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Measurement of Personal Exposure to Volatile Organic Compounds and Particle Associated PAH in Three UK Regions

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Personal exposures to 15 volatile organic compounds (VOC) and 16 polycyclic aromatic hydrocarbons (PAH) of 100 adult nonsmokers living in three UK areas, namely London, West Midlands, and rural South Wales, were measured using an actively pumped sampler carried around by the volunteers for 5/1 (VOC/PAH) consecutive 24-h periods, following their normal lifestyle. Results from personal exposure measurements categorized by geographical location, type of dwelling, and exposure to environmental tobacco smoke (ETS) are presented. The average personal exposure concentration to benzene, 1,3-butadiene, and benzo(a)pyrene representing the main carcinogenic components of the VOC and PAH mixture were $2.2 \pm 2.5\ \mu g/m^3$, $0.4 \pm 0.7\ \mu g/m^3$, and 0.3 ± 0.7 ng/m^3 respectively. The association of a number of generic factors with personal exposure concentrations was investigated, including first-line property, traffic, the presence of an integral garage, and ETS. Only living in houses with integral garages and being exposed to ETS were identified as unequivocal contributors to VOC personal exposure, while only ETS had a clear effect upon PAH personal exposures. The measurements of personal exposures were compared with health-based European and UK air quality guidelines, with some exceedences occurring. Activities contributing to high personal exposures included the use of a fireplace in the home, ETS exposure, DIY (i.e., construction and craftwork activities), and photocopying, among others.

1. Introduction

Air toxics are ubiquitous in outdoor and indoor air, and therefore of public health concern. The International Agency for Research on Cancer (IARC) classifies benzene, 1,3-butadiene, and benzo(a)pyrene as known human carcinogens, dibenzo(a, h)anthracene as a probable human carcinogen, and several others (e.g., ethylbenzene) as possible human carcinogens (*1*). To date, while the study of personal exposure to air pollution is a rather well developed science, it has been restricted mainly to benzene and BTEX (benzene, toluene, ethylbenzene, and the xylenes) (*2, 3*), while studies where personal exposures to other VOC such as styrene, ETS markers, 1,3-butadiene, and PAH have been assessed are very limited (*4*−*6*).

The implementation of the European air quality daughter Directive 2000/69/EC involves a European limit value for benzene of 5 $\mu g/m^3$ (annual mean) to be achieved by 2010. The Directive 2004/107/EC involves a target value for B(a)P of 1 ng/m^3 (annual mean) to be achieved by 2012. The UK Air Quality Strategy for England, Scotland, Wales, and Northern Ireland involves a UK national air quality objective, reiterated in 2007, of 5 $\mu g/m^3$ (annual mean) for benzene, 2.25 $\mu g/m^3$ (running annual average) for 1,3-butadiene, and 0.25 ng/m^3 for benzo(a)pyrene.

The Measurement and Modeling of Air Toxic Concentrations for Health Effect Studies (MATCH) Project aimed to provide a significant strengthening of the VOC and PAH personal exposure and microenvironment measurement database through generating new data via direct measurements. The study sought to lead to advances in understanding the causes and magnitude of exposures to VOC and PAH and to establish whether collecting lifestyle information is sufficient to model personal exposures reliably when compared with exposures evaluated independently by personal samplers. In this paper, the results for VOC and PAH personal exposure are presented and considered in light of the target values for benzene, 1,3-butadiene, and benzo(a)pyrene set by European Directives and the UK Air Quality Strategy. It also tries to elucidate factors influencing the exposure of the subjects to VOC and PAH including living in a first-line property, living in houses with integral garage, and being exposed to environmental tobacco smoke (ETS) as well as traffic in terms of geographical location and location of the dwelling within the city boundaries.

2. Materials and Methods

2.1. Volunteer Recruitment. The MATCH project recruited 100 healthy non-smoker adult volunteer subjects between 2005 and 2007 for non-occupational personal exposure (PE). Three areas were chosen for their expected gradient in PE concentrations: London, West Midlands, and rural South Wales. Subjects were chosen to participate based upon four key determinants (i.e., possible VOC/PAH sources), namely the location where they lived, if they were exposed to ETS, if their house incorporated an integral garage, and by the proximity of the house to a major road (coded as first line). First line (FL) subjects lived in properties located on an A road (>20,000 vehicles per day including heavy vehicles) or a busy B road (lower volumes of traffic including heavy vehicles) in an urban/suburban area. Subjects were considered ETS exposed whenever they reported themselves in the activity diary to be in the company of a smoker (friend/relative) or to be in places with smokers (e.g., pub).

Subjects were excluded if they were smokers, in poor health, under 18 years of age, unable to carry the personal sampler, exposed to VOC and PAH at work, if they traveled more than 2 h per day professionally, if the journey to work took more than 2 h traveling time for a return trip, or if their workplace was more than 20 miles from their house.

2.2. Sampling Methods. VOC and 1,3-butadiene were collected in separate preconditioned sorbent tubes (*4*), while PAH were collected on 47-mm filters. For the first 33 subjects glass fiber filters were used but for the subsequent work quartz fiber filters prebaked for 48 h at 400 °C were used due to a lower blank (*7*).

