London Wide Environment Programme

Polycyclic Aromatic Hydrocarbon Survey 2004-2005

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London Wide Polycyclic Aromatic Hydrocarbons Survey

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Prepared By

J Brookes
Consultant

J Booker
Principal Consultant

Prepared For

London Borough of Bexley
London Borough of Brent
London Borough of Greenwich
London Borough of Hackney
London Borough of Hammersmith and Fulham
London Borough of Richmond
City of Westminster

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Summary

The primary aim of the London-Wide PAH survey, which has been in operation since 1991, is to assess the exposure of the London population to PAHs. The data from this survey have been compared with guidelines for PAHs in order to increase the understanding of the scale of the pollution problem posed by PAHs in London.

There is some evidence from epidemiological studies to implicate inhaled PAHs as a cause of lung cancer. It is estimated that exposure to Benzo(a)pyrene *(B[a]P)* results in a unit lifetime risk of about 10^{-4} per ng m^{-3}. This risk has been expressed in terms of B[a]P rather than total PAH because B[a]P is the best-known carcinogen in the PAH group, and 'total PAH' is not a well-defined substance.

At present, there are no national standards for PAHs in the UK. However, in a report issued during 1999, the UK Expert Panel on Air Quality Standards *(EPAQS)* recommended setting a national Standard for PAHs using annual average concentrations of B[a]P as a marker for PAH. A guideline of 0.25 ng m^{-3} B[a]P had been recommended as the maximum desirable level for PAHs in ambient air if health effects are to be avoided[17]. Since the Air quality Strategy for England, Scotland, Wales and Northern Ireland was published in January 2000, an addendum to the Air Quality Strategy was issued during 2003. This adopted a new objective of achieving the EPAQS recommended standard of 0.25 ng m^{-3} B[a]P as an annual average by the end of 2010. This will not be a national standard and objective for the time being and will not be placed in regulations for the purposes of LAQM[21].

Until recently, no EU ambient air quality limit values for PAHs existed, however following a consultation with the European Commission, in December 2004 the European Parliament published its 4th Daughter directive (2004/107/EC). As with EPAQS proposed limit values, the directive adopted target values based on maximum annual average concentration of B[a]P as a marker for PAH. The PAH target value is 1.0ng m^{-3} to be achieved by 2012, and is based on the total benzo(a)pyrene content in the PM_{10} fraction.

With respect to sources and chemical origin of PAHs, estimates of atmospheric emissions of PAHs by source type in the UK indicate that the major sources are stationary and sensitive to the type of plant and fuel. In urban areas, however, mobile sources are likely to be the major contributors to PAH emissions, and diesel emissions are thought to be the primary source of urban PAH. The measurement programme for the London-Wide PAH survey was devised to reflect this fact and that PAH

levels tend to vary seasonally. Therefore, most of the locations chosen for sampling were close to busy roads with measurements taken in both summer and winter seasons. For comparison a background and roadside (intermediate) site were also included. Sixteen PAH compounds were selected for measurement, based upon US EPA recommendations with respect to air monitoring programmes.

Seven measurement sites were chosen, one in each of the Boroughs participating in the 2004-05 survey. Sites were classified as roadside (busy), roadside (intermediate) and background. Roadside (busy) sites were generally within 20 m of a busy road, for example at the facade of buildings adjoining the road; roadside (intermediate) sites were within 20m of an intermediately busy roadside; background sites were those located at a distance greater than 40 m from any roadside.

The survey included one background, one roadside (intermediate) and five roadside (busy) sites. Results obtained from monitoring at these locations within the survey highlighted some general trends in the data set:

- Species measured throughout the year followed a similar pattern across all sites with a clear pattern in concentration of species. There were consistently higher concentrations of compounds, which have been associated with vehicular emissions, such as fluoranthene and pyrene. This is a general feature of PAH concentrations surveyed in urban areas.

- Use of particulate and vapour phase B[a]P as an index of carcinogenicity indicates that the concentration of B[a]P at all sites would not exceed the EPAQS guideline.

Continued policy measures to reduce emissions from diesel cars and heavy goods vehicles have included stringent emission standards *(Euro Standards)* and an improvement in the quality of diesel fuel. Within the UK, road transport sources of B[a]P declined from 5.3t (1990) to 0.69t (2000) and are predicted to fall even further by 2020 to 0.22t [(17)]. Industrial sources of B[a]P related to aluminium production have fallen dramatically since 1990 reducing B[a]P concentration in the vicinity of plants, which is expected to decrease further due to improvements in industrial abatement and process controls [(17)].

1 Introduction

Polycyclic aromatic hydrocarbons *(PAHs)* are a large group of organic compounds found throughout the environment. Chemically, PAHs are groups of hydrocarbons consisting of two or more benzene type rings. They are mainly produced during pyrolysis or because of incomplete combustion of carbon compounds *(fossil fuels)*. In urban and industrial atmospheres, the formation of PAHs is almost entirely anthropogenic in origin with vehicle emissions representing a major source. Many PAH species, including the best known of them, benzo(a)pyrene *(B[a]P)* as shown in Figure 1, have been identified as carcinogenic. Airborne PAH concentrations are typically higher in urban areas compared with rural areas and exposure of the urban population to PAHs may be linked to the higher incidence of respiratory problems in towns compared with rural areas.

PAHs are present in both vapour and particulate phases in the atmosphere. The vapour phase consists of predominantly lower molecular weight PAHs whilst higher weight PAHs condense onto very small particulate nuclei to form particulate phase PAHs. Partition between the vapour and particulate phases is known to be temperature dependent and hence seasonal variation occurs in the concentrations of PAHs in the particulate phase. Studies have shown that many PAHs are bound to particles, such as B[a]P which is predominantly found in the particulate phase. However, the potential contribution of the vapour phase should not be ignored and various studies incorporate the gas phase to provide the total PAH concentration.

The primary aim of the London-Wide PAH survey, which has been in operation since 1991, is to assess the exposure of the London population to PAHs. The sixteen PAH species, as recommended by the United States Environmental Protection Agency *(US EPA)* [11] for air pollution monitoring programmes, are monitored at sites in participating London Boroughs each month of the year. This enables possible identification of seasonal and intersite variations in the relative proportions of PAH species. The London-wide PAH survey is developing an important database that is increasing the understanding of the scale of the pollution problem posed by PAHs in the capital.

This report summarises and comments on the PAH measurements taken on behalf of London Boroughs as part of the 2004-05 monitoring programme. The results are assessed in relation to guidelines for PAHs and the results of other surveys of PAH concentrations in urban areas in the UK. The potential effects of air pollution on human health are also described, in particular the specific effects that concentrations of PAHs

recorded in the 2004-05 survey may have on the health of the London population.

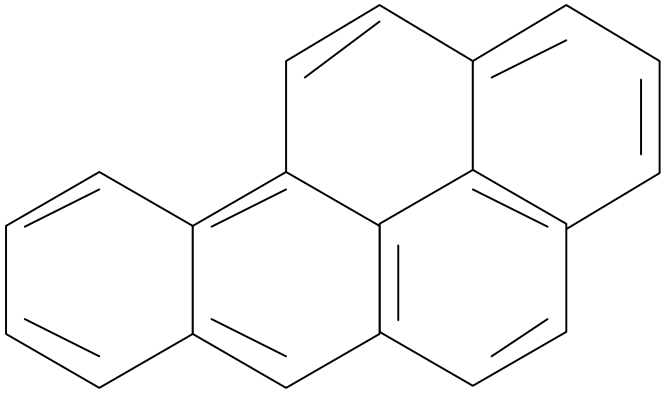

Figure A. The Molecular Structure and Chemical Formula of Benzo(a)pyrene B[a]P [13].

Benzo(a)pyrene $C_{20}H_{12}$

Alternative names by which benzo(a)pyrene has been known.

- Benzo[a]pyrene
- Benzo[α]pyrene
- Benz[α]pyrene
- 1,2-benzopyrene
- PAH EPA no. 73

2 Sources and Chemical Origin of PAH

Atmospheric emissions of B[a]P for the UK since 1990 and projected future emissions from 2000 are shown in Table A. It can be seen from the Table that certain industrial processes, such as anode baking for aluminium production, which were major sources of B[a]P have declined since 1990 from 23t to 1.7t. These sources would strongly influence the B[a]P concentration near plants. Such a decline in emissions is due to reduced industrial coal use, field burning and improvements in industrial abatement [17]. Projected emissions of B[a]P across the source types are expected to decline further due to reductions in the use of domestic solid fuel, improved controls on aluminium anode baking processes and importantly, lower emissions from vehicles. The future trend in concentration of B[a]P indicates that emissions are likely to plateau in 2005 onwards to a level of around 6.5t per year across all sectors.

Table A: UK Annual B[a]P Emissions 1990-99 and Projections to 2010 (tonnes)[17]

Tonnes	1990	1995	2000	2005	2010	2015	2020
Road Transport	**5.3**	2.1	**0.69**	0.34	0.24	0.22	0.22
Fires	31.0	2.9	2.9	2.9	2.9	2.9	2.9
Aluminium production	1.9	1.5	0.18	0.24	0.25	0.26	0.27
Anode baking	23.0	15.0	1.7	0.35	0.37	0.39	0.40
Coke and SSF production	0.16	0.13	0.13	0.1	0.11	0.11	0.10
Domestic solid fuels	6.3	3.4	3.7	2.2	2.2	2.3	2.4
Industrial combustion	0.42	0.21	0.10	0.15	0.14	0.13	0.16
Other sources	0.28	0.24	0.20	0.21	0.21	0.21	0.21
Total	**68.0**	**25.0**	**9.5**	**6.5**	**6.4**	**6.5**	**6.6**

In previous years road traffic emissions have been a dominant source of total PAH and B[a]P. Studies have shown that up to 90% of total PAH and B[a]P at roadside locations and 40% at background locations can be attributed to this source [15, 16]. Contrary to such studies recent assessments of future PAH concentrations in the UK suggest that vehicles may no longer be a significant source of B[a]P [17]. Evidence from measurements at the Marylebone Road monitoring site in London *(a kerbside site)* shows that concentrations adjacent to heavily trafficked roads are not significantly higher than at urban background locations [17]. Updated vehicle emissions inventories also indicate smaller releases from vehicles than previously estimated.

The First Report of the Department of the Environment Quality of Urban Air Review Group[1] reported that firm evidence had been provided

to demonstrate that the bulk of particulate derives from unburned fuel. It also reported that the air/fuel ratio *(AFR)* is established as a significant determinant of PAH emissions from spark-ignition vehicles, and cold-starts were found to increase emissions substantially.

Particulate PAHs contaminate rain and, together with dry deposition of particles and vapour, leads to contamination of soil, crops and plants that may then be responsible for concentrations of PAHs found in food and water. More substantial amounts of PAH, including B[a]P, may be found in food because of various methods of cooking, preservation and food storage[13].

3 Effects of PAH on Human Health

Determination of the risk to human health from air pollutants requires knowledge of five basic factors;

- the source and emission rates of the pollutant;
- its dispersion and transport in the atmosphere;
- the exposure of individuals to the pollutant;
- the dose of pollutant absorbed by these individuals;
- the adverse health effect resulting from these doses.

The distinction between exposure and dose is an important one.

EXPOSURE refers to contact between an airborne pollutant and the human body: it is usually expressed in terms of the pollutant concentration, the duration of exposure, and the frequency of exposure.

DOSE, in contrast, refers to the amount of pollutant, which is actually absorbed by the body and is a function of concentration and time.

3.1 General Assessment of Human Exposure to Air Pollution

The PAH concentrations reported in this document reflect the emissions, dispersion, chemical transformation and transport of pollutants. However, these measurements are based on a limited number of outdoor locations in the London area, so it is important to assess how well these are likely to reflect the actual exposures of individuals as they move around this area.

Defining human exposure to air pollutants is not straightforward. Most pollutant measurements are based on static devices that provide a record of the concentrations at one point in space. However, humans are highly mobile and in the course of a day may spend time in many different locations, e.g. in the house, in the garden, in the office, in the car, on the street, etc. Thus, the exposure of an individual over the course of the day will be a complex function of the temporal variation in pollutant concentration, the spatial variation in pollutant concentration, and the activity pattern of the individual. Since it is the actual exposure of an individual which will determine the likelihood of any adverse effect on the health of that individual, the ability to determine personal exposure is of crucial importance in assessing the health effects of air pollution.

There are several methods of obtaining an estimate of personal exposure to pollutants, which are described as:

- The DIRECT approach where personal monitors are worn or carried by individuals. Most personal monitors, such as those developed for NO_2 or respirable particles, can only provide a time-averaged concentration, although continuous personal monitors have been used for CO and could be developed for other pollutants. The need for portability, robustness and lightweight units means that personal monitors are usually less sensitive than the equivalent fixed-site monitor *(FSM),* and this can restrict their use at ambient concentrations. The use of direct personal monitoring is expensive and time-consuming, and is thus less practical for the estimation of the personal exposure of populations, or large groups;

- The INDIRECT approach where estimates of personal exposure are based on models, which combine information on pollutant concentrations with a number of other pieces of information. Such models are usually based on the concept of the microenvironment, which refers to locations, such as kitchen, living room, office, street, restaurant, car, or garage, in which pollutant concentrations are broadly determined by the same factors. To obtain an indirect estimate of personal exposure, a model must combine information on the time-activity patterns of individuals - when and for how long they occupy each microenvironment and on the characteristics of these microenvironments - for example, what type of cookers are used in the kitchen, the traffic densities on the streets, and on the pollutant concentrations. Estimates of pollutant concentrations may be obtained from personal monitoring, from monitoring at a fixed location in each microenvironment, or from computer models of indoor or outdoor pollutant dispersion.

3.2 Assessment of Health Effects of Air Pollution

Broadly speaking, there are two major types of concern in relation to health effects of air pollution. The first relate to the short-term, *(acute)* effects of episodes of elevated concentrations of pollutants, which historically have been associated with short-term increases in mortality and hospital admissions. The second relate to the longer-term *(chronic)* effects to lower pollutant concentrations, which may influence the prevalence of a range of respiratory and other diseases. A number of approaches have been used to assess these health impacts, but each of these has important limitations, which need to be recognised.

- **Laboratory studies of animals**. These have been very important in demonstrating the potential health effects of a range of compounds, and in investigating the possible mechanisms involved. However, the differences in physiology between humans and animals, and the uncertainty in the actual dose to be compared, mean that they are of limited value in predicting likely human effects of exposure to specific concentrations of pollutants.

  Car exhaust emissions have been linked to a decrease in fertility. Benzo[a]pyrene which is a common car exhaust compound causes a significant reduction in fertility in test animals with fertility further lowered when animals were exposed to both B[a]P and lead simultaneously. Results showed a reduction in ovarian weight and a "marked reduction in ovarian follicles"[20].

- **Laboratory studies of humans**. In theory, exposure of human subjects to a range of pollutant concentrations and combinations can provide valuable information relevant to the assessment of the health effects of ambient pollutant concentrations. In practice, there are a number of important limitations of this approach. Firstly, it can only be used to study the effects of short-term exposure. Secondly, for ethical reasons, it is not possible to conduct experiments on many of the individuals - elderly people, children, and those with severe respiratory diseases - who are most at risk during short-term episodes of high concentrations. In addition, many studies are of limited value because of specific features of their design, e.g. low replication and a lack of definition of prior exposure of subjects to air pollutants.

- **Epidemiological studies**. The key advantage of the epidemiological approach is that the health effects are assessed using actual exposure levels and patterns incorporating pollutant mixtures and environmental conditions. Some of these studies can be carried out using routinely collected data, and it is relatively easy to place observations into a public health context. On the other hand, such studies are subject to problems with accuracy of measuring exposure and outcome, and with methodological problems such as confounding factors.

