

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

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24 bally with a half-life of  $6.3 \pm 2.3$  years. While indoor/outdoor ratios for benzo[*a*]pyrene toxicity  
25 equivalents (BaP<sub>eq</sub>) declined in North America with a half-life of  $12.2 \pm 3.2$  years, no significant  
26 decline was observed when data from all regions were considered. Comparison of the global da-  
27 tabase, revealed that I/O ratios for  $\Sigma$ PAH (average =  $4.3 \pm 1.3$ ), exceeded significantly those of  
28 BaP<sub>eq</sub> (average =  $1.7 \pm 0.4$ ) in the same samples. The significant decline in global I/O ratios  
29 suggests that indoor sources of PAH have been controlled more effectively than outdoor sources.  
30 Moreover, the significantly higher I/O ratios for  $\Sigma$ PAH compared to BaP<sub>eq</sub>, imply that indoor  
31 sources of PAH emit proportionally more of the less carcinogenic PAH than outdoor sources.  
32 Dietary exposure to PAH ranges from 137 to 55000 ng/day. Definitive spatiotemporal trends in  
33 dietary exposure were precluded due to relatively small number of relevant studies. However, al-  
34 though reported in only one study, PAH concentrations in Chinese diets exceeded those in diet  
35 from other parts of the world, a pattern consistent with the spatial trends observed for concentra-  
36 tions of PAH in indoor air. Evaluation of human exposure to  $\Sigma$ PAH via inhalation, dust and diet  
37 ingestion, suggests that while intake via diet and inhalation exceeds that via dust ingestion; all  
38 three pathways contribute and merit continued assessment.

39

40 **Keywords:** polycyclic aromatic hydrocarbons, indoor air, indoor dust, dietary intake, temporal  
41 and geographical trends

42

### 43 **1. Introduction**

44 People spend an average of approximately 90% of their time indoors (Diffey, 2011;  
45 Health Canada, 1989; U.S. Environmental Protection Agency, 1989). Indoor occupants poten-  
46 tially have contact with pollutants generated from building materials, electronics, toys, furniture,

47 carpets, paints, household chemicals (e.g. glues, detergents, insecticides), and domestic combus-  
48 tion activities (e.g. cooking, heating, smoking) (CDC, 2013). Outdoor pollutants can also enter  
49 through infiltration and ventilation depending on home insulation conditions and ventilation fre-  
50 quencies (CDC, 2013).

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

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

68 Depending on factors such as the vapor pressure, a proportion of the indoor burden of  
69 airborne contaminants, such as PAH, may exist in the vapor phase, with the remainder partition-

70 ing to suspended or re-suspended particulates as well as dust deposited to room surfaces (settled  
71 dust) (Harrad et al., 2010). Thus, inhalation of air (both gas and particle phases) and ingestion of  
72 settled house dust (SHD) are two prominent pathways of human exposure to such indoor conta-  
73 minants as PAHs (Harrad et al., 2010). Additional exposure may occur via contact with outdoor  
74 air and soil, and also the ingestion of diet. Contamination of food with PAH may occur during its  
75 production (e.g. accumulation by shellfish and plants from the surrounding ambient environ-  
76 ment), subsequent processing/preservation (e.g. smoking of fish), and cooking (e.g. barbequing  
77 and broiling etc.) (CDC, 2013).

78 While the published literature on PAH contamination of the indoor environment is sub-  
79 stantial, hitherto there has been little systematic attempt to analyze temporal and geographical  
80 trends in such contamination, to evaluate the significance of human exposure via contact with  
81 indoor dust as well as inhalation, and to place this in line with dietary exposure. Thus, the re-  
82 view aims to fill the knowledge gap on PAH's occurrence and distribution in the indoor envi-  
83 ronment.

84

## 85 **2. Strategy of the review**

86 In this study, we review critically the data on PAH concentrations in indoor air and set-  
87 tled dust published between the beginning of 1984 and January 2014, as well as that available on  
88 human dietary exposure published between the 1970s and January 2014. All the data were ob-  
89 tained from peer-reviewed journals, conference proceedings, and official reports from govern-  
90 ment agencies. Key words of “polycyclic aromatic hydrocarbons” combined with “indoor air”,  
91 “indoor dust”, and “total dietary” were searched in SciFinder<sup>®</sup> interface. For evaluation of tem-  
92 poral and geographical trends, data were classified by sampling year, and country from which

93 samples originated. All data from studies used in this review were averaged and treated as ob-  
94 servations for further statistical analysis. Specifically, the global or regional averages and ranges  
95 cited here are the average and range of the average values reported by each study. As a conse-  
96 quence of this approach, two studies based on analysis of e.g. 10 and 100 samples respectively  
97 will contribute equally to the global or regional average. Moreover, the numbers and identities of  
98 PAH reported varies between studies, with the result that studies reporting a greater number of  
99 more prevalent PAH will tend to influence disproportionately the global or regional average  
100  $\Sigma$ PAH value. These necessarily introduce elements of uncertainty into our interpretations of the  
101  $\Sigma$ PAH database. We believe the impact of the first of these factors is relatively minor for indoor  
102 air, for which more studies are available, but will impact more on regional averages and indoor  
103 dust and diet for which the database is smaller. To reduce uncertainty due to the second of these  
104 factors, we also calculate benzo[*a*]pyrene equivalent concentrations, recognising that the PAH  
105 used to calculate this metric are not identical in each study.

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

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

115

## 116 **3. Results and discussions**

### 117 **3.1 Concentrations of PAH in indoor air**

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

123 While the number of PAH monitored is roughly similar (average of 18 PAH commonly  
124 reported) in all studies reviewed, there are some important variations between studies. Naphtha-  
125 lene, which originates mostly from moth repellents, toilet deodorant blocks, and polyvinyl chlo-  
126 ride (PVC) (Jia and Batterman, 2010), accounts for approximately 50% of the airborne concen-  
127 tration of  $\Sigma$ PAH (vapor + particle phases). However, naphthalene levels were not reported in 8  
128 of the total 35 studies. Moreover, reported naphthalene concentrations may depend on the sam-  
129 pling media employed. Naphthalene has a greater affinity for XAD-2 resin than polyurethane  
130 foam (PUF) – the predominant sorbents used to retain vapor phase PAH (Chuang et al., 1987).  
131 As approximately one-third of the studies reviewed used only PUF as the vapor phase sorbent,  
132 the data provided in these instances likely underestimate the overall airborne concentration of  
133 naphthalene. To improve comparability between studies, we thus report  $\Sigma$ PAH concentrations in  
134 indoor air excluding naphthalene. Concentrations of  $\Sigma$ PAH in indoor air thus adjusted, range  
135 globally from 7 to 14300 ng/m<sup>3</sup> with an average of 1124 ± 449 ng/m<sup>3</sup>. As indicated above in sec-  
136 tion 2, these values are the range and average of the averages reported in the 35 studies consi-  
137 dered.

138 **Table 1.** A summary of average PAH concentrations (ng/m<sup>3</sup>) in indoor air by country and year.

