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Measurement and Modeling of Exposure to Selected Air Toxics for Health Effects Studies and Verification by Biomarkers

Roy M. Harrison, Juana Maria Delgado-Saborit,
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with a Critique by the HEI Health Review Committee

Research Report 143

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ABOUT HEI

The Health Effects Institute is a nonprofit corporation chartered in 1980 as an independent research organization to provide high-quality, impartial, and relevant science on the effects of air pollution on health. To accomplish its mission, the institute

- Identifies the highest-priority areas for health effects research;
- Competitively funds and oversees research projects;
- Provides intensive independent review of HEI-supported studies and related research;
- Integrates HEI's research results with those of other institutions into broader evaluations; and
- Communicates the results of HEI research and analyses to public and private decision makers.

HEI receives half of its core funds from the U.S. Environmental Protection Agency and half from the worldwide motor vehicle industry. Frequently, other public and private organizations in the United States and around the world also support major projects or certain research programs. HEI has funded more than 280 research projects in North America, Europe, Asia, and Latin America, the results of which have informed decisions regarding carbon monoxide, air toxics, nitrogen oxides, diesel exhaust, ozone, particulate matter, and other pollutants. These results have appeared in the peer-reviewed literature and in more than 200 comprehensive reports published by HEI.

HEI's independent Board of Directors consists of leaders in science and policy who are committed to fostering the public-private partnership that is central to the organization. The Health Research Committee solicits input from HEI sponsors and other stakeholders and works with scientific staff to develop a Five-Year Strategic Plan, select research projects for funding, and oversee their conduct. The Health Review Committee, which has no role in selecting or overseeing studies, works with staff to evaluate and interpret the results of funded studies and related research.

All project results and accompanying comments by the Health Review Committee are widely disseminated through HEI's Web site (www.healtheffects.org), printed reports, newsletters, and other publications, annual conferences, and presentations to legislative bodies and public agencies.

ABOUT THIS REPORT

Research Report 143, *Measurement and Modeling of Exposure to Selected Air Toxics for Health Effects Studies and Verification by Biomarkers*, presents a research project funded by the Health Effects Institute and conducted by Dr. Roy M. Harrison of the Division of Environmental Health and Risk Management, University of Birmingham, Birmingham, United Kingdom, and his colleagues. This report contains three main sections.

The HEI Statement, prepared by staff at HEI, is a brief, nontechnical summary of the study and its findings; it also briefly describes the Health Review Committee's comments on the study.

The Investigators' Report, prepared by Harrison et al., describes the scientific background, aims, methods, results, and conclusions of the study.

The Critique is prepared by members of the Health Review Committee with the assistance of HEI staff; it places the study in a broader scientific context, points out its strengths and limitations, and discusses remaining uncertainties and implications of the study's findings for public health and future research.

This report has gone through HEI's rigorous review process. When an HEI-funded study is completed, the investigators submit a draft final report presenting the background and results of the study. This draft report is first examined by outside technical reviewers and a biostatistician. The report and the reviewers' comments are then evaluated by members of the Health Review Committee, an independent panel of distinguished scientists who have no involvement in selecting or overseeing HEI studies. During the review process, the investigators have an opportunity to exchange comments with the Review Committee and, as necessary, to revise their report. The Critique reflects the information provided in the final version of the report.

PREFACE

HEI's Research Program on Air Toxics Hot Spots

Air toxics comprise a large and diverse group of air pollutants that, with sufficient exposure, are known or suspected to cause adverse effects on human health, including illness and death. These compounds are emitted by a variety of indoor and outdoor sources. Even though the ambient levels of air toxics are generally low, the compounds are a cause for public health concern because large numbers of people are exposed to them over long periods. Tools and techniques for assessing specific health effects of air toxics are very limited, in part because of the generally low ambient levels of the compounds.

Air toxics are not regulated by the U.S. Environmental Protection Agency (EPA) under the National Ambient Air Quality Standards, but the EPA is required under the Clean Air Act and its amendments to characterize, prioritize, and address the effects of air toxics on public health and the environment, and EPA has the statutory authority to control and reduce the release of air toxics in the environment. The law also requires the EPA to regulate or consider regulating air toxics from motor vehicles in the form of standards for fuels, vehicle emissions, or both. HEI has recently published a critical review of the literature on exposure and health effects associated with high-priority air toxics from mobile sources (HEI Special Report 16, 2007).

In trying to understand the potential health effects of exposure to toxic compounds, scientists often turn first to evaluating responses in highly exposed populations, such as occupationally exposed workers. However, workers and their on-the-job exposures are not representative of the general population, and therefore such studies may be somewhat limited in value. Another strategy is to study populations living in areas thought to have high concentrations of these pollutants (so-called hot spots). Such areas offer the potential to conduct health investigations in groups that are more similar to the general population.

Hot spots are generally specific areas that are expected to have elevated levels of one or more air

toxics owing to their proximity to one or more sources. Some hot spots may have sufficiently high pollutant concentrations that they may be studied to determine whether there is a link between exposure to air toxics and an adverse health outcome. Before health effects studies can be initiated, however, actual exposures to pollutants in such hot-spot areas must first be characterized — including their spatial and temporal distributions. Understanding exposures in hot spots, as well as the sources of these exposures, will improve our ability to select the most appropriate sites, populations, and end points for subsequent health studies.

In January 2003, HEI issued a Request for Applications (RFA 03-1) entitled "Assessing Exposure to Air Toxics." The main goal of the RFA was to support research to identify and characterize exposure to air toxics from a variety of sources in areas or situations where concentrations of air toxics are elevated. HEI was particularly interested in studies that focused on air toxics emitted from mobile sources. Five studies, chosen to represent a diversity of sites and toxic compounds, were funded under this RFA. The study by Harrison and colleagues — presented in this Research Report — is the first of the five studies to be published. The remaining studies have been completed and are currently at varying stages of the HEI review and publications process. All are expected to be released within the next year.

**"Measurement and Modeling of Exposure to Air Toxics and Verification by Biomarker,"
Roy M. Harrison, University of Birmingham,
Birmingham, United Kingdom (Principal Investigator)**

In the study presented in this report (HEI Research Report 143), Roy M. Harrison and colleagues investigated personal exposure to a broad range of air toxics, with the goal of developing detailed personal exposure models that would take various microenvironments

Preface

into account. Repeated measurements of exposure to selected air toxics were made for each of 100 healthy nonsmoking adults who resided in urban, suburban, or rural areas of the United Kingdom, among which exposures to traffic were expected to differ; repeated urine samples were also collected for analysis. Harrison and colleagues developed models to predict personal exposure on the basis of microenvironmental concentrations and data from time–activity diaries; they then compared measured personal exposure with modeled estimates of exposure.

“Assessing Exposure to Air Toxics,” Eric Fujita, Desert Research Institute, Reno, Nevada (Principal Investigator)

The study by Fujita and colleagues assessed air toxics concentrations on major California freeways and compared them with corresponding measurements obtained at fixed monitoring stations. The diurnal and seasonal variations in concentrations of selected pollutants and the contribution of diesel- and gasoline-powered vehicles to selected air toxics and elemental carbon were also determined.

“Assessing Personal Exposure to Air Toxics in Camden, New Jersey,” Paul Lioy, Environmental and Occupational Health Sciences Institute, Piscataway, New Jersey (Principal Investigator)

Lioy and colleagues measured personal and ambient residential concentrations of air toxics and fine particulate matter in two areas of Camden, New Jersey. One site was a potential hot spot with mobile sources and a high density of industrial facilities, and the other neighborhood was considered an urban reference site. Simultaneous measurements were made of air toxics in personal air samples for 107 nonsmoking participants and in air samples from fixed monitoring sites in the two areas. The degree of variation in the ambient concentrations of air toxics was assessed during three sampling periods. In addition to the measurements of actual ambient and personal exposures, the investigators

used modeling to estimate the contribution of ambient sources to personal exposure.

“Air Toxics Exposure from Vehicular Emissions at a U.S. Border Crossing,” John Spengler, Harvard School of Public Health, Boston, Massachusetts (Principal Investigator)

The study by Spengler and colleagues assessed concentrations of mobile-source air toxics surrounding the Peace Bridge Plaza, a major border crossing between the United States and Canada, located in Buffalo, New York. Three fixed monitoring sites were used to compare concentrations upwind and downwind of the plaza. Meteorologic measurements and hourly counts of trucks and cars were used to examine the relationship between the concentrations of air toxics and traffic density. To study spatial patterns, staff members walked along four established routes in a residential neighborhood in West Buffalo while making measurements with mobile instruments and global positioning system (GPS) devices.

“Air Toxics Hot Spots in Industrial Parks and Traffic,” Thomas Smith, Harvard School of Public Health, Boston, Massachusetts (Principal Investigator)

The study by Smith and colleagues was added to an ongoing study, funded by the National Cancer Institute, of the relationship between exposure to diesel exhaust and mortality from lung cancer among dockworkers and truck drivers at more than 200 truck terminals in the United States. With support from HEI, Smith and colleagues measured levels of air toxics and particulate matter in truck cabins and in 15 truck terminals throughout the United States. Twelve-hour measurements were made at upwind and downwind locations around the perimeter of each terminal and at loading docks. The degree of variation among terminals at various locations and the influence of wind direction were evaluated with the goal of identifying the potential impact of truck terminals on the surrounding areas. Continuous sampling was performed inside GPS-equipped truck cabins while they were being driven.

HEI STATEMENT

Synopsis of Research Report 143

Measurement and Modeling of Exposure to Selected Air Toxics

BACKGROUND

Air toxics are a diverse group of air pollutants that are known or suspected, with sufficient exposure, to cause adverse health effects including cancer, damage to the immune, neurologic, reproductive, developmental, or respiratory systems, or other health problems. Limited monitoring has been performed by some state and local agencies, but substantial uncertainty regarding exposure to air toxics remains, largely because of their presence in the ambient environment at low concentrations. Although environmental exposures to air toxics are generally low, the potential for widespread chronic exposure and the large number of people who are exposed have led to concerns regarding their impact on public health. Estimation of the health risks of exposure to air toxics is complicated by the fact that there are multiple sources of air toxics. These may be outdoor and indoor (e.g., environmental tobacco smoke, building materials, consumer products, and cooking).

APPROACH

Dr. Roy Harrison investigated personal exposures to a broad group of air toxics, with the goal of developing detailed personal exposure models that take various microenvironments into account. In order to provide important information on personal exposures to air toxics, the study was designed to capture adequate variation in exposure concentrations. Repeated measurements of exposure to selected air toxics were made for each of 100 healthy adult non-smoking participants residing in urban, suburban, and rural areas of the United Kingdom expected to have different traffic exposures. Measurements included five repeated 24-hour measurements of personal exposure to volatile organic compounds

(VOCs; including 1,3-butadiene) per participant; five urine samples collected to test for urinary biomarkers (polycyclic aromatic hydrocarbon [PAH] metabolites, cotinine, and *trans*-3'-hydroxycotinine) per participant; and one 24-hour measurement of particle-phase PAHs per participant; plus concurrent measurement of microenvironmental exposures at participants' homes and workplaces—a total of 200 VOC, 190 1,3-butadiene, and 168 PAH samples, as well as measurements in other major microenvironments.

Dr. Harrison developed models to predict personal exposures on the basis of microenvironmental concentrations and data from time-activity diaries, and compared measured personal exposures with modeled estimates of exposure. The goal was to use these data to produce a scheme for categorizing exposure (by compound) according to the location of residence and other lifestyle and exposure factors, including environmental tobacco smoke, for use in the design of health studies of cancer incidence.

RESULTS AND IMPLICATIONS

This study serves as a rich source of recent information on personal exposures to selected air toxics across a range of residential locations and exposures to non-traffic sources, with attention to spatial variation and areas in which air toxics exposures were likely to be elevated. However, most participants in the study were young adults, thus limiting the study's generalizability to other age groups. Also, owing to challenges in recruitment, the study sample was not balanced.

Personal exposures were most heavily influenced by the home microenvironment and were higher in the presence of fossil fuel combustion, environmental tobacco smoke, solvent use, use of selected

This Statement, prepared by the Health Effects Institute, summarizes a research project funded by HEI and conducted by Dr. Roy M. Harrison at University of Birmingham, Division of Environmental Health and Risk Management, Birmingham, U.K., and colleagues. Research Report 143 contains both the detailed Investigators' Report and a Critique of the study prepared by the Institute's Health Review Committee.

consumer products, and commuting. After the home microenvironment, the workplace and commuting were the largest contributors to personal exposure. The reported concentrations of selected air toxics and levels of personal exposure were somewhat lower than those observed in other studies.

Harrison and colleagues used an innovative approach to modeling to predict personal exposures on the basis of microenvironmental concentration data and time–activity diaries, with the idea that models could inform the design of future health studies. The most predictive statistical models did only a fair-to-moderate job of predicting personal exposures, however. Statistical models based on

microenvironmental factors and lifestyle were able to explain a fair amount of the variance in personal exposures for selected VOCs but were less predictive of PAH exposures. While part of the inability to effectively model exposures may be due to the lack of measured characteristics of home ventilation, particularly air exchange rates in the home, this study underscores the challenges of accurately predicting personal exposures. Personal exposure monitoring requires extensive time and equipment, but the science is not yet at a point at which exposures to VOCs and PAHs can be reliably predicted from time–activity patterns and microenvironmental concentrations alone.

Measurement and Modeling of Exposure to Selected Air Toxics for Health Effects Studies and Verification by Biomarkers

Roy M. Harrison, Juana Maria Delgado-Saborit, Stephen J. Baker, Noel Aquilina, Claire Meddings, Stuart Harrad, Ian Matthews, Sotiris Vardoulakis, and H. Ross Anderson

Division of Environmental Health and Risk Management, University of Birmingham, Birmingham (R.M.H., J.M.D.-S., S.J.B., N.A., C.M., S.H.); Department of Epidemiology, Statistics, and Public Health, Cardiff University, Cardiff (I.M.); Public and Environmental Health Research Unit, London School of Hygiene and Tropical Medicine (S.V.); Department of Community Health Sciences, St. George's Hospital Medical School, London (H.R.A.)—all in the United Kingdom.

ABSTRACT

The overall aim of our investigation was to quantify the magnitude and range of individual personal exposures to a variety of air toxics and to develop models for exposure prediction on the basis of time–activity diaries. The specific research goals were (1) to use personal monitoring of non-smokers at a range of residential locations and exposures to non-traffic sources to assess daily exposures to a range of air toxics, especially volatile organic compounds (VOCs*) including 1,3-butadiene and particulate polycyclic aromatic hydrocarbons (PAHs); (2) to determine microenvironmental concentrations of the same air toxics, taking account of spatial and temporal variations and hot spots; (3) to optimize a model of personal exposure using microenvironmental concentration data and time–activity diaries and to compare modeled exposures with exposures independently estimated from personal monitoring data; (4) to determine the relationships of urinary biomarkers with the environmental exposures to the corresponding air toxic.

This Investigators' Report is one part of Health Effects Institute Research Report 143, which also includes a Critique by the Health Review Committee and an HEI Statement about the research project. Correspondence concerning the Investigators' Report may be addressed to Dr. Roy Harrison, University of Birmingham, Division of Environmental Health and Risk Management, Edgbaston Park Road, School of Geography, Earth, and Environmental Sciences, Birmingham B15 2TT, United Kingdom, r.m.harrison@bham.ac.uk.

Although this document was produced with partial funding by the United States Environmental Protection Agency under Assistance Award CR-83234701 to the Health Effects Institute, it has not been subjected to the Agency's peer and administrative review and therefore may not necessarily reflect the views of the Agency, and no official endorsement by it should be inferred. The contents of this document also have not been reviewed by private party institutions, including those that support the Health Effects Institute; therefore, it may not reflect the views or policies of these parties, and no endorsement by them should be inferred.

*A list of abbreviations and other terms appears at the end of the Investigators' Report.

Personal exposure measurements were made using an actively pumped personal sampler enclosed in a briefcase. Five 24-hour integrated personal samples were collected from 100 volunteers with a range of exposure patterns for analysis of VOCs and 1,3-butadiene concentrations of ambient air. One 24-hour integrated PAH personal exposure sample was collected by each subject concurrently with 24 hours of the personal sampling for VOCs. During the period when personal exposures were being measured, workplace and home concentrations of the same air toxics were being measured simultaneously, as were seasonal levels in other microenvironments that the subjects visit during their daily activities, including street microenvironments, transport microenvironments, indoor environments, and other home environments. Information about subjects' lifestyles and daily activities were recorded by means of questionnaires and activity diaries.

VOCs were collected in tubes packed with the adsorbent resins Tenax GR and Carbotrap, and separate tubes for the collection of 1,3-butadiene were packed with Carbopack B and Carbosieve S-III. After sampling, the tubes were analyzed by means of a thermal desorber interfaced with a gas chromatograph–mass spectrometer (GC–MS). Particle-phase PAHs collected onto a quartz-fiber filter were extracted with solvent, purified, and concentrated before being analyzed with a GC–MS. Urinary biomarkers were analyzed by liquid chromatography–tandem mass spectrometry (LC–MS–MS).

Both the environmental concentrations and personal exposure concentrations measured in this study are lower than those in the majority of earlier published work, which is consistent with the reported application of abatement measures to the control of air toxics emissions. The environmental concentration data clearly demonstrate the influence of traffic sources and meteorologic conditions leading to higher air toxics concentrations in the winter and during

peak-traffic hours. The seasonal effect was also observed in indoor environments, where indoor sources add to the effects of the previously identified outdoor sources.

The variability of personal exposure concentrations of VOCs and PAHs mainly reflects the range of activities the subjects engaged in during the five-day period of sampling. A number of generic factors have been identified to influence personal exposure concentrations to VOCs, such as the presence of an integral garage (attached to the home), exposure to environmental tobacco smoke (ETS), use of solvents, and commuting. In the case of the medium- and high-molecular-weight PAHs, traffic and ETS are important contributions to personal exposure. Personal exposure concentrations generally exceed home indoor concentrations, which in turn exceed outdoor concentrations. The home microenvironment is the dominant individual contributor to personal exposure. However, for those subjects with particularly high personal exposures, activities within the home and exposure to ETS play a major role in determining exposure.

Correlation analysis and principal components analysis (PCA) have been performed to identify groups of compounds that share common sources, common chemistry, or common transport or meteorologic patterns. We used these methods to identify four main factors determining the makeup of personal exposures: fossil fuel combustion, use of solvents, ETS exposure, and use of consumer products.

Concurrent with sampling of the selected air toxics, a total of 500 urine samples were collected, one for each of the 100 subjects on the day after each of the five days on which the briefcases were carried for personal exposure data collection. From the 500 samples, 100 were selected to be analyzed for PAHs and ETS-related urinary biomarkers. Results showed that urinary biomarkers of ETS exposure correlated strongly with the gas-phase markers of ETS and 1,3-butadiene. The urinary ETS biomarkers also correlated strongly with high-molecular-weight PAHs in the personal exposure samples.

Five different approaches have been taken to model personal exposure to VOCs and PAHs, using 75% of the measured personal exposure data set to develop the models and 25% as an independent check on the model performance. The best personal exposure model, based on measured microenvironmental concentrations and lifestyle factors, is able to account for about 50% of the variance in measured personal exposure to benzene and a higher proportion of the variance for some other compounds (e.g., 75% of the variance in 3-ethenylpyridine exposure). In the case of the PAHs, the best model for benzo[*a*]pyrene is able to account for about 35% of the variance among exposures, with a similar result for the rest of the PAH compounds.

The models developed were validated by the independent data set for almost all the VOC compounds. The models developed for PAHs explain some of the variance in the independent data set and are good indicators of the sources affecting PAH concentrations but could not be validated statistically, with the exception of the model for pyrene.

A proposal for categorizing personal exposures as low or high is also presented, according to exposure thresholds. For both VOCs and PAHs, low exposures are correctly classified for the concentrations predicted by the proposed models, but higher exposures were less successfully classified.

INTRODUCTION

The term “air toxics” embraces a range of gaseous or particulate pollutants that are present in the air in low concentrations and have characteristics such as toxicity or persistence such that they are a hazard to human, plant, or animal life. VOCs and PAHs are among the several categories of compounds considered to be air toxics.

VOCs and PAHs are emitted into ambient air from a wide range of sources. Major anthropogenic sources of VOCs in ambient air include industrial processes, fossil fuel combustion for purposes of transportation, domestic heating and electricity generation, fuel distribution, solvent use, and landfills and waste treatment plants. With regard to indoor air, primary sources of VOCs include outdoor air, ETS, fuel combustion, building materials, furnishings, furniture and carpet adhesives, paints and solvents, cleaning agents, air fresheners, and cosmetics (Jurvelin 2003). PAHs are produced by high-temperature reactions such as incomplete combustion and pyrolysis of fossil fuels and other organic materials (Harrison et al. 1996). Major anthropogenic sources of ambient-air PAHs include heating (with coal, oil, or wood), refuse burning, coke production, industrial processes, and operation of motor vehicles (Benner et al. 1989). In indoor environments, PAHs are generated from cooking, smoking, and the burning of natural gas, wood, candles, or incense, as well as being transported from the outdoors (Chuang et al. 1991; Naumova et al. 2002).

Air toxics are ubiquitous in outdoor and indoor air and are therefore of public health concern. There is evidence that cancer, birth defects, genetic damage (International Agency for Research on Cancer 1982), immunodeficiency (U.S. Environmental Protection Agency [EPA] 2007), respiratory disorders, (Andersson et al. 1997) and nervous system disorders (EPA 2007) can be linked to exposure to occupational levels of air toxics. The International Agency

for Research on Cancer (2006) classifies 1,3-butadiene, benzene, and benzo[*a*]pyrene as known human carcinogens; dibenz[*a,h*]anthracene as probably carcinogenic to humans; and ethylbenzene, styrene, naphthalene, benzo[*a*]anthracene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, and indeno[1,2,3-*cd*]pyrene as possibly carcinogenic to humans. Therefore, although air toxics may be associated with a number of adverse health outcomes, the most important, especially in the public mind, is likely to be cancer. Currently, however, evidence for carcinogenicity derives primarily from epidemiologic studies of occupationally exposed individuals (Armstrong et al. 2004), or even from laboratory studies of animal models. In the case of pollutants such as benzene and the PAHs, for which the evidence for carcinogenicity from occupational exposures is very strong, the exposures greatly exceed typical environmental concentrations to which the general public is exposed, and therefore evaluation of carcinogenic risk by means other than extrapolation is very difficult.

There is growing international recognition of the potential health risks associated with exposure to air toxics and the need for action to minimize these risks. Recently, the contribution of indoor air to the exposure of an individual to pollutants has been increasingly recognized as being of importance (Samet and Spengler 2003; Adgate et al. 2004a,b; Phillips et al. 2005). The determination of an individual's exposure to air pollution depends on the individual and his or her activity patterns, which are reflected in the time spent in various microenvironments (Harrison et al. 2002). Numerous international studies have reported that people spend 80–93% of their time indoors, 1–7% in transit in an enclosed vehicle, and 2–7% outdoors (Jenkins et al. 1992; Hinwood et al. 2003; Brunekreef et al. 2005; Koutrakis et al. 2005). It has been estimated that of the time spent indoors, approximately 60–70% is spent in the home (Thatcher and Layton 1995; Hinwood et al. 2003), which suggests that significant exposures may occur in the home. In addition, other indoor microenvironments, and particularly the workplace, are important determinants of overall personal exposure to air toxics (Harrison et al. 2002).

Despite the research community recognizing the importance of indoor environments in personal exposure, policy makers have focused their attention on outdoor air quality. Environmental laws, standards, and other regulations have traditionally focused on the release of pollution into the environment, and on outdoor concentrations, rather than on the extent of human exposure caused by the release. For this reason, the amounts of environmental pollutants to which general populations are actually exposed are rarely quantified.

Non-occupational air pollution regulations have typically been applied to outdoor rather than indoor air, which

means that toxic pollutants emitted from indoor sources have been ignored (Ott and Roberts 1998; Jurvelin 2003). In addition, monitoring for compliance with ambient air quality standards is limited to a relatively small number of stationary outdoor monitoring sites. Given the likely spatial variation in air toxics concentrations in ambient air, it is questionable to what extent such a monitoring strategy represents an accurate reflection of personal exposure (Kim et al. 2002). Furthermore, since people in developed countries spend most of their time indoors, it can be argued that fixed monitoring should not be used in risk assessment for personal exposures, as individual exposures are not well represented.

Current assessments of public exposure to atmospheric pollutants have found that the personal exposures of the urban population to many airborne pollutants are vastly different from, and often greater than, the outdoor air concentrations measured at fixed monitoring stations (Michael et al. 1990; Hartwell et al. 1992; Edwards et al. 2001a,b; Kim et al. 2002; Adgate et al. 2004a,b; Lai et al. 2004; Payne-Sturges et al. 2004; Phillips et al. 2005). Therefore, risk estimates based on ambient measurements may misestimate risks, leading to ineffective or inefficient management strategies (Payne-Sturges et al. 2004). Consequently, personal exposures cannot be determined directly from ambient background measurements from fixed monitoring stations. The alternative is personal exposure monitoring, which takes into account the mobility of people across various microenvironments, according to their daily activities (Carrer et al. 2000). Personal exposure monitoring can occur either via direct or indirect measurement. Direct measurement is undertaken by means of personal samplers, whereas indirect exposure is determined by time–activity diaries and microenvironmental measurements (Ott 1985).

To date, even though the study of personal exposure to air pollution is a rather well-developed science, it has been restricted to a relatively limited range of pollutants. Among the air toxics, benzene has been heavily studied (Wallace 1989b; Lofgren et al. 1991; Edwards and Jantunen 2001), and benzene, toluene, and the xylenes have been the focus for many studies of environmental concentrations. Edwards and colleagues (2001), as part of the Air Pollution Exposure Distributions of Adult Urban Populations in Europe (EXPOLIS)–Helsinki study, reported microenvironmental and personal exposure concentrations of 30 target VOCs, finding ETS to be an important factor in exposure. Studies have also been conducted in Germany (Hoffmann et al. 2000; Ilgen et al. 2001a), the United Kingdom (Kim et al. 2002), the United States (Wallace et al. 1985; Pellizzari et al. 1995; Turpin et al. 2007), Asia (Baek et al. 1997; Chang et al. 2005), and Australia (Hinwood et al. 2003). Studies assessing personal exposures to PAHs are

few, however (Georgiadis et al. 2001; Levy et al. 2001; Reff et al. 2005; Loh et al. 2007; Turpin et al. 2007).

In addition, for certain air toxics (e.g., 1,3-butadiene and styrene), the extent of data from outdoor measurements is quite limited, and those from indoor measurements generally even more limited, despite their carcinogenicity and higher unit risk factor. Furthermore, the available data for 1,3-butadiene are limited to specific microenvironments such as automobiles, buses, homes, or workplaces (Heavener et al. 1996; Duffy and Nelson 1997; Kim et al. 1999, 2001b; Klepeis et al. 2001). In previous studies, extensive measurement programs were carried out, determining concentrations of 15 VOCs in a wide range of urban microenvironments including homes, offices, restaurants, pubs, department stores, bus and train stations, cinemas, libraries, perfume shops, heavily trafficked roadside locations, buses, trains and automobiles (Kim et al. 2001a; Fedoruk and Kerger 2003; Lau and Chan 2003; Guo et al. 2004a). Yet the number of measurements of VOCs and especially PAHs is still insufficient to allow for exposure estimations representative of various microenvironments. Finally, most previous studies of personal exposure have been limited to 1–2 days of sampling per subject (Hartwell et al. 1987; Hartwell et al. 1992; Leung and Harrison 1998; Jurvelin et al. 2001), with some exceptions, in which diurnal variations in concentrations in individual exposures were studied for 5–10 days (Kim et al. 2002; Koutrakis et al. 2005). As a result, daily variations in exposure of individuals to VOCs have not been evaluated extensively. Thus, our study will considerably strengthen the database of VOC and PAH personal exposure and microenvironmental measurements by generating new data via direct measurements.

Personal exposure can also be estimated via indirect measurements. An earlier study (Leung and Harrison 1998) showed that personal exposures modeled from microenvironmental concentrations and personal-activity diaries can provide a good prediction of overall measured personal exposure. Microenvironmental modeling offers an effective means of estimating population exposures to pollutants without the considerable logistical difficulties of personal sampling. This approach would be improved by determining the range of variability in pollutant concentrations over space and time for key microenvironments, allowing a probabilistic approach to be taken in the use of these models (Harrison et al. 2002). Activity patterns have a significant influence on personal exposure. Therefore, a study of the typical means and ranges of activity patterns of susceptible groups would permit microenvironmental models to be included in probabilistic models (Harrison et al. 2002). The power of this approach can be strengthened by measuring exposures of more individuals and in microenvironments under various conditions.

Although previous studies have shed light on the distribution of concentrations seen in personal exposures and in various microenvironments, and much work has been conducted with the aim of modeling population exposures to air pollutants using information collected in time–activity diaries and using microenvironmental concentrations, very little has been done toward validating such models at the level of the individual. The present study aimed to determine the statistical confidence with which personal exposures can be reconstructed using measured microenvironmental concentrations, to demonstrate the confidence with which individual personal exposures can be categorized as low-risk or high-risk on the basis of limited lifestyle information such as that which can be collected in a questionnaire.

Finally, a major goal of environmental epidemiology is to establish quantitative relationships between exposures to air toxics and the associated risks of disease. Biological monitoring has been increasingly viewed as a desirable alternative to air sampling for characterizing environmental exposures, not only because it accounts for all possible exposure routes but also because it covers unexpected or accidental exposures and reflects inter-individual differences in uptake or genetic susceptibility (Lin et al. 2005). Urinary biomarkers have been widely used for assessing occupational exposure to air toxic concentrations because the dose–response relationship between air toxics in such exposures and biomarkers is of importance in setting threshold values (Jacob et al. 2007). Nevertheless, to date there are few studies in which urinary biomarkers have been used to assess non-occupational exposures (Buckley et al. 1995; Scherer et al. 1999; Scherer et al. 2000; Hu et al. 2006). In this study, we aimed to collect urine samples, analyze urinary metabolites, and relate them to corresponding air toxic exposures to establish the exposure–response relationship in non-occupational environmental exposures.

This study seeks to lead to advances in understanding the causes and magnitudes of exposures to relevant air toxic substances — 15 VOCs and 16 PAHs — and to establish whether collecting lifestyle information and/or urinary biomarker data is sufficient to model personal exposures reliably, as compared with collecting independent data on exposures by using personal samplers, for a range of air toxic substances.

SPECIFIC AIMS

This research is concerned primarily with the issue of quantifying personal exposures to air toxics according to the location of documented exposure and associated emissions from specific sources such as road traffic and ETS.

The aim is to provide a validated approach to the estimation of personal exposure to determine whether personal exposures can be predicted using data from a lifestyle questionnaire, allowing individuals to be differentiated into a range of personal exposure groupings that could be used in a subsequent epidemiological study of either a case-control or ecological (spatial analysis) design.

Since air toxic substances are not emitted uniformly from a single source, their concentrations may not correlate among various exposure environments; therefore, it would be desirable to measure microenvironmental concentrations of a wide range of air toxics to establish the inter-relationships among them while also determining the relationships between microenvironmental concentrations and personal exposures through direct measurements and modeling.

OVERALL AIM

To quantify the magnitude and range of individual personal exposures to a group of selected air toxics and to develop models for exposure prediction on the basis of time-activity diaries.

SPECIFIC GOALS

The specific goals of this research were as follows:

- To use personal monitoring of volunteer nonsmokers with a range of residential locations and exposures to non-traffic sources to assess daily exposures.
- To determine microenvironmental concentrations of a range of air toxics, taking spatial and temporal variations and hot spots into account.
- To study the trend in the relationship of environmental exposures to selected air toxics and urinary biomarker levels.
- To optimize a model of personal exposures based on microenvironmental concentration data and time-activity diaries and to compare modeled exposures with exposures independently estimated from personal monitoring data.
- To produce a method for categorizing exposures (on the basis of compound) according to location of residence and other lifestyle and exposure factors (e.g., ETS) for use in the design of case-control and ecological studies of cancer incidence.

The targeted air toxics were categorized into three groups that require different methods of sampling and analysis.

1. VOCs excluding 1,3-butadiene: Benzene, ethylbenzene, *n*-hexane, naphthalene, styrene, toluene, *o*-xylene,

m-xylene, *p*-xylene, 1,3,5-trimethylbenzene, 1,2,4-trimethylbenzene, *p*-isopropyltoluene, pyridine, and 3-ethenylpyridine. (Note that naphthalene was grouped with the VOCs for purposes of sampling and analysis, despite being the most abundant PAH in the gas phase.)

2. 1,3-Butadiene. (Because of its high volatility, 1,3-butadiene was considered separately from the rest of the VOCs for sampling and analysis, but it is grouped with the VOCs throughout this Investigators' Report.)
3. PAHs: Acenaphthylene, acenaphthene, fluorene, phenanthrene, anthracene, fluoranthene, pyrene, benzo[*a*]anthracene, chrysene, benzo[*b*]fluoranthene, benzo[*j*]fluoranthene, benzo[*k*]fluoranthene, benzo[*a*]pyrene, indeno[1,2,3-*cd*]pyrene, benzo[*g,h,i*]perylene, dibenz[*a,h*]anthracene, and coronene.

METHODS AND STUDY DESIGN

RECRUITMENT OF SUBJECTS

We recruited 100 healthy adult volunteers for measurement of personal exposure and indoor microenvironmental (home and office) concentrations. Selection was performed without regard to age, sex, or ethnic background. Potential subjects were excluded if they were smokers, under 18 years old, unhealthy (e.g., had chronic respiratory or coronary disease or cancer), unable to carry the personal sampler for any reason, or exposed to PAHs or VOCs at work or if they traveled more than 2 hours per day in the course of their work, their commute from work to home was more than 2 hours in duration, or the distance from home to the workplace was more than 20 miles.

Subjects resided in three different areas of the United Kingdom: London (11 volunteers), where some of the highest traffic exposures were anticipated; West Midlands (79 volunteers), where traffic exposures were expected to be intermediate; and South Wales (10 volunteers), which has a broad gradient between clean rural areas and more heavily contaminated urban environments.

After applying the exclusion criteria, we used four key determinants to further define our selection of subjects, including the number of subjects we sought to enroll in each category:

1. Location: Urban (40 subjects in London and Birmingham, the urban center of West Midlands), suburban (40 subjects in suburban Birmingham, West Midlands), or rural (20 subjects in rural West Midlands and South Wales).

2. Exposure to ETS: No (> 80 subjects), or yes (< 20 subjects).
3. Integral garage: No (70–80 subjects) or yes (20–30 subjects).
4. Proximity to major road: Within the first line of properties (50 subjects) or beyond the first line (50 subjects). Homes were considered as first-line properties if they were located on an A-class road (a road designed for large volumes of traffic and heavy vehicles) running through an urban or suburban area, on a road traveled by more than 20,000 vehicles per day, on a busy A road in rural area, or on a busy B-class road (a road designed for lower volumes of traffic including heavy vehicles) in an urban or suburban area.

A subject-selection matrix with 24 different subcategories of volunteers was developed to ensure enrollment that was representative of all possible combinations of the four

specified key determinants (Table 1). A modification of the initial proposal of subject distribution per subcategory was made because subcategories 5 to 8, which represented urban residents with integral garages, proved impossible to recruit into owing to the fact that in Britain there are very few residential buildings with integral garages in the downtown area of cities. To address this situation, the volunteers targeted in those categories were added to subcategories 1 to 4 (Table 1).

Different recruitment strategies were implemented to recruit volunteers in the different subcategories. These included a press release, advertisements in local papers, regional radio and TV interviews, leafleting, mass mailing, and posters and advertisements in various key institutions and environmental authority bodies to recruit the necessary number of participants in the three different regional areas.

These recruitment strategies led to responses from 762 individuals. Potential volunteers were informed in writing

Table 1. Subject Distribution Among Proposed Subcategories, According to Key Determinant Criteria and Proposal

Location, Subcategory I.D.	Key Determinant Criterion			Number of Volunteers (N = 100)		
	Integral Garage	ETS Exposure	First-Line Property	Initial Proposal	Modified Proposal	Recruited
Urban						38
1	No	No	Yes	10	13	12
2	No	No	No	10	13	14
3	No	Yes	Yes	5	7	4
4	No	Yes	No	5	7	8
5	Yes	No	Yes	3	0	0
6	Yes	No	No	3	0	0
7	Yes	Yes	Yes	2	0	0
8	Yes	Yes	No	2	0	0
Suburban						42
9	No	No	Yes	9	9	10
10	No	No	No	9	9	8
11	No	Yes	Yes	5	5	3
12	No	Yes	No	5	5	10
13	Yes	No	Yes	3	3	4
14	Yes	No	No	3	3	2
15	Yes	Yes	Yes	3	3	1
16	Yes	Yes	No	3	3	4
Rural						20
17	No	No	Yes	4	4	8
18	No	No	No	4	4	5
19	No	Yes	Yes	3	3	0
20	No	Yes	No	3	3	2
21	Yes	No	Yes	2	2	2
22	Yes	No	No	2	2	1
23	Yes	Yes	Yes	1	1	0
24	Yes	Yes	No	1	1	2

of the aims of the study and of the activities that were expected of them, none of which required any change in the normal pattern of activities; hence, they were exposed to no additional risks. Volunteers were asked to complete a screening questionnaire containing information about personal data, the key determinant criteria, and exclusion criteria in order to be classified into subcategories. They were also asked to give their consent to participate in the study, which was required for recruitment. The subject information sheets and screening questionnaires, as well as the consent forms, were approved by the Local Research Ethics Committee for South Birmingham. Among the original 762 respondents, 49 were unable to be contacted, 252 did not return their screening questionnaire and consent form, 51 met one or more of the exclusion criteria, and 14 expressed their wish to no longer participate in the study. Therefore, the final number of volunteers suitable for recruitment was 396. The numbers of subjects recruited with each method is summarized in Table 2.

Although overall we had a large number of people interested in participating in the project, our requirement of grouping them into 20 specific subcategories relating to the location of their home, proximity to a busy road, presence or absence of integral garage, and exposure to ETS meant that we had certain subcategories such as 12 and 17 (see Table 1) with large number of volunteers but other subcategories such as 15, 19, and 23 which had few or no volunteers.

The main concern for recruitment of participants was exposure to ETS. This criterion could not be specifically

evaluated during the recruitment process, unlike whether the home was a first-line property or had an integral garage. Although we had plenty of volunteers who met a given requirement, finding those who met some combinations of requirements (e.g., having an integral garage and a first-line home in a rural location and being exposed to ETS) was problematic. Because of such difficulties in recruitment, after exhausting all the recruitment methods, the final distribution of subjects per subcategory was slightly different than that initially proposed (Table 1). Only 16 recruited subjects had integral garages, when initially the proposal was to recruit 20–30 such subjects; a total of 34 subjects were exposed to ETS on at least one day, when initially the proposal was to recruit 30–40 subjects; and finally, 44 subjects lived on a busy road (resided in a first-line property), when the proposal was 50 subjects. Nevertheless, the overall balance among the various key factors was not compromised and the subjects recruited represent a wide range of residential locations with varying degrees of automotive pollution, ETS exposures, and concentrations of selected air toxics, which enabled the work to proceed.

SAMPLING METHODS

Sampling Devices

Adsorption tubes were used for collection of VOC compounds including 1,3-butadiene, whereas quartz-fiber filters were the sampling medium used to collect particle-phase PAHs. The sorbent tubes used for VOC and 1,3-butadiene sampling were stainless-steel tubes 17.8 cm (7 inches) in length, 0.6 cm (0.25 inches) in external diameter, and 0.49 cm (0.20 inches) in internal diameter. The tubes were packed with a dual-adsorbent bed separated and plugged with unsilanised glass wool, fitted with appropriate ferrules and Swagelok caps, and conditioned at the University of Birmingham. Different adsorbents were used for sampling the main group of VOC compounds and 1,3-butadiene (Table 3).

Table 2. Suitable Volunteers Recruited, According to Recruitment Method

Method of Recruitment	Number of Volunteers
Media (local press, radio, and TV)	229
Newspaper article 1/17/2006	17
Posters	2
Referrals	12
Leafleting in West Midlands	23
Leafleting in Wales	16
E-mail distribution in London (via environment authority bodies)	13
Leafleting in London	0
Mass mailing	62
MATCH Internet site	1
Active recruitment by Swansea city council	20
Active recruitment by Birmingham city council	1
Total	396

Table 3. Sorbent Tubes Used for VOC Sampling

VOC	Sorbent Material	Quantity of Sorbent Material (mg)
Any studied, except 1,3-butadiene	Tenax [®] GR (60/80 mesh)	300
	Carbotrap [™] (20/40 mesh)	600
1,3-Butadiene	Carbopack [™] B (60/80 mesh)	1000
	Carbosieve [™] SIII (60/80 mesh)	150

The adsorbent materials were purchased from Supelco (Dorset, United Kingdom). Tubes were conditioned with helium for one hour at 275°C for use in sampling VOCs and at 360°C for 1,3-butadiene, either before use or after the analysis of highly polluted samples. Conditioned sorbent tubes were stored, wrapped in aluminum foil, inside airtight metal tins at 4°C in a refrigerator.

All collected samples were kept under refrigeration after sampling and before analysis. Tubes containing samples were stored, wrapped in aluminum foil, inside metal tins in a refrigerator dedicated to either 1,3-butadiene or all other sampled VOCs.

The sampling material used to collect PAHs was filter media. For the first 33 subjects, glass-fiber filters were used (Whatman GF/A glass microfiber filter, 47 mm in diameter); sampling for the remaining subjects was performed with preconditioned quartz-fiber filters (Millipore AQFA reinforced quartz fiber filter, 47 mm in diameter). This change in filter media occurred after the method for extraction and analysis of PAHs from filters was developed and validated during the second year of the project. The method developed showed that the best filter on which to collect PAHs was the quartz-fiber filter, pre-baked for 48 hours at 400°C. Before sampling, the pre-treated filters were stored inside metal tins that were wrapped in aluminum foil and placed in an airtight glass jar. All filters that contained samples were individually stored inside metal tins wrapped in aluminum foil, in a separate freezer.

Personal Exposures

Personal exposure to selected air toxics was monitored by using actively pumped personal sampling devices to collect the following air toxics:

1. VOCs excluding 1,3-butadiene: Benzene, ethylbenzene, *n*-hexane, naphthalene, styrene, toluene, *o*-xylene, *m*-xylene, *p*-xylene, 1,3,5-trimethylbenzene, 1,2,4-trimethylbenzene, *p*-isopropyltoluene, pyridine, and 3-ethenylpyridine.
2. 1,3-Butadiene.
3. PAHs (particle phase only): Acenaphthylene, acenaphthene, fluorene, phenanthrene, anthracene, fluoranthene, pyrene, benzo[*a*]anthracene, chrysene, benzo[*b*]fluoranthene, benzo[*j*]fluoranthene, benzo[*k*]fluoranthene, benzo[*a*]pyrene, indeno[1,2,3-*cd*]pyrene, benzo[*g,h,i*]perylene, dibenz[*a,h*]anthracene, and coronene.