3.3 Acute Effects of Short-Term Exposures to Air Pollutants

The considerations that apply to exposure measurement have already been described. The health outcome variables in acute studies are as follows;

- mortality (all-cause and cause-specific);
- hospital admissions (all-cause and cause-specific);
- attendance at Accident and Emergency Departments;
- general practitioner attendance's;
- school absences;
- changes in pulmonary function in susceptible groups;
- increase in medication use in susceptible groups;
- symptoms in susceptible groups;
- changes in pulmonary function in normal individuals;
- general symptomatic changes in normal individuals.

The interpretation of outcome data from acute studies is not necessarily straightforward as outcome data is affected by many variables. For example; the influence of meteorological conditions on pollutant concentrations; factors associated with health outcome, such as temperature, humidity, pollen count and indoor air pollution; reporting bias when respondent's knowledge of the research topic influences their recording of symptoms; and problems with comparability e.g. between different hospitals.

3.4 Chronic Effects of Long-Term Exposures to Air Pollution

It is important to know whether the presence of pollutants, even at quite low concentrations, increases the risk of major diseases such as chronic respiratory disease and cancer. The theoretically ideal research design is a cohort study, in which large groups of people with known exposures are followed up many years later, and the risk (incidence or mortality rate) of those with high exposures is compared with that of non-exposed or low-exposed individuals. The long latent period in the development of such diseases means that such studies require a long follow-up period, and are therefore expensive.

A more commonly used design is the cross-sectional study, in which disease rates are compared with current concentrations of pollutants. However, the latter do not necessarily reflect historical levels accurately.

Both types of design have the problems outlined in the previous section, together with additional problems. The frequency of the occurrence of serious disease conditions is low, which means that study populations must be very large to have sufficient statistical power. The list of potential confounding variables is long, as it includes not only time-dependent ones as in acute studies, but any factor that may be associated with both the outcome and the exposure level, for example cigarette smoke, housing conditions and other socio-economic variables.

3.5 Human Exposure to Polycyclic Aromatic Hydrocarbons *(PAHs)*

Owing to the limited data available, it is not possible to make an accurate assessment of the exposure of the general population to PAH. However, it is recognised that exposure to ambient levels of PAHs in the atmosphere constitutes a health issue. Concentrations outdoors are likely to be highest in cities where domestic coal burning is still significant; close to major industrial sources and at roadside locations. In addition, PAHs are an important carcinogenic component of cigarette smoke, although the concentration is lower in modern low-tar cigarettes. There is some limited and dated information regarding the relative dose of PAHs received from air, water and food which suggests that total PAH intake through ingestion of food is substantially greater than that through inhalation. The study of personal exposure and health effects is further complicated by the lack of knowledge of comparative importance of short-term exposure to high concentrations or background levels. For non-smokers the greatest risk is likely to arise via food contaminated by PAHs deposited from the atmosphere. Assessment of human exposure to PAHs is further complicated by the likely temporal and spatial differences in the composition of PAH mixtures.

3.6 Effects of Polycyclic Aromatic Hydrocarbons on Human Health

One of the earliest reports of occupational cancers and probable recorded effects of PAH on health were recorded in the 18th century when Sir Percival Pott described the unusual prevalence of tumours in chimney sweeps[8]. Elevated incidence of tumours were reported later in workers in early coal tar and oil processing plants. The positive link between PAH compounds and cancer was identified during the 1920's and 30's by Kennay and his co-workers at the Research Institute of the Cancer Hospital in London and proved to be a major step in the isolation of carcinogenic compounds [8]. One of the first such isolated chemicals to be proven to be linked to tumour formation was Benzo[a]pyrene [8].

Subsequent observations from experimental studies have shown several compounds within the PAH group to be mutagenic and carcinogenic, and this is true of actual air or of condensates taken from certain industrial sources, vehicle exhausts, and ambient air. The carcinogenic and mutagenic activity of PAHs appears to be concentrated in the 4-7 ring of PAH-containing fractions and derives from the formation of breakdown products as the body attempts to detoxify the pollutant. Benzo[a]pyrene is broken down to produce its diol epoxide, which is then able to rotate one of the amino acids in DNA so potentiating mutagenicity [(9)]. The higher molecular weight PAHs, which tend to be found in the particulate phase, are reported to be the most carcinogenic [(10)]. The associated particulate usually measure less than a micron in diameter and can penetrate deep into the lungs. Particulate in themselves pose a significant health threat *(EPAQS 2001)*, but there is also a strong likelihood of a synergistic effect between particulate and PAHs as some particulate are known to promote the carcinogenic effects of other chemicals. A classification of the carcinogenity of PAH compounds is shown in Table E *(Section 6.1)*.

The most obvious links between health and PAHs have been derived from workers exposed to high concentrations and there is evidence, from epidemiological studies on coke-oven workers and coal-gas workers, to implicate inhaled PAHs as a cause of lung cancer. The evidence is sufficient to form a quantitative estimate of the risk, and several such estimates have been published during the past decade. Two of the most authoritative are those of the World Health Organisation *(WHO)* [(2)] and Doll and Peto [(3)]; these are in broad agreement and suggest that exposure to benzo[a]pyrene *(B[a]P)* at a concentration of 1ng of B[a]P per cubic metre of air, throughout life, carried a risk of lung cancer of 1 in 10,000 or 0.0001%. This is a risk over and above the already existing risk of lung cancer, which in this country is about 6% averaged over the whole population *(smokers plus non-smokers)*; for non-smokers alone the risk is about 0.8%.

The risk has been expressed in terms of B[a]P rather than total PAH as most research has been conducted on this carcinogenic species, and 'total PAH' is not a well-defined class of compounds. However, it is important to remember that in deriving risk estimates for B[a]P in this way, a number of assumptions have been made. These include the assumption that the coke workers are exposed to the same PAH profile as the general public. It seems likely that occupational exposure to diesel exhausts may be associated with an increased risk of lung cancer, but it has not been proven. Epidemiological studies into the effect of PAHs on human health are subject to problems of accuracy of measuring exposure and outcome in light of the long latency of some effects and by confounding effects. In the case of PAH exposure the influence of exposure to cigarette smoking is of particular importance.

Furthermore, with respect to the influence of smoking, it seems likely that the combined effects of smoking and B[a]P are much more than additive. The unit risk of 10^{-4} is therefore valid only for a population having the same smoking habits as the exposed worker population. The risk for non-smokers, or for any group who smokes less than the worker group did, would be expected to be less than 10^{-4}.

4 Guidelines and Standards

There are currently no national standards or objectives for PAHs in the UK. However, the Expert Panel on Air Quality Standards *(EPAQS)* recommended in its report *(published in 1999)* a PAH guideline of 0.25 ng m^{-3} B[a]P as an annual average which applies to the total PAH concentration. This is intended to represent the maximum desirable level for PAHs in ambient air if health effects are to be avoided. Since the Air Quality Strategy for England, Scotland, Wales and Northern Ireland was published in January 2000, an addendum to the Air Quality Strategy was issued during 2003. This adopted a new objective of achieving the EPAQS recommended standard of 0.25ng m^{-3} B[a]P as an annual average by the end of 2010. This will not be a national standard and objective for the time being and will not be placed in regulations for the purposes of LAQM[21].

Benzo[a]pyrene is used as a marker for the purposes of the guideline and represents the total mixture of PAH in the UK. This is considered to be the most carcinogenic PAH and thus choosing a level of this PAH species that is considered to pose a risk to the public that is so small as to be undetected would provide an estimate of a worse case scenario. It was recommended that techniques and monitoring should be consistent with those used by the Department for Environment, Food and the Rural Affairs.

4.1 European PAH Target Values

The European Union (EU) values are generally derived from WHO guideline values. Thus if an UK objective is derived from an EU limit value, it will be the WHO guideline value rather than the relevant EPAQS recommendation, that forms the basis of the objective.

Until recently, no EU ambient air quality limit values for PAHs existed and some European countries set their own national guidelines for PAH concentrations in ambient air. In the Netherlands, an interim goal is to reduce the annual average concentration of B[a]P to 5 ng m^{-3} [4]. A guideline for the annual average B[a]P concentration proposed by the German Environmental Agency is 10 ng m^{-3} [4].

Following consultation with the European Commission, in December 2004 the European Parliament published its 4th Daughter directive (2004/107/EC). As with EPAQS proposed limit values, the directive adopted a target value based on maximum annual average concentration of B[a]P as a marker for PAH. The PAH target value was a value of 1.0ng

m^{-3} to be achieved by 2012. This is based on the total benzo(a)pyrene content in the PM_{10} fraction.

In order for member states to assess ambient air concentrations and deposition rates, other PAHs are required to be monitored at a limited number of sites where B[a]P is monitored. These compounds should include at least: benzo(a)anthracene, benzo(b)flouranthene, benzo(j)flouranthene, benzo(k)flouranthene, indeno(1,2,3-cd)pyrene, and dibenz(a,h)anthracene. In addition, monitoring sites should be selected in such a way that geographical variation and long term trends can be identified.

5 Concentrations of PAH in Urban Areas

Compared with other pollutants, the monitoring of speciated organic compounds in urban areas of the UK is still in its infancy and long time-series databases are not currently available.

Two National monitoring networks for the measurement of PAH have been established by AEA Technology's National Environmental Technology Centre ***(netcen)*** on behalf of the Department for Environment, Food and Rural Affairs *(Defra)* and devolved administrations. The UK TOMPS *(Toxic Organic Micropollutants)* network *(6-sites)* was established in the early 1990's to provide information on long-term trends in ambient concentrations of dioxins and PAHs. The more recently established PAH Network was designed to improve knowledge of the levels of PAHs throughout the UK [18]. There are currently 23 national PAH monitoring sites measuring 54 compounds with a view to improve the characterisation of ambient PAH levels in the UK [17]. These additional sites are being located mainly at urban background locations whereas the 15 sites are situated at various city centre, rural and near to industrial sources. Sites from the TOMPS network have been established in London *(Westminster)* and Manchester since 1991 and additionally at Middlesborough and Hazelrigg since 1992 to monitor PAH, polychlorinated biphenyls *(PCBs)* and dioxins.

Past data from the TOMPS network have generally shown that site specific influences are important and significant differences are discernible between what might have been thought to be similar locations. The data variability between urban sites was found greater than expected. **netcen** recommended that greater sampling flexibility and a wider range of TOMPS sites would be required in order to quantify the influence of geographical location, proximity of potential emission sources and seasonal variation on PAH concentration. In 1999 a new PAH network was established. Table B lists the national PAH monitoring sites including potential additional sites and includes the annual average B[a]P concentration from 1999 to 2004.

Table B: Currently Operating National PAH Monitoring Sites and the Annual Average B[a]P Concentrations (ng m^{-3}) from 1999 to 2004 [17].

Site	Site Type	Monitoring commenced	Concentration (ng/m3) 1999	2000	2001	2002	2003	2004
Ashington	Urban Industrial	1999	0.20	0.17	0.20	0.15	0.19	0.15
Bolsover	Industrial	1999	0.24	0.25	0.28	0.24	0.46	0.22
Glasgow	Urban	1999	0.20	0.12	0.12	0.12	0.07	0.07
Hazelrigg	Semi-rural	1992	0.06	0.06	0.08	0.04	0.04	0.02
Holyhead	Urban Industrial	1999	0.11	0.11	0.15	0.18	0.14	
High Muffles	Rural	1997	0.06	0.04	0.05	0.03	0.05	0.02
Kinlochleven	Urban Industrial	1999	7.40	2.27	0.34	0.38	0.21	0.32
Lisburn	Urban	1999	0.75	0.95	0.94	0.65	1.00	0.62
London	Urban	1991	0.34	0.14	0.14	0.13	0.12	0.07
Manchester	Urban	1991	0.15	0.24	0.34	0.12	0.24	0.11
Middlesborough	Urban Industrial	1992	0.24	0.28	0.37	0.15	0.24	0.14
Newport	Urban Industrial	1999	0.23	0.35	0.36	0.19	0.11	0.10
Port Talbot	Urban Industrial	1999	0.24	0.59	0.40	0.34	0.47	0.29
Scunthorpe	Urban Industrial	1999	0.38	1.16	0.34	1.35	1.30	
Stoke Ferry	Rural	1997	0.11	0.09	0.09	0.08	0.08	0.04
New Sites			**1999**	**2000**	**2001**	**2002**	**2003**	**2004**
Belfast		2001			0.37	0.13	0.08	0.15
Birmingham		2001			0.16	0.13	0.16	
Brent	Suburban	2002				0.18	0.21	0.10
Bromley	Roadside	2001			0.20	0.25	0.10	0.19
Hove		2002				0.18	0.16	0.09
Leeds		2001			0.16	0.18	0.21	
Newcastle		2001			0.11	0.12	0.16	0.06
Speke		2001			0.08	0.14	0.14	0.10

(Extracted from the Air Quality Strategy - Consultation Document 2001[17] and updated with the means calculated data from the UK National Air Quality Information Archive)

PAH measurements made during 2004 at the Manchester, Middlesborough and London TOMPS sites are given in Table C. Measurements for one rural site also in the TOMPS network are shown for comparison.

Table C: 2004 Annual Average Particulate PAH Concentrations (ngm^{-3}) for London, Manchester, Middlesbrough and High Muffles

Compound	London	Manchester	Middlesbrough	High Muffles
Acenaphthylene	0.45	-	-	-
Acenaphthene	0.94	2.20	12.0	0.29
Fluorene	4.00	2.60	21.0	1.20
Phenanthrene	19.00	45.00	56.0	3.20
Anthracene	0.43	1.40	2.9	0.13
Fluoranthene	2.00	3.00	2.6	0.54
Pyrene	1.60	2.50	1.2	0.30
Benz[a]anthracene	0.08	0.13	0.12	0.03
Chrysene	0.15	0.32	0.29	0.06
Benzo[b]fluoranthene *	0.19	0.19	-	-
Benzo[k]fluoranthene	0.04	-	0.09	0.02
Benzo[a]pyrene	**0.76**	**0.11**	**0.14**	**0.03**
Indeno[1,2,3 cd]pyrene	0.10	-	-	-
Dibenz[ac/ah]anthracene	0.02	-	-	-
Benzo[ghi]perylene	0.16	0.20	0.16	0.03
Total PAH	**29.44**	**35.98**	**28.49**	**9.2**

(Means calculated from National Air Quality Information Archive, TOMPS data.)

6 The Measurement Programme

6.1 General

Mobile sources are contributors to PAH emissions and exhaust emissions from traffic are an important source of PAHs in London and many other towns. Most of the locations chosen for sampling were therefore close to busy roads, with the remainder located at greater distances from the roadside, in order to provide some information on intermediately busy roads and background pollution levels.

Air pollution levels tend to be higher during winter than summer because of less favourable conditions for atmospheric dispersion during the winter months. For PAHs, this effect is likely to be enhanced by the increased use of fossil fuels for the heating of homes and offices during winter. Measurements were therefore made throughout the year.

It is generally agreed that B[a]P by itself is not a satisfactory index of total PAH, but there is no universally agreed selection of PAHs, which performs such a role. About 500 PAH compounds have been detected in ambient air and it is impracticable to measure all but a small fraction of this number.

The most authoritative recommendation with respect to the selection of a species for measurement is probably that of United States Environmental Protection Agency *(US EPA)*. They have listed 16 PAHs as priority pollutants for air monitoring programmes, due to their toxicity and common occurrence. Thus, for the purpose of the current assessment, monitoring has followed convention set by the US EPA. Other agencies such as the International Agency for Research on Cancer *(IARC)* have identified a sub-set of six of these PAHs as probable or possible carcinogens. The United Nations Economic Commission for Europe Protocol on Persistent Organic Pollutants *(POPS)* also focuses upon four of these compounds. In addition, for compilation of some EU inventories, a sub-set Borneff 6 has been used [17]. These sub-sets and PAH species are shown in Table D.

