



## London Wide Environment Programme



Polycyclic Aromatic Hydrocarbon Survey - 2005-2006

Ref: AGG04101



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## DOCUMENT INFORMATION AND CONTROL SHEET

**Report Title:** London Wide Environmental Programme  
Polycyclic Aromatic Hydrocarbon Survey -

**Report Ref:** BV/AQ/AGG04101/DM

**Client**

**London Borough of Bexley**  
**London Borough of Brent**  
**London Borough of Greenwich**  
**London Borough of Hackney**  
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**Document Status and Approval Schedule**

Issue	Status	Description	Prepared by: Daniel Moon Assistant Consultant Signed/Dated	Reviewed by: Jeff Booker Principal Consultant Signed/Dated
1	Draft Report	Issued to client by Email	29/08/06	12/09/06
2	Final Report	Issued to client by email and hard copy	15/09/06	15/09/06

# Contents

<b>CONTENTS .....</b>	<b>1</b>
<b>SUMMARY.....</b>	<b>2</b>
<b>1. INTRODUCTION .....</b>	<b>4</b>
<b>2. SOURCES AND CHEMICAL ORIGIN OF PAH.....</b>	<b>7</b>
<b>3. EFFECTS OF PAH ON HUMAN HEALTH .....</b>	<b>9</b>
3.1 GENERAL ASSESSMENT OF HUMAN EXPOSURE TO AIR POLLUTION .....	9
3.2 ASSESSMENT OF HEALTH EFFECTS OF AIR POLLUTION.....	10
3.3 ACUTE EFFECTS OF SHORT-TERM EXPOSURES TO AIR POLLUTANTS .....	11
3.4 CHRONIC EFFECTS OF LONG-TERM EXPOSURES TO AIR POLLUTION .....	12
3.5 HUMAN EXPOSURE TO POLYCYCLIC AROMATIC HYDROCARBONS (PAHS) .....	12
3.6 EFFECTS OF POLYCYCLIC AROMATIC HYDROCARBONS ON HUMAN HEALTH .....	13
<b>4. GUIDELINES AND STANDARDS .....</b>	<b>15</b>
<b>5. CONCENTRATIONS OF PAH IN URBAN AREAS.....</b>	<b>17</b>
<b>6. THE MEASUREMENT PROGRAMME .....</b>	<b>21</b>
6.1 MEASUREMENT SITES AND SAMPLING PERIODS .....	23
6.2 SCOPE AND PRINCIPLE OF SAMPLING.....	25
6.2.1 SAMPLING EQUIPMENT AND PREPARATION .....	26
6.2.2 ANALYTICAL PROCEDURE .....	28
<b>7. RESULTS .....</b>	<b>29</b>
7.1 CONCENTRATIONS – OVERVIEW .....	29
7.2 COMPARISON OF ROADSIDE (BUSY), ROADSIDE (INTERMEDIATE) AND BACKGROUND SITES .....	30
7.3 THE PAH PROFILE.....	30
7.4 BENZO(A)PYRENE CONCENTRATION AS A PERCENTAGE OF ALL MAJOR CARCINOGENIC PAHS .....	31
7.5 COMPARISON OF RESULTS WITH GUIDELINES FOR BENZO(A)PYRENE .....	32
<b>8. ASSESSMENT OF EFFECTS OF PAH ON THE LONDON POPULATION.....</b>	<b>33</b>
<b>9. CONCLUSION.....</b>	<b>35</b>
<b>10. RECOMMENDATIONS.....</b>	<b>37</b>
10.1 FUTURE DEVELOPMENTS TO PAH LEGISTRATION .....	37
<b>11. DISCLAIMER .....</b>	<b>40</b>
<b>12. REFERENCES.....</b>	<b>41</b>
<b>APPENDIX A .....</b>	<b>43</b>
<b>SITE DESCRIPTIONS .....</b>	<b>43</b>
<b>APPENDIX B .....</b>	<b>45</b>
<b>TABLES OF RESULTS 1 – 7.....</b>	<b>45</b>
<b>APPENDIX C .....</b>	<b>53</b>
<b>FIGURES 1A – 1G.....</b>	<b>53</b>
<b>APPENDIX D .....</b>	<b>61</b>
<b>FIGURES 2 – 5.....</b>	<b>61</b>

## 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 has been compared with guidelines for PAHs in order to increase the understanding of the scale of the pollution problem posed by PAHs in London.

Epidemiological studies of occupational exposure to PAHs have produced evidence that shows increased incidence of tumours of the lungs, skin and possibly bladder. Lung cancer is the most clearly linked to inhalation of PAHs in the air. Consumption of food that has been contaminated with PAHs through air pollution is also a major source of human exposure. It is estimated from a number of Benzo(a)pyrene (B[a]P) studies that exposure to B[a]P results in a range of unit lifetime risks from  $1.1 \times 10^{-3}$  to  $4.8 \times 10^{-3} \mu\text{g m}^{-3}$ . 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 <sup>(25)</sup>.

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  $0.25 \text{ ng m}^{-3}$  B[a]P had been recommended as the maximum desirable level for PAHs in ambient air if health effects are to be avoided <sup>(17)</sup>. 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 an objective for the whole UK of achieving the EPAQS recommended standard of  $0.25 \text{ ng m}^{-3}$  B[a]P as an annual average by the end of 2010. This will not be placed in regulations for the purposes of LAQM due to the high monitoring and analytical costs associated with this pollutant <sup>(21, 22)</sup>. In 2006 a consultation document on further improvements in air quality was published by Department for Environment, Food and Rural Affairs (Defra). This document reported that the current objective on PAHs will be retained with a view to consolidate current achievement and generate further improvements where feasible <sup>(22)</sup>.

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  $1.0 \text{ ng m}^{-3}$  to be achieved by 2012, and is based on the total benzo(a)pyrene content in the PM10 fraction. Since the EU

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.

Six measurement sites were chosen, one in each of the Boroughs participating in the 2005-06 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 four 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 current air quality objective for PAHs of  $0.25 \text{ ng m}^{-3}$  of B[a]P.

## 1. Introduction

Polycyclic aromatic hydrocarbons (PAHs) are a large group of organic compounds found throughout the environment. Chemically, PAHs are a group of hydrocarbons consisting of two or more fused benzene type rings. They are mainly produced from combustion of saturated hydrocarbon compounds (fossil fuels) under oxygen deficient conditions. Hydrocarbons with a low molecular mass form PAHs via a process called pyrosynthesis. This occurs at 500°C when all carbon-carbon and carbon-hydrogen bonds are broken to form free radicals. These radicals can then go onto dehydrogenate and form stable PAHs, which are resistant to thermal degradation <sup>(27)</sup>. Hydrocarbons with high molecular mass form PAHs first via pyrolysis first, then pyrosynthesis. Pyrolysis, otherwise known as 'cracking' is a process where large alkane chains are thermally broken down into smaller alkane chains. These smaller alkane chains then undergo pyrosynthesis. Many PAH species, including the best known of them, benzo(a)pyrene (B[a]P) as shown in Figure A, have been identified as precursors to cancer causing metabolites. The enzyme system responsible for PAH metabolism is a microsomal mixed function oxidase system. This system converts the non polar PAHs into polar hydroxy and epoxy derivatives, which can then be further enzymatically metabolised to other harmful compounds such as dihydrodiols and phenols. The epoxy derivative of B[a]P is also shown in Figure A. The system is extensively distributed in the cells and tissues of humans and animals. Airborne PAH concentrations are typically higher in urban and industrial areas compared with rural areas as PAHs are predominantly emitted by anthropogenic sources. Exposure of the urban population to PAHs may be linked to the higher incidence of respiratory problems in towns compared with rural areas <sup>(23)</sup>.

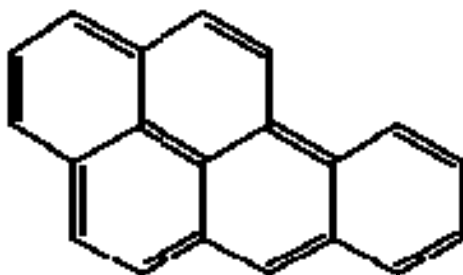
PAHs are present in both vapour and particulate phases in the atmosphere. The vapour phase consists of predominantly lower molecular weight PAHs whilst higher weight PAHs condense onto very small particulate nuclei to form particulate phase PAHs. Partition between the vapour and particulate phases is known to be temperature dependent and hence seasonal variation occurs in the concentrations of PAHs in the particulate phase. Many PAHs such as B[a]P are bound onto particulate matter. These PAHs are predominantly found in the particulate phase. However, the potential contribution of the vapour phase to ill health should not be ignored and various studies incorporate the gas phase to provide the total PAH concentration. The carcinogenic activity in occupational studies are due to the whole mixture of PAHs however for practical reasons a marker compound is needed to provide an indicator of this carcinogenic activity. The reason B[a]P was chosen as a marker for PAHs is because it provides a reasonably constant contribution to the carcinogenic effect of the PAH mixture as shown in Table A. In Table A the potential carcinogenic contribution of each compound is assessed by multiplying the relative concentration (RC) by the relative potency (RP) <sup>(26)</sup>.

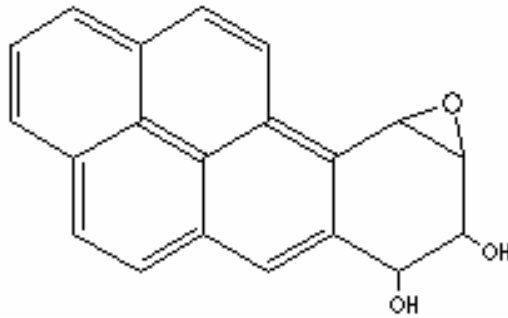
**Table A:** Estimated contribution relative to B[a]P of selected PAH compounds to the carcinogenic activity of PAH mixtures at different sites <sup>(26)</sup>.

Site Type	London: Mean 1991-95			Middlesbrough: Mean 1993-95			Aluminium Smelter		
Source	DETR, 1997			DETR, 1997			Farant & Gariepy, 1998		
PAH	RC	RP	RC x RP	RC	RP	RC x RP	RC	RP	RC x RP
Benzo[a]pyrene	1	1	1	1	1	1	1	1	1
Benz[a]anthracene	1.2	0.1	0.1	1.3	0.1	0.1	1.8	0.1	0.2
Dibenzo[ah]anthracene	0.3	1.91	0.6	0.5	1.91	1.0	0.1	1.91	0.2
Benzo[b]fluoranthene	2.4	0.11	0.3	2.4	0.11	0.3	3.8	0.11	0.4
Benzo[k]fluoranthene	2.1	0.03	0.1	1.9	0.03	0.1	1.2	0.03	0.1
Indeno[123cd]pyrene	1.8	0.08	0.1	1.5	0.08	0.1	0.7	0.08	0.1
Chrysene	2.0	0.03	0.1	2.3	0.03	0.1	3.5	0.03	0.1
% Contribution of B[a]P to total carcinogenic activity	44.6			37.5			49.3		

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) <sup>(11)</sup> 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 inter-site 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 2005-06 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 2005-06 survey may have on the health of the London population.

Benzo(a)pyrene, C<sub>20</sub>H<sub>12</sub>



7,8-Diol-9,10-epoxide of Benzo(a)pyrene,  $C_{20}H_{14}O_3$

**Figure A.** The Molecular Structure and Chemical Formula of Benzo(a)pyrene B[a]P <sup>(13)</sup> and 7,8-Diol-9,10-epoxide of Benzo(a)pyrene (carcinogenic metabolic product of B[a]P) <sup>(22)</sup>.

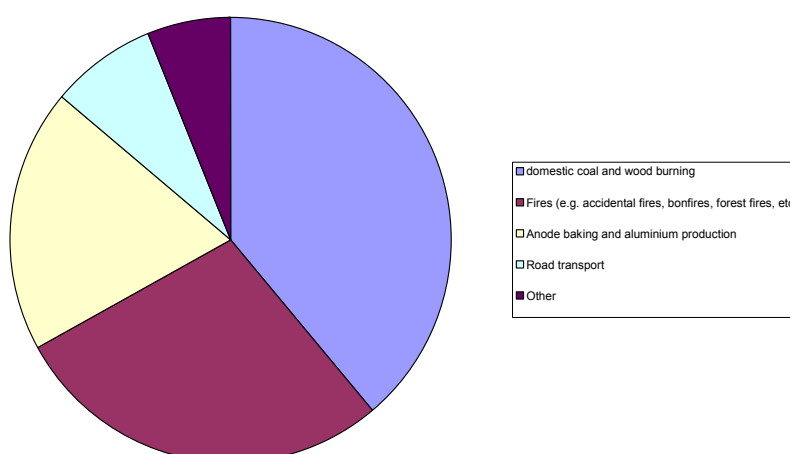
Other alternative names by which benzo(a)pyrene has been known.