Location	Sampling Year	Media <sup>a</sup>	N <sup>b</sup>	# PAH	ΣPAH (ng/m <sup>3</sup> )	BaP <sub>eq</sub> (ng/m <sup>3</sup> )	Reference
OH, USA	1986-1987	QFF, XAD-2	10	15	165	1.30	Chuang et al., 1991
NC, USA	1989	QFF, XAD-2	20	15	390	0.78	Wilson et al., 1989
Burundi	1993	GFF, other	16	12	14300		Viau et al., 2000
Chicago, USA	1994-1995	QFF, XAD-2/PUF	45	17	229	0.41	Van Winkle and Scheff, 2001
NC, USA	1994-1995	QFF, XAD-2	25	18	280	1.87	Chuang et al., 1999
Taiwan	1995	GFF, XAD-2	14	15	196	6.35	Li and Ro, 2000
Taiwan	1996	GFF, PUF	6	21	4299	133.66	Lin et al., 2002
Shizuoka, Japan	1996	Millipore filter, XAD-2	5	12	59	2.34	Zhu et al., 1997
NC, USA	1997	QFF, XAD-2/PUF	13	20	99	0.23	Wilson et al., 2003
NC, USA	1997	QFF, XAD-2	10	19	242	0.19	Wilson et al., 2001
Hangzhou, China	1999	GFF, XAD-2	16	12	3802	37.0	Liu et al., 2001
CA, USA	1999-2000	QFF, PUF	61	27	31	0.15	Naumova et al., 2002
NJ, USA	1999-2000	QFF, PUF	51	27	57	0.25	Naumova et al., 2002
TX, USA	1999-2000	QFF, PUF	45	27	55	0.10	Naumova et al., 2002
Fuji, Japan	1999-2000	GFF, XAD-2	41	39	455	1.10	Ohura et al., 2002
Hangzhou, China	2000	GFF, XAD-2	10	12	8328	198.55	Zhu and Wang, 2003
Chicago, USA	2000-2001	QFF, XAD-2/PUF	115	16	36	0.44	Li et al., 2005
Taiwan	2000-2001	TFF, PUF	8	16	1248		Lung et al., 2004
Shimizu, Japan	2000-2001	GFF, XAD-2	27	39	220	0.50	Ohura et al., 2004
Quebec, Canada	2002 <sup>c</sup>	QFF, XAD-2	12	17	280	0.35	Sanderson and Farant, 2004
Yunnan, China	2002-2004	QFF, PUF	9	16	1646	165.22	Lv et al., 2009
Sweden	2003	QFF, PUF	23	22	28	0.57	Gustafson et al., 2008
Lancaster, UK	2003	GFF, PUF	7	15	132	0.49	Halsall et al., 2008
Kuwait	2004	PUF	24	15	7	0.80	Gevao et al., 2007
Hong kong, China	2005 <sup>c</sup>	GFF, PUF/XAD-4/PUF	6	22	492	4.15	Chen et al., 2007
New York, USA	2005-2010	QFF, PUF	301	16	61	0.37	Jung et al., 2010
CA, USA	2006	QFF, XAD-2	50	9	31	0.02	Rudel et al., 2010
Hangzhou, China	2006	TFF, XAD-2	22	15	1080	7.72	Lu et al., 2008
Hangzhou, China	2006-2007	TFF, XAD-2	104	16	715	7.00	Zhu et al., 2009



Agra, India	2006-2007	TFF, XAD-2	10	23	1021	29.38	Masih et al., 2010
Agra, India	2006-2007	TFF, XAD-2	20	23	856	14.83	Masih et al., 2012
Atlanta, USA	2007	QFF, XAD/PUF	88	17	25	0.08	Li et al., 2010
Krakow, Poland	2007	QFF, PUF	74	9	21	4.95	Choi et al., 2008
Hangzhou, China	2007-2010	GFF, XAD-2	61	14	356	4.77	Lv and Zhu, 2013
Alaska, USA	2009	PUF	8	20	45	0.01	Gouin et al., 2010
Porto, Portugal	2009	TFF, PUF	38	16	37	4.80	Castro et al., 2011
Kaunas, Lithuania	2011-2012	GFF, XAD-2	150	15	253		Krugly et al., 2014
<b>Range:</b>			<b>5-301</b>	<b>9-39</b>	<b>7-14300</b>	<b>0.01-199</b>	
<b>Overall average:</b>			<b>42 ± 9</b>	<b>18 ± 1</b>	<b>1124 ± 449</b>	<b>19 ± 8</b>	

a. QFF: quartz fibre filter; GFF: glass fibre filter; TFF: Teflon fibre filter; PUF: polyurethane foam.

b. N: sample size.

c. the year of sampling was not provided, we assumed samples were collected 2 years prior to the publication date.

139 It is also common practice to express concentrations of complex mixtures of PAH in  
140 terms of their overall carcinogenicity. To do so, concentrations of each PAH monitored are mul-  
141 tiplied by a potency equivalency factor (PEF) that expresses the carcinogenic potency of that  
142 PAH relative to benzo[*a*]pyrene, which is assigned an arbitrary PEF of 1. The overall carcinoge-  
143 nicity of a PAH mixture (referred to as benzo[*a*]pyrene equivalents - BaP<sub>eq</sub>) is thus the sum of  
144 the concentrations of each PAH multiplied by the relevant PEF. Delgado-Saborit et al. reviewed  
145 three major existing PEF schemes and discussed the strengths and limitations of each (Delgado-  
146 Saborit et al., 2011). In this study, we apply one set of PEFs (Nisbet and Lagoy, 1992) of PAH  
147 employed by Delgado-Saborit et al. to calculate BaP<sub>eq</sub> as shown in equation 1:

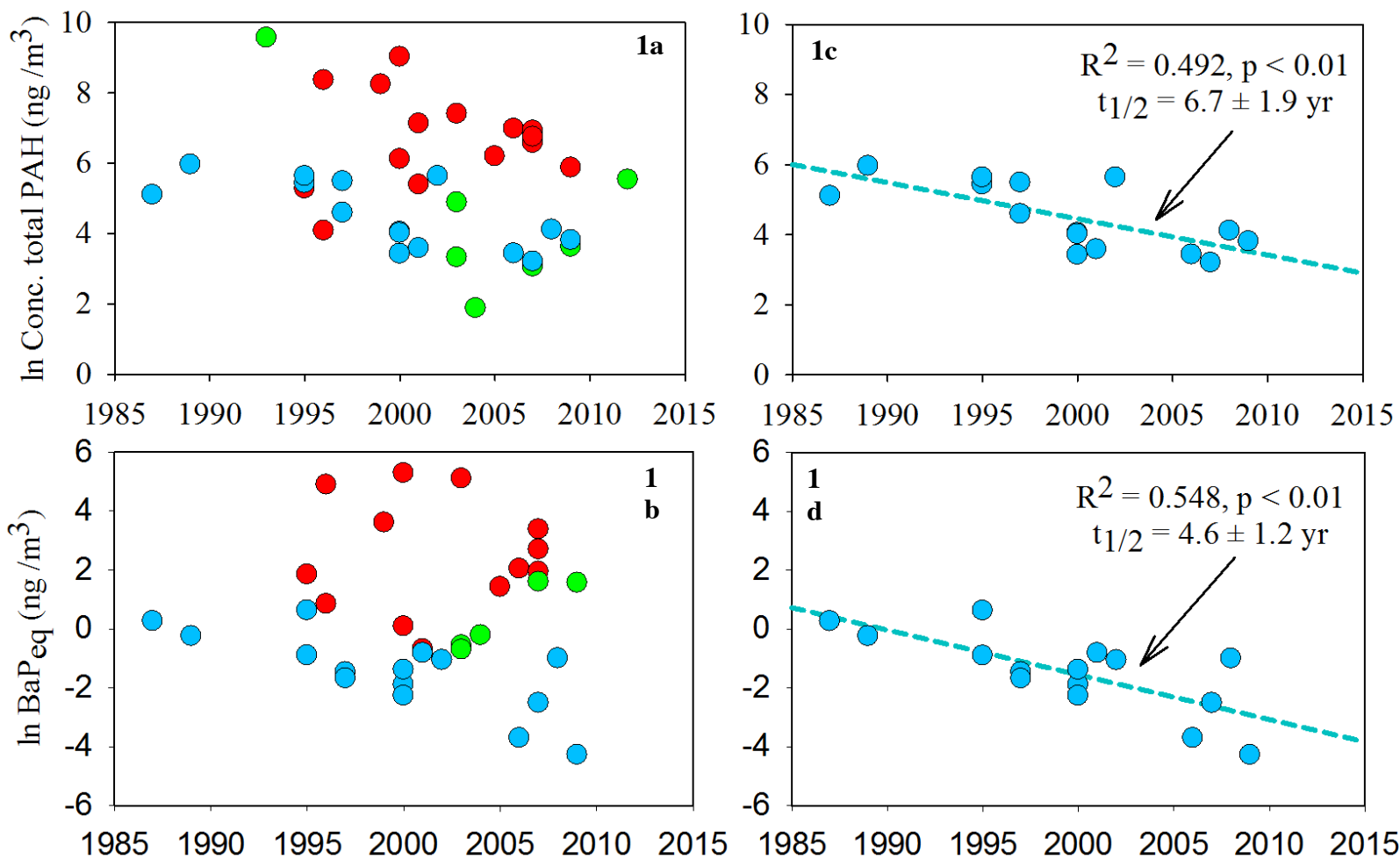
$$\text{BaP}_{\text{eq}} = 0.001 \times (\text{Acy} + \text{Ace} + \text{Fle} + \text{Phe} + \text{Fla} + \text{Pyr}) + 0.01 \times (\text{Ant} + \text{Chr} + \text{BghiP}) \\ + 0.1 \times (\text{BaA} + \text{BbF} + \text{BkF} + \text{IcdP}) + \text{BaP} + \text{DahA} \quad (1)$$

