of food with PAH may occur during its production (e.g. accumulation by shellfish and plants from the surrounding ambient environment), subsequent processing/preservation (e.g. smoking of fish), and cooking (e.g. barbequeing and broiling etc.) (CDC, 2013).

While the published literature on PAH contamination of the indoor environment is substantial, hitherto there has been little systematic attempt to analyze temporal and geographical trends in such contamination, to evaluate the significance of human exposure via contact with indoor dust as well as inhalation, and to place this in line with dietary exposure. Thus, the review aims to fill the knowledge gap on the occurrence and distribution of PAH in the indoor environment.

2. Strategy of the review

In this study, we review critically the data on PAH concentrations in indoor air and settled dust published between the beginning of 1984 and January 2014, as well as that available on human dietary exposure published between the 1970s and January 2014. All the data were obtained from peer-reviewed journals, conference proceedings, and official reports from government agencies. Key words of "polycyclic aromatic hydrocarbons" combined with "indoor air", "indoor dust", and "total dietary" were searched in SciFinder® interface. For evaluation of temporal and geographical trends, data were classified by sampling year, and country from which samples originated. All data from studies used in this review were averaged and treated as observations for further statistical analysis. Specifically, the global or regional averages and ranges cited here are the average and range of the average values reported by each study. As a consequence of this approach, two studies based on analysis of e.g. 10 and 100 samples respectively will contribute equally to the global or regional average. Moreover, the numbers and identities of PAH reported varies between studies, with the result that studies reporting a greater number of more prevalent PAH will tend to influence disproportionally the global or regional average ΣPAH value. These necessarily introduce elements of uncertainty into our interpretations of the ΣPAH database. We believe the impact of the first of these factors is relatively minor for indoor air, for which more studies are available, but will impact more on regional averages and indoor dust and diet for which the database is smaller. To reduce uncertainty due to the second of these factors, we also calculate benzo[*a*]pyrene equivalent concentrations, recognizing that the PAH used to calculate this metric are not identical in each study.

Temporal analysis was conducted via linear regression of the log-transformed concentration versus sampling year. Where a study collected samples over more than one year, we used the middle year for purposes of regression analysis. If the samples were collected in two consecutive years, we used the year during which most samples were collected. In instances where the year of sampling was not provided, we assumed samples were collected 2 years prior to the publication date.

Spatial analysis was performed via t-tests with the assumption that the averages from the literature accurately represent a normally distributed population and each study contributes equal weight to the combined regional dataset.

3. Results and discussions

3.1. Concentrations of PAH in indoor air

We found a total of 35 studies (a sum of 1545 samples) reporting concentrations of PAH associated with both vapor and particulate phases in indoor air as shown in Table 1. Studies reporting concentrations of PAH associated only with indoor suspended airborne particulates were excluded, as such data have been reviewed elsewhere (Delgado-Saborit et al., 2011) and because reporting particulate phase concentrations only, underestimates atmospheric PAH concentrations.

While the number of PAH monitored is roughly similar (average of 18 PAH commonly reported) in all studies reviewed, there are some important variations between studies. Naphthalene, which originates mostly from moth repellents, toilet deodorant blocks, and polyvinyl chloride (PVC) (Jia and Batterman, 2010), accounts for approximately 50% of the airborne concentration of ΣPAH (vapor + particle phases). However, naphthalene levels were not reported in 8 of the total 35 studies. Moreover, reported naphthalene concentrations may depend on the sampling media employed. Naphthalene has a greater affinity for XAD-2 resin than polyurethane foam (PUF) — the predominant sorbents used to retain vapor phase PAH (Chuang et al., 1987). As approximately one-third of the studies reviewed used only PUF as the vapor phase sorbent, the data provided in these instances likely underestimate the overall airborne concentration of naphthalene. To improve comparability between studies, we thus report ΣPAH concentrations in indoor air excluding naphthalene. Concentrations of ΣPAH in indoor air thus adjusted, range globally from 7 to 14,300 ng/m^3 with an average of 1124 ± 449 ng/m^3. As indicated above in Section 2, these values are the range and average of the averages reported in the 35 studies considered.

It is also common practice to express concentrations of complex mixtures of PAH in terms of their overall carcinogenicity. To do so, concentrations of each PAH monitored are multiplied by a potency equivalency factor (PEF) that expresses the carcinogenic potency of that PAH