- Benzo[ $\alpha$ ]pyrene
- 1,2-benzopyrene
- PAH EPA no. 73
- 4-benz[a]pyrene
- 3,4-Benzopyrene
- 3,4-BP
- 6,7-Benzopyrene
- Benzo[d,e,f]chrysene



## 2. Sources and Chemical Origin of PAH

PAHs are for the most part emitted by anthropogenic (human activity) sources where incomplete combustion of organic compounds occurs. In previous years road traffic emissions have been a dominant source of total PAH and B[a]P. The First Report of the Department of the Environment Quality of Urban Air Review Group <sup>(1)</sup> reported that firm evidence had been provided to demonstrate that the bulk of particulate PAHs derive from incomplete combustion of 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. 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 <sup>(15, 16)</sup>. However, 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 <sup>(17)</sup>. Measurements at the Marylebone Road monitoring site (a kerbside site) and past LWEP PAH Survey's in London have provided evidence that shows concentrations adjacent to heavily trafficked roads are not significantly higher than at urban background locations <sup>(17)</sup>. 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 <sup>(17)</sup>. Updated vehicle emissions inventories also indicate smaller releases from vehicles than previously estimated and now report that wood and coal burning is the UK's largest PAH source. The Air Quality Strategy Addendum (2003) reports that within the UK; wood and coal burning now accounts for 39% of the total PAH sources, fires 28%, anode baking and aluminium production 19% and road transport 8%. This information is represented in a pie chart in figure B <sup>(21)</sup>.



**Figure B.** Contribution of different sources of PAHs to the total concentration of PAHs in the UK (2003).

There has been a 75% reduction in the total PAH emissions since 1990. Emissions of B[a]P have fallen from 68 tonnes per annum in 1990 to 9.5 tonnes in 2000 and are projected to fall even further to 6.4 tonnes in 2010. However beyond 2010 emissions may increase due to a forecast increase in domestic coal, anthracite and solid smokeless fuel combustion. Emissions beyond 2010 are hard to project as there is a high amount of uncertainty beyond 2010. Atmospheric emissions of B[a]P for the UK since 1990 and projected future emissions from 2000 are shown in Table B. It can be seen from the Table B 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, which is expected to decrease further due to improvements in industrial abatement and process controls <sup>(17)</sup>. 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 <sup>(17)</sup>. 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 B:** UK Annual B[a]P Emissions 1990-99 and Projections to 2010 (tonnes) <sup>(17)</sup>

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
<b>Total</b>	<b>68.0</b>	<b>25.0</b>	<b>9.5</b>	<b>6.5</b>	<b>6.4</b>	<b>6.5</b>	<b>6.6</b>

PAH concentrations are also likely to be reduced as a result of measures in place to control particles. 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 <sup>(13)</sup>.

### 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 is where personal monitors are worn or carried by individuals. Most personal monitors, such as those developed for NO<sub>2</sub> or 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 is 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 <sup>(8)</sup>. Elevated incidences 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 <sup>(8)</sup>. One of the first such isolated chemicals to be proven to be linked to tumour formation was Benzo[a]pyrene <sup>(8)</sup>. 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 rings 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 <sup>(9)</sup>. The higher molecular mass PAHs, which tend to be found in the particulate phase, are reported to be the most carcinogenic <sup>(10)</sup>. The associated particulate usually measures less than a micron in diameter and can penetrate deep into the lungs. Particulates 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 carcinogenicity 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 over the past two decades. Two of the most authoritative are those of the World Health Organisation (WHO) [1987] <sup>(2)</sup> and Doll and Peto [1983] <sup>(3)</sup>; 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  $1 \times 10^{-4}\%$ . 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 an additive effect. 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 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  $0.25 \text{ ng m}^{-3}$  B[a]P as an annual average which applies to the total PAH concentration. 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 provisional objective of achieving the EPAQS recommended standard of  $0.25 \text{ ng m}^{-3}$  B[a]P as an annual average by the end of 2010. This objective is intended to reduce any risk to the population from exposure to PAHs. However the EPAQS commented that they believe that all exposure above this standard carries a significant risk as a tenfold safety factor was used while deriving the standard. This will not be a national standard for the time being and will not be placed in regulations for the purposes of LAQM due to high monitoring and analytical cost for this pollutant; however this will be reassessed at the next periodic review of the Air Quality Strategy <sup>(21)</sup>. In 2006 a consultation document on further improvements in air quality was published by Department for Environment, Food and Rural Affairs (Defra). This document reported that the current objective on PAHs will be retained with a view to consolidate current achievement and generate further improvements where feasible <sup>(22)</sup>.

Benzo[a]pyrene is used as a marker for the purposes of the guideline and represents the total mixture of PAH in the UK due to its remarkable stability and its relatively high and constant contribution to the carcinogenicity of PAH mixtures.

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  $5 \text{ ng m}^{-3}$  <sup>(4)</sup>. A guideline for the annual average B[a]P concentration proposed by the German Environmental Agency is  $10 \text{ ng m}^{-3}$  <sup>(4)</sup>.

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  $1.0 \text{ ng m}^{-3}$  to be achieved by 2012. This is based on the total benzo(a)pyrene content in the PM<sub>10</sub> 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)fluoranthene,
- benzo(j)fluoranthene,
- benzo(k)fluoranthene,
- indeno(1,2,3-cd)pyrene,
- dibenz(a,h)anthracene.

In addition, monitoring sites should be selected so that geographical variations and long term trends can be identified.

## 5. Concentrations of PAH in Urban Areas

Compared with other pollutants, the monitoring of organic compounds in urban areas of the UK is still in its infancy and long time-series databases are not currently available. However measurements that have been taken in particularly problematic areas since 1990 of B[a]P have shown a decrease of 87% in levels and a 75% reduction in total PAH emissions <sup>(22)</sup>.

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 <sup>(18)</sup>. 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 <sup>(17)</sup>. These additional sites are being located mainly at urban background locations whereas the 15 sites are situated at various city centres, rural and near to industrial sources. Sites from the TOMPS network have been established in London (Westminster) and Manchester since 1991 and additionally at Middlesbrough 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 C lists the national PAH monitoring sites including potential additional sites and includes the annual average B[a]P concentration from 1999 to 2004.

**Table C:** Currently Operating National PAH Monitoring Sites and the Annual Average B[a]P Concentrations (ng m<sup>-3</sup>) from 2000 to 2005 <sup>(17)</sup>.

Site	Site Type	Monitoring commenced	Concentration (ngm <sup>-3</sup> )					
			2000	2001	2002	2003	2004	2005
Ashington	Urban Industrial	1999	0.17	0.20	0.15	0.19	0.15	0.16
Bolsover	Industrial	1999	0.25	0.28	0.24	0.46	0.22	0.23
Glasgow	Urban	1999	0.12	0.12	0.12	0.07	0.07	0.1
Hazelrigg	Semi-rural	1992	0.06	0.08	0.04	0.04	0.02	0.02
Holyhead	Urban Industrial	1999	0.11	0.15	0.18	0.14		
High Muffles	Rural	1997	0.04	0.05	0.03	0.05	0.02	0.03
Kinlochleven	Urban Industrial	1999	2.27	0.34	0.38	0.21	0.32	0.31
Lisburn	Urban	1999	0.95	0.94	0.65	1.00	0.62	0.61
London	Urban	1991	0.14	0.14	0.13	0.12	0.07	0.08
Manchester	Urban	1991	0.24	0.34	0.12	0.24	0.11	0.10
Middlesbrough	Urban Industrial	1992	0.28	0.37	0.15	0.24	0.14	0.18
Newport	Urban Industrial	1999	0.35	0.36	0.19	0.11	0.10	0.1
Port Talbot	Urban Industrial	1999	0.59	0.40	0.34	0.47	0.29	0.37
Scunthorpe	Urban Industrial	1999	1.16	0.34	1.35	1.30		0.95
Stoke Ferry	Rural	1997	0.09	0.09	0.08	0.08	0.04	0.06
<b>New Sites</b>			<b>2000</b>	<b>2001</b>	<b>2002</b>	<b>2003</b>	<b>2004</b>	<b>2005</b>
Belfast		2001		0.37	0.13	0.08	0.15	0.27
Birmingham		2001		0.16	0.13	0.16		0.12
Brent	Suburban	2002			0.18	0.21	0.10	0.11
Bromley	Roadside	2001		0.20	0.25	0.10	0.19	0.17
Hove		2002			0.18	0.16	0.09	0.01
Leeds		2001		0.16	0.18	0.21		0.17
Newcastle		2001		0.11	0.12	0.16	0.06	0.08
Speke		2001		0.08	0.14	0.14	0.10	0.1

(Extracted from the Air Quality Strategy - Consultation Document 2001<sup>(17)</sup> and updated with the means calculated data from the UK National Air Quality Information Archive)

PAH measurements made during 2005 at the Manchester, Middlesbrough and London TOMPS sites are given in Table D. Measurements for one rural site also in the TOMPS network are shown for comparison.

**Table D:** 2005 Annual Average Particulate PAH Concentrations (ngm<sup>-3</sup>) for London, Manchester, Middlesbrough and High Muffles

Compound	London	Manchester	Middlesbrough	High Muffles
Acenaphthylene	0.35	0.37	0.2	0.024
Acenaphthene	0.59	0.63	1.4	0.17
Fluorene	2.7	2.8	3.8	1.1
Phenanthrene	12	14	12	3
Anthracene	0.38	0.55	0.42	0.05
Fluoranthene	2	2.3	2.6	0.59
Pyrene	1.6	2	1.4	0.36
Benzo[a]anthracene	0.11	0.17	0.24	0.038
Chrysene	0.22	0.31	0.44	0.069
Benzo[b]fluoranthene *				
Benzo[k]fluoranthene				
Benzo[a]pyrene	0.08	0.10	0.18	0.025
Indeno[1,2,3 cd]pyrene	0.14	0.19	0.26	0.037
Dibenz[ac/ah]anthracene	0.02	0.025	0.033	0.009
Benzo[ghi]perylene	0.18	0.2	0.23	0.035
<b>Sum of PAHs</b>	<b>20.37</b>	<b>23.645</b>	<b>23.203</b>	<b>5.507</b>

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

The number of people exposed to B[a]P above the air quality objective concentration is expected to decrease from 2.4 million in 2003 to 1.66 million in 2010. Most of these people are likely to be situated in London and Belfast. The data reported in the technical annex to the 2006 consultation document is shown in Table E. It shows expected trends in the amount of people and background area that are exposed to concentrations of B[a]P above the current objective for 2003, 2010, 2015, and 2020.

**Table E:** Background area, and population in the background area exceeding the B[a]P objective of  $0.25 \text{ ng m}^{-3}$  in 2003, 2010, 2015 and 2020 by region.

	Area Assessed (km <sup>2</sup> )	2003		2010		2015		2020	
		Area (km <sup>2</sup> )	No. of people (x10 <sup>3</sup> )	Area (km <sup>2</sup> )	No. of people (x10 <sup>3</sup> )	Area (km <sup>2</sup> )	No. of people (x10 <sup>3</sup> )	Area (km <sup>2</sup> )	No. of people (x10 <sup>3</sup> )
London	1629	174	1317	124	988	118	947	117	940
Rest of England	128547	81	162	58	70	58	70	58	70
Scotland	77473	26	68	6	16	6	16	6	16
Wales	20665	2	0	2	0	2	0	2	0
Northern Ireland	13724	439	861	238	587	257	620	256	619
Total UK	242038	772	2407	428	1661	441	1653	439	1645
Population – weighted mean B[a]P concentrations (ng m <sup>-3</sup> )									
		2003		2010		2015		2020	
Inner London		0.20		0.19		0.19		0.19	
Outer London		0.20		0.20		0.20		0.20	
Rest of England		0.12		0.11		0.11		0.11	
Scotland		0.13		0.12		0.12		0.12	
Wales		0.09		0.09		0.09		0.09	
Northern Ireland		0.31		0.23		0.24		0.24	
UK		0.14		0.13		0.13		0.13	

## 6. The Measurement Programme

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 <sup>(17)</sup>. These sub-sets and PAH species are shown in Table F

**Table F:** PAH Species and Sub-sets <sup>(17)</sup>

Species	US EPA priority pollutants <sup>1</sup>	IARC Group 2a <sup>3</sup>	IARC Group 2b <sup>3</sup>	UNECE POPs Protocol	“Borneff 6”
Napthalene	3				
Acenaphthene	3				
Acenaphthylene	3				
Fluorene	3				
Anthracene	3				
Phenanthrene	3				
Fluoranthene	3				3
Pyrene	3				
Benz[a]anthracene	3	3			
Chrysene	3				
Benzo[b]fluoranthene	3		3	3	3
Benzo[l]fluoranthene			3		
Benzo[k]fluoranthene	3		3	3	3
Benzo[a]pyrene	3	3		3	3
Dibenzo[a,h]anthracene	3	3			
Dibenzo[a,c/a,h]anthracene					
Dibenzo[a,e]pyrene			3		
Dibenzo[a,h]pyrene			3		
Dibenzo[a,l]pyrene			3		
Dibenzo[a,i]pyrene			3		
Indeno[1,2,3-c,d]pyrene	3		3	3	3
5-methylchrysene			3		
Benzo[ghi]perylene	3				3
Coronene					
All Species are currently monitored within the PAH network					
<sup>1</sup> Included in National Atmospheric Emissions Inventory. Sum of emissions corresponds to total PAH					
<sup>2</sup> Possible human carcinogen					
<sup>3</sup> 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 G.

**Table G:** The PAH Compounds Measured

Compound and Abbreviation		Cancer Rating(a)	Rings	Mol wt
Naphthalene	Np	?	2	128
Acenaphthene	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:

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.