149 where Acy, Ace, Fle, Phe, Fla, Pyr, Ant, Chr, BghiP, BaA, BbF, BkF, IcdP, and DahA denote  
150 acenaphthylene, acenaphthene, fluorene, phenanthrene, fluoranthene, pyrene, anthracene, chry-  
151 sene, benzo[*g,h,i*]perylene, benzo[*a*]anthracene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, in-  
152 deno[*1,2,3-c,d*]perylene, and dibenzo[*a,h*]anthracene respectively. Across the 35 indoor air stu-  
153 dies reviewed here, BaP<sub>eq</sub> concentrations ranged from 0.01 to 199 ng/m<sup>3</sup> with an average of 19 ±  
154 8 ng/m<sup>3</sup>. In the same studies, the concentration of B[*a*]P was between 0.04 and 118 ng/m<sup>3</sup> with  
155 an average of 12 ± 5 ng/m<sup>3</sup>. The average relative carcinogenic potential of B[*a*]P to BaP<sub>eq</sub> was  
156 48 ± 3 % with a wide range from 20 to 86 %. Only 12 of the 35 studies in this review reported  
157 all 15 PAH listed in equation 1, while the rest reported between 10 and 14 of the 15 PAH. Con-  
158 sidering the difficulties in estimating concentrations of such not reported PAH, we assumed  
159 those PAH as contributing zero concentrations for the purposes of our BaP<sub>eq</sub> calculations.

160 Using these data, we examined the temporal trend in PAH contamination of indoor air  
161 over the period of this review. To do so, we conducted linear regression of the natural logarithm

162 of  $\Sigma$ PAH and of BaP<sub>eq</sub> in each study in Table 1 against the year of sampling. Overall, no signifi-  
163 cant linear regression trend was observed when the entire dataset was examined. However, as  
164 the level and pattern of PAH contamination may vary between different areas, we divided our  
165 dataset into three geographical areas: North America, Asia, and elsewhere. We then subjected  
166 these three separate databases to the same linear regression approach used to examine the full  
167 global database. In North America, for which 15 data points were available,  $\Sigma$ PAH concentra-  
168 tions have decreased significantly ( $p < 0.01$ ) with a half-life of  $6.7 \pm 1.9$  years over the period  
169 reviewed (Fig. 1). In the same region, concentrations of BaP<sub>eq</sub> decreased at a slightly faster rate  
170 ( $t_{0.5} = 4.6 \pm 1.2$  years (Fig. 1)). In contrast, concentrations of both  $\Sigma$ PAH and BaP<sub>eq</sub> in Asian  
171 countries ( $n = 15$ ) displayed no significant temporal trend. Elsewhere in the world, there were  
172 too few data to support substantial conclusions on temporal trends. Notwithstanding this, it is  
173 noteworthy that the  $\Sigma$ PAH concentration recorded in Burundi, Africa in 1993 was the highest  
174 found at  $14300 \text{ ng/m}^3$ , with the authors attributing this very high concentration to poor ventila-  
175 tion of fumes from wood combustion employed as the major energy source (Viau et al., 2000).  
176 Sufficient data were available to permit comparison of concentrations of both  $\Sigma$ PAH and BaP<sub>eq</sub>  
177 in North America with Asia. Owing to the positively skewed distribution of both datasets, we  
178 log-transformed concentrations before conducting a t-test comparison. This revealed concentra-  
179 tions of  $\Sigma$ PAH were significantly higher in Asia than in North America (t-value = 5.29,  $p$ -value <  
180 0.01) as well as those of BaP<sub>eq</sub> (t-value = 6.53,  $p$ -value < 0.01).