Table D: PAH Species and Sub-sets [17]

Species	USEPA priority pollutants[1]	IARC Group 2a[3]	IARC Group 2b[3]	UNECE POPs Protocol	"Borneff 6"
Napthalene	✓				
Acenapthene	✓				
Acenapthylene	✓				
Fluorene	✓				
Anthracene	✓				
Phenanthrene	✓				
Fluoranthene	✓				✓
Pyrene	✓				
Benz[*a*]anthracene	✓	✓			
Chrysene	✓				
Benzo[*b*]fluoranthene	✓		✓	✓	✓
Benzo[*j*]fluoranthene			✓		
Benzo[*k*]fluoranthene	✓		✓	✓	✓
Benzo[*a*]pyrene	✓	✓		✓	✓
Dibenzo[*a,h*]anthracene	✓	✓			
Dibenzo[*a,c*/*a,h*]anthracene					
Dibenzo[*a,e*]pyrene			✓		
Dibenzo[*a,h*] pyrene			✓		
Dibenzo[*a,I*] pyrene			✓		
Dibenzo[*a,L*] pyrene			✓		
Indeno[*1,2,3-c,d*]pyrene	✓		✓	✓	✓
5-methylchrysene			✓		
Benzo[*g,h,I*]perylene	✓				✓
Coronene					

All Species are currently monitored within the PAH network

[1] Included in National Atmospheric Emissions Inventory. Sum of emissions corresponds to total PAH

[2] Possible human carcinogen

[3] Possible human carcinogen

Many factors influence the inclusion of a particular compound in a measurement programme. These include its carcinogenic potential, sufficient atmospheric concentration to permit reliable measurement using the analytical techniques and reference standards currently available, and whether the relative concentrations of particular compounds give an indication of the main source of PAHs.

The US EPA list therefore contains a selection of compounds likely to occur at relatively high levels and includes members across a wide range of

molecular weights, from the volatile 2-ring naphthalene to those of higher molecular weight, which are likely to be predominantly in the particle phase.

Human carcinogenicity data are available only for PAH mixtures, and our knowledge of the carcinogenicity of individual PAHs therefore comes from *in vitro* and animal studies. There is no definitive 'carcinogenicity classification' of PAH compounds, but two authoritative commentaries are in general accord [2 & 3]. A rough classification based on these two commentaries is given in Table E.

Table E: The PAH Compounds Measured

Compound and Abbreviation		Cancer Rating[a]	Rings	Mol wt
Naphthalene	Np	?	2	128
Acenapthene	ACE	-	3	166
Fluorene	FL	-	3	166
Phenanthrene	PHE	?	3	178
Anthracene	ANT	-	3	178
Fluoranthene	FLH	?	4	202
Pyrene	PYR	-	4	202
Benzo(a)anthracene	BaA	+	4	228
Chrysene	CHR	+	4	228
Benzo(b)fluoranthene	BbF	+ +	5	252
Benzo(k)fluoranthene	BkF	+ +	5	252
Benzo(a)pyrene	B[a]P	+ + +	5	252
Dibenz(ah)anthracene	DahA	+ + +	5	278
Benzo(ghi)perylene	BghiP	+	6	276
Coronene	COR	-	7	300

Note:

(a) Carcinogenic classification: a dash (-) indicates that there is no evidence for carcinogenicity, a question mark (?) that there is insufficient evidence, and one or more plus signs (+) that there is sufficient evidence.

6.2 Measurement Sites and Sampling Periods

Seven sites were chosen, one in each of the Boroughs participating in the survey. At each site, samples of approximately two weeks duration were taken within each month for the period May 2004 –March 2005. Table F below shows sampling periods across all Boroughs but excludes invalid samples identified from subsequent quality control checks.

Table F: Sampling Periods and Site Classification Across all Boroughs

Authority	Classification	Sampling Periods
Bexley	Background	27/04/04-06/05/04, 20/05/04-03/06/04, 24/06/04-08/07/04, 22/07/04-04/08/04, 26/08/04-13/09/04, 30/09/04-13/10/04, 26/10/04-09/11/04, 24/11/04-08/12/04, 05/01/04-11/01/05, 27/01/05-09/02/05, 24/02/05-08/03/05, 31/03/05-12/04/05,
Greenwich	Roadside (busy)	27/04/04-06/05/04, 20/05/04-03/06/04, 24/06/04-08/07/04, 22/07/04, 22/07/04
Brent	Roadside (busy)	26/04/04-07/05/04, 19/05/04-02/06/04, 25/06/04-09/07/04, 20/07/04-03/08/04, 27/08/04-14/09/04, 01/10/04-14/10/04, 29/10/04-08/11/04, 22/11/04-07/12/04, 04/01/04-13/01/05, 25/01/05-08/02/05, 25/02/05-11/03/05, 30/03/05-13/04/05,
Hackney	Roadside (busy)	26/04/04-06/05/04, 19/05/04-02/06/04, 25/06/04-08/07/04, 20/07/04-03/08/04, 27/08/04-14/09/04, 01/10/04-14/10/04, 29/10/04-08/11/04, 22/11/04-07/12/04, 04/01/05-13/01/05, 25/01/05-27/01/05, 25/02/05-11/03/05, 30/03/05- 13/04/05
Westminster	Roadside (busy)	26/04/04-07/05/04, 19/05/04-02/06/04, 25/06/04-08/07/04, 20/07/04-03/08/04, 27/08/04-14/09/04, 01/10/04-14/10/04, 29/10/04-08/11/04, 22/11/04-07/12/04, 04/01/05-13/01/05, 25/01/05-08/02/05, 25/02/05-11/03/05, 30/03/05-13/04/05
H & Fulham	Roadside (busy)	20/05/04-02/06/04, 20/07/04-30/07/04, 07/09/04-14/09/04, 01/10/04-14/10/04, 29/10/04-08/11/04, 22/11/04-07/12/04, 04/01/05-13/01/05, 25/01/05-08/02/05, 25/02/05-11/03/05, 30/03/05-13/05/05
Richmond	Roadside (intermediate)	27/04/04-06/05/04, 20/05/04-03/06/04, 26/06/04-08/07/04, 22/07/04-03/08/04, 26/08/04-13/09/04, 30/09/04-13/10/04, 26/10/04-09/11/04, 24/11/04-08/12/04, 05/01/05-11/01/05, 27/01/05-09/02/05, 24/02/05-09/03/05, 31/03/05-12/04/05

Sites were classified as "roadside (busy)", "roadside (intermediate)" and "background". Generally, "roadside (busy)" sites were located within 20 m of a busy road, for example at the facade of buildings adjoining the road; "roadside (intermediate) sites" were those within 20m from a

intermediately busy road; and "background" sites were classed as those located at a distance greater than 40 m from the roadside. A summary of site descriptions is shown below in Table G. Descriptions of each site are also provided in Appendix A.

Table G: Summary of Site Descriptions and Classification for Participants in the Polycyclic Aromatic Hydrocarbon Survey 2004-05.

Local Authority	Location	Classification
Site 1 – Bexley	Air monitoring cabin located at Whitehall day centre in Bexley, approx. 200m from the nearest busy road.	Background
Site 2 – Brent Harlesden	Air monitoring cabin located at Budgen car park in Harlesden. Approx. 2m from the nearest busy road.	Roadside (Busy)
Site 3 – Greenwich	Air monitoring cabin located near Crown Woods Way, approx. 10m from the A2.	Roadside (Busy)
Site4 – Hammersmith & Fulham	Air monitoring cabin next to Hammersmith Broadway tube station. Approx. 2m from A315.	Roadside (Busy)
Site 5 – Richmond	Air monitoring cabin located at 75 Castelnau SW13 9RT at the library, approx. 2m from an intermediately busy road.	Roadside (intermediate)
Site 6 – Hackney	Air monitoring cabin located beside the fire station on Old Street, approx. 6m from busy road.	Roadside (Busy)
Site 7 – Westminster	Basement of Westminster Council offices, approx. 6m from Marylebone Road.	Roadside (Busy)

6.3 Scope and Principle of Sampling

Monitoring of polycyclic aromatic hydrocarbons in ambient air was carried out according to TP45-AIR(C), an in-house quality-assured method for the measurement of particulate and vapour-phase PAH. Sample air was first drawn through a filter to collect particulate phase and particulate-bound vapour phase PAH, and then through an adsorbent material to collect vapour phase PAH. The sampling train containing the filter and the adsorbent material was then returned to a central laboratory for analysis.

6.3.1 Sampling Equipment and Preparation

The sampling train consisted of a quartz fibre filter (47mm diameter) in conjunction with a polyurethane foam plug (PUF) (25mm x 76mm). Using an adapter, the filter holder was connected to the by-pass flow of a Tapered Element Oscillating Microbalance (TEOM) fitted with a USEPA approved PM_{10} inlet to perform a 10μm particle size cut-off. Figure B below shows the type of filter and PUF sampler used for sampling.

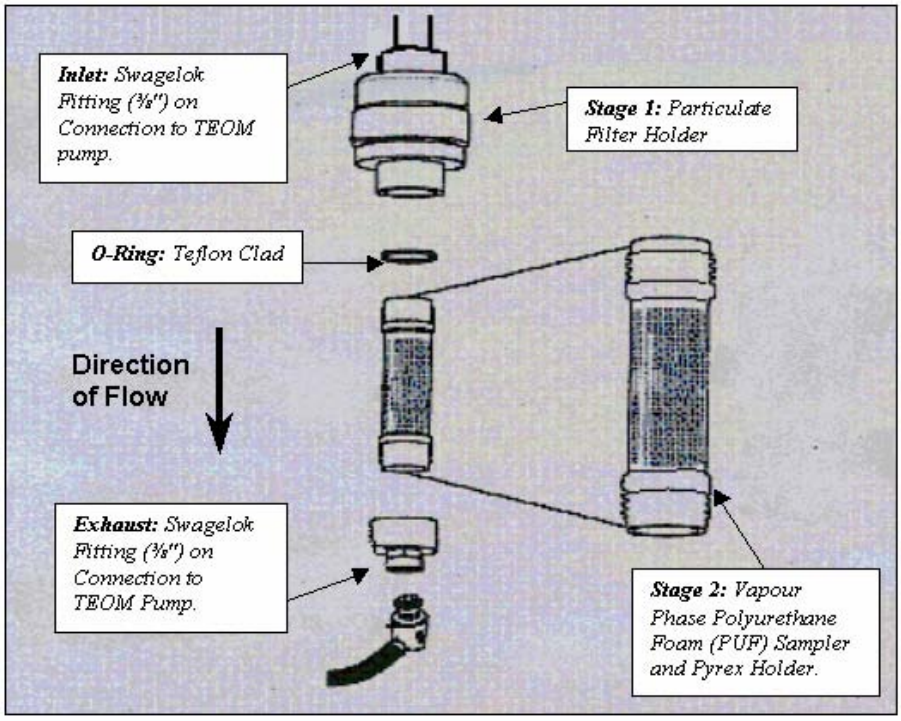

Figure B: Particulate/Vapour-Phase Filter and PUF Sampler.

Prior to sampling the filter holder was thoroughly rinsed with 'Pesticide Residue' quality acetone and air-dried. Dichloromethane solvent was used to soxhlet *(vessel used which refluxes at temperature extracting concentrate PAH; a recycling process*) extract the filter paper. After 8 hours, the filter paper was air dried until no solvent was detected and wrapped with hexane rinsed aluminium foil. The PUF and filter media were then air-dried in a fume hood until no smell of solvent was detected. The sampler was wiped down with dichloromethane and allowed to dry in a fume hood, prior to being assembled and spiked with recovery standards. In order to minimise possible contamination from ultraviolet sources, the Pyrex cartridge was wrapped with hexane or dichloromethane rinsed aluminium foil. The sampling train was assembled and sealed at both ends with *URG* Teflon

screw caps, ready for use. Before sampling the filter was loaded into a filter holder, capped and placed in a sealed polythene bag ready for use.

Once on site a Teflon adapter was used to connect the sampling train into the by-pass flow line of the TEOM. The separation of particle size at 10μm diameter takes place as the sample passes through the PM_{10} inlet. This inlet also protects the sample from adverse weather conditions and ultraviolet sources. A flow splitter separates the total flow of 16.7l/m into two parts: a main flow of 3.0 l/m that enters the TEOM sensor unit through the sample tube, and the auxiliary (bypass) flow of 13.7l/m. Both sample flows entering the TEOM monitor are mass-flow controlled ensuring a constant flow rate for the sample duration. It is the bypass flow rate, which is used for the sampling of PAH. Sampling periods of approximately two weeks were used in order to collect a sample volume of between 217-335m^3 of air ± 3 days.

6.3.2 Analytical Procedure

The multi component samples on the filter medium were pre-spiked with a sampling standard (1000ng of each of D8-naphthalene, D8-acenaphthene, D10-anthracene, D10-pyrene, D12-benzo(b)fluoranthene, D14-dibenzo(ah)anthracene), which are extracted with dichloromethane.

The sample extracts were then evaporated down to a few mls in volume. After the addition of a recovery spike (1000ng of each of D10-fluorene, D12-chrysene and D12-benzo(ghi)perylene) the sample was cleaned up by column chromatography and a syringe standard (1000ng of each of D10-fluoranthene and D12-benzo(a)pyrene) added. The samples were analysed using a Hewlett-Packard 6890/5973 GC-MS system running in selected ion monitoring (SIM) mode.

Prior to analysis, the system was calibrated with reference standards, and a solvent and system blank obtained to ensure accuracy of results. Samples were analysed by a UKAS (United Kingdom Accreditation service) subcontract laboratory for PAH *(USEPA-16*). The subcontract laboratory, currently Harwell Scientifics Ltd, carried out combined analysis of particulate and vapour phase PAH. The limits of detection were calculated from the analysis of blank samples with the result of various detection limits.

7 Results

The concentrations of particulate PAH measured at each site during the survey are given in Appendix B, Tables 1 to 7 respectively. Measured concentrations below the analytical detection limit have not been included nor has any data that failed quality control and which is assumed, suspect. For each compound, at each site, the average concentration values were calculated to give an estimate of the overall annual mean concentration. These estimates are given in Appendix B, Table 8. Figure 2 below summarises mean B[a]P concentration for the particulate phase measured for participants within the survey. Mean values have been calculated using all available data.

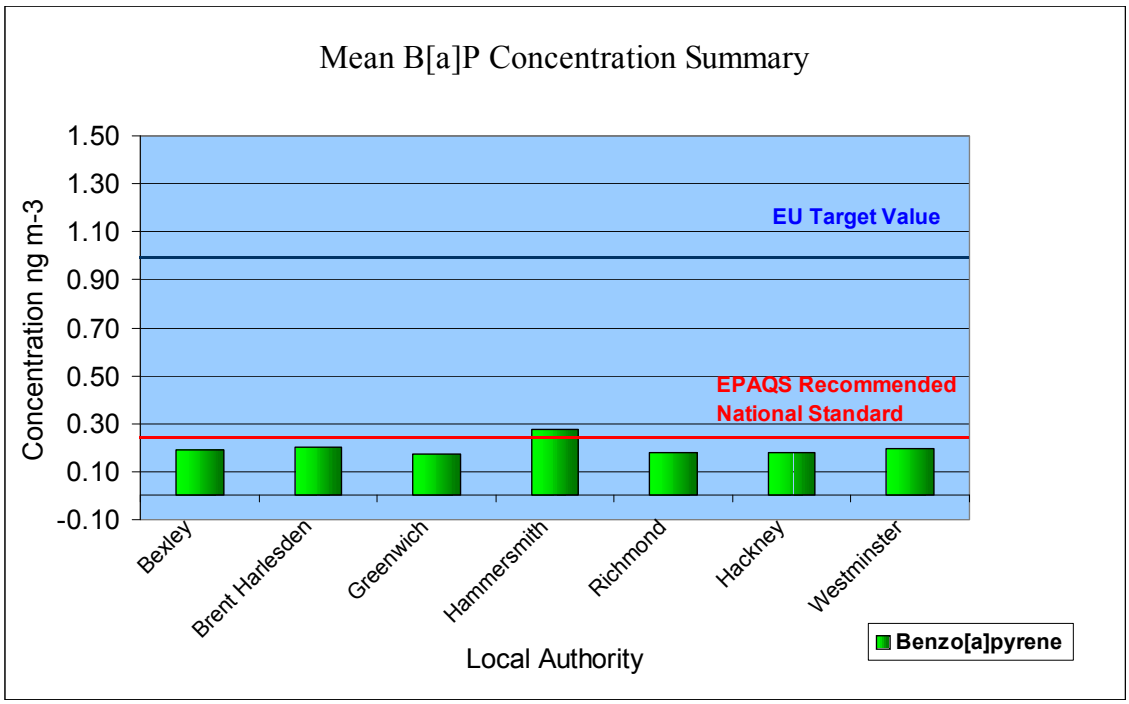

Figure 2. Summary of Calculated Mean B[a]P Concentration for the Seven Site Locations within the Survey.