### 6.1 Measurement Sites and Sampling Periods

Six 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 April 2005 – March 2006. Table H below shows sampling periods across all Boroughs but excludes invalid samples identified from subsequent quality control checks.

**Table H:** Sampling Periods and Site Classification Across all Boroughs

Authority	Classification	Sampling Periods
Bexley	Background	(03/05/05 - 16/05/05), (01/06/05 - 14/06/05), (05/07/05 - 19/07/05), (02/08/05 - 10/08/05), (22/08/05 - 06/09/05), (22/09/05 - 05/10/05), (20/10/05 - 03/11/05), (24/11/05 - 08/12/05), (21/12/05 - 05/01/06), (26/01/06 - 07/02/06), (21/02/06 - 07/03/06), (22/03/06 - 06/04/06)
Greenwich	Roadside (busy)	(03/05/05 - 16/05/05), (01/06/05 - 14/06/05), (05/07/05 - 19/07/05), (02/08/05 - 10/08/05), (22/08/05 - 06/09/05), (22/09/05 - 05/10/05), (20/10/05 - 03/11/05), (24/11/05 - 08/12/05), (21/12/05 - 05/01/06), (26/01/06 - 07/02/06), (21/02/06 - 07/03/06), (22/03/06 - 06/04/06)
Brent	Roadside (busy)	(29/04/05 - 10/05/05), (31/05/05 - 10/06/05), (04/07/05 - 18/07/05), (01/08/05 - 09/08/05), (25/08/05 - 05/09/05), (23/09/05 - 04/10/05), (25/01/06 - 08/02/06), (20/02/06 - 06/03/06), (23/03/06 - 05/04/06)
Hackney	Roadside (busy)	(29/04/05 - 10/05/05), (31/05/05 - 10/06/05), (04/07/05 - 18/07/05), (01/08/05 - 09/08/05), (25/08/05 - 05/09/05), (23/09/05 - 04/10/05), (25/01/06 - 08/02/06), (20/02/06 - 06/03/06), (23/03/06 - 05/04/06)
Richmond	Roadside (intermediate)	(03/05/05 - 16/05/05), (01/06/05 - 14/06/05), (05/07/05 - 19/07/05), (02/08/05 - 10/08/05), (22/08/05 - 06/09/05), (22/09/05 - 05/10/05), (20/10/05 - 03/11/05), (24/11/05 - 08/12/05), (21/12/05 - 05/01/06), (26/01/06 - 07/02/06), (21/02/06 - 07/03/06), (22/03/06 - 06/04/06)
Hammersmith & Fulham	Roadside (busy)	(29/04/05 - 10/05/05), (31/05/05 - 10/06/05), (04/07/05 - 18/07/05), (01/08/05 - 09/08/05), (25/08/05 - 05/09/05), (23/09/05 - 04/10/05), (25/01/06 - 08/02/06), (20/02/06 - 06/03/06), (23/03/06 - 05/04/06)

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 I. Descriptions of each site are also provided in Appendix A.

**Table I:** Summary of Site Descriptions and Classification for Participants in the Polycyclic Aromatic Hydrocarbon Survey 2005-06.

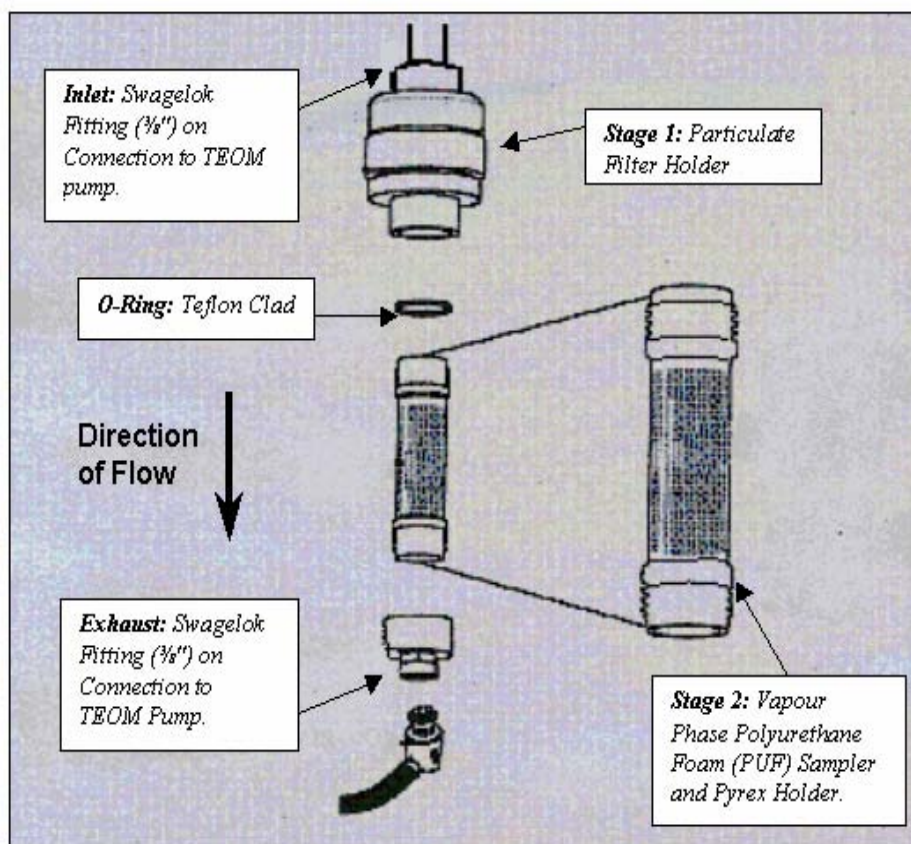
Local	Location	Classification
Site 1 – Bexley	Air monitoring cabin located at Whitehall day centre in Bexley, approx. 200m from the nearest busy road.	Background
Site 2 – Brent Harlesden	Air monitoring cabin located at Budgen car park in Harlesden. Approx. 2m from the nearest busy road.	Roadside (Busy)
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)

## 6.2 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.

### 6.2.1 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 PM10 inlet to perform a 10µm particle size cut-off. Figure C below shows the type of filter and PUF sampler used for sampling and Figure D is a photograph of the actual Filter/PUF sampler used in the survey.



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



**Figure D:** Photograph of Particulate/Vapour-Phase Filter and PUF Sampler used in the project.

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  $10\mu\text{m}$  diameter takes place as the sample passes through the PM10 inlet. This inlet also protects the sample from adverse weather conditions and ultraviolet sources. A flow splitter separates the total flow of  $16.7\text{l/m}$  into two parts: a main flow of  $3.0\text{l/m}$  that enters the TEOM sensor unit through the sample tube, and the auxiliary (bypass) flow of  $13.7\text{l/m}$ . 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<sup>3</sup> of air ± 3 days.

### *6.2.2 Analytical Procedure*

The multi component samples on the filter medium were pre-spiked with a sampling standard which are extracted with dichloromethane.

The sampling standard comprises of 1000ng of:

- D8-naphthalene,
- D8-acenaphthene,
- D10-anthracene,
- D10-pyrene,
- D12-benzo(b)fluoranthene,
- D14-dibenzo(ah)anthracene.

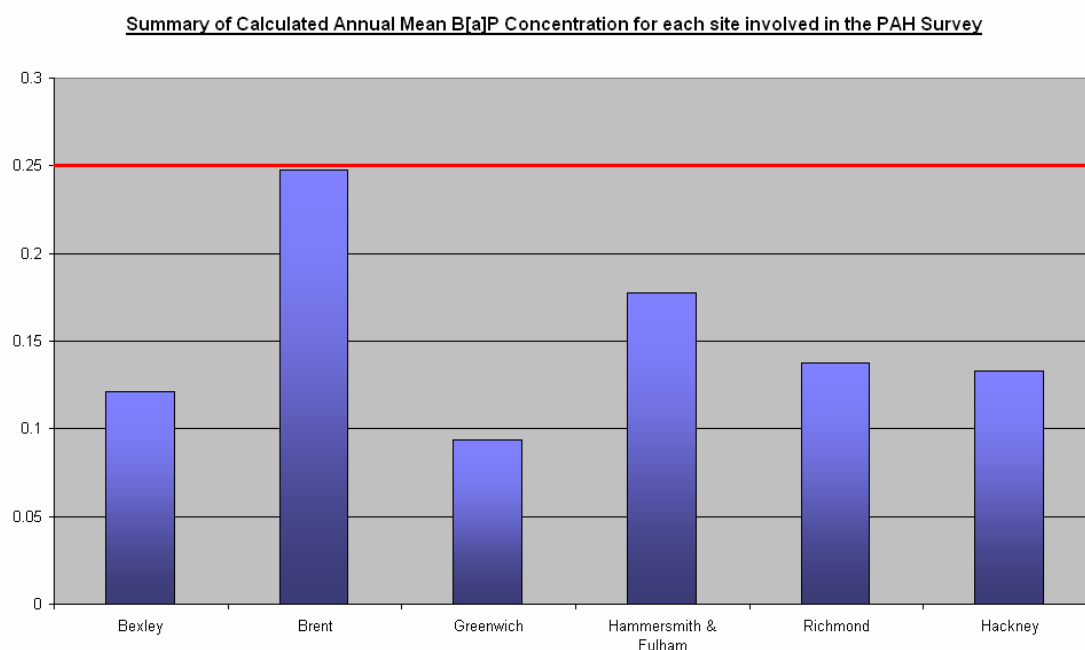
The sample extracts were then evaporated down to a few mls in volume. After the addition of a recovery spike (1000ng of each of D10-fluorene, D12-chrysene and D12-benzo(ghi)perylene) the sample was cleaned up by column chromatography and a syringe standard (1000ng of each of D10-fluoranthene and D12-benzo(a)pyrene) 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 monthly concentrations of particulate PAH measured at each site during the survey are given in Appendix B, Tables 1 to 6 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 7. Figure E below summarises annual mean B[a]P concentration for the particulate phase measured for participants within the survey. Annual mean values have been calculated using all available data.

**Figure E:** Bar chart of annual mean B[a]P concentration for each site in LWEP PAH Survey 2005-2006



### 7.1 Concentrations – Overview

Annual mean B[a]P concentrations of between 0.09ng m<sup>-3</sup> – 0.25ng m<sup>-3</sup> was calculated for the six participants in the survey. The highest mean B[a]P concentration of 0.25ng m<sup>-3</sup> was recorded for Brent, a roadside (busy) site. The lowest mean value of 0.09ng m<sup>-3</sup> was recorded for the Greenwich site a roadside (busy) site located next to the A2.

Monthly mean concentrations varied from 0.02 ng m<sup>-3</sup> to 1.57 ng m<sup>-3</sup>. The highest mean concentration was recorded at Brent, a roadside (busy) and the lowest mean concentration was recorded at Bexley and Greenwich. The seasonal variation in the concentration of B[a]P is represented in Figure 5,

Appendix D. Figure 5 shows us that the concentration of PAHs increases in the winter months. This could be associated with an increase in the use of cars and fuel to heat homes.

Mean PAH concentrations measured for Bexley, Brent, Greenwich, Hammersmith and Fulham, Richmond and Hackney are illustrated in Appendix C, Figures 1a to 1f respectively. The concentrations for each of the USEPA 16 listed species from the particulate phase are presented. Figure 1g 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. A possible reason such low B[a]P level was recorded at Greenwich is that the predominant source of PAH around the Greenwich site is not industry but road traffic which is only a small contributor to annual B[a]P emissions in the UK.

## *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 with some 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 total PAH obtained for each of the sites within the survey for the past three surveys. Over past surveys the total amount of PAHs sampled at roadside (busy) sites have been consistently higher than at roadside (intermediate) and background sites.

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:

$$\text{Equation 1: } \% \text{ [B[a]P]} = \frac{[\text{B[a]P}] \times 100}{[\text{BaA}] + [\text{CHR}] + [\text{BbF}] + [\text{BkF}] + [\text{B[a]P}] + [\text{DahA}] + [\text{BghiP}]}$$

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

**Table J:** 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	% B[a]P
Bexley	Background	8.76
Brent Harlesden	Roadside (Busy)	10.91
Greenwich	Roadside (Busy )	5.71
Hammersmith & Fulham	Roadside (Busy)	8.42
Richmond	Roadside (intermediate)	8.87
Hackney	Roadside (Busy)	7.59
<b>Arithmetic Mean</b>		<b>8.38</b>

A percentage of 8.76% was recorded at the background site; 8.87% at the roadside (intermediate) site; and values at roadside (busy) sites ranged from 5.71% to 10.91%.

### 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 ( $0.25 \text{ ng m}^{-3}$ ) 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 K: Tabular Summary of Annual Mean B[a]P Concentration Measured from the Particulate Phase.

Borough	Concentration $\text{ng m}^{-3}$ (2d.p.)
Bexley	0.12
Brent Harlesden	0.25
Greenwich	0.09
Hammersmith and Fulham	0.18
Richmond	0.14
Hackney	0.13

The survey results indicate that the estimated annual mean B[a]P concentration recorded at all sites do not exceed the current EU directive value ( $1 \text{ ng m}^{-3}$ ) for PAHs in ambient air. In addition the annual mean B[a]P concentration at all sites did not exceed the current objective ( $0.25 \text{ ng m}^{-3}$ ) for PAHs in ambient air.