181

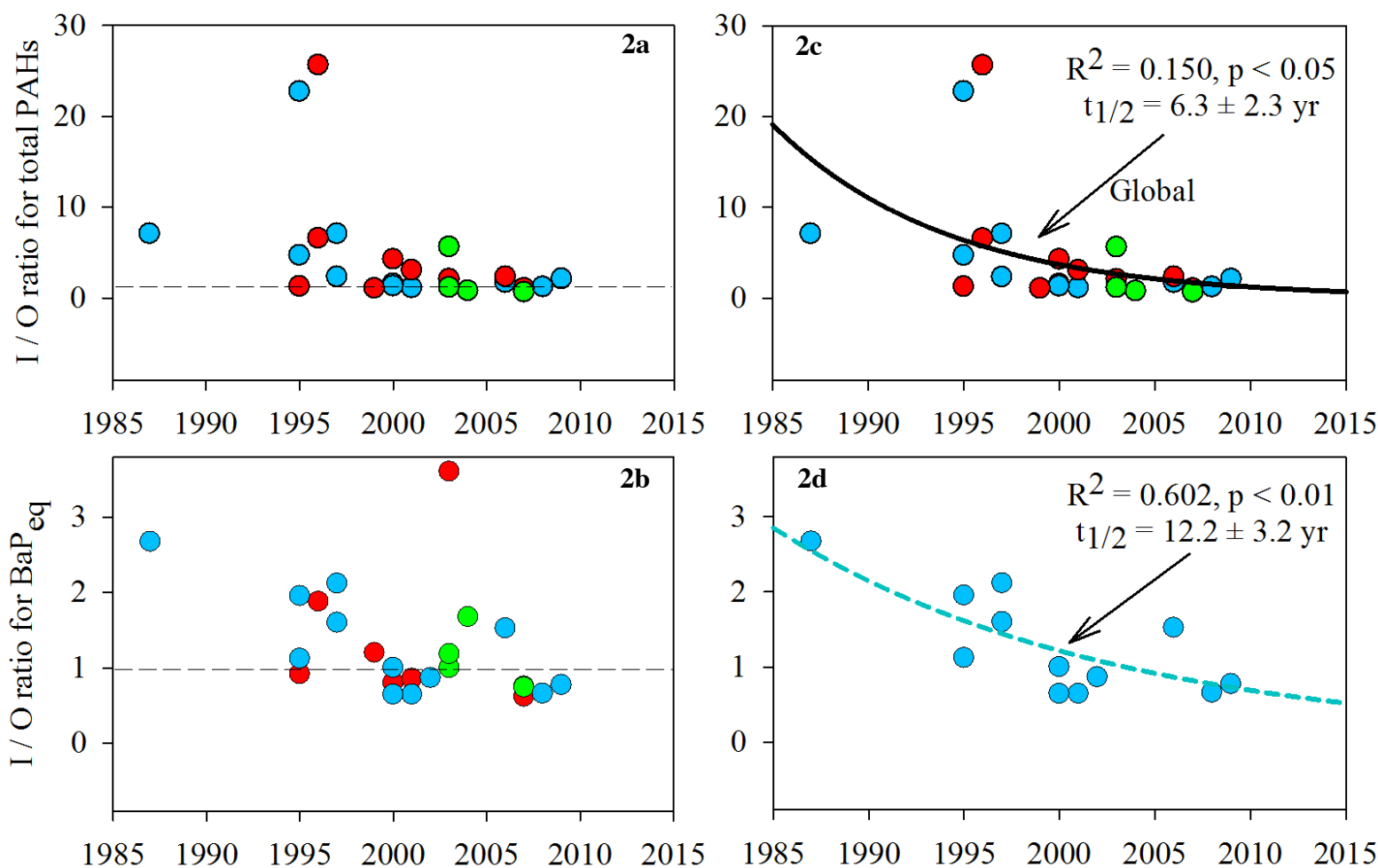


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**Fig. 1.** Natural log-transformed concentrations of  $\Sigma$ PAH (1a) and BaP<sub>eq</sub> (1b) in indoor air as a function of the year (1c and 1d, respectively) in which the sample was collected. The blue color indicates samples from North America, red from Asian countries, green from elsewhere. The blue dashed lines indicate the regression line obtained for North American data

187 A total of 27 studies of indoor air, also reported PAH concentrations in co-located out-  
188 door air samples, thereby permitting the calculation of indoor/outdoor (I/O) concentration ratios  
189 for both  $\Sigma$ PAH and BaP<sub>eq</sub>. As the I/O ratios provide an indicator of the relative importance of in-  
190 door and outdoor sources, we examined temporal trends in these ratios for both  $\Sigma$ PAH and BaP<sub>eq</sub>  
191 (Fig. 2). While the I/O ratios for  $\Sigma$ PAH displayed a clear decline with a halving time of  $6.3 \pm 2.3$   
192 years ( $p < 0.05$ ) across the full global dataset; no significant decline in such ratios was observed  
193 when the three regions were analyzed individually. This is likely due to the small datasets in-  
194 volved –  $n = 10, 13,$  and  $4$  for Asia, North America, and elsewhere respectively. In contrast,  
195 while examination of I/O ratios for BaP<sub>eq</sub> revealed no significant temporal decline when the full  
196 global dataset was analysed; for the 13 studies reporting BaP<sub>eq</sub> I/O ratios in North America, such  
197 ratios declined with a half-life time of  $12.2 \pm 3.2$  years ( $p < 0.01$ ). Comparison of the global da-  
198 taset, revealed that I/O ratios for  $\Sigma$ PAH, (average =  $4.3 \pm 1.3$ ), exceeded significantly (paired t-  
199 test on log-transformed I/O ratios,  $t$ -value =  $3.97$ ,  $p$ -value  $< 0.01$ ) those of BaP<sub>eq</sub> (average =  $1.7 \pm$   
200  $0.4$ ) in the same samples. Combined, these observations concerning I/O ratios suggest that over  
201 the period covered by this review: (a) indoor sources of PAH have been controlled more effec-  
202 tively than outdoor sources because of the significant decline in global I/O ratios, and (b) indoor  
203 sources of PAH emit proportionally more of the less carcinogenic PAH than outdoor sources ow-  
204 ing to the significantly higher I/O ratios for  $\Sigma$ PAH compared to BaP<sub>eq</sub>.

205



206  
207

208 **Fig. 2.** I/O ratio values for  $\Sigma$ PAH (2a) and BaP<sub>eq</sub> (2b) in indoor air as a function (first-order exponential decay) of the year (2c and 2d,  
209 respectively) in which the sample was collected. The blue color indicates samples from North America, red from Asian countries,  
210 green from elsewhere. The black dashed line represents an I/O ratio of 1. The black straight line relates to the regression analysis  
211 conducted on all data points. The blue dashed line relates to the regression analysis conducted only on the data points from North  
212 America indicated in blue.

### 213 **3.2 PAH concentrations in settled indoor dust**

214 PAH in settled house dust (SHD) represent an exposure hazard with the potential for  
215 human exposure to occur via hand to mouth behavior and subsequent oral ingestion, as well as  
216 via dermal contact. Table 2 summarizes the 35 studies (a sum of 3005 samples) reporting con-  
217 centrations of PAH in indoor dust. A major issue encountered when comparing these studies is  
218 variability in both the sampling methods employed and the dust particle size fractions subjected  
219 to analysis. As indicated in Table 2, while American Society for Testing and Materials (ASTM)-  
220 recommended high-volume small surface samplers (HVS3) were the sampling method of choice  
221 in early studies; household vacuum cleaners have found increasing use more recently. A compar-  
222 ison of the two sampling methods concluded that the household vacuum cleaner can be a cheaper,  
223 easier to use, and acceptable replacement for the HVS3 (Colt et al., 1998; Colt et al., 2008).

224 In 21 out of the 35 studies reviewed, the particle size cut-off points were either 150  $\mu\text{m}$  or  
225 63  $\mu\text{m}$ . Driver et al. suggested particles exceeding 150  $\mu\text{m}$  in size do not easily and efficiently  
226 adhere to hands or skins and are thus less relevant in the context of exposure via ingestion or  
227 dermal pathways (Driver et al., 1989). Chuang et al. explained that 150  $\mu\text{m}$  was generally se-  
228 lected according to the ASTM procedure D 5438-93 and a 62  $\mu\text{m}$  cut-off was chosen for sam-  
229 pling in sandy locations (particles  $>63 \mu\text{m}$  are more likely of sandy composition) (Chuang et al.,  
230 1997a). Lewis et al. reported the distribution of PAH concentrations between different particle  
231 size ranges (Lewis et al., 1999).  $\Sigma\text{PAH}$  concentrations were: 2.1  $\mu\text{g/g}$  on particles  $< 500 \mu\text{m}$ , 2.6  
232  $\mu\text{g/g}$  on  $< 150 \mu\text{m}$  particles, 3.1  $\mu\text{g/g}$  on  $< 53 \mu\text{m}$  particles, and 10.1  $\mu\text{g/g}$  on particles  $<4 \mu\text{m}$   
233 (Lewis et al., 1999). Taking this concentration distribution into consideration, alongside the ob-  
234 servation that the largest contributions to overall dust mass were from particles between 53 and  
235 106  $\mu\text{m}$  and those  $< 25 \mu\text{m}$ ; the authors concluded that while for most samples collecting the

236 <150 or <500  $\mu\text{m}$  fraction is acceptable, a < 53 or 63  $\mu\text{m}$  (depending on the sieving technology)

237 particle size may be more appropriate for samples containing a high proportion of sand.

238



239 **Table 2.** A summary of average PAH concentrations (ng/g) in indoor dust by country and year.