7.1 Concentrations - Overview

Mean B[a]P concentrations of between 0.17ng m^{-3} – 0.28ng m^{-3} were calculated for the seven participants in the survey. The highest mean B[a]P concentration of 0.28ng m^{-3} was recorded for Hammersmith, a roadside (busy) site located at the air monitoring cabin next to Hammersmith Broadway tube station. The lowest mean value of 0.17ng m^{-3} was recorded for the roadside (busy) site located next to the A2.

Mean PAH concentrations measured for Bexley, Brent, Greenwich, Hammersmith and Fulham, Richmond, Hackney and Westminster are illustrated in Appendix C, Figures 1a to 1g respectively. The concentrations for each of the USEPA 16 listed species from the particulate phase are presented. Figure 1h provides an illustration of the above, averaged across the seven participating Boroughs.

Concentrations of each measured PAH species varied from site to site. A similar pattern in PAH species distribution was observed across all sites – concentrations of phenanthrene, fluoranthene and pyrene were higher than concentrations of the other species measured.

The major PAH species present in car exhaust emissions are reported to be fluoranthene and pyrene. The results show that levels of fluoranthene and pyrene for all sites were generally elevated above other PAHs during each of the monitoring periods. Such results indicate that emissions from motor vehicles were the largest contributing source to PAH level measured in this study.

Concentrations of particulate phase PAHs are generally highest near busy roadsides, concentrations falling off rapidly with increasing distance from the source. The wide variations in measured concentrations from compound to compound and site to site are a feature of PAH concentrations in urban areas and are likely to reflect traffic flows and prevailing weather conditions.

7.2 Comparison of Roadside (Busy), Roadside (Intermediate) and Background Sites

The mean concentration of each compound has been averaged for each of the three site categories and these results are illustrated in Appendix D, Figure 3. Due to a low number of sites across all three-site classifications, any comparison should only be used as an indication of comparability.

Concentrations of species measured were similar across all three site categories. Concentrations of species measured at roadside (busy) sites were slightly higher than at roadside (intermediate) and background sites. Many of the species, including some of those associated with vehicular emissions, e.g. fluoranthene, phenanthrene, pyrene and chrysene, measured higher concentrations than expected for the background site. This may suggest that levels recorded at the background site may have been influenced by local traffic sources or industry.

7.3 The PAH Profile

The use of the relative proportions of the individual PAHs in a given sample or series of samples - the 'PAH profile' - has often been tried as a method of determining the relative contribution of different sources. For example, benzo(g,h,i)perylene (BghiP) and chrysene (CHR) have been suggested as markers for vehicle emissions. Benzo(a)pyrene (B[a]P) is readily produced by coal and coke-burning as well as being present in vehicle emissions. There is no general consensus as to the use of PAH profiles for source apportionment, one reason for this may be that the effect of atmospheric transport, degradation and deposition processes tend to blur any initial sharp differences in the emitted PAH concentrations. However, in order to investigate the use of profiles in this study, the graph shown in Appendix D Figure 3 was constructed. The PAHs chosen were the seven most carcinogenic of molecular weight 228 or greater, and the average concentrations from sampling were used.

Appendix D, Figure 4 illustrates the PAH profiles obtained for each of the sites within the survey. The concentrations of benzo(b)fluoranthene were the highest at all sites and a similar profile was observed at all sites with dibenz(a,h)anthracene (DahA) producing the lowest levels followed in succession by either benzo(k)fluoranthene (BkF) or benzo(a)pyrene (B[a]P), then either benzo(a)pyrene (B[a]P) or benzo(a)anthracene (BaA), then either Chrysene (CHR) or benzo(g,h,i)perylene B(ghi)P, and finally benzo(b)fluoranthene (BbF). The similar profiles suggest that no sites were affected significantly by a specific source, e.g. a localised industrial process, and can be attributed to a much wider source.

7.4 Benzo(a)Pyrene Concentration as a Percentage of all Major Carcinogenic PAHs

Benzo(a)pyrene (B[a]P) is the only PAH for which there are any authoritative recommendations as to an appropriate guideline or standard. It is often stated that the B[a]P concentration on its own is not a satisfactory index of the total carcinogenic potential of a mixture of PAHs, so it is of interest to find the B[a]P concentration expressed as a percentage of all the major carcinogenic PAHs. In the context of the present measurements, this is:

Equation 1: $$\%\ B[a]P = \frac{B[a]P\ \text{concentration} \times 100}{\text{Sum of conc. (BaA+CHR + BbF + BkF + B[a]P + DahA + BghiP)}}$$

This percentage has been calculated for the estimated annual average concentrations and the values are given in Table H.

Table H: B[a]P Concentrations as a Percentage of the Sum of the Concentrations of (BaA+CHR + BbF + BkF + B[a]P + DahA + BghiP)

Borough	Site Classification	Percent B[a]P
Bexley	Background	11.20
Brent Harlesden	Roadside (Busy)	10.19
Greenwich	Roadside (Busy)	10.64
Hammersmith and Fulham	Roadside (Busy)	11.42
Richmond	Roadside (intermediate)	11.15
Hackney	Roadside (Busy)	10.40
Westminster	Roadside (Busy)	11.55
Arithmetic Mean		**10.49**

A percentage of 11.20% was recorded at the background site; 11.15% at the roadside (intermediate) site; and values at roadside (busy) sites ranged from 10.19% to 11.55 %.

7.5 Comparison of Results with Guidelines for Benzo(a)Pyrene

If B[a]P is used as an index of PAH carcinogenicity, then some comparison of the LWEP PAH survey with the EPAQS guideline for B[a]P *(0.25 ng m^{-3})* is possible. However, it must be noted that these guidelines relate to annual mean concentrations, and the short sampling periods used in this survey mean that any comparison can only be approximate.

Table I: Tabular Summary of Estimated Annual Mean B[a]P Concentration Measured from the Particulate Phase.

Borough	Concentration ng m^{-3}
Bexley	0.19
Brent Harlesden	0.20
Greenwich	0.17
Hammersmith and Fulham	0.28*
Richmond	0.18
Hackney	0.18
Westminster	0.20

(*<11-months data available from May 2004-March 2005 due to site power failures)

The survey results indicate that the estimated annual mean B[a]P concentration recorded at all sites do not exceed the current EU target value (1ng m^{-3}) for PAHs in ambient air. In addition the annual mean B[a]P concentration at all sites except Hammersmith*, do not exceed the current EPAQS guideline (0.25ng m^{-3}) for PAHs in ambient air.

* As less than 12 months data was captured for Hammersmith, the estimated annual mean for B[a]P may not be as representative of a full years data as the sites that did successfully collect 12 months data.

8 Assessment of Effects of PAH on the London Population

The PAH standard of 0.25ng m^{-3} B[a]P recommended by EPAQS is considered to be the maximum desirable level for total PAH in ambient air if health effects are to be avoided. However, there is no threshold for the level at which B[a]P is considered to have no effect, so it can be assumed that even at levels below the recommended standard some risk may still exist.

Based on occupational studies, the WHO has calculated an estimated risk for B[a]P exposure at one cancer per ten thousand for a person exposed to 1ng m^{-3} for a working lifetime. The annual mean concentration of B[a]P recorded at the background site in the present survey was 0.19ng m^{-3}. Hence, for an individual living at a background location over a lifetime, the respiratory cancer risk would be $1.9x10^{-6}$. The urban background site has been used in this calculation as such sites are considered representative of levels to which the majority of people are exposed to for significant periods. The risk should be correspondingly higher for individuals who spend a significant length of time at roadside (busy) locations. For example, the highest annual concentration of B[a]P recorded at the roadside sites in the present survey was 0.28ng m^{-3}, resulting in a respiratory cancer risk of $2.8x10^{-6}$.

The risk to the population of PAH exposure may however be much greater than these calculations imply. B[a]P as a percentage of total carcinogenic PAH in this study was just under 10.5%, a very small percentage of total carcinogenic PAH. Using B[a]P alone does not take into account any risk arising from the 'cocktail' of PAH in ambient air to which people are exposed.

Furthermore, there are other significant sources of inhaled PAH, in particular cigarette smoke. A single low tar cigarette is estimated to deliver 10ng B[a]P[(2)] in the mainstream smoke. For comparison assuming a daily-inhaled volume of $20m^3$, an individual spending 8 hours per day at the most polluted site in this study would inhale 1.9ng check against previous years data - equivalent to smoking just over one cigarette per week.

Equation 2:

1. $\frac{8\text{hrs}}{24\text{hrs}} \times 20\text{m}^3 = 6.67\text{m}^3$ breathed.

2. 6.67m^3 breathed x 0.28ng = 1.9ng B[a]P cumulative

The PAH composition of coke-oven emissions, cigarette smoke, diesel exhausts and ambient London air may be very different. Since there is only limited information on the relative carcinogenicity of different PAHs, it is uncertain to what extent it is possible to extrapolate data from exposure to one of these to the effects of other types of exposure. Given these and the many other uncertainties involved in risk assessment based on occupational exposures, the risks quoted here are very uncertain. As far as can be ascertained, the risks for individuals with the highest exposure to roadside PAH levels in London are finite but very small; for smokers, the risks appear trivial compared with those through PAH exposure from cigarette smoke.

9 Conclusion

This survey was designed to give a snapshot of the PAH concentrations across London, and the short sampling periods used do not, therefore, permit a detailed analysis of intersite differences. There are also many uncertainties relating to the measurement of PAHs, which include handling/storage of samples and differences in analytical techniques. However, despite the limitations of the survey, it is possible to identify some general trends in the data set.

- Measurement of the US EPA suite of compounds followed a similar trend with variations in concentration from site to site. Consistently higher concentrations of compounds associated with vehicular emissions, such as fluoranthene and pyrene were evident across all sites. This is a general feature of PAH concentrations surveyed in urban areas.

- The PAH profiles for the seven most carcinogenic compounds monitored were consistent across all sites. The similar profiles suggest that no sites were affected significantly by a specific source and can be attributed to a much wider mobile source.

- Use of particulate B[a]P as an index of carcinogenicity indicates that a concentration of B[a]P at all sites would not exceed the new air quality objective for England and Wales of 0.25 ng m^{-3} B[a]P by the end of 2010. In addition, the higher EU target value of 1ng m^{-3} would not be expected to be exceeded by any sites by the end of 2012.

- The average particulate B[a]P concentrations recorded across all three site categories were similar. At background, roadside (intermediate) and roadside (busy) sites mean concentrations were 0.19, 0.18 and 0.21ng m^{-3} respectively. The similarity in mean concentrations and assessment of data suggests influence from motor vehicle emissions at the background location. This can be confirmed by the location of a car park and movement of vehicles near the sampling point. For an individual exposed to these concentrations over a lifetime, this would represent a 1.9 x 10^{-6}, 1.8 x 10^{-6} and 2.1 x 10^{-6} risk of respiratory cancer. It is considered that the health risks for individuals with the highest exposure to roadside PAH levels in London are finite but very small; for smokers the risks would appear trivial compared with those associated with exposure to PAH from cigarette smoke.

Vehicular emissions are still considered a primary source of PAH in urban areas as data suggests. Some PAH species are emitted in higher

concentrations from diesel engines including the known carcinogen benzo(a)pyrene. Continued policy measures to reduce emissions from diesel cars and heavy goods vehicles have included stringent emission standards *(Euro Standards)* and an improvement in the quality of diesel fuel, reducing the sulphur content. Summaries of dates for implementation of such Euro Standards are shown in Table J [18] [19].

Table J: Summary of Euro Standards and Dates of Implementation

	Directive	**Standard**	**Date of Implementation**
Heavy duty vehicles (first-step)	91/542/EEC	Euro I	July 1992/ 1 October 1993
(second-step)	96/1/EC	Euro II, TA & COP	30 September 1997/ 1 October 1996/ 30 September 1998
Light duty diesel	98/69/EC	Euro III	1 January 2001
		Euro IV	1 January 2006
Heavy duty diesel	1999/96/EC	Euro III	1 October 2001
		Euro IV	1 October 2006

Reductions in the sulphur content of diesel fuel will reduce emissions of particulate matter *(PM),* but is not likely to affect B[a]P emissions, however, changes in fuel formulation affecting the polyaromatic content of fuel will likely influence B[a]P emissions, although the UK is already within limits set by European fuel directives[18].

Road transport sources of B[a]P have declined since 1990 from 5.3t to 0.69t in 2000 and are predicted to fall even further by 2020 to 0.22t [17]. Industrial sources of B[a]P related to aluminium production have fallen dramatically since 1990 reducing B[a]P concentration in the vicinity of plants, which is expected to decrease further due to improvements in industrial abatement and process controls[17].

In view of the current standard and future objective, further monitoring of PAHs is required to assist in assessing the impact of recent legislation on London's air quality, and to continue expanding the valuable database of PAH concentrations within London.

10 Recommendations

For comparison both High Performance Liquid Chromatography (HPLC) and GC-MS analytical methods were used to acquire data for 2001-2002. The GC-MS showed a substantial reduction in levels of PAHs measured due to improvements in quality control.

Subsequently the 2002-2003, 2003-2004 and 2004-2005 studies have used the GC-MS analytical method to coincide with the method used in Europe. However the sampling method was modified to include both the PM_{10} particulate fraction and the vapour phase component for collection of 'total PAH'. The analysis of samples was covered by a UKAS accredited laboratory to improve the credibility of the data. As would be predicted, the sampling of PM_{10} particulate, as compared to the PM_{16} (estimated) particulate in the 2001-2002 study, reduced the concentrations of B[a]P and other PAHs. This year levels of B[a]P remained below the EPAQS guideline of 0.25 ng m^{-3} as an annual average at all sites except for Hammersmith and all sites B[a]P levels were below the EU target value.

It is therefore recommended that preparation and GC-MS analysis of samples be covered by a UKAS laboratory to continue maintaining a high quality and credible monitoring strategy. Following the implementation of the 4th Daughter directive (2004/107/EC) there is no longer a requirement for the vapour phase component be measured from the PM10 fraction for collection of 'total PAH'. This is because the directive has only adopted target values for B[a]P which is found in the particulate phase. As such it is thought that the monitoring strategy could be modified to monitor B[a]P in particulate phase only. For those wishing to maintain their databases for trend analysis an option to continue with the vapour phase can be offered.

11 Report Statement.

Bureau Veritas completed this report on the basis of a defined program of works and within the terms and conditions agreed with the client.

This report was compiled with all reasonable skill and care, bearing in mind the project objectives, the agreed scope of works, prevailing site conditions and the degree of manpower and resources allocated to the project as agreed.

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In addition to the above, in reports containing results for UKAS accredited procedures, all non-accredited activities, subcontracted results, recommendations and professional opinions will be disclaimed.

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Appendix A
Site Descriptions

Appendix A

Site Descriptions

Site 1 - Bexley
A background site air monitoring cabin at Whitehall Day Centre in Bexley, approx. 200m from the nearest busy road.

Site 2 – Brent Harlesden
A roadside (busy) air monitoring cabin at the Budgen car park in Harlesden approximately 2m from the nearest busy road.

Site 3 - Greenwich
A roadside (busy) site located at an air monitoring cabin near Crown Woods Way, approximately 10m from the A2.