## 8. Assessment of Effects of PAH on the London Population

The PAH standard of  $0.25\text{ng m}^{-3}$  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  $1\text{ng m}^{-3}$  for a working lifetime. The annual mean concentration of B[a]P recorded at the background site in the present survey was  $0.12\text{ng m}^{-3}$ . Hence, for an individual living at a background location over a lifetime, the respiratory cancer risk would be  $1.2 \times 10^{-6}$ . 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  $0.25\text{ng m}^{-3}$ , resulting in a respiratory cancer risk of  $2.5 \times 10^{-6}$ .

The risk to the population of PAH exposure may however be much greater than these calculations imply. B[a]P as a percentage of the total carcinogenic PAH in this study was 8.38%, a very small percentage of totals carcinogenic PAHs. Using B[a]P alone does not take into account any risk arising from the mixture 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  $10\text{ng B[a]P(2)}$  in the mainstream smoke. For comparison assuming a daily-inhaled volume of  $20\text{m}^3$ , an individual spending 8 hours per day at the most polluted site in this study would inhale  $1.9\text{ng}$  check against previous year's data - equivalent to smoking just over one cigarette per week.

Equation 2:

1.  $8\text{hrs} \times 20\text{m}^3 = 6.67\text{m}^3$  breathed 24hrs
2.  $6.67\text{m}^3$  breathed  $\times 0.25\text{ng} = 1.7\text{ng B[a]P}$  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.

High PAH concentrations were recorded at all sites during the winter months. This could be associated with the Buncefield fire in the southeast of England on the 11<sup>th</sup> December 2005. Three of the twenty four PAH network sites in the UK are located in the southeast of England; at Brent (Kingsbury High School), London (Victoria Street) and Bromley (Crystal Palace Parade). Analysis of the data from these sites showed that there was a rise in PAH concentrations during the week in which the Buncefield site was burning. Brent showed the greatest increase while Victoria Street showed the least. This could be due to seasonal variations or other local/regional sources. To confirm that the increase in PAH concentration was due to the Buncefield fire further dispersion modelling and analysis of samples from the PAH network would be needed <sup>(28)</sup>.

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 current air quality objective for England and Wales of 0.25 ng m<sup>-3</sup> B[a]P by the end of 2010. In addition, the higher EU target value of 1ng m<sup>-3</sup> 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 0.12, 0.14 and 0.16ng m<sup>-3</sup> 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 1.2 x 10<sup>-6</sup>, 1.4 x 10<sup>-6</sup> and 1.6 x 10<sup>-6</sup> 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 L <sup>(18)</sup> <sup>(19)</sup>.

**Table L:** 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 <sup>(18)</sup> .

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 <sup>(17)</sup>. 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 <sup>(17)</sup>.

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.

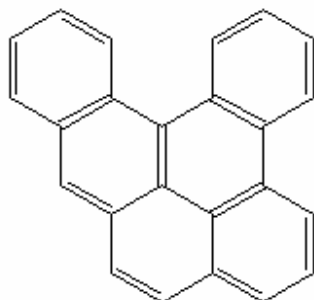
Subsequently the following years to present studies have used the GC-MS analytical method to coincide with the method used in Europe. However the sampling method was modified to include both the PM10 particulate fraction and the vapour phase component for collection of 'total PAH'. The analysis of samples was covered by a UKAS accredited laboratory to improve the credibility of the data. As would be predicted, the sampling of PM10 particulate, as compared to the PM16 (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  $0.25 \text{ ng m}^{-3}$  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.

### *10.1 Future Developments to PAH Legislation*

Some new evidence has come to light that the PAH marker chosen, B[a]P may be not as good as once thought. PAHs such as Dibenzo[a,l]pyrene (DB[a,l]P), as shown in Figure F have been found to be particularly carcinogenic

(22).

Dibenzo[a,l]pyrene, C<sub>24</sub>H<sub>14</sub>

**Figure F:** The Molecular Structure and Chemical Formula of Dibenzo[a,l]pyrene (DB[a,l]P) <sup>(24)</sup>

The Committee of Carcinogenicity (COC) was asked in 2003 to categorise DB[a,l]P into the COC hazard ranking scheme and to consider from data available the relative carcinogenic potency of DB[a,l]P compared with B[a]P, particularly when inhaled. The COC agreed that the in-vitro mutagenicity tests and information on in-vivo DNA adduct formation was consistent with dibenzo(a,l)pyrene being an in-vivo mutagen. When DB[a,l]P was dermatologically applied to mice and rats tumours occurred at various sites (including the skin, lung, and malignant lymphoma of the spleen and malignant lymphoma with multiple organ involvement) and intraperitoneal administration to rats produced lung tumours. Intramammary instillation in rats resulted in mammary tumours. DB[a,l]P was also found to be an initiator in mouse skin carcinogenicity promotion assays. Due to these findings the COC decided that DB[a,l]P should be assigned to group A of its hazard ranking scheme for PAHs. This category includes chemicals for which 'there is a high level of concern about a carcinogenic hazard for humans because the compound is an in-vivo mutagen and/or a multi-site carcinogen in more than one species.' The COC also agreed that DB[a,l]P is a very potent genotoxic carcinogen which is dependent on species, dose, route of administration and site of tumour is produced. From data where a comparison can be made, depending on the particular test DB[a,l]P is usually found to be anywhere from 10 to 100 times more potent than B[a]P. The measurement of DNA adducts (Time Integrated DNA Adduct Levels: TIDAL) following intraperitoneal dosing of DB[a,l]P showed that DB[a,l]P bound much more extensively to DNA, in comparison to other PAHs. This is seemingly due to the higher reactivity of its diol-epoxide metabolite. However due to insufficient data the committee was unable to conclude on the relative potency of DB[a,l]P compared to B[a]P by the inhalation route of exposure and there was a need to address this compound <sup>(23)</sup>.

If such PAHs were present in some places and not present in others or were not present in the atmospheres of aluminium smelters, which were used as a starting point for deriving the current PAH objective from EPAQS, then B[a]P would not represent the carcinogenicity of PAH mixtures well. The consequence,



if this was to be confirmed, would be a reformulation of the current objective. However, there is insufficient spatial data about variations in concentration of these extremely potent PAHs to reformulate the objective. The relative potency of different PAHs would also be needed as to gain an overall picture of the total carcinogenic effect that PAHs have upon human health. Dibenzo[a,l]pyrene is found in a relatively small concentration around the UK compared to other PAHs. Typical concentrations taken from the air quality archive are shown in Table H below. However, it is not recommended that this PAH be included in future LWEP monitoring programmes since:

- a. analytical costs are high due to the compound's steric bulk causing a long eluent time during the chromatography stage of analysis, and
- b. there is a lot unknown about the compounds carcinogenic potency via the inhalation route of exposure <sup>(22)</sup>.

**Table H:** Annual mean concentrations of Dibenzo[a,l]pyrene ( $\text{ng m}^{-3}$ ) taken at different sites across the UK.

Site	2001	2002	2003	2004	2005
Belfast	0.31	0.1	0.04	0.071	0.073
Bolsover	0.16	0.081	0.19	0.067	0.066
London	0.048	0.04	0.028	0.01	0.021
Glasgow	0.058	0.041	0.025	0.018	0.013

## 11. Disclaimer

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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 & 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 is within the enclosure at Castelnau Library.

#### **Site 6 – Hackney**

A roadside (busy) air monitoring cabin located beside the fire station on Old Street, approx. 6m from busy road.

**Appendix B**  
**Tables of Results 1 – 7**

**Table 1 - The London Borough of Bexley - Monthly Concentrations of PAH / ng m<sup>-3</sup>**

	Apr-05	May-05	Jun-05	Jul-05	Aug-05	Sep-05	Oct-05	Nov-05	Dec-05	Jan-06	Feb-06	Mar-06
Naphthalene	0.39	0.24	0.58	0.18	0.12	0.47	0.37	<20	0.16	0.48	0.68	0.10
Acenaphthylene	0.02	0.02	0.02	0.02	0.03	0.05	0.02	0.00	0.06	0.19	0.06	0.02
Acenaphthene	0.14	0.12	0.09	0.16	0.11	0.08	0.05	0.01	0.08	0.09	0.11	0.07
Fluorene	0.39	0.44	0.54	0.71	0.63	0.35	0.14	0.34	0.35	0.59	0.68	0.38
Phenanthrene	3.64	5.85	5.41	9.47	6.32	5.10	3.04	14.24	4.76	14.67	7.09	2.83
Anthracene	0.01	0.14	0.03	0.08	0.08	0.12	0.04	0.58	0.20	0.48	0.25	0.07
Fluoranthene	1.37	2.19	2.40	2.37	3.16	2.00	1.92	2.81	2.54	7.14	2.60	1.87
Pyrene	0.67	0.99	0.91	1.01	1.39	1.06	1.01	1.90	1.68	4.36	1.62	1.02
Benz(a)anthracene	0.05	0.05	0.04	0.07	0.08	0.13	0.06	0.24	0.23	0.99	0.21	0.08
Chrysene	0.11	0.13	0.12	0.14	0.30	0.19	0.14	0.34	0.41	1.66	0.47	0.17
Benzo(b)fluoranthene	0.17	0.17	0.14	0.18	0.23	0.26	0.18	0.68	0.63	1.55	0.32	0.22
Benzo(k)fluoranthene	0.05	0.04	0.03	0.04	0.03	0.08	0.02	0.12	0.03	0.38	0.13	0.04
<b>Benzo(a)pyrene</b>	<b>0.05</b>	<b>0.05</b>	<b>0.04</b>	<b>0.06</b>	<b>0.06</b>	<b>0.09</b>	<b>0.02</b>	<b>0.19</b>	<b>0.15</b>	<b>0.52</b>	<b>0.16</b>	<b>0.08</b>
Indeno(1,2,3-cd)pyrene	0.09	0.08	0.07	0.09	0.11	0.13	0.08	0.30	0.27	0.95	0.29	0.09
Dibenz(a,h)anthracene	0.01	0.01	0.01	0.01	0.02	0.02	0.01	0.03	0.04	0.11	0.03	0.01
Benzo(g,h,i)perylene	0.09	0.11	0.07	0.12	0.14	0.15	0.09	0.32	0.31	0.79	0.41	0.11
Total PAH (ng m3)	7.24	10.62	10.51	14.70	12.80	10.26	7.19	22.11	11.92	34.94	15.11	7.14



**Table 2 - The London Borough of Brent - Monthly Concentrations of PAH / ng m<sup>-3</sup>**

	Apr-05	May-05	Jun-05	Jul-05	Aug-05	Sep-05	Oct-05	Nov-05	Dec-05	Jan-06	Feb-06	Mar-06
Naphthalene	0.51	0.61	<20	0.56	0.56	0.27	0.44	0.67	0.46	0.95	0.69	0.59
Acenaphthylene	0.09	0.26	0.03	0.16	0.24	0.11	0.05	0.37	0.15	0.28	0.28	0.17
Acenaphthene	0.16	0.46	0.13	0.58	0.40	0.08	0.08	0.16	0.08	0.23	0.32	0.20
Fluorene	0.44	1.93	1.02	2.28	1.53	0.63	0.32	0.87	0.54	0.88	1.49	0.67
Phenanthrene	10.21	17.79	26.22	26.64	15.75	10.17	6.55	12.99	8.04	28.83	14.55	6.32
Anthracene	0.22	0.92	0.73	0.95	0.39	0.47	0.13	0.91	0.62	1.90	0.84	0.25
Fluoranthene	4.01	3.86	4.37	3.93	5.09	2.74	2.77	4.72	4.29	7.30	2.77	4.34
Pyrene	2.95	2.80	2.91	2.92	3.24	2.54	1.89	3.94	3.75	5.47	2.29	3.28
Benz(a)anthracene	0.11	0.12	0.11	0.12	0.10	0.23	0.09	0.38	0.43	1.06	0.27	0.08
Chrysene	0.18	0.22	0.23	0.20	0.33	0.25	0.15	0.67	0.62	1.39	0.47	0.17
Benzo(b)fluoranthene	0.32	0.26	0.32	0.32	0.24	0.36	0.22	0.94	0.97	3.39	0.35	0.23
Benzo(k)fluoranthene	0.09	0.08	0.09	0.08	0.03	0.05	0.04	0.22	0.23	1.13	0.13	0.04
<b>Benzo(a)pyrene</b>	<b>0.06</b>	<b>0.08</b>	<b>0.09</b>	<b>0.11</b>	<b>0.07</b>	<b>0.15</b>	<b>0.04</b>	<b>0.35</b>	<b>0.19</b>	<b>1.57</b>	<b>0.20</b>	<b>0.05</b>
Indeno(1,2,3-cd)pyrene	0.15	0.17	0.15	0.20	0.14	0.20	0.13	0.47	0.54	2.63	0.33	0.07
Dibenz(a,h)anthracene	0.01	0.03	0.02	0.02	0.02	0.02	0.01	0.04	0.05	0.22	0.03	0.01
Benzo(g,h,i)perylene	0.23	0.27	0.15	0.28	0.19	0.26	0.16	0.63	0.59	2.23	0.55	0.15
Total PAH (ng m3)	19.74	29.87	36.58	39.35	28.30	18.53	13.05	28.31	21.53	59.46	25.56	16.61