Each subject was sampled for VOC and 1,3-butadiene for a total of five consecutive 24-h periods using one personal sampler pump, connected to the sorbent tubes and in one

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TABLE 1. Characterization of VOC (μg/m^3) and PAH (ng/m^3) Personal Exposure Concentrations by Location in All Subjects and by Key Determinant

Location / Key Determinant		Statistics		VOC									PAH							
				Number of VOC samples	Benzene	Toluene	Ethylbenzene	Σ-Xylenes	Styrene	3-Ethenylpyridine	Naphthalene	1,3-Butadiene	Number of PAH samples	Benzo(a)anthracene	Benzo(b)fluoranthene	Benzo(k)fluoranthene	Benzo(a)pyrene	Indeno(1,2,3-cd)pyrene	Dibenz(a,h)anthracene	Σ8-PAH[a]	Σ16-PAH
ALL		Max/Min	Min	500	0.1	0.1	0.1	0.1	0.1	n.d.	0.1	n.d.	91	n.d.	n.d.	0.01	n.d.	n.d.	0.01		
			Max		30.2	407	182	963	61.7	6.9	12.6	6.3		3.33	5.18	5.62	6.22	5.36	3.51		
		Arithmetic	Mean		2.2	19.7	3.2	14.4	1.3	0.3	0.7	0.4		0.45	0.42	0.37	0.30	0.23	0.20	**2.21**	**3.28**
			SD		2.5	34.3	10.6	53.6	4.6	0.6	1.1	0.7		0.59	0.70	0.68	0.69	0.60	0.41		
		Geometric	Mean		1.6	11.5	1.5	6.1	0.6	0.1	0.5	0.1		0.22	0.22	0.18	0.14	0.09	0.10	**1.03**	**1.61**
			SD		2.0	2.7	2.7	8.9	2.4	3.9	2.0	5.2		3.67	2.98	3.12	3.20	3.91	2.94		
URBAN	ALL URBAN	Max/Min	Min	191	0.2	0.1	0.1	0.1	0.1	n.d.	0.1	n.d.	35	0.01	0.02	0.02	n.d.	0.01	n.d.		
			Max		18.9	205	221	51.9	61.3	6.9	12.7	5.9		0.97	1.49	1.32	1.19	0.93	0.19		
		Arithmetic	Mean		1.6	16.4	9.9	2.0	1.8	0.3	0.8	0.4		0.16	0.26	0.24	0.19	0.18	0.03	**1.74**	**3.12**
			SD		1.0	16.5	21.8	4.6	6.6	0.6	1.5	0.8		0.24	0.31	0.28	0.27	0.21	0.05		
		Geometric	Mean		1.4	10.9	5.3	1.1	0.6	0.1	0.5	0.1		0.08	0.18	0.17	0.11	0.14	0.01	**1.18**	**2.08**
			SD		1.8	2.4	7.7	2.5	2.6	3.8	2.2	5.5		2.91	2.75	2.34	2.81	2.20	7.17		
	LONDON	Max/Min	Min	55	2.7	0.4	1.2	0.2	0.1	0.1	0.1	n.d.	11	0.03	0.02	0.04	0.03	0.03	n.d.		
			Max		7.2	98.0	164	28.7	1.2	2.5	12.7	0.8		0.18	0.24	0.18	0.14	0.19	0.03		
		Arithmetic	Mean		1.8	17.7	11.2	2.2	0.5	0.3	1.0	0.1		0.07	0.13	0.12	0.07	0.09	0.01	**0.84**	**2.09**
			SD		1.3	21.1	24.2	4.3	0.2	0.5	1.9	0.1		0.04	0.07	0.04	0.03	0.04	0.01		
		Geometric	Mean		1.5	10.5	5.7	1.2	0.4	0.1	0.6	0.1		0.06	0.10	0.12	0.07	0.09	0.01	**0.81**	**1.75**
			SD		1.8	2.7	8.2	2.7	1.6	3.6	2.2	3.1		1.49	2.51	1.59	1.69	1.72	3.38		
	BIRMINGHAM	Max/Min	Min	136	0.3	0.1	0.1	0.1	0.1	n.d.	0.1	n.d.	24	0.01	0.02	0.02	n.d.	0.01	n.d.		
			Max		18.9	205	221	51.9	61.3	6.9	10.8	5.9		0.97	1.49	1.32	1.19	0.93	0.19		
		Arithmetic	Mean		1.6	15.9	9.5	1.9	2.3	0.3	0.7	0.6		0.20	0.33	0.30	0.24	0.22	0.04	**2.15**	**3.59**
			SD		0.9	14.6	20.9	4.7	7.7	0.6	1.3	0.9		0.28	0.35	0.32	0.32	0.24	0.05		
		Geometric	Mean		1.3	11.1	5.2	1.0	0.7	0.1	0.4	0.2		0.10	0.25	0.22	0.15	0.18	0.02	**1.53**	**2.46**
			SD		1.9	2.4	7.6	2.5	2.8	3.8	2.1	5.8		3.71	2.55	2.61	3.30	2.31	10.40		
SUBURBAN		Max/Min	Min	209	0.2	0.1	0.1	0.1	0.1	n.d.	0.1	n.d.	39	0.01	0.01	n.d.	n.d.	0.01	n.d.		
			Max		30.3	377	182	965	41.7	5.2	6.4	6.3		0.70	2.24	1.59	0.80	0.75	0.20		
		Arithmetic	Mean		2.6	22.3	4.4	20.5	1.1	0.3	0.7	0.4		0.10	0.38	0.23	0.16	0.14	0.04	**1.61**	**2.49**
			SD		3.3	40.4	15.4	78.9	3.0	0.7	0.8	0.6		0.15	0.49	0.30	0.19	0.15	0.04		
		Geometric	Mean		1.8	11.9	1.7	6.8	0.7	0.1	0.6	0.1		0.07	0.27	0.18	0.11	0.14	0.02	**1.20**	**1.91**
			SD		2.1	2.8	2.9	10.3	2.2	4.2	2.0	5.8		2.69	2.50	2.17	2.94	2.34	3.75		
RURAL	ALL RURAL	Max/Min	Min	100	1.4	0.2	0.9	0.2	0.1	0.1	0.1	0.1	17	n.d.	0.03	0.05	0.01	0.01	n.d.		
			Max		15.9	403	46.3	8.5	10.8	3.3	2.8	4.0		5.55	5.62	6.22	5.36	3.51	0.94		
		Arithmetic	Mean		2.5	20.8	9.7	2.0	0.9	0.2	0.7	0.4		0.44	0.56	0.56	0.49	0.39	0.10	**3.62**	**5.46**
			SD		2.3	41.9	9.2	1.9	1.7	0.4	0.5	0.6		1.32	1.29	1.45	1.29	0.85	0.24		
		Geometric	Mean		1.9	11.8	6.2	1.3	0.6	0.1	0.6	0.2		0.12	0.42	0.28	0.18	0.22	0.03	**1.84**	**3.01**
			SD		2.0	2.7	8.0	2.7	2.4	3.4	1.9	3.9		5.43	4.35	4.74	5.37	4.40	5.79		
	WEST MIDLANDS	Max/Min	Min	50	3.3	0.4	1.3	0.2	0.2	0.1	0.2	0.1	9	n.d.	0.03	0.05	0.01	0.01	n.d.		
			Max		8.7	403	45.6	8.5	1.8	3.2	2.8	4.0		0.22	0.50	0.40	0.37	0.32	0.09		