Site 4 - Hammersmith and Fulham
A roadside (busy) site located situated next to the Hammersmith Broadway tube station approximately 2m from the A315.

Site 5 - Richmond
A roadside (intermediate) monitoring site located approximately 2m from the intermediately busy road at Castelnau SW13. Sampling point within enclosure at Castelnau Library.

Site 6 - Hackney
Air monitoring cabin located beside the fire station on Old Street, approx. 6m from busy road.

Site 7 - Westminster
A roadside (busy) site located at the Westminster Council offices. The sampling point is approximately 6m from Marylebone Road.

Appendix B
Tables of Results 1 – 7

Table 1 - The London Borough of Bexley - Monthly Concentrations of PAH Expressed as ng m-

	Apr-04	May-04	Jun-04	Jul-04	Aug-04	Sep-04	Oct-04	Nov-04	Dec-04	Jan-05	Feb-05	Mar-05
Naphthalene	0.64	0.16	0.07	ND	ND	0.48	0.47	0.81	0.55	0.55	0.47	0.38
Acenaphthylene	N.D	0.01	0.02	0.01	0.01	0.02	0.12	0.47	0.09	0.11	0.09	0.04
Acenaphthene	0.21	0.05	0.04	0.03	0.02	0.05	0.07	0.29	0.14	0.09	0.11	0.12
Fluorene	0.74	0.26	0.20	0.44	0.09	0.28	0.40	1.15	0.53	0.66	0.55	0.51
Phenanthrene	7.44	2.54	4.41	9.48	1.84	3.27	9.10	10.83	5.03	10.21	6.71	5.53
Anthracene	ND	2.37	0.10	0.20	0.04	0.07	0.94	1.08	0.33	0.36	0.33	0.17
Fluoranthene	3.61	1.66	2.37	4.01	2.57	3.27	4.38	3.32	1.65	3.54	2.13	2.13
Pyrene	1.75	0.68	1.19	1.57	1.08	1.95	3.24	2.78	1.57	2.66	1.50	1.19
Benz(a)anthracene	0.08	0.04	0.09	0.05	0.06	0.10	0.61	0.64	ND	0.32	0.22	0.11
Chrysene	0.24	0.10	0.15	0.19	0.19	0.22	0.88	0.88	0.13	0.47	0.47	0.26
Benzo(b)fluoranthene	0.13	0.05	0.10	0.16	0.21	0.37	1.21	1.29	0.13	0.91	0.71	0.32
Benzo(k)fluoranthene	0.16	0.05	0.10	0.03	0.04	0.06	0.37	0.44	0.04	0.22	0.21	0.11
Benzo(a)pyrene	0.10	0.02	0.06	0.03	0.03	0.10	0.47	0.68	0.05	0.36	0.25	0.11
Indeno(1,2,3-cd)pyrene	0.15	0.05	0.10	0.06	0.09	0.00	0.64	0.71	0.10	0.51	0.33	0.19
Dibenz(a,h)anthracene	0.01	0.00	ND	0.01	0.02	ND	0.12	0.11	0.00	0.06	0.05	0.03
Benzo(g,h,i)perylene	0.18	0.06	0.11	0.08	0.11	0.14	0.71	0.95	0.10	0.62	0.43	0.21
Total PAH (ng m3)	15.45	8.10	9.11	16.36	6.41	10.37	23.73	26.44	10.46	21.65	14.59	11.40

Table 2 - The London Borough of Brent - Monthly Concentrations of PAH Expressed as ng m-3

	Apr-04	May-04	Jun-04	Jul-04	Aug-04	Sep-04	Oct-04	Nov-04	Dec-04	Jan-05	Feb-05	Mar-05
Naphthalene	1.20	0.47	0.28	0.16	0.18	0.51	1.22	0.95	0.95	0.51	0.71	0.41
Acenaphthylene	0.52	0.08	0.12	0.12	0.03	0.18	0.52	0.73	0.53	0.47	0.17	0.09
Acenaphthene	0.73	0.17	0.15	0.21	0.04	0.13	0.22	0.41	0.37	0.21	0.22	0.17
Fluorene	2.28	0.58	0.41	0.68	0.13	0.47	1.27	1.07	0.90	1.22	0.91	0.41
Phenanthrene	19.36	6.15	7.11	6.76	2.41	6.21	16.92	14.53	9.48	12.52	15.89	5.77
Anthracene	0.82	0.22	0.22	0.19	0.09	0.17	1.93	2.18	0.79	0.81	1.35	0.26
Fluoranthene	6.02	2.93	3.28	5.07	2.46	4.02	4.23	4.42	2.74	5.08	5.07	3.29
Pyrene	4.73	2.10	2.30	3.38	1.37	3.21	3.71	4.11	2.74	4.74	4.73	2.78
Benz(a)anthracene	0.25	0.06	0.09	0.07	0.05	0.12	0.44	0.66	0.09	0.37	0.37	0.16
Chrysene	0.52	0.12	0.13	0.19	0.15	0.23	0.66	0.88	0.26	0.64	0.68	0.33
Benzo(b)fluoranthene	0.31	0.07	0.11	0.18	0.16	0.40	0.94	1.26	0.26	0.95	0.98	0.37
Benzo(k)fluoranthene	0.22	0.05	0.05	0.04	0.03	0.09	0.30	0.35	0.05	0.26	0.31	0.13
Benzo(a)pyrene	0.19	0.04	0.07	0.05	0.04	0.10	0.40	0.70	0.05	0.37	0.30	0.13
Indeno(1,2,3-cd)pyrene	0.34	0.09	0.13	0.10	0.08	0.16	0.56	0.79	0.18	0.54	0.64	0.25
Dibenz(a,h)anthracene	0.02	0.01	ND	0.02	0.01	0.03	0.09	0.11	ND	0.07	0.07	0.03
Benzo(g,h,i)perylene	0.47	0.13	0.18	0.15	0.11	0.24	0.80	1.20	0.23	0.81	0.91	0.34
Total PAH (ng m3)	37.99	13.26	14.63	17.36	7.33	16.28	34.21	34.36	19.62	29.58	33.32	14.89

Table 3 - The London Borough of Greenwich - Monthly Concentrations of PAH Expressed as ng

	Apr-04	May-04	Jun-04	Jul-04	Aug-04	Sep-04	Oct-04	Nov-04	Dec-04	Jan-05	Feb-05	Mar-05
Naphthalene	ND	0.11	0.04	0.37	0.15	0.61	0.61	0.61	0.71	0.62	0.67	0.36
Acenaphthylene	0.08	0.02	0.00	0.09	0.07	0.05	0.17	0.41	0.09	0.11	0.25	0.04
Acenaphthene	0.18	0.02	0.03	0.08	0.04	0.07	0.08	0.20	0.15	ND	0.08	0.07
Fluorene	0.74	0.27	0.30	0.66	0.26	0.34	0.57	1.05	0.40	0.36	0.51	0.34
Phenanthrene	11.39	3.29	7.13	6.95	3.68	5.13	9.80	8.81	4.18	8.76	9.48	4.75
Anthracene	ND	0.20	0.33	0.23	0.29	0.11	1.11	0.98	0.27	0.44	0.83	0.17
Fluoranthene	4.78	2.34	2.41	4.75	1.26	2.79	4.05	4.07	1.50	4.74	4.35	3.01
Pyrene	3.42	1.42	1.39	3.04	1.05	1.94	3.38	3.39	1.50	4.01	3.67	2.06
Benz(a)anthracene	0.13	0.11	0.05	0.08	0.04	0.07	0.44	0.58	ND	0.34	0.40	0.13
Chrysene	0.29	0.12	0.12	0.20	0.07	0.14	0.71	0.85	0.14	0.58	0.71	0.30
Benzo(b)fluoranthene	0.18	0.06	0.06	0.21	0.08	0.27	0.98	1.25	0.15	0.99	1.07	0.34
Benzo(k)fluoranthene	0.21	0.08	0.04	0.05	0.01	0.03	0.30	0.41	0.04	0.24	0.33	0.11
Benzo(a)pyrene	0.09	0.04	0.03	0.07	0.03	0.12	0.41	0.64	0.05	0.30	0.34	0.11
Indeno(1,2,3-cd)pyrene	0.20	0.06	0.05	0.07	0.04	0.07	0.51	0.71	0.10	0.51	0.51	0.19
Dibenz(a,h)anthracene	0.01	0.01	ND	0.01	ND	0.00	0.09	0.09	ND	0.04	0.08	0.02
Benzo(g,h,i)perylene	0.26	0.10	0.08	0.15	0.08	0.14	0.64	0.91	0.11	0.66	0.71	0.24
Total PAH (ng m3)	21.95	8.25	12.07	16.99	7.15	11.60	23.85	24.96	9.38	22.70	23.99	12.23

Table 4 - The London Borough of Hammersmith - Monthly Concentrations of PAH Expressed as ng m-3

	Apr-04	May-04	Jun-04	Jul-04	Aug-04	Sep-04	Oct-04	Nov-04	Dec-04	Jan-05	Feb-05	Mar-05
Naphthalene	unavailable	0.21	unavailable	0.86	0.14	0.23	1.22	1.08	0.63	1.05	1.05	0.31
Acenaphthylene	unavailable	0.06	unavailable	0.26	0.10	0.09	0.28	0.89	0.68	0.64	0.10	0.18
Acenaphthene	unavailable	0.11	unavailable	0.53	0.06	0.10	0.16	0.27	0.36	0.18	0.12	0.10
Fluorene	unavailable	0.58	unavailable	1.53	0.75	0.55	0.99	1.23	0.95	1.12	0.67	0.58
Phenanthrene	unavailable	9.22	unavailable	21.03	14.23	6.57	14.57	14.24	8.96	10.82	9.78	7.11
Anthracene	unavailable	0.38	unavailable	0.76	0.66	0.44	1.74	1.96	1.37	0.81	0.71	0.58
Fluoranthene	unavailable	2.46	unavailable	11.47	10.16	3.58	4.37	4.11	4.11	4.06	3.71	2.98
Pyrene	unavailable	1.84	unavailable	9.08	8.13	3.58	4.09	4.11	4.11	3.72	3.24	2.71
Benz(a)anthracene	unavailable	0.07	unavailable	0.19	0.25	0.14	0.44	0.66	0.17	0.37	0.30	0.17
Chrysene	unavailable	0.14	unavailable	0.53	0.49	0.27	0.66	0.92	0.31	0.51	0.57	0.31
Benzo(b)fluoranthene	unavailable	0.07	unavailable	0.48	0.62	0.40	0.99	1.33	0.09	0.81	0.91	0.37
Benzo(k)fluoranthene	unavailable	0.08	unavailable	0.08	0.14	0.08	0.31	0.44	0.40	0.21	0.25	0.12
Benzo(a)pyrene	unavailable	0.04	unavailable	0.16	0.15	0.16	0.47	0.82	0.13	0.37	0.33	0.14
Indeno(1,2,3-cd)pyrene	unavailable	0.07	unavailable	0.19	0.22	0.09	0.56	0.79	0.19	0.47	0.44	0.21
Dibenz(a,h)anthracene	unavailable	0.01	unavailable	0.00	0.03	ND	0.09	0.10	0.00	0.06	0.06	0.02
Benzo(g,h,i)perylene	unavailable	0.12	unavailable	0.43	0.54	0.36	0.80	1.17	0.36	0.68	0.67	0.34
Total PAH (ng m3)	unavailable	15.45	unavailable	47.58	36.66	16.63	31.74	34.14	22.83	25.87	22.91	16.23

Table 5 - The London Borough of Richmond - Monthly Concentrations of PAH Expressed as ng

	Apr-04	May-04	Jun-04	Jul-04	Aug-04	Sep-04	Oct-04	Nov-04	Dec-04	Jan-05	Feb-05	Mar-05
Naphthalene	0.79	0.09	0.28	0.43	0.20	0.44	0.47	0.64	0.65	0.95	0.48	0.35
Acenaphthylene	0.22	0.03	0.04	0.06	0.07	0.15	0.13	0.64	0.21	0.14	0.12	0.06
Acenaphthene	0.29	0.06	0.05	0.16	0.04	0.11	0.07	0.37	0.21	0.09	0.14	0.13
Fluorene	1.19	0.29	0.28	0.46	0.14	0.40	0.37	1.02	0.71	0.62	0.55	0.51
Phenanthrene	16.44	3.13	4.07	4.26	3.42	4.02	6.74	9.83	6.94	9.86	6.22	4.35
Anthracene	0.74	0.07	0.06	0.07	0.18	3.40	0.64	1.12	0.65	0.62	0.33	0.08
Fluoranthene	4.08	1.71	2.75	3.60	4.47	2.74	2.80	3.19	2.21	3.65	2.49	2.14
Pyrene	2.95	1.02	1.59	1.82	3.15	2.08	2.29	2.98	2.21	3.07	1.97	1.42
Benz(a)anthracene	0.11	0.04	0.04	0.05	0.16	0.06	0.28	0.68	0.07	0.33	0.25	0.09
Chrysene	0.27	0.09	0.09	0.17	0.37	0.14	0.47	0.85	0.23	0.55	0.48	0.23
Benzo(b)fluoranthene	0.15	0.04	0.05	0.14	0.39	0.28	0.74	1.32	0.21	0.88	0.73	0.22
Benzo(k)fluoranthene	0.14	0.05	0.03	0.03	0.09	0.05	0.23	0.47	0.05	0.19	0.22	0.06
Benzo(a)pyrene	0.11	0.03	0.03	0.04	0.06	0.08	0.27	0.81	0.07	0.33	0.27	0.07
Indeno(1,2,3-cd)pyrene	0.17	0.06	ND	0.05	0.19	0.00	0.40	0.81	0.06	0.51	0.40	0.14
Dibenz(a,h)anthracene	0.01	0.00	ND	0.01	0.03	ND	0.07	0.13	ND	0.05	0.06	0.02
Benzo(g,h,i)perylene	0.25	0.08	0.06	0.09	0.24	0.15	0.57	1.08	0.16	0.66	0.55	0.20
Total PAH (ng m3)	27.91	6.80	9.43	11.44	13.20	14.09	16.54	25.95	14.62	22.50	15.24	10.07

Table 6 - The London Borough of Hackney - Monthly Concentrations of PAH Expressed as ng m-3

	Apr-04	May-04	Jun-04	Jul-04	Aug-04	Sep-04	Oct-04	Nov-04	Dec-04	Jan-05	Feb-05	Mar-05
Naphthalene	1.27	0.22	0.22	0.64	0.16	1.20	0.72	1.42	1.63	5.08	0.71	0.47
Acenaphthylene	0.40	0.05	0.08	0.15	0.02	0.20	0.30	0.38	0.63	1.59	0.22	0.16
Acenaphthene	0.45	0.08	0.14	0.20	0.03	0.13	0.14	0.18	0.53	0.67	0.16	0.16
Fluorene	1.88	0.44	0.48	0.54	0.11	0.44	1.16	0.79	1.26	5.08	1.08	0.68
Phenanthrene	18.30	5.89	7.40	6.44	1.92	6.20	15.91	11.05	10.01	14.32	11.48	9.14
Anthracene	0.92	0.18	0.23	0.16	0.07	0.09	1.83	1.26	1.26	0.69	1.08	0.61
Fluoranthene	6.61	3.09	5.55	6.78	2.89	4.38	4.82	4.42	2.53	3.23	4.05	4.06
Pyrene	5.59	2.21	4.07	4.75	1.68	4.01	4.58	4.42	2.53	2.77	3.38	3.39
Benz(a)anthracene	0.19	0.06	0.06	0.08	0.07	0.09	0.40	0.57	0.12	0.30	0.29	0.18
Chrysene	0.41	0.13	0.16	0.21	0.17	0.29	0.63	0.79	0.23	0.39	0.54	0.34
Benzo(b)fluoranthene	0.20	0.06	0.08	0.19	0.19	0.35	0.92	1.07	0.24	0.55	0.74	0.41
Benzo(k)fluoranthene	0.20	0.08	0.06	0.04	0.05	0.04	0.29	0.35	0.26	0.55	0.23	0.13
Benzo(a)pyrene	0.16	0.03	0.03	0.05	0.05	0.10	0.44	0.60	0.07	0.25	0.25	0.12
Indeno(1,2,3-cd)pyrene	0.27	0.07	0.06	0.07	0.10	0.10	0.53	0.63	0.17	0.28	0.44	0.24
Dibenz(a,h)anthracene	0.01	0.00	ND	0.01	0.02	0.00	0.08	0.08	ND	ND	0.05	0.03
Benzo(g,h,i)perylene	0.42	0.11	0.14	0.14	0.12	0.26	0.77	0.95	0.28	0.06	0.64	0.37
Total PAH (ng m3)	**37.29**	**12.71**	**18.75**	**20.43**	**7.65**	**17.88**	**33.52**	**28.94**	**21.78**	**35.84**	**25.34**	**20.49**