Location	Sampling Year	Sampler <sup>a</sup>	N <sup>b</sup>	Particle Size	# PAHs	ΣPAH (ng/g)	BaP <sub>eq</sub> (ng/g)	Reference
OH, USA	1992-93	HVS3	24	<150 μm	19	115817	15530	Chuang et al., 1993; Chuang et al., 1995
WA, USA	1992-93	HVS3	9	<150 μm	16	10249	1235	Chuang et al., 1994
TX, USA	1993	HVS3	15	<53 μm	15	1715	198 <sup>c</sup>	Mukerjee et al., 1997
NC, USA	1994	HVS3	24	<150 μm	19	4200	439	Chuang et al., 1999
NC, USA	1995	HVS3	4	<150 μm	19	3936	421	Chuang et al., 1997a
MD, USA	1995-96	HVS3	126	<150 μm	11	81190	12169	Egeghy et al., 2005; U.S. Environmental Protection Agency, 2011
KY, USA	1995-96	HVS3	3	<150 μm	19	3034	327	Chuang, 1996
NC, USA	1996	HVS3	13	<150 μm	19	3230	286	Chuang et al., 1997b
NC, USA	1996	VC	25	<150 μm	10	20100	3268	Lewis et al., 1999
AZ, USA	1996	HVS3	22	<62 μm	19	1769	200	Chuang et al., 1997b
NC, USA	1997	HVS3	10	<150 μm	19	2729	351	Wilson et al., 2001
NC, USA	1997	HVS3	13	<150 μm	19	2180	267	Wilson et al., 2003
MI/IA/CA/WA, USA	1998-2000	VC	616	<150 μm	7	8570	2103	Camann et al., 2002
MA, USA	1999	VC	6	<150 μm	2	5810	3191	Rudel et al., 2001
Berlin, Germany	1997-98, 2000 <sup>e</sup>	VC	123	fine dust	18	6140 <sup>c</sup>	485	Fromme et al., 2004
MA, USA	1999-2001	VC	120	<150 μm	4	5761	1680	Rudel et al., 2003
Ottawa, Canada	2002-2003	VC	51	<150 μm	13	29300	4724	Maertens et al., 2008
Brisbane, Australia	2003	NA	11	<1 mm	14	7440	106	Ayoko et al., 2005
Warsaw, Poland	2003-04	VC	48	<150 μm	16	35030	2389	Tatur et al., 2009
Kuwait	2004	VC	24	<63 μm	15	540	162	Gevao et al., 2007
CA, USA	2003-05	HVS3	68	<150 μm	9	384 <sup>c</sup>	63	Whitehead et al., 2012
Shanghai, China	2005	HB	27	NA	16	20674	4393	Ren et al., 2006
Palermo, Italy	2006	HB	45	NA	16	5111	262	Mannino and Orecchio, 2008
CA, USA	2005-07	HVS4	132	NA	16	810 <sup>c</sup>	78	Hoh et al., 2012
CA, USA	2001-07	HVS3/VC	583	<150 μm	9	425 <sup>c</sup>	72	Whitehead et al., 2011

CA, USA	2001-07, 2010 <sup>f</sup>	VC	494	<150 µm	12	857 <sup>c</sup>	71	Whitehead et al., 2013
TX, USA	2008	HVS3	23	<500 µm	16	73521	7449	Mahler et al., 2010
Brazil	2008	HB	9	NA	16	4091	288	Coronas et al., 2013
Hong Kong, China	2008 <sup>d</sup>	VC	55	<100 µm	16	6070	635	Kang et al., 2010
Delta State, Nigeria	2009	HB	30	NA	17	127	NR <sup>g</sup>	Iwegbue, 2011
Guangzhou, China	2010	VC	20	<100 µm	16	5916	788	Wang et al., 2013a
CO, USA	2010	VC	3	<63 µm	15	3358	126	Anders et al., 2012
Greece	2010	VC	11	<62 µm	16	397	19	Christopoulou et al., 2012
Cambridge, UK	2010	VC	1	<63 µm	15	5095	345	Anders et al., 2012
Shanghai, China	2010	HB	22	NA	16	11575	829	Peng et al., 2012
Pearl Delta, China	2010	VC	55	<100 µm	16	5910	934	Kang et al., 2011
Guangzhou, China	2011-2012	VC	70	<100 µm	16	8130	843	Wang et al., 2013b
Qingyang, China	2011-2012	VC	70	<100 µm	16	34800	3446	Wang et al., 2013b
<b>Range:</b>			<b>1-616</b>		<b>2-19</b>	<b>127-115817</b>	<b>19-15530</b>	
<b>Overall average:</b>			<b>79±24</b>		<b>15±1</b>	<b>14105±4025</b>	<b>1897±552</b>	

a. HVS3: high volume small surface sampler; VC: vacuum cleaner; HB: hand brushing; NA: not available.

b. N: sample size.

c. Medians instead of averages.

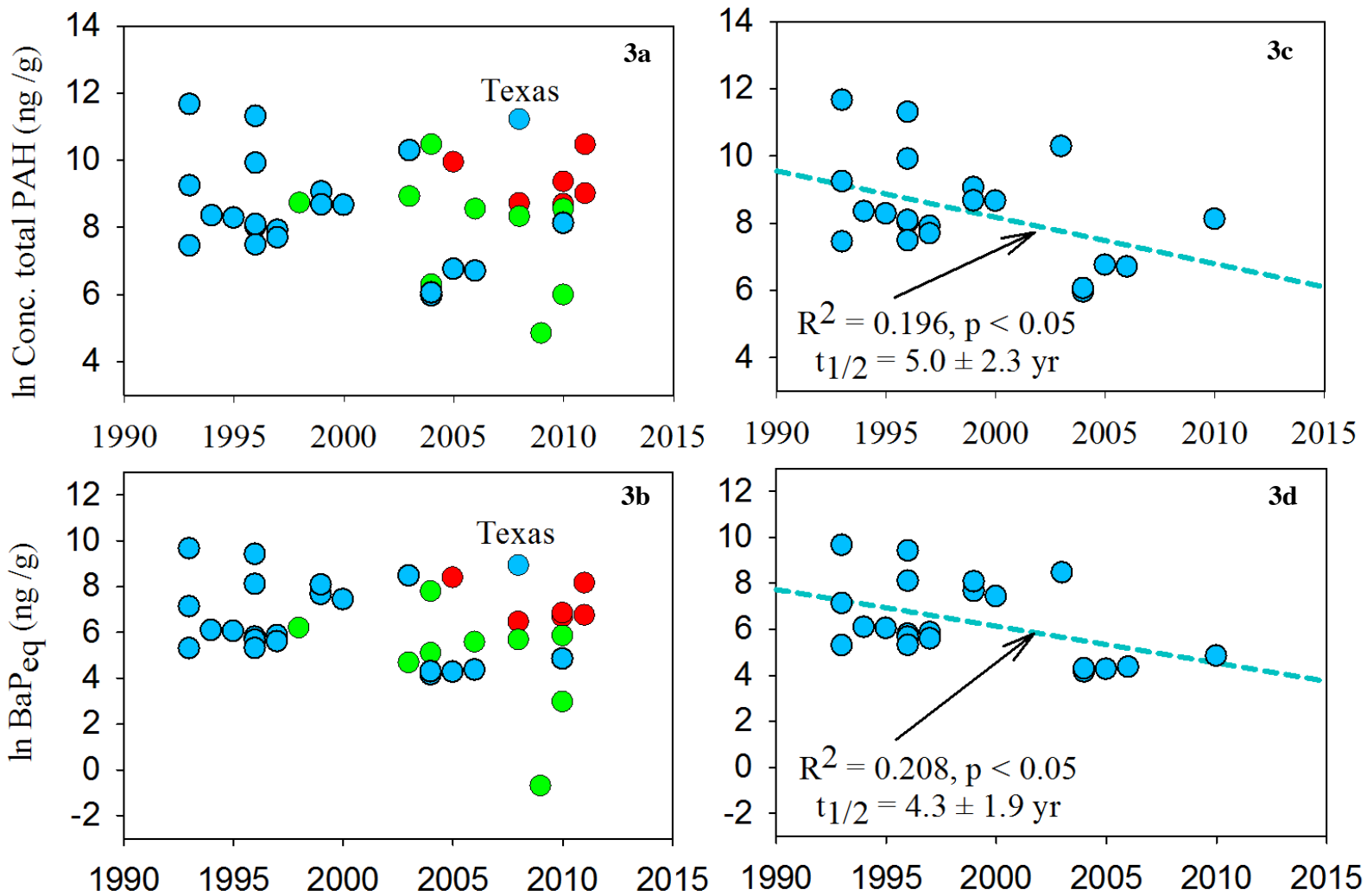
d. the year of sampling was not provided, we assumed samples were collected 2 years prior to the publication date.

e. year 1999 was used for regression analysis.