**Table 3 - The London Borough of Greenwich - Monthly Concentrations of PAH / ng m<sup>-3</sup>**

	Apr-05	May-05	Jun-05	Jul-05	Aug-05	Sep-05	Oct-05	Nov-05	Dec-05	Jan-06	Feb-06	Mar-06
Naphthalene	0.29	1.02	0.36	0.48	0.54	0.51	0.51	0.73	0.51	0.68	0.51	0.37
Acenaphthylene	0.04	0.03	0.03	0.07	0.21	0.13	0.02	0.18	0.22	0.20	0.10	0.03
Acenaphthene	0.09	0.07	0.05	0.10	3.73	0.13	0.13	0.10	0.11	0.17	0.09	0.10
Fluorene	0.33	0.37	0.23	0.76	8.15	0.55	0.36	0.51	0.58	0.94	0.73	0.44
Phenanthrene	4.30	5.10	5.08	13.98	12.22	7.80	6.16	6.55	7.15	22.96	11.25	4.08
Anthracene	0.14	0.33	0.09	0.41	0.54	0.34	0.10	0.36	0.51	1.57	0.62	0.16
Fluoranthene	2.03	3.18	3.99	2.86	3.73	2.54	2.61	3.60	3.10	8.93	3.99	2.72
Pyrene	1.53	2.08	2.36	1.97	2.07	1.76	1.38	2.84	2.42	6.38	3.45	1.73
Benz(a)anthracene	0.09	0.09	0.11	0.10	0.10	0.14	0.06	0.27	0.27	1.28	0.35	0.08
Chrysene	0.16	0.20	0.19	0.18	0.29	0.18	0.13	0.44	0.48	2.21	0.65	0.15
Benzo(b)fluoranthene	0.31	0.25	0.21	0.24	0.22	0.29	0.20	0.73	0.71	1.83	0.51	0.20
Benzo(k)fluoranthene	0.07	0.07	0.05	0.04	0.03	0.07	0.03	0.11	0.03	0.55	0.21	0.04
<b>Benzo(a)pyrene</b>	<b>0.08</b>	<b>0.07</b>	<b>0.06</b>	<b>0.08</b>	<b>0.05</b>	<b>0.11</b>	<b>0.02</b>	<b>0.16</b>	<b>0.19</b>	<b>0.02</b>	<b>0.24</b>	<b>0.06</b>
Indeno(1,2,3-cd)pyrene	0.11	0.11	0.11	0.13	0.10	0.13	0.08	0.33	0.33	1.45	0.40	0.08
Dibenz(a,h)anthracene	0.02	0.03	0.02	0.01	0.01	0.02	0.00	0.04	0.04	0.11	0.05	0.01
Benzo(g,h,i)perylene	0.14	0.17	0.12	0.19	0.16	0.17	0.11	0.36	0.37	1.11	0.65	0.14
Total PAH (ng m <sup>3</sup> )	9.71	13.17	13.05	21.60	32.16	15.02	11.89	17.32	17.02	50.38	23.80	10.41

**Table 4 - The London Borough of Hammersmith & Fulham**  
**- Monthly Concentrations of PAH / ng m<sup>-3</sup>**

	Apr-05	May-05	Jun-05	Jul-05	Aug-05	Sep-05	Oct-05	Nov-05	Dec-05	Jan-06	Feb-06	Mar-06
<b>Naphthalene</b>	0.13	0.71	0.58	0.70	1.30	0.60	1.92	1.75	0.35	0.51	0.51	1.02
<b>Acenaphthylene</b>	0.03	0.13	0.26	0.22	0.46	0.38	0.25	0.90	0.10	0.23	0.12	0.07
<b>Acenaphthene</b>	0.04	0.37	0.12	0.51	0.28	0.24	0.12	0.31	0.05	0.15	0.13	0.13
<b>Fluorene</b>	0.20	1.58	0.84	2.09	1.62	1.39	0.65	1.25	0.38	0.80	0.73	0.39
<b>Phenanthrene</b>	5.09	13.26	11.66	20.93	12.99	16.69	10.88	14.80	6.70	22.62	9.82	6.69
<b>Anthracene</b>	0.12	1.07	0.44	0.82	0.56	0.65	0.73	1.28	0.56	1.28	0.58	0.43
<b>Fluoranthene</b>	3.52	4.54	4.37	4.12	5.57	3.80	4.35	4.67	4.02	6.93	3.17	5.12
<b>Pyrene</b>	3.02	4.08	3.46	3.42	4.45	2.78	3.99	4.28	3.48	5.11	2.80	4.33
<b>Benz(a)anthracene</b>	0.10	0.16	0.10	0.13	0.17	0.25	0.25	0.62	0.46	0.91	0.29	0.20
<b>Chrysene</b>	0.20	0.26	0.21	0.23	0.46	0.32	0.34	0.78	0.67	1.24	0.55	0.37
<b>Benzo(b)fluoranthene</b>	0.27	0.31	0.26	0.30	0.35	0.46	0.51	1.21	0.91	1.20	0.36	0.71
<b>Benzo(k)fluoranthene</b>	0.06	0.09	0.06	0.06	0.07	0.14	0.08	0.30	0.13	0.29	0.16	0.12
<b>Benzo(a)pyrene</b>	<b>0.08</b>	<b>0.11</b>	<b>0.07</b>	<b>0.10</b>	<b>0.11</b>	<b>0.18</b>	<b>0.15</b>	<b>0.36</b>	<b>0.22</b>	<b>0.44</b>	<b>0.17</b>	<b>0.14</b>
<b>Indeno(1,2,3-cd)pyrene</b>	0.15	0.17	0.13	0.16	0.17	0.32	0.23	0.62	0.43	0.77	0.33	0.25
<b>Dibenz(a,h)anthracene</b>	0.01	0.02	0.01	0.02	0.02	0.02	0.01	0.05	0.05	0.08	0.04	0.02
<b>Benzo(g,h,i)perylene</b>	0.20	0.32	0.20	0.29	0.31	0.35	0.44	0.78	0.54	0.73	0.62	0.39
<b>Total PAH (ng m3)</b>	13.21	27.16	22.79	34.11	28.88	28.58	24.89	33.96	19.06	43.30	20.39	20.37

**Table 5 - The London Borough of Richmond - Monthly Concentrations of PAH / ng m<sup>-3</sup>**

	Apr-05	May-05	Jun-05	Jul-05	Aug-05	Sep-05	Oct-05	Nov-05	Dec-05	Jan-06	Feb-06	Mar-06
Naphthalene	0.28	0.39	0.31	1.40	0.41	1.17	0.69	0.77	0.44	0.93	0.69	0.32
Acenaphthylene	0.04	0.04	0.02	0.08	0.12	0.27	0.03	0.36	0.12	0.17	0.23	0.08
Acenaphthene	0.14	0.12	0.06	0.22	0.25	0.12	0.04	0.16	0.08	0.11	0.13	0.13
Fluorene	0.47	0.51	0.25	1.08	0.75	0.47	0.12	0.66	0.41	0.80	0.80	0.51
Phenanthrene	4.30	5.89	5.78	10.78	6.11	6.25	3.59	6.93	6.45	20.34	9.83	3.76
Anthracene	0.04	0.23	0.08	0.21	0.11	0.21	0.10	0.47	0.44	1.10	0.44	0.16
Fluoranthene	1.56	2.59	2.89	3.11	3.19	2.19	1.74	4.01	3.36	6.78	3.06	2.39
Pyrene	1.02	1.73	1.50	1.59	1.66	1.45	1.20	2.92	2.82	5.08	2.48	1.57
Benz(a)anthracene	0.05	0.06	0.04	0.07	0.06	0.14	0.07	0.40	0.34	1.02	0.29	0.10
Chrysene	0.11	0.13	0.12	0.15	0.26	0.18	0.14	0.51	0.58	1.48	0.58	0.21
Benzo(b)fluoranthene	0.17	0.17	0.15	0.17	0.17	0.27	0.18	0.98	0.82	1.36	0.40	0.25
Benzo(k)fluoranthene	0.04	0.05	0.04	0.04	0.02	0.07	0.02	0.22	0.05	0.30	0.16	0.05
<b>Benzo(a)pyrene</b>	<b>0.05</b>	<b>0.05</b>	<b>0.04</b>	<b>0.06</b>	<b>0.04</b>	<b>0.11</b>	<b>0.03</b>	<b>0.26</b>	<b>0.21</b>	<b>0.47</b>	<b>0.24</b>	<b>0.09</b>
Indeno(1,2,3-cd)pyrene	0.09	0.09	0.07	0.10	0.10	0.15	0.11	0.55	0.37	0.93	0.36	0.09
Dibenz(a,h)anthracene	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.05	0.05	0.09	0.04	0.01
Benzo(g,h,i)perylene	0.11	0.15	0.08	0.15	0.14	0.18	0.14	0.51	0.48	0.80	0.58	0.17
Total PAH (ng m3)	8.47	12.20	11.43	19.21	13.39	13.24	8.19	19.76	17.01	41.76	20.32	9.89

**Table 6 - The London Borough of Hackney - Monthly Concentrations of PAH / ng m<sup>-3</sup>**

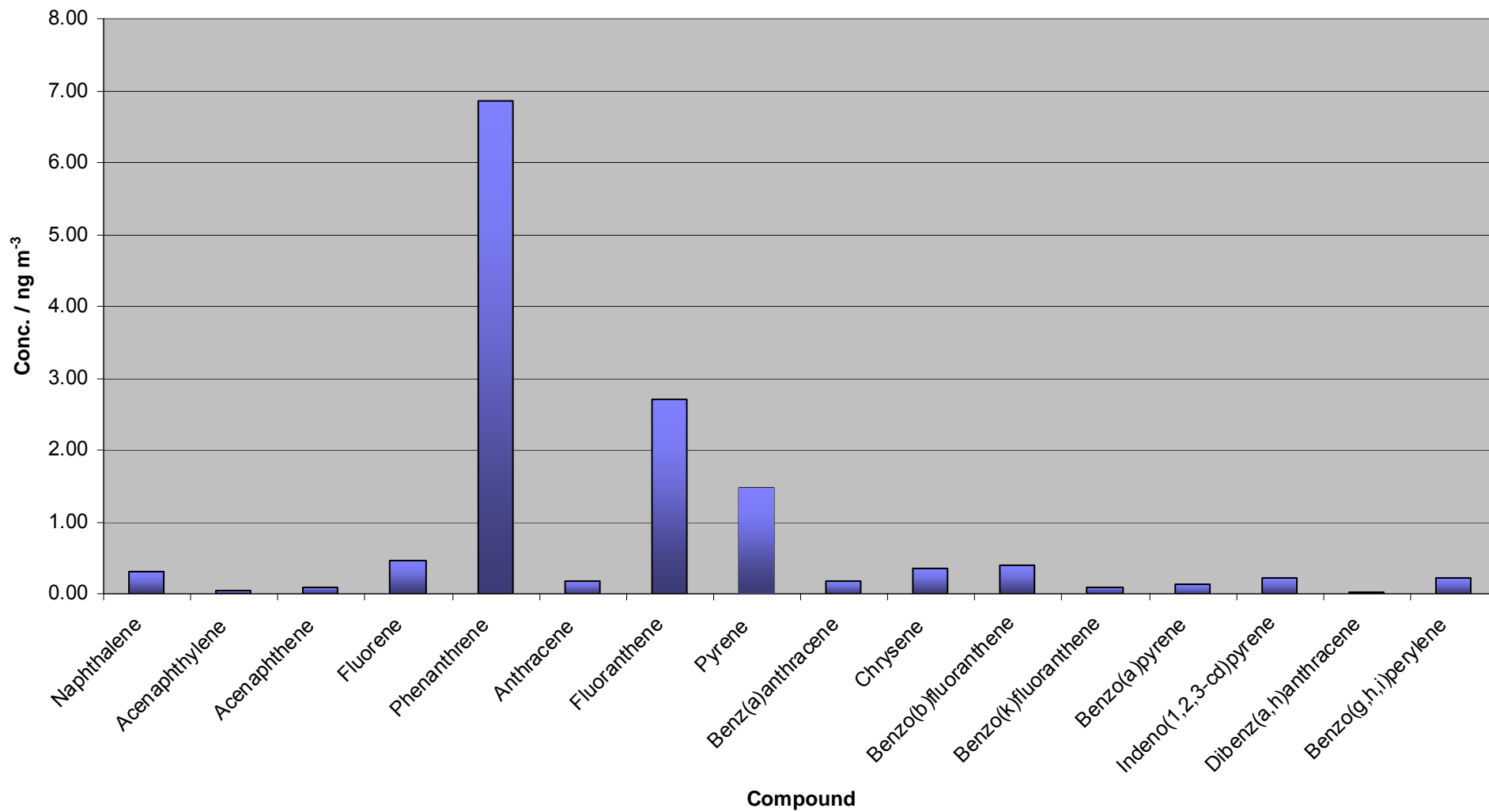
	Apr-05	May-05	Jun-05	Jul-05	Aug-05	Sep-05	Oct-05	Nov-05	Dec-05	Jan-06	Feb-06	Mar-06
Naphthalene	0.51	0.45	0.31	0.44	0.56	0.51	0.47	<20	0.64	0.55	0.58	0.29
Acenaphthylene	0.05	0.12	0.09	0.20	0.17	0.17	0.11	0.13	0.16	0.20	0.16	0.07
Acenaphthene	0.08	0.23	0.22	0.31	0.26	0.15	0.09	0.09	0.09	0.11	0.12	0.09
Fluorene	0.28	1.53	0.91	1.65	1.02	0.67	0.36	0.75	0.51	0.80	0.80	0.39
Phenanthrene	6.21	13.23	6.92	14.59	8.79	8.64	6.90	16.10	8.12	20.76	13.10	6.68
Anthracene	0.14	0.81	0.15	0.34	0.27	0.33	0.22	1.18	0.57	1.31	0.76	0.26
Fluoranthene	4.02	5.09	4.74	5.20	5.55	3.50	3.38	4.71	4.06	7.28	4.00	4.32
Pyrene	3.14	4.17	3.13	3.87	3.80	2.71	2.76	3.93	3.72	5.83	4.00	3.93
Benz(a)anthracene	0.08	0.14	0.07	0.12	0.11	0.21	0.13	0.37	0.31	0.98	0.33	0.10
Chrysene	0.18	0.25	0.15	0.20	0.38	0.25	0.20	0.51	0.51	1.57	0.58	0.20
Benzo(b)fluoranthene	0.25	0.31	0.17	0.27	0.25	0.33	0.26	0.82	0.78	1.27	0.44	0.27
Benzo(k)fluoranthene	0.05	0.09	0.04	0.05	0.03	0.09	0.05	0.14	0.10	0.35	0.16	0.06
Benzo(a)pyrene	<b>0.06</b>	<b>0.09</b>	<b>0.04</b>	<b>0.10</b>	<b>0.06</b>	<b>0.13</b>	<b>0.07</b>	<b>0.24</b>	<b>0.14</b>	<b>0.44</b>	<b>0.19</b>	<b>0.06</b>
Indeno(1,2,3-cd)pyrene	0.11	0.14	0.08	0.13	0.11	0.17	0.15	0.35	0.34	0.73	0.35	0.11
Dibenz(a,h)anthracene	0.01	0.02	0.01	0.02	0.01	0.01	0.01	0.04	0.04	0.07	0.04	0.01
Benzo(g,h,i)perylene	0.19	0.27	0.13	0.23	0.21	0.27	0.23	0.51	0.47	0.84	0.91	0.30
Total PAH (ng m3)	15.37	26.95	17.16	27.71	21.59	18.14	15.38	29.88	20.56	43.08	26.54	17.15