Table 7 - The London Borough of Westminster - Monthly Concentrations of PAH Expressed as ng m

	Apr-04	May-04	Jun-04	Jul-04	Aug-04	Sep-04	Oct-04	Nov-04	Dec-04	Jan-05	Feb-05	Mar-05
Naphthalene	0.46	0.30	0.33	0.41	0.37	0.84	0.85	1.33	1.69	1.18	0.71	0.64
Acenaphthylene	0.65	0.02	0.10	0.17	0.04	0.34	0.52	0.92	1.42	1.29	0.47	0.34
Acenaphthene	0.88	0.07	0.16	0.22	0.05	0.31	0.32	0.06	0.90	0.54	0.41	0.27
Fluorene	3.09	0.16	0.51	0.71	0.21	1.02	2.02	1.67	2.11	2.06	1.96	1.22
Phenanthrene	38.79	1.49	11.67	9.83	4.22	16.42	25.86	23.05	14.76	17.94	17.58	16.95
Anthracene	3.37	0.04	0.40	0.33	0.15	0.58	2.96	3.47	1.69	0.78	1.25	1.15
Fluoranthene	5.08	1.45	4.01	5.09	3.96	5.47	4.09	4.10	3.06	3.22	2.91	3.36
Pyrene	3.88	1.41	3.32	4.07	3.17	4.74	3.81	4.10	3.06	2.94	2.37	2.78
Benz(a)anthracene	0.16	0.07	0.09	0.09	0.09	0.13	0.44	0.51	0.13	0.28	0.24	0.16
Chrysene	0.30	0.15	0.15	0.21	0.22	0.29	0.66	0.73	0.24	0.44	0.41	0.28
Benzo(b)fluoranthene	0.17	0.07	0.11	0.20	0.24	0.47	0.89	1.01	0.25	0.71	0.61	0.30
Benzo(k)fluoranthene	0.19	0.08	0.06	0.05	0.05	0.05	0.28	0.31	0.09	0.17	0.19	0.08
Benzo(a)pyrene	0.12	0.04	0.04	0.06	0.06	0.15	0.47	0.66	0.11	0.32	0.20	0.11
Indeno(1,2,3-cd)pyrene	0.20	0.08	ND	0.06	0.11	0.00	0.47	0.57	0.16	0.37	0.30	0.17
Dibenz(a,h)anthracene	0.01	0.01	ND	0.01	0.01	0.02	0.08	0.08	0.00	0.04	0.04	0.02
Benzo(g,h,i)perylene	0.30	0.13	0.17	0.14	0.20	0.36	0.75	0.98	0.31	0.54	0.44	0.26
Total PAH (ng m3)	57.67	5.56	21.12	21.64	13.15	31.21	44.49	43.55	29.96	32.83	30.07	28.08

Table 8 - Average PAH Concentrations at Each Site (ng m-3)

Compound	Bexley	Brent Harlseden	Greenwich	Hammersmith	Richmond	Hackney	Westminster	Average of all boroughs
Naphthalene	0.46	0.63	0.39	0.68	0.48	1.15	0.76	0.65
Acenaphthylene	0.09	0.30	0.11	0.33	0.16	0.35	0.52	0.26
Acenaphthene	0.10	0.25	0.09	0.20	0.14	0.24	0.35	0.20
Fluorene	0.49	0.86	0.47	0.89	0.55	1.16	1.40	0.83
Phenanthrene	6.37	10.26	6.66	11.65	6.61	9.84	16.55	9.70
Anthracene	0.54	0.75	0.43	0.94	0.66	0.70	1.35	0.77
Fluoranthene	2.89	4.05	3.26	5.10	2.98	4.37	3.82	3.78
Pyrene	1.76	3.33	2.44	4.46	2.21	3.61	3.30	3.02
Benz(a)anthracene	0.21	0.23	0.21	0.28	0.18	0.20	0.20	0.21
Chrysene	0.35	0.40	0.33	0.47	0.33	0.36	0.34	0.37
Benzo(b)fluoranthene	0.47	0.50	0.44	0.61	0.43	0.42	0.42	0.47
Benzo(k)fluoranthene	0.15	0.16	0.15	0.21	0.13	0.19	0.13	0.16
Benzo(a)pyrene	0.19	0.20	0.17	0.28	0.18	0.18	0.20	0.20
Indeno(1,2,3-cd)pyrene	0.24	0.32	0.24	0.32	0.26	0.25	0.23	0.27
Dibenz(a,h)anthracene	0.04	0.05	0.04	0.04	0.04	0.03	0.03	0.04
Benzo(g,h,i)perylene	0.31	0.46	0.32	0.55	0.34	0.36	0.38	0.39
Total PAH (ng m3)	14.50	22.74	15.64	27.00	15.65	23.38	29.94	21.27

Appendix C
Figures 1a – 1h

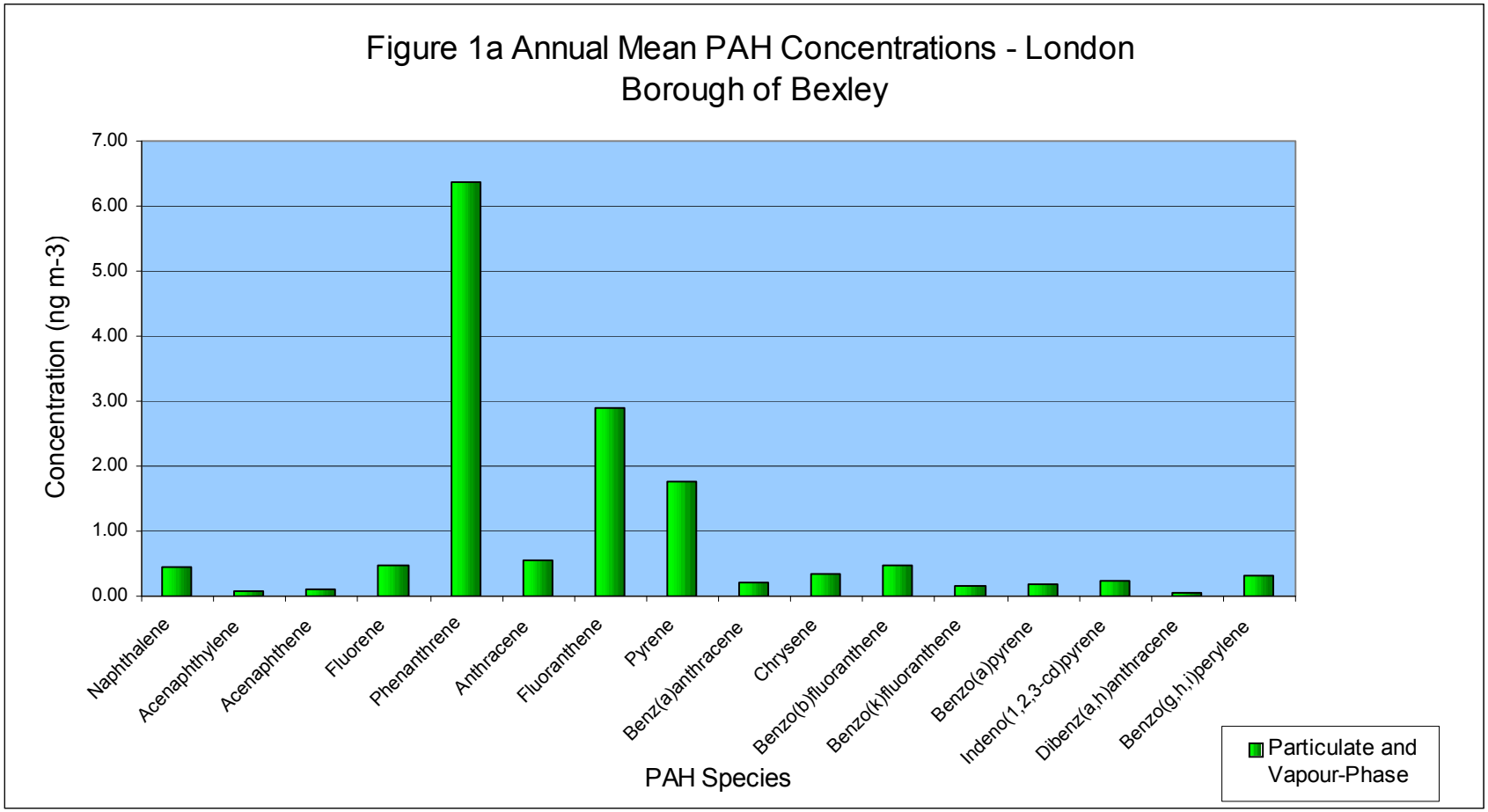


Figure 1a Annual Mean PAH Concentrations - London Borough of Bexley
Concentration (ng m-3)
7.00
6.00
5.00
4.00
3.00
2.00
1.00
0.00
Naphthalene
Acenaphthylene
Acenaphthene
Fluorene
Phenanthrene
Anthracene
Fluoranthene
Pyrene
Benz(a)anthracene
Chrysene
Benzo(b)fluoranthene
Benzo(k)fluoranthene
Benzo(a)pyrene
Indeno(1,2,3-cd)pyrene
Dibenz(a,h)anthracene
Benzo(g,h,i)perylene
PAH Species
Particulate and Vapour-Phase

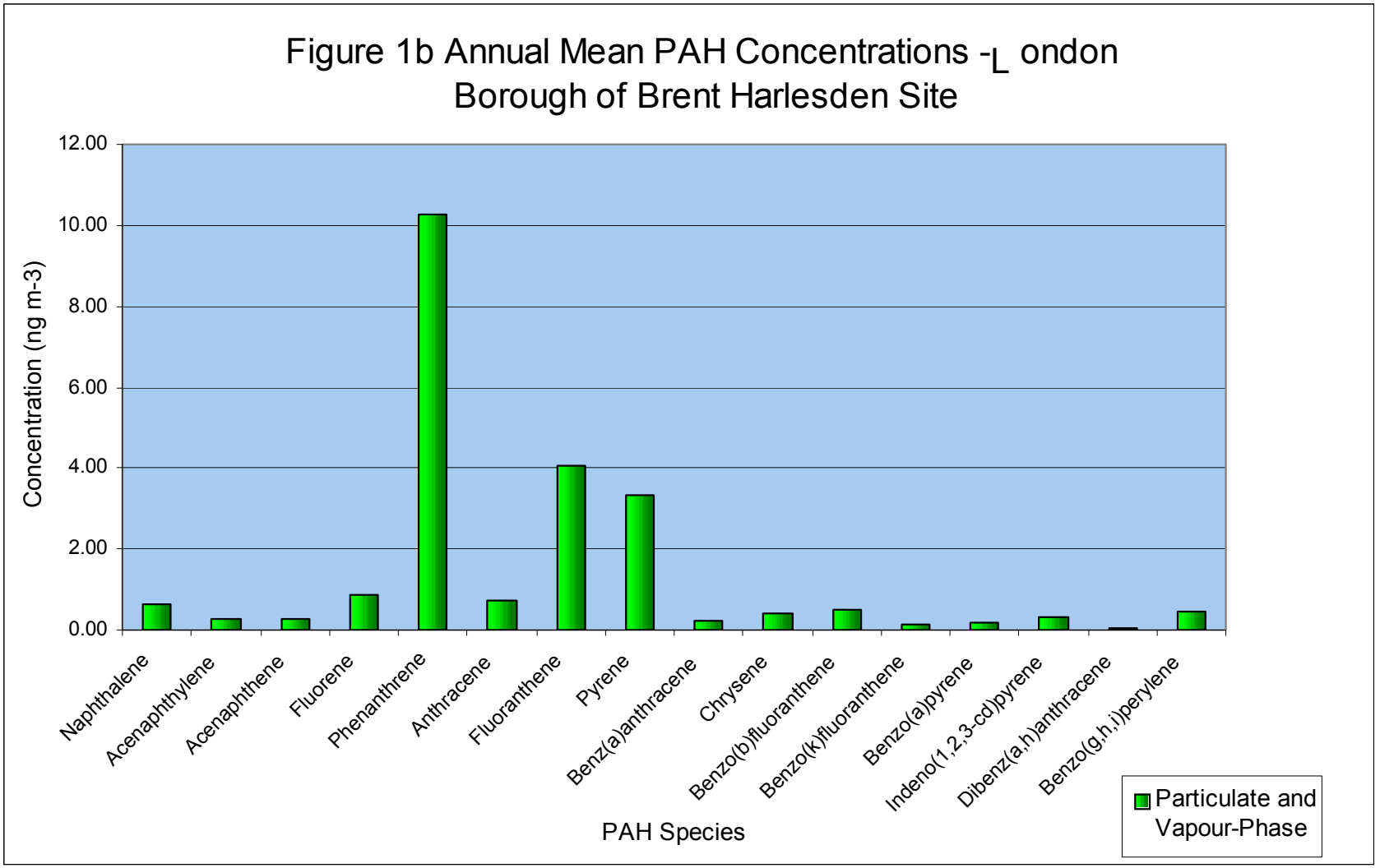


Figure 1b Annual Mean PAH Concentrations -London
Borough of Brent Harlesden Site
Concentration (ng m-3)
12.00
10.00
8.00
6.00
4.00
2.00
0.00
Naphthalene
Acenaphthylene
Acenaphthene
Fluorene
Phenanthrene
Anthracene
Fluoranthene
Pyrene
Benz(a)anthracene
Chrysene
Benzo(b)fluoranthene
Benzo(k)fluoranthene
Benzo(a)pyrene
Indeno(1,2,3-cd)pyrene
Dibenz(a,h)anthracene
Benzo(g,h,i)perylene
PAH Species
Particulate and Vapour-Phase