The design of the personal exposure sampler involved a direct-current personal sampler pump (SKC model 224-PCXR8) connected to the sorbent tubes and a second DC pump connected to the PAH filter. The setup was fitted in

small aluminum briefcases (Figure A1.1, Appendix 1; all appendices are available on the HEI Web site) and extra power was supplied via additional batteries contained within the briefcase. The personal exposure sampler monitored air toxics with the following design conditions:

1. *VOCs in the gas phase, excluding 1,3-butadiene*: The sampling method (Kim et al. 2001a) involved drawing air, at 40 mL min⁻¹, through an adsorbent tube.
2. *1,3-Butadiene in the gas phase*: 1,3-Butadiene was collected, at a flow rate of 30 mL min⁻¹, on an adsorbent tube (Kim et al. 1999).
3. *PAHs in the particulate phase*: Particle-phase PAHs were collected onto a quartz-fiber filter at a flow rate of 3 L min⁻¹ (Lim et al. 1999).

The setup of the sampling equipment was optimized to minimize noise and temperature levels, as well as weight. Samplers proved robust and fit for our purposes. The recruited subjects were generally happy to carry their personal samplers at all times. They carried the briefcases either from the handle, with the inlets at hip height, or with the use of a strap carried over the shoulder.

Subject-Related Microenvironments

Microenvironmental samplers (ME samplers) were designed and constructed (Figure A1.2, Appendix 1) to measure selected air toxics at each volunteer's home and workplace. The sampler was alternating current-powered, programmable, and was designed to collect two consecutive samples.

The ME samplers were designed to monitor air toxics under the following conditions:

1. *VOCs in the gas phase, excluding 1,3-butadiene*: VOCs were collected at 80 mL min⁻¹ for 12 hours in the home and at 120 mL min⁻¹ for 8 hours in the workplace.
2. *1,3-Butadiene in the gas phase*: 1,3-Butadiene was collected at 60 mL min⁻¹ for 12 hours in the home and at 90 mL min⁻¹ for 8 hours in the workplace.
3. *PAHs in the particulate phase*: Particle-phase PAHs were collected onto a quartz-fiber filter at a flow rate of 6 L min⁻¹ for 12 hours in the home and at a flow rate of 9 L min⁻¹ for 8 hours in the workplace.

Other Microenvironments

An "other microenvironmental" (OME) sampler was also designed and constructed, powered by direct current, to be used in microenvironments where no power supply was available for sampling during short time periods (2 hours or less).

The OME sampler (Figure A1.3, Appendix 1) was designed to monitor air toxics as follows:

1. *VOCs in the gas phase, excluding 1,3-butadiene*: VOCs were collected at 480 mL min⁻¹ for 2 hours.
2. *1,3-Butadiene in the gas phase*: 1,3-Butadiene was collected at 360 mL min⁻¹ for 2 hours.
3. *PAHs in the particulate phase*: Particle-phase PAHs were collected onto a quartz-fiber filter at a flow rate of 12 L min⁻¹ for 2 hours.

Urine Sampling

Urine samples were collected with the purpose of analyzing urinary biomarkers related to the selected air toxics under study. The first midstream urine sample in the morning was collected every day for each volunteer for analysis of the urinary biomarkers corresponding to the previous 24 hours of sampling. Urine samples were collected in 100-mL polypropylene cups and transferred to the laboratory in portable coolers containing ice packs. Once in the laboratory, 30 mL of the urine sample was transferred to smaller polypropylene vials, which were stored in the freezer at -20°C. After a short time, the frozen urine samples were stored in a -80°C freezer.

A total of 500 urine samples were collected, five for each of the 100 subjects. From among those samples, 100 were chosen for analysis. They had to have been either collected on the morning after days where subjects were exposed to tobacco smoke or after days when PAHs were sampled. About 15 mL of each urine sample was sent in dry ice to the Division of Clinical Pharmacology at the University of California, San Francisco. In the research facilities of the group led by Dr. Peyton Jacob III, urine samples were analyzed for the ETS metabolites cotinine and *trans*-3'-hydroxycotinine and the PAH metabolites 1-hydroxyfluorene, 2-hydroxyfluorene, 3-hydroxyfluorene, 1-hydroxyphenanthrene, 2-hydroxyphenanthrene, 3+4-hydroxyphenanthrene, and 1-hydroxypyrene.

SAMPLING PROGRAMS

Personal Exposures

With the personal exposure sampler, the exposure of each volunteer was sampled for VOCs (including 1,3-butadiene) for a total of five consecutive 24-hour periods. In addition, particle-phase PAHs were sampled during one 24-hour period for 95 of the 100 volunteers.

Collection of the samples was spread over two years, from May 2005 to May 2007. Subjects in London were sampled in spring and summer (May–June 2006), subjects in Wales were sampled in winter (October 2006–February

2007) and subjects in West Midlands were sampled throughout the four seasons (May 2005–May 2007).

Duplicate samples were taken for 3% of the study population, and in 3%, weekend samples were obtained. A total of 521 samples were collected for VOCs and 1,3-butadiene, and 99 samples were collected for particle-phase PAHs.

Subject-Related Microenvironments

During the period when personal exposure was measured, there was a simultaneous program of measurement of subject-related microenvironments such as in the workplace and the home. Detailed information on the sampling days, which were concurrent with those for personal sampling, and background pollutant levels is available in Appendix 2.

VOC, 1,3-butadiene, and PAH samples were collected in the subjects' homes (as two 12-hour samples, one during the day and the other at night). The preferred place to locate the ME sampler was the living room, where the subjects spent most of their active time at home. Samples of VOCs, 1,3-butadiene, and PAHs were also collected in some of the subjects' workplaces, concurrently with personal exposure samples for eight hours. As most of the volunteers were office workers, most of the workplaces were offices. Other workplaces that were sampled were a health center, a nursery/playschool, a laboratory and a garden center. The numbers of samples collected in the home and workplace are listed in Table 4.

Other Microenvironments

In addition to the sampling of subject-related microenvironments, we carried out a seasonal program of measurement of other microenvironments that the public visits in the course of their daily activities. These other microenvironmental measurements were independent from the subject's sampling program. The locales sampled included street microenvironments (e.g., trafficked roadside locations, background streets, and parks), transport microenvironments (e.g., cars, trains, buses, and bus stations), indoor microenvironments (e.g., restaurants and libraries) and

Table 4. Numbers of Samples Collected in Subject-Related Microenvironments

Air Toxic	Home, Day	Home, Night	Workplace	Total
VOCs	80	80	40	200
1,3-Butadiene	76	76	38	190
PAHs	66	66	36	168

Table 5. Numbers of Samples Collected in Other Microenvironments

Microenvironment, Sub-microenvironment	VOCs				1,3-Butadiene				PAHs			
	Summer	Winter	Sub- total	Total	Summer	Winter	Sub- total	Total	Summer	Winter	Sub- total	Total
Street				150				97				57
Background street	28	28	56		16	14	30		8	10	18	
Park	4	3	7		4	3	7		3	2	5	
Pedestrian street	10	8	18		8	6	14		4	4	8	
Street canyon	10	10	20		9	6	15		6	4	10	
Trafficked roadside	28	21	49		16	15	31		8	8	16	
Transport				122				74				43
Car	7	6	13		6	6	12		2	2	4	
Train	20	6	26		8	5	13		6	2	8	
Subway	10	0	10		4	0	4		4	0	4	
Bus	14	6	20		10	4	14		7	2	9	
Main train station	8	8	16		4	4	8		2	2	4	
Bus station	3	4	7		3	0	3		2	2	4	
Local train station	8	8	16		4	4	8		2	2	4	
Bus stop	6	4	10		4	4	8		2	2	4	
Car park	2	2	4		2	2	4		1	1	2	
Indoor				39				39				23
Pub	9	5	14		9	5	14		5	6	11	
Restaurant	6	4	10		6	4	10		4	4	8	
Department store	2	0	2		2	0	2		0	0	0	
Supermarket	2	2	4		2	2	4		0	0	0	
Hair salon	1	0	1		1	0	1		0	0	0	
Library	2	3	5		2	3	5		1	1	2	
Museum	1	2	3		1	2	3		1	1	2	
Homes Other Than Subjects'				83				83				37
Living room and kitchen (concurrently)	16	12	28		16	12	28		9	8	17	
Spare bedroom	8	12	20		8	12	20		4	4	8	
Garage	12	12	24		12	12	24		4	4	8	
Living room and backyard (concurrently)	7	4	11		7	4	11		2	2	4	

other home microenvironments (in the kitchen, living room, backyard, garage, and spare bedroom). The number distribution of other microenvironments sampled is summarized in Table 5.

Summer samples were collected during May–August 2006 for all microenvironments except streets in Wales, which were sampled in May 2007. Winter samples were collected during November 2006–February 2007 in all locations. The sole sampling program in transport microenvironments in London was carried out in October 2006.

The sampler was supervised by a researcher at all times during the sampling period. Meteorologic data and a description of each microenvironmental sampling event

were recorded on appropriate forms, as described in the Data Collection section.

Streets Two-hour VOC, 1,3-butadiene, and PAH samples were collected according to a seasonal program conducted in various street microenvironments (Table 5): trafficked roadsides, background streets, pedestrian streets during rush hour (7:00–9:00 a.m.) and the afternoon (1:00–3:00 p.m.). Samples were also collected in parks during the afternoon (1:00–3:00 p.m.).

Trafficked roadsides were selected in the city center of London (zone 1, which refers to the central zone of London, covering the West End, the Holborn district, Kensington,

Paddington, and the city of London) and the city center and suburban areas of West Midlands and among trafficked rural roads crossing through a village (A roads) in Wales. Background streets were selected in the city center of London (zone 1); in the city center and suburban areas of Birmingham, and in a rural village in Wales. Pedestrian streets were located in the city centers of London and Birmingham as well as in a suburban area in Birmingham. Parks were located in the city center of London and in suburban areas in Birmingham.

Samples were collected mainly in Birmingham, for logistical reasons. However, samples from trafficked roadsides and background streets were also collected in London and Wales, in two different locations, during each season and each time period mentioned above. One sample from a pedestrian street and one from a park were collected in London for each season and each time period.

The OME sampler was located on top of a portable table on the curb, with the inlets facing the road or street. The sampler was placed in the same position and location every time a given site was sampled, regardless of time of day or season. In the case of the park and the pedestrian street, the sampler was located away from local sources and trees or walls, to avoid shadowing effects.

Transport Microenvironments Two-hour VOC, 1,3-butadiene, and PAH samples were collected in the West Midlands area in a seasonal program in various mobile transport microenvironments (Table 5), namely bus, train, and car during rush hour (7:00–9:00 a.m.) and the afternoon (1:00–3:00 p.m.). Samples were also collected in London transport systems in one sampling event carried out in the autumn, in which samples were collected in two different buses, two trains, and two subway trains at rush hour and in the afternoon.

The buses and trains sampled covered linear routes from the city center outward into suburban areas, and vice versa, with an average traveled distance of 30 miles per leg. The car samples were taken in Birmingham city center and suburban areas with typical traffic patterns for cities and suburban areas, respectively.

Sampling in vehicles was performed with the sampler secured with ropes to the passenger seat. The inlets faced the center of the car or the aisle of the train or bus. In double-decker buses, the sampler was located on the bottom deck.

In addition, 2-hour VOC, 1,3-butadiene, and PAH samples were collected during a seasonal program at various transport stations (Table 5). Two samples were collected in summer and in winter during rush hour and in the afternoon, as defined above, at main train stations, local train stations, and local bus stops. Obtaining permission for sampling in bus stations proved to be very difficult, and

only one location could be sampled at the two proposed times in summer and winter. In the case of the car parks, two samples were taken in two different locations in both seasons, in the afternoon.

Main train and bus stations were located in the city centers of Birmingham and London, respectively. The local bus stops and train stations were located in suburban areas of Birmingham. The car parks were multi-storey buildings located in the city center of Birmingham. The samplers were placed on a portable table in the main passenger areas: the waiting area of the bus stations, the platform of the train stations, on the curb at local bus stops, and in a parking space within the car parks. The inlets faced the flow of traffic, and the position was replicated each time a given location was sampled.

Indoor Areas To assess air toxic levels in indoor locations other than homes and workplaces, a seasonal program was designed to measure VOC and PAH concentrations in various indoor environments (Table 5). Pubs, restaurants, libraries, museums, supermarkets, department stores, and hair salons were the indoor microenvironments chosen to reflect common places frequented by people during their normal day-to-day activities.

A total of 24 VOC and 1,3-butadiene samples and 19 PAH samples were collected in pubs and restaurants. Fifteen VOC and 1,3-butadiene samples were monitored in other indoor environments, whereas just four PAH samples were obtained from a library and museum (Table 5). All samples were collected during daytime hours over 2-hour intervals, preferably from 1:00–3:00 p.m., although some pub samples were collected during the evening (6:00–8:00 p.m.). Because of the difficulty in getting permission to measure indoor microenvironments, just two libraries, one museum, and one supermarket were sampled during both seasons. We obtained permission from managers of one department store and one hair salon to collect one sample during the summer. On the other hand, permission was given by managers of five pubs and four restaurants for sampling in their premises in summer and winter.

All these indoor microenvironments were sampled in Birmingham. The pubs and restaurants were located in urban, suburban, and rural areas. The other indoor environments were located mainly in a suburban area. The sampler was placed on a table with the inlets facing the center of the microenvironment in a location representative of the environment (e.g., a table for customers in a pub).

Homes Other Than Subjects' Residences VOC, 1,3-butadiene, and PAH samples were collected as part of a seasonal program in homes other than subjects' home, mainly

in suburban areas, in various home microenvironments such as kitchens, living rooms, spare bedrooms, and garages. This sampling was conducted over 12-hour periods during the day and 12-hour periods during the night (Table 5). Samples were also collected in backyards (over 6-hour daytime periods). Some of these microenvironments were sampled concurrently, such as living rooms and kitchens or living rooms and backyards.

DATA COLLECTION

Subject-Related Information

First, to enable reconstruction of exposures from micro-environmental measurements, the subjects kept activity diaries in which they recorded their location and mode of activity every 30 minutes. If a number of activities were conducted within the sampling day, they were asked to list these. Basic lifestyle information and important factors influencing exposure, including the presence of a smoker, the degree of ventilation, and the presence of an integral garage at the home, were also recorded by subjects, as was the precise location of residence and workplace and their proximity to neighboring highways and busy roads. The information collected from the subjects was recorded in the following forms (Appendix 3).

- The time–activity diaries gave information, for every 30 minutes, about where the volunteers had been, what they were doing, whether there was ventilation, and whether there were people smoking.
- The traveling description sheet gave information about the trips the subjects took every day (e.g., means of transport, time spent traveling, state of the roads, and places traveled through).
- The location description sheet gave information about the places the subjects visited (e.g., the location, time spent in each microenvironment, state of the nearby roads, and previous redecoration).
- The activity questionnaires gave detailed information about different activities performed during the day that could affect the personal exposure (e.g., use of aerosols and perfumes, use of a photocopier, refueling of a car, and home repair and improvement).
- The storage questionnaire gave information about products that the volunteers stored in their homes and/or garages that could affect their personal exposure. This questionnaire was implemented after the start of the study, used by the 27th recruited subject onwards.
- The home questionnaire gave basic information about the home of each subject, the heating and cooking system used, the ventilation, redecoration events,

integral garages, and smoking events that might have affected the interior air.

- The screening questionnaire gave general information about each volunteer, the location of the home and work with reference to busy roads, traveling patterns, and presence or absence of an integral garage at the home and a source of ETS.
- The ETS questionnaire gave information about the smoking events that the volunteers had been exposed to during a specific day. This questionnaire was implemented for the last 50 volunteers.

Compliance of the volunteers with the instructions for completing the forms was assessed every sampling day by the researcher in charge. The assessment consisted of checking the filled-out forms and checking whether there was any situation precluding the subject from carrying the briefcase (and if so, noting that in the activity diary). Accuracy of the filled forms was also assessed, involving checking that the complementary information entered in different forms was consistent and detailed (e.g., if a subject reported exposure to ETS in the activity diary, the researcher had to make sure that an appropriate ETS form had been completed). On the other hand, the fact that the forms were focused more on collecting information by location than by activity reduced the discrepancies between activity patterns and reported activity diaries (Robinson 1988).

Microenvironmental Data

Each time a microenvironment was monitored, information was recorded to describe the microenvironment and to aid the interpretation of the results at a later stage. For home and workplace microenvironments, a photograph was taken or a schematic of the location of the sampler in the room was drawn. In addition, the position of the ME sampler with respect to walls, windows, doors, and heating systems was recorded. For the last 40 volunteers recruited, meteorologic conditions such as temperature and relative humidity were also collected.

In the case of samples from microenvironments other than those related to the subjects, a series of description sheets was developed to collect information about the sampling conditions representative of the various microenvironments such as street, transport, and indoor environments. In those forms, information about the microenvironmental location, the state of the roads, smoking events, weather conditions, ventilation, and redecoration was recorded. In all cases photographs were taken or schematics were drawn. Meteorologic conditions (temperature and relative humidity) were recorded in all cases. Wind speed was also recorded in outdoor microenvironments.

A copy of each form is presented in Appendix 4.

ANALYTICAL METHODS

Analysis of VOCs

All the VOCs samples were analyzed at the University of Birmingham according to standard operating procedures based on a previously described method (Kim 2001; Kim et al. 2001a). Briefly, the method comprises the thermal desorption of the compounds from sampling tubes and subsequent analysis with a GC-MS.

Sampled tubes loaded on the thermal desorption unit (Aerotrap 6016 Autosampler, Tekmar) were heated to 250°C for 20 minutes to allow for the complete release of the target compounds from the adsorbent. Released compounds were transferred in the carrier gas to a liquid nitrogen-cooled cryotrap (Aerotrap Desorber 6000, Tekmar) containing Tenax GR, cooled to -30°C. When desorption was completed, the cryotrap was heated to 250°C for 4 minutes, releasing the trapped compounds onto the GC column, where they were separated and identified using a MS detector. After desorption, the tube was baked at 275°C for 10 minutes to condition the tube for subsequent sampling.

Compounds were separated using a column (Varian CP7723 CP Wax 52CB column, 50 m in length \times 0.25 mm in internal diameter \times 0.20 μ m in film thickness), a GC oven (Agilent Technologies 6890 N Network GC System) and a MS detector (Agilent Technologies 5973 Network Mass Selective Detector). Samples were injected at a split ratio of 100:1 at 250°C. The initial temperature of the GC oven was held at 40°C for 5 minutes, then ramped up at 5°C per minute to a final temperature of 180°C, held for 1 minute. The carrier gas was helium, with a constant flow rate of 1 mL min⁻¹.

The detector was set to quantify the analytes in single ion monitoring mode, covering specific masses ranging from 43.1–128 atomic mass units with a single ion monitoring of 50–100 milliseconds per ion. The MS quad and source temperatures were 150°C and 230°C, respectively. The analysis time per sample was 34 minutes.

After the sample was analyzed with a GC-MS, each chromatogram was checked using MSD ChemStation software and the data were transferred to an Excel file to enable the concentration of each target VOC in the samples to be calculated. The samples were analyzed and quantified using a minimum-five-point calibration graph of concentration against peak area, using linear regression. The calibration graphs were determined for each VOC with the use of tubes spiked with known concentrations of standard solutions prepared in methanol from commercially purchased, certified, standard solutions of the target compounds (LGC Promochem, Greyhound Chemservice, and UltraScientific). This procedure was carried out each time the column was replaced on the GC.

Quality assurance (QA) and quality control (QC) during analysis of the VOCs included an instrument performance check and tuning before initial calibration, preparation of an initial five-point calibration curve, analysis of laboratory blanks at the beginning of a batch run, standard check at the beginning and end of each batch run, re-conditioning of highly exposed tubes, and analysis of randomly selected duplicate standards.

Analysis of 1,3-Butadiene

1,3-Butadiene was sampled and analyzed separately from the other VOCs because of its high volatility. All 1,3-butadiene samples were analyzed at the University of Birmingham, according to a standard operating procedure based on a technique previously developed and validated in our laboratory (Kim et al. 1999). Briefly, the method comprises the thermal desorption of the 1,3-butadiene from the sampling tube and subsequent analysis on a GC-MS.

Sampled tubes loaded on the thermal desorption unit were heated to 350°C for 15 minutes to allow for the complete release of 1,3-butadiene from the adsorbent. The released gas was transferred and trapped at -60°C on Tenax GR in the liquid nitrogen-cooled cryotrap. When desorption was complete, the cryotrap was heated to 350°C for 5 minutes, releasing the 1,3-butadiene onto the GC column. After desorption, the tube was baked at 360°C for 10 minutes to condition the tube for subsequent sampling.

Two columns were used to analyze 1,3-butadiene. Samples quantified before March 2006 were analyzed using a capillary column (Varian CP7552 CP-PoraPLOT Q; 52.5 m in length \times 0.32 mm in internal diameter \times 10 μ m in film thickness), and the remaining samples were quantified using a different column (Varian CP7352 CP-PoraBOND Q; 50.0 m in length \times 0.32 mm in internal diameter \times 5 μ m in film thickness). The change in the capillary column was based on the criteria of using the best available capillary column for 1,3-butadiene separation, after a crack was discovered in the PoraPLOT column. Results were compared between the two capillary columns, and the new PoraBOND column was validated for the analysis of 1,3-butadiene using the same analytical methods.

1,3-Butadiene was isolated using the same GC (Agilent Technologies 6890 N Network GC System) and MS detector (Agilent Technologies 5973 Network Mass Selective Detector) as for the VOC samples. Samples were injected at a split ratio of 50:1 at 250°C. The initial temperature was held at 90°C for 3 minutes, then ramped up at 8°C per minute to a final temperature of 220°C, held for 3 minutes. The carrier gas was helium, with a constant flow rate of 1 mL min⁻¹.

The detector was set to quantify 1,3-butadiene in single ion monitoring mode, covering the specific masses 39.0,

53.1, and 54.1 atomic mass units, with a dwell time of 100 milliseconds per ion. The MS quad and source temperatures were 150°C and 230°C, respectively. The analysis time per sample was 32 minutes.

Each chromatogram was checked using MSD ChemStation software and the data were transferred to an Excel file to enable the concentration of 1,3-butadiene in the samples to be calculated. The samples were analyzed and quantified using a six-point calibration graph of concentration against peak area, using linear regression. Standard tubes were prepared by injecting an appropriate amount of a certified 1,3-butadiene sample into a nitrogen standard gas mixture (Restek Corporation) by means of a valve and a sample loop. This procedure was carried out each time the column was replaced in the GC-MS.

QA and QC during the 1,3-butadiene analysis included an instrument performance check and tuning before initial calibration, preparation of an initial six-point calibration curve, analysis of a laboratory blank at the beginning of a batch run, a standard check at the beginning and end of each batch run, re-conditioning of highly exposed tubes, and analysis of randomly selected duplicate standards.

Analysis of PAHs

All the PAH samples were analyzed at the University of Birmingham, according to a standard operating procedure based on a method specifically developed and validated in this study. Briefly, the method comprises the extraction, concentration, and purification of the PAH compounds from the filter media and subsequent analysis with a GC-MS.

PAH filters were spiked with internal standards before extraction, to enable monitoring of the extraction procedure. Filters were placed in conical flasks with 15 mL of dichloromethane (high-performance liquid chromatography [HPLC] grade) and were shaken for 15 minutes at 1400 revolutions per minute. The extract was pre-concentrated to approximately 0.5 mL by blowing down with nitrogen and was subsequently dried and cleaned by removing the remaining filter fiber with a chromatography column filled with anhydrous sodium sulfate. The cleaned extract was then further concentrated by blowing down with nitrogen to 25 μ L. The solvent was changed from dichloromethane to nonane (purum, 99%; Sigma-Aldrich Company) with a final volume of 25 μ L. Extracted samples were stored in GC vials in a freezer at -20°C. Before analysis, 25 μ L of recovery standard was spiked into the insert vial to monitor the extraction process with regard to the recovery of the internal standards. Samples were stirred to allow for mixing of the recovery standard into the sample and were then placed on the GC autosampler (Agilent 7683 Series Autosampler).

A 1- μ L aliquot of sample was injected in a splitless and non-pulsed injection mode at 300°C. Compounds were separated using a column (Agilent HP-5MS column; 30 m in length \times 0.25 mm in internal diameter \times 0.25 μ m in film thickness), a GC oven (Agilent Technologies 6890 N Network GC System) and a MS detector (Agilent Technologies 5973 Network Mass Selective Detector). The initial temperature was held at 120°C for 2 minutes, then ramped up at 4°C per minute to a final temperature of 300°C, held for 10 minutes. The carrier gas was helium, with a constant flow rate of 1 mL min⁻¹. The solvent delay time was set to 3.8 minutes.

The detector was set to quantify the analytes in single ion monitoring mode covering specific masses ranging from 122–300 atomic mass units, with a dwell time of 75–100 milliseconds per ion. The mass spectrometer quad and source temperatures were 150°C and 230°C, respectively. The analysis time per sample was 57 minutes.

Each chromatogram was checked using MSD ChemStation software and the data were transferred to an Excel file to enable the concentration of each target PAH in the sample to be calculated.

The samples were analyzed and quantified using a six-point calibration graph of the concentration ratio of the analyte and the internal standard against the corresponding peak area ratios, using linear regression. This procedure curve was performed every time the column was replaced in the GC or when routine maintenance (e.g., source cleaning) was carried out in the GC-MS. The calibration graphs were determined for each PAH with the use of standard solutions prepared in nonane (purum, 99%) containing the certified standard 16 EPA Priority PAHs in toluene (LGC Promochem), coronene standard solution in toluene (Greyhound ChemService), nine deuterium-labeled internal standards (Greyhound ChemService and UltraScientific) and *p*-terphenyl-D14 in methylene chloride as recovery standard (UltraScientific).

QA and QC during the PAH analysis included an instrument performance check and tuning before initial calibration, preparation of an initial six-point calibration curve, analysis of laboratory method blanks after every 10 samples had been run, a standard check at the beginning and end of each batch run as well as after every 10 samples had been run, and the extraction and analysis of standard reference material 1649a (Greyhound ChemService) after every 25 samples had been run.

Analysis of Urinary Biomarkers

Analysis of urinary biomarkers was performed by the research group led by Dr. Peyton Jacob III in the Division of

Clinical Pharmacology at the University of California, San Francisco.

PAH metabolites in human urine, such as monophenolic metabolites of naphthalene (2-naphthol), fluorene (1-hydroxyfluorene, 2-hydroxyfluorene, 3-hydroxyfluorene), phenanthrene (1-hydroxyphenanthrene, 2-hydroxyphenanthrene, 3+4-hydroxyphenanthrene), and pyrene (1-hydroxypyrene) were analyzed with LC–MS–MS. This analytical method involves conversion of the metabolites to the pentafluorobenzyl ether derivatives, in order to enhance sensitivity by making use of the electron-capture atmospheric-pressure chemical ionization technique. LC–MS–MS analyses were carried out with a Surveyor HPLC instrument interfaced to a TSQ Quantum Ultra triple-stage quadrupole mass spectrometer (Thermo-Finnigan, San Jose, CA). A PAH Green column (4.6 × 150 mm; Thermo-Hypersil-Keystone, Bellefonte, PA) was used for the chromatography (Jacob et al. 2007).

Concentrations of cotinine and *trans*-3'-hydroxycotinine in urine were determined using LC–MS–MS. The method is similar to a published method for determining cotinine concentrations in plasma of non-smokers (Bernert et al. 1997). Deuterium-labeled internal standards, cotinine-D9 and *trans*-3'-hydroxycotinine-D9, were used as internal standards. The MS is operated in the positive ion mode using atmospheric pressure chemical ionization. Quantitation is achieved using selected reaction monitoring of the transitions from the mass-to-charge ratio (m/z) of 177 to m/z 80 for cotinine, m/z 193 to m/z 80 for *trans*-3'-hydroxycotinine, and m/z 186 to m/z 84 and m/z 202 to m/z 84 for the respective internal standards (P. Jacob, personal communication, April 2006).

DATABASE DESIGN

All data collected in the Measurement and Modelling of Exposure to Air Toxic Concentrations for Health Effect Studies (MATCH) study was input into and stored in a custom database. The MATCH database was developed using Microsoft Access (version 2007). The goals of the database were to store all data collected, to identify all data with a unique code, to link appropriately the data stored in various tables to facilitate data entry and data recovery, to have procedures to check the correctness and completeness of data entry, and to protect the privacy of the volunteers.

Data stored in the MATCH database included all the information from volunteers, after being made anonymous, from the eight forms used, the VOC and PAH personal exposure concentrations, urine biomarker concentrations, microenvironmental data collected on the four forms according to microenvironment type, the VOC and PAH microenvironmental concentrations, and meteorologic information.

QA–QC AND RECORD KEEPING

Standard Operating Procedures

Standard operating procedures were developed to facilitate the homogeneity, reproducibility, and accuracy of the sampling and analysis for the following major activities:

- Subject screening and sampling visits,
- Preparation of sampling tubes for VOCs and 1,3-butadiene,
- Personal exposure monitoring,
- Microenvironmental monitoring in subject-related areas,
- Microenvironmental monitoring in other areas,
- Sampling of urine and storage of these samples,
- Analysis of VOCs using a GC–MS,
- Analysis of 1,3-butadiene using a GC–MS,
- Analysis of PAHs using a GC–MS, and
- Extraction of PAHs.

Besides the standard operating procedures, some instructions were developed to cover specific activities that required detailed information:

- Changing the column in the GC–MS,
- Filling the nitrogen vessel connected to the thermal desorption unit,
- Loading the thermal desorption unit and running the GC–MS,
- Conditioning the sampling tubes for the analysis of VOCs and 1,3-butadiene in air, and
- Setting up, running the samplers within, and dismantling the briefcase.

Finally, some of the standard operating procedures and instructions were accompanied by checklists to ensure that all the steps were followed and all the parameters were checked when carrying out certain activities as well as when sampling away from the research center in London and Wales. Checklists were written for:

- Loading the thermal desorption unit with 1,3-butadiene tubes and running the GC–MS,
- Loading the thermal desorption unit with VOC tubes and running the GC–MS,
- Sampling personal exposures (general procedures),
- Sampling personal exposures in London and Wales, and
- Sampling exposures in microenvironments other than subject-related areas, in London and Wales.

QA–QC During Sampling

Blanks For QA purposes, enough blanks were prepared to be equivalent in number to 6% of the total number of samples obtained during both the personal exposure and the microenvironmental sampling. The types of blanks collected were travel blanks, travel and exposure blanks, and travel and environment blanks.

A travel blank was a clean sample (e.g., a conditioned filter or sampling tube) that was carried to the sampling site, returned to the laboratory without being opened, and treated as an environmental sample. In this study, a travel blank was a capped sorbent tube or a filter enclosed in a metal tin that was placed in the air sampler during the period of travel to the site but was not placed in the briefcase during the period of sampling.

A travel and environment blank was a clean sample carried to the sampling site, exposed to sampling conditions without being opened, returned to the laboratory, and treated as an environmental sample. In this study, a travel and environment blank was a sorbent tube or a filter enclosed in a metal tin that was placed in the air sampler at the same time as the normal sample but was kept closed during the preparation and sampling periods.

A field blank was a clean sample that was calibrated, closed, carried to the sampling site, exposed to sampling conditions without being opened, returned to the laboratory, and treated as an environmental sample. In this study, a field blank was a sorbent tube or a filter placed in the air sampler, calibrated, closed later with cap locks (if a sorbent tube) or enclosed in a metal tin (if a PAH filter), placed in the air sampler for the same time as normal samples, but kept closed during the sampling time.

Duplicates For assessing the precision of measurements, duplicate samples were collected for 3% of the measurements. Duplicate samples were two samples taken with two identical samplers in the same environment during the same time period. Duplicate samples were carried through all the steps of sampling and analysis in an identical manner. Therefore, they were used to assess the total variance of the sampling and analytical procedures.

Calibration of Flow Rates The flow rates of all the pumps used in sampling (personal and microenvironmental) were calibrated with rotameters covering the appropriate range of values before and after the sampling period; flow rates were calculated as the average of both values. All the rotameters were calibrated regularly using an air flow calibration system (Giliblator) that, in turn, was calibrated and serviced annually. All pumps were serviced and calibrated by the supplier annually.

Tests of Stability of Sample Storage An experiment was conducted to test the stability of sampled VOC and 1,3-butadiene tubes stored in the refrigerator before analysis. Sets of VOC and 1,3-butadiene standards were prepared with a midrange standard concentration, with target preparation times of 8, 6, 4, 2 and 0 weeks before analysis.

Owing to the technical problems with the GC–MS system at the time of the tests of sample storage stability, it was not possible to analyze the test tubes for VOCs at the target times; instead, storage times of 21, 19, 17, 15, and 0 weeks before analysis were used. Fortunately, the 1,3-butadiene stability tests could be performed on the target timescale.

A second storage stability test was performed for 1,3-butadiene tubes. In this case, six samples were collected in a high-pollution environment, and another six in a low-pollution environment. Half of each set of samples were analyzed immediately after sampling, and the other half 20 weeks after collection. In addition, two replicate standard tubes were spiked with medium concentrations of 1,3-butadiene and two with high concentrations; these were stored 20 weeks before analysis.

QA–QC During Analysis

The QA–QC protocol for the analysis was developed on the basis of guidelines already in place within our research group (Harrad 2005). QA–QC results are presented in Appendix 5.

Blanks For QA–QC purposes, enough laboratory method blanks were prepared to be equivalent in number to 10% of the total number of analyzed samples. A laboratory method blank was a tube or filter that was not removed from the laboratory and that underwent the same analytical procedure as a field sample.

GC–MS Performance Check and Method Calibration During the analysis stage, before performing the method calibration, the GC–MS system was checked against the mass spectral ion abundance criteria for the instrument performance check standard containing perfluorotributylamine. After these criteria had been met, the GC–MS system was fully calibrated with standards that spanned the monitoring range of interest. The method-calibration sequence was used to determine the instrument sensitivity and the linearity of GC–MS response to the target compound concentrations. Calibration curves were constructed by plotting the concentration versus the peak area of the analyte for five data points for VOCs and six points for 1,3-butadiene, using linear regression. In the case of PAHs, calibration curves of six points were constructed by plotting

concentrations against peak area ratios of the analyte and its internal standard. Analytical methods for urinary biomarkers also involved calibration with standards.

Standard Check During the analysis stage, standard checks were conducted to monitor the performance of the GC–MS system. In the case of VOCs and 1,3-butadiene, one midrange standard was placed at the front of each batch, immediately after the laboratory method blank, and a second standard was placed at the end of the batch. Each batch of VOC or 1,3-butadiene samples contained a maximum of 10 samples. PAH sample batches were considerably larger, and therefore a midrange standard was run at the beginning and end of the batch run as well as after every ten samples.

Limits of Detection Two categories of limits of detection were calculated for all the analytical methods used: the instrument detection limit and the sample detection limit.

The instrument detection limit was defined as that amount of pollutant that gives a signal-to-noise ratio of 3:1. It was best determined by calculating the signal-to-noise ratio for the pollutant with the lowest concentration in the calibration standard. The sample detection limit was defined as the instrument detection limit divided by the sample size.

Precision The precision was calculated for all methods used. Precision was defined as the relative standard deviation of concentrations obtained from replicate analyses (n) of the same sample (i.e., $100 \times \sigma_{n-1}/\text{average}$). In the case of the VOC and 1,3-butadiene methods, precision was calculated from replicate standard tubes. For the PAH method, precision was calculated from replicate standard solutions and from the replicate analysis of standard reference material.

Accuracy Accuracy of the VOC and 1,3-butadiene methods was determined via analysis of standard tubes prepared from certified standard solutions. Accuracy was calculated as the difference between the value given in the analysis and the nominal value of the standard, divided by the nominal value of the standard. Accuracy of the PAH method was calculated in two ways: via the analysis of standards prepared from certified standard solutions, and via analysis of standard reference material 1649a.

Recovery of Internal Standards All PAH samples were spiked with deuterated internal standards before the extraction was performed, and with a deuterated recovery standard after the extraction was performed, to monitor the performance of the extraction.

Data Integrity and Record Keeping

Protocols for data integrity, data traceability, sample referencing, and making the subject data anonymous were prepared. The requirements of the relevant European Union legislation (Directive 95/46/EC on the protection of individuals with regard to the processing of personal data and on the free movement of such data) were taken into account.

STATISTICAL METHODS AND DATA ANALYSIS

Data were analyzed using SPSS (version 15.0 for Windows; SPSS Inc. 1989–2006), Excel (version 2002; Microsoft Corporation, 1985–2001) and Access (version 2007; Microsoft Corporation, 2006). The main components of the data analysis presented:

- Characterization of personal exposure and microenvironmental concentrations of VOCs, including 1,3-butadiene, and PAHs.
- Characterization of urinary biomarkers and examination of the correlation of biomarker concentrations with personal exposure to selected air toxics.
- Source apportionment of the data set of personal exposure and microenvironmental concentrations.
- Development and validation of a predictive model of personal exposure for selected air toxics.

CHARACTERIZATION OF PERSONAL EXPOSURES AND MICROENVIRONMENTAL CONCENTRATIONS

Personal exposure and microenvironmental concentrations were tested for normality using the skewness statistic. Concentrations of all the VOCs tested, including 1,3-butadiene, and PAHs were found to have right-skewed distributions. For this reason, the VOC and PAH data were \log_{10} -transformed, geometric means (GMs) and geometric standard deviations (GSDs) were calculated, and measures of association were characterized by Pearson correlation coefficients (R) for both the transformed and nontransformed data.

Personal exposures and microenvironmental concentrations were characterized using descriptive statistics and graphical analysis. The techniques used include univariate distribution analysis and graphical distribution analysis using bar charts to characterize data sets and Pearson correlation analysis to assess relations between compounds. Univariate distribution analysis reported medians, 25th and 75th percentiles, and arithmetic means and standard deviations (SDs), GMs and GSDs, and maxima and minima for all pollutant concentrations. Summaries of these values are reported in Appendices 4 through 12. Personal

exposure and microenvironmental concentrations are presented here as arithmetic means \pm SDs, with the GM in parentheses.

Statistical differences between two strata were tested in the \log_{10} -transformed database with a *t* test for equality of means and Kolmogorov–Smirnov test in cases in which the variance was heterogeneous. Analyses of variance performed on the \log_{10} -transformed database were used to assess differences among more than two strata in cases in which the variance was homogeneous, whereas the Kruskal–Wallis chi-square statistic was also used for the \log_{10} -transformed database in cases in which the variance was heterogeneous. For personal exposure values, statistical differences were calculated among the five-day average concentration levels ($N = 100$) and also the individual concentration levels ($N = 500$). Results were considered significant if *P* values were < 0.05 , and all *P* values < 0.10 were reported. Variance was considered homogeneous provided that $P > 0.05$ from the Levene test. In the case of personal exposure, only *P* values from tests involving the five-day average concentrations are discussed here. The results of these analyses are presented in Appendices 7 through 13.

As a consequence of the air toxic concentrations having \log_{10} -normal distributions, bar graphs with a log scale were used to present GMs and error bars representing the 95% confidence intervals. If fewer than five data points were valid for a compound, confidence intervals were not shown.

The analyses were done on a compound-by-compound basis. In addition to the analysis of all the data, pooled, analyses were done according to several stratifying variables. For the personal exposure and home microenvironmental data set, the strata were locations of the residences (i.e., urban, suburban, or rural), presence or absence of integral garages, presence or absence of ETS exposures, first-line or non-first-line properties, and city. The same strata, plus daytime or nighttime, were used to analyze data collected from home microenvironments. For the rest of the microenvironmental data set, the strata were season, daytime vs. nighttime, the presence or absence of ETS exposures, and sub-microenvironment type (as described in Table 5). For simplicity, multivariable analyses to estimate mutually adjusted effects of characteristics were not undertaken.

The statistical inference we used ignores, for simplicity's sake, some of the correlation expected among the data, in particular between repeated measurements from the same subject on different days. Including this type of correlation would have resulted in confidence intervals that were somewhat too narrow and *P* values that were too low. We have addressed the omission by being cautious in our interpretation.

Concentrations of VOCs, including 1,3-butadiene, are reported in micrograms per cubic meter, whereas PAH concentrations are reported in nanograms per cubic meter. Data below the detection limit (for a summary of these, see Table A14.9, Appendix 14) were replaced with half the value of the detection limit for the purpose of statistical analysis. When duplicate samples were collected, the average of both measurements was used in the data analyses. Personal exposure data were considered for inclusion in the analysis only if the sampling time was over 1100 minutes, therefore avoiding all the data collected with faulty battery pumps. In the same way, data for which the final flow rate could not be measured owing to a rotameter breakage were not included in analyses.

CHARACTERIZATION OF URINARY BIOMARKERS AND CORRELATION WITH PERSONAL EXPOSURES TO SELECTED AIR TOXICS

Urinary biomarker concentrations were characterized using univariate distribution analysis reporting medians, 25th and 75th percentiles, and arithmetic means and SDs, GMs and GSDs, and maxima and minima for each biomarker.

The urinary biomarker distribution was right-skewed, so the data were \log_{10} -transformed for most analyses. To assess relations between urinary biomarkers and concentrations of the parent compounds, bivariate scatterplotting was used, as well as Pearson correlation coefficients (*R*) in transformed data and nontransformed data.

SOURCE APPORTIONMENT

PCA was the technique used to assess source apportionment of VOC and PAH samples. As the data set was found to be not normally distributed, PCA was performed on the natural-log-transformed concentration data set. Analyses were done in SPSS (version 15.0 for Windows), using the Varimax rotation method.

Data below the detection limit were replaced with half the value of the detection limit. Species for which more than 20% of the data were below detection limits were not included in the PCA. Absolute factor-loading coefficients of < 0.40 have been omitted from tables to facilitate identification of sources.

DEVELOPMENT OF THE PERSONAL EXPOSURE MODEL

The personal exposure data were split into two different and independent data sets. The first set contains 75% of the data and was used for “training” the model. The other 25% of the data was saved as an independent-contrast validation data set to check and validate the model developed

with the training data set. Samples included in each data set (training and validation) were chosen among all person-days in order to ensure an equal distribution of high and low concentrations in each data set.

Various models were implemented and tested to develop and identify the model that best predicts personal exposure to selected air toxics.

Model 1

Model 1 assesses associations between personal exposure and microenvironmental concentrations, as measured in homes and workplaces directly related to each subject. The equations used in model 1 are:

$$\text{Log}(Y_i) = \alpha + \beta \times \log(X_{i, \text{home}}) + \varepsilon_i \quad (1.1)$$

and

$$\text{Log}(Y_i) = \alpha + \beta \times \log(X_{i, \text{workplace}}) + \varepsilon_i \quad (1.2)$$

where Y_i is the five-day average measured personal exposure for a subject i , α is the intercept of the model, β is the slope of the model, $X_{i, \text{home}}$ is the average home concentration for subject i and $X_{i, \text{workplace}}$ is the workplace concentration for subject i . The ε_i terms account for random error. Equations 1.1 and 1.2 were fitted to the data separately.

Model 2

Model 2 predicts the personal exposure, integrating the fraction of time spent in each microenvironment multiplied by the concentration in each microenvironment visited, as reflected in equation 2:

$$Y_{ij} = \sum \frac{t_{ijk} \times X_{ik}}{T_{ij}} \quad (2)$$

where Y_{ij} is the predicted personal exposure for a subject i on a day j , t_{ijk} is the time spent in microenvironment k by subject i on a day j , X_{ik} is the concentration representative of microenvironment k for subject i , and T_{ij} is the total time spent in all microenvironments by subject i on a day j , which in turn is the same as the sampling time for subject i on a day j .