**Table 7 - Annual Mean of each PAH analysed at each site - ng m<sup>-3</sup>**

	Bexley	Brent	Greenwich	H & Fulham	Richmond	Hackney	All Boroughs
Naphthalene	0.31	0.53	0.59	0.84	0.65	0.44	0.56
Acenaphthylene	0.04	0.18	0.10	0.26	0.13	0.14	0.14
Acenaphthene	0.09	0.24	0.38	0.20	0.13	0.15	0.20
Fluorene	0.46	1.05	1.10	0.99	0.57	0.81	0.83
Phenanthrene	6.87	15.34	8.60	12.68	7.50	10.84	10.30
Anthracene	0.17	0.69	0.42	0.71	0.30	0.53	0.47
Fluoranthene	2.70	4.18	3.57	4.52	3.07	4.65	3.78
Pyrene	1.47	3.17	2.46	3.77	2.08	3.75	2.78
Benz(a)anthracene	0.19	0.26	0.23	0.30	0.22	0.25	0.24
Chrysene	0.35	0.41	0.42	0.47	0.37	0.42	0.40
Benzo(b)fluoranthene	0.39	0.66	0.46	0.57	0.42	0.45	0.49
Benzo(k)fluoranthene	0.08	0.18	0.11	0.13	0.09	0.10	0.12
<b>Benzo(a)pyrene</b>	<b>0.12</b>	<b>0.25</b>	<b>0.09</b>	<b>0.18</b>	<b>0.14</b>	<b>0.13</b>	<b>0.15</b>
Indeno(1,2,3-cd)pyrene	0.21	0.43	0.27	0.31	0.25	0.23	0.28
Dibenz(a,h)anthracene	0.03	0.04	0.03	0.03	0.03	0.02	0.03
Benzo(g,h,i)perylene	0.23	0.47	0.30	0.43	0.29	0.38	0.35
Total PAH (ng m <sup>3</sup> )	13.71	28.07	19.13	26.39	16.24	23.29	21.14