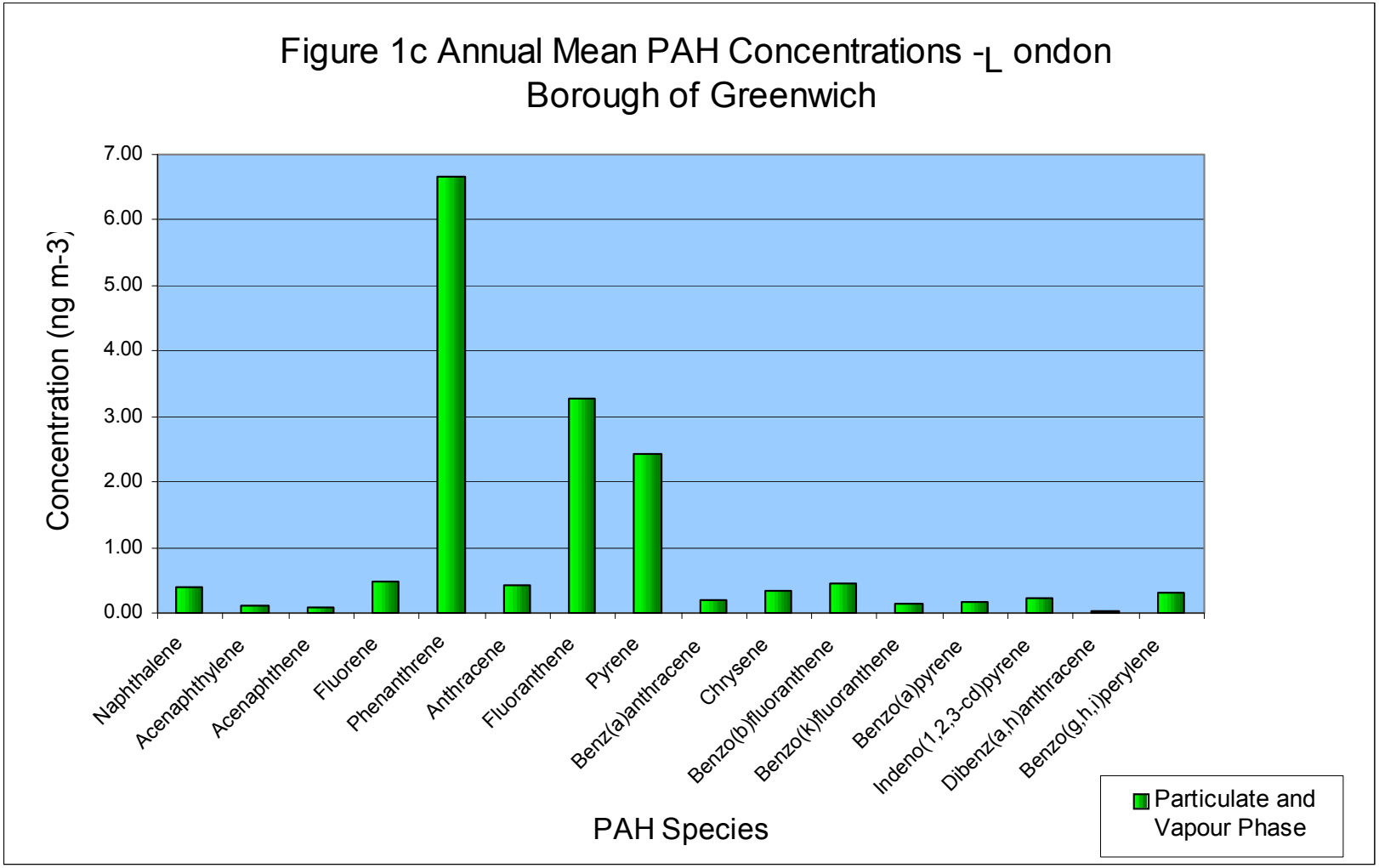


Figure 1c Annual Mean PAH Concentrations -L ondon
Borough of Greenwich
Concentration (ng m-3)
7.00
6.00
5.00
4.00
3.00
2.00
1.00
0.00
Naphthalene
Acenaphthylene
Acenaphthene
Fluorene
Phenanthrene
Anthracene
Fluoranthene
Pyrene
Benz(a)anthracene
Chrysene
Benzo(b)fluoranthene
Benzo(k)fluoranthene
Benzo(a)pyrene
Indeno(1,2,3-cd)pyrene
Dibenz(a,h)anthracene
Benzo(g,h,i)perylene
PAH Species
Particulate and Vapour Phase

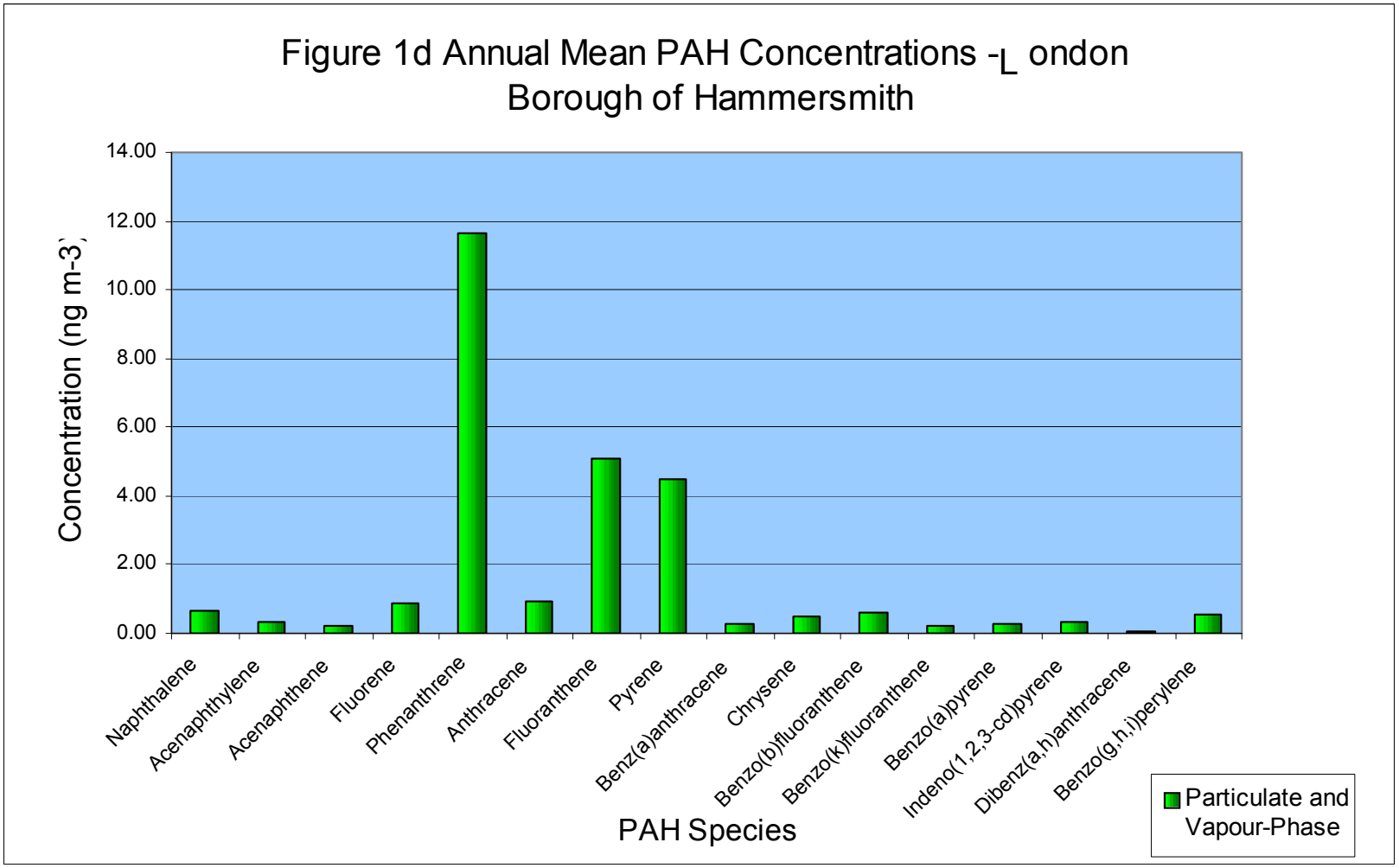


Figure 1d Annual Mean PAH Concentrations -L ondon
Borough of Hammersmith
Concentration (ng m-3)
14.00
12.00
10.00
8.00
6.00
4.00
2.00
0.00
Naphthalene
Acenaphthylene
Acenaphthene
Fluorene
Phenanthrene
Anthracene
Fluoranthene
Pyrene
Benz(a)anthracene
Chrysene
Benzo(b)fluoranthene
Benzo(k)fluoranthene
Benzo(a)pyrene
Indeno(1,2,3-cd)pyrene
Dibenz(a,h)anthracene
Benzo(g,h,i)perylene
PAH Species
Particulate and Vapour-Phase

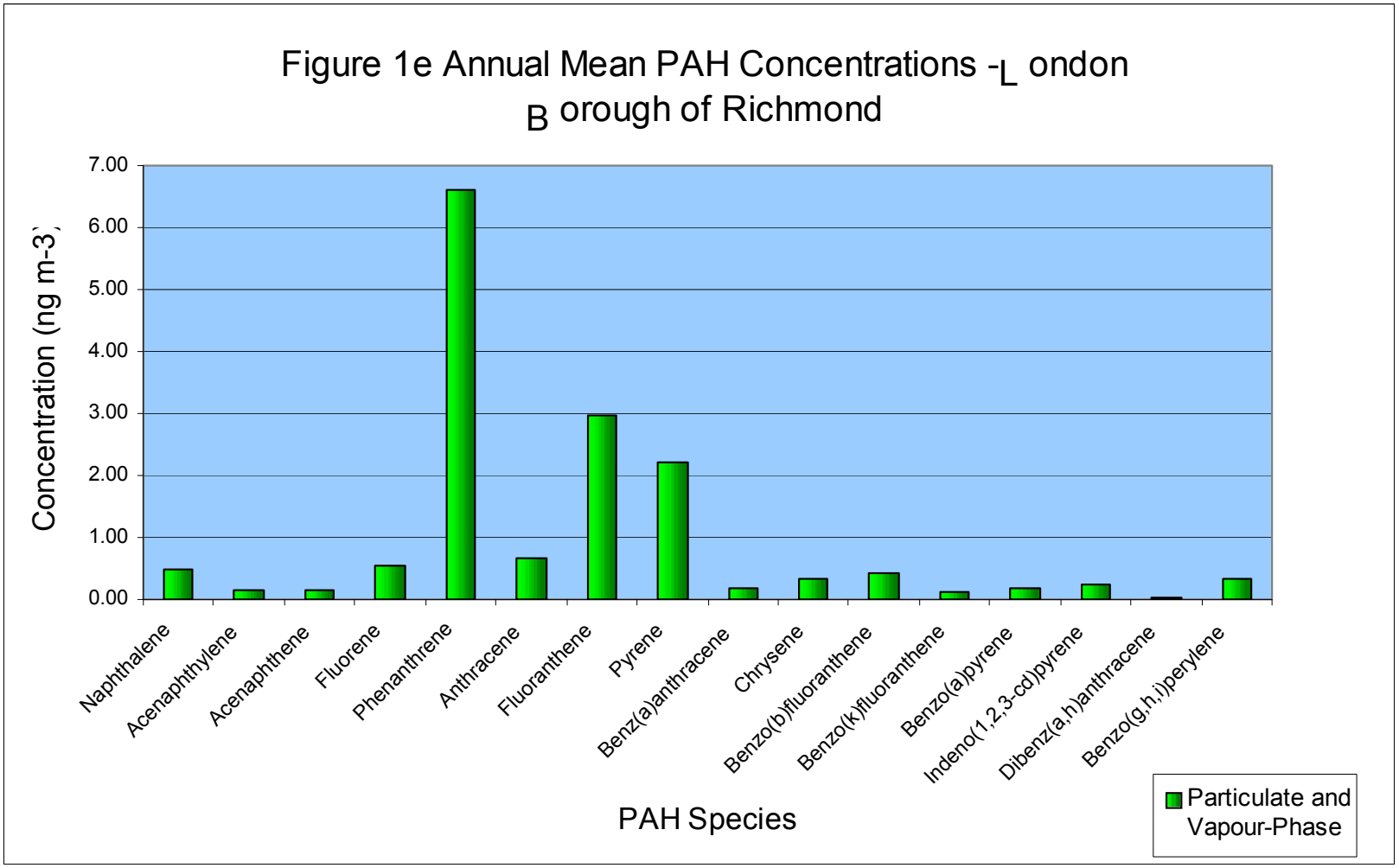


Figure 1e Annual Mean PAH Concentrations -L ondon
B orough of Richmond
Concentration (ng m-3`,
7.00
6.00
5.00
4.00
3.00
2.00
1.00
0.00
Naphthalene
Acenaphthylene
Acenaphthene
Fluorene
Phenanthrene
Anthracene
Fluoranthene
Pyrene
Benz(a)anthracene
Chrysene
Benzo(b)fluoranthene
Benzo(k)fluoranthene
Benzo(a)pyrene
Indeno(1,2,3-cd)pyrene
Dibenz(a,h)anthracene
Benzo(g,h,i)perylene
PAH Species
Particulate and Vapour-Phase

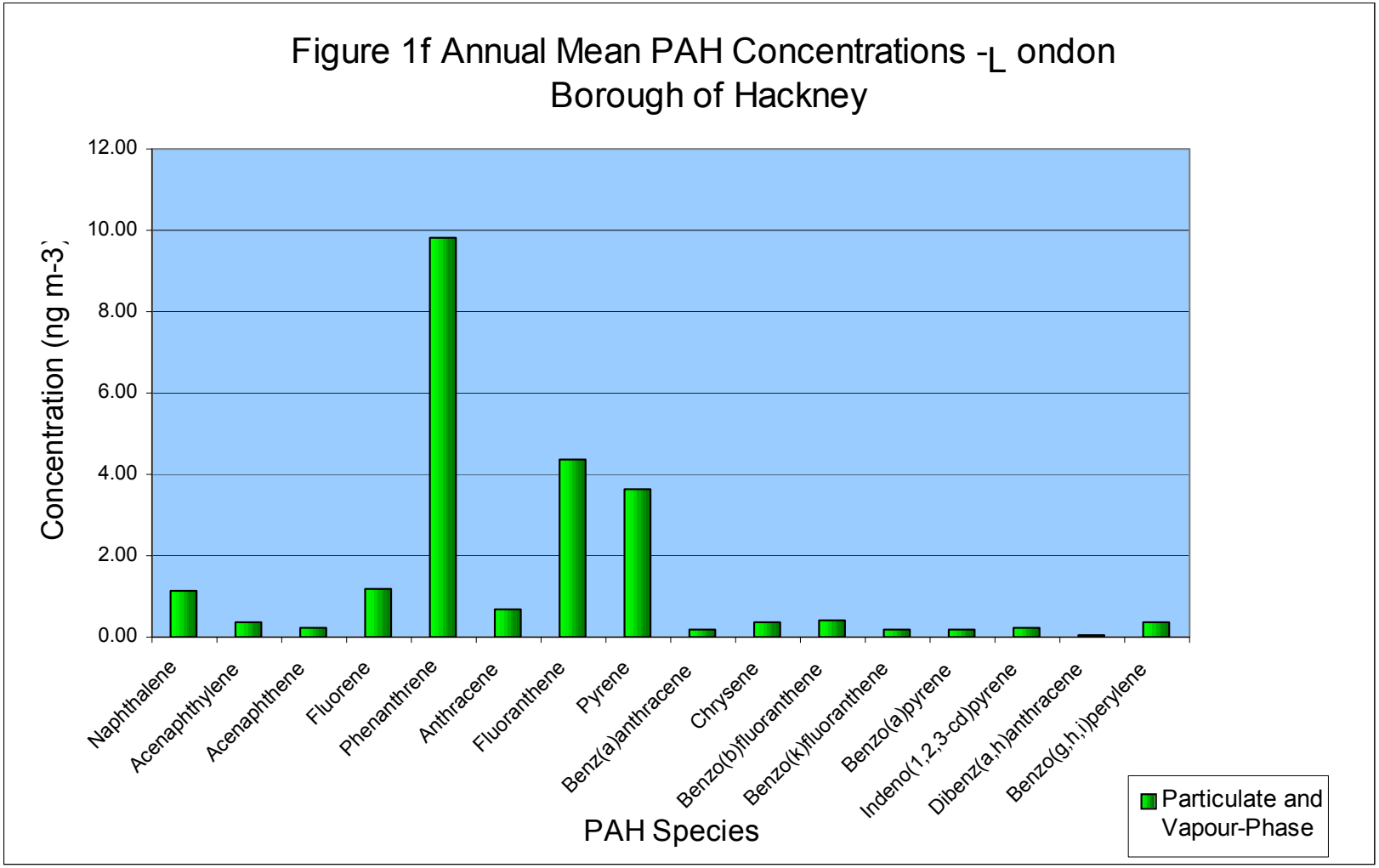


Figure 1f Annual Mean PAH Concentrations -L ondon
Borough of Hackney
Concentration (ng m-3)
12.00
10.00
8.00
6.00
4.00
2.00
0.00
Naphthalene
Acenaphthylene
Acenaphthene
Fluorene
Phenanthrene
Anthracene
Fluoranthene
Pyrene
Benz(a)anthracene
Chrysene
Benzo(b)fluoranthene
Benzo(k)fluoranthene
Benzo(a)pyrene
Indeno(1,2,3-cd)pyrene
Dibenz(a,h)anthracene
Benzo(g,h,i)perylene
PAH Species
Particulate and Vapour-Phase