The microenvironmental concentrations used in model 2 for homes and workplaces were the data collected directly in each volunteer's home and workplace. For the volunteers with no data for home or work available, and for the non-subject-related microenvironments (streets, transport settings, and other indoor microenvironments), an average concentration representative of each microenvironment was used. The concentration value for each microenvironment was obtained by averaging the concentrations measured in each specific microenvironment (e.g.,

taking the arithmetic mean of all measurements in pubs, to represent the pub concentration in model 2). The table containing these representative concentrations is available in Appendix 19 (Table A19.1).

Model 3

Model 3 predicts the personal exposure by integrating the fraction of time spent in each microenvironment multiplied by the concentration in each microenvironment visited, as reflected in equation 3. Model 3 differs from model 2 in that the microenvironmental concentrations used in model 3 for homes and workplaces were not the data collected directly in each volunteer's home and workplace but instead a pooled value representative of the microenvironment. That is:

$$Y_{ij} = \sum \frac{t_{ijk} \times X_k}{T_{ij}} \quad (3)$$

with the variables the same as those defined above for equation 2, except that X_k is used (the concentration representative of microenvironment k , without specific regard to subject i). For equation 3, a detailed list of stratified microenvironmental concentrations was developed for all the microenvironments—homes, workplaces, streets, transport, and other indoor microenvironments—taking into account various strata such as location, season, traffic exposure, ETS exposure, and time of day, as appropriate. The concentration value for each stratum was the arithmetic mean of all the concentrations measured with regard to that stratum. The list of concentrations can be found in Appendix 19 (Tables A19.2 and A19.3).

Model 4

Model 4 predicts the personal exposure by integrating the fraction of time spent in each microenvironment, multiplied by the concentration in each microenvironment visited, and by accounting for external factors that might affect exposure, as add-on variables:

$$Y'_{ij} = \alpha \times Y_{ij} + \sum \beta_m A_m + \sum \gamma_n F_n \quad (4)$$

where Y'_{ij} is the observed personal exposure for a subject i on a day j , α is the coefficient associated with personal exposure, Y_{ij} is the personal exposure predicted for a subject i on a day j as calculated with model 3, A_m is an explanatory variable describing activities performed on a day j by a subject i or characteristics associated to a volunteer i , β_m is the coefficient associated with the explanatory variable A_m , F_n represents the time spent doing various activities, and γ_n is the coefficient associated with F_n .

Values of the explanatory variable A_m , related to activities such as burning incense, being exposed to ETS, or living in

a house with integral garage, was extracted from the time-activity diary, the ETS questionnaire, and the activity questionnaire. The explanatory variables related to characteristics were extracted from the home questionnaire, storage questionnaire, and the screening questionnaire. The explanatory variables were assigned a value of 1 if the activity was performed or the characteristic is present, and 0 if not.

The variables representing the time associated with various activities, F_n , were measured in minutes and were extracted from the time-activity diaries and location sheets for traveling. Examples of this sort of variable are “time exposed to ETS” or “time spent in a car.”

A total of 112 add-on variables, A_m and F_n , extracted from the collected information were included in the model. The list of the add-on variables included, and their meanings, can be found in Table A21.3 in Appendix 21.

The model was developed and run with SPSS (version 15.0) in three steps:

- Step 1. Development of the model using the Stepwise option from the Linear Regression Menu. This option gave the optimum number of variables to yield the highest correlation coefficient. The criterion for entering new variables was a probability of $F \leq 0.05$. The criterion for removal of variables was a probability of $F \geq 0.10$.
- Step 2. The variables selected automatically by the SPSS program were reviewed according to criteria of scientific meaning and to try to explain as much variation using the least number of variables.
- Step 3. A new model with the variables selected in Step 2 was run with the SPSS program using the Enter option from the Linear Regression Menu.

Model 5

Model 5 predicts the personal exposure by focusing explicitly on the factors initially set as key determinants: traffic effects (first-line property or not), having an integral garage at home or not, being exposed to ETS or not, and living in an urban, suburban, or rural area. The equation was as follows:

$$Y_{ij} = a + \sum \delta_l D_l \quad (5)$$

where Y_{ij} is the observed personal exposure for a subject i on a day j , a is a constant, D_l is the key determinant, and δ_l is the coefficient associated with key determinant D_l .

The model was developed with the SPSS (version 15.0) program using the Stepwise option from the Linear Regression Menu. This option gave the optimum number of variables to get the highest correlation coefficient. The

criterion for entering new variables was a probability of $F \leq 0.05$. The criterion for removing variables was a probability of $F \geq 0.10$.

To assess the improvements made in the method development and the bias in the prediction, we used linear regression to compare the measured personal exposure versus the predicted personal exposure in the training set; scatterplots of predicted versus measured data were presented, and the Pearson correlation coefficient (R) and the standard error of the estimate were calculated and analysis of variance of the regression was performed.

VALIDATION OF THE PERSONAL EXPOSURE MODEL

The best model developed with the training set was later validated with the independent data set. Therefore, the selected prediction model (i.e., the model with the regression coefficients obtained in the training data set) was used to predict concentrations in the validation data set. We calculated the Pearson correlation coefficient (R), the normalized mean bias, the mean fractional bias, and the percentages of predicted values that reflected a difference between the predicted and measured values within a factor of 2 and within a factor of 3 were calculated.

The normalized mean bias was calculated as follows:

$$NMB(\%) = \frac{\sum_{i=1}^N (Y_{\text{Predicted}} - Y_{\text{Measured}})}{\sum_{i=1}^N Y_{\text{Measured}}} \times 100 \quad (6)$$

where NMB (%) is the percentage of the normalized mean bias, $Y_{\text{Predicted}}$ is the concentration of the air toxic as predicted by the selected model, Y_{Measured} is the concentration of the air toxic as measured, and N is the total number of cases in the validation data set.

The fractional bias was calculated as follows:

$$FrB = \frac{2 \times (Y_{\text{Predicted}} - Y_{\text{Measured}})}{(Y_{\text{Predicted}} + Y_{\text{Measured}})} \quad (7)$$

where FrB is the fractional bias, $Y_{\text{Predicted}}$ is the concentration of the air toxic as predicted by the selected model and Y_{Measured} is the concentration of the air toxic as measured.

The mean fractional bias is calculated by averaging the fractional bias, as follows:

$$MFB(\%) = \frac{1}{N} \sum_{i=1}^N \frac{2 \times (Y_{\text{Predicted}} - Y_{\text{Measured}})}{(Y_{\text{Predicted}} + Y_{\text{Measured}})} \times 100 \quad (8)$$

where MFB (%) is the mean fractional bias, $Y_{\text{Predicted}}$ is the concentration of air toxic as predicted by the selected model,

Table 6. Selected Housing Characteristics of the Subjects ($N = 100$)

Characteristic	Urban		Suburban	Rural		All
	London	Birmingham	Birmingham	West Midlands	Wales	
Total number of dwellings	11	27	42	10	10	100
Type of dwelling						
First-line property	5	11	18	4	6	44
Integral garage	0	0	11	2	3	16
ETS exposure at home	1	3	8	0	0	12
House	1	0	34	10	10	55
Flat	10	27	8	0	0	45
Built before 1991	11	9	36	9	8	73
Built in or after 1991	0	18	6	1	2	27
Heat						
Natural gas	9	5	32	6	7	59
Electricity	0	19	8	2	0	29
Other fuel	0	0	1	2	3	6
Missing data	2	3	1	0	0	6
Cooking						
Natural gas	9	3	26	3	6	47
Electricity	2	24	16	7	4	53
Cooker hood used	3	11	17	2	3	36
Cooker hood not used	8	16	25	8	7	64
Redecoration within previous year						
Wallpapering	0	1	4	1	0	6
Carpeting or linoleum installed	3	2	7	1	0	13
Sanding or stripping performed	2	1	7	3	2	15
Use of glue or sealants	4	6	16	2	1	29
Use of air fresheners	4	5	19	3	4	35

Y_{Measured} is the concentration of air toxic as measured, and N is the total number of cases in the validation data set.

The fraction of data within a factor of 2 was calculated as the percentage of data for which $0.5 < Y_{\text{Predicted}}/Y_{\text{Measured}} < 2$. The fraction of data within a factor of 3 was calculated as the percentage of data for which $0.33 < Y_{\text{Predicted}}/Y_{\text{Measured}} < 3$.

Concentrations predicted with the model were regressed against concentrations measured and the corresponding scatterplots were presented.

PERCENT CONTRIBUTION OF VARIOUS MICROENVIRONMENTS TO OVERALL PERSONAL EXPOSURES

The average contribution from microenvironment K to the personal exposure to compound Z was calculated on the basis of the results of model 2, as follows:

$$\text{Percent exposure to compound } Z \text{ due to microenvironment } K = \frac{\sum_{i=1}^I \sum_{j=1}^J \frac{t_{ijk} \times X_{ik}}{T_{ij}}}{\sum_{k=1}^K \sum_{i=1}^I \sum_{j=1}^J \frac{t_{ijk} \times X_{ik}}{T_{ij}}} \times 100 \quad (9)$$

where t_{ijk} is the time spent in microenvironment k by subject i on a day j , X_{ik} is the concentration representative of microenvironment k for subject i , and T_{ij} is the total time spent in all microenvironments by subject i on a day j , which in turn is the same as the sampling time for subject i on day j .

RESULTS

STUDY POPULATION

In the study, a total of 100 subjects living in three locations across the United Kingdom were recruited. Most participants

Table 7. Subject Characteristics (*N* = 100)

Characteristic	Number of subjects
Sex	
Female	57
Male	43
Age (years)	
18–25	18
26–35	31
36–45	15
46–55	13
56–65	17
≥ 66	6
Occupation category	
Administration or office worker	48
Cleaning	1
Education	6
Food or hospitality	7
Health	7
Housewife	4
Manufacturing	1
Police	1
Research and development	7
Retired	7
Student	10
Unemployed	1
Time spent traveling to workplace (minutes)	
< 5	47
5–15	26
> 15–30	6
> 30–45	3
> 45–60	1
Not applicable	17
ETS exposure	
Yes, at home	12
Not at home	88
Yes, at work	8
Not at work	92

Table 9. Percentage of Subjects That Performed Various Activities

Activity	%
Cleaning	61
Dusting	27
Vacuuming	41
Aerosol or perfume use	55
Solvent use	8
Dry cleaning	4
Candle burning	13
Photocopier, printer, or fax machine use	41
Lighted fire in fireplace	11
Other fossil fuel use	4
Refueling car or visiting petrol station	23
Home repair or improvement	12
Gardening	8
Other	22

were recruited in the West Midlands, with 27 subjects living in Birmingham city center, 42 subjects in suburban areas of Birmingham, and 10 subjects living in the rural West Midlands. Eleven subjects were recruited in London, where some of the highest exposures were anticipated, and 10 were recruited in Wales, where some of the lowest exposures were anticipated. In terms of type of location, 38 subjects lived in urban areas, 42 lived in suburban areas, and 20 subjects lived in rural areas.

The attributes of the subjects' homes most directly relevant to the analysis reported on here are shown in Table 6. The attributes were extracted from the subjects' answers to the home questionnaire.

Table 7 summarizes some demographic data (e.g., sex, age, and occupation) of the 100 volunteers and gives information about the time they spent traveling to the workplace and whether they reported being exposed to ETS at

Table 8. Percentages of Overall Time Spent in Each Microenvironment

Microenvironment	Urban			Suburban Birmingham	Rural			All
	All	London	Birmingham		All	West Midlands	Wales	
Indoors at home	58	54	60	64	65	67	63	62
Indoors at work	21	27	19	15	10	7	14	16
Other indoors	11	7	12	12	16	18	14	12
Total indoors	90	87	91	91	91	91	91	91
Outdoors	5	6	5	4	2	2	2	4
In transit	5	6	4	6	7	7	7	6

Table 10. Percentages of Subjects That Reported the Use of Products and Activities

Product or Activity	%
Toiletries	
Deodorant, body spray, or perfume	42
Hair products	9
Nail varnish or remover	1
Shampoo, conditioner, or toothpaste	1
Air freshener	
Plug in or automatic	13
Spray	12
Burning of candle, incense, or oil	14
Cleaning	
Antibacterial wipes	5
Cleaning fireplace, sweeping, or vacuuming	44
Dry cleaning	5
Ironing	6
Sprays or polish	23
Washing machine tablets, detergent, or fabric conditioner; dishwasher tablets; or cleaning fluid	44
Cleaning or dusting without use of products	16
Home repair or improvement	
Painting	7
Drilling holes, sawing, assembling furniture, putting up plasterboard, moving furniture, or sanding	8
Fire for cooking or other purpose	
Bonfire	1
Electric burner	1
Gas burner	4
Outdoor gas stove	1
Wood or paper, burned or lighter used	5
Gardening	
Compost or "grow bags"	1
Fertilizer	2
Fertilizer or insect spray	7
Lawnmower, hedge trimmer, or generator fueled by gasoline	4
Gas smelled	1
Glue or filler	3
Laboratory or other solvent or sterilizing agent	6
Cat litter or birdseed	3
Photocopier, fax machine, or printer	41
Refueling	
Diesel	3
Unleaded gasoline	20
Artificial snow	1
Spray-on sunscreen	1
Anti-chlorine hair conditioner	2
Correction fluid	2
Visiting hair, tanning, or beauty salon	3
Other	12

home or at work. All the information reflected in Table 7 was collected in the screening questionnaire.

BEHAVIORAL INFORMATION

Table 8 shows the distribution of time that the subjects spent in each microenvironment. Table 9 and Table 10 present the distribution of activities performed by the subjects, and the products used in those activities, respectively, as reported in the activity questionnaires. Table 11 through Table 14 provide information about ETS events, as collected on the ETS questionnaire, location description sheet, and traveling description sheet.

PRESENTATION OF PERSONAL EXPOSURE AND MICROENVIRONMENTAL CONCENTRATION DATA

All personal exposure and microenvironmental concentrations had some instances of large GSDs. This may imply that the data encompassed more than one population of samples and that the populations had different modes.

In figures, the bars represent the GM of the VOC and PAH concentrations and the error bars represent the 95% confidence intervals calculated on a log scale. Unless otherwise specified, GMs are presented rather than arithmetic means, owing to the fact that the data are \log_{10} -normally distributed.

Table 11. Percentages of Subjects Exposed to ETS

Number of Cigarettes Smoked per Exposure	Within	
	≤ 2 m	> 2 m
0	24	56
1	23	4
2–5	28	10
6–10	5	4
11–20	3	2
21–50	4	1
51–100	2	1
Missing data	12	22

Table 12. Relationships of Subjects with Smokers Exposing Them to ETS

Smoker	% of Subjects
Friend or relative in my company	56
Person who was not in my company	25
Passerby	20

Table 13. Places Where Subjects Were Exposed to ETS

Location	% of Subjects Exposed
Outside in an open space	31
Private garden	8
Park	6
Street	47
Bus stop	11
Other	25
Missing data	3
Inside in an enclosed space	67
Restaurant, café, or tea room	4
Friend or relative's house	13
Home	24
Other	13
Pub, bar, or social club	44
Work or educational institute or office	1
Other	2

In the cases of sub-samples consisting of fewer than five valid data points, error bars are not presented in the graphs and results are not discussed, because of the low representation of the subsample for purposes of determining subsequent comparisons.

Data for the lower-molecular-weight PAHs (those of molecular weight < 200, i.e., acenaphthylene, acenaphthene, fluorene, phenanthrene, and anthracene) are presented but should be viewed with great caution. These compounds are present mainly in the vapor phase, but our measurement method accounts for only the amounts associated with particles. This amount is affected heavily by temperature, through the vapor-particle partition, and hence if samples are not collected under identical climate conditions, they will not be comparable. The same caveat applies to four-ring PAHs (those of molecular weight between 200 and 228; i.e., fluoranthene, pyrene, benzo[*a*]anthracene, and chrysene), albeit to a lesser degree. Five- and six-ring PAHs are largely associated with particles and therefore could be accurately measured using our methods.

PERSONAL EXPOSURES

VOC, 1,3-butadiene, and PAH samples were collected during the study to assess personal exposure. A summary of the VOC and PAH personal exposure data is presented in Table 15. Figures showing the results of personal exposure are given in Appendix 6, and the statistics summarizing personal exposure values are given in Appendix 7. Figure A6.1 and Figure A6.6 present the 24-hour GMs of

Table 14. Descriptions of Indoor Spaces Where Subjects Were Exposed to ETS

Location	% of Subjects Exposed
Level of smokiness	
Very smoky — people constantly smoking	7
Smoky — people frequently smoking	18
Slightly smoky — people occasionally smoking	60
Not smoky	15
Level of ventilation	
Adequate ventilation	15
Some ventilation	44
No ventilation	35
Don't know	6
Type of ventilation	
Open window	2
Open door	38
Fan	9
Ceiling fan	4
Air extractor	14
Air conditioning	3
Passive ventilation	0
Don't know	13

the personal exposure concentrations obtained, according to where the subjects lived (urban, suburban, or rural dwellings) for VOCs and PAHs, respectively. Figure A6.2 and Figure A6.7 show the patterns of behavior of the various groups of VOCs and PAHs, on the basis of the number of benzene rings, in each geographic location. The effect on personal exposure of living close to trafficked roadsides (in first-line homes) compared to living in homes located away from traffic (in non-first-line homes) is shown (with data expressed as GMs) in Figure A6.3 for VOCs and Figure A6.8 for PAHs.

Figure A6.4 and Figure A6.9 present the personal exposure measurements for VOCs and PAHs, respectively, associated with subjects living in homes with integral garages and subjects living in homes without integral garages. The effect of exposure to ETS on personal exposure concentrations is shown in Figure A6.5 for VOCs and Figure A6.10 for PAHs.

The effect of age on personal exposures is presented in Figure 1; age was categorized as 18–65 years old and ≥ 66 years old. Figure 2 and Figure 3 show the effects of occupational categories on personal exposure concentrations of VOCs and PAHs, respectively.

Table 15. Personal Exposure and Microenvironmental Concentrations of VOCs and PAHs

Compound	Personal Exposure		Home		Workplace		Street		Transport Vehicles		Transport Stations		Pubs and Restaurants		Other Indoor	
	GM	GSD	GM	GSD	GM	GSD	GM	GSD	GM	GSD	GM	GSD	GM	GSD	GM	GSD
<i>n</i> -Hexane	1.67	3.04	1.57	2.90	0.79	3.05	0.24	12.73	0.92	2.13	0.65	2.98	0.94	2.04	0.80	4.19
Benzene	1.64	2.01	1.50	2.07	1.05	1.77	1.05	2.83	1.84	2.11	1.43	2.36	1.94	2.78	1.21	2.02
Toluene	11.53	2.65	10.88	2.81	3.54	1.76	2.48	3.44	6.82	2.26	4.06	2.49	6.14	2.64	3.77	2.89
Ethylbenzene	1.47	2.72	1.17	2.49	0.85	2.67	0.47	3.30	1.13	2.13	0.66	2.40	1.03	2.57	0.69	2.68
<i>p</i> -Xylene	1.26	3.06	1.03	2.75	0.78	2.48	0.43	3.55	1.08	2.35	0.60	2.64	0.78	2.62	0.50	2.77
<i>m</i> -Xylene	3.23	3.00	2.55	2.75	1.97	2.89	0.99	4.05	2.88	2.25	1.55	2.69	2.27	2.90	1.34	2.70
Pyridine	0.15	2.56	0.12	1.99	0.10	1.96	0.03	2.56	0.05	1.57	0.05	2.16	0.89	5.52	0.07	1.75
<i>o</i> -Xylene	1.61	2.83	1.29	2.62	0.92	2.39	0.52	3.71	1.44	2.20	0.82	2.67	1.01	2.60	0.68	2.45
1,3,5-Trimethylbenzene	0.44	2.80	0.37	2.75	0.22	1.86	0.13	4.16	0.34	2.28	0.21	2.65	0.23	3.18	0.34	1.75
Styrene	0.63	2.38	0.58	2.21	0.48	2.04	0.11	2.77	0.43	2.19	0.18	2.11	0.56	2.87	0.38	3.52
<i>p</i> -Isopropyltoluene	0.80	2.28	0.79	2.18	0.33	2.77	0.00	15.92	0.11	5.91	0.00	20.80	0.78	2.76	0.22	2.21
1,2,4-Trimethylbenzene	1.57	2.94	1.30	2.96	0.78	2.08	0.41	4.51	1.34	2.14	0.78	2.40	0.96	2.89	1.21	1.47
3-Ethenylpyridine	0.10	3.95	0.05	2.81	0.03	2.68	0.02	3.26	0.06	1.94	0.05	2.93	1.16	6.36	0.04	1.61
Naphthalene	0.53	2.03	0.52	2.33	0.33	1.49	0.15	2.65	0.56	1.99	0.23	1.81	0.32	2.09	0.53	2.93
1,3-Butadiene	0.14	5.24	0.12	4.37	0.05	2.85	0.04	6.06	0.13	3.62	0.09	3.06	0.70	12.18	0.05	8.81
Acenaphthene	0.27	3.67	0.13	5.84	0.11	3.16	0.44	7.16	2.63	2.30	0.43	1.97	1.22	3.51	1.15	2.35
Fluorene	0.12	15.93	0.52	4.24	0.44	3.63	0.38	8.69	0.33	3.10	1.00	1.00	0.53	3.09	0.88	3.32
Phenanthrene	0.16	11.31	0.58	3.60	1.00	1.00	1.54	9.96	0.19	4.44	0.21	2.06	0.41	5.22	—	—
Anthracene	0.22	2.64	0.24	4.23	0.19	2.05	0.50	3.34	0.83	2.33	0.49	2.11	0.50	3.13	1.14	3.23
Fluoranthene	0.05	3.08	0.05	3.95	0.08	1.65	0.10	2.78	0.25	2.87	0.09	1.86	0.14	1.97	0.29	1.68
Pyrene	0.56	2.30	0.54	2.95	0.82	2.27	1.76	2.56	2.41	2.21	1.50	1.58	1.62	2.28	2.16	1.54
Benzo[<i>a</i>]anthracene	0.35	2.41	0.36	3.35	0.35	2.41	0.77	2.49	1.66	1.81	0.95	1.87	1.04	2.34	0.85	1.30
Chrysene	0.09	3.63	0.06	4.47	0.13	3.12	0.09	4.02	0.37	3.31	0.12	1.79	0.32	7.07	0.04	5.32
Benzo[<i>b</i>]fluoranthene	0.27	2.74	0.19	3.07	0.30	2.44	0.26	2.60	0.88	2.52	0.31	1.44	1.23	4.74	0.34	1.33
Benzo[<i>k</i>]fluoranthene	0.27	3.29	0.22	2.82	0.51	2.79	0.17	3.37	0.51	1.98	0.26	1.37	0.66	3.65	0.12	1.95
Benzo[<i>a</i>]pyrene	0.21	3.06	0.18	2.74	0.30	2.46	0.16	3.17	0.36	2.63	0.16	1.61	0.57	3.54	0.11	2.16
Indeno[1,2,3- <i>cd</i>]pyrene	0.13	3.65	0.09	4.03	0.16	3.22	0.13	2.52	0.28	3.31	0.12	1.98	0.37	5.13	0.05	2.98
Dibenz[<i>a,h</i>]anthracene	0.16	2.97	0.13	2.95	0.20	2.28	0.15	2.42	0.23	1.61	0.18	1.63	0.26	3.08	0.08	2.92
Benzo[<i>g,h,i</i>]perylene	0.02	5.49	0.02	5.58	0.02	9.45	0.02	3.96	0.02	4.01	0.01	2.06	0.11	5.47	—	—
Coronene	0.22	2.66	0.19	2.63	0.27	1.99	0.20	2.76	0.43	1.71	0.30	1.62	0.53	3.14	0.12	2.45

MICROENVIRONMENTAL CONCENTRATIONS

Subjects' Homes

A summary of VOC and PAH concentrations in the home microenvironment is shown in Table 15 (expressed as GMs and GSDs) and is presented diagrammatically in Appendix 6; the statistics summarizing home microenvironmental values are listed in Appendix 8.

Figure A6.11 and Figure A6.17 present the GM concentrations of VOCs and PAHs, respectively, measured in homes located in urban, suburban and rural environments; Figure A6.12 and Figure A6.18 present data concerning geographic location.

The effect of traffic on the levels of VOCs and PAHs in the home microenvironment is explored in Figure A6.13

and Figure A6.19. VOC and PAH concentrations are shown according to the presence or absence of an integral garage in Figure A6.14 and Figure A6.20, respectively, and according to the presence or absence of ETS exposure in Figure A6.15 and Figure A6.21, respectively.

The effect of daytime on VOC and PAH concentrations is presented in Figure A6.16 and Figure A6.22, respectively.

Homes Other than the Subjects'

Figures showing measurements obtained in microenvironments in homes other than the subjects' are presented in Appendix 6, with the summary statistics given in Appendix 9.

The GMs of VOC concentrations in various home microenvironments ranged from 0.04 $\mu\text{g}/\text{m}^3$ (for 3-ethenylpyridine)

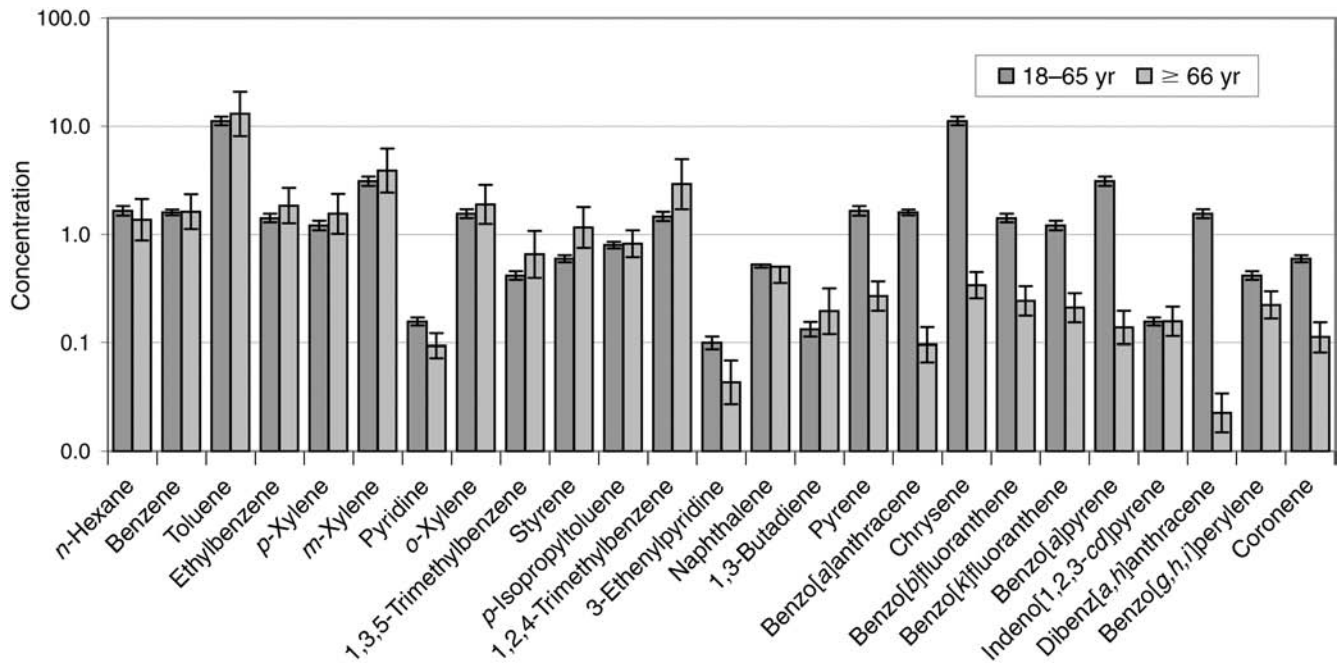


Figure 1. Personal exposure concentrations of VOCs (in $\mu\text{g}/\text{m}^3$) and PAHs (in ng/m^3), according to subject age. Data are presented as the geometric mean \pm 95% CI (error bars). For VOCs, $N = 448$ for 18–65 years of age and $N = 29$ for ≥ 66 years; for PAHs, $N = 59$ for 18–65 years and $N = 6$ for ≥ 66 years.

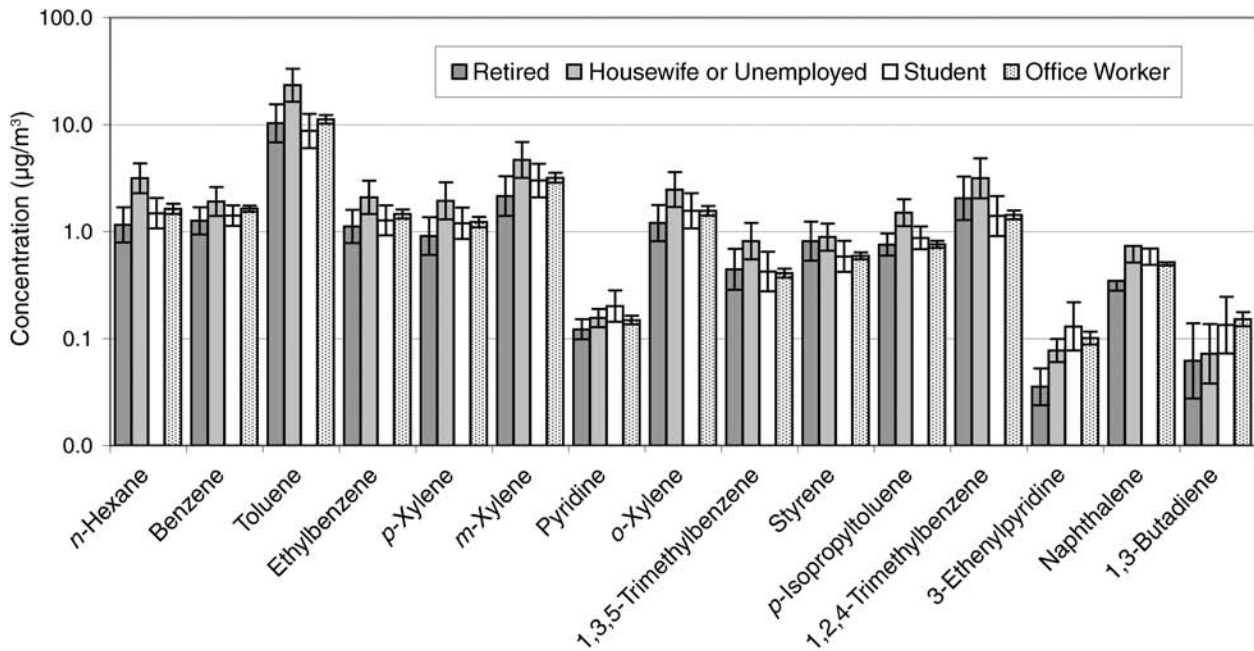


Figure 2. Personal exposure concentrations of VOCs (in $\mu\text{g}/\text{m}^3$), according to occupational category. Data are presented as the geometric mean \pm 95% CI (error bars). $N = 35$ for retired subjects, $N = 24$ for housewives or unemployed subjects, $N = 46$ for students, and $N = 372$ for office workers.

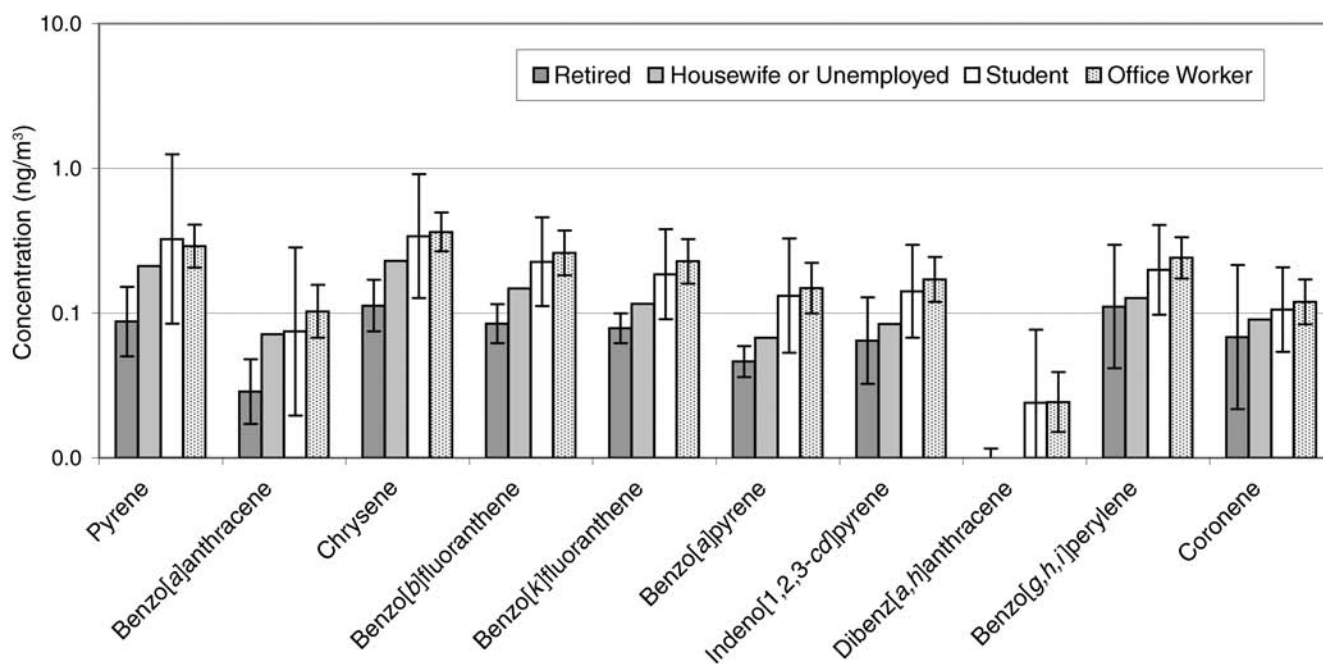


Figure 3. Personal exposure concentrations of PAHs (in ng/m^3), according to occupational category. Data are presented as the geometric mean \pm 95% CI (error bars). $N = 6$ for retired subjects, $N = 5$ for housewives or unemployed subjects, $N = 6$ for students, and $N = 50$ for office workers.

to $4.7 \mu\text{g}/\text{m}^3$ (for toluene). The compound with the second-highest concentration was *n*-hexane, with a GM of $0.8 \mu\text{g}/\text{m}^3$. The compounds with the next-lowest concentrations, after 3-ethenylpyridine, were pyridine and 1,3-butadiene, with GMs of $0.08 \mu\text{g}/\text{m}^3$ and $0.13 \mu\text{g}/\text{m}^3$, respectively. Figure A6.23 and Figure A6.28 show the VOC and PAH concentrations in the various home microenvironments measured. Figure A6.24 and Figure A6.29 compare VOC and PAH concentrations sampled in microenvironments in homes other than the subjects' in summer and in winter, and Figure A6.26 and Figure A6.31 show the results of concurrent measurements made indoors in the living room and outdoors in the backyard ($N = 6$) of houses located away from trafficked roads. Concurrent samples were also taken in living rooms and kitchens to observe the effect of cooking and other activities in the home (Figure A6.27 for VOCs and Figure A6.32 for PAHs). Figure A6.25 and Figure A6.30 present data for VOCs and PAHs, respectively, measured during the day as compared to during the night.

Workplaces

A summary of VOC and PAH concentrations measured in offices is presented in Table 15 as GMs and GSDs, are presented diagrammatically in Appendix 6, and the statistics summarizing work microenvironmental concentrations are accessible in Appendix 10.

Figure A6.33 and Figure A6.36 show the VOCs and PAH concentrations measured in each workplace-related microenvironment.

The effect of traffic on workplaces is examined by comparing offices located on trafficked roadsides (First-line Offices) with offices located away from traffic (Non-first-line Offices) in Figure A6.34 and Figure A6.37 and offices located in the city center with offices located in a suburban area (Figure A6.35 and Figure A6.38).

Streets

VOC and PAH concentrations in street microenvironments are presented as GMs and GSDs in Table 15, as well as diagrammatically in Appendix 6. The summary statistics of the street microenvironmental data are given in Appendix 11.

Figure A6.39 and Figure A6.44 show the VOC and PAH concentrations measured in various street microenvironments. Figure A6.40 analyzes the effect of traffic associated with the three geographic locations under study. The traffic effects were determined by comparing trafficked roadsides and background streets, as these microenvironments were measured in all three locations.

Samples collected in all types of street microenvironments in summer were compared with corresponding samples collected in winter, to analyze the seasonal effect on

VOC and PAH concentrations measured in the street (Figure A6.41 and Figure A6.45). The effect of traffic in streets was examined by comparing all samples collected during rush hour (7:00–9:00 a.m.) with samples collected during the middle of the day (1:00–3:00 p.m.) (Figure A6.42 for VOCs and Figure A6.57 for PAHs).

Transport Microenvironments

Table 15 shows the GMs and GSDs of the VOC and PAH concentrations measured in transport microenvironments (categorized as vehicles and stations), which are presented diagrammatically in Appendix 6. Summary statistics for vehicle (mobile) transport and stations are given in Appendix 12 and Appendix 13, respectively.

Figure A6.48 and Figure A6.52 present the VOC and PAH concentrations measured in the transport-vehicle microenvironments. Figure A6.49 and Figure A6.53 show the distribution of VOC and PAH concentrations among various types of transport stations.

Samples collected during the summer were plotted against samples collected in winter (Figure A6.50 for VOCs and Figure A6.54 for PAHs), to study the seasonal effect on transport microenvironments. Similarly, samples collected during rush hour were plotted against samples collected in the afternoon (Figure A6.51 and Figure A6.55), to observe any trend in the concentration data according to characteristic times of day.

Indoor Areas

Table 15 presents the GMs and GSDs of VOC and PAH concentrations measured in indoor environments (categorized as pubs and restaurants and as other indoor microenvironments). The data are also presented in figures in Appendix 6, and the summary statistics are given in Appendix 14.

Figure A6.57 and Figure A6.60 compare the VOC and PAH levels recorded in various indoor microenvironments. Samples collected in indoor environments during the summer were compared with corresponding samples collected in winter (Figure A6.58 and Figure A6.61), to analyze the seasonal effect on VOC concentrations measured indoors. Samples from pubs and restaurants were excluded from this analysis to avoid masking of the seasonal effect with the ETS effect.

Some restaurants and pubs already had a non-smoking policy before July 1, 2007. Thus, to assess the ETS effect, samples collected in pubs and restaurants where people were smoking were plotted alongside samples collected in ETS-free environments (Figure A6.59 and Figure A6.62). The ratio of measurements in winter and corresponding

Table 16. Arithmetic Ratios of Selected PAH Concentrations in Winter and Summer from Pubs and Restaurants

Compound	Pubs	Restaurants
Number of samples	20	16
Benzo[a]pyrene	13.0	3.5
Chrysene	12.8	1.9

measurements in summer are given in Table 16 for benzo[a]pyrene and chrysene, the latter being a possible PAH marker for exposure to ETS.

SUMMARY OF PERSONAL EXPOSURES AND MICROENVIRONMENTAL CONCENTRATIONS

Personal exposure and microenvironmental measurements are plotted side by side in Figure 4 and Figure 5 for VOCs and PAHs, respectively. Summary statistics are given in Table 15 and Appendix 15.

URINARY BIOMARKERS

Concurrent with collection of the air toxics samples, 500 urine samples (one for each day that each subject carried the sampling briefcase for personal exposure data collection) were obtained. Among these, 100 urine samples were analyzed for ETS and for urinary metabolites of PAHs. In all, 68 of the 100 urine samples were collected on the morning after the PAH sampling and 32 on a day not corresponding to the PAH sampling. Eight samples from five subjects were not included in the data analysis, as the cotinine values were extremely high, suggesting that these subjects smoked at some point before the urine collection. Three subjects confirmed that they had smoked on the day of PAH sampling; the other two subjects were unable to be contacted.

The statistics summarizing urinary biomarker concentrations and the Pearson coefficients from the correlation analysis performed on data from the log₁₀-transformed and nontransformed databases are presented in Appendix 16.

Correlations between the log₁₀-transformed concentrations of urinary biomarkers and concentrations of the respective PAH parent compounds and some VOCs present in ETS are given in Table 17. Table 18 shows the correlations between the biomarker concentrations and concentrations of other PAHs (non-parent compounds). Correlations that were nominally significant at the 0.05 level (two-tailed) are highlighted in bold type. The same correlation analysis was also performed on the nontransformed data (Appendix 16).

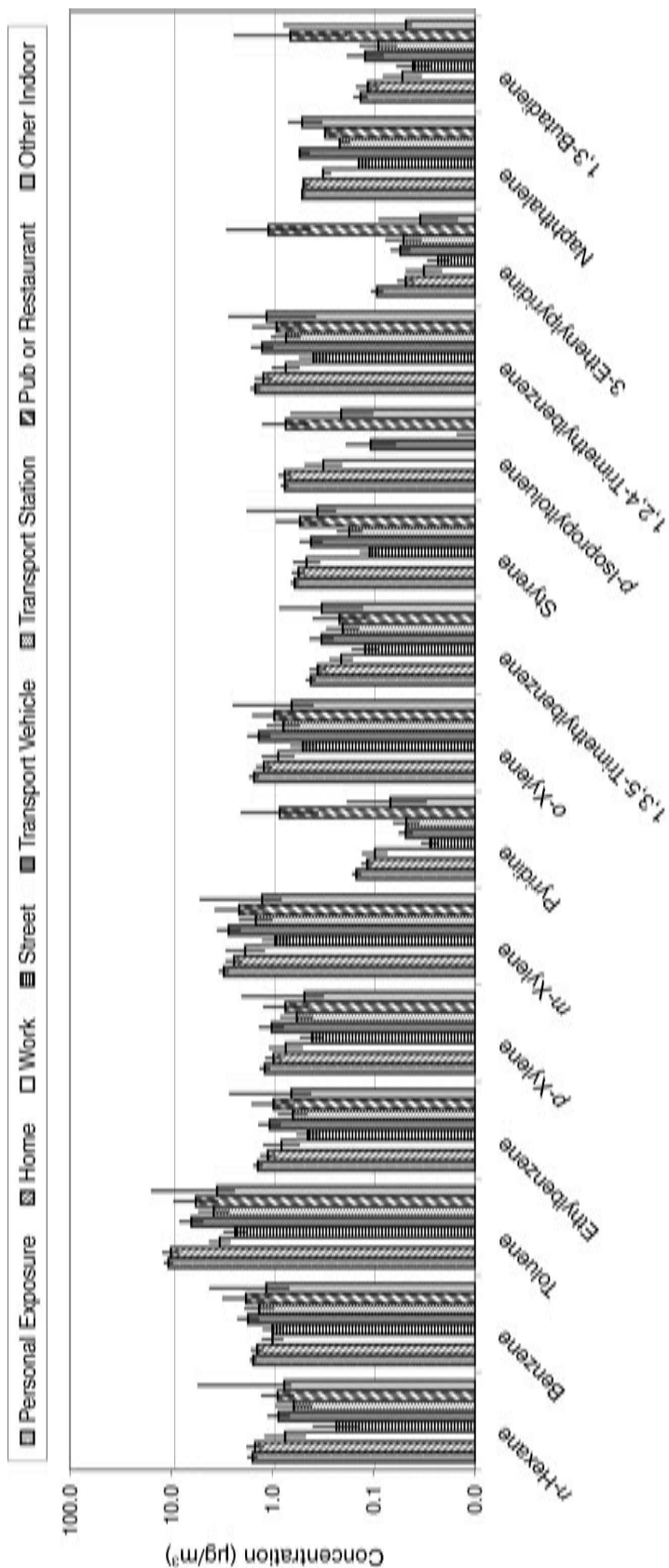


Figure 4. Personal exposure and microenvironmental concentrations of VOCs (in $\mu\text{g}/\text{m}^3$), according to exposure site. Data are presented as the geometric mean \pm 95% CI (error bars, with the bars absent if there were fewer than five data points). $N = 500$ for personal exposure, $N = 160$ for home, $N = 40$ for work, $N = 150$ for street, $N = 69$ for transport vehicle, $N = 53$ for transport station, $N = 24$ for pub or restaurant, and $N = 15$ for other indoor microenvironments.