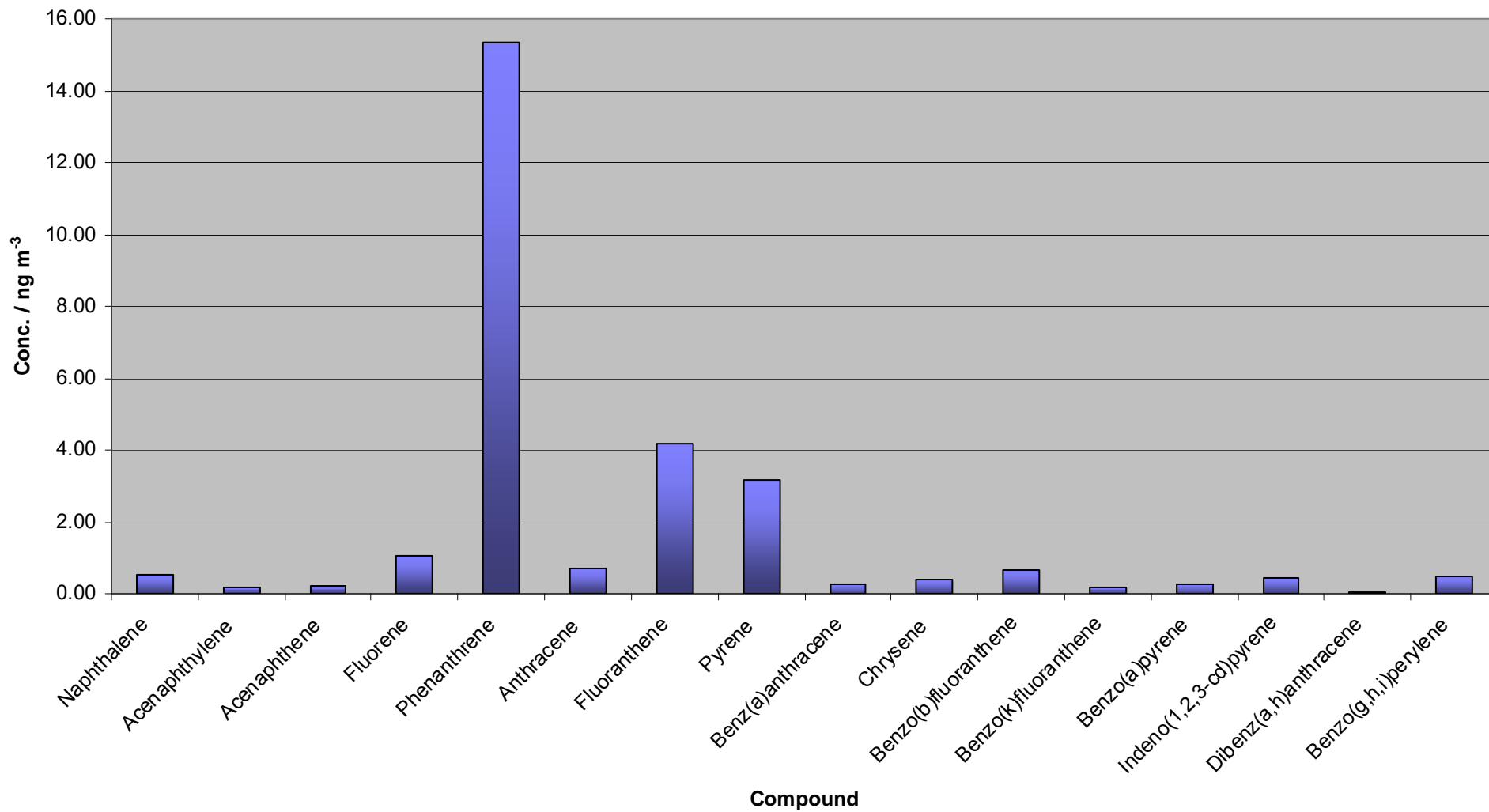
**Appendix C**  
**Figures 1a – 1g**

**Figure 1a. Annual Mean PAH Concentrations - London Borough of Bexley**

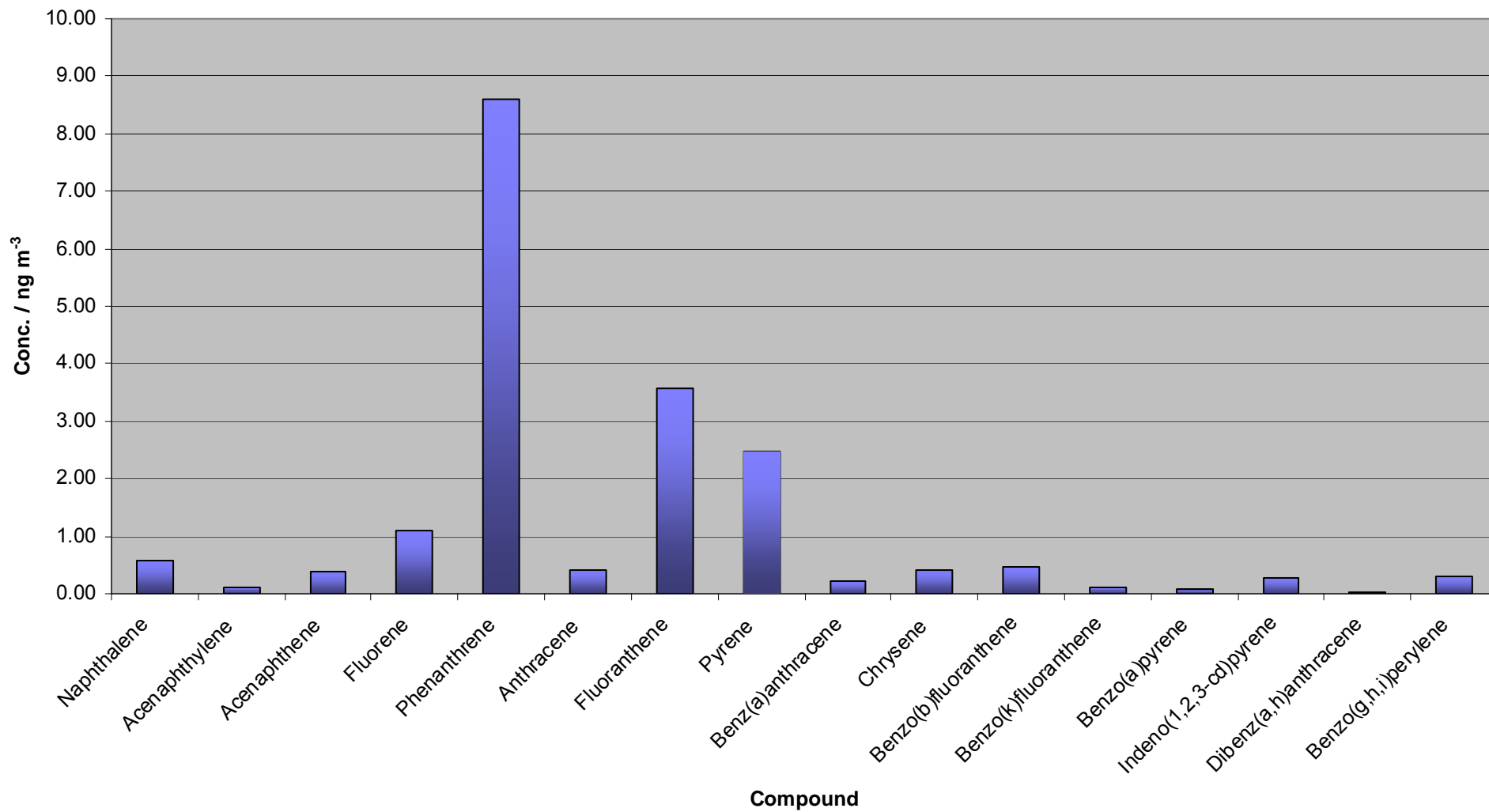




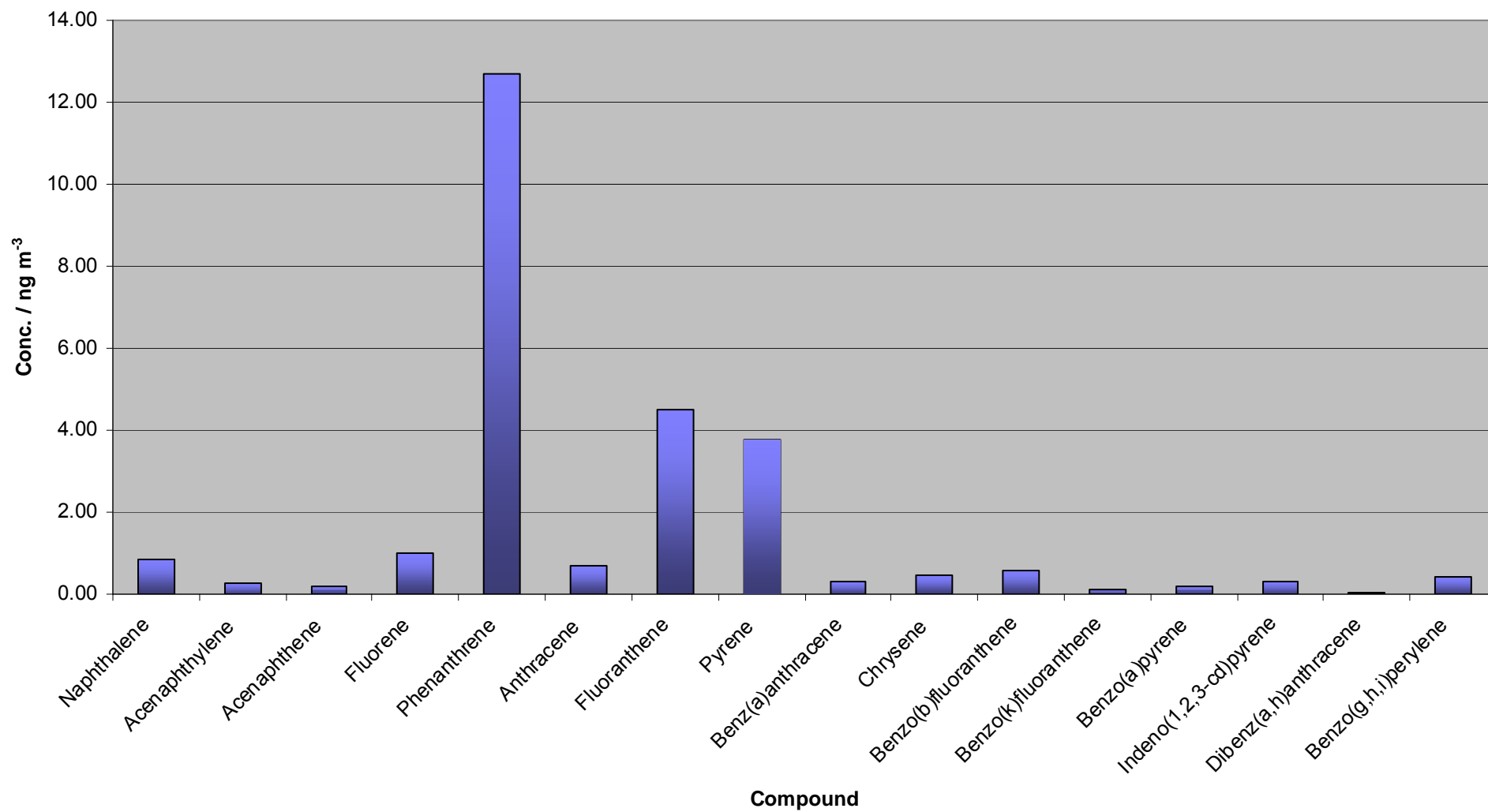
**Figure 1b. Annual Mean PAH Concentrations - London Borough of Brent**



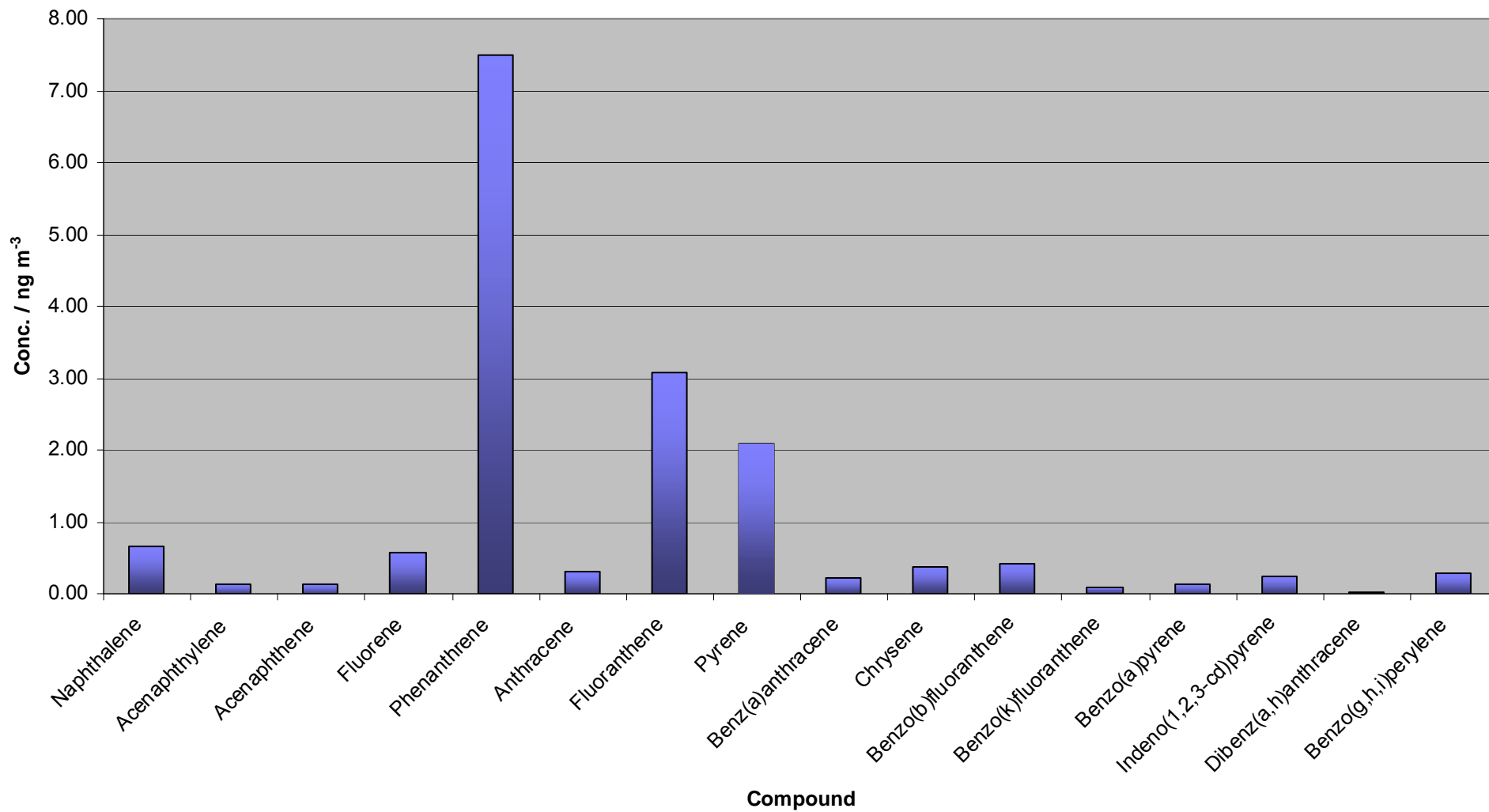
**Figure 1c. Annual Mean PAH Concentrations - London Borough of Greenwich**



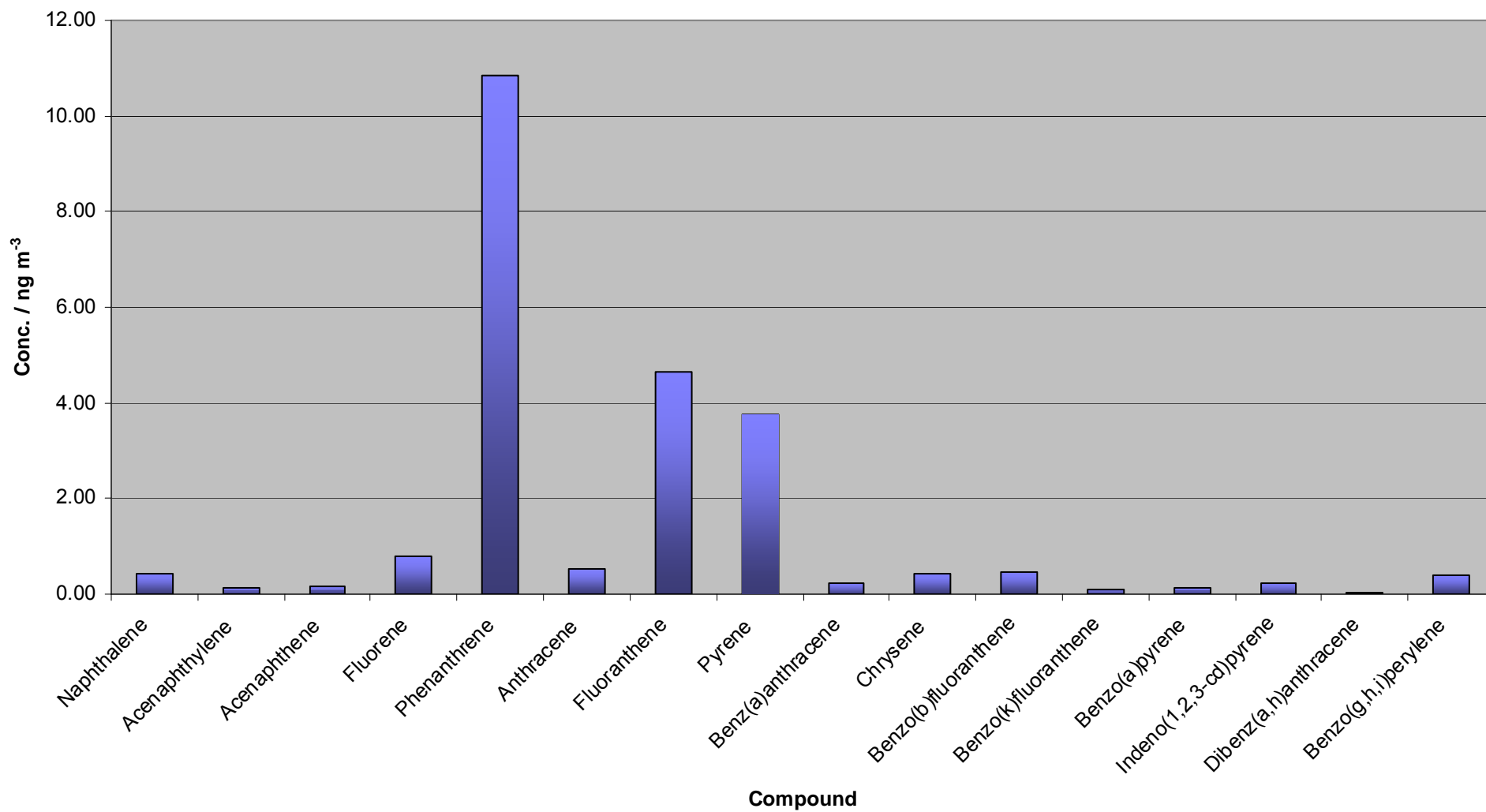
**Figure 1d. Annual Mean PAH Concentrations - London Borough of Hammersmith and Fulham**



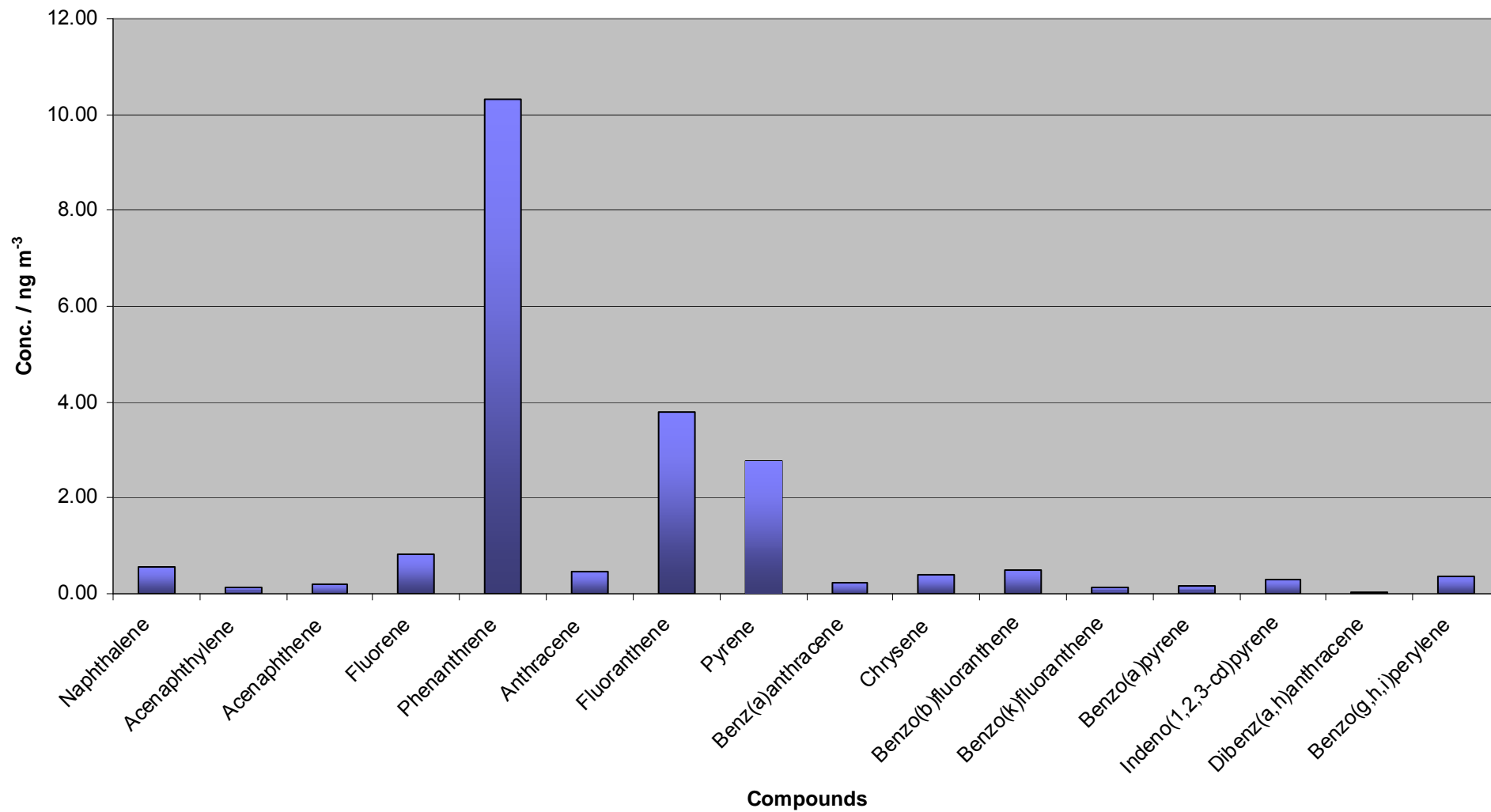
**Figure 1e. Annual Mean PAH Concentrations - London Borough of Richmond**