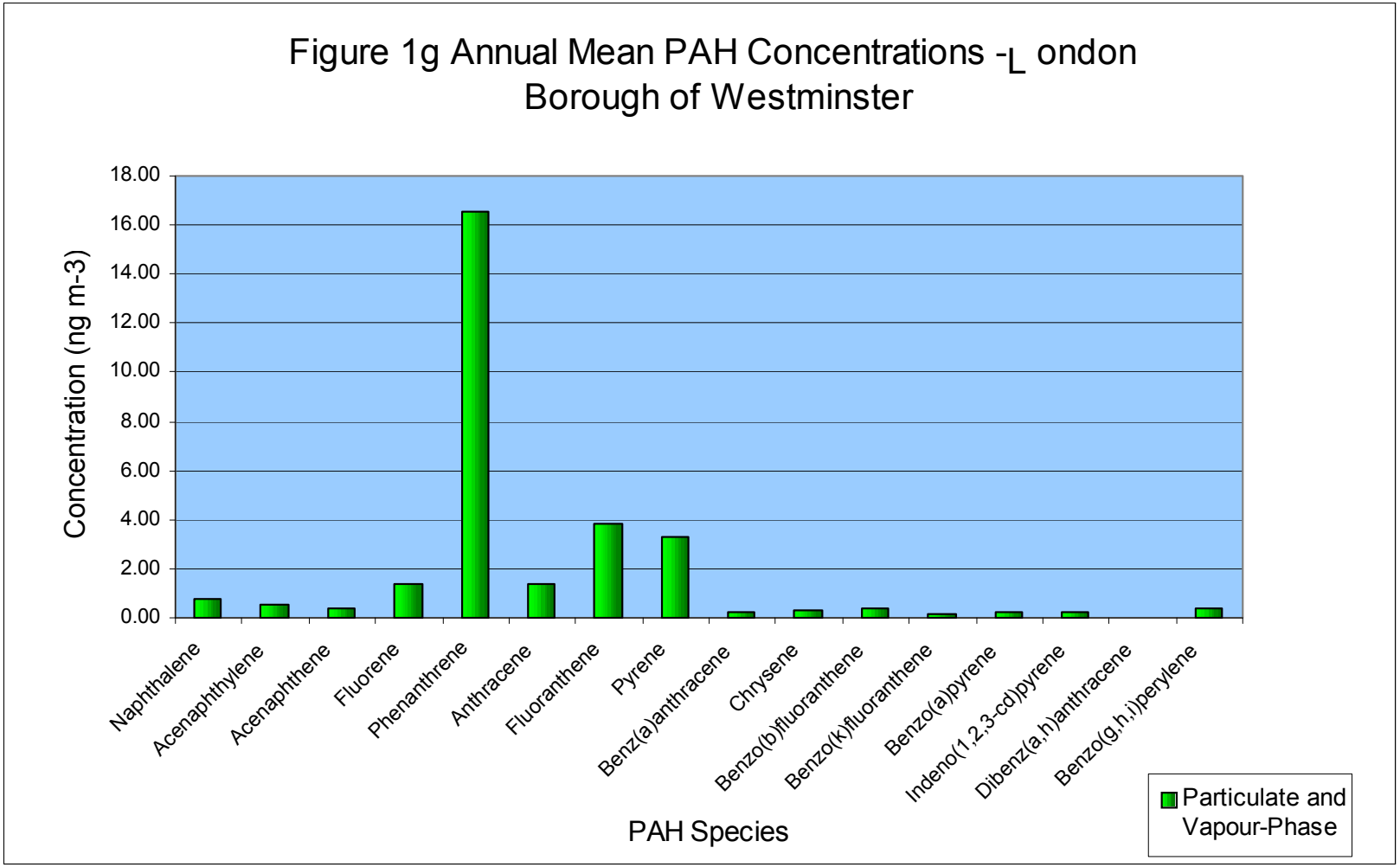
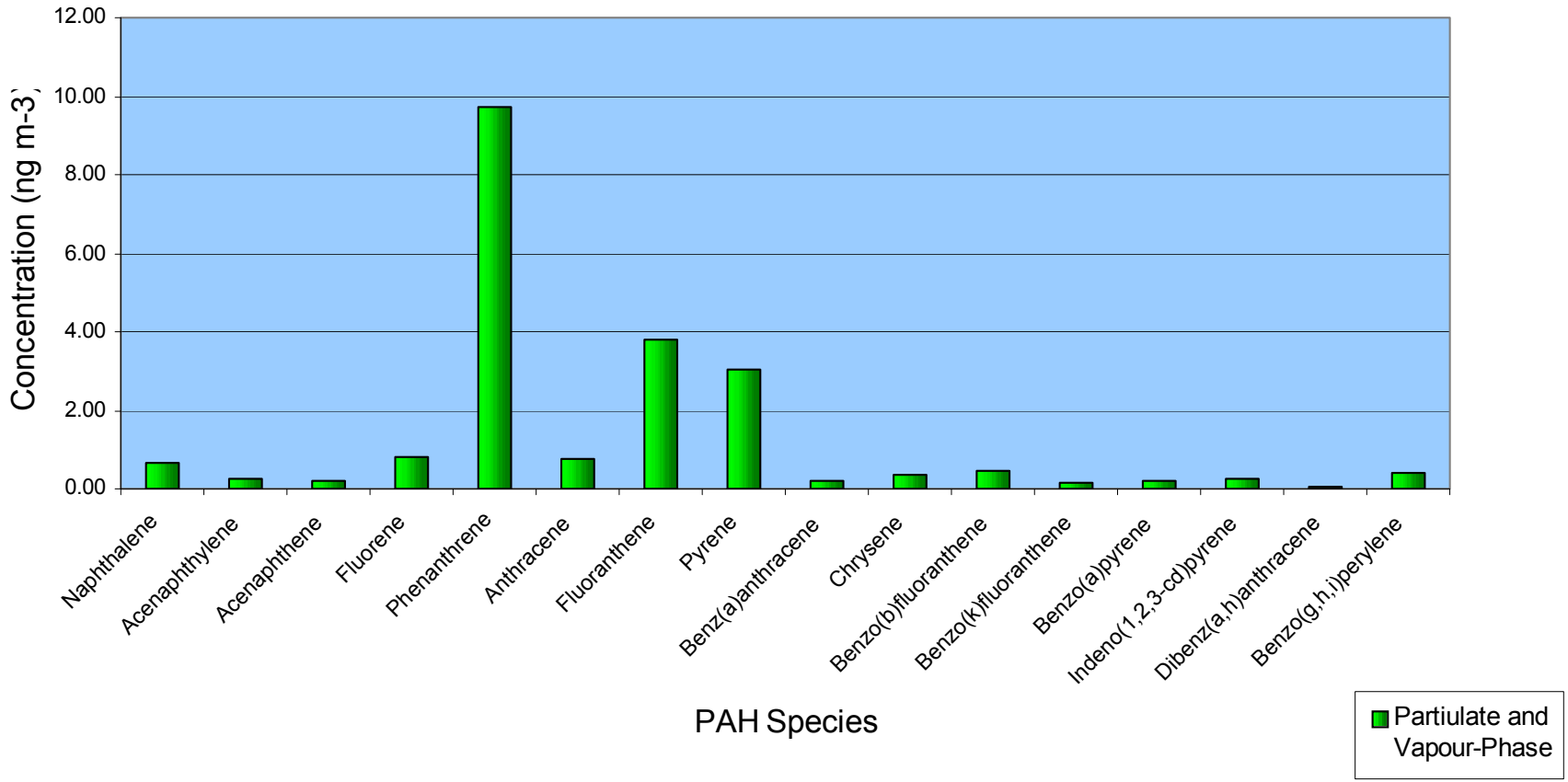


Figure 1g Annual Mean PAH Concentrations -L ondon Borough of Westminster
Concentration (ng m-3)
18.00
16.00
14.00
12.00
10.00
8.00
6.00
4.00
2.00
0.00
Naphthalene
Acenaphthylene
Acenaphthene
Fluorene
Phenanthrene
Anthracene
Fluoranthene
Pyrene
Benz(a)anthracene
Chrysene
Benzo(b)fluoranthene
Benzo(k)fluoranthene
Benzo(a)pyrene
Indeno(1,2,3-cd)pyrene
Dibenz(a,h)anthracene
Benzo(g,h,i)perylene
PAH Species
Particulate and Vapour-Phase

Figure 1h Annual Mean PAH Concentrations - All Borough



Appendix D
Figures 2 - 4

Figure 2. Mean Concentration of PAH Species at Roadside (Busy), Roadside (Intermediate) and Background Sites

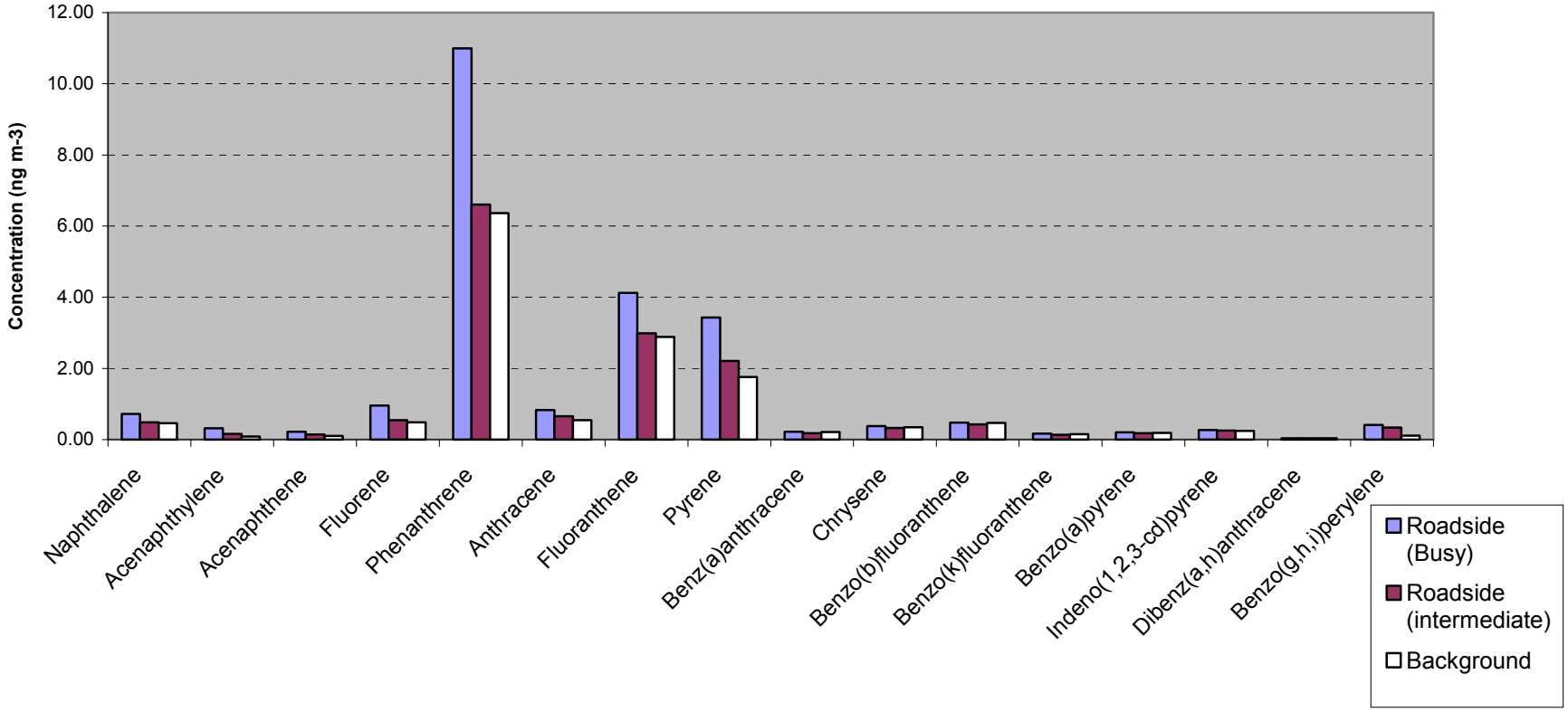


Figure 3. Mean Concentration of the Most Carcinogenic Compounds at Each Site

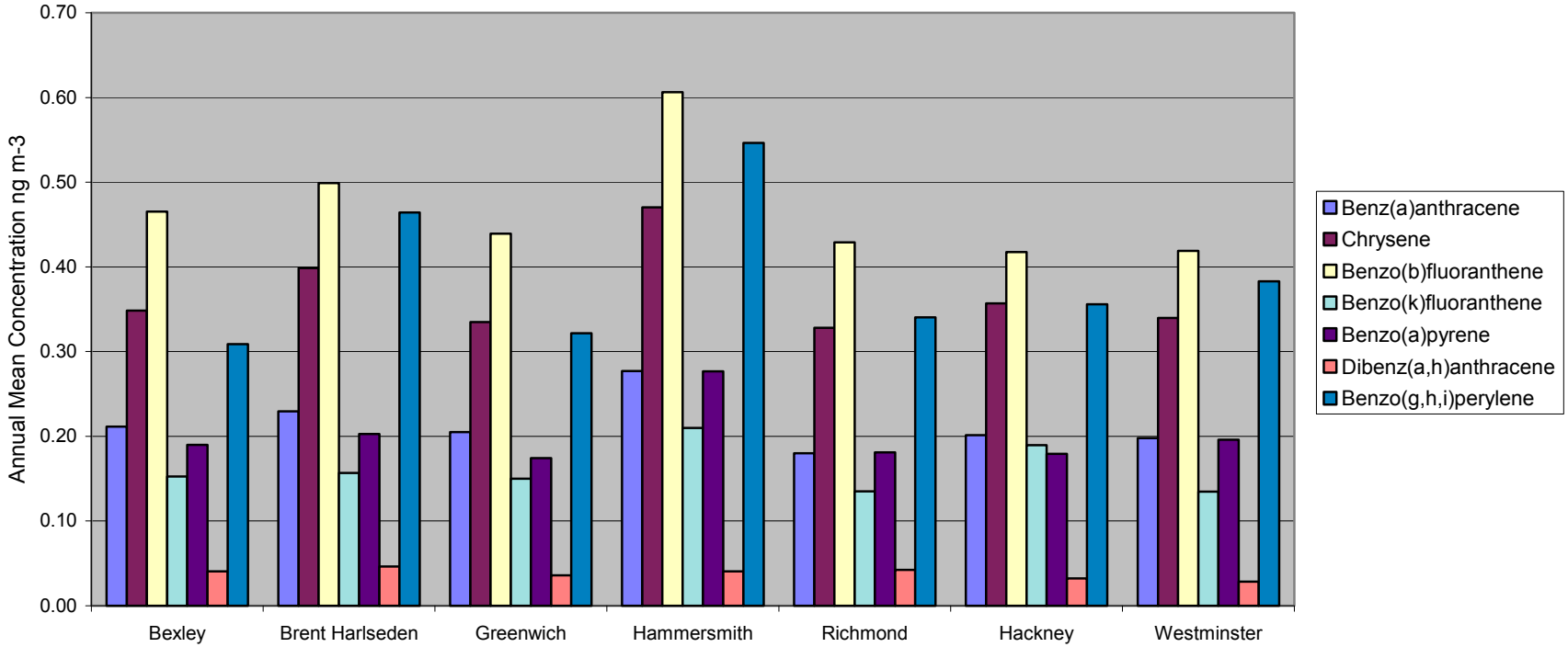


Figure 4. Particulate and Vapour Phase Annual Total PAH Concentration 2002/03 to 2004/05