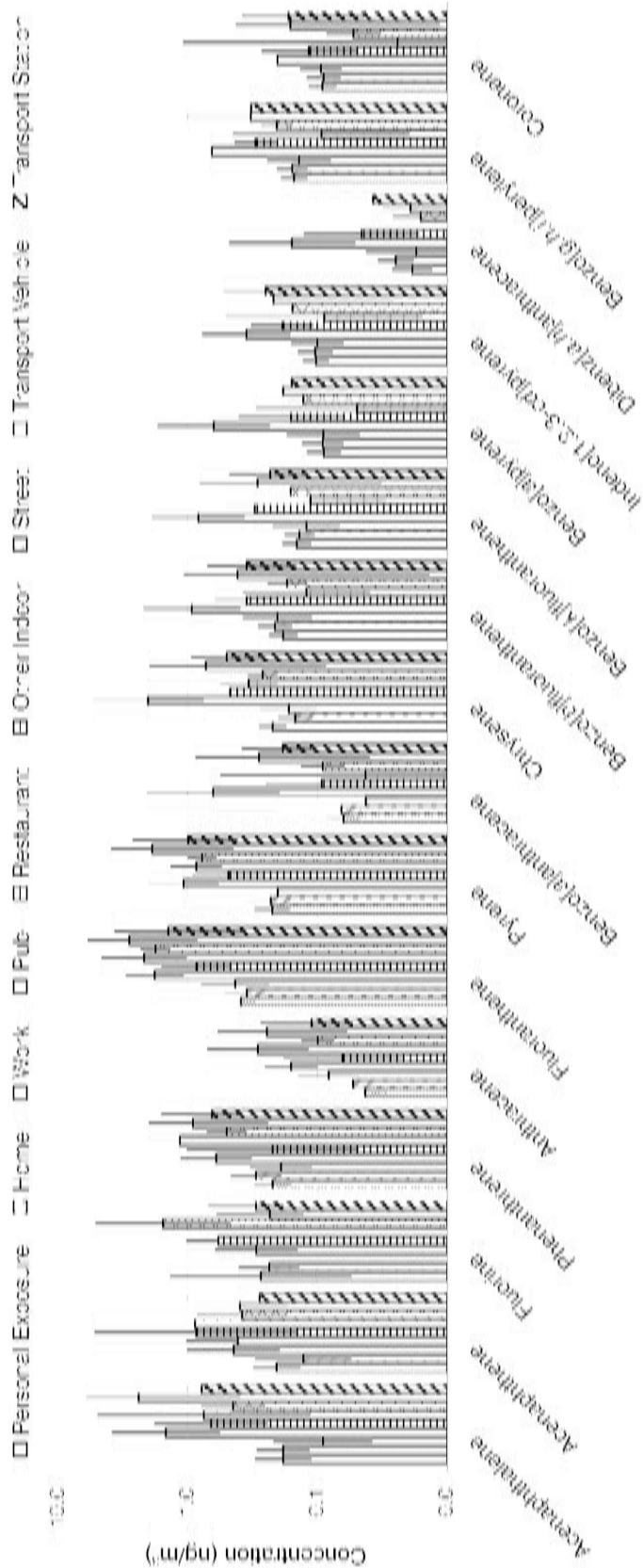


Figure 5. Personal exposure and microenvironmental concentrations of PAHs (in ng/m³), according to exposure site. Data are presented as the geometric mean \pm 95% CI (error bars). N = 100 for personal exposure, N = 132 for home, N = 36 for work, N = 11 for pub, N = 8 for restaurant, N = 4 for other indoor microenvironments, N = 57 for street, N = 25 for transport vehicle, and N = 18 for transport station.

Table 17. Pearson *R* Correlation Coefficients for Urinary Biomarkers and PAH Parent Compounds or VOCs Found in ETS (Log₁₀-Transformed Data)^a

Urinary Biomarker	Fluorene	Phenanthrene	Pyrene	3-Ethenyl- pyridine	Naphthalene (gas phase)	1,3-Butadiene
Cotinine	-0.23	0.11	0.07	0.75	0.05	0.47
Cotinine, creatinine corrected	-0.26	0.13	0.02	0.74	0.05	0.43
<i>trans</i> -3'-Hydroxycotinine	0.11	0.04	0.09	0.64	-0.05	0.44
<i>trans</i> -3'-Hydroxycotinine, creatinine corrected	0.07	0.09	0.03	0.68	-0.03	0.42
2-Naphthol	-0.73	-0.13	0.17	-0.09	0.03	0.07
2-Naphthol, creatinine corrected	-0.78	-0.06	0.10	-0.07	0.08	0.03
1-Hydroxyfluorene	— ^b	0.05	0.18	0.05	0.09	0.07
1-Hydroxyfluorene, creatinine corrected	— ^b	0.40	0.31	0.05	0.18	0.01
2-Hydroxyfluorene	-0.26	0.12	0.31	-0.02	0.00	0.15
2-Hydroxyfluorene, creatinine corrected	-0.33	0.22	0.24	0.12	0.12	0.10
3-Hydroxyfluorene	0.33	0.26	0.16	0.10	0.15	0.06
3-Hydroxyfluorene, creatinine corrected	0.05	0.29	0.04	0.11	0.12	-0.03
1-Hydroxyphenanthrene	0.53	-0.12	0.17	-0.05	-0.10	-0.03
1-Hydroxyphenanthrene, creatinine corrected	0.29	0.01	0.16	-0.02	-0.13	-0.09
2-Hydroxyphenanthrene	-0.05	-0.01	0.15	0.01	-0.20	0.00
2-Hydroxyphenanthrene, creatinine corrected	-0.07	0.09	0.22	0.08	-0.02	-0.06
3+4-Hydroxyphenanthrene	-0.13	0.22	0.30	-0.01	-0.07	0.05
3+4-Hydroxyphenanthrene, creatinine corrected	-0.11	0.24	0.21	0.09	-0.03	0.00
1-Hydroxypyrene	0.48	0.10	0.18	-0.08	-0.06	0.15
1-Hydroxypyrene, creatinine corrected	0.30	0.24	0.26	0.10	0.01	0.23

^a *N* = 92 for the VOCs and *N* = 68 for the PAH parent compounds. Correlations that were nominally significant at the 0.5 level (two-tailed) are given in bold type. "Creatinine corrected" refers to division by the creatinine level, to normalize the data.

^b Correlation could not be computed because there was only one sample.

Among all the biomarkers that were significantly correlated ($P < 0.05$) with selected VOCs or PAHs, those with Pearson correlation coefficients > 0.4 are shown in Figure 6.

The effect of ETS on the urinary biomarker concentrations was also investigated. Figure 7 shows the arithmetic mean concentrations and SDs of the ETS and PAH metabolites, the corresponding parent PAHs, and 3-ethenylpyridine, 1,3-butadiene, and naphthalene (in the gas phase) for the 92 urine samples (excluding the eight with the extremely high cotinine concentrations, typical of smokers), categorized as those from subjects exposed to ETS and those not exposed, on the basis of the measured personal exposures to 3-ethenylpyridine.

Normalizing data to the creatinine concentration is a common practice used to correct for differences in urinary flow. Nevertheless, various methods for creatinine correction have been suggested in the literature. To assess the effect of creatinine correction in urinary biomarker data, we calculated correlations for normalized biomarker data

(i.e., those for which the concentration was divided by creatinine levels) and non-normalized data (Table 17 and Table 18).

CORRELATIONS BETWEEN THE VOC AND PAH DATA SETS

To assess the relationships among various compounds a correlation analysis was performed using data from the VOC data set, the PAH data set, and the two data sets combined. Because the two data sets are log₁₀-normally distributed, Pearson correlation coefficients were calculated for both the linear and the log₁₀-transformed data. The correlation matrices are presented in Appendix 17.

SOURCE APPORTIONMENT

PCA was used for source apportionment in the VOC and PAH data sets. PCA identifies the various main factors that could be interpreted as the sources contributing to the final

Table 18. Pearson *R* Correlation Coefficients for Urinary Biomarkers and Non-Parent PAHs (Log₁₀-Transformed Data)^a

Urinary Biomarker	1	2	3	4	5	6	7	8	9	10
Benzo[<i>a</i>]anthracene	0.35	0.28	0.32	0.25	0.20	0.11	0.17	0.13	-0.12	0.00
Chrysene	0.55	0.44	0.52	0.43	0.30	0.15	0.28	0.21	0.11	0.12
Benzo[<i>b</i>]fluoranthene	0.42	0.36	0.38	0.34	0.15	0.06	0.17	0.18	0.10	0.20
Benzo[<i>k</i>]fluoranthene	0.45	0.38	0.42	0.38	0.16	0.08	0.26	0.21	0.15	0.19
Benzo[<i>a</i>]pyrene	0.38	0.30	0.36	0.29	0.22	0.10	0.28	0.23	0.14	0.18
Indeno[1,2,3- <i>cd</i>]pyrene	0.06	0.04	0.09	0.07	0.16	0.15	0.14	0.18	0.05	0.14
Dibenz[<i>a,h</i>]anthracene	0.37	0.29	0.32	0.25	0.08	-0.08	0.07	0.00	0.00	-0.12
Benzo[<i>g,h,i</i>]perylene	0.30	0.23	0.31	0.26	0.20	0.11	0.16	0.12	0.12	0.12
Coronene	0.23	0.19	0.21	0.19	0.01	-0.03	0.06	0.06	0.08	0.07
Combination of PAHs ^b										
Low molecular weight	0.17	0.16	0.11	0.14	-0.08	-0.06	0.08	0.18	0.08	0.12
Medium molecular weight	0.27	0.18	0.31	0.23	0.33	0.21	0.26	0.25	0.06	0.17
High molecular weight	0.37	0.28	0.37	0.31	0.24	0.13	0.21	0.15	0.15	0.09
16 PAH	0.26	0.17	0.31	0.22	0.24	0.10	0.25	0.24	0.13	0.20

^a *N* = 68 for the non-parent PAHs. Correlations that were nominally significant at the 0.5 level (two-tailed) are given in bold type. "Creatinine corrected" refers to division by the creatinine level, to normalize the data. The non-parent PAHs are indicated as 1 through 10, as follows, with the even number in each pair representing the data after division by the creatinine level, for normalization: 1 and 2, cotinine; 3 and 4, *trans*-3'-hydroxycotinine; 5 and 6, 2-naphthol; 7 and 8, 2-hydroxyfluorene; and 9 and 10, 1-hydroxypyrene.

^b Low-molecular-weight PAHs consisted of naphthalene, acenaphthylene, acenaphthene, fluorene, phenanthrene, and anthracene. Medium-molecular-weight PAHs consisted of fluoranthene, pyrene, benzo[*a*]anthracene, and chrysene. High-molecular-weight PAHs consisted of benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, benzo[*a*]pyrene, indeno[1,2,3-*cd*]pyrene, dibenz[*a,h*]anthracene, benzo[*g,h,i*]perylene, and coronene. The 16 PAHs consisted of naphthalene, acenaphthylene, acenaphthene, fluorene, phenanthrene, anthracene, fluoranthene, pyrene, benzo[*a*]anthracene, chrysene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, benzo[*a*]pyrene, indeno[1,2,3-*cd*]pyrene, dibenz[*a,h*]anthracene, and benzo[*g,h,i*]perylene.

concentration of a sample or common transport or meteorologic factors or common chemistries. PCA incorporating Varimax rotation was performed using SPSS for Windows (version 15.0). In the PAH data set, the first five components (acenaphthylene, acenaphthene, fluorene, phenanthrene, and anthracene) were not included in the factor analysis, as more than 20% of the data points were below the limit of detection.

PCA was performed on concentrations of all VOCs and PAHs detected in all samples collected for personal exposure. The factor-loading results of these analyses are shown in Table 19 for VOCs, Table 20 for PAHs, and Table 21 for the two combined.

As seen in Figure 8 and Figure 9 and in the results of the *t* test (Appendix 7), having an integral garage and being exposed to ETS has an important influence on personal exposure to VOC. Hence, to examine the relative contribution of these two sources, we conducted a factor analysis of concentration data from samples associated with ETS and integral garages and compared the results with those from all other types of samples (e.g., all ETS-related samples were compared with all non-ETS-related samples). The results of these analyses are shown in Table 19 for VOCs and Table 20 for PAHs.

DEVELOPMENT OF THE PERSONAL EXPOSURE MODEL

For the purposes of development of the personal exposure model, the data were split in two independent sets. The first set, containing 75% of the data, was used for "training" the model. The other set, containing the remaining 25% of the data, was used to test model performance.

Four different stages were then used in the development of the model to test which model best describes and predicts the personal exposure to VOCs and PAHs. Each model tested is described in the Statistical Methods and Data Analysis section above.

Briefly, the models tested were as follows. Model 1 considers the relationship between personal exposure and micro-environmental concentrations measured in each subject's home or workplace as described in equations 1.1 and 1.2.

Model 2 and model 3 predict the personal exposure by integrating the fractions of time spent in various micro-environments multiplied by the micro-environmental concentrations, as shown in equations 2 and 3, respectively. The difference between the two models lies in the micro-environmental concentrations used. Model 2 uses micro-environmental concentrations measured directly in the homes and workplaces of the subjects and uses pooled

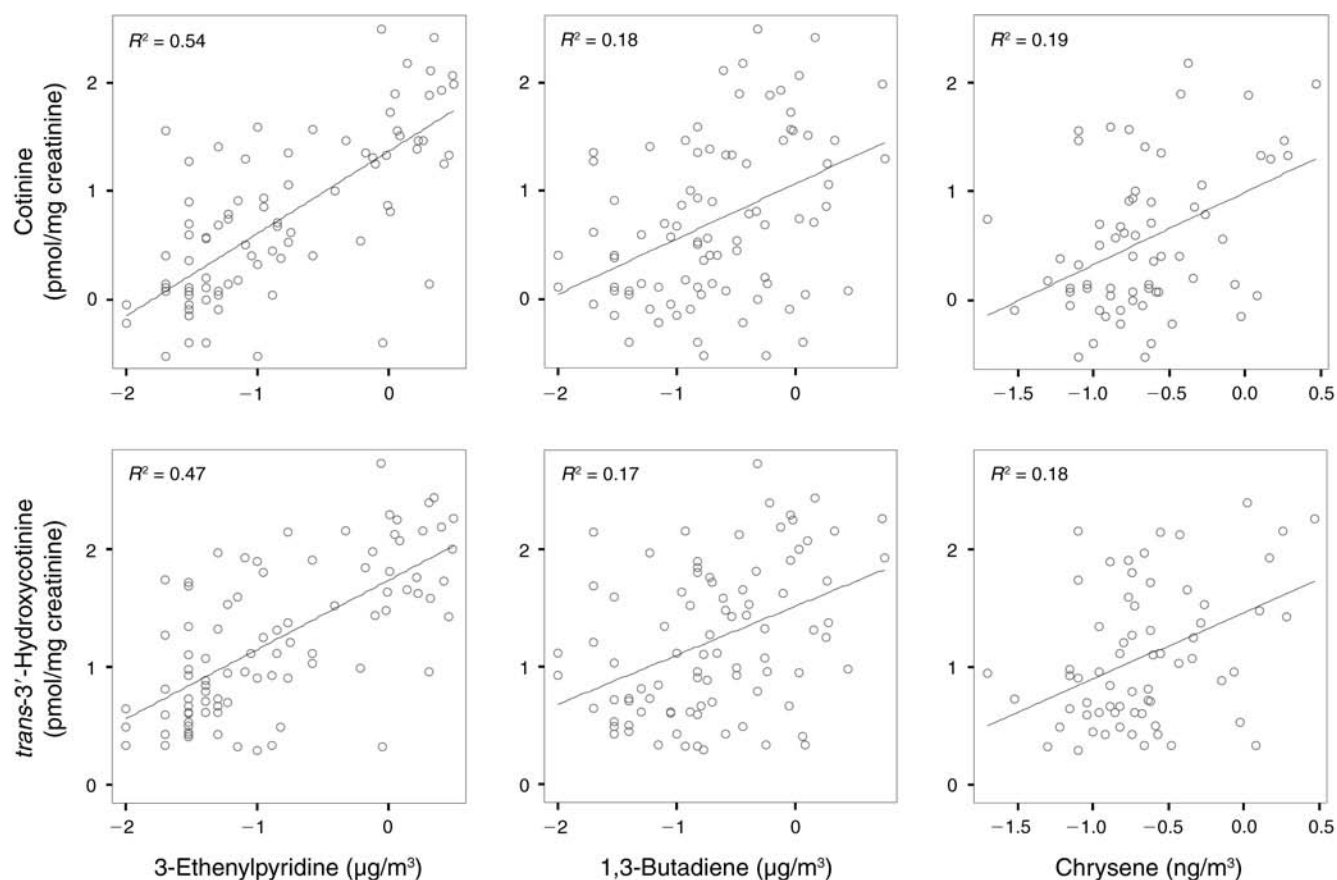


Figure 6. Significant correlations between urinary biomarkers and personal exposures to selected VOCs and a PAH. The figure shows some of the correlations for which the two-tailed P was < 0.05 and the Pearson correlation coefficients were > 0.4 . $N = 92$ for the VOC data (for 3-ethenylpyridine and 1,3-butadiene) and $N = 68$ for the PAH data (for chrysene).

data representative of each microenvironment for the rest of the microenvironments (street, transport and other indoor) as well as for the homes and workplaces not sampled. Model 3 uses pooled data segregated into various strata for each microenvironmental concentration instead of directly measured subject-specific microenvironmental concentrations. The pooled microenvironmental concentrations used in model 2 are presented in Table A19.1, and the pooled and stratified microenvironmental concentrations used in model 3 are presented in Table A19.2 and Table A19.3, of Appendix 19.

Model 4 predicts the personal concentrations by considering the personal exposure concentration calculated as in model 3 and incorporating add-on variables that explain external factors such as ETS events, home repair and improvement activities, and others (equation 4). Hence, model 4 used the same stratified concentrations as model 3 (Table A19.2 and Table A19.3 of Appendix 19).

Model 5 predicts the personal exposure by focusing on the key determinants proposed in the recruitment criteria

of this study, such as living in a first-line property, living in a house with an integral garage, being exposed to ETS, or living in an urban, suburban, or rural area.

Modeling of the lower-molecular-weight PAH compounds (acenaphthylene, acenaphthene, fluorene, phenanthrene, and anthracene) was not performed, since these compounds exist mainly in the vapor phase, and what is sampled on the filter is probably mainly an adsorption artifact of the gas-phase material rather than PAHs genuinely associated with particles.

Some outliers became immediately clear in the analysis of the training data set. These were characterized by extremely high concentrations, and although in some cases the sources were identified (e.g., use of fireplace or home repair and improvement), the cases could not be modeled using the information provided by the subjects. These results were excluded from the model development. Details of the outliers are given in Appendix 18.

Table 22 and Table 23 present the results of the model development as correlation coefficients and standard

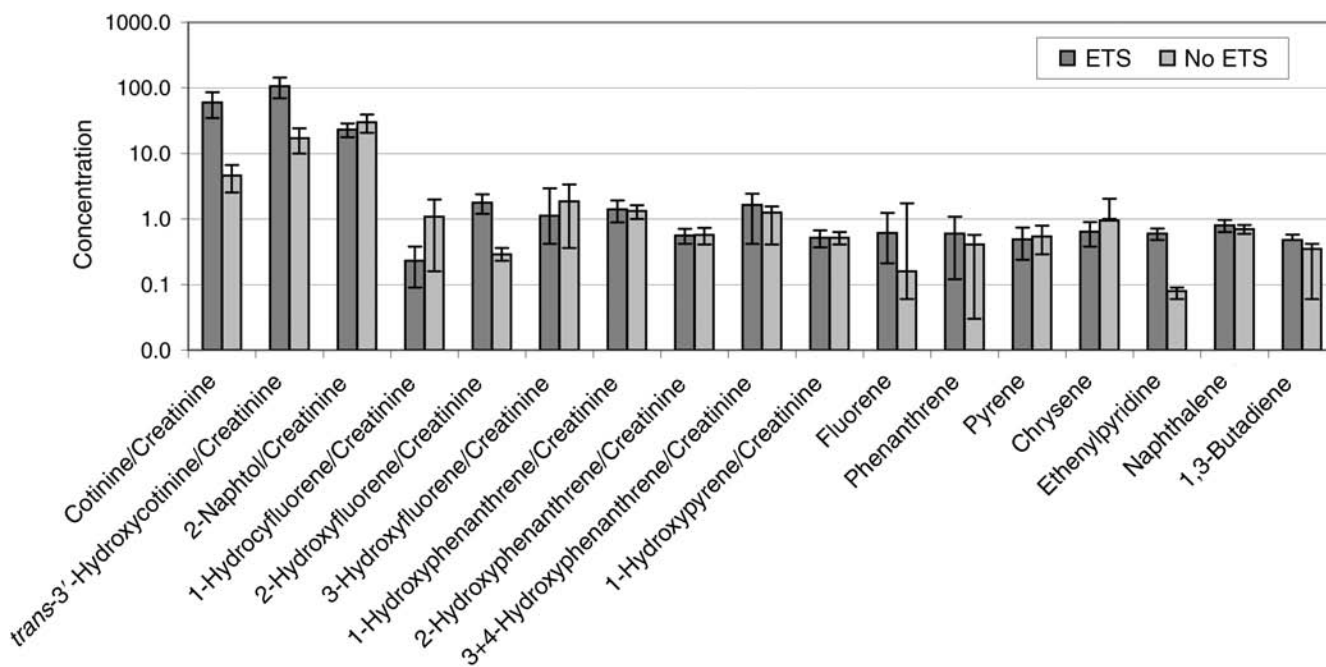


Figure 7. Personal exposure concentrations of urinary PAH metabolites and selected parent PAHs and VOCs, according to presence or absence of ETS exposure. The data are presented as the arithmetic mean \pm standard deviation (error bars). The urinary PAH biomarkers are expressed in relation to creatinine (pmol/mg creatinine), the PAHs (fluorene, phenanthrene, pyrene, and chrysene) are expressed in ng/m^3 , and the VOCs (ethynylpyridine, naphthalene, and 1,3-butadiene) are expressed in $\mu\text{g}/\text{m}^3$. For subjects with ETS exposure, $N = 31$ for urinary biomarkers and VOCs and $N = 16$ for PAHs; and for subjects without ETS exposure, $N = 61$ for urinary biomarkers and VOCs and $N = 52$ for PAHs.

errors, according to the models developed with the VOC and PAH training data sets, respectively. The scatterplots showing the correlations between the personal exposures predicted using the results of all the models versus the personal exposures measured in the training data set are presented in Appendix 20. The personal exposure database is \log_{10} -normally distributed and, as observed in the scatterplots for models 2 through 5, some models were driven by skewed distributions. A sensitivity analysis was performed, regressing the \log_{10} -transformed personal exposures measured against the \log_{10} -transformed personal exposures predicted in each of models 2 through 5. The results of this analysis are shown in Table 24 and Table 25.

The outcomes of model 4 are summarized in Table A21.1 and Table A21.2 of Appendix 21, for VOCs and PAHs, respectively. Table A21.6 presents the results of model 5 for VOCs and PAHs.

Detailed information for model 4, including the detailed list of add-on variables selected for each compound (A_m and F_n), as well as values of the nonstandardized-variable coefficients (β_m and γ_n) and standard errors, the standardized variable coefficient (β) and variable correlations (zero-order, partial, and semi-partial) are presented for all the VOCs in Table A21.4 and all the PAHs in Table A21.5. The

information contained in these tables is summarized in Table 26 for the compounds that are either carcinogenic to humans, representative of several other compounds (e.g., the *p*-xylene model is given as a representative of the similar xylene and ethylbenzene models), or related to ETS (e.g., 3-ethynylpyridine).

Figure 10A and Figure 11A show, for VOCs and PAHs, the correlation between personal exposures predicted using the results of model 4 and the measured personal exposures in the training data set. Solid lines represent the 1:1 relationship, and dashed lines represent the factor-of-two relationship.

The scatterplots showing the results of regression analysis for all the studied compounds and all the proposed models (1 through 5) are presented in Appendix 20.

VALIDATION OF THE PERSONAL EXPOSURE MODEL

According to Table 22, the model that best predicts the personal exposures in the training data set was the one developed using model 4. This model was tested in the independent-contrasts data set that contains 25% of the measured data.

Table 27 and Table 28 present, for VOCs and PAHs, respectively, the correlation coefficients between the predicted and measured values, the normalized mean bias,

Table 19. Source Apportionment in Personal Exposure: VOC Factor-Loading Coefficients, According to Key Determinant and Component Number

	All (N = 500)		ETS Exposure (n = 195)			No ETS Exposure (n = 305)			Integral Garage (n = 80)			No Integral Garage (n = 420)	
	1	2	1	2	3	1	2	3	1	2	3	1	2
Variance explained (%)	47.5	19.2	49.5	19.1	9.2	44.6	18.1	8.8	48.2	20.9	13.2	44.1	20.5
Compound													
<i>n</i> -Hexane	0.67		0.69			0.70			0.81			0.63	
Benzene	0.69		0.72	0.46		0.68			0.83			0.61	
Toluene	0.69		0.72			0.60			0.85			0.59	0.45
Ethylbenzene	0.94		0.96			0.93			0.94			0.94	
<i>p</i> -Xylene	0.95		0.95			0.96			0.88			0.96	
<i>m</i> -Xylene	0.95		0.96			0.96			0.90			0.95	
Pyridine		0.90		0.90			0.87				0.93		0.89
<i>o</i> -Xylene	0.96		0.97			0.96			0.93			0.96	
1,3,5-Trimethylbenzene	0.85		0.89			0.77	0.43		0.71	0.58		0.83	
Styrene	0.46	0.53		0.62	0.45	0.50	0.46			0.67		0.41	0.60
<i>p</i> -Isopropyltoluene		0.46			0.93		0.55			0.89			0.52
1,2,4-Trimethylbenzene	0.85		0.89			0.78			0.73	0.58		0.82	
3-Ethenylpyridine		0.85		0.89			0.75				0.93		0.83
Naphthalene	0.57		0.68			0.44	0.46		0.73			0.50	
1,3-Butadiene		0.42		0.55				0.92		0.61			0.45

Table 20. Source Apportionment in Personal Exposure: PAH Factor-Loading Coefficients, According to Key Determinant and Component Number

	All (N = 91)		ETS Exposure (n = 35)			No ETS Exposure (n = 56)			Integral Garage (n = 16)		No Integral Garage (n = 75)	
	1	2	1	2	3	1	2	3	1	2	1	2
Variance explained (%)	53.9	13.1	52.6	15.7	10.1	56.1	13.0	7.9	51.8	17.3	55.3	13.1
Compound												
Fluoranthene		0.93		0.85			0.93			0.91		0.91
Pyrene		0.94		0.87			0.94			0.88		0.92
Benzo[<i>a</i>]anthracene	0.65	0.48		0.79		0.56	0.63			0.59		0.76
Chrysene	0.65	0.42	0.66	0.47		0.52	0.62		0.72	0.60	0.58	0.55
Benzo[<i>b</i>]fluoranthene	0.82		0.85		0.40	0.78	0.37		0.93		0.45	0.75
Benzo[<i>k</i>]fluoranthene	0.87		0.84		0.44	0.93			0.92			0.85
Benzo[<i>a</i>]pyrene	0.76		0.40		0.56	0.78			0.68		0.59	0.60
Indeno[1,2,3- <i>cd</i>]pyrene	0.83		0.52	0.66		0.92			0.83		0.57	0.62
Dibenz[<i>a,h</i>]anthracene	0.76		0.55	0.68		0.72			0.88		0.66	0.43
Benzo[<i>g,h,i</i>]perylene	0.90		0.88			0.91			0.98		0.46	0.81
Coronene	0.74		0.94			0.58	0.67		0.83	0.40	0.61	0.53
1,3-Butadiene			0.83		0.79	0.24		0.52		0.51		0.60
3-Ethenylpyridine			0.70		0.82			0.87				0.62

Table 21. Source Apportionment in Personal Exposure: VOC and PAH Factor-Loading Coefficients ($N = 91$)

	Component			
	1	2	3	4
Variance explained (%)	33.7	29.0	10.3	8.7
Compound				
<i>n</i> -Hexane	0.59	0.44		
Benzene	0.87			
Toluene	0.62	0.47		
Ethylbenzene	0.79	0.46		
<i>p</i> -Xylene	0.74	0.51		
<i>m</i> -Xylene	0.79	0.49		
Pyridine			0.76	
<i>o</i> -Xylene	0.78	0.47		
1,3,5-Trimethylbenzene	0.69	0.52		
Styrene	0.53			0.43
<i>p</i> -Isopropyltoluene		0.51		0.41
1,2,4-Trimethylbenzene	0.67	0.54		
3-Ethenylpyridine	0.51		0.68	
Naphthalene	0.64			
1,3-Butadiene	0.49			0.56
Benzo[<i>a</i>]anthracene	0.65	-0.49		
Chrysene	0.78	-0.50		
Benzo[<i>b</i>]fluoranthene	0.76	-0.53		
Benzo[<i>k</i>]fluoranthene	0.77	-0.61		
Benzo[<i>a</i>]pyrene	0.78	-0.58		
Indeno[1,2,3- <i>cd</i>]pyrene	0.76	-0.60		
Dibenz[<i>a,h</i>]anthracene	0.75	-0.49		
Benzo[<i>g,h,i</i>]perylene	0.74	-0.62		
Coronene	0.66	-0.62		

the mean fractional bias, and the percentages of cases predicted within a factor of two and three. Figure 10B and Figure 11B show the correlation between the predicted and measured personal exposures to VOCs and PAHs, respectively, for the validation data set. Solid lines represent the 1:1 relationship, whereas dashed lines represent the factor-of-two relationship.

CATEGORIZATION OF LOW AND HIGH PERSONAL EXPOSURES

In order to analyze the performance of the proposed models in predicting personal exposure, we tested their ability to correctly categorize cases. The patterns in Figure 10 and 11 suggested that the measured and modeled personal exposures typically cluster into two groups: subjects with relatively low exposures (the main cluster) and subjects with higher exposures. On the basis of a visual assessment of the scatterplots, we estimated threshold exposures

that differentiate personal exposures into two main categories, low exposure and high exposure (see Table 29 for VOC thresholds and Table 30 for PAH thresholds). The thresholds proposed for benzene, 1,3-butadiene, and benzo[*a*]pyrene are those recommended in the guidelines of the U.K. Department for Environment, Food, and Rural Affairs (2003, 2006): 5 $\mu\text{g}/\text{m}^3$ for benzene, 2.25 $\mu\text{g}/\text{m}^3$ for 1,3-butadiene and 0.25 ng/m^3 for benzo[*a*]pyrene.

The percentages of subjects in the independent validation data sets that were correctly classified as having low or high exposures are given for VOCs in Table 29 and for PAHs in Table 30.

PERCENT CONTRIBUTIONS OF VARIOUS MICROENVIRONMENTS TO OVERALL PERSONAL EXPOSURES

The average contributions of various microenvironments to overall personal exposures to VOCs were calculated on the basis of the results of model 2. Model 2 was selected because it accounts for the largest proportion of variance for almost all the compounds, and it predicts personal exposure by integrating the fractions of time spent in various microenvironments and uses the concentrations directly measured within the subjects' homes and workplaces. Despite model 4 being able to explain a high proportion of variance, it was not considered for this analysis, because it predicts personal exposure to VOCs by taking into account the joint effect of various activities and microenvironments in personal exposure. This analysis could not be performed in the PAH data set, as the total variance explained by model 2 or model 3 was not significant.

The average exposure in microenvironment *K* to compound *Z*, and the total average exposure in all microenvironments, was calculated by integrating the individual values from all volunteers, all days, and all microenvironments (Appendix 23). Figure 12 presents the percent contribution of each microenvironment to personal exposure to VOCs, averaged across the studied population. The same analysis was performed for the subpopulation of ETS-exposed subjects (Figure 13).

DISCUSSION

STUDY POPULATION

A total of 100 subjects living in three locations across the United Kingdom were included in the study. In addition, there were several key determinants defined among the recruitment criteria for selection of the subjects, as shown in Table 1 (i.e., location, first-line property or not,

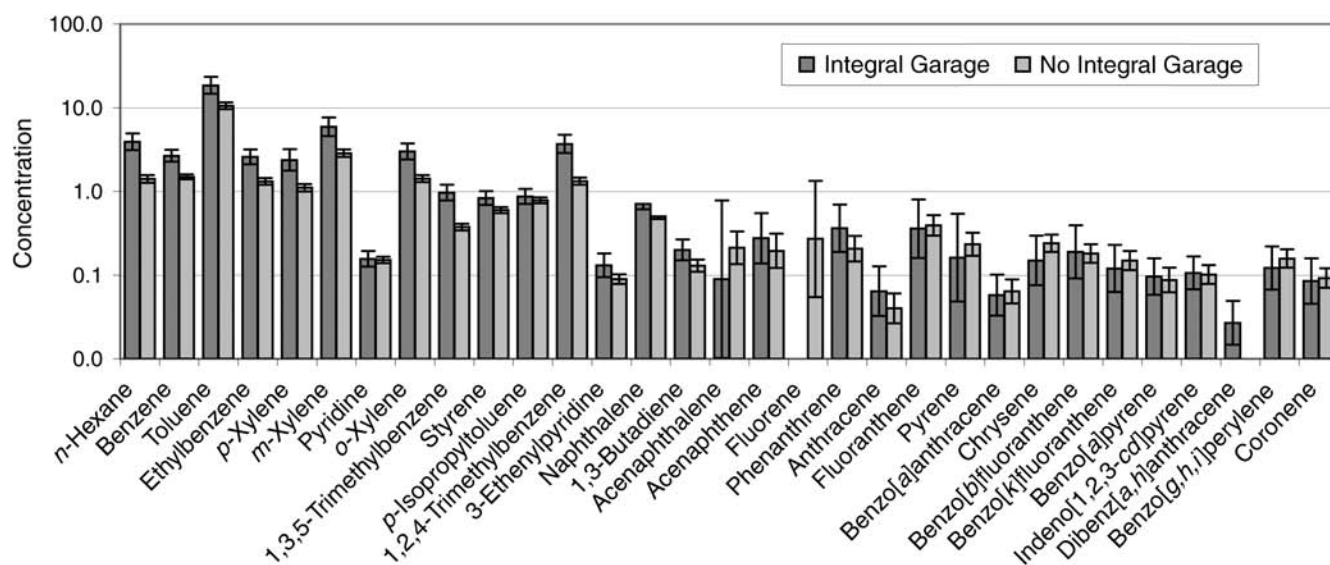


Figure 8. Personal exposure concentrations of VOCs (in $\mu\text{g}/\text{m}^3$) and PAHs (in ng/m^3), according to presence or absence of integral garage. Data are presented as the geometric mean \pm 95% CI (error bars). For VOCs, $N = 80$ for subjects with an integral garage and $N = 420$ for those without an integral garage; and for PAHs, $N = 16$ for those with an integral garage and $N = 75$ for those without.

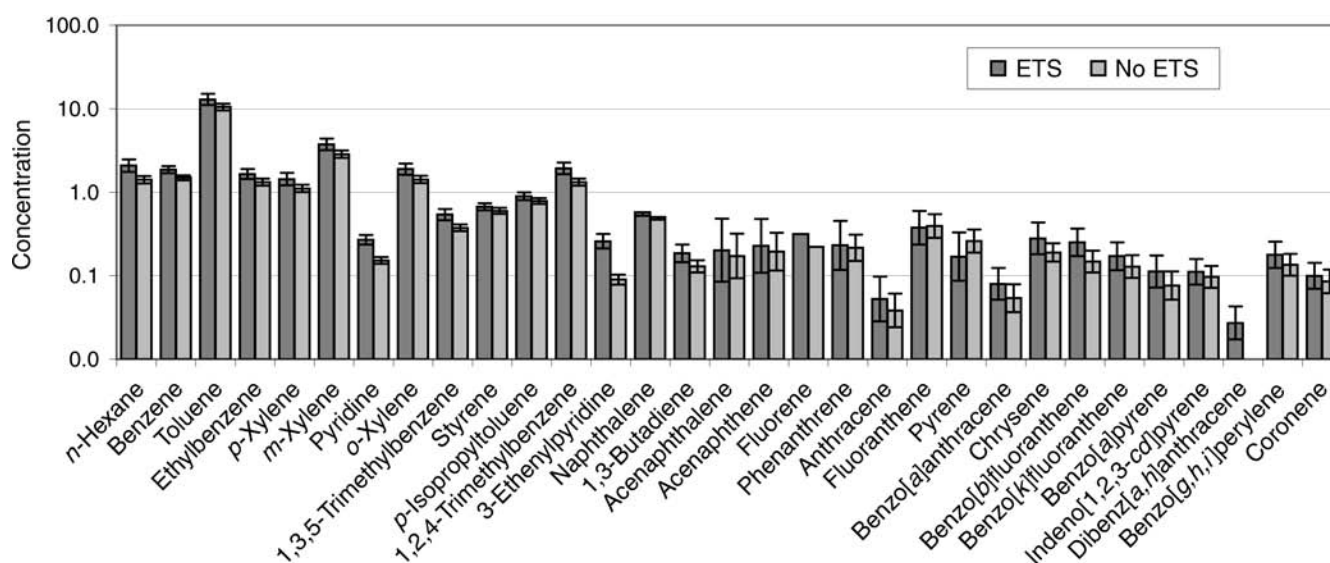


Figure 9. Personal exposure concentrations of VOCs (in $\mu\text{g}/\text{m}^3$) and PAHs (in ng/m^3), according to presence or absence of ETS exposure. Data are presented as the geometric mean \pm 95% CI (error bars). For VOCs, $N = 195$ for subjects with ETS exposure and $N = 305$ for subjects without ETS exposure; and for PAHs, $N = 35$ for subjects with ETS exposure and $N = 56$ for those without. No error bars are shown for fluorene because there are fewer than five data points.

Table 22. Correlation Coefficients and Standard Errors from the VOC Training Set, According to Proposed Model^a

VOC	Model 1											
	Home (<i>n</i> = 77)		Workplace (<i>n</i> = 40)		Model 2 (<i>n</i> = 375)		Model 3 (<i>n</i> = 375)		Model 4 (<i>n</i> = 375)		Model 5 (<i>n</i> = 375)	
	<i>R</i> ²	SE	<i>R</i> ²	SE	<i>R</i> ²	SE	<i>R</i> ²	SE	<i>R</i> ²	SE	<i>R</i> ²	SE
Hexane	0.331	1.95	0.243	2.24	0.513	1.24	0.017	1.13	0.385	2.85	0.126	3.38
Benzene	0.675	1.38	0.236	1.66	0.439	0.89	0.159	0.98	0.469	0.97	0.090	1.25
Toluene	0.666	1.62	0.288	2.04	0.525	11.3	0.112	9.81	0.513	12.31	0.020	17.17
Ethylbenzene	0.436	1.78	0.137	2	0.559	0.89	0.073	0.94	0.813	1.43	0.014	3.25
<i>p</i> -Xylene	0.497	1.78	0.239	2.04	0.631	0.91	0.126	1.08	0.819	1.42	NVE	NVE
<i>m</i> -Xylene	0.474	1.82	0.268	2.04	0.604	2.19	0.126	2.6	0.833	3.39	0.013	8.16
Pyridine	0.25	1.78	0.003	1.95	0.242	0.22	0.192	0.22	0.699	0.23	0.215	0.36
<i>o</i> -Xylene	0.502	1.74	0.184	2	0.63	0.98	0.147	1.34	0.832	1.67	0.015	4.46
1,3,5-Trimethylbenzene	0.693	1.7	0.124	2.34	0.617	0.33	0.197	0.33	0.788	1.24	0.041	2.61
Styrene	0.359	1.86	0.084	1.74	0.791	0.38	0.284	0.64	0.868	1.18	0.012	3.21
<i>p</i> -Isopropyltoluene	0.518	1.62	0.061	1.7	0.43	0.35	0.046	0.26	0.484	0.72	NVE	NVE
1,2,4-Trimethylbenzene	0.731	1.7	0.159	2.34	0.667	1.23	0.315	1.28	0.862	3.31	0.047	7.42
3-Ethenylpyridine	0.26	2.24	0	2.4	0.304	0.26	0.264	0.25	0.750	0.34	0.274	0.59
Naphthalene	0.478	1.58	0.215	1.7	0.798	0.38	0.128	0.47	0.418	0.9	0.054	1.16
1,3-Butadiene	0.266	2.14	0.015	2.04	0.121	0.28	0.077	0.41	0.487	0.39	0.097	0.50

^a The model 1 data are dimensionless; the data for models 2 through 5 are expressed in $\mu\text{g}/\text{m}^3$. Bold values indicate a significant correlation at the 0.01 level. NVE indicates no value entered in the stepwise regression.

Table 23. Correlation Coefficients and Standard Errors from the PAH Training Set, According to Proposed Model^a

PAH	Model 1											
	Home (<i>n</i> = 36)		Workplace (<i>n</i> = 20)		Model 2 (<i>n</i> = 68)		Model 3 (<i>n</i> = 68)		Model 4 (<i>n</i> = 68)		Model 5 (<i>n</i> = 68)	
	<i>R</i> ²	SE	<i>R</i> ²	SE	<i>R</i> ²	SE	<i>R</i> ²	SE	<i>R</i> ²	SE	<i>R</i> ²	SE
Pyrene	0.226	3.16	0.002	3.39	0.066	0.47	0.147	0.42	0.247	0.44	0.061	0.68
Benzo[<i>a</i>]anthracene	0.184	2.88	0.009	2.34	0.024	0.19	0.015	0.19	0.661	0.27	0.068	1.89
Chrysene	0.361	2.04	0.191	2.09	0.006	0.40	0.049	0.39	0.334	0.17	0.056	2.32
Benzo[<i>b</i>]fluoranthene	0.121	2.82	0.502	2.40	0.000	0.28	0.023	0.28	0.278	0.26	0.054	2.61
Benzo[<i>k</i>]fluoranthene	0.023	2.29	0.131	2.09	0.003	0.25	0.022	0.25	0.303	0.23	0.059	0.29
Benzo[<i>a</i>]pyrene	0.107	2.75	0.194	2.45	0.000	0.23	0.040	0.22	0.346	0.20	0.063	2.68
Indeno[1,2,3- <i>cd</i>]pyrene	0.068	2.51	0.307	2.24	0.000	0.23	0.032	0.22	0.282	0.21	0.060	2.01
Dibenz[<i>a,h</i>]anthracene	0.434	2.34	0.009	2.04	0.003	0.04	0.042	0.04	0.575	0.03	0.091	0.16
Benzo[<i>g,h,i</i>]perylene	0.076	2.51	0.378	2.14	0.000	0.33	0.084	0.31	0.259	0.30	0.063	1.80
Coronene	0.008	2.51	0.250	2.04	0.001	0.18	0.101	0.16	0.367	0.15	0.059	1.08

^a The model 1 data are dimensionless; the data for models 2 through 5 are expressed as ng/m^3 . Bold values indicate a significant correlation at the 0.01 level.