		Arithmetic	Mean		2.2	23.8	9.9	2.1	0.6	0.3	0.8	0.5		0.05	0.24	0.17	0.15	0.15	0.04	**1.36**	**3.01**
			SD		1.6	56.9	9.1	1.9	0.4	0.5	0.7	0.8		0.07	0.19	0.13	0.14	0.11	0.03		
		Geometric	Mean		1.8	13.0	6.9	1.4	0.6	0.1	0.6	0.2		0.04	0.24	0.14	0.09	0.13	0.01	**0.99**	**2.31**
			SD		1.8	2.4	7.2	2.4	1.7	3.8	2.0	4.0		1.66	2.01	2.29	2.93	1.98	7.63		
	WALES	Max/Min	Min	50	1.4	0.2	0.9	0.2	0.1	0.1	0.2	0.1	8	0.02	0.06	0.05	0.02	0.02	0.01		
			Max		15.9	60.6	36.6	8.2	10.8	0.6	1.9	1.8		5.55	5.62	6.22	5.36	3.51	0.94		
		Arithmetic	Mean		2.9	17.8	9.4	2.0	1.3	0.1	0.6	0.2		0.77	0.88	0.95	0.79	0.63	0.17	**5.75**	**7.73**
			SD		2.8	17.5	9.4	2.1	2.3	0.1	0.3	0.3		1.80	1.81	2.02	1.75	1.18	0.34		
		Geometric	Mean		2.1	10.6	5.5	1.1	0.6	0.1	0.5	0.1		0.19	0.52	0.36	0.23	0.26	0.04	**2.31**	**3.45**
			SD		2.2	2.9	8.8	3.0	3.1	2.2	1.7	3.5		5.77	5.13	5.44	6.19	5.26	4.90		
First Line		Max/Min	Min	219	0.3	0.1	0.1	0.1	0.1	n.d.	n.d.	n.d.	41	n.d.	0.02	0.02	n.d.	0.01	n.d.		
			Max		30.0	377	99.0	441	42.0	6.0	6.0	3.0		5.55	5.62	6.22	5.36	3.51	0.94		
		Arithmetic	Mean		2.5	24.3	3.1	14.0	1.1	0.2	0.7	0.3		0.25	0.41	0.38	0.32	0.27	0.06	**2.52**	**3.78**
			SD		3.3	40.3	8.6	39.8	3.1	0.5	0.8	0.5		0.86	0.89	0.98	0.87	0.59	0.16		
		Geometric	Mean		1.7	12.9	1.4	6.0	0.6	0.1	0.5	0.1		0.08	0.24	0.18	0.12	0.14	0.02	**1.23**	**2.04**
			SD		2.2	2.9	2.8	9.0	2.4	3.7	2.2	5.2		2.62	2.29	2.22	2.53	2.32	3.08		
Non-First Line		Max/Min	Min	281	0.2	0.1	0.1	0.1	0.1	n.d.	0.1	n.d.	50	0.01	0.01	n.d.	n.d.	0.01	n.d.		
			Max		19.0	403	182	965	61.0	7.0	13.0	6.0		0.97	2.24	1.59	1.19	0.93	0.20		
		Arithmetic	Mean		2.0	16.1	3.3	14.6	1.5	0.3	0.8	0.5		0.14	0.34	0.23	0.17	0.15	0.03	**1.67**	**3.03**
			SD		1.5	27.3	12.0	62.1	5.5	0.6	1.2	0.9		0.21	0.46	0.29	0.22	0.16	0.04		
		Geometric	Mean		1.6	10.5	1.5	6.2	0.7	0.1	0.6	0.2		0.09	0.30	0.23	0.14	0.18	0.02	**1.48**	**2.80**
			SD		1.8	2.4	2.7	8.9	2.3	4.0	1.9	5.3		4.53	4.11	3.73	4.63	3.47	7.86		
Integral Garage		Max/Min	Min	80	0.2	0.1	0.1	0.1	0.1	n.d.	0.1	n.d.	16	0.01	0.01	n.d.	0.02	0.02	0.01		
			Max		19.0	403	99.0	441	11.0	5.0	6.0	2.0		0.29	1.98	0.48	0.33	0.27	0.10		
		Arithmetic	Mean		3.5	33.5	4.6	21.0	1.3	0.4	0.9	0.4		0.09	0.39	0.19	0.14	0.14	0.04	**1.41**	**2.40**
			SD		3.0	61.3	11.2	49.6	1.9	0.7	1.0	0.4		0.08	0.50	0.14	0.11	0.10	0.03		
		Geometric	Mean		2.7	18.5	2.6	11.3	0.8	0.1	0.7	0.2		0.07	0.24	0.14	0.09	0.12	0.02	**1.03**	**1.78**
			SD		2.1	2.8	2.5	9.7	2.3	4.3	2.0	3.6		2.24	2.82	2.42	2.84	2.18	2.68		
Non-Integral Garage		Max/Min	Min	420	0.3	0.1	0.1	0.1	0.1	n.d.	n.d.	n.d.	75	n.d.	0.02	n.d.	n.d.	0.01	n.d.		
			Max		30.0	313	182	965	61.0	7.0	13.0	6.0		5.55	5.62	6.22	5.36	3.51	0.94		
		Arithmetic	Mean		2.0	17.0	2.9	13.0	1.3	0.3	0.7	0.4		0.21	0.37	0.32	0.25	0.22	0.05	**2.18**	**3.54**
			SD		2.3	24.4	10.5	53.9	4.9	0.6	1.1	0.8		0.67	0.72	0.75	0.66	0.45	0.12		
		Geometric	Mean		1.5	10.5	1.3	5.4	0.6	0.1	0.5	0.1		0.09	0.28	0.23	0.14	0.17	0.02	**1.45**	**2.52**
			SD		1.9	2.6	2.7	8.4	2.4	3.9	2.0	5.6		3.94	3.42	3.15	3.81	3.12	6.22		
ETS		Max/Min	Min	195	0.2	0.1	0.1	0.1	0.1	n.d.	n.d.	n.d.	36	0.01	0.01	n.d.	0.01	0.01	n.d.		
			Max		30.0	403	99.0	441	4.0	7.0	11.0	6.0		0.78	2.24	1.59	0.80	0.75	0.20		
		Arithmetic	Mean		2.5	25.1	3.3	16.1	0.9	0.6	0.8	0.5		0.17	0.42	0.28	0.21	0.17	0.05	**2.11**	**3.33**
			SD		3.0	49.3	9.1	43.1	0.7	0.8	1.2	0.8		0.21	0.48	0.30	0.21	0.16	0.04		
		Geometric	Mean		1.9	12.9	1.7	7.1	0.7	0.3	0.6	0.2		0.10	0.34	0.29	0.23	0.23	0.05	**2.01**	**2.91**
			SD		2.0	2.9	2.6	9.2	2.0	4.0	2.0	5.5		3.13	1.87	1.98	2.22	2.14	2.22		
Non-ETS		Max/Min	Min	305	0.3	0.1	0.1	0.1	0.1	n.d.	0.1	n.d.	55	n.d.	0.02	n.d.	n.d.	0.01	n.d.		
			Max		19.0	159	182	965	61.0	2.0	13.0	6.0		5.55	5.62	6.22	5.36	3.51	0.94		
		Arithmetic	Mean		2.0	16.3	3.1	13.2	1.6	0.1	0.7	0.4		0.21	0.33	0.31	0.25	0.22	0.05	**2.00**	**3.36**
			SD		2.1	17.2	11.5	58.9	5.8	0.1	1.0	0.7		0.77	0.79	0.85	0.76	0.51	0.14		
		Geometric	Mean		1.9	12.9	1.7	7.1	0.7	0.3	0.6	0.2		0.09	0.25	0.20	0.13	0.16	0.02	**1.33**	**2.24**
			SD		2.0	2.9	2.6	9.2	2.0	4.0	2.0	5.5		4.06	3.98	3.67	4.03	3.45	5.93		