**Figure 1f. Annual Mean PAH Concentrations - London Borough of Hackney**

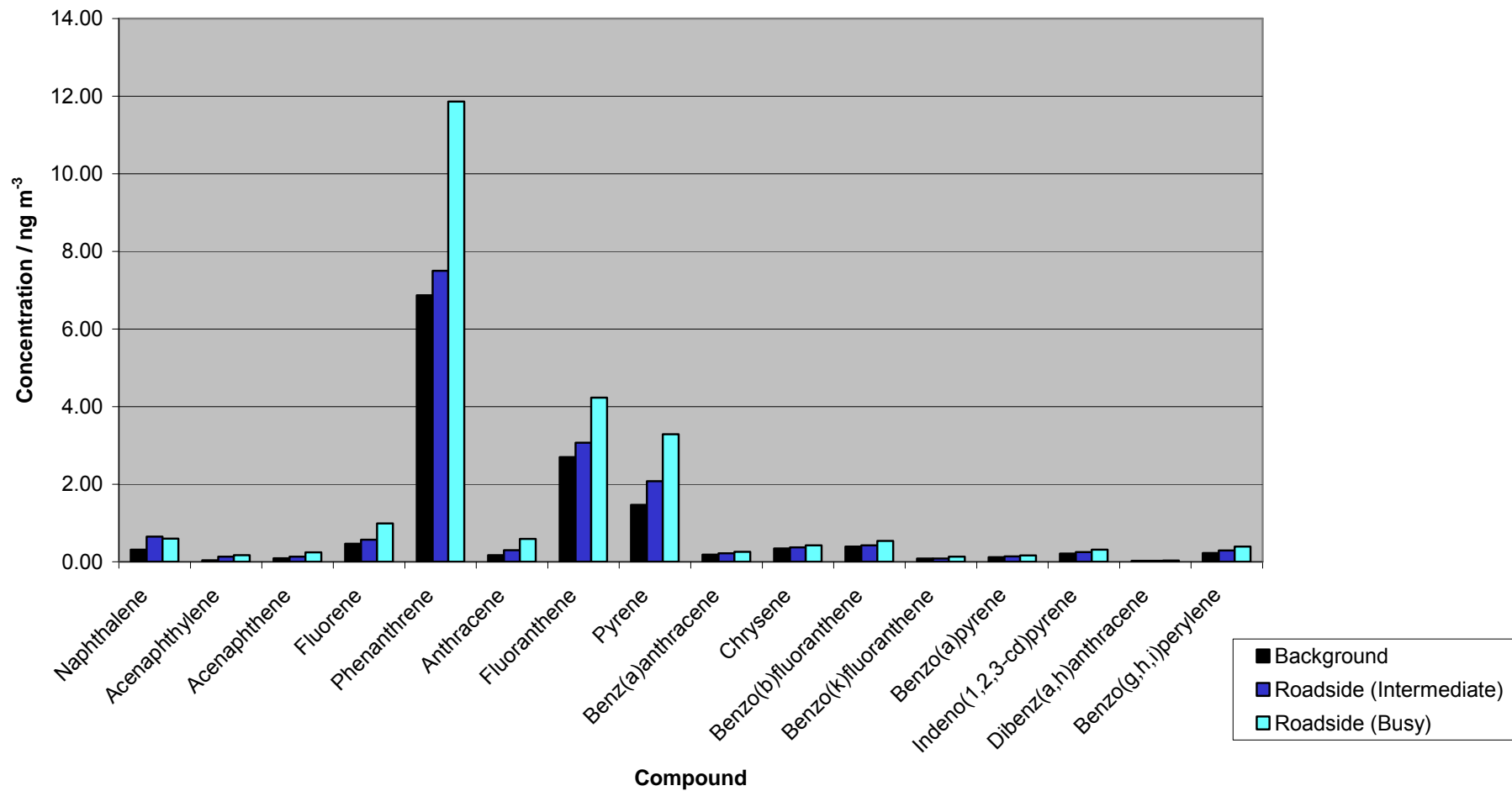


**Figure 1g. Annual Mean PAH Concentrations - All London Boroughs**



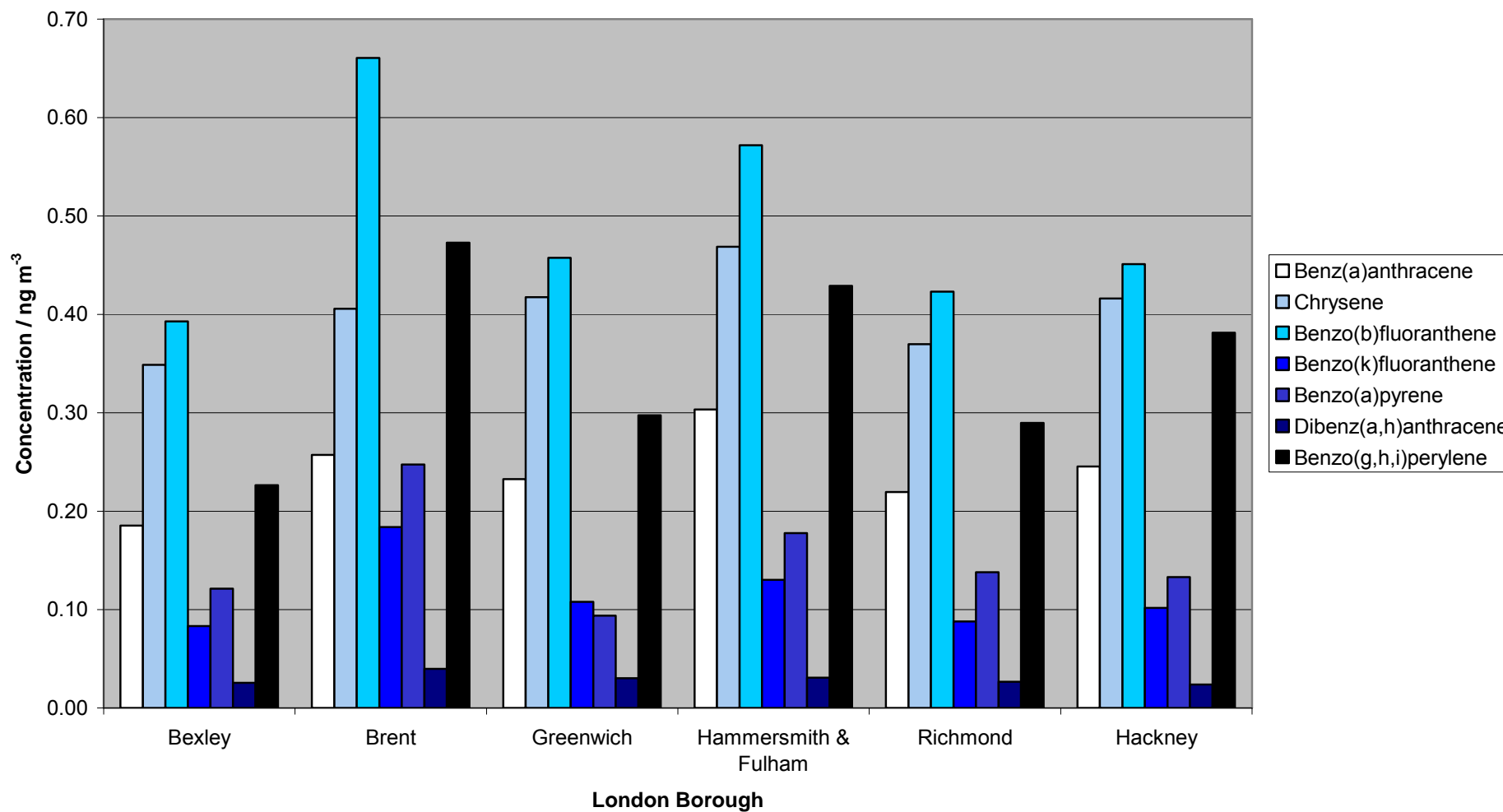
**Appendix D**  
**Figures 2 – 5**

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

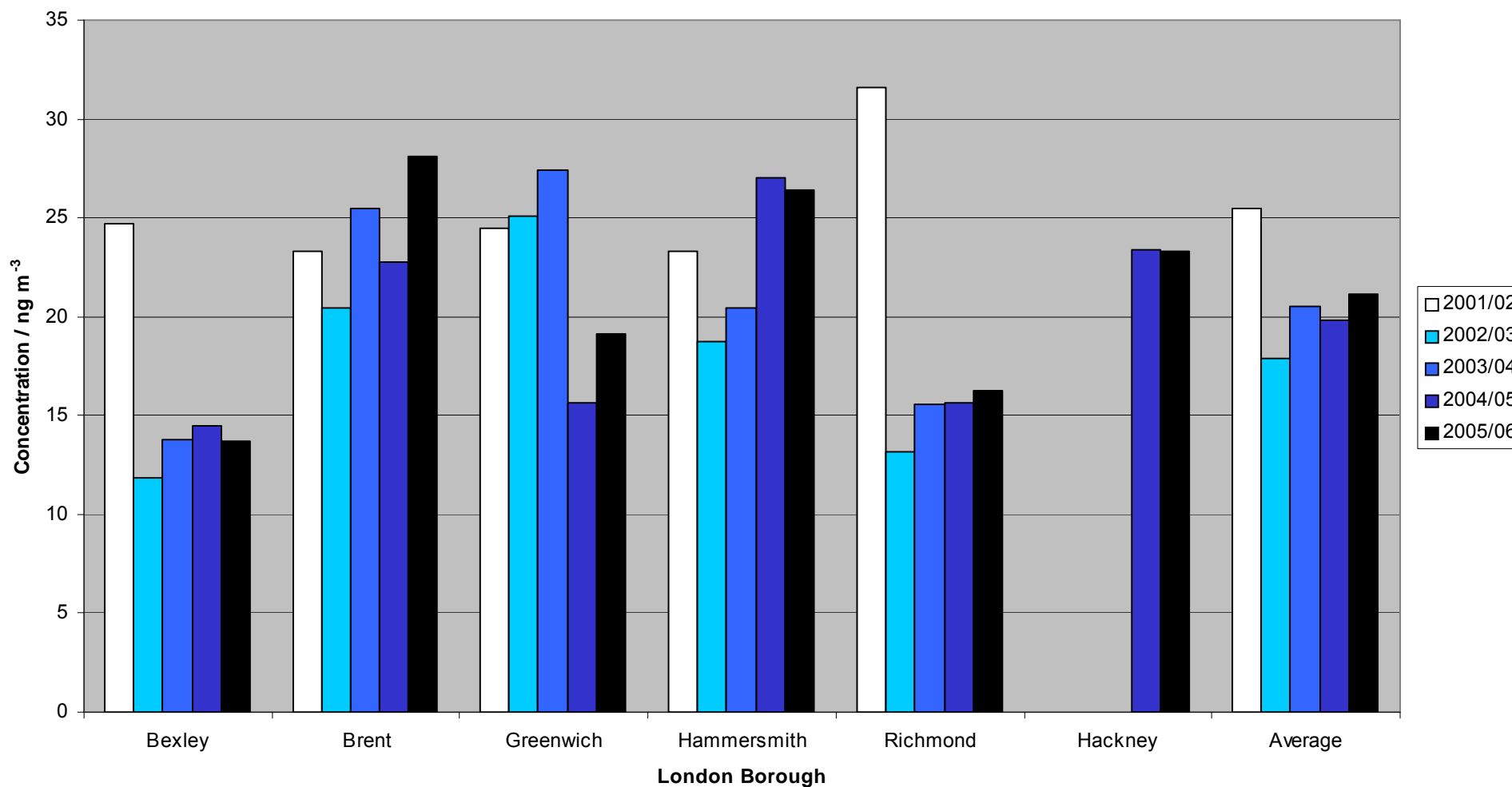




**Figure 3. Annual Mean Concentration of the Most Carcinogenic Compounds at each Site**



**Figure 4. Annual Mean Total PAH Concentration over the past Five Surveys at each Site**



**Figure 5. Monthly Variation in B[a]P Concentration**