Table 24. Model Evolution Measured as Correlation Coefficient and Standard Error from the Log₁₀-Transformed VOC Training Set^a

VOC	Model 2 (n = 375)		Model 3 (n = 375)		Model 4 (n = 375)		Model 5 (n = 375)	
	R ²	SE	R ²	SE	R ²	SE	R ²	SE
Hexane	0.401	0.24	0.025	0.22	0.289	0.19	0.127	0.16
Benzene	0.392	0.17	0.120	0.18	0.354	0.14	0.130	0.08
Toluene	0.496	0.26	0.114	0.26	0.288	0.20	0.005	0.06
Ethylbenzene	0.457	0.20	0.078	0.21	0.450	0.18	—	—
<i>p</i> -Xylene	0.470	0.21	0.096	0.23	0.461	0.19	0.018	0.09
<i>m</i> -Xylene	0.422	0.23	0.115	0.22	0.412	0.20	NVE	NVE
Pyridine	0.427	0.24	0.263	0.27	0.283	0.23	0.221	0.24
<i>o</i> -Xylene	0.468	0.21	0.127	0.22	0.462	0.19	0.017	0.10
1,3,5-Trimethylbenzene	0.601	0.21	0.161	0.24	0.302	0.38	0.030	0.17
Styrene	0.464	0.19	0.105	0.21	0.317	0.18	0.001	0.14
<i>p</i> -Isopropyltoluene	0.439	0.19	0.048	0.15	0.253	0.15	NVE	NVE
1,2,4-Trimethylbenzene	0.599	0.22	0.196	0.25	0.386	0.30	0.116	0.17
3-Ethenylpyridine	0.506	0.34	0.387	0.36	0.322	0.35	0.330	0.39
Naphthalene	0.511	0.18	0.092	0.17	0.282	0.19	0.008	0.14
1,3-Butadiene	0.132	0.42	0.036	0.33	0.288	0.26	0.122	0.21

^a Bold values indicate a significant correlation at the 0.01 level. NVE indicates no value entered in the stepwise regression.

Table 25. Model Evolution Measured as Correlation Coefficient and Standard Error from the Log₁₀-Transformed PAH Training Set^a

PAH	Model 2 (n = 68)		Model 3 (n = 68)		Model 4 (n = 68)		Model 5 (n = 68)	
	R ²	SE	R ²	SE	R ²	SE	R ²	SE
Pyrene	0.036	0.10	0.181	0.20	0.267	0.24	0.030	0.13
Benzo[<i>a</i>]anthracene	0.013	0.13	0.058	0.31	0.242	0.18	0.034	0.41
Chrysene	0.002	0.13	0.115	0.24	0.419	0.24	0.029	0.26
Benzo[<i>b</i>]fluoranthene	0.001	0.11	0.018	0.25	0.170	0.32	0.011	0.31
Benzo[<i>k</i>]fluoranthene	0.021	0.11	0.010	0.26	0.126	0.34	0.017	0.38
Benzo[<i>a</i>]pyrene	0.016	0.12	0.045	0.27	0.228	0.20	0.016	0.21
Indeno[1,2,3- <i>cd</i>]pyrene	0.016	0.10	0.024	0.17	0.078	0.24	0.017	0.38
Dibenz[<i>a,h</i>]anthracene	0.011	0.14	0.059	0.28	0.418	0.21	0.048	0.26
Benzo[<i>g,h,i</i>]perylene	0.008	0.10	0.067	0.16	0.084	0.31	0.013	0.31
Coronene	0.004	0.10	0.049	0.14	0.175	0.31	0.110	0.29

^a Bold values indicate a significant correlation at the 0.01 level.

Table 26. Details of Model 4 for VOCs and PAHs

Model, Add-On Variables ^a	Nonstandardized Coefficients		Standardized Beta	t	P Value	Correlation Coefficients		
	β	SE				Zero-Order	Partial	Semi-Partial
Benzene (R² = 0.469)								
(Constant)	0.609	0.196		3.108	0.002			
Benzene modeled	0.140	0.082	0.096	1.696	0.091	0.316	0.093	0.068
Storage_of_paints_in_garage	1.905	0.210	0.405	9.082	0.000	0.464	0.447	0.364
Storage_of_car_in_garage	1.049	0.215	0.219	4.889	0.000	0.382	0.260	0.196
Urban	0.380	0.133	0.140	2.854	0.005	-0.094	0.155	0.114
Time_Const_Freq_ETS	0.003	0.001	0.279	5.994	0.000	0.285	0.313	0.240
Visited_hospital	1.320	0.316	0.170	4.171	0.000	0.183	0.223	0.167
Use_all_trains	0.350	0.179	0.083	1.962	0.051	0.023	0.107	0.079
Time_car	0.005	0.001	0.153	3.597	0.000	0.243	0.194	0.144
Gas_main_heating	0.455	0.123	0.172	3.714	0.000	0.032	0.200	0.149
Additional_other_heating	2.087	0.668	0.148	3.122	0.002	0.175	0.169	0.125
p-Xylene (R² = 0.819)								
(Constant)	0.620	0.184		3.374	0.001			
p-Xylene modeled	0.423	0.150	0.095	2.814	0.005	0.180	0.152	0.066
Storage_of_lawn_mower_in_garage	1.760	0.337	0.131	5.222	0.000	0.200	0.275	0.122
Storage_of_car_in_garage	2.157	0.321	0.179	6.717	0.000	0.205	0.345	0.157
Time_paint	0.092	0.003	0.828	35.487	0.000	0.818	0.889	0.828
Visited_hospital	2.028	0.460	0.103	4.409	0.000	0.091	0.235	0.103
Carpet_fumigated	5.857	0.828	0.165	7.072	0.000	0.141	0.361	0.165
Additional_other_heating	2.510	1.157	0.071	2.169	0.031	0.113	0.118	0.051
Work_factory	5.552	0.828	0.157	6.707	0.000	0.134	0.345	0.156
1,2,4-Trimethylbenzene (R² = 0.862)								
(Constant)	-0.138	0.280		-0.494	0.621			
1,2,4-Trimethylbenzene modeled	0.716	0.154	0.100	4.639	0.000	0.115	0.246	0.094
Storage_of_car_in_garage	4.164	0.625	0.151	6.665	0.000	0.191	0.343	0.135
Time_direct_paint	0.416	0.011	0.822	37.466	0.000	0.873	0.899	0.758
Kitchen_garage	4.337	0.709	0.135	6.113	0.000	0.177	0.317	0.124
Use_gas_cooker	0.642	0.081	0.173	7.879	0.000	0.465	0.396	0.159
Work_factory	3.902	1.645	0.048	2.372	0.018	0.025	0.129	0.048
3-Ethenylpyridine (R² = 0.750)								
(Constant)	0.109	0.022		4.912	0.000			
3-Ethenylpyridine modeled	-0.337	0.052	-0.318	-6.472	0.000	0.370	-0.333	-0.172
ETS	0.178	0.051	0.116	3.482	0.001	0.468	0.187	0.092
Time_ETS	0.001	0.000	0.227	3.970	0.000	0.761	0.212	0.105
Time_constant_ETS	0.003	0.000	0.215	5.204	0.000	0.711	0.273	0.138
Time_const_freq_ETS	0.004	0.000	0.647	9.808	0.000	0.777	0.472	0.260
1,3-Butadiene (R² = 0.487)								
(Constant)	0.115	0.036		3.232	0.001			
Solvent_use	0.112	0.135	0.034	0.834	0.405	0.117	0.046	0.033
Time_ETS	0.001	0.000	0.149	1.969	0.050	0.389	0.108	0.077
Time_const_freq_ETS	0.001	0.000	0.236	3.150	0.002	0.375	0.171	0.124
Petrol_station	0.178	0.106	0.068	1.678	0.094	0.107	0.092	0.066
Use_bus	0.108	0.070	0.062	1.538	0.125	0.094	0.084	0.060
Time_traveling	0.001	0.000	0.059	1.458	0.146	0.042	0.080	0.057

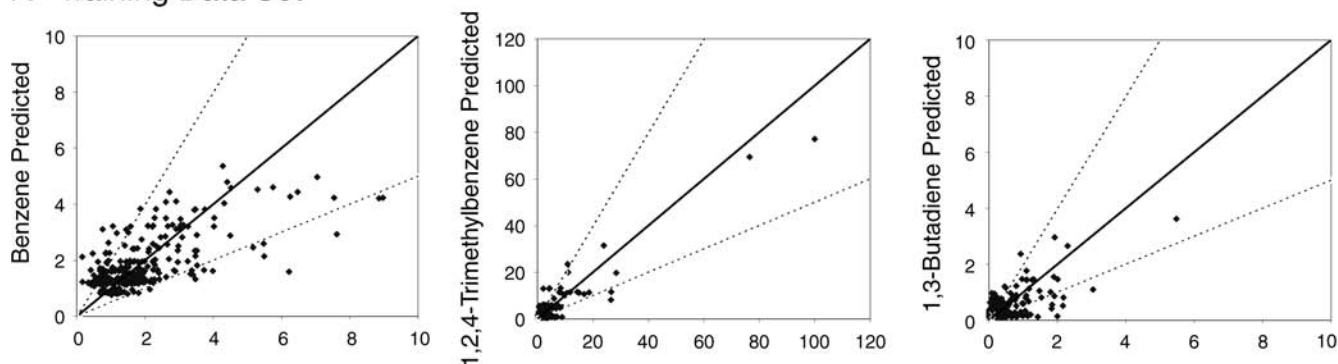
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Table 26 (Continued). Details of Model 4 for VOCs and PAHs

Model, Add-On Variables ^a	Nonstandardized Coefficients		Standardized Beta	<i>t</i>	<i>P</i> Value	Correlation Coefficients		
	β	SE				Zero-Order	Partial	Semi-Partial
1,3-Butadiene ($R^2 = 0.487$) (Continued)								
Kitchen_garage	0.401	0.091	0.176	4.403	0.000	0.145	0.235	0.173
Additional_gas_heating	0.666	0.112	0.238	5.969	0.000	0.198	0.312	0.234
Time_wrapping_presents	0.015	0.001	0.431	10.685	0.000	0.448	0.506	0.419
Inv_time_since_antimold	2.001	0.338	0.241	5.925	0.000	0.247	0.310	0.232
Benzo[a]anthracene ($R^2 = 0.661$)								
(Constant)	0.300	0.149		2.015	0.049			
Benzo[a]anthracene modeled	-0.096	0.417	-0.059	-0.230	0.819	0.400	-0.031	-0.018
Summer	-0.229	0.080	-0.258	-2.874	0.006	-0.341	-0.364	-0.228
Urban	0.211	0.073	0.241	2.893	0.005	0.115	0.366	0.229
No_of_cig_within_2m	0.109	0.016	0.561	6.856	0.000	0.655	0.682	0.543
Use_bus	0.209	0.113	0.151	1.840	0.071	0.096	0.243	0.146
Not_cooker_hood	-0.287	0.175	-0.141	-1.642	0.106	-0.029	-0.218	-0.130
Use_gas_cooker	-0.004	0.018	-0.018	-0.206	0.838	-0.143	-0.028	-0.016
Time_frequent_ETS	0.002	0.001	0.409	1.584	0.119	0.442	0.211	0.125
Benzo[a]pyrene ($R^2 = 0.346$)								
(Constant)	0.167	0.070		2.396	0.020			
Benzo[a]pyrene modeled	0.162	0.292	0.062	0.555	0.582	0.109	0.075	0.061
Summer	-0.125	0.059	-0.260	-2.122	0.038	-0.353	-0.277	-0.233
Car_sometimes_garage	0.373	0.146	0.286	2.557	0.013	0.323	0.329	0.281
No_of_cig_within_2m	0.036	0.012	0.355	3.150	0.003	0.412	0.394	0.347
Not_cooker_hood	-0.113	0.121	-0.106	-0.938	0.352	-0.082	-0.127	-0.103
Use_gas_cooker	0.000	0.013	-0.001	-0.006	0.996	-0.141	-0.001	-0.001
Dibenz[a,h]anthracene ($R^2 = 0.575$)								
(Constant)	0.038	0.010		3.725	0.001			
Dibenz[a,h]anthracene modeled	0.076	0.178	0.045	0.426	0.672	0.213	0.064	0.042
Summer	-0.027	0.010	-0.303	-2.759	0.008	-0.432	-0.384	-0.271
No_of_cig_within_2m	0.010	0.002	0.560	5.543	0.000	0.651	0.641	0.545
ETS_home	0.031	0.016	0.209	1.872	0.068	0.243	0.272	0.184
Not_cooker_hood	-0.021	0.022	-0.094	-0.941	0.352	-0.060	-0.141	-0.093
Use_gas_cooker	-0.001	0.002	-0.077	-0.681	0.499	-0.176	-0.102	-0.067

^a Explanation of the add-on variables A_m and F_n is given in Appendix 21.

A Training Data Set



B Validation Data Set

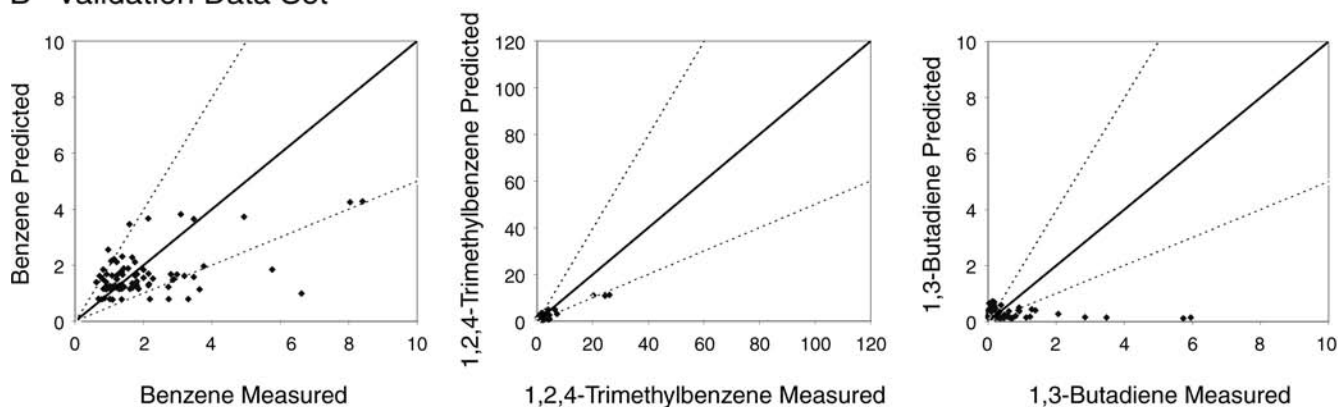


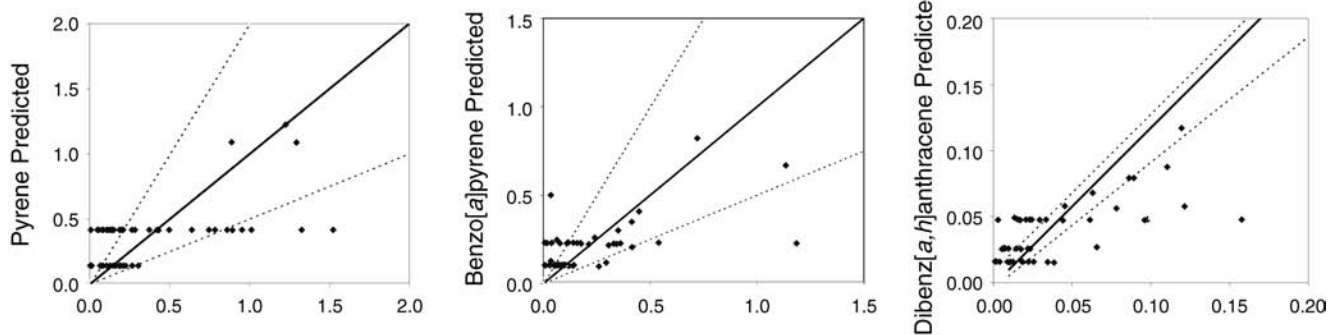
Figure 10. VOC concentrations (in $\mu\text{g}/\text{m}^3$) predicted with model 4 vs. the concentration measured, for the training data set (A) and the validation data set (B). The training set contained data for 375 subjects, and the validation set contained data for 125 subjects. The solid lines represent the 1:1 relationship, and the pairs of dashed lines represent the factor-of-two relationship.

and presence or absence of integral garage and ETS exposure). Most of the recruitment criteria were met. However, some problems were encountered in recruiting subjects living in homes with integral garages, with only 16 subjects meeting the criterion even though the original proposal was for between 20 and 30 subjects. Few subjects were exposed to ETS at home in urban or suburban homes and none were exposed to ETS at home in a rural dwelling. However, 34 subjects were exposed to ETS either at home, in a pub, or while socializing in other indoor environments. This fact resulted in a different proportion of subjects fulfilling each key determinant in each type of location (i.e., urban, suburban, and rural) and also a different distribution of key determinants met in London, West Midlands, and Wales. Table 31 shows that, although the integral garages were evenly distributed between suburban and rural areas (present for approximately 25% of the sampled population in each area), these structures will not be a source of exposures for urban subjects. In addition, within rural areas, integral garages were more frequent among Welsh subjects

than among West Midlands subjects (with 30% and 20%, respectively, of the integral garages within rural areas). Exposure to ETS was reported by a greater proportion of suburban subjects (43%) than urban subjects (32%) or rural subjects (20%). With respect to the distribution of ETS-exposed subjects by geographic location, West Midlands was the area with the highest percentage of subjects exposed to ETS (38% of the sampled population), as compared with London (27%) or Wales (only 10%). Finally, the proportion of subjects living in first-line properties was quite evenly distributed among all the types of location: around 40% in both urban and suburban areas and 50% in rural areas. With regard to the distribution within each geographic area studied, homes were first-line properties for a majority of Welsh subjects (60%) but for lesser proportions of subjects in London (45%) and West Midlands (42%).

The recruitment and sampling of subjects was done in various seasons. Each subject was sampled on five consecutive days in only one season. Sampling was not planned to occur in both warm and cold seasons for each subject, as

A Training Data Set



B Validation Data Set

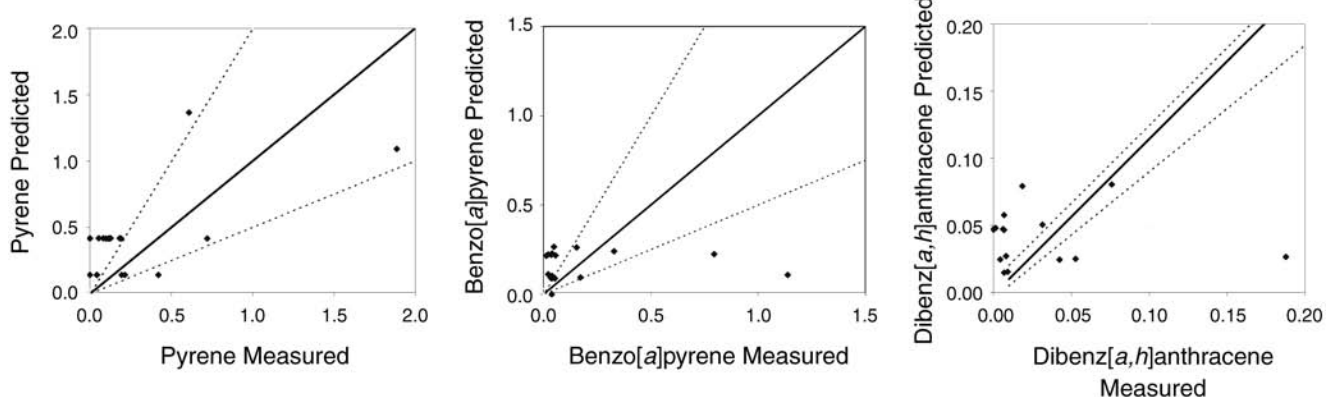


Figure 11. PAH concentrations (in ng/m^3) predicted with model 4 vs. the concentration measured, for the training data set (A) and the validation data set (B). The training set contained data for 68 subjects, and the validation set contained data for 23 subjects. The solid lines represent the 1:1 relationship, and the pairs of dashed lines represent the factor-of-two relationship.

this would have considerably increased the sample size. Table 32 shows the percentages of subjects sampled in each season, according to geographic location, type of location, and key determinants.

These “snapshot” measurements cannot address temporal and seasonal variation. Hence, the differences in sampling season and in the sources affecting subjects in different locations and different cities may have repercussions for the comparison of sub-samples (see the Personal Exposure Levels section below).

With regard to home characteristics, all the urban subjects lived in flats except one subject in London, who lived in a house. All the flats in London were built before 1991, whereas the flats in Birmingham were predominantly new homes built after 1991. Suburban subjects lived mainly in old houses, which had higher incidences of redecoration in the previous year than other dwellings. Very few suburban subjects lived in flats. All the rural subjects lived in houses, most of which had been built before 1991. Natural gas was the predominant main heating fuel in homes, except in Birmingham city center, where it was electricity,

and in some rural homes, which used other fuels such as kerosene. The main fuel used for cooking was also natural gas, though in most homes in Birmingham city center and in rural West Midlands, electricity was used. Most subjects reported not using a stovetop hood during cooking, although some did use one (principally in urban and suburban Birmingham).

As regards the demographic distribution of the study subjects, although there was no intended bias in recruitment, a majority were female (Table 7). The age range of the subjects was very wide, with about half between 26 and 35 years old. Few were 66 years of age or older. The main occupation of the subjects was administration or office worker. The majority of subjects spent less than 15 minutes commuting to the workplace. ETS exposure at home and work was reported by only 12 and 8 subjects, respectively.

BEHAVIORAL INFORMATION

Analysis of the data for time spent in each microenvironment (Table 8) showed that all the subjects in all locations

Table 27. Summary Statistics for the Validation of the Personal Exposure Model Using the VOC Validation Set ($N = 125$)^a

VOC	Pearson <i>R</i>	Normalized Mean Bias (%)	Mean Fractional Bias (%)	Factor of 2 (%)	Factor of 3 (%)
Hexane	0.69	-25	44	43	61
Benzene	0.66	-20	-8	80	92
Toluene	0.73	-7	14	61	83
Ethylbenzene	0.80	-10	6	73	87
<i>p</i> -Xylene	0.79	-15	11	69	90
<i>m</i> -Xylene	0.72	-23	4	66	87
Pyridine	0.81	-6	25	63	75
<i>o</i> -Xylene	0.79	14	41	56	84
1,3,5-Trimethylbenzene	0.84	-22	-24	49	72
Styrene	0.97	8	16	57	84
<i>p</i> -Isopropyltoluene	0.60	-6	13	76	91
1,2,4-Trimethylbenzene	0.91	-28	-7	60	83
3-Ethenylpyridine	0.78	-39	16	42	62
Naphthalene	0.24	17	23	64	84
1,3-Butadiene	0.29	-40	27	31	49

^a Bold values indicate a significant correlation at the 0.01 level.

spent more time indoors than outdoors or commuting. Among all locations, subjects spent 87–91% of their time indoors, with subjects living in London spending the least time at home (54%) and the most time at work (27%) and subjects living in the rural West Midlands spending the most time at home (67%) and the least time at work (7%), mainly because some of them were retired. Overall, subjects spent very little time outdoors, with the rural volunteers spending less time outdoors (2%) than the suburban dwellers (4%) and the urban dwellers (5%). On the other

hand, rural subjects spent more time commuting (7% of their time) than did suburban subjects (6%) or urban subjects (5%).

The most frequent activities performed by the subjects were associated with housekeeping (cleaning and vacuuming), personal hygiene (aerosol or perfume use), refueling, and use of a photocopier or printer (Table 9). Activities that had clear repercussions for personal exposure were burning of candles or incense, fireplace use, and home repair and improvement. These frequently performed

Table 28. Summary Statistics for Validation of the Personal Exposure Model Using the PAH Validation Set ($N = 23$)^a

PAH	Pearson <i>R</i>	Normalized Mean Bias (%)	Mean Fractional Bias (%)	Factor of 2 (%)	Factor of 3 (%)
Pyrene	0.93	66	91	19	38
Benzo[<i>a</i>]anthracene	0.51	177	108	19	38
Chrysene	0.27	-51	-15	62	86
Benzo[<i>b</i>]fluoranthene	0.60	-50	-80	14	33
Benzo[<i>k</i>]fluoranthene	0.43	63	147	14	38
Benzo[<i>a</i>]pyrene	0.43	-22	148	19	29
Indeno[1,2,3- <i>cd</i>]pyrene	0.38	314	-47	19	29
Dibenz[<i>a,h</i>]anthracene	0.51	25	326	19	29
Benzo[<i>g,h,i</i>]perylene	0.35	-39	-26	24	38
Coronene	0.62	-27	185	24	33

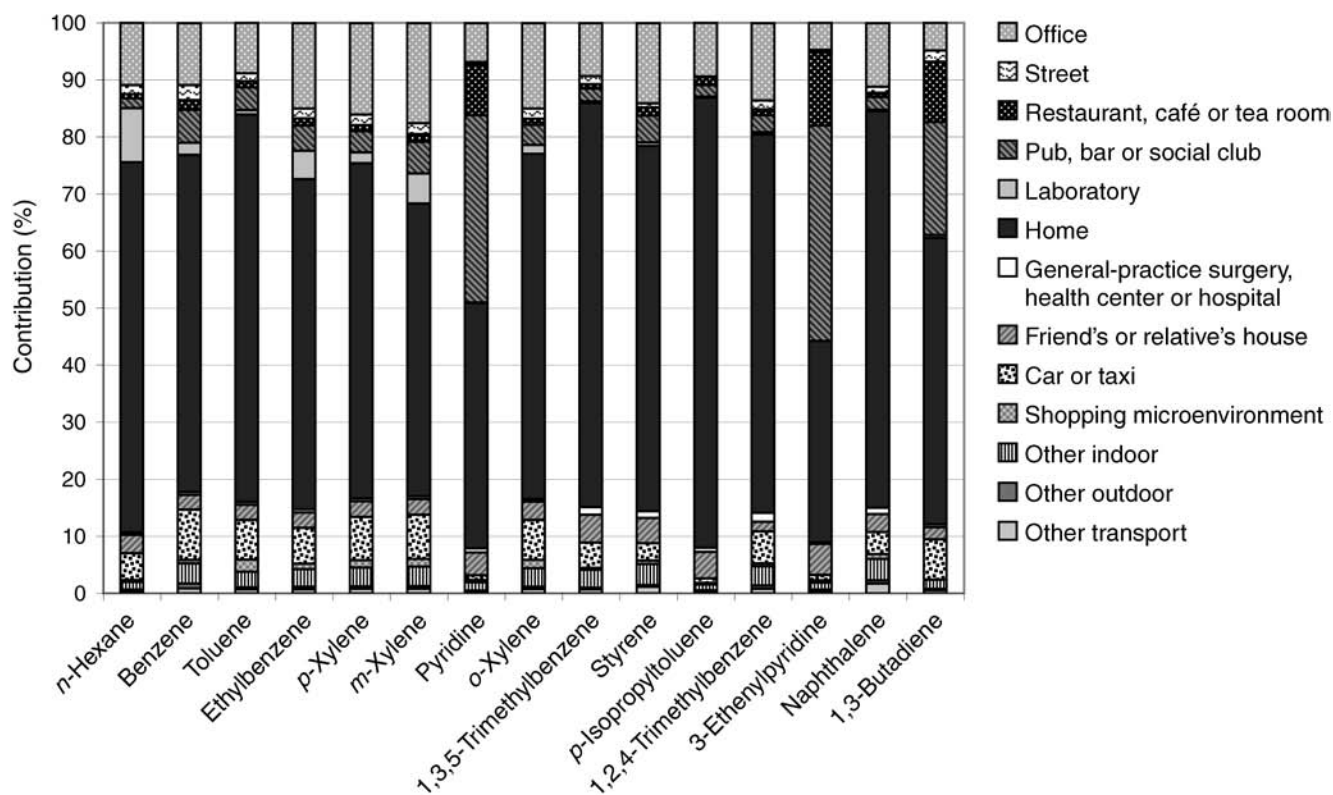
^a Bold value indicates a significant correlation at the 0.01 level.

Table 29. Percentage of Cases Correctly Classified According to the Proposed Threshold Value, for the VOC Validation Set ($N = 125$)

VOC	Exposure Categorization Threshold Value ($\mu\text{g}/\text{m}^3$)	% Cases Correctly Classified	
		Low Exposure	High Exposure
<i>n</i> -Hexane	5	95	58
Benzene	5	100	11
Toluene	30	100	27
Ethylbenzene	6	100	25
<i>p</i> -Xylene	5	98	50
<i>m</i> -Xylene	10	100	33
Pyridine	0.4	100	59
<i>o</i> -Xylene	5	98	58
1,3,5-Trimethylbenzene	2	97	50
Styrene	5	100	40
<i>p</i> -Isopropyltoluene	4	100	0
1,2,4-Trimethylbenzene	5	100	38
3-Ethenylpyridine	0.5	100	42
Naphthalene	4	100	0
1,3-Butadiene	2.25	100	0

Table 30. Percentage of Cases Correctly Classified According to the Proposed Threshold Value, for the PAH Validation Set ($N = 23$)

PAH	Exposure Categorization Threshold Value (ng/m^3)	% Cases Correctly Classified	
		Low Exposure	High Exposure
Pyrene	0.50	100	67
Benzo[<i>a</i>]anthracene	0.50	95	0
Chrysene	0.20	85	63
Benzo[<i>b</i>]fluoranthene	0.60	100	0
Benzo[<i>k</i>]fluoranthene	0.60	89	0
Benzo[<i>a</i>]pyrene	0.25	89	0
Indeno[1,2,3- <i>cd</i>]pyrene	0.50	90	0
Dibenz[<i>a,h</i>]anthracene	0.05	83	33
Benzo[<i>g,h,i</i>]perylene	0.50	100	0
Coronene	0.30	100	0

**Figure 12.** Percent contributions of various microenvironments to VOC personal exposures ($N = 375$).

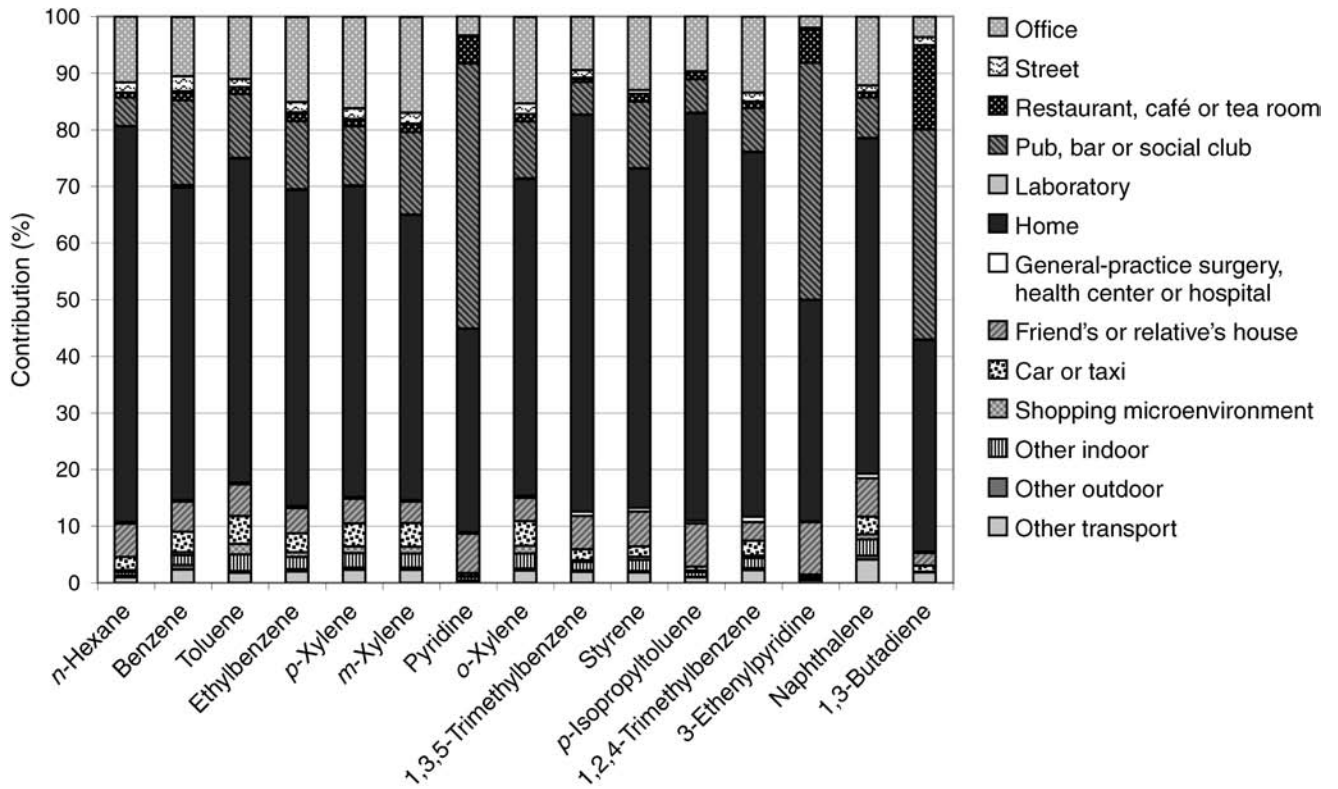


Figure 13. Percent contributions of various microenvironments to VOC personal exposures in the ETS-exposed population (N = 127).

Table 31. Percentage of Subjects Exposed to Each Key Determinant in Each Geographic Location and the Total Percentages, According to Type of Location

Type of Location ^a	Integral Garage (%)				ETS Exposure (%)				First-Line Properties (%)			
	London	West Midlands	Wales	All	London	West Midlands	Wales	All	London	West Midlands	Wales	All
Urban	0.0	0.0		0.0	27.3	33.3		31.6	45.5	40.7		42.1
Suburban		26.2		26.2		42.9		42.9		42.9		42.9
Rural		20.0	30.0	25.0		30.0	10.0	20.0		40.0	60.0	50.0
All	0.0	16.5	30.0	16.0	27.3	38.0	10.0	34.0	45.5	41.8	60.0	44.0

^a For urban subjects, the numbers of subjects living in London, West Midlands, Wales, and All was 11, 27, 0, and 38, respectively. For suburban subjects, the numbers of subjects living in London, West Midlands, Wales, and All was 0, 42, 0, and 42, respectively. For rural subjects, the numbers of subjects living in London, West Midlands, Wales, and All was 0, 10, 10, and 20, respectively. For all subjects, the numbers of subjects living in London, West Midlands, Wales, and All was 11, 79, 10, and 100, respectively.

Table 32. Percentage of Subjects Sampled Each Season, According to Geographic Location, Type of Location, and Key Determinant

Location or Determinant	Spring (%)	Summer (%)	Autumn (%)	Winter (%)
West Midlands	16	25	28	30
London	100	0	0	0
Wales	0	0	40	60
Urban	32	21	24	24
Suburban	26	21	26	26
Rural	5	15	30	50
Integral garage	25	13	19	44
ETS	26	18	23	33
First-line property	20	16	39	25

activities are also reflected in the use of products by the subjects (Table 10), the most frequent being deodorants or perfumes, cleaning products, photocopiers or printers, and refueling. Products used in home repair and improvement, gardening, candle or incense burning, and fireplaces were also used frequently.

As regards ETS exposure (Tables 11–14), most of the subjects who were exposed to between one and five cigarettes were less than 2 m away from the smoker, who was a friend or relative with whom they were spending time in 56% of the cases. Subjects were mainly exposed to ETS indoors (67%), the pub being the most common place (44%) followed by the home (24%) and friend or relative's house (13%). (Legislation outlawing smoking in public places came into force in England and Wales in July 2007, too late to influence this study.) People exposed to ETS outdoors were mainly exposed in the street by a passer-by or a person not in their company, in most cases. Although most subjects exposed to ETS indoors were less than 2 m away from the ETS source, most of them reported that the place was just slightly smoky. Regarding the level of ventilation, there was an even distribution between places which were not ventilated and those with some ventilation, mainly from open doors and air extractors.

PERSONAL EXPOSURES

VOCs Including 1,3-Butadiene

The VOC personal exposure levels observed in this study (Table 33; also see detailed statistics in Appendix 7 and summary statistics in Appendix 15) are significantly lower than those found in similar studies conducted previously in various locations in the United States and Europe

(Wallace 1986; Carrer et al. 2000; Gonzalez-Flesca et al. 2000; Hoffmann et al. 2000).

The VOC concentrations measured in EXPOLIS-Helsinki (Edwards et al. 2001b) are higher than those reported in the present study, for almost all the compounds studied except styrene. On the other hand, the values reported for EXPOLIS-Oxford (Lai et al. 2004) show mean values for ethylbenzene and the xylenes that are similar to those reported here but higher mean concentrations for benzene, toluene, trimethylbenzenes, and *n*-hexane. In addition, the results of a study performed in Hanover, Germany (Ilgen et al. 2001c) gave values of ethylbenzene and xylenes similar to ours, but the levels of toluene and benzene were nonetheless higher than ours. On the other hand, the Population Exposure to Air Pollutants in Europe (PEOPLE) study (Pérez Ballesta et al. 2006) found higher benzene levels than those in our study, except for people who spent most of their time at home.

A study conducted in Birmingham, United Kingdom, in 1998 (Leung and Harrison 1998) reported higher concentrations than in this study for all compounds except for *o*-xylene. In a later study, Kim and colleagues (2002) reported concentrations higher than ours for benzene, toluene and *n*-hexane but similar to ours for all the other compounds. However, studies carried out in Minneapolis in 2000–2001 (Adgate et al. 2004a) and in Oklahoma in 1999–2000 (Phillips et al. 2005) found median VOC concentrations for personal exposure similar to those reported here.

A rough comparison of the Total Exposure Assessment Methodology (TEAM) and similar studies and more recent data suggests that personal exposure to benzene and other VOCs has decreased since the 1980s (HEI Air Toxics Review Panel 2007). The decrease in exposure to VOCs, as exemplified by benzene (Figure 14), especially exposures from traffic, is consistent with the sharp downward trends in airborne concentrations (Dollard et al. 2007) mainly due to the use of reformulated gasolines, the lowering of vehicle emissions, and other control measures (Fruin et al. 2001).

A list of previous studies of personal exposures and microenvironmental concentrations of VOCs and PAHs is given in Appendix 24.

The impact of traffic on personal exposure has been assessed in various ways: first, by considering the geographic location of subjects; second, the area within the city where the subject's home is located; and third, classifying the home with respect to road traffic. Homes are the most influential microenvironment for all VOCs (Kim et al. 2002); in some cases the distributions of personal and indoor home concentrations overlap considerably (Phillips

Table 33. Characterization of VOC and PAH Personal Exposures

Compound ^a	<i>N</i>	25th Percentile	75th Percentile	Arithmetic Mean	Geometric Mean
VOC					
<i>n</i> -Hexane	500	0.76	3.19	3.61	1.67
Benzene	500	1.02	2.41	2.21	1.64
Toluene	500	5.92	21.36	19.76	11.53
Ethylbenzene	500	0.75	2.59	3.21	1.47
<i>p</i> -Xylene	500	0.62	2.28	3.07	1.26
<i>m</i> -Xylene	500	1.58	6.23	7.69	3.23
Pyridine	500	0.08	0.26	0.25	0.15
<i>o</i> -Xylene	500	0.80	2.90	3.58	1.61
1,3,5-Trimethylbenzene	500	0.22	0.75	0.95	0.44
Styrene	500	0.39	0.94	1.32	0.63
<i>p</i> -Isopropyltoluene	500	0.50	1.33	1.07	0.80
1,2,4-Trimethylbenzene	500	0.76	2.82	3.48	1.57
3-Ethenylpyridine	500	0.03	0.20	0.28	0.10
Naphthalene	500	0.34	0.77	0.74	0.53
1,3-Butadiene	500	0.05	0.42	0.40	0.14
PAH					
Acenaphthylene	91	0.06	0.62	0.48	0.27
Acenaphthene	91	0.11	0.50	0.63	0.12
Fluorene	91	0.03	0.84	0.46	0.16
Phenanthrene	91	0.10	0.47	0.47	0.22
Anthracene	91	0.02	0.13	0.10	0.05
Fluoranthene	91	0.18	0.93	0.89	0.56
Pyrene	91	0.11	0.64	0.62	0.35
Benzo[<i>a</i>]anthracene	91	0.02	0.13	0.46	0.09
Chrysene	91	0.11	0.42	0.76	0.27
Benzo[<i>b</i>]fluoranthene	91	0.08	0.42	0.74	0.27
Benzo[<i>k</i>]fluoranthene	91	0.08	0.33	0.72	0.21
Benzo[<i>a</i>]pyrene	91	0.04	0.24	0.61	0.13
Indeno[1,2,3- <i>cd</i>]pyrene	91	0.04	0.23	0.51	0.16
Dibenz[<i>a,h</i>]anthracene	91	0.01	0.06	0.06	0.02
Benzo[<i>g,h,i</i>]perylene	91	0.08	0.34	0.57	0.22
Coronene	91	0.05	0.17	0.32	0.11

^a The concentration data are expressed as $\mu\text{g}/\text{m}^3$ for VOCs and ng/m^3 for PAHs.

et al. 2005). Therefore, the impact of traffic on personal exposure is mainly due to the impact of traffic emissions on the home microenvironment. The assessment of the effect of traffic on homes shows patterns similar to those of the effect on personal exposure.

As regards the geographic locations of the subjects and homes (Figure 15), those in London had similar concentrations to those in urban Birmingham for most of the compounds. These results should be carefully interpreted, however, as the London subjects and homes were sampled in summer, when both outdoor and indoor concentrations are lower (Schneider et al. 2001) and the microenvironmental

air exchange rate and proportion of time spent outdoors are higher (Kim et al. 2002); the Birmingham subjects and homes were sampled across all four seasons. In addition, some sources affecting Birmingham subjects are less commonly experienced by London subjects, such as ETS exposure (found for 33% of subjects in Birmingham [West Midlands] but only 27% in London) (Table 31).

On the other hand, subjects and homes in the rural West Midlands and rural Wales had similar concentrations ($P > 0.10$), except for ETS related compounds, which were lower in rural Wales. The similar concentrations in West Midlands and Wales (where the lower exposures were

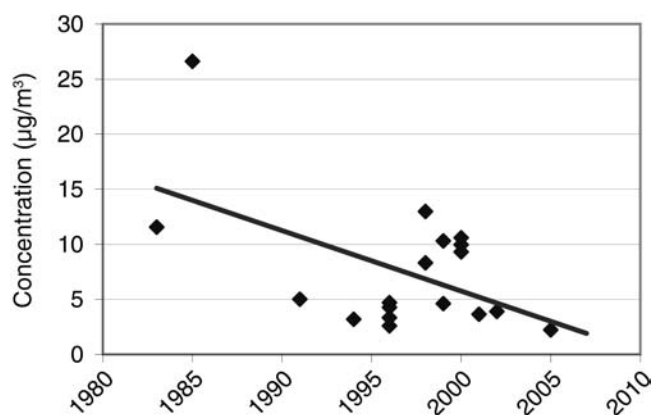


Figure 14. Reported personal exposures to benzene since 1980 in the United States and Europe. The data points represent the arithmetic means, from Table A24.1 in Appendix 24.

anticipated) could be a consequence of the higher percentage of integral garages in homes in Wales (for 30% of subjects) than in homes in West Midlands (for 20%), a higher proportion of subjects living in first-line properties in Wales (60%, compared to 40% in West Midlands), and the fact that the Welsh subjects were sampled in cold months, which could be influenced by additional indoor sources (e.g., heating systems). The fact that Welsh subjects show lower concentrations of ETS-related compounds is in accordance

with Wales having the lowest percentage of ETS-exposed subjects (Table 31).