[a] ∑8-PAH is the sum of B(a)A, Chry, B(b)F, B(k)F, B(a)P, D(a,h)A, B(ghi)P, I(1,2,3-cd)P.

concurrent day an additional pump sampled PAH. The active-sampling setup was enclosed in a small aluminum briefcase. The flow rates used were 40 mL/min for VOC, 30 mL/min for 1,3-butadiene, and 3 L/min for PAH (*7*).

Quality assurance and quality control procedures are described in detail in the Supporting Information (SI).

2.3. Subject-Related Information. The atmospheric sampling was backed up with information related to the volunteer subjects. Several questionnaires, filled out daily, included 30-min activity diaries, travel description sheets, location description sheet, and activity questionnaires. Information about the subject's demographics, home, and products stored within the house were collected once per subject. An ETS questionnaire was filled when the subjects were exposed to ETS (once per event). Further details

describing the questionnaires can be found in Harrison et al. (*7*). Researchers checked the accuracy of forms daily.

2.4. Analytical Methodology. Three methods were employed for analyzing 1,3-butadiene, the rest of the VOC (*n*-hexane to naphthalene), and all the PAHs (*7*) as described in detail in the SI.

2.5. Statistical Analysis. PE were tested for normality and distributions were found to be right-skewed. Therefore arithmetic and geometric means and standard deviation are reported. Statistical differences between two strata were tested in the logged database with a *t* test for equality of means.

3. Results and Discussion

3.1. VOC Personal Exposures. The VOC personal exposure levels observed in this study (Table 1 and Table S6, SI) are substantially lower (e.g., by 15−80%) than those found in similar studies, conducted in different locations in the United States and Europe and at earlier times (*8*−*11*).

The concentrations for VOC measured in EXPOLIS-Helsinki (*6*) are higher than the present study for almost all the compounds except for styrene. On the other hand, the values reported by EXPOLIS-Oxford (*12*), show similar mean values for ethylbenzene and the xylenes, but higher mean concentrations for benzene, toluene, trimethylbenzenes, and *n*-hexane. Similarly, the same comparison can be drawn with the results of a German study (*2*), whose values of ethylbenzene and xylenes are comparable to ours, but the levels of toluene and benzene nonetheless were higher. Conversely, the European study PEOPLE (*13*) found higher benzene levels than those in our study for all the sampled population except for subjects who spent most of the time at home.

A previous study conducted in the West Midlands (UK) in 1998 (*14*) reported higher concentrations than those in this study for all the compounds except for *o*-xylene. In a later study in the West Midlands, Kim and co-workers reported concentrations higher for benzene, toluene, and *n*-hexane, but similar to those in the present study for all the other compounds (*4*). Two recent studies in Minneapolis (2000−2001) and Oklahoma (1999−2000), found similar median VOC concentrations for PE (*3, 15*).

A rough comparison of the TEAM and similar studies and the more recent data suggests that PE to benzene and other VOC compounds has decreased since the 1980s (*16*). The decrease in exposure to VOC and especially, those from traffic, is consistent with the sharp downward trends in airborne concentrations (*17*) mainly due to controls on emissions from both road vehicles and industrial sources.

3.2. PAH Personal Exposures. The subjects in this study went about their life as normal, and the measured exposures to PAH are summarized in Table 1 (and Table S6, SI). This table, as well as Figures 1−3 omit two extreme outlying points (see below) which were measured in two rural subjects burning wood in a fireplace. Information on PAH PE reported in this study is comparable with data reported previously (*18*−*20*).