f. year 2005 was used for regression analysis.

g. NR = not reported

240           The global average total PAH concentration in residential dust reported in the 35 studies  
241 reviewed, is  $14105 \pm 4025$  ng/g with a range of 127 to 115817 ng/g. In the same studies,  $BaP_{eq}$   
242 concentrations range from 19 to 15530 ng/g with a global average of  $1897 \pm 552$  ng/g. Fig. 3  
243 shows the global temporal trend of both  $\Sigma PAH$  and  $BaP_{eq}$  in indoor dust. Regression analysis re-  
244 vealed no significant global temporal trend in either  $\Sigma PAH$  or  $BaP_{eq}$ . Using a similar approach to  
245 that used above to interpret the database on concentrations of PAH in indoor air, we examined  
246 temporal trends in concentrations of PAH in dust from Asia, North America, and elsewhere sepa-  
247 rately (Fig. 3). While no significant trend was observed for other areas, concentrations of  $\Sigma PAH$   
248 and  $BaP_{eq}$  in indoor dust declined significantly ( $p = 0.044$  and  $0.038$ ) in North America with half-  
249 lives of  $5.0 \pm 2.3$  years and  $4.3 \pm 1.9$  years, respectively. Interestingly, concentrations of  $BaP_{eq}$  in  
250 indoor dust are declining with a half-life that is consistent with the half-life observed for the de-  
251 cline of the same parameter in indoor air ( $4.9 \pm 1.3$  years). Also pertinent, concentrations of PAH  
252 in indoor dust are significantly higher in Asia than elsewhere (excluding North America due to  
253 limited data points for comparisons) in studies conducted between 2005 and 2011 (natural loga-  
254 rithm transformed concentrations,  $t$ -value = 3.69,  $p$ -value = 0.004) with 7 studies for Asia and 8  
255 for elsewhere. More data for PAH in indoor dust from all regions is needed to confirm the exis-  
256 tence and magnitude of this apparent “Asian high-rise”. A noteworthy anomaly is the highly ele-  
257 vated concentrations of PAH in dust from homes in Texas, USA located close to parking lots  
258 treated with coal-tar-based sealcoat, a known PAH source (Mahler et al., 2010; Van Metre et al.,  
259 2009). While of interest, this data point (represented as a blue circle in Fig. 3) was treated as an  
260 outlier and excluded from half-life calculations.



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**Fig. 3.** Natural log-transformed concentrations in indoor dust of  $\Sigma$ PAH (3a) and BaP<sub>eq</sub> (3b) as a function of the year (3c and 3d, respectively) in which the sample was collected. The blue color indicates samples from North America, red from Asian countries, green from elsewhere. The blue dashed line was from regression analysis on the data points in blue color (North America).

### 266 3.3 Current knowledge of PAH sources in indoor air and dust

267 Four PAH ratios, Ant/(Ant+Phe), Fla/(Fla+Pyr), BaA/(BaA+Chr), and IcdP/(IcdP+BghiP)  
268 (Yunker et al., 2002), are commonly used as indicators of whether the PAH in a sample originate  
269 from pyrogenic or petrogenic sources. Pyrogenic PAH arise from incomplete combustion while  
270 petrogenic PAH are associated with fossil fuels, petroleum, and oil products (Yunker et al.,  
271 2002). Fig. S1, Table S1, and Table S2 show all four ratios for both indoor air and dust samples.  
272 The average ratios for Ant/(Ant+Phe), Fla/(Fla+Pyr), and BaA/(BaA+Chr) in both indoor dust  
273 and air exceed 0.10, 0.50, and 0.35 respectively; indicating the origins of such PAH to be pyro-  
274 lytic (Fraser et al., 1998). In contrast, the average IcdP/(IcdP+BghiP) ratios (0.44 and 0.43 in in-  
275 door air and dust respectively – Tables S1 and S2) in the same samples were more in line with  
276 those associated with petrogenic sources such as liquid fossil fuel (vehicle and crude oil) com-  
277 bustion (between 0.20 and 0.50; Fraser et al., 1998). Combined, interpretation of these source in-  
278 dicator ratios, suggests indoor PAH arise from a complex mixture of sources, including both  
279 combustion for space heating and cooking, as well as ingress of outdoor emissions from traffic  
280 and fossil fuel combustion.

281 A number of papers have reported on the relative contributions of different putative  
282 sources of PAH in indoor environments (Bhargava et al., 2004; Mannino and Orecchio, 2008; Li  
283 et al., 2010; Masih et al., 2010; Li et al., 2005; Liu et al., 2001; Van Winkle and Scheff, 2001).  
284 For example, Li et al. reported a significant positive correlation between house age and the con-  
285 centration of PAH in indoor air and attributed this to re-volatilization of PAH emitted in the past  
286 and reversibly sorbed by a variety of surfaces. The same authors also discussed the possible as-  
287 sociations of elevated indoor concentrations of PAH, with cooking activities and proximity to  
288 outdoor sources like traffic and industry (Li et al., 2005). The potential role of cooking emissions

289 is underlined by Mannino et al., who reported kitchen dust to display the highest PAH concentra-  
290 tions in their study of indoor dust from a range of microenvironments (Mannino and Orecchio,  
291 2008). In summary, while the relative importance of specific source activities varies between re-  
292 gions (e.g. coal-tar-based pavement sealcoat in America, Mahler et al., 2010), there is consensus  
293 about the importance of biomass burning, smoking, ingress of outdoor contamination, and cook-  
294 ing as sources of indoor PAH.

295 To sum up, in Asia, traffic emission, cooking methods, and biomass burning are major  
296 PAH sources (Qi et al., 2014; Bhargava et al., 2004); while in North America, gas heating, resi-  
297 dence age, outdoor generated PAH infiltration, coal-tar-based parking lot sealcoat, and environ-  
298 mental tobacco smoke are prevailing contributions to indoor PAH levels (Whitehead et al., 2011;  
299 Mahler et al., 2010; Hoh et al., 2012). A very limited number of studies have explored PAH  
300 source changes over time. Policy interventions to reduce traffic emissions (Jung et al., 2014)  
301 may provide one plausible explanation for the decreasing trend we observed in North America.

302

### 303 **3.4 Dietary intake of PAH**

304 PAH enter food via uptake from air, soil, and water, as well as through cooking and pre-  
305 servation processes that involve combustion (grilling, smoking, roasting, etc.) (WHO, 2005). As  
306 a result, they are found in a wide range of comestibles, including: cereals, mollusks, crustaceans,  
307 bread, and vegetable oils (Veyrand et al., 2013). Given this, dietary exposure to PAH is influ-  
308 enced strongly by individual eating choices (Purcaro et al., 2013). Assessment of dietary expo-  
309 sure at a population level is commonly conducted via the total diet study approach, where con-  
310 centrations in representative samples of foodstuffs are combined with data on consumption rates  
311 for different population sectors (EFSA, 2011). Overall, 12 studies of dietary exposure to PAH

312 were found that used such an approach (Table 3). In the 1970s and 80s, average US and UK  
313 adult dietary intakes of  $\Sigma$ PAH were 3000 and 3700 ng/day respectively (Menzie et al., 1992;  
314 Dennis et al., 1983), lower than the 5220 ng  $\Sigma$ PAH/day reported for the Netherlands in 1984-86  
315 (De Vos et al., 1990). In more recent years, total dietary intakes in the UK, Netherlands, and Ita-  
316 ly (summarized in Table 3), vary widely between 1415-4140 ng  $\Sigma$ PAH/day, while intakes in  
317 Spain of between 6720 and 12040 ng  $\Sigma$ PAH /day have been recorded (Falcó et al., 2003; Martí-  
318 Cid et al., 2008; Martorell et al., 2010). The elevated exposure figures for Spain may be at least  
319 partly attributable to the fact that in each of the three Spanish studies concerned; data points be-  
320 low detection limits were assumed for the calculation of exposure estimates to equal half the de-  
321 tection limit – which may overestimate exposure if actual concentrations in food are well below  
322 detection limits. Another possible explanation relates to the food categories included in the re-  
323 spective dietary studies. For example, EFSA only studied dried and processed fruit while the  
324 three Spanish studies included several types of fresh fruit.