With regard to the effect that home location within a city has on personal exposure and concentrations within the home, we expected that urban subjects would have the highest VOC exposures and rural subjects the lowest. This was not borne out in our study. Figure 16 shows similar concentrations for urban, suburban, and rural subjects and homes. This finding is at odds with previously reported results of suburban volunteers and home microenvironments having lower concentrations than urban ones (Leung and Harrison 1998; Ilgen et al. 2001b,c; Mann et al. 2001). As shown in Table 31, suburban and rural subjects have a pollutant source that urban subjects do not have: an integral garage within the home. Suburban subjects also show a greater prevalence of ETS exposure (43%) compared with subjects living in urban (32%) and rural areas (20%). Additionally, rural subjects were sampled mainly in cold months, during which there are more indoor sources, because of house heating, and indoor sources have a greater impact on microenvironmental concentrations and exposures because of low air exchange rates; also 25% of the rural subjects used fuels other than gas or electricity (e.g., solid fuel and kerosene) for home heating and 25% of the rural subjects had recently redecorated their homes. These different seasonal patterns and source distributions for subjects living in different types of locations might have led to higher VOC exposures for

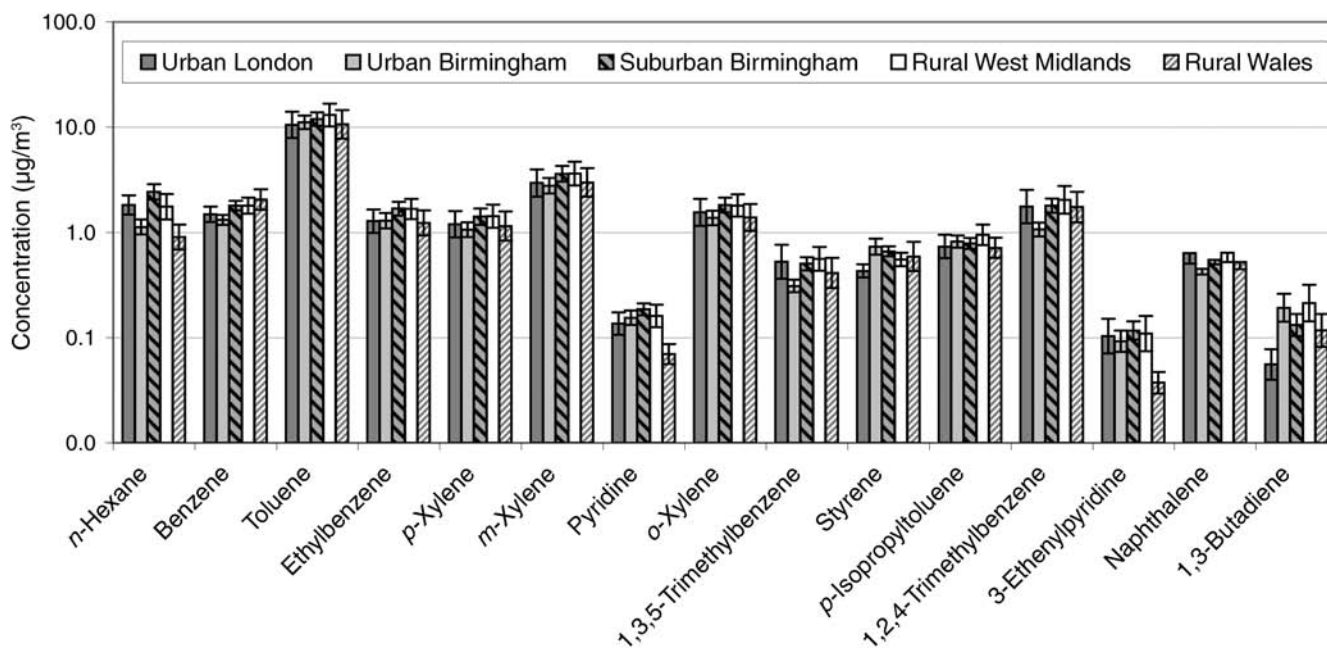


Figure 15. Personal exposure concentrations of VOCs (in $\mu\text{g}/\text{m}^3$), according to geographic location. Data are presented as the geometric mean \pm 95% CI (error bars). $N = 55$ for subjects in urban London, $N = 136$ for subjects in urban Birmingham, $N = 209$ for subjects in suburban Birmingham, $N = 50$ for subjects in rural West Midlands, and $N = 50$ for subjects in rural Wales.

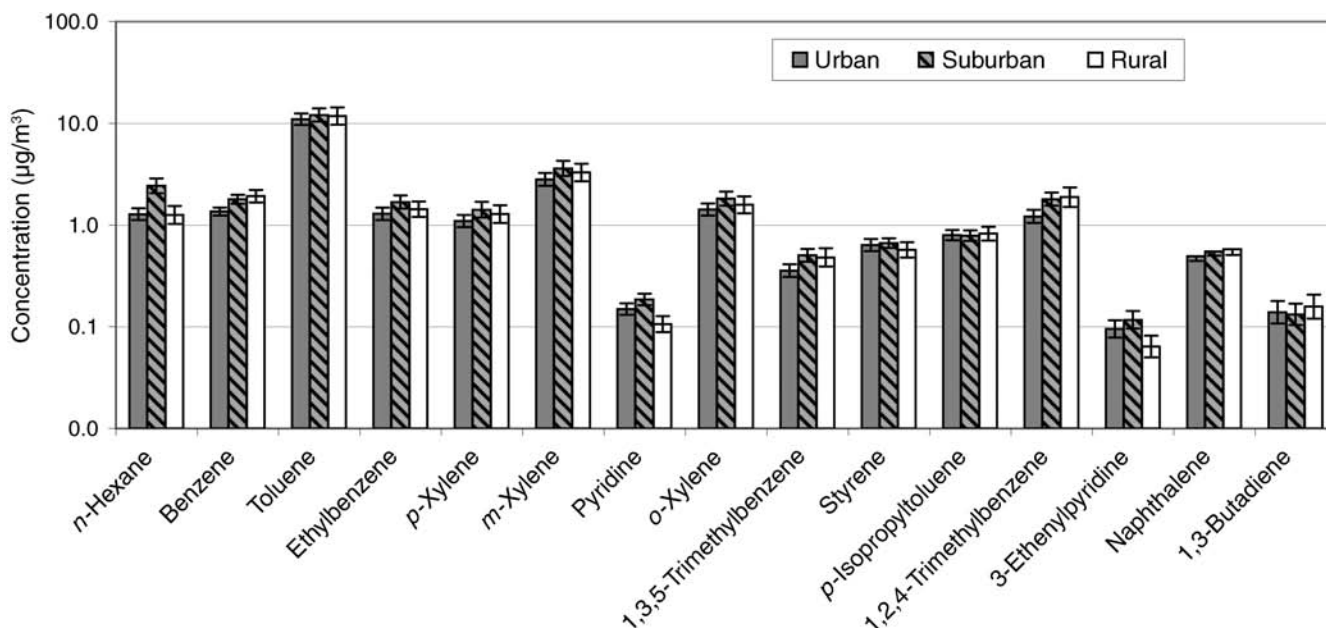


Figure 16. Personal exposure concentrations of VOCs (in $\mu\text{g}/\text{m}^3$), according to type of location. Data are presented as the geometric mean \pm 95% CI (error bars). $N = 191$ for subjects in urban locations, $N = 209$ for subjects in suburban locations, and $N = 100$ for subjects in rural locations.

rural subjects and, to a lesser extent, suburban subjects, which in turn could have produced a pattern different than the expected trend of personal exposures being highest for urban subjects, followed by suburban and then rural subjects.

We performed a similar assessment focused on subjects living in the West Midlands area only. Within West Midlands, more subjects living in suburban areas than in urban or rural areas had homes with integral garages (26%), were exposed to ETS at home (20%) and lived in first-line properties (43%). This higher proportion of subjects with within-home sources is reflected in the higher suburban home concentrations in the West Midlands area (Figure A6.12). On the other hand, personal exposures of West Midlands subjects were similar in the suburban and rural areas; those in rural West Midlands spent more time commuting (7% of their time) than did the other West Midlands sub-populations. Therefore, these findings emphasize the fact that not only home concentrations, but also a wide range of other sources, contribute to personal exposure concentrations (Leung and Harrison 1998)—including a variety of activities that the subjects are engaged in during their normal life, such as exposure to ETS, commuting, use of consumer products, home repair and improvement, use of solvents, and photocopying. The home microenvironment is influenced by a wide range of indoor sources, such as the presence of integral garages, ETS, paints, consumer products, past redecoration, and heating and cooking systems (Wallace 2001; Adgate et al. 2004a).

Personal exposure levels and the corresponding home microenvironmental levels experienced by subjects living in houses located on trafficked roadsides (first-line houses) are similar to those in non-first-line homes, except for toluene, which is found at slightly higher concentrations for subjects living in first-line homes ($P < 0.10$) as well as within the home itself ($P < 0.05$) (Figure 17). This pattern is in contrast, however, to those found in other studies, with homes located close to traffic sources having higher VOC concentrations than those farther from traffic (Heavener et al. 1996; Ilgen et al. 2001a,b; Kim et al. 2001b).

One important observation is that levels of compounds associated with ETS (e.g., pyridine, 3-ethenylpyridine) were higher in subjects not living in first-line properties. Further investigation of the effect of first-line versus non-first-line status in those households without an integral garage or without ETS exposure showed that, in general, home VOC concentrations were higher in homes located on a trafficked roadside (Figure 18), although the increase was significant only for toluene ($P < 0.10$) and 1,2,4-trimethylbenzene ($P < 0.05$). On the other hand, controlling for the effect of ETS exposure and an integral garage in the analysis showed that personal VOC concentrations were higher for subjects living on a non-trafficked roadside (Figure 19). This finding may indicate that, once the presence of integral garages and ETS are controlled for, the influence of other indoor sources (e.g., redecoration, heating by means of fuels other than electricity or natural gas)

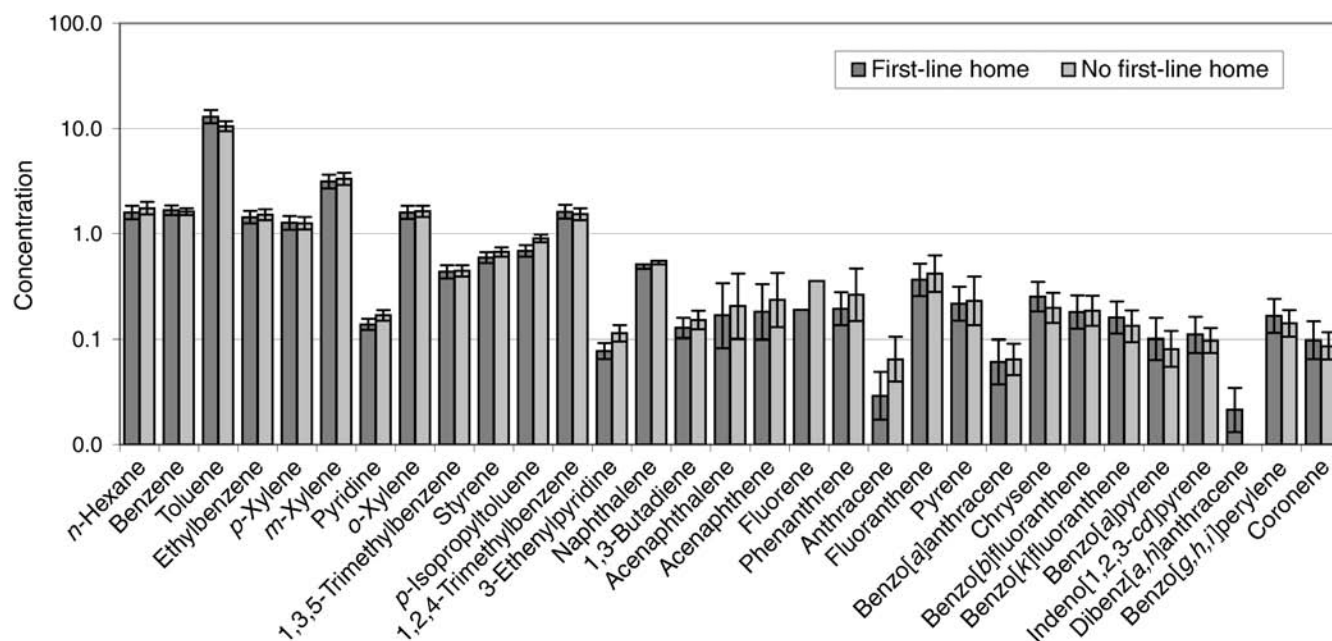


Figure 17. Personal exposure concentrations of VOCs (in $\mu\text{g}/\text{m}^3$) and PAHs (in ng/m^3), according to proximity of home to trafficked road. Data are presented as the geometric mean \pm 95% CI (error bars). For VOCs, $N = 219$ for subjects with first-line homes and $N = 281$ for those without first-line homes; and for PAHs, $N = 41$ for subjects with first-line homes and $N = 50$ for those without. No error bars are shown for fluorene because there are fewer than five data points.

and activities that the subjects perform during the day (e.g., commuting, home repair and improvement, incense burning) might have masked the effect of traffic on personal exposures and concentrations in the home, with the effect being prevalent for only a few compounds in home environments (e.g., toluene).

Living in houses with integral garages (Figure 8) was associated with increased levels of personal exposure and in the home microenvironment ($P < 0.01$) for most of the compounds. The average ratio of the personal exposures (GM \pm GSD) of subjects who lived in houses with integral garages and the concentrations of those without integral garages is $(1.9 \pm 0.6):1$; the ratio of the corresponding home concentrations is $(2.0 \pm 0.7):1$. These findings are in accordance with results previously reported (Heavner et al. 1995; Ilgen et al. 2001b; Marshall et al. 2003; Batterman et al. 2006a,b, 2007). Heavner and colleagues (1995) reported that levels of xylenes and 1,3,5-trimethylbenzene increased in homes where gasoline was stored. Sources of VOCs in garages were investigated on the basis of information provided by the subjects in the storage questionnaire, related to parked cars, storage of products used in home repair and improvement and gardening, and location of the heating system unit.

The effect of ETS in personal exposures and home microenvironments is clear (Figure 9), leading to higher concentrations of all the VOCs ($P < 0.05$, $N = 500$), with

an average ratio for the presence of ETS and its absence of $(1.4 \pm 0.4):1$. The greatest differences observed were for ETS-related compounds like 3-ethenylpyridine or pyridine (average ratio for each, 1.8:1). Similarly, homes with ETS show higher VOC concentrations than homes that are ETS-free, with a corresponding average ratio of $(2.4 \pm 2.1):1$. The ratio is greater for ETS-related compounds like 3-ethenylpyridine (9.4:1), pyridine (2.1:1), and 1,3-butadiene (4.6:1). These data are consistent with those in other studies (Heavner et al. 1995, 1996; Wallace 1996; Leung and Harrison 1998; Carrer et al. 2000; Pérez Ballesta et al. 2006).

When personal exposures of subjects exposed to ETS were averaged across the sampling week, only the exposures to ETS-related compounds such as 3-ethenylpyridine and pyridine remained significantly higher than those experienced by subjects not exposed to ETS ($P < 0.001$, $N = 100$). This is a consequence of the fact that subjects with ETS exposures were not necessarily exposed to ETS on every day of sampling, and therefore the ETS effect was diluted when personal exposures were averaged across the 5 days.

PAHs

PAHs are semi-VOCs formed during the incomplete combustion of organic material. Although they are also released into the atmosphere from natural sources, such as forest and prairie fires and volcanic eruptions, their occurrence

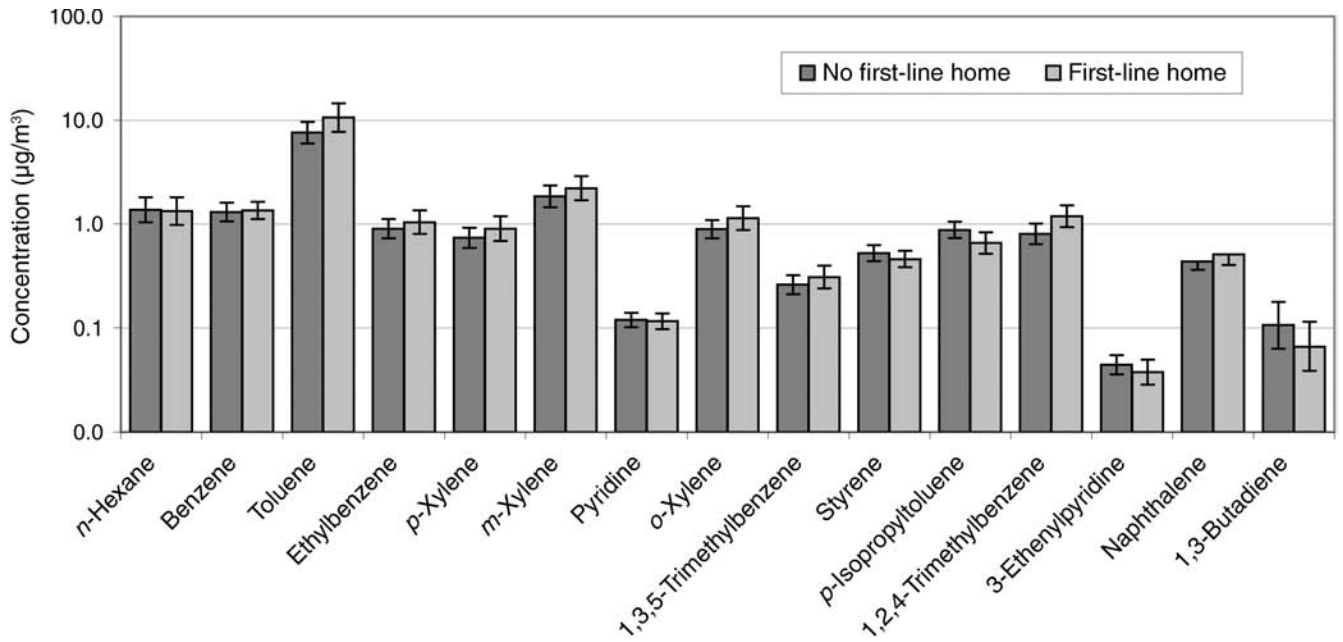


Figure 18. Home concentrations of VOCs (in $\mu\text{g}/\text{m}^3$) among subjects with no integral garage and no ETS exposure, according to proximity of home to trafficked road. Data are presented as the geometric mean \pm 95% CI (error bars). $N = 56$ for subjects with first-line homes and $N = 47$ for those without first-line homes.

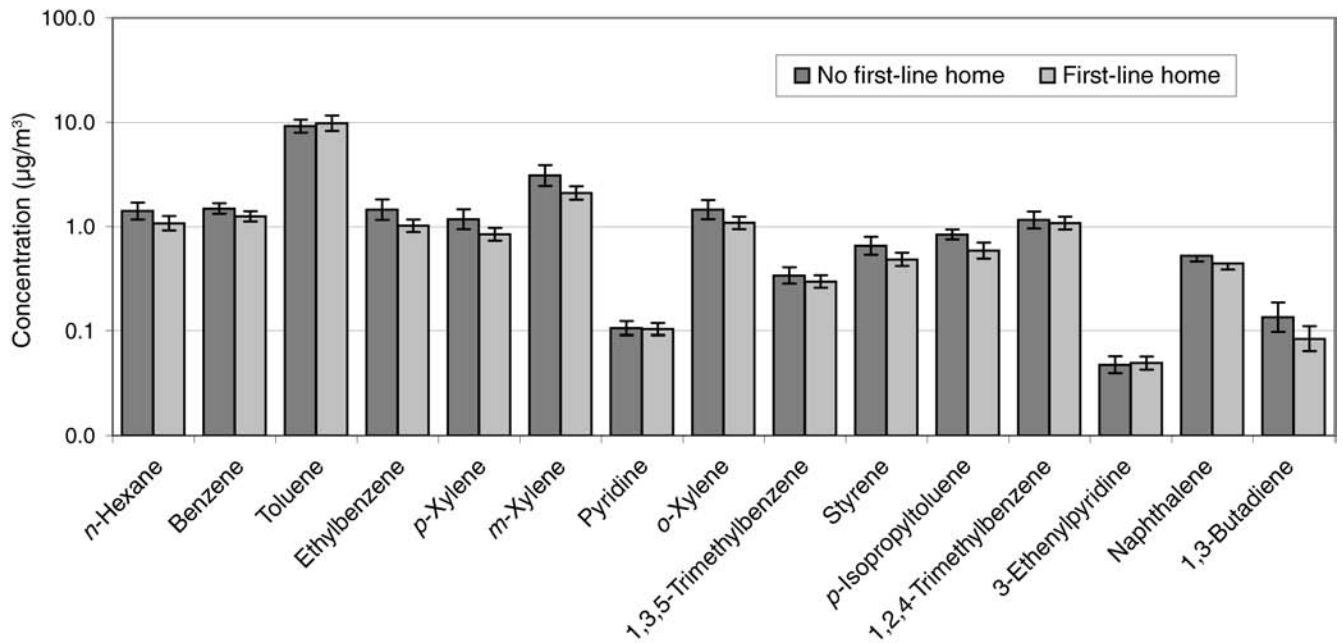


Figure 19. Personal exposure concentrations of VOCs (in $\mu\text{g}/\text{m}^3$) among subjects with no integral garage and no ETS exposure, according to proximity of home to trafficked road. Data are presented as the geometric mean \pm 95% CI (error bars). $N = 128$ for subjects with first-line homes and $N = 111$ for those without first-line homes.

is largely a result of anthropogenic emissions from coal-, oil- and gas-burning facilities, motor vehicles, waste incineration, and industrial activities such as oil refining, coke and asphalt production, and aluminum production (Mitra and Ray 1995; Baek et al. 1997; Ohura et al. 2004b). After PAHs are emitted, the atmosphere is the determining factor influencing their distribution and fate. The extent to which humans are exposed to PAHs depends on several parameters, including the prevailing atmospheric conditions, concentrations in ambient air, partitioning between the gas and particle phases, and the size distribution of airborne particulates (Georgiadis et al. 2001; Naumova et al. 2003; Ohura et al. 2004a; Chang et al. 2006).

Studies related to exposure to PAHs have focused on either occupational exposure or on exposure in specific microenvironments like trafficked roadsides (Lim et al. 1999), vehicles (Levy et al. 2002), homes in which smoking occurs (Chuang et al. 1991), and areas of social interaction like pubs, restaurants (Levy et al. 2002), and kitchens (Zhu and Wang 2003). Little information is available on exposure in specific rooms in the home like kitchens, living rooms, and dining areas. This study presents personal exposure data and concentrations measured in various microenvironments (i.e., indoor, outdoor, and transport microenvironments) of PAHs.

The personal exposure data for two rural subjects living in Wales were considered as extreme outliers. The causes of these high exposures (e.g., 25.31 ng/m³ and 5.36 ng/m³ for benzo[*a*]pyrene) were associated with the use of a fireplace at home. Data for these two highly exposed subjects were removed from the analysis for the assessment of the effect of key determinants (exposure to ETS or living in a first-line property or with an integral garage) or geographic location and type of location.

The personal exposures to PAHs recorded in the present study (excluding the two outliers) are summarized in Table 33. Results obtained for the lower-molecular-weight PAH compounds (acenaphthylene, acenaphthene, fluorene, phenanthrene, and anthracene) should be considered with great caution, since these compounds exist mainly in the vapor phase and our method determines only PAHs associated with particles. For example, concentrations of phenanthrene (sum of both particle and vapor phases) are typically 10–20 ng/m³, so these are substantially underestimated in the present study (e.g., the average arithmetic personal exposure mean for phenanthrene is 0.44 ng/m³). The vapor–particle partition is severely affected by temperature and therefore samples that were not collected under identical climate conditions will not be comparable.

Georgiadis and coworkers (2001) carried out a campaign of sampling PAH personal exposures in Athens, Greece, a

heavily trafficked city with moderate-to-high air pollution, and in Halkida, a nearby small town, expected to have lower pollution levels. Eight PAHs—benzo[*a*]anthracene, chrysene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, benzo[*a*]pyrene, dibenz[*a,h*]anthracene, benzo[*g,h,i*]perylene, and indeno[1,2,3-*cd*]perylene—were measured, and the data were summed across all eight. The mean personal exposure to all eight PAHs for Athens was 7.95 ng/m³ and for Halkida it was 4.53 ng/m³. In our study, for London, the mean personal exposure to the same group of compounds was 0.77 ng/m³. In Birmingham it was 1.85 ng/m³ and Wales it was 1.99 ng/m³ (Appendix 7). The air quality standards have become more stringent in recent years in the United Kingdom and this change, in combination with different meteorologic conditions and different traffic patterns, might explain the difference between the Athens and the London values.

The exposure to lower-molecular-weight, more-volatile PAHs within the class containing acenaphthylene, acenaphthene, fluorene, phenanthrene, and anthracene tended to be higher in suburban and rural areas than in urban areas (Figure 20), but earlier caveats about data quality apply. The personal exposure to the medium-to-high-molecular-weight PAHs—fluorene, phenanthrene, anthracene, fluoranthene, pyrene, benzo[*a*]anthracene, chrysene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, benzo[*a*]pyrene, indeno[1,2,3-*cd*]pyrene, dibenz[*a,h*]anthracene, benzo[*g,h,i*]perylene, and coronene—was surprisingly high in rural areas, with an exposure very similar to that in urban areas and higher than in suburban areas. Nevertheless, these observations of personal exposure in different locations within a city were not significantly different ($P > 0.10$). On the other hand, rural homes showed higher concentrations of high-molecular-weight PAHs ($P < 0.05$) than did urban or suburban homes. This finding might be associated with use of fireplaces and fuel types other than natural gas or electricity for heating, as was reported by the subjects in the forms, especially as most of the rural homes were sampled during winter. The fact that rural homes showed the highest concentration of high-molecular-weight PAHs clearly impacts the personal exposures of rural subjects, reflecting the general fact that home microenvironments have a large effect on personal exposures. On the other hand, when we considered just the subjects living in urban, suburban, and rural areas in West Midlands, we found higher personal exposures for urban subjects and higher concentrations in rural homes, although these differences are not significant. The apparent mismatch between home concentrations and personal exposure concentrations in the West Midlands subsample shows the relevance of personal activities, in addition to home concentrations, to personal exposures.

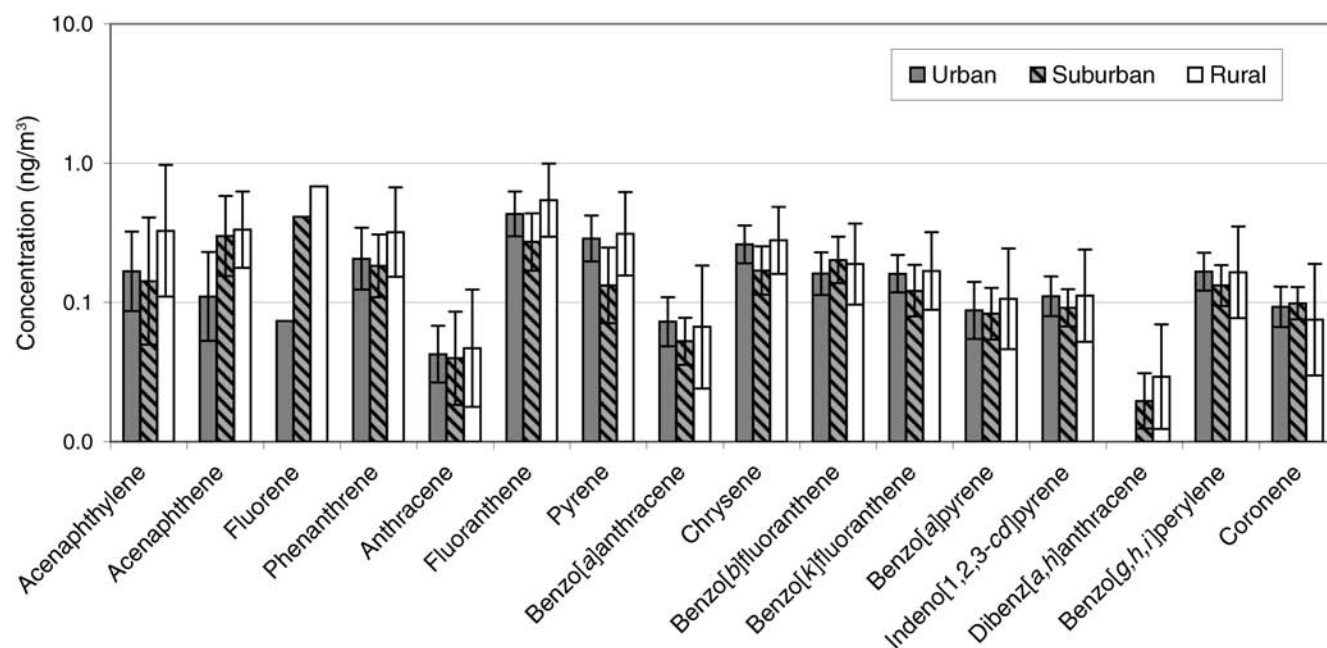


Figure 20. Personal exposure concentrations of PAHs (in ng/m^3), according to type of location. Data are presented as the geometric mean \pm 95% CI (error bars). $N = 35$ for subjects in urban locations, $N = 37$ for subjects in suburban locations, and $N = 19$ for subjects in rural locations. No error bars are shown for fluorene because there are fewer than five data points.

Personal exposures to PAHs do not appear to differ significantly according to the subjects' geographic location (Figure 21), although concentrations were higher among Welsh subjects than those in London or West Midlands for some compounds, such as benzo[*a*]anthracene, chrysene, benzo[*k*]fluoranthene ($P < 0.05$), benzo[*a*]pyrene, and ideno[1,2,3-*cd*]pyrene. The fact that the Welsh homes were sampled in winter, when ambient PAH levels are higher and additional indoor sources (such as fireplace use or space heating) are present, might have influenced this result.

The effect of residential traffic on personal exposure and home concentrations does not show a clear distinction between people living in first-line properties as compared to non-first-line properties (Figure 17). Although the average arithmetic and GMs suggest that first-line homes have higher levels of the higher-molecular-weight PAHs, this difference was not significant ($P > 0.10$). The results were similar when classifying personal exposure and home micro-environmental concentrations according to the presence or absence of an integral garage (Figure 8). Homes with an integral garage do not seem to have higher PAH concentrations, with the exception of benzo[*b*]fluoranthene ($P < 0.01$) and anthracene, benzo[*k*]fluoranthene, benzo[*a*]pyrene, and coronene ($P < 0.10$). These results are consistent with the observation that, in home microenvironments, unless ETS

is the major source indoors, the exposure to PAHs has a multitude of sources (Li et al. 2005).

As reported in various studies, ETS is the main contributor to PAHs in personal exposures (Georgiadis et al. 2001) and indoors (Chuang et al. 1991; Phillips 1994; Mitra and Ray 1995; Harrison et al. 1996; Liu 2001; Ohura et al. 2004a; Gee et al. 2005; Lu 2006). Our results are consistent with regard to home concentrations (Figure 9), although there is not a clear-cut difference between personal exposures for subjects exposed to second-hand smoke and those not exposed to ETS. Li and colleagues (2005) propose that, apart from ETS, indoor sources exist for two- and three-ring PAHs, whereas outdoor air may contribute significantly to indoor levels of PAHs with four or more rings that are associated with particles $\leq 2.5 \mu\text{m}$ in aerodynamic diameter (Sugiyama 2000; Koyano 2001). Further investigation of the supporting information and activity diaries for the non-ETS group showed that three subjects can be considered to be outliers. After the data for these three subjects were removed from analyses, the mean benzo[*a*]pyrene concentration in the non-ETS group ($N = 52$) dropped to $0.20 \text{ ng}/\text{m}^3$ as compared with $0.80 \text{ ng}/\text{m}^3$ in the ETS group ($N = 36$), and the sum of eight PAHs (benzo[*a*]anthracene, chrysene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, benzo[*a*]pyrene, dibenz[*a,h*]anthracene, benzo[*g,h,i*]perylene, and ideno[1,2,3-*cd*]perylene) is reduced to $2.87 \text{ ng}/\text{m}^3$,

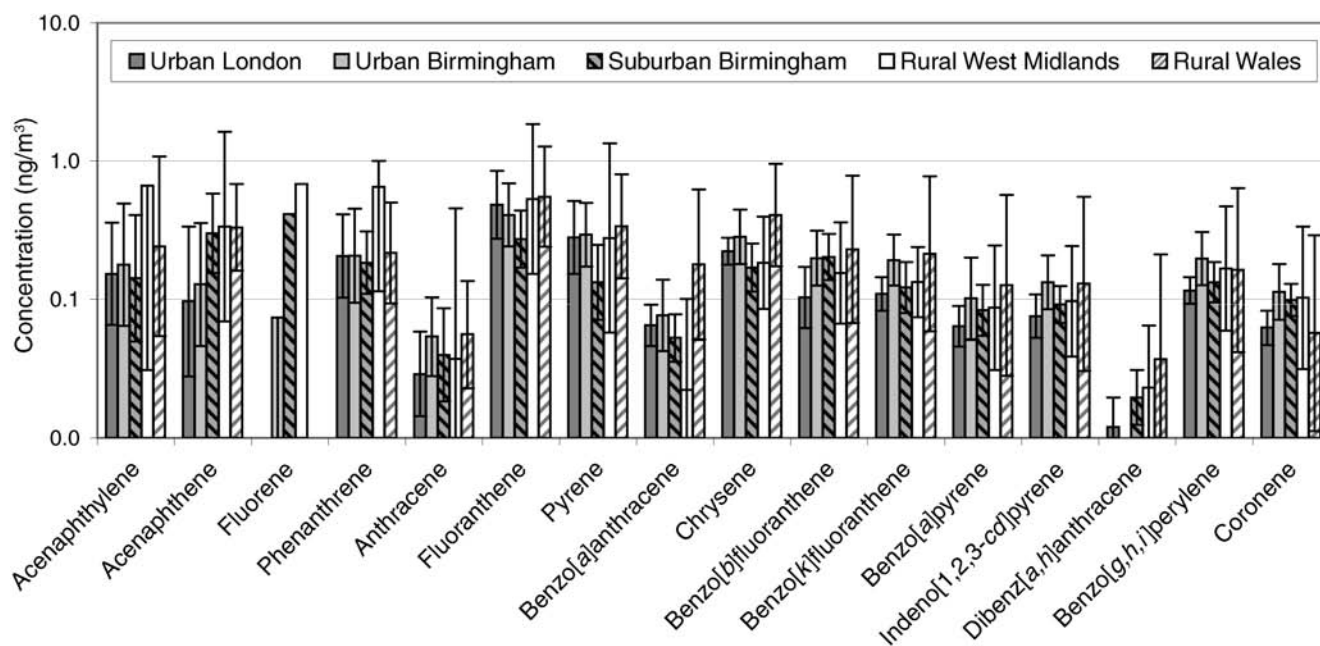


Figure 21. Personal exposure concentrations of PAHs (in ng/m^3), according to geographic location. Data are presented as the geometric mean \pm 95% CI (error bars). $N = 11$ for subjects in urban London, $N = 24$ for subjects in urban Birmingham, $N = 37$ for subjects in suburban Birmingham, $N = 9$ for subjects in rural West Midlands, and $N = 10$ for subjects in rural Wales. No error bars are shown for fluorene because there are fewer than five data points.

compared with $3.33 \text{ ng}/\text{m}^3$ in the ETS group — yielding a pronounced, significant ($P < 0.10$) difference between the ETS and the non-ETS groups. Our results, like those of Mitra and Ray (1995) emphasize the impact of ETS on indoor air quality and also indicate that, in the absence of ETS, background sources (e.g., gas utilities) contribute significantly to total PAH exposures (Mitra and Ray 1995).

The majority of subjects selected for this study were between 18 and 65 years old. Nevertheless, those subjects who were 66 years old or older had an interesting pattern of VOC and PAH concentrations (Figure 1). Although they are generally exposed to higher VOC concentrations (albeit not significantly higher), their PAH exposures are significantly lower than those in younger age groups. This result may reflect the different activities that the two groups perform. The senior population (≥ 66 years) may be less exposed to strong sources of PAHs, such as ETS or commuting. This finding is consistent with the pattern of PAH concentrations according to occupational category (Figure 3). The retired subjects, most of whom were over 66 years old, were the subset with the lowest PAH concentrations. The subsets of subjects with more time spent commuting and more activities performed outside the home (office workers and students) were the ones who showed the highest PAH concentrations. On the other hand, the VOC concentrations were similar between retired people and the students

and office workers, with the housewives and the unemployed having the highest VOCs concentrations (Figure 2) — possibly a consequence of the greater time spent at home doing various activities involving solvents (e.g., cleaning agents, products for home repair and improvement).

MICROENVIRONMENTAL CONCENTRATIONS

VOCs Including 1,3-Butadiene

The levels of VOCs in subjects' homes in this study (Appendix 8) were mainly measured in living rooms and dining rooms. The levels are significantly lower than those reported in other studies in the United States (Wallace 1989a,b; Heavner et al. 1996), Hong Kong, China (Lee et al. 2002a,b), Korea (Baek et al. 1997), the Netherlands, Germany (Brown et al. 1994), and the United Kingdom (Brown and Crump 1998; Leung and Harrison 1998; Lai et al. 2004).

As compared with our study, studies carried out in Columbus, Ohio (Heavner et al. 1995), Chicago, Illinois (Van Winkle and Scheff 2001), and Melbourne, Australia (Brown 2002), reported higher concentrations for all the compounds except for toluene and 3-ethenylpyridine (in Columbus); toluene, styrene, and 3-ethenylpyridine (in Chicago); and toluene, styrene and *n*-hexane (in Melbourne). Studies reported over the last decade in the United States (Adgate et al. 2004a; Phillips et al. 2005) showed median

VOC values similar to those in our study, with the exception of a higher level of benzene reported by Adgate and colleagues and toluene reported by Phillips and coworkers.

In Europe, studies carried out in Helsinki, Finland, in 1995 (Kostiainen 1995) and 2001 (Edwards et al. 2001b) reported VOC concentrations similar to those in our study except for higher concentrations for the xylenes and ethylbenzene in both studies, benzene in the 1995 study, and trimethylbenzene in the 2001 study. Two studies carried out in German cities in 2001 showed concentrations similar to those in our study for all VOCs except toluene (Schneider et al. 2001) and higher concentrations for all except benzene and *o*-xylene (Ilgen et al. 2001a). A study completed in the West Midlands in 2001 (Kim et al. 2001a) demonstrated concentrations similar to those in the present study for almost all the aromatic compounds but higher concentrations for benzene, toluene and 1,3-butadiene.

Daytime and nighttime concentrations appear to be fairly similar both for VOCs and PAHs (Figure 22), with slightly higher levels measured during the day than during the night, with an average ratio of $(1.10 \pm 0.07):1$. An exception was 1,3-butadiene, which has a very short atmospheric lifetime with respect to photolysis; higher concentrations were observed during the night than during the day, with a geometric ratio of 1.4:1. Nevertheless, daytime and nighttime concentrations were not significantly different ($P > 0.10$) for any of the studied compounds. In contrast, higher

daytime concentrations have been reported in previous studies (Phillips et al. 2005). The similar levels seen in the current study might imply that the general day–night pattern is confounded by evening indoor activities. The concentrations of VOCs and PAHs in homes during the day, when subjects are usually at the workplace and therefore activity in the home is reduced and the heating systems of the house are turned off, might be more influenced by outside air. In the evening, subjects are normally at home carrying out various activities (e.g., cooking), and although outdoor ambient concentrations are lower then, indoor activities might affect indoor VOC and PAH levels. A shortcoming of this project is that minimal information was gathered on the actual activities going on during the period of home sampling in the specific rooms, unless performed by the subject, and direct quantification of the air exchange rate was not possible. In the case of PAHs, there could also be a mixed effect of source strength (greater during the day) and temperature that leads to greater partitioning of the lower-molecular-weight compounds into the particles at night.

With regard to concentrations measured in different microenvironments within the house, caution should be exercised because of the low number of measurements performed (i.e., three in each season and each microenvironment). In this study, the highest concentrations were found in the garage, followed by the bedrooms, living

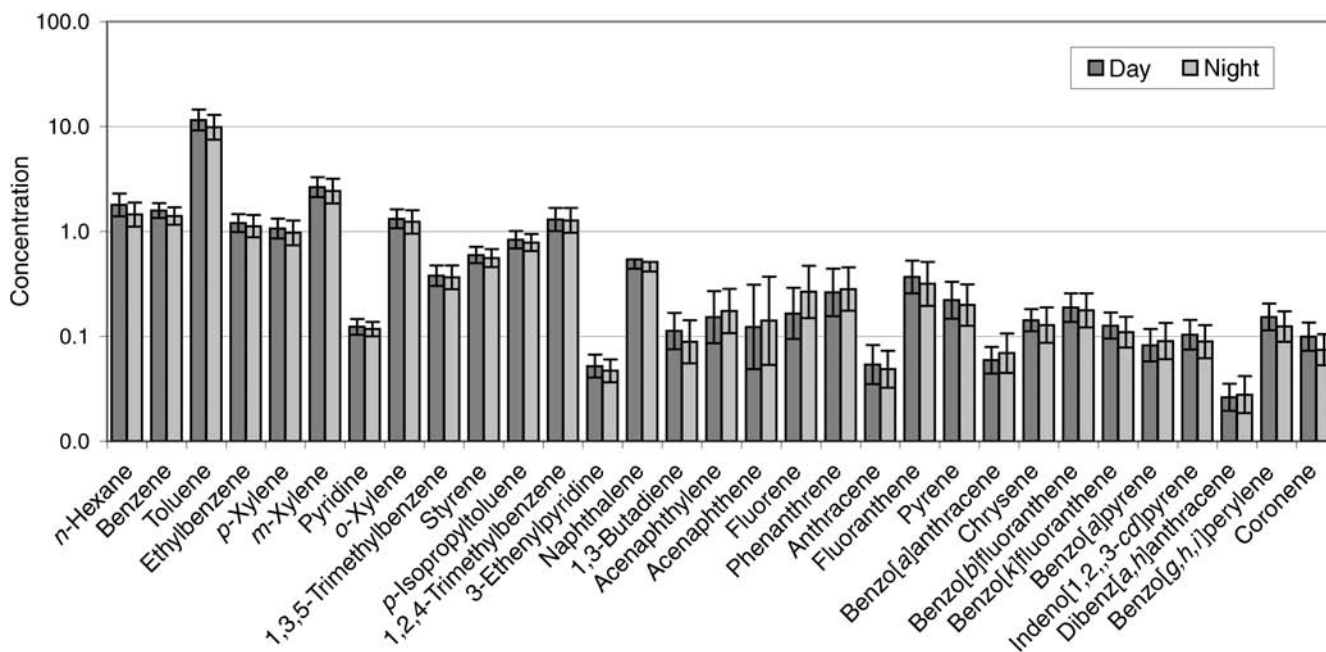


Figure 22. Home microenvironmental concentrations of VOCs (in $\mu\text{g}/\text{m}^3$) and PAHs (in ng/m^3), according to time of day. Data are presented as the geometric mean \pm 95% CI (error bars). For VOCs, $N = 80$ for day and $N = 80$ for night; for PAHs, $N = 39$ for day and $N = 38$ for night.

room, and kitchen. The levels in these microenvironments are lower than those reported in other studies to date, such as those carried out in Helsinki, Finland (Edwards et al. 2001a), Avon, United Kingdom (Mann et al. 2001), Hanover, Germany (Ilgen et al. 2001b), and Hong Kong, China (Lee et al. 2002b).