Georgadis et al. (*29*) carried out a PAH PE campaign in Athens and Halkida, a nearby small town. The following PAH: B(a)A, Chry, B(b)F, B(k)F, B(a)P, D(a, h)A, B(ghi)P, and I(1,2,3-cd)P, were measured and data were reported as the sum of the 8 PAH (∑8-PAH). The mean PE to the ∑8-PAH was 7.95 ng/m^3 and 4.53 ng/m^3 for Athens and Halkida, respectively. In this study for urban London the ∑8-PAH PE mean was 0.84 ng/m^3. In Birmingham it was 2.15 ng/m^3 and 1.61 ng/m^3 for urban and suburban areas, respectively, whereas in rural Wales it was 5.75 ng/m^3 (Table 1). Regulation of air quality has become more stringent in recent years in the UK and this combined with different meteorological conditions and different traffic patterns in the two countries might explain the difference between levels recorded in Greek cities and those measured in this study in different UK areas. According to Coleman and co-workers, from the air data sets available from 1991 to 1995, on average, for the two urban sites in London and Manchester the ∑8-PAH concentrations declined by 30%/yr (*21*). Data from Birmingham collected in 1977/1978 and 1992 also indicate a strong downward trend (*22*).

3.3. Effect of Key Determinants. The impact that traffic has on PE has been assessed in different ways. These include considering the geographical location of subjects, the area within the city where the subject's home is located, and classifying the home with respect road traffic load. Homes are expected to be the most influential microenvironment for all VOC (*4*), with in some cases the distributions of personal and indoor home concentrations highly overlapped (*3*). Therefore, the impact that traffic has on PE is mainly due to the impact of the traffic emissions in the home.

As regards the geographical location of the subjects (Table 1), those in London showed concentrations similar to those subjects in urban Birmingham for most of the compounds, while those in rural Wales showed concentrations similar to those located in rural West Midlands for VOC but higher concentrations for PAH. These results should be carefully interpreted as due to logistic and recruitment problems: all the London subjects were sampled in summertime, when both outdoor and indoor concentrations are lower (*23*), there is a higher outdoor−indoor air exchange rate, and a higher proportion of time is spent outdoors (*4*), while Birmingham subjects were sampled across the four seasons and Wales subjects were sampled mainly in wintertime. To overcome this seasonality effect, urban London subjects were compared with urban subjects living in Birmingham sampled in summertime whereas rural Welsh subjects were compared with rural West Midlands subjects sampled in wintertime. Figure 1 shows that London subjects generally show higher PEs than subjects living in Birmingham for most of the VOC ($p < 0.10$, i.e., probability of random correlation less than 10%) and some PAH compounds (e.g., benzo(a)pyrene, $p < 0.05$) in accordance with the expected traffic volumes characteristics of each city, which emphasizes the idea of outdoor traffic as a source of VOC and PAH in PEs. As regards rural subjects, those living in West Midlands show slightly higher concentrations for ETS-related compounds (e.g., pyridine, 3-ethenylpyridine, 1,3-butadiene, and chrysene, $p < 0.05$) consistent with information reported in the questionnaires, and higher although not significant ($p > 0.10$) PAH concentrations for those living in Wales. This result is consistent with the fact that in wintertime people spend most of their time indoors and therefore, personal activities, heating, and cooking characteristics are expected to have a larger influence on PE than outdoor traffic.

As regards the effect that home location within a city has on PE concentration (Table 1) urban subjects have previously been reported to be more exposed to VOC than rural subjects (*2, 14*). However, this is not the situation reflected in this study (Table 1) with similar VOC concentrations for urban, suburban, and rural subjects, and higher rural PAH concentrations ($p < 0.05$). Analyzing the information gathered about home description (Tables S3 and S4, Supporting Information) it can be observed that many suburban and rural subjects have a pollutant source that urban subjects do not have, which is the presence of an integral garage within their homes. In addition, our suburban subjects showed a greater prevalence of ETS exposure (43%) compared with subjects living in urban (32%) and rural areas (20%). Additionally, rural subjects were sampled mainly in colder months, during which indoor sources related to home heating are greater and air exchange rates are lower. Also, 25% of the rural subjects had recently redecorated their homes, were not ETS exposed, and the percentage of houses heated by natural gas was 65%, 25% with other carbonaceous fuels (coal and kerosene) and only 10% with electricity. In comparison,