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345 **Table 3.** A summary of total dietary intakes of ΣPAH (ng/day) by country and sampling year.  
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Country	Sampling year	ΣPAH <sup>a</sup>	Ref.
US	1970s	3000	Menzie et al., 1992
US	2001	20-120 <sup>b</sup>	Kazerouni et al., 2001
UK	1983	3700	Dennis et al., 1983
UK	2000	4140	Food Standards Agency, 2002
UK	2008	1415	EFSA, 2008
Netherlands	1984-86	5220	De Vos et al., 1990
Netherlands	2008	1785	EFSA 2008
Italy	1995	3000	Lodovici et al., 1995
Italy	2008	1962	EFSA 2008
Spain	2000	8420	Falcó et al., 2003
Spain	2006	12040	Martí-Cid et al., 2008
Spain	2008	6720	Martorell et al., 2010
France	2006-2007	137	Veyrand et al., 2013
France	2008	1814	EFSA 2008
China	2010	55000	Zhang et al., 2014

347 a. ΣPAH includes at least 8 PAHs.

348 b. This study reported B[a]P only.

349

350 The European Food Safety Authority (EFSA) recently suggested two suites of 8  
 351 (BaA,Chr, BbF, BkF, BaP, DahA, BghiP, IcdP = PAH<sub>8</sub>) and 4 PAH (BaA, Chr, BbF, BaP =  
 352 PAH<sub>4</sub>) as better indicators of PAH contamination of food than B[a]P alone (EFSA 2008). In Sep-  
 353 tember 2012, the European Commission introduced regulatory limits for concentrations of both  
 354 B[a]P and PAH<sub>4</sub> in various foods (European Commission Regulation, 2011). A self-consistent  
 355 meta-analysis conducted by EFSA in 2008 of 9714 analyses of PAH in food from 16 European  
 356 countries showed average European dietary intake for a 60 kg adult was 1163 ± 32 ng/day for  
 357 PAH<sub>4</sub> and 1726 ± 47 ng/day for PAH<sub>8</sub> (EFSA 2008). The EFSA meta-analysis suggests that  
 358 when consistent exposure assessment methodology is applied, there appears no substantial varia-  
 359 tion between dietary exposures to PAH reported for different European countries – see Table 3,  
 360 2008 estimates for France, Italy, Netherlands, and the UK. Globally, the highest dietary intake of  
 361 approximately 55000 ng/day was recorded in China in 2010 (Zhang et al., 2014).

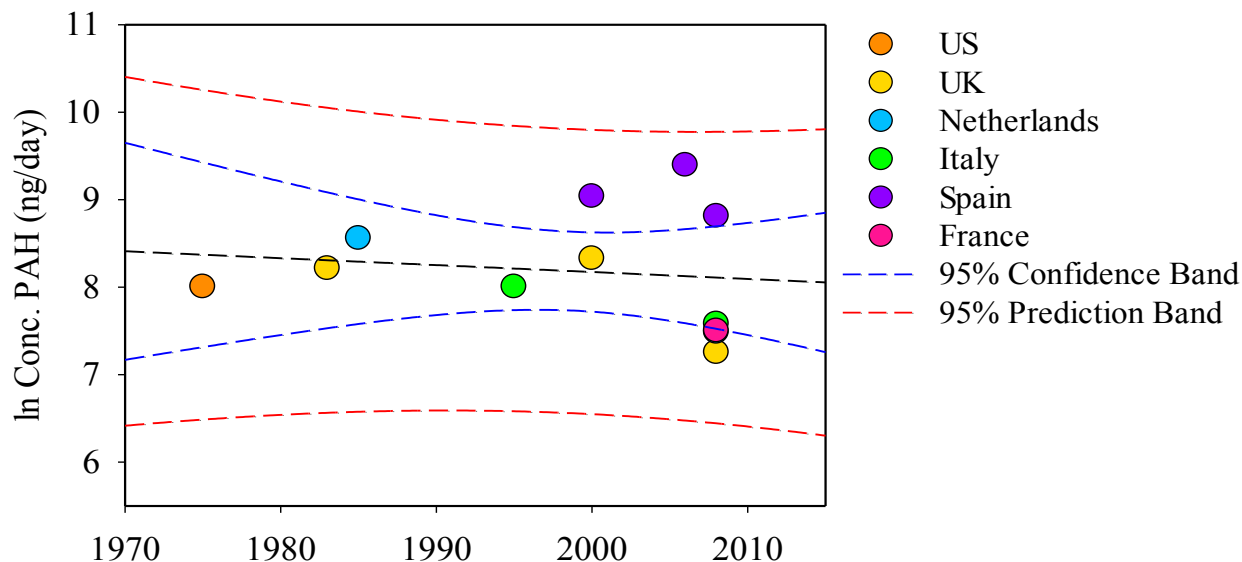


362 While the published literature is too small and geographically disparate to permit defini-  
363 tive analysis of temporal trends, in general, dietary intakes of ΣPAH appear to be slowly decreas-  
364 ing with time over the period of this review as shown in Figure 4. Three studies (Kazerouni et al.,  
365 2001; Veyrand et al., 2013; Zhang et al., 2014) were treated as outliers and thus excluded in the  
366 linear regression in Figure 4. With the limitation on the number of available data, this trend is  
367 not statistically significant ( $p = 0.67$ ), although all the regression data were within the 95% con-  
368 fidence or prediction band.

369 To illustrate, the exposure estimates provided for 2008 in the EFSA meta-analysis for the  
370 Netherlands, the UK and Italy; all appear lower than those reported for the same countries in  
371 previous decades (Dennis et al., 1983; De Vos et al., 1990; Lodovici et al., 1995; Food Standards  
372 Agency, 2002; EFSA, 2008). In contrast, for France, the EFSA meta-analysis reported a level of  
373 PAH dietary exposure 10 times higher than that reported in an earlier separate study conducted  
374 between 2006-07 (EFSA, 2008). While the EFSA data exposure estimates were determined on  
375 the assumption that concentrations of PAH below detection limits equal the detection limit, the-  
376 reby overestimating the real PAH dietary intakes (the earlier French study assumed concentra-  
377 tions below detection limits to equal half the detection limit); a more plausible explanation for  
378 the order-of-magnitude higher EFSA exposure estimate for France, is that the EFSA study in-  
379 cluded more food categories considered to be “PAH-rich” than the earlier French study.

380 Outside Europe, the only total dietary study in the United States since the 1970s – albeit  
381 confined to B[a]P only – reported adult exposure to fall between 20 and 120 ng/day B[a]P for 90%  
382 of the investigated population (Kazerouni et al., 2001).