Despite the levels recorded in the garages being the highest in the home microenvironment, these levels are far below those reported in the United States (Batterman et al. 2006a,b, 2007) and previously in the United Kingdom (Mann et al. 2001). A possible explanation may be the fact that, much of the time, cars are not parked in the sampled garages. This idea is consistent with the findings of Ilgen and associates (2001b), who found that garages had very low concentrations of VOCs when no cars or solvents were stored inside, as compared with periods when cars were parked and solvents were kept inside the garages.

The levels of VOCs in living rooms in this study were higher than the levels in kitchens, except for *n*-hexane and 1,3-butadiene. The ratio of GM concentrations in living rooms and kitchens, for all compounds, is $(1.4 \pm 0.5):1$, and the ratio for 1,3-butadiene alone is $0.35:1$. There appears to be an ETS effect on the living room:kitchen ratio, as evidenced by the data for pyridine and 3-ethenylpyridine, which obscures the usefulness of the ratio. Other studies performed in Hong Kong, however, have reported higher concentrations of VOCs in kitchens than in living rooms (Lee et al. 2002a,b). This difference might be a consequence of the reported presence of ETS in the living room and the fact that, in our study, cooking was not occurring in several sampled kitchens. Other determinants such as ventilation patterns, cooking styles, and kitchen materials used may differ between the United Kingdom and Hong Kong, influencing the results. On the other hand, the higher levels of 1,3-butadiene recorded in the kitchen than in the living room might be a consequence of the combustion that occurs in the kitchen during cooking. Similarly, the higher levels of *n*-hexane in the kitchen might be attributable to the presence of sealants and the storage of paints (Scorecard 2006) in the kitchen.

An unexpected result is that high concentrations of benzene, toluene, ethylbenzene and the xylenes were found in yard samples. This result should be viewed with caution, owing to the low number of samples collected and also because one yard was the site of construction (of an addition to the house and a new conservatory). The cause of this high level of benzene, toluene, ethylbenzene and the xylenes in yards could also be explained by the use of a gasoline-fueled lawnmower in the subject's or a neighbor's yard.

The results of several concurrent samples collected outdoors in the backyard and indoors in the living or dining

room of homes located away from traffic show a clear pattern, with higher concentrations indoors than outdoors for all VOC compounds. Figure A6.26 (Appendix 6) clearly shows that concentrations indoors are higher, with an average ratio of $(3.6 \pm 5.3):1$. The same pattern is observed when comparing concentrations measured in street microenvironments and any indoor microenvironment. This finding is largely supported in the literature (Brown et al. 1994; Wallace 1996; Baek et al. 1997; Edwards et al. 2001b; Ilgen et al. 2001a; Schneider et al. 2001; Phillips et al. 2005; Batterman et al. 2007). However, some other studies of certain subpopulations reported indoor:outdoor ratios close to, or less than, unity for benzene, ethylbenzene, and *o*-xylene (Wallace et al. 1988). The indoor-air loading of benzene, toluene, and the xylenes in homes originates in part from the outdoors. Another source is human activities performed indoors, such as smoking, cooking, heating, cleaning, redecoration, fumigation, and the use of varnish and solvents. Other sources of benzene, toluene, and the xylenes may be residues in furniture and floor and wall coverings (Schneider et al. 2001). Therefore, the higher concentrations indoors are a consequence of a higher number of indoor VOC sources that contribute to the outdoor background VOC level.

Levels of VOC compounds recorded in workplaces (Appendix 10) are lower than personal exposure and home microenvironmental levels. As with personal and home concentrations, VOC levels measured in the present study are lower than those from earlier studies in the West Midlands (Leung and Harrison 1998), England (Brown and Crump 1998; Lai et al. 2004), Europe (Carrer et al. 2000; Ilgen et al. 2001c), the United States (Heavner et al. 1996), and Singapore (Zuraimi et al. 2006). A study performed in West Midlands offices in late 1990s showed similar concentrations of the xylenes, trimethylbenzenes, styrene, and *p*-isopropyltoluene but higher concentrations of benzene, toluene, ethylbenzene, *n*-hexane, naphthalene, and 1,3-butadiene (Kim et al. 2001a).

The effect of traffic on workplace microenvironments can be examined by comparing offices located on trafficked roadsides with offices located away from traffic and offices located in the city center with those located in a suburban area. Although values are similar in first-line offices to non-first-line offices and in urban offices and suburban offices, compounds like toluene and 1,2,4-trimethylbenzene have higher concentrations in the locations more affected by traffic. This observation could not be proven statistically, however ($P > 0.10$).

Street levels of VOCs recorded in the present study (Appendix 11) are mostly lower than those reported in studies carried out in the United States (Wallace 1996), Europe (Pérez Ballesta et al. 2006), Germany (Ilgen et al.

2001a), the United Kingdom (Lai et al. 2004), and Turkey (Muezzinoglu et al. 2001). Studies performed in the United States in the past decade reported VOC levels similar to those in the present study, except for some compounds: higher benzene levels have been reported in urban sites in Maryland (Sapkota and Buckley 2003) and in five U.S. cities with low traffic, and higher styrene concentrations have been found for high-traffic cities (Rappaport and Kupper 2004). The median levels of VOCs in outdoor air reported in Minnesota (Adgate et al. 2004a) and arithmetic means reported in the Multiple Air Toxics Exposure Study (MATES) III (Ospital et al. 2008) were, however, similar to the corresponding data in our study. The levels of VOCs reported in EXPOLIS-Helsinki (Edwards et al. 2001b) were similar to our data for most of the compounds, except for toluene and *n*-hexane, for which the EXPOLIS concentrations were higher.

In the ambient air, traffic is frequently a dominant source of VOCs. Therefore, higher concentrations appear first in areas with high traffic loads (Schneider et al. 2001; Pérez Ballesta et al. 2006). This is consistent with the outdoor levels recorded in our study, since VOC concentrations decrease as one moves from trafficked roadsides to background streets, pedestrian streets, and finally parks (Figure 23). In the same way, VOC levels measured in London streets are higher than those in Birmingham streets, with concentrations lowest in Wales streets (Figure 24); the data

correspond directly with the traffic load. Concentrations measured at rush hour, with peak traffic, are significantly higher ($P < 0.05$) than levels measured in the same streets during the afternoon, when traffic is less (Figure 25), with an average ratio of rush hour GM and afternoon GM of $(1.7 \pm 0.3):1$. These results are similar to those reported by other researchers, with VOC levels being higher during peak hours (Leung and Harrison 1999; Muezzinoglu et al. 2001; Sapkota and Buckley 2003; Ho et al. 2004) and generally lower in rural outdoor areas than in urban outdoor areas (Begerow et al. 1995; Ilgen et al. 2001a; Sapkota and Buckley 2003; Rappaport and Kupper 2004) and higher on trafficked roadsides than on background streets (Leung and Harrison 1999).

On the other hand, among measurements in different mobile-transport microenvironments, samples collected in the London Underground (subway) show the highest concentrations for ETS-related compounds (3-ethenylpyridine, pyridine, naphthalene and 1,3-butadiene) and *p*-isopropyltoluene. The reason for this is unclear, as smoking is not permitted in the subway.

As regards in-vehicle VOC concentrations (Appendix 12), the levels recorded in this study are lower than those from other studies carried out in vehicles (Wallace 1996; Carrer et al. 2000; Ilgen et al. 2001c; Chan et al. 2003; Lau and Chan 2003; Shiohara et al. 2005). The levels reported in two previous studies by our research group also showed

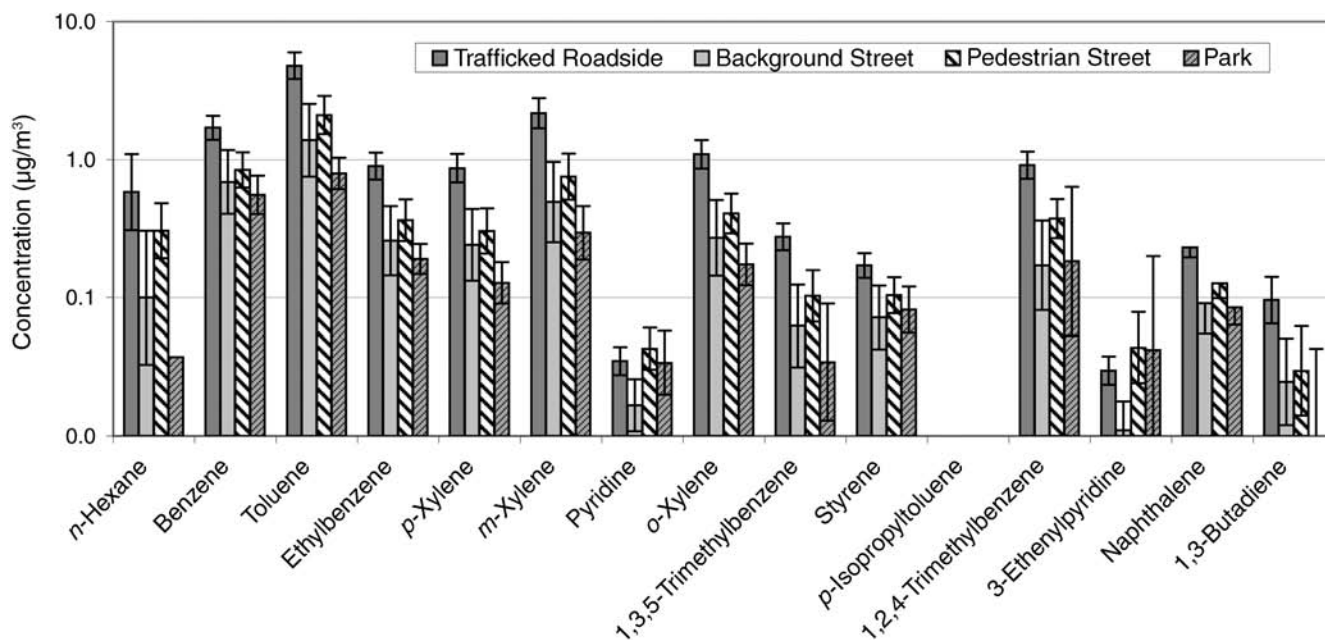


Figure 23. Street microenvironmental concentrations of VOCs (in µg/m³), according to street site. Data are presented as the geometric mean ± 95% CI (error bars). *N* = 21 for trafficked roadside, *N* = 26 for background street, *N* = 9 for pedestrian street, and *N* = 4 for park.

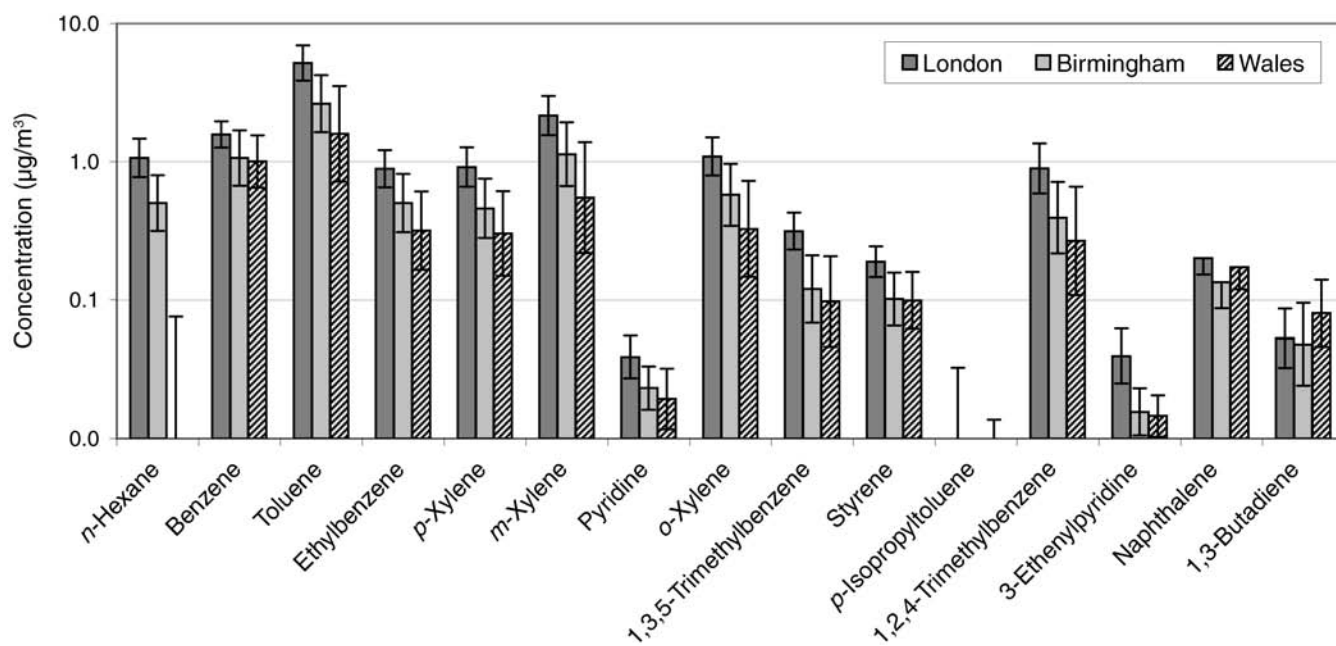


Figure 24. Microenvironmental concentrations of VOCs (in $\mu\text{g}/\text{m}^3$) on trafficked roadsides and background streets combined, according to geographic location. Data are presented as the geometric mean \pm 95% CI (error bars). $N = 8$ for London, $N = 39$ for Birmingham, and $N = 8$ for Wales.

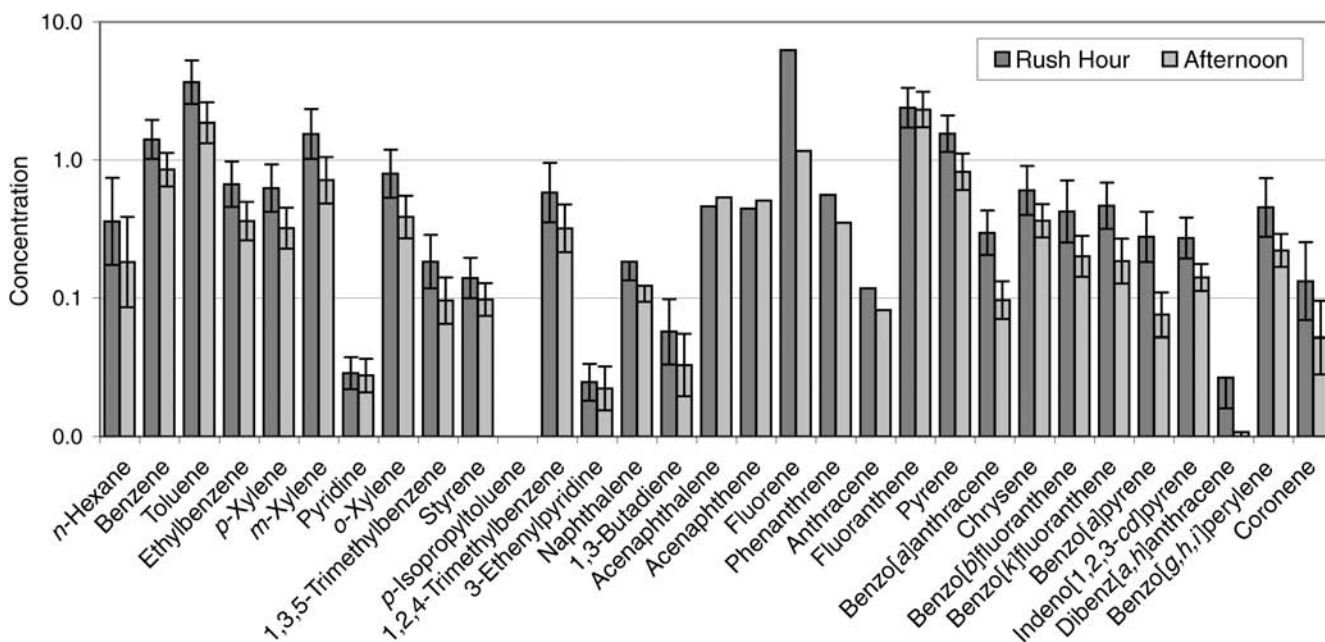


Figure 25. Street microenvironmental concentrations of VOCs (in $\mu\text{g}/\text{m}^3$) and PAHs (in ng/m^3), according to time of day with regard to traffic. Data are presented as the geometric mean \pm 95% CI (error bars, shown if there are more than five data points). For VOCs, $N = 50$ for rush hour and $N = 70$ for afternoon; for PAHs, $N = 30$ for rush hour and $N = 30$ for afternoon.

VOC concentrations in all bus, train, car, and London Underground microenvironments that were higher than those in the current study (Leung and Harrison 1999; Kim et al. 2001b). However, a study of VOC concentrations in cars in California in 1997 (Fedoruk and Kerger 2003) reported concentrations to those described here.

Of the transport vehicle microenvironments sampled, trains in London had the lowest VOC concentrations, and London buses and subways had the highest (Figure 26). Nevertheless, caution should be exercised in interpretation, as London transportation was sampled only twice per microenvironment type at rush hour and in daytime, and all sampling was carried out in autumn; in Birmingham, some samples were collected in summer and winter. Another microenvironment with low concentrations was the train in Birmingham. The sample size for this microenvironment was larger than for the London train; it appears that in general, trains are the commuting vehicle with the lowest VOC concentrations. These findings are consistent with those in other studies (Barrefors and Petersson 1996; Kingham et al. 1998; Leung and Harrison 1999; Lau and Chan 2003; Guo et al. 2004a). Cars and buses sampled in Birmingham show similar concentrations, although levels of *n*-hexane, benzene, and toluene are higher in cars than in buses. This finding is, however, divergent from those of other studies, in which cars have higher VOC concentrations than buses do (Jo and Park 1998; Carrer et al. 2000;

Kim et al. 2001b; Chan et al. 2003; Fedoruk and Kerger 2003; Shiohara et al. 2005). Since VOC concentrations in both cars and buses derive from the air on the road in front of the vehicle, these differences may not be important.

As regards the VOC levels recorded in various transport stations (Figure 27 and Appendix 13), the highest levels were recorded in car parks, although these values are lower than those reported previously (Leung and Harrison 1999). The lowest VOC levels were sampled in local train stations ($P < 0.01$), all of which were outdoors, dedicated exclusively to rail transit, and located away from road traffic. In contrast to local train stations, local bus stops showed the second highest concentrations after the car park, exceeding even the bus stations. This finding could be due to the fact that bus stations were sampled in the passenger waiting area, whereas local bus stops were in close contact with not only bus traffic but also general traffic and therefore had sources of pollution other than bus traffic. VOC levels measured in passenger areas in main train and bus stations (i.e., platforms and waiting areas, respectively) were similar. Both environments are influenced by the need of transport vehicles to idle for long periods to allow passengers to board and exit.

The diurnal traffic pattern is reflected in VOC concentrations measured within vehicles, with higher levels recorded during rush hour than during the day ($P < 0.05$). These results are similar to those reported by other researchers:

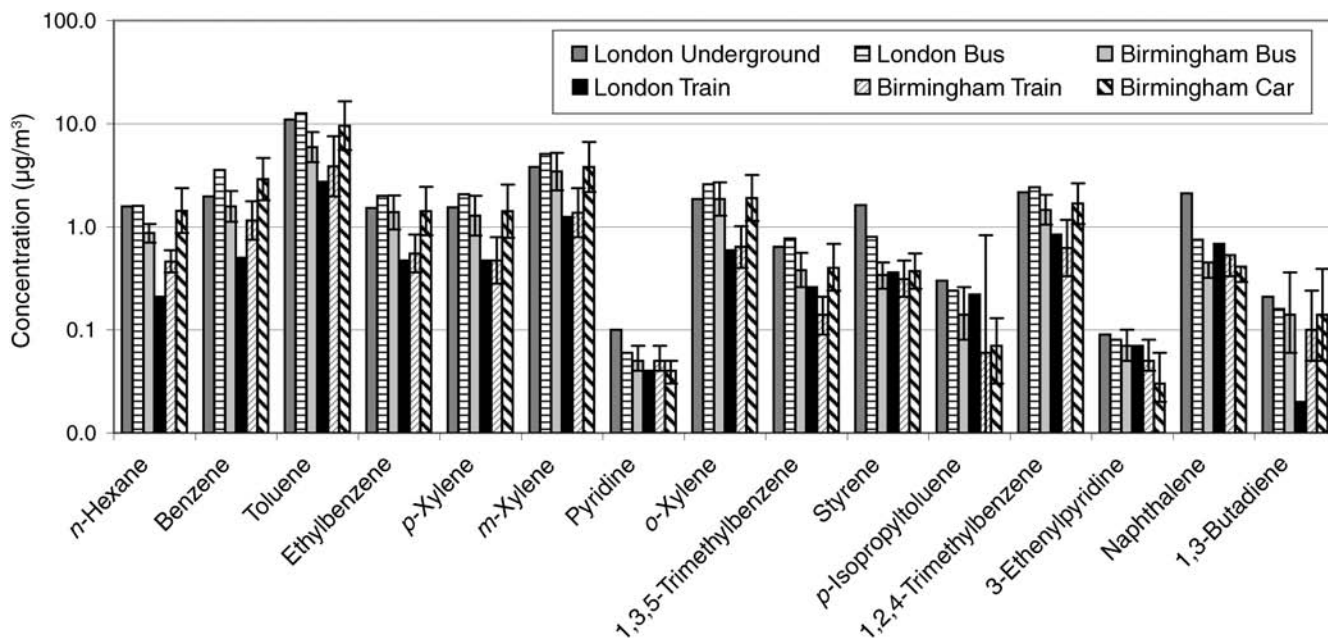


Figure 26. Mobile-transport microenvironmental concentrations of VOCs (in $\mu\text{g}/\text{m}^3$), according to mobile site. Data are presented as the geometric mean \pm 95% CI (error bars, shown if there are more than five data points). $N = 4$ for London Underground (subway), $N = 4$ for London bus, $N = 12$ for Birmingham bus, $N = 4$ for London train, $N = 11$ for Birmingham train, and $N = 12$ for Birmingham car.

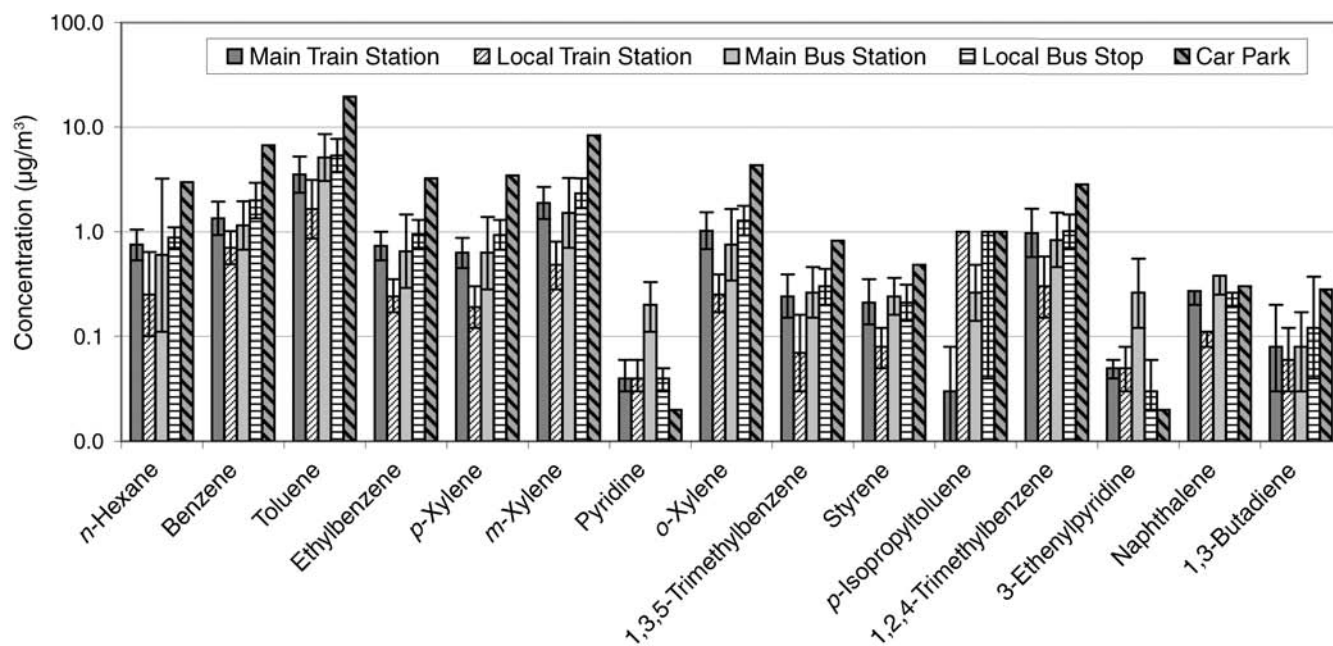


Figure 27. Transport-station microenvironmental concentrations of VOCs (in $\mu\text{g}/\text{m}^3$), according to station type. Data are presented as the geometric mean \pm 95% CI (error bars, shown if there are more than five data points). $N = 8$ for main train station, $N = 8$ for local train station, $N = 5$ for main bus station, $N = 8$ for local bus stop, and $N = 4$ for car park.

that VOC levels were higher during peak hours than non-peak hours (Leung and Harrison 1999; Chan et al. 2003; Fedoruk and Kerger 2003). In contrast, the diurnal traffic pattern does not apparently have much effect in transport stations. This might be due to the traffic there being constant and scheduled, rather than being concentrated in peak hours, resulting in less variation in the degree of traffic.

As regards the VOC concentrations measured in other indoor environments (Figure 28), caution should be taken when interpreting results obtained for hair salons, department stores, museums, and supermarkets, because only a small number of samples were collected. In contrast, more than four samples each were collected in pubs, restaurants, and libraries. Among these three microenvironments, pubs exhibited the highest VOC concentrations, especially for the two ETS-marker compounds (3-ethenylpyridine and pyridine). Restaurants and libraries showed very similar concentrations, except for the ETS markers, for which restaurants had higher concentrations. These results clearly reflect the practice of smoking, which is more frequent in pubs (before the smoking ban), less frequent in restaurants, which have ETS-free dining areas, and absent in libraries (in which smoking is forbidden). The average ratio for VOC compounds in ETS-exposed pubs and restaurants versus non-exposed pubs and restaurants is $(4.4 \pm 2.2):1$ (Figure 29). This result illustrates that the presence of ETS not only increases the levels of ETS-related compounds like

3-ethenylpyridine but also the levels of all VOC compounds ($P < 0.05$), as reported by other authors (Heavner et al. 1995; Kim et al. 2002).

With regard to a seasonal effect in microenvironmental VOC levels (e.g., Figure 30), all the microenvironments (i.e., homes, streets, vehicles, transport stations, and other indoor areas) show a clear pattern of higher VOC concentrations in the winter than the summer, both indoors and outdoors. In the home microenvironment, winter levels show significantly higher values than summer ($P < 0.01$ for most compounds), with an average ratio of GMs of $(1.5 \pm 0.4):1$. An exception is the two ETS-related compounds (3-ethenylpyridine and pyridine), which have ratios close to 1. The average ratio of winter and summer GMs in street microenvironments is $(1.3 \pm 0.8):1$. This overall pattern is consistent with findings in other studies measuring indoor and outdoor air in various seasons (Baek et al. 1997; Ilgen et al. 2001b; Mann et al. 2001; Schneider et al. 2001; Ho et al. 2004; Curren et al. 2006), reflecting the combined effects of lower outdoor concentrations, moderated by higher ventilation, and a lack of indoor combustion processes (Schneider et al. 2001).

PAHs

In this study, some sampling was carried out in microenvironments such as kitchens, living rooms, spare bedrooms, and backyards in homes other than the subjects'.

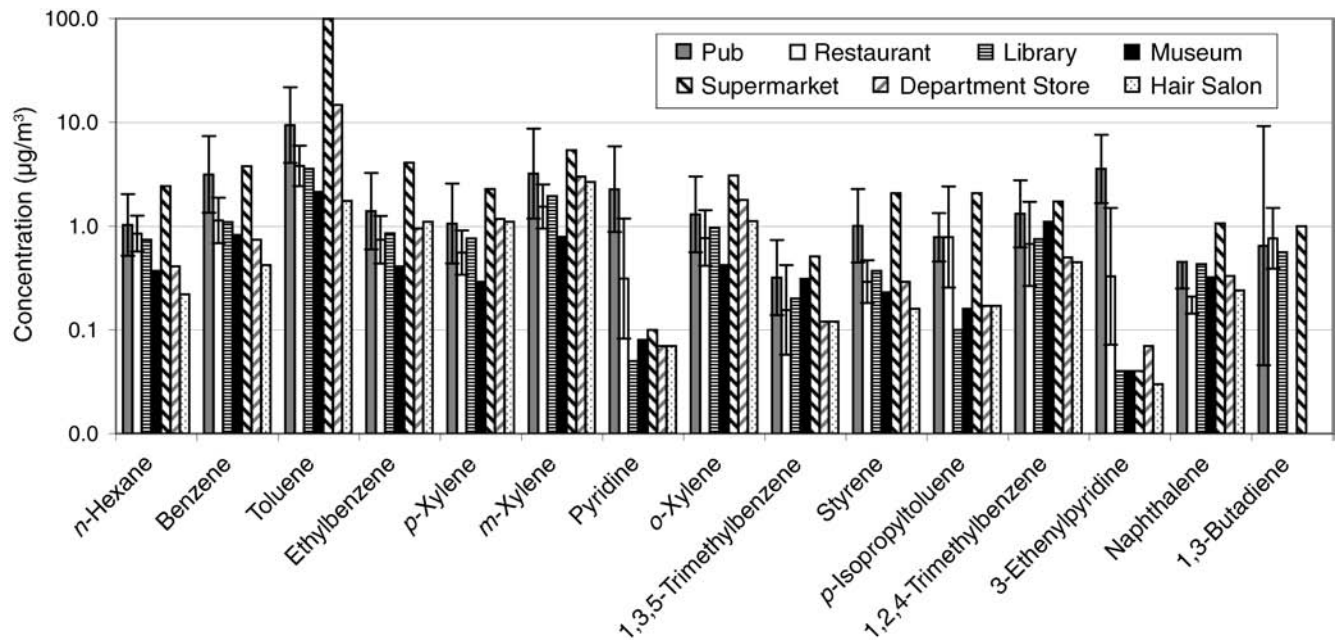


Figure 28. Indoor microenvironmental concentrations of VOCs (in $\mu\text{g}/\text{m}^3$), according to indoor site. Data are presented as the geometric mean \pm 95% CI (error bars, shown if there are more than five data points). $N = 10$ for pub, $N = 8$ for restaurant, $N = 4$ for library, $N = 2$ for museum, $N = 2$ for supermarket, $N = 1$ for department store, and $N = 1$ for hair salon.

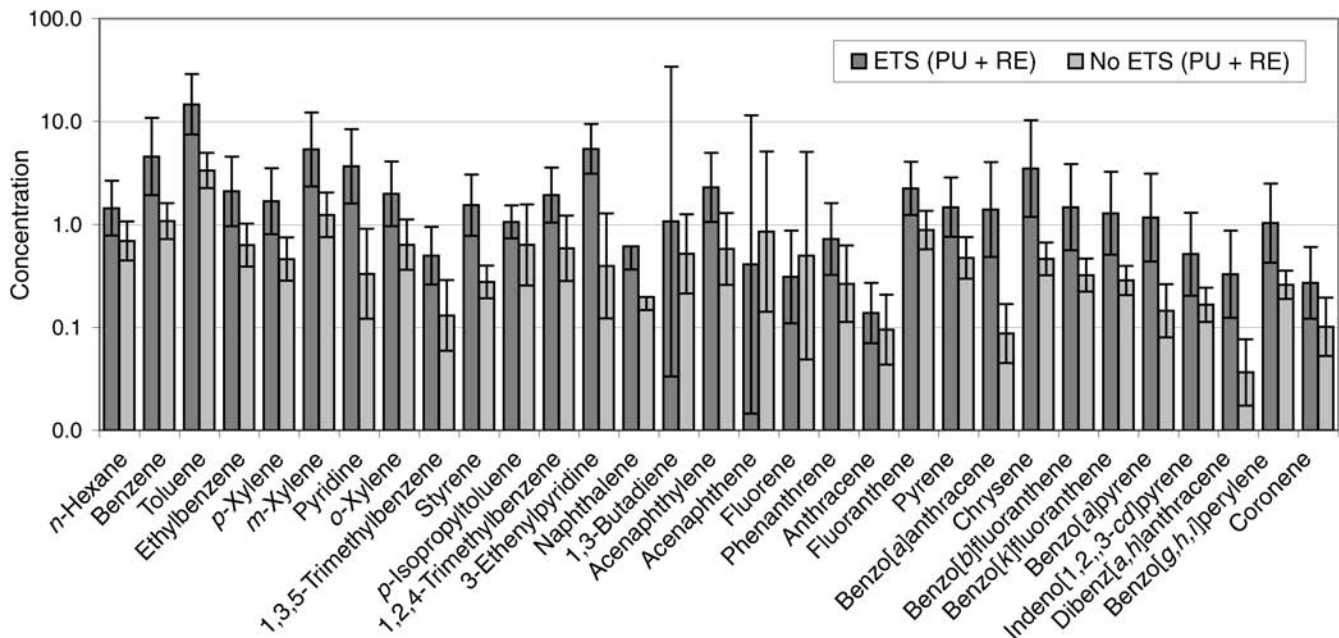


Figure 29. Pub and restaurant microenvironmental concentrations of VOCs (in $\mu\text{g}/\text{m}^3$) and PAHs (in ng/m^3), according to ETS exposure. Data are presented as the geometric mean \pm 95% CI (error bars). For VOCs, $N = 8$ for ETS exposure and $N = 10$ for no ETS exposure; and for PAHs, $N = 16$ for ETS exposure and $N = 20$ for no ETS exposure.

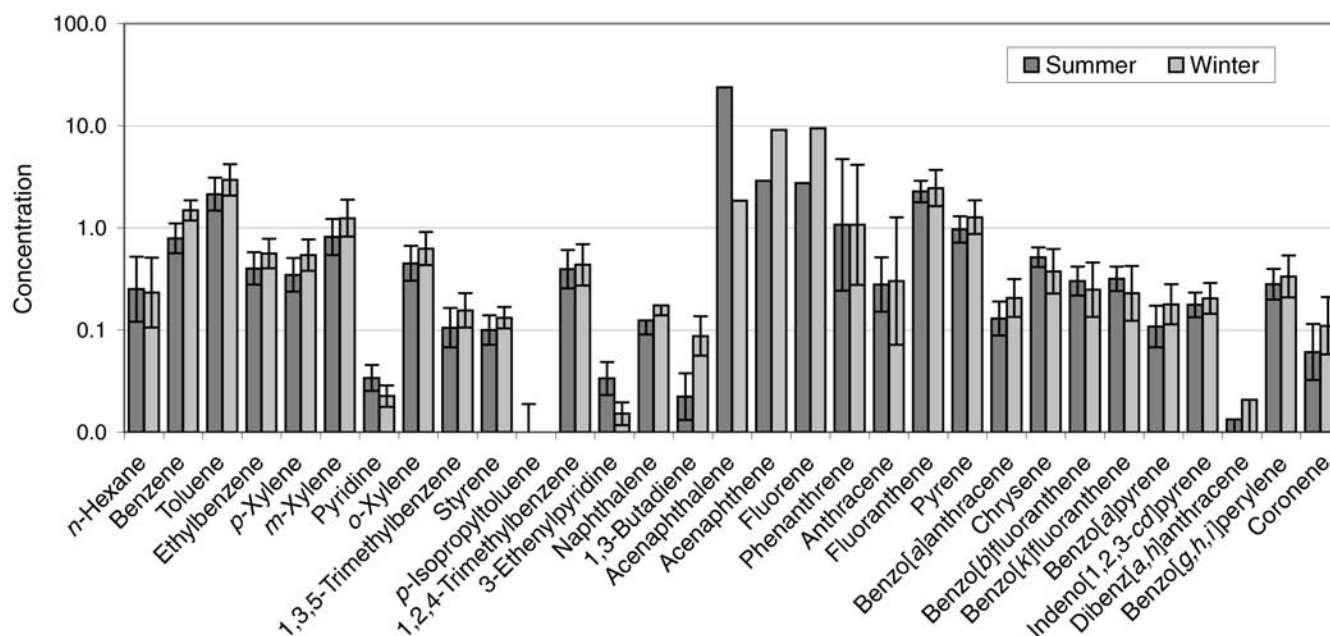


Figure 30. Street microenvironmental concentrations of VOCs (in $\mu\text{g}/\text{m}^3$) and PAHs (in ng/m^3), according to season. Data are presented as the geometric mean \pm 95% CI (error bars, shown if there are more than five data points). For VOCs, $N = 60$ for summer and $N = 60$ for winter; and PAHs, $N = 30$ for summer and $N = 30$ for winter.

The sample size for each of these microenvironments was small and cooking was not occurring in some of the kitchens sampled; therefore extrapolation to general home microenvironments might not be possible. Results of subsets of data with more than four representative data points will be discussed below.

We found microenvironmental concentrations of most PAHs to be highest in the living room, followed by the kitchen and then the bedroom. This ranking is consistent with that reported by Chuang and colleagues (1991). The relatively higher concentrations in kitchens and living rooms as compared to the bedrooms probably result from a lesser source influence in the bedrooms and the greater height of the bedrooms above traffic sources (Ilgen et al. 2001a). The ranking of the indoor concentrations is influenced by different ventilation conditions, house age, and indoor activities; it clearly indicates that indoor sources affecting the living room, such as ETS and fuel combustion, have a greater influence than do sources in the kitchen when cooking is not occurring. Our ranking is not consistent with that reported by Zhu and Wang (2003), who classified the concentrations in indoor air to be as follows: bedroom > kitchen > living room > balcony. Liu and associates (2001) argue that the air of the kitchen is polluted not only by outdoor air but also by indoor emissions such as cooking and use of other gas appliances. It seems that cooking oil fumes, if a stovetop hood is not in use, generate PAHs

as the oil evaporates in the air (Srogi 2007). Moret and Conte (2000) argue that, at high temperatures, organic compounds are partially broken down into unstable smaller fragments by pyrolysis and can then recombine to form stable PAHs (Moret and Conte 2000). The contribution of cooking oil fumes could not be tested in this study, as cooking was not ongoing when many of the kitchen samples were collected. Therefore, our ranking may be not able to be directly extrapolated to households where cooking is a regular activity.

When comparing PAH data collected in microenvironments in homes other than the subjects' in summer and in winter, the higher-molecular-weight group (benzo[a]-anthracene–coronene), with the exception of chrysene, have higher concentrations in winter than in summer ($P < 0.05$), consistent with the generally higher ambient concentrations and an increase in indoor sources such as heating at this time of year.

The effect of traffic on workplaces was examined by comparing offices located on trafficked roadsides (first-line offices) with offices located away from traffic (non-first-line offices) and offices located in the city center with offices located in a suburban area. The high-molecular-weight PAH concentrations were higher in first-line offices than in the other locations ($P < 0.05$ for benzo[a]anthracene, chrysene, and benzo[k]fluoranthene and $P < 0.10$ for benzo[b]fluoranthene and ideno[1,2,3-cd]pyrene). This

observation seems to be concordant with what was observed in first-line and non-first-line homes. As for homes, for offices the situation for the low-molecular-weight PAHs is less clear ($P > 0.10$). In general, high-molecular-weight PAHs showed higher concentrations in suburban than urban offices. This might be because suburban offices tend to be located at ground level, whereas urban offices could be located high above the street. Therefore, a dilution factor might exist for urban offices but not suburban offices.

As regards outdoor levels measured in streets (Appendix 11), the air quality in Birmingham seems to have improved, as evidenced by the concentrations of particulate-phase benzo[a]pyrene in 1996, 0.48 ng/m^3 (Harrison et al. 1996), and in 2008 in our study, 0.26 ng/m^3 . This trend is consistent with that in previous studies indicating a decrease in PAH levels in ambient air in Germany (Schauer et al. 2003) and the United States (Ospital et al. 2008). The benzo[a]pyrene values in this study are similar to, though generally lower than, typical values obtained elsewhere in Europe. Measurements taken by Menichini and coworkers (2007) at road level in high-traffic areas in Rome, Italy, gave mean values for benzo[a]pyrene varying from 0.7 to 2.3 ng/m^3 , with means of 0.82 ng/m^3 in urban Flanders, Belgium (Rockens et al. 2000), and 2.97 ng/m^3 in trafficked Naples, Italy (Caricchia et al. 1999). Some countries outside Europe may not yet have such stringent emission controls, and thus the benzo[a]pyrene levels are higher: 2.13 ng/m^3 in trafficked Hong Kong, China (Guo et al. 2003); 1.8 ng/m^3

in urban Mumbai, India, and 9.32 ng/m^3 in Lahore, Pakistan (Smith et al. 1996); and 37.01 ng/m^3 in trafficked Tainan, Taiwan (Sheu et al. 1997).

In street microenvironments (Figure 31), traffic is a major source of PAHs, and therefore, the magnitude of PAH concentrations should be associated with traffic volume. PAH concentrations measured in trafficked roadsides were generally higher ($P < 0.01$) than in other street types, with parks being the outdoor environment with the lowest recorded PAH concentrations; these data are consistent with traffic loads, as reported in previous studies (Lim et al. 1999; Wu et al. 2005; Chang et al. 2006).