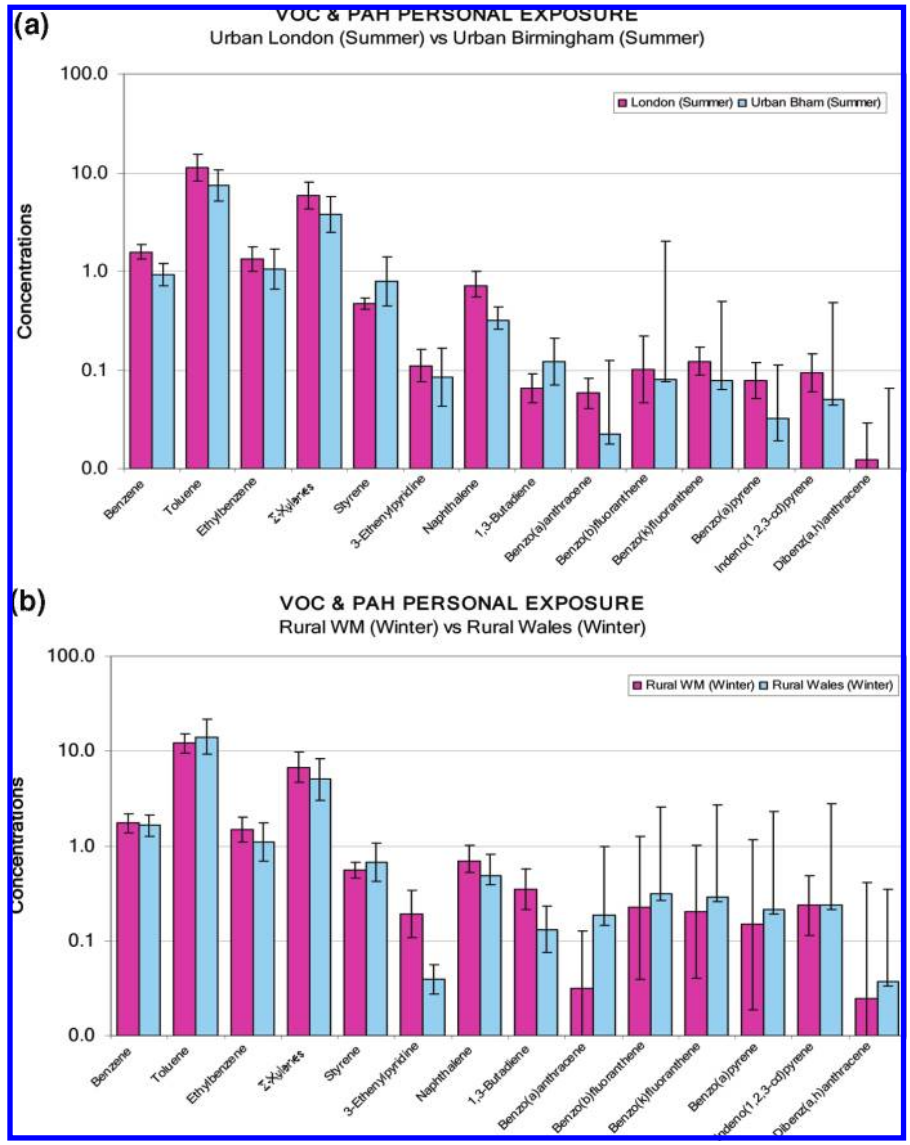

FIGURE 1. Geographic effect upon VOC ($\mu g/m^3$) and PAH (ng/m^3) personal exposure concentrations matched by season. (a) Urban London vs urban Birmingham and (b) rural West Midlands vs rural Wales.

76% heated the house by natural gas, 19% with electricity, 2.4% with other fuels, and 2.4% reported missing data in suburban Birmingham. The higher use of more polluting fuels to heat the houses might explain a higher PAH PE in rural areas. Use of a fireplace (including heating stoves) was associated with increased exposures. For benzo(a)pyrene, the mean exposures for those using a gas fire was 0.37 ng/m^3 ($n = 5$) and for wood burning was 6.24 ng/m^3 ($n = 5$), compared with 0.16 ng/m^3 for those subjects who were using only central heating or whose houses were unheated ($n = 83$). The figure for wood burning includes two extreme outliers of 25.3 ng/m^3 and 5.4 ng/m^3 whose data were excluded from the statistical analysis. Another activity that might influence PAH exposure is that in rural areas 75% of the people do not use a cooker hood while cooking as opposed to the 60% in suburban Birmingham. Rural dwellers may also spend longer (7% of their time) commuting by car than those who live in cities. A similar assessment performed within West Midlands subjects confirms the fact that a higher proportion of within-home sources masks the trend of Urban > Suburban > Rural (Table 1). These findings emphasize the fact that not only outdoor concentrations contribute to PE but also other sources contribute (*14*) including personal activities such as exposure to ETS, commuting, use of consumer products, DIY (i.e., construction and craftwork activities), use of solvents, photocopying, etc.

The effect of residential traffic on PE to VOC and PAH does not show a clear distinction between people living on trafficked roadsides (first line (FL) subjects) as compared to the non-FL subjects. Although the mean suggests that FL-subjects have higher levels for the higher molecular weight PAH, toluene is the only compound that exhibits slightly higher concentrations in FL-subjects ($p < 0.10$) (Table 1 and Table S7, SI). This pattern is, however, in contrast to other studies where subjects living close to traffic sources recorded higher VOC concentrations (*2*) in addition to the fact that traffic is one of the known sources of VOC and PAH compounds (*4, 24*). On the other hand, although the means of both subpopulations are not statistically different, in the case of traffic-related compounds such as benzene or the high molecular weigh PAH (e.g., BaP), the comparison of the frequency of PE distribution for subjects living in homes near traffic (Figure 2a, FL = 1) with those living away from traffic (Figure 2a, FL = 0) shows that there is a higher percentage of population exposed to high levels of VOC within the subpopulation living close to traffic sources. However, one important observation is that components linked with ETS (e.g., pyridine, 3-ethenylpyridine) were higher in subjects