383



384  
 385 **Figure 4.** Natural log-transformed daily dietary intakes of PAH as a function of the year in  
 386 which the data were reported.  $R^2 = 0.019$ ,  $p = 0.67$ , slope =  $-0.0079$ . The normality and constant  
 387 variance tests were passed.  
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389  
 390 **3.5 Relative significance of different exposure pathways to PAH**

391 To estimate the relative contributions of inhalation, as well as the ingestion of dust and  
 392 diet to overall adult exposure to  $\Sigma$ PAH; we used the average PAH concentration data reported in  
 393 Tables 1-3 for indoor air, dust, and diet. We then assumed daily dust ingestion and air inhalation  
 394 rates of 20 mg/day and 20 m<sup>3</sup>/day for adults, as well as 50 mg/day and 7.6 m<sup>3</sup>/day for toddlers  
 395 (Oomen et al., 2008; Jones-Otazo et al., 2005). We also assumed dietary intake of toddlers to be  
 396 57% of adult dietary intake (Oomen et al., 2008; Jones-Otazo et al., 2005). Given the geographi-  
 397 cal variation in contamination of all three media, we conducted separate comparisons for Asia,  
 398 North America, and “other areas” (primarily but not exclusively Europe). For indoor air and dust,  
 399 we used the average concentrations for each region since 2000; while for diet, we used the 2001  
 400 US dietary intake data (B[a]P only instead of BaP<sub>eq</sub>, due to the data availability ) for North  
 401 America (Kazerouni et al., 2001), the reported 2008 dietary intake from EFSA (EFSA, 2008) for

402 Europe and elsewhere, while for Asia we used the 2010 dietary intake data from China (Zhang et  
403 al., 2014). The data from 1970s study were used for a rough estimation of the dietary intake of  
404  $\Sigma$ PAH in North America (Menzie et al., 1992). The time periods covered by these studies used  
405 for the exposure estimation and comparisons here were broadly temporally consistent (mostly  
406 between 2000 and 2010), thereby minimizing (though not eliminating) the impact of temporal  
407 variations. As earlier, we excluded as outliers, the studies reporting highly elevated concentra-  
408 tions in indoor air and dust from Burundi and Texas (Viau et al., 2000; Van Metre et al., 2009).

409         Table 4 summarizes the daily intakes of  $\Sigma$ PAHs and B[a]P via the three major exposure  
410 pathways in different regions for both adults and toddlers. Notwithstanding the uncertainties in-  
411 herent in these calculations, it is evident that for both adults and toddlers, that a) ingestion of in-  
412 door dust and diet constitute the principal pathways of exposure to B[a]P to North Americans; b)  
413 diet and inhalation are the predominant pathways of human exposure in Asia; c) diet ingestion is  
414 the most important exposure pathway elsewhere in the world. With respect to exposure to  $\Sigma$ PAH,  
415 inhalation and diet are the major sources for both adults and toddlers in most cases, although  
416 toddlers outside Asia and North America receive substantial exposure via all three pathways. In-  
417 takes of PAH via indoor dust ingestion are broadly similar in all regions, while exposure via in-  
418 halation and diet ingestion is an order of magnitude higher in Asia than elsewhere. These find-  
419 ings differ from other studies, which have reported either dietary ingestion (Chuang et al., 1999;  
420 Wilson et al., 2001; Menzie et al., 1992) or inhalation (Wilson et al., 2003) as the single domi-  
421 nant exposure pathway, rather than highlighting the importance of both such pathways, as well as  
422 – in some scenarios – ingestion of indoor dust. While acknowledging the uncertainties of our es-  
423 timates (and others) and the likely substantial inter-individual variability in the relative contribu-

424 tion of these pathways; our findings suggest each of the three exposure routes studied here, war-  
 425 rant attention in future studies.

426  
 427 **Table 4.** Comparison of estimated PAH intakes (ng/day) from the three major exposure path-  
 428 ways for adults and toddlers in Asia, North America and elsewhere.  
 429

<b>ΣPAH intakes (ng/day)</b>						
<b>Pathway</b>	<b>Adult</b>			<b>Toddler</b>		
	Asia	North America	Elsewhere	Asia	North America	Elsewhere
<b>Indoor air</b>	29848	1504	1592	11342	571	605
<b>Indoor dust</b>	266	117	145	665	292	361
<b>Total diet</b>	55000	3000	1729	31350	1710	986

<b>B[a]P intakes (ng/day)</b>						
<b>Pathway</b>	<b>Adult</b>			<b>Toddler</b>		
	Asia	North America	Elsewhere	Asia	North America	Elsewhere
<b>Indoor air</b>	509	2	19	193	1	7
<b>Indoor dust</b>	17	13	4	42	34	10
<b>Total diet</b>	472	20-80	235	269	11-46	134

430 See main text (section 3.5) for explanation of how exposure estimates were derived.  
 431

#### 432 **4. Discussion and conclusion**

433 In both indoor air and dust, ΣPAH concentrations in North America have decreased over  
 434 the past 30 years with a halving time of 5-7 years. In contrast, indoor PAH concentrations in  
 435 Asia have remained steady. Indoor/outdoor ratios of atmospheric concentrations of ΣPAH have  
 436 declined globally, while those for benzo[a]pyrene toxicity equivalents declined in North America  
 437 only – no significant decline was observed globally. Comparison of the global database, revealed  
 438 that I/O ratios for ΣPAH exceeded significantly those of BaP<sub>eq</sub> in the same samples. Combined,  
 439 these two findings suggest that over the period covered by this review: (a) indoor sources of  
 440 PAH have been controlled more effectively than outdoor sources because of the significant de-  
 441 cline in global I/O ratios, and (b) indoor sources of PAH emit proportionally more less carcino-

442 genic PAH than outdoor sources owing to the significantly higher I/O ratios for  $\Sigma$ PAH compared  
443 to  $BaP_{eq}$ . Definitive spatiotemporal trends in dietary exposure were not possible because of the  
444 relatively small number of relevant studies. However, although reported in only one study, PAH  
445 concentrations in Chinese diets exceeded those in diet from other parts of the world, a pattern  
446 consistent with the spatial trends observed for concentrations of PAH in indoor air. Evaluation of  
447 human exposure to  $\Sigma$ PAH via inhalation, dust and diet ingestion, suggests that while intake via  
448 diet and inhalation exceeds that via dust ingestion; all three pathways contribute and merit con-  
449 tinued assessment. The uncertainty of the conclusions is possibly limited by the heterogeneity in  
450 the numbers of PAH reported in each study as well as the variances of the sample sizes. Thus,  
451 the precautions should be taken as the review was based on the best available data.

452

## 453 **5. Research recommendations**

454 This review summarizes current knowledge about human exposure to PAH in the indoor  
455 environment. Although indoor PAH concentrations in Asia appear higher than in the rest of the  
456 world, this is based on comparison of a disparate range of *ad hoc* studies, and systematic com-  
457 parison of international trends is required if this apparent “Asian high-rise” is to be confirmed.  
458 Significantly elevated concentrations of PAH indoors were reported from locations considered  
459 impacted by emissions from the nearby outdoor use of coal-tar car park sealant in the US. This  
460 points to a possible focus of future research being the characterization of sources of indoor PAH  
461 additional to those currently recognized, like cooking and smoking. Moreover, information on  
462 PAH emission rates from such indoor sources remains scant and more such data are required.

463 Research is also required into the relative sampling efficiency of widely used vapor phase  
464 sorbents like PUF and XAD-2 for different PAH. This will ensure not only that the most appro-

465 piate sorbent is used in future, but provide reassurance of the comparability of previous studies  
466 using different sorbents. There also appears a need for research that examines whether the abso-  
467 lute concentrations and relative abundance of different PAH in indoor settled dust, is influenced  
468 significantly by whether the dust is collected from the floor, or elevated surfaces such as tables  
469 and chairs etc. Given that toddlers are more likely exposed to floor dust, and adults to elevated  
470 surface dust, any such differences will impact exposure and risk assessment.

471 Finally, more carefully designed studies are needed to assess fully the relative contribu-  
472 tions of different exposure pathways to overall human exposure to PAH. Given the evidence pre-  
473 sented here of substantial geographical and temporal variations in PAH contamination of indoor  
474 air and dust, as well as food; more exposure assessments based on samples collected in a spatial-  
475 ly and temporally consistent fashion are recommended (Wilson et al., 2003; Zhang et al., 2014).

476

#### 477 **Acknowledgements**

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480 project).

481

#### 482 **Appendix A. Supplementary data**

483 Details of PAH profiles in dust and air samples can be found online at  
484 <http://dx.doi.org/10.1016/j.envint.2014.xx.xxx>.

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