As regards the effect of geographic location on PAH street concentrations (Figure 32), the measurements were not concurrent among the cities, and therefore meteorologic conditions during the sampling period might have affected the average concentrations considerably, given that the number of samples is quite limited. London had higher concentrations of PAHs such as fluorene, anthracene, indeno[*g,h,i*]perylene, and coronene than the other cities ($P < 0.05$). Birmingham had higher concentrations than Wales for fluorene, benzo[*g,h,i*]perylene, and coronene ($P < 0.01$). Wales showed the lowest concentrations of acenaphthalene, chrysene, and benzo[*k*]fluoranthene ($P < 0.05$). Phenanthrene and especially benzo[*g,h,i*]perylene and coronene are considered to be markers of emissions from gasoline-fueled cars. Coronene was found at higher concentrations in trafficked roadsides in London and Wales

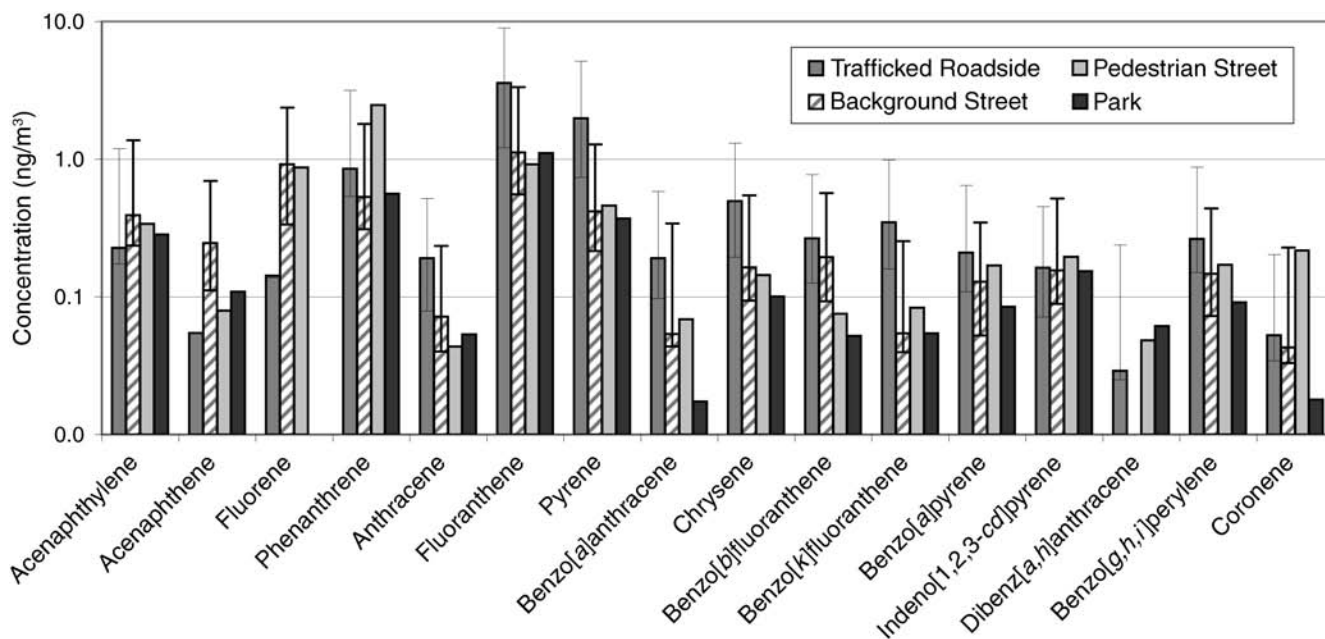


Figure 31. Street microenvironmental concentrations of PAHs (in ng/m^3), according to street site. Data are presented as the geometric mean \pm 95% CI (error bars, shown if there are more than five data points). $N = 8$ for trafficked roadside, $N = 4$ for pedestrian street, $N = 10$ for background street, and $N = 4$ for park.

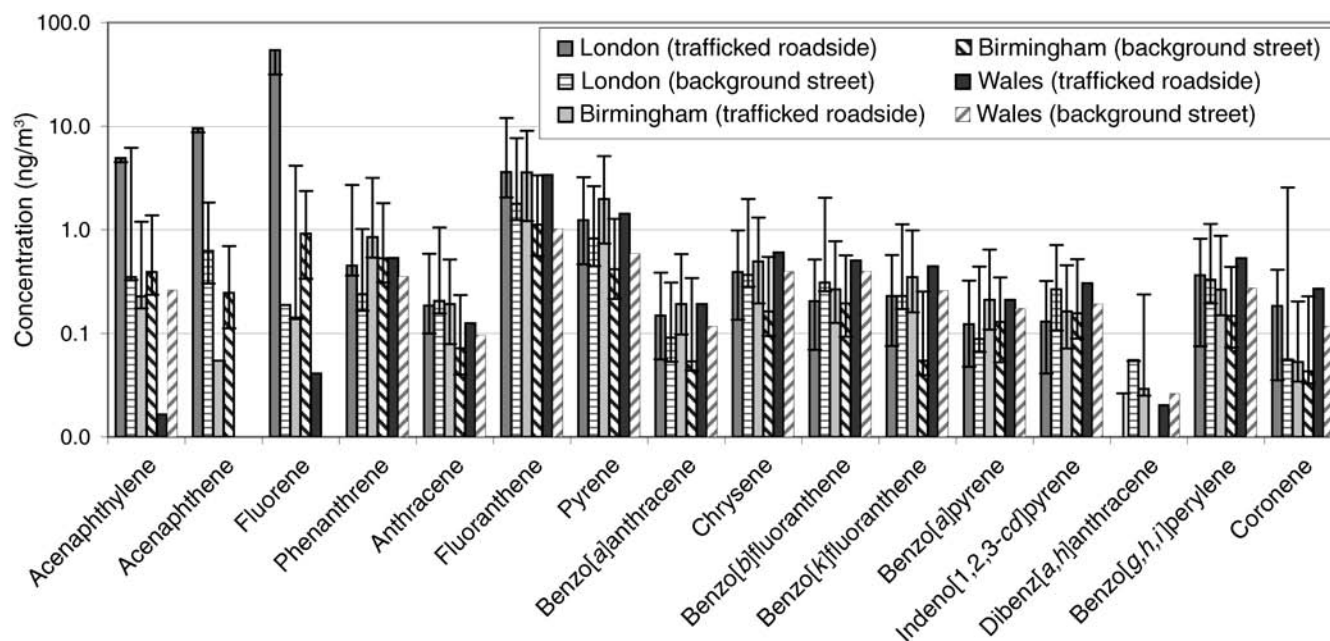


Figure 32. Street microenvironmental concentrations of PAHs (in ng/m^3), according to geographic location and street site. Data are presented as the geometric mean \pm 95% CI (error bars, shown if there are more than five data points). $N = 8$ for London trafficked roadside, $N = 4$ for London background street, $N = 8$ for Birmingham trafficked roadside, $N = 10$ for Birmingham background street, $N = 4$ for Wales trafficked roadside, and $N = 4$ for Wales background street.

than on background streets in the corresponding city. Unexpectedly, the coronene concentrations in Birmingham did not vary much among the street microenvironments. An interesting observation is the relatively high concentrations of acenaphthalene, acenaphthene, and fluorene measured on trafficked roadsides in London compared with the other cities, whereas in Birmingham these compounds were, on average, higher in background streets than on trafficked roadsides. As these are the highly volatile PAHs, the explanation may be the meteorologic conditions on the day of sampling. For the other four- and five-ring PAHs, the average concentrations on trafficked roadsides were generally higher than in background streets; however, the levels in Birmingham and Wales were very similar to those in London.

With regard to the effect of traffic in streets, higher PAH concentrations ($P < 0.05$) were recorded during rush hour than during the afternoon, when less traffic is present in the streets. These results are similar to those previously reported (Dubowsky et al. 1999; Lim et al. 1999; Sapkota and Buckley 2003; Chang et al. 2006).

Smith and coworkers (1996) found that in winter, at an urban site, the PAH concentrations were five times those measured in summer. This is consistent with measurements made on trafficked roadsides and background streets in Wales, which presented an average winter-to-summer ratio of 5 for benzo[a]pyrene. Data reported in a number of

previous studies are similar (Harrison et al. 1996a; Fromme et al. 1998; Georgiadis et al. 2001; Rehwagen et al. 2005; Tang et al. 2005; Wu et al. 2005). In London and Birmingham, the situation is less clear, which is consistent with the findings of Ohura and associates (2004b), who reported that seasonal differences for some high-molecular-weight PAHs were negligible. The small number of samples collected and the unclear findings may imply that the information gathered in our study is heavily dependent on the meteorologic conditions during sampling. A similar problem of interpretation was found for the benzo[g,h,i]perylene and coronene winter-to-summer ratios for the three areas.

Among all mobile-transport microenvironments measured (Figure 33), samples collected on the London Underground show the highest concentrations for phenanthrene, anthracene, fluoranthene, pyrene, benzo[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, indeno[1,2,3-cd]pyrene, dibenz[a,h]anthracene, benzo[g,h,i]perylene, and coronene ($P < 0.01$), whereas Birmingham trains showed the lowest concentrations, similar to the behavior of VOCs. When comparing transportation systems sampled in the two cities, in the London buses, the fluoranthene and pyrene concentrations were less than in Birmingham buses. For benzo[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, indeno[1,2,3-cd]pyrene, and dibenz[a,h]anthracene, the concentrations in London buses were higher than in

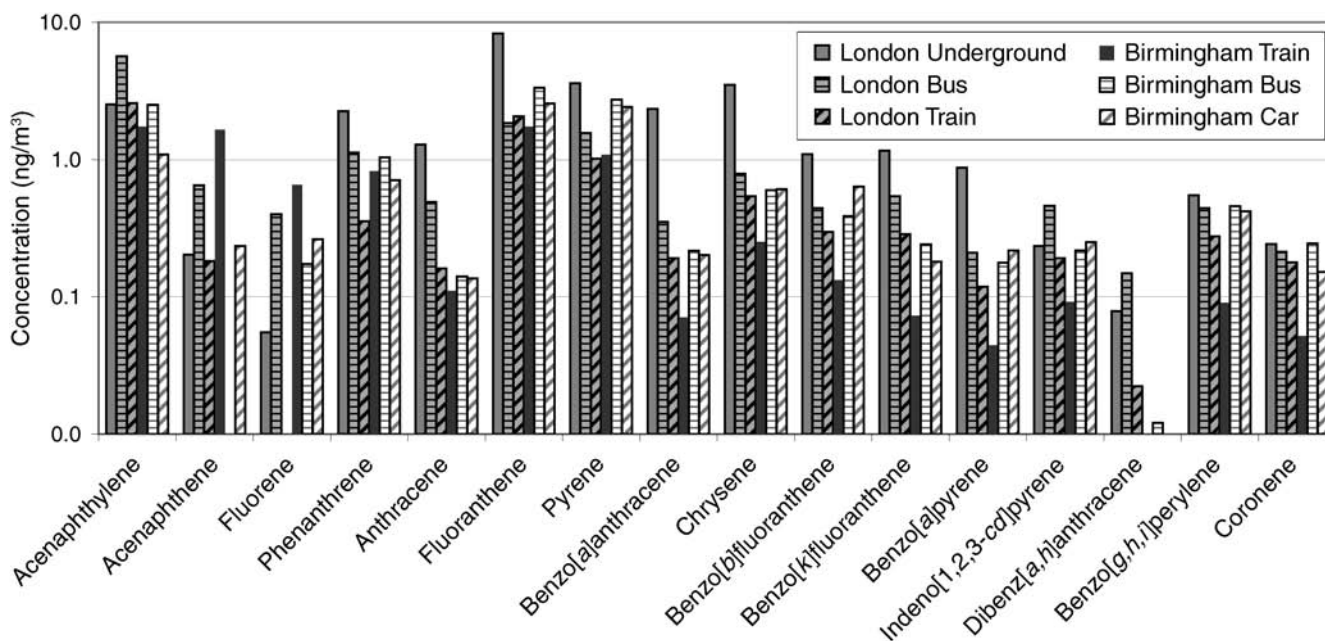


Figure 33. Mobile-transport microenvironmental concentrations of PAHs (in ng/m^3), according to mobile site. Data are presented as geometric means. $N = 4$ for London Underground (subway), $N = 4$ for London bus, $N = 3$ for Birmingham bus, $N = 4$ for London train, $N = 4$ for Birmingham train, and $N = 4$ for Birmingham car. No error bars are shown because the data are based on fewer than five data points.

Birmingham, whereas the benzo[*g,h,i*]perylene and coronene levels in the two settings are similar, probably due to infiltration from the surrounding emissions from gasoline-fueled cars. The PAH concentrations in gasoline-fueled cars, under various conditions of heating and ventilation, were similar to those in London buses. For trains, the lower-molecular-weight PAHs—acenaphthene, fluorene, and phenanthrene—had higher levels in Birmingham than in London; the finding was opposite for fluoranthene, pyrene, benzo[*a*]anthracene, chrysene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, benzo[*a*]pyrene, indeno[1,2,3-*cd*]pyrene, dibenz[*a,h*]anthracene, benzo[*g,h,i*]perylene, and coronene.

In transportation systems, mobile and stationary, very little PAH monitoring has been carried out to date. Leutwyler and colleagues (2002) took particle-phase PAH measurements in an underground electric train commuting non-stop from Zurich to Bern, Switzerland. The train contained a mechanical ventilation system to regulate the indoor air quality, as the windows could not be opened, and the train contained cabins for smokers and cabins for non-smokers. Although the authors do not give information on individual PAHs, the indoor PAH concentrations were two to three times higher than the outdoor concentrations and five times higher in smoker cabins than non-smoker cabins. Fromme and coworkers (1998) conducted sampling in an underground train in a non-smoker cabin and in a two-year-old

car fitted with a three-way catalyst that traveled the same route. The mean benzo[*a*]pyrene values in the car were $1.0 \text{ ng}/\text{m}^3$ in summer and $3.2 \text{ ng}/\text{m}^3$ in winter. In the subway, the mean benzo[*a*]pyrene values were $0.7 \text{ ng}/\text{m}^3$ in summer and $4.0 \text{ ng}/\text{m}^3$ in winter. Levy and colleagues (2002) conducted PAH sampling in buses and subways, with the former having higher PAH concentrations. Their results differs from ours, with the highest benzo[*a*]pyrene level obtained in the London Underground ($3.43 \text{ ng}/\text{m}^3$) (Figure 33) and the mean level in a gasoline-fueled car was $0.25 \text{ ng}/\text{m}^3$; both of these measurements are lower than those in the abovementioned studies. The mean benzo[*a*]pyrene was $0.10 \text{ ng}/\text{m}^3$ for Birmingham trains, all of which are electric—a measurement lower than that obtained in the Underground. However, trains in Birmingham do not travel underground. The PAH concentrations were highest at main train stations and local bus stops (Figure 34), possibly because of the stop-and-go movement of diesel-powered buses that might lead to higher emissions. Train stations are located at a distance from roads, whereas local bus stops, besides being exposed to bus emissions, have as a major source of PAHs the passing traffic. These results should be viewed in the context of the atmospheric conditions.

As regards the effect of traffic in transport microenvironments, higher PAH concentrations were recorded at rush hour than during the afternoon ($P < 0.05$) in mobile-transport

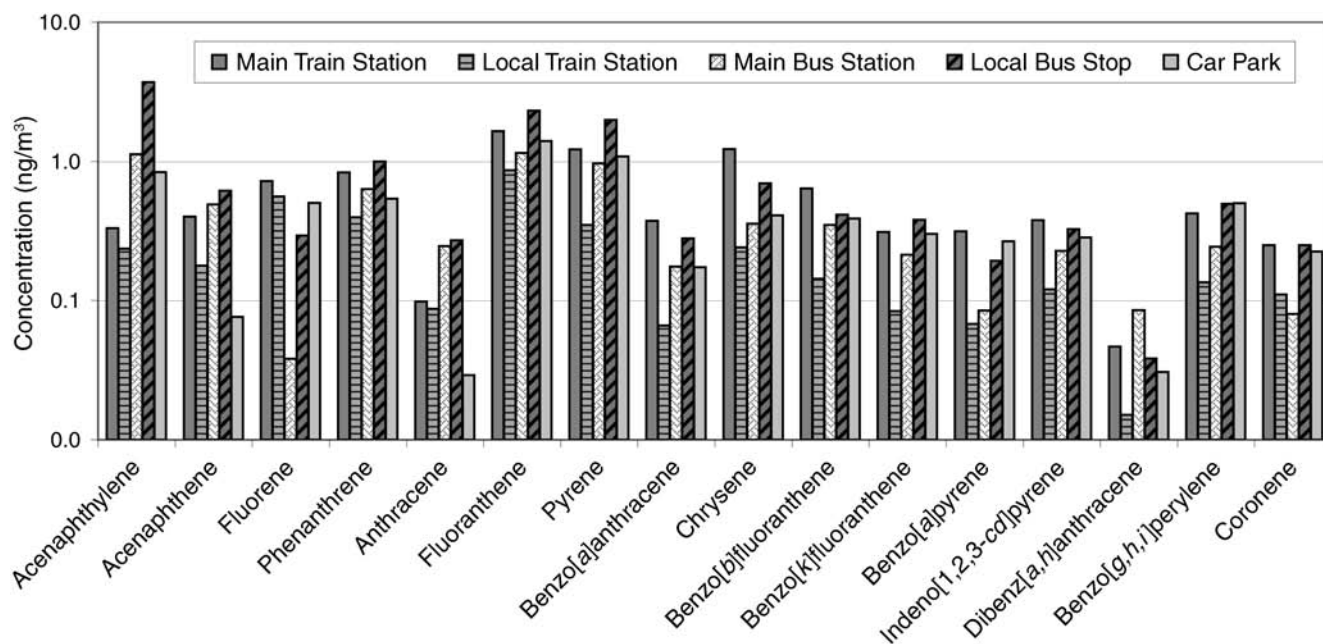


Figure 34. Transport-station microenvironmental concentrations of PAHs (in ng/m^3), according to station type. Data are presented as the geometric mean. $N = 4$ for main train station, $N = 4$ for local train station, $N = 4$ for main bus station, $N = 7$ for local bus stop, and $N = 2$ for car park. No error bars are shown because the data are based on fewer than five data points.

settings, but similar concentrations were obtained in transport stations ($P > 0.10$). These results are similar to previously reported findings (Chang et al. 2006) and with the results obtained here for the VOCs.

The seasonal effect in transport environments consisted of higher concentrations in winter than in summer for both mobile-transport microenvironments and stations.

The indoor environments sampled were pubs and restaurants, chosen as typical social places that subjects visit frequently. A library and museum were also sampled. The pubs were dominated by high concentrations of high-molecular-weight PAHs, from ETS ($P < 0.05$). This result is consistent with those published previously that implicate ETS as the main contributor to PAHs indoors (Chuang et al. 1991; Phillips 1994; Mitra and Ray 1995; Harrison et al. 1996; Liu 2001; Ohura et al. 2004a; Gee et al. 2005; Lu 2006). On average, the PAH concentrations were higher in pubs than in restaurants (Figure 35), because the latter had restricted smoking or had a restricted area for smoking within the eating area, unlike in pubs. Pubs can be considered a microenvironment that is ETS-dominated, with homogeneous, well-mixed air, unless an efficient ventilation system is used to exchange indoor air effectively with outside air, thus minimizing the effect of a strong source like ETS. All the sampling in our study was carried out before the smoking ban was instituted in England, on July 1, 2007.

Regarding the seasonal effect in pubs and restaurants, lower concentrations were sampled in summer than in winter ($P < 0.01$). A likely explanation for the noticeable increase of PAH concentrations in winter is that in winter, owing to the cold weather, people tend to stay inside the pubs, which could lead to an increase in the number of cigarettes smoked per night. Additionally, air conditioning systems may be switched off and windows not opened, thus reducing the exchange of air into and out of the pub. In summer, the situation probably changes, with better ventilation and more use of beer gardens or other open spaces. The winter-to-summer ratio for benzo[a]pyrene levels in pubs shows that if there is not an efficient ventilation system exchanging the air between the ETS environment and the outside, the exposure to PAHs will be extremely high. In restaurants, the situation is slightly better, with a winter-to-summer ratio of 3.5 for benzo[a]pyrene; most of the restaurants had already started to enforce a non-smoking environment at the time of sampling.

SUMMARY OF PERSONAL EXPOSURES AND MICROENVIRONMENTAL CONCENTRATIONS

Comparison of the measured personal exposure levels with the levels in the home and workplace microenvironments and outdoor microenvironments shows a pattern of personal exposure concentrations being highest, followed closely by home and in-vehicle concentrations, workplace

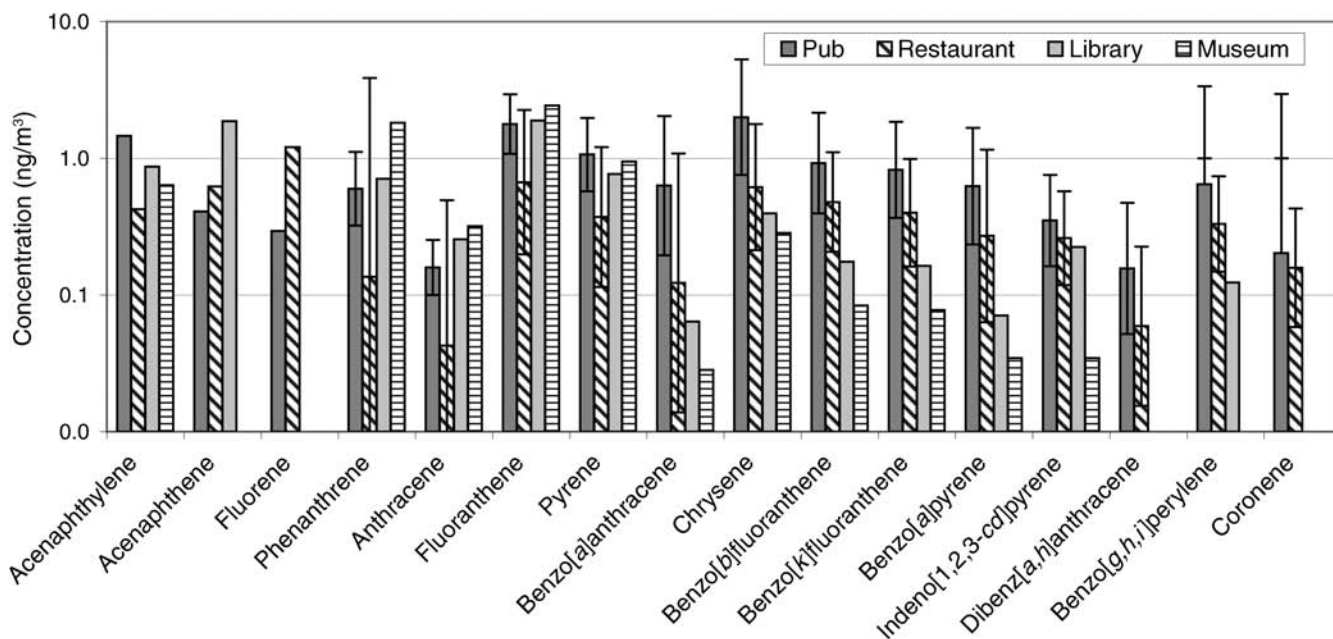


Figure 35. Indoor microenvironmental concentrations of PAHs (in ng/m^3), according to indoor site. Data are presented as the geometric mean \pm 95% CI (error bars, shown if there are more than five data points). $N = 11$ for pub, $N = 7$ for restaurant, $N = 4$ for library, and $N = 2$ for museum.

levels, and lastly, outdoor levels (see Figure 4 for VOCs and Figure 5 for PAHs). Exceptions to this trend were ETS-related compounds (3-ethenylpyridine, pyridine, and 1,3-butadiene), for which the highest levels were in pubs and restaurants. The close relationship between personal exposure concentrations and home microenvironmental concentrations is due to the fact that subjects spent between 58% and 67% of their time in their own house. Therefore, concentrations seem to be controlled by home indoor sources and are modified according to the activities carried out and places visited during the day.

These results are consistent with those of previous studies in which researchers reported personal exposure levels of VOCs to be, on average, higher than concentrations measured in the home or workplace and much higher than the concentration in outdoor air in low-traffic areas (Wallace 1996; Gonzalez-Flesca et al. 2000; Edwards et al. 2001b; Ilgen et al. 2001c; Kim 2001). In contrast, other researchers have shown home levels to be higher than personal exposure levels for children, who spend longer periods outdoors (Adgate et al. 2004a), or that aromatic compound levels are highest in the workplace, followed by personal exposures, and then residential indoor and residential outdoor concentrations (Lai et al. 2004). Nevertheless, all the studies agree that outdoor concentrations fall well below indoor concentrations (Phillips et al. 2005).

On the other hand, concentrations measured in transport vehicles were in some cases lower than, and in other

cases similar to, those in homes. Office VOC concentrations were always lower than personal exposures or levels in home or transport microenvironments. Finally, the lowest VOC concentrations were recorded consistently in the streets, where natural ventilation favors the dispersion of the pollutants. The studied population spent just 2% to 5% of their time outdoors, where air is less polluted with VOCs than indoors or in vehicles.

As regards PAH levels in various microenvironments, office concentrations of PAHs, unlike VOCs, were similar to personal exposure and home concentrations. This result may indicate that the sources of PAHs are similar in the office and the home, given that such a large proportion of time is spent inside in either place. Unlike VOCs, PAHs were not at the lowest levels in the streets. The highest levels of PAHs—especially benzo[*a*]anthracene, chrysene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, benzo[*a*]pyrene, indeno[1,2,3-*cd*]pyrene, dibenz[*a,h*]anthracene, benzo[*g,h,i*]perylene, and coronene—were found in pubs and restaurants, where ETS, a major source of PAHs, dominates. Transportation systems and stations exhibit the second-highest exposures to phenanthrene, anthracene, fluoranthene, pyrene, benzo[*a*]anthracene, chrysene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, benzo[*a*]pyrene, indeno[1,2,3-*cd*]pyrene, dibenz[*a,h*]anthracene, benzo[*g,h,i*]perylene, and coronene. The high levels of benzo[*b*]fluoranthene and benzo[*k*]fluoranthene may indicate exposure to diesel emissions, and high levels of

benzo[*g,h,i*]perylene and coronene indicate exposures to gasoline emissions.

URINARY BIOMARKERS

Initial analysis for the specific benzene biomarker *S*-phenylmercapturic acid (S-PMA) in urine was carried out. The 24-hour mean airborne benzene concentrations to which each subject was exposed were plotted against levels of S-PMA (normalized to the creatinine level) detected in urine samples collected just after the exposure period (Figure 36). The correlation was very poor ($R^2 = 0.03$) and the S-PMA concentrations appeared to be unreasonably high (20–25 nmol/L). A subsequent literature review revealed that the proposed method was not suitable for low concentrations of benzene (Kivisto et al. 1997; Fustinoni et al. 1999; Farmer et al. 2005; Fustinoni et al. 2005a,b), and therefore the analysis was stopped.

Urinary metabolites of ETS and PAHs were analyzed and compared with the respective parent air toxic compounds. The figures presented in this study (e.g., Figure 6) show a trend in the exposure–response relationship for the selected air pollutant and its urinary biomarker, with regard to microenvironmental exposures. Nevertheless, the data gathered in this study are not sufficient to determine exposure–response relationships, as there are several confounding factors (e.g., sex, age, dietary exposures) that were not taken into account into this analysis.

The effect of exposure to ETS was studied. The measured concentrations were higher for *trans*-3'-hydroxycotinine

than cotinine (Table A16.1 in Appendix 16), whereas cotinine levels correlated better with levels of ETS-related VOCs than *trans*-3'-hydroxycotinine levels ($R = 0.74$ for cotinine and $R = 0.68$ for *trans*-3'-hydroxycotinine, vs. 3-ethenylpyridine; Table 17). Cotinine is the primary metabolite of nicotine, is very stable in the body (half life, approximately 18–20 hours), and can be reliably measured in blood, saliva, and urine for monitoring nicotine exposure in humans (Bernert et al. 1997). However, consistent with our results, Tuomi and colleagues (1999) suggest that even though cotinine has been used extensively as a nicotine marker in the urine of both active and passive smokers, *trans*-3'-hydroxycotinine is the predominant nicotine metabolite, corresponding to 40% of the total nicotine excretion, and thus it should be measured along with cotinine when monitoring passive ETS exposure (Tuomi et al. 1999).

Previous studies have indicated that levels of urinary cotinine in non-smokers are usually < 20 µg/L and that the threshold values distinguishing active and passive smokers are 50–100 µg/L (Carrer et al. 2000). In our study, the mean urinary cotinine level was 2.33 µg/L, showing that the non-smokers had ETS exposures near the lower end of the range. The samples identified as outliers that we excluded from the analysis had cotinine levels well above 120 µg/L.

Concentrations of urinary cotinine and *trans*-3'-hydroxycotinine, both normalized to the creatinine level, were higher for ETS-exposed subjects and were highly correlated with the gas-phase VOC ETS markers 3-ethenylpyridine and 1,3-butadiene (Figure 7). On the other hand, there seems to be no significant difference between the ETS-exposed and non-ETS-exposed subjects for the PAH metabolites analyzed. Interestingly, chrysene exposures were very strongly correlated with levels of the ETS markers 3-ethenylpyridine, cotinine, and *trans*-3'-hydroxycotinine (Table 18). This is consistent with earlier work emphasizing that chrysene is a main constituent of sidestream smoke, the primary contributor to ETS (Georgiadis et al. 2001).

The effect of normalizing with creatinine has been assessed by comparing the correlations of creatinine-normalized biomarkers with ETS-related VOCs and PAHs and the corresponding correlations of the non-normalized data (Figure 37, Table 17, and Table 18). Non-creatinine normalized data for cotinine show a significant correlation with 3-ethenylpyridine ($R^2 = 0.46$), with 1,3-butadiene ($R^2 = 0.28$), and with chrysene ($R^2 = 0.64$) (among others), and all three correlation coefficients are lower when the creatinine-normalized data are used. This begs the question of the appropriateness of normalizing the ETS urinary biomarkers levels with creatinine. Thomson and coworkers (1990) reported that the correlation between the cotinine concentration and data for serum collected from smokers

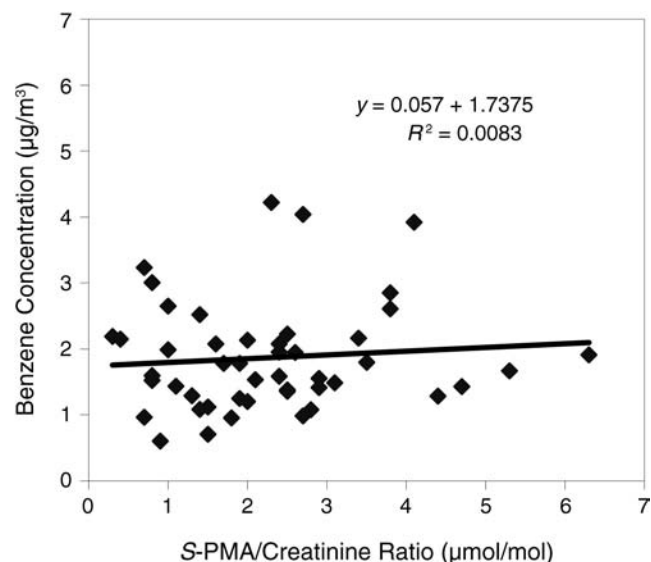
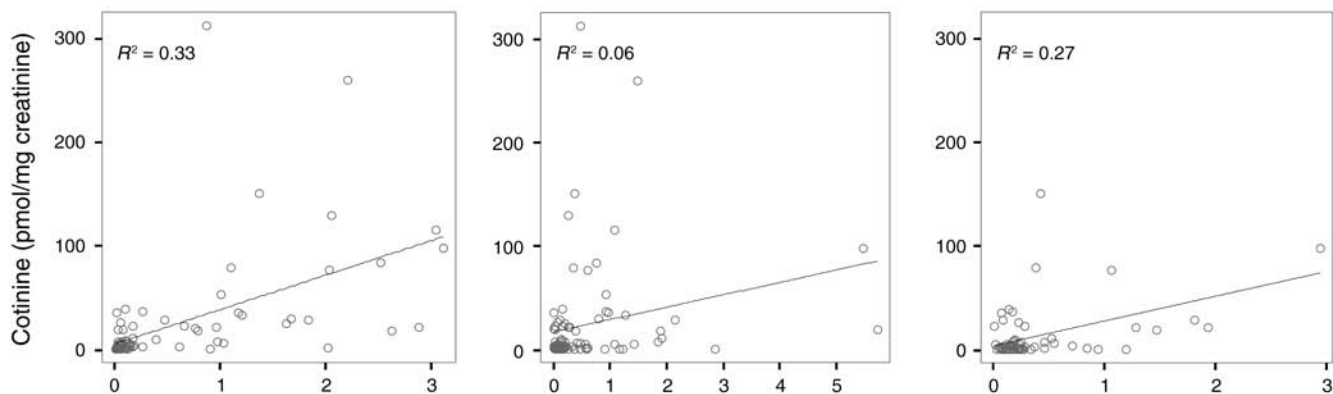


Figure 36. Benzene concentration (24-hr time-weighted average), according to S-PMA/creatinine ratio in urine ($N = 35$).

A Creatinine-normalized data



B Non-creatinine-normalized data

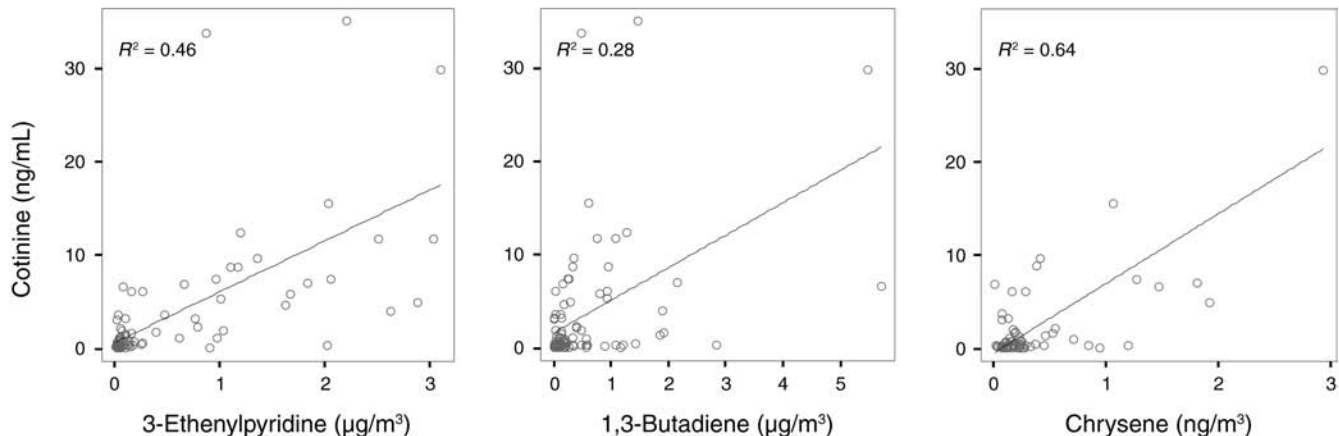


Figure 37. Significant correlations between urinary biomarkers and personal exposures to selected VOCs and a PAH, shown after (A) or before (B) normalization for creatinine level ($N = 92$ for 3-ethenylpyridine and 1,3-butadiene and $N = 68$ for chrysene). The figure shows correlations for which the two-tailed P was < 0.05 and the Pearson correlation coefficients were > 0.4 .

increased significantly by adjusting the urinary cotinine levels for the urinary creatinine concentration. The adjustment they made was based on the observed regression relationship between urinary cotinine and urinary creatinine, instead of the common method of expressing urinary cotinine and urinary creatinine as a ratio (Thompson et al. 1990).

Along with the traditional method of normalizing biomarker data with creatinine (i.e., dividing the biomarker concentration by the creatinine concentration), we also tested the method proposed by Thompson and associates (1990). In our study, however, there was a very weak correlation between the \log_{10} -transformed cotinine and \log_{10} -transformed creatinine data ($N = 92$; $R^2 = 0.004$) and for \log_{10} -transformed *trans*-3'-hydroxycotinine and \log_{10} -transformed creatinine levels ($N = 92$; $R^2 = 0.07$), suggesting that the method of Thomson and colleagues (1990) is

not valid for non-smokers. The fact that ETS biomarkers (i.e., cotinine and *trans*-3'-hydroxycotinine) and creatinine do not correlate might indicate that the two types of urinary substances have different excretion mechanisms. This might explain the fact that the ETS-related compounds are better correlated with the parent compounds when the data are not normalized (Figure 37).

As regards the PAH urinary metabolites, Jacob and coworkers (2007) reported that levels of the hydroxyfluorenes, hydroxyphenanthrenes, 1-hydroxypyrene, and 2-naphthol were all significantly higher in smokers than non-smokers (Jacob et al. 2007; Wilhelm et al. 2007). In our study, there were no distinct differences between the ETS-exposed and non-ETS exposed groups, either because the ETS exposure was not high enough to reveal a difference (as it depends on the number of cigarettes smoked, duration of exposure, distance from smokers, and degree of

ventilation) or because other sources of PAHs contribute to higher exposure in the non-exposed group (Figure 7). Nor was there a correlation between the abovementioned metabolites and the ETS metabolites or the ETS VOC markers, perhaps because the subjects in our study who were ETS-exposed had far lower exposures than smokers have, and this study did not include smokers. However, cotinine and *trans*-3'-hydroxycotinine levels showed a correlation ($P < 0.05$) with exposures to the low-molecular-weight PAHs (naphthalene, acenaphthylene, acenaphthene, fluorene, phenanthrene, and anthracene), which include the PAH parent compounds.

It is unfortunate that the PAH metabolites measured in urine derive from the low-molecular-weight compounds for which our exposure data are not reliable, as the sampling technique collects data on the particle phase only, and these compounds are mainly found in the gas phase. Therefore, we observed little correlation of PAH urinary metabolites and parent compounds. The personal exposure data for naphthalene should be reliable, as it was measured with the VOCs. The relatively weak correlation between naphthalene exposures and the 2-naphthol data (normalized to creatinine) is probably due to other, non-respiratory sources of naphthalene exposure, of which there are many (Price and Jayjock 2008). Dietary exposure to PAHs may be a greater source of PAH intake into the body than is exposure to airborne PAHs, as the estimated average adult dietary intake of benzo[*a*]pyrene and benzo[*a*]anthracene was 1.6 and 0.8 ng/kg of body weight per day, respectively, in 2000 in the United Kingdom (Food Standards Agency 2002). The U.K. air quality standard for benzo[*a*]pyrene is 0.25 ng/m³; thus, assuming a 70-kg person inhales 20 m³ of air daily, benzo[*a*]pyrene intake from the atmosphere is much lower than the estimate, at 0.07 ng/kg body weight per day.

On the other hand, high correlations were observed between the ETS urinary biomarkers and the low-molecular-weight PAH compounds (Table 18). This finding may suggest that the ETS urinary biomarkers studied are good biological indicators of personal exposure to some PAHs.

CORRELATIONS WITHIN AND BETWEEN THE VOC AND PAH DATABASES

Correlations among various VOCs and PAHs were examined for both personal exposures and microenvironments (Appendix 17). These correlations might suggest some sources.

Generally, personal exposures and home and workplace microenvironments all showed similar correlations among the compounds. The strongest correlations were between ethylbenzene and the xylenes ($R > 0.9$) and between the two trimethylbenzenes and the two ETS markers ($R > 0.94$).

The strength of the correlations may be the result of associations with traffic, use of solvents, and ETS. Although VOC concentrations were elevated in smoking environments, only pyridine was correlated significantly with 3-ethenylpyridine, as was reported by Heavner and colleagues (1995). The correlation between pyridine and 3-ethenylpyridine was not found in workplaces, however, where smoking is forbidden. High correlations were observed among the high-molecular-weight PAHs ($R > 0.8$), which could be in association with traffic or combustion sources. When analyzing the correlations among VOCs and PAHs in the personal exposure data set, we found correlations between benzene and the high-molecular-weight PAHs; between *m*-xylene and the low-molecular-weight PAHs; between 3-ethenylpyridine and compounds such as benzo[*a*]anthracene, chrysene, benzo[*b*]fluoranthene and benzo[*a*]pyrene; and between 1,3-butadiene and phenanthrene, anthracene, benzo[*a*]anthracene, and chrysene. Benzene and naphthalene correlate with the high-molecular-weight PAHs, and in the office microenvironment, so do ethylbenzene, the xylenes, and styrene. These correlations suggest that the high-molecular-weight PAHs and benzene may share the same source, which the literature suggests is traffic (Lim et al. 1999; Ho and Lee 2002) or combustion processes (Levy et al. 2002; Lung et al. 2003) such as ETS (McBride et al. 1999; Georgiadis et al. 2001; Levy et al. 2001).

The street and all transport microenvironments had an increased number of correlating compounds, including benzene, toluene, ethylbenzene, and the xylenes being correlated with the trimethylbenzenes, *n*-hexane, styrene, and naphthalene (e.g., $0.85 < R < 0.99$ in street and $R > 0.75$ in mobile-transport settings). This correlation among VOCs is attributed to traffic (Kim et al. 2001b). The correlation between the high-molecular-weight PAHs was, however, lower than that seen for personal exposures and indoor samples. Correlations between VOC and PAH compounds were stronger, including among the high-molecular-weight PAH compounds and the VOCs well known to be generated from gasoline and diesel combustion, a finding consistent with traffic being a single source (Lim et al. 1999; Dimashki et al. 2001; Levy et al. 2001; Sakai et al. 2002).

The correlations observed among the PAHs were weaker in street microenvironments than in all the indoor microenvironments studied. This may be a consequence of the influence of regional atmospheric transport and dilution on outdoor PAH concentrations. A weaker correlation was also observed in transport-station microenvironments within the PAH data set, possibly due to the effect of ventilation and the distance between the source and the sampling point.

Correlations found in ETS-influenced indoor environments clearly showed the influence of tobacco smoke. VOCs

were well correlated, with $R > 0.7$ ($P < 0.05$). Specifically, benzene and toluene were correlated; toluene, ethylbenzene, and the xylenes were correlated with trimethylbenzenes; 3-ethenylpyridine was correlated with pyridine; and naphthalene was correlated with xylenes and styrene. All the high-molecular-weight PAHs were correlated not only with one another ($R > 0.75$) but also with VOCs ($R > 0.75$). This suggests a similar predominant source of both pollutant types, namely ETS (McBride et al. 1999; Georgiadis et al. 2001; Levy et al. 2001).

SOURCE APPORTIONMENT BY FACTOR ANALYSIS

Three potential sources of VOCs affecting personal exposure were studied: traffic, ETS, and integral garages. As previously discussed, having an integral garage at home and being exposed to ETS leads to an increase in personal exposure to VOCs; hence, these two sources have been evaluated.

Although a number of factors were extracted from our data, here we discuss, and use for source apportionment, only the factors that explained most of the variance in the original data set. Factor analysis performed on all pooled personal exposure data for VOCs reveals two factors (Table 19). High loadings of benzene, toluene, ethylbenzene, the xylenes, trimethylbenzenes, *n*-hexane, styrene, and naphthalene are associated with a mix of sources such as vehicle use and solvents, the first factor (Mukund et al. 1996; Hinwood et al. 2006; Brown et al. 2007; Song et al. 2007). The second factor is associated with ETS, owing to the high loadings of the ETS markers 3-ethenylpyridine and pyridine (Kim et al. 2002).

The same two factors were present in other factor analyses performed on other subsets of the VOC database, demonstrating the importance of fossil fuel combustion, solvent use, and ETS in everyday personal exposures. Analysis according to the presence of integral garages revealed a third significant factor, with high loadings of trimethylbenzenes, styrene, *p*-isopropyltoluene, and 1,3-butadiene.

As regards the PAH data set, factor analysis performed on all pooled personal exposure data revealed two factors (Table 20). Factor 1 presented high loadings of all the high-molecular-weight PAHs (benzo[*a*]anthracene, chrysene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, benzo[*a*]pyrene, indeno[1,2,3-*cd*]pyrene, dibenz[*a,h*]anthracene, benzo[*g,h,i*]perylene, and coronene), which could be associated with gasoline emissions and combustion sources (Lim et al. 1999; Chang et al. 2006). Factor 2 could be associated with cooking (Oanh et al. 2000; Zhu and Wang 2003) or diesel-vehicle emissions (Chang et al. 2006), because of the high loadings of low-molecular-weight PAHs.

The same two factors were present in other factor analyses performed on other PAH data sets (related to ETS and integral garages), demonstrating the importance of traffic, combustion sources, and cooking on personal exposure to PAHs.

On the other hand, when performing factor analysis on the combined VOC and PAH database (Table 21), the previous factor 1 identified in the VOC data set, which explained 48% of the variance and was associated with a mixture of fuel combustion and use of solvents, was now broken into two different factors explaining 34% and 30% of the variance in the combined data set. The new factor 1 