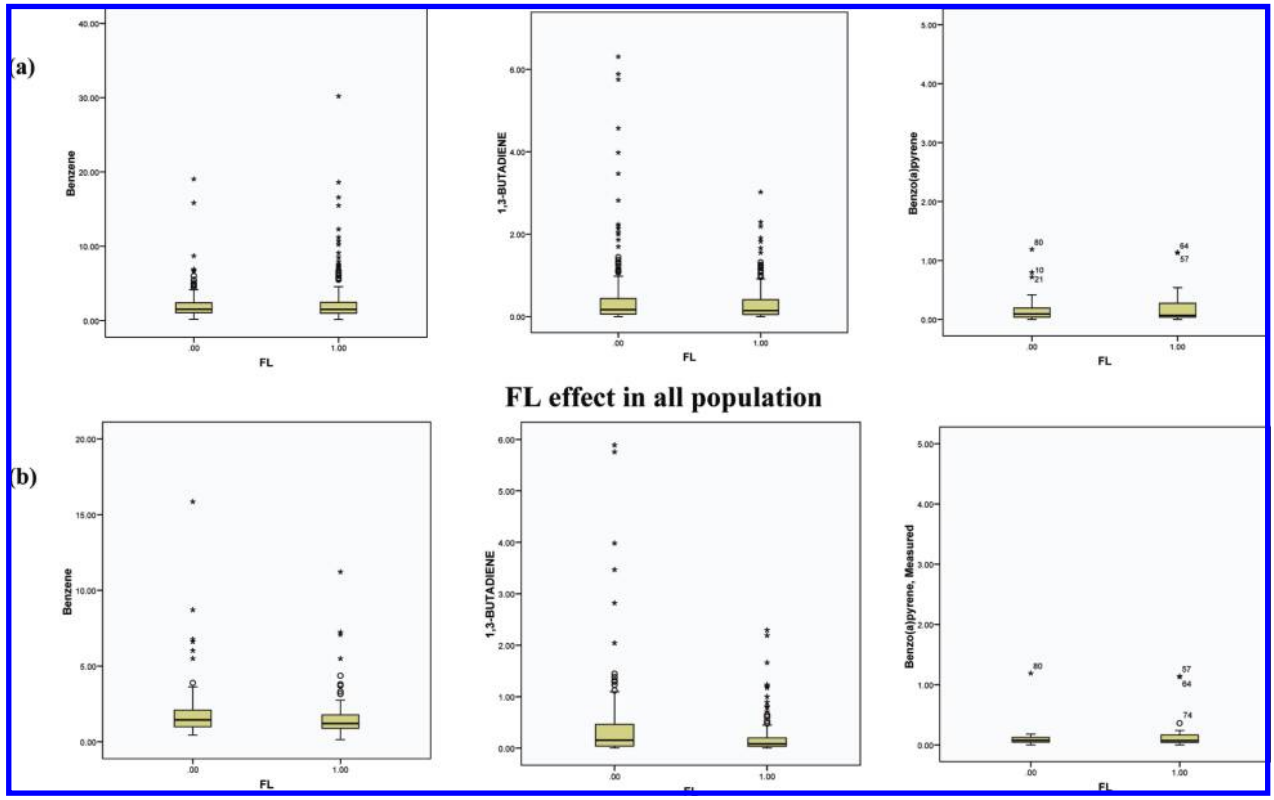

FIGURE 2. Boxplots for benzene (μg/m^3), 1,3-butadiene (μg/m^3) and benzo(a)pyrene (ng/m^3) personal exposure for (a) all subjects living in first line (FL = 1) properties (N_{VOC} = 219; N_{PAH} = 41) and all subjects living in houses away from traffic (FL = 0) (N_{VOC} = 281; N_{PAH} = 50); (b) subjects living in houses in FL properties (FL = 1) (N_{VOC} = 119; N_{PAH} = 20) and subjects living in houses away from traffic (FL = 0) (N_{VOC} = 129; N_{PAH} = 24) with no ETS exposure and no integral garage.

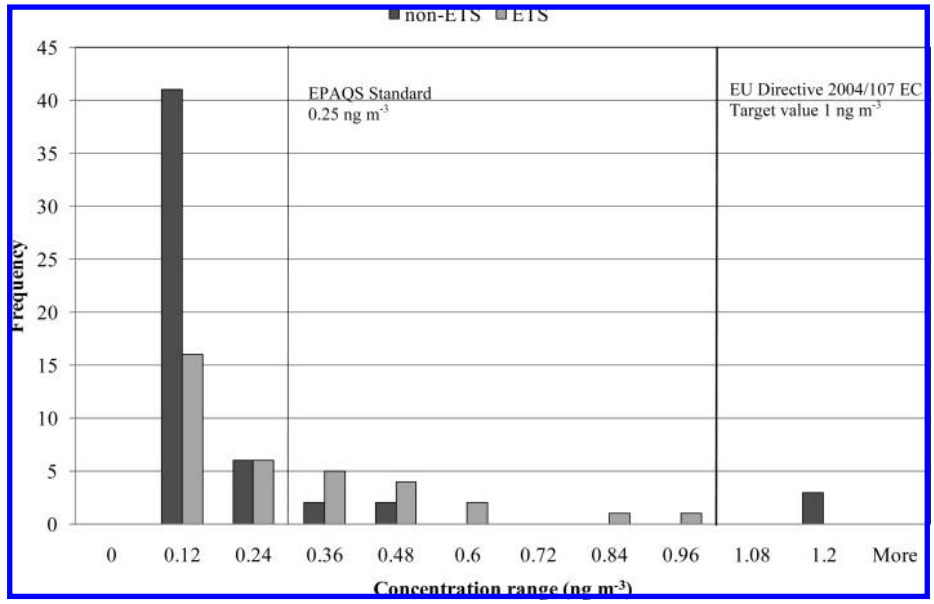

FIGURE 3. Frequency distribution of B(a)P personal exposure (ng/m^3) for the ETS-exposed (N = 36) and non-ETS exposed (N = 55) groups (excludes two extreme outliers, see text).

not living in first line properties. Further investigation controlling for the effect of ETS exposure and integral garage (IG) in the analysis of the effect of first line properties on PE showed that VOC concentrations were higher in the subjects living on a non-trafficked roadside (Figure 2b), but remained higher in FL subjects for the PAH data set. This finding suggests that once controlled for integral garages and ETS, the influence of other indoor sources (e.g., redecoration, heating by means of alternative fuels to electricity or natural gas), and activities that the subjects perform during the day (e.g., commuting, DIY, incense burning) might have masked the effect of traffic on PEs on the VOC data set, other than for a few compounds (e.g., toluene). On the contrary, once controlled for ETS and IG, residential traffic remains as a factor that increases PAH concentrations regardless of other indoor sources (e.g., ETS), as we observe in the higher percentage of population exposed to high levels of PAH within the subpopulation living close to traffic sources (Figure 2b).

Living in houses with integral garages (IG) (Table 1) was associated with increased PE levels ($p < 0.01$) for most of the

VOC compounds and with no effect in the PAH data set. The average ratio of geometric mean VOC concentrations of subjects that lived in houses with IG compared to those without IG is (1.9 ± 0.6):1. These findings of higher VOC levels are in accordance with results previously reported in other studies (*25, 26*), while a small effect in the PAH data set is consistent with the observation in home microenvironments that unless ETS is the major source indoors, the exposure to PAH would be due to a multitude of sources (*27*). Integral garages have been associated with higher exposures to air toxics (e.g., benzene, xylenes, and 1,3,5-trimethylbenzene) with major evaporative sources (e.g., stored gasoline) (*25*), but this does not apply to combustion-generated pollutants such as PAH, unless heating boilers are situated in the integral garage. Sources of VOC in garages were investigated from information provided by the subjects in the questionnaires, and were related to parked cars, storage of DIY and gardening products, and location of the heating system unit.

The effect of ETS in VOC PE is also clear (Table 1), leading to higher concentrations for all the VOC compounds ($p < 0.05$, $N = 500$) with an average ratio of geometric means being (1.4 ± 0.4):1. The highest differences observed were for ETS related compounds like 3-ethenylpyridine and pyridine, with an average ratio of geometric means being 1.8:1 for both compounds, which is consistent with other studies (*10, 13, 14, 25, 28*). When VOC personal exposures of ETS subjects are averaged across the sampling week, only ETS related compounds, such as 3-ethenylpyridine and pyridine, remain significantly higher compared with non-ETS compounds ($p < 0.001$, $N = 100$). This is a consequence of the fact that not all the ETS subjects had been exposed to ETS on all the sampled days, and therefore the ETS effect gets diluted when PE are averaged throughout the 5 sampling days.

On the contrary, results for the PAH data set show no significant difference between ETS-affected and non-ETS subjects (Table 1). This is inconsistent with results reported in various studies where ETS is the main contributor to PAH in PE (*29*) and in indoor air (*30, 31*). Li et al. (2005) propose that apart from ETS, indoor sources exist for 2- and 3-ring PAH (*27*) while the outdoor air may contribute significantly to the 4 or more ring PAH inside, associated with particles of diameters ≤2.5 μm (*32*). Source apportionment carried out by Mitra and Ray (1995) showed that there is not a clear-cut difference between nonsmokers' homes and smokers' homes (*33*). In the present study, a distinction between ETS and non-ETS exposed groups is shown in Figure 3, if in the non-ETS group 3 subjects are considered as outliers for a concentration range more than 1.08 ng/m^3 and up to 1.2 ng/m^3 (the two extreme outliers due to fireplace use were already excluded). When these 3 subjects were removed, the B(a)P mean concentration in the non-ETS group drops to 0.20 ng/m^3 and the ∑PAH reduces to 2.87 ng/m^3, making a pronounced statistically significant ($p < 0.10$) difference between the ETS and the non-ETS groups. The difference is such that as the results of Mitra and Ray (1995) emphasize, in the absence of ETS, background sources contribute significantly to the total PAH.

3.4. European and UK guidelines Compliance. The majority of the subjects were exposed to levels of benzene, 1,3-butadiene, and benzo(a)pyrene below the European limit or target value (benzene, 5 μg/m^3; benzo(a)pyrene, 1 ng/m^3) and UK air quality objectives (benzene, 5 μg/m^3; 1,3-butadiene, 2.25 μg/m^3, benzo(a)pyrene, 0.25 ng/m^3). However, 33 cases of benzene (6.6%) and 10 cases (2%) of 1,3-butadiene were higher than the limit values/air quality objectives. As for the PAH, 5 cases (5%) were higher than the European target value and 16 cases (17%) exceeded the standard value recommended by the UK EPAQS on which the UK air quality objective is based, and it would appear from Figure 3 that ETS exposure was an important contributory factor in those exceeding the EPAQS standard. It should, however, be noted that the EPAQS standard is based upon benzo(a)pyrene as a surrogate for the carcinogenic potency of the PAH mixture. Since the relative amounts of the different carcinogenic PAH may differ between indoor air and outdoor air (for which the EPAQS standard was derived) the level of protection provided by the standard may differ when applied to indoor air from that afforded in relation to outdoor air exposures at the same B(a)P concentration. It is also important to bear in mind that these air quality standards relate to health impacts of chronic exposure. The PEs measured in this study are however short-term (24-h) exposures due to resource constraints. Hence caution should be taken when extrapolating the short-term exposures reported in this study to facilitate comparison with such standards.

The highest benzene level (30.3 μg/m^3) corresponded to an ETS-exposed subject living in a suburban first line property, while the highest 1,3-butadiene level (6.3 μg/m^3) was found in an ETS-exposed subject living in a suburban house away from traffic. Other cases of higher VOC exposures were attributed to photocopying (e.g., ethylbenzene, 182 μg/m^3), painting (e.g., ethylbenzene, 25.7 μg/m^3), fumigating carpets (e.g., styrene, 61.6 μg/m^3), and using moth balls at home (e.g., naphthalene, 12.6 μg/m^3). As regards the PAH extremes, the two highest BaP levels (25.3 ng/m^3 and 5.4 ng/m^3) were measured in subjects burning wood in the fireplace. Three other subjects had BaP levels higher than 1 ng/m^3. The possible causes for two of these extremes were related to shopping in a heavily trafficked road in Birmingham (A38 Road) for 1.5 h and to driving for a total of 3 h on that day and doing a lot of photocopying at work (*34, 35*). However, the activity diary of the third extreme subject, who lived in Birmingham city center and worked from home, did not justify the higher PAH values though data for toluene, benzene, *m*-,*o*-,*p*-xylenes, and 1,3-butadiene (5.73 μg/m^3) were high. Elevated concentrations of 1,3-butadiene generally arise from exposure to traffic fumes or ETS, and hence this remains unexplained.

A number of generic factors have been shown to be associated with PE concentrations. The presence of an integral garage within the home (for VOC) and exposure to environmental tobacco smoke (for VOC and PAH) are associated with higher PEs. When the home is a first-line property there is a statistically significant higher level of toluene but other VOCs and PAHs are not significantly elevated. Personal activities such as wood-burning fireplace use, ETS exposure, photocopying, and DIY activities were identified as contributors to high personal exposures. The accuracy of reporting of time-activity diaries proves to be essential in interpreting sources affecting personal exposures.

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Supporting Information Available

Further details regarding quality control and quality assurance procedures, analytical methodology, subject characteristics and key determinants distribution. This information is available free of charge via the Internet at http://pubs.acs.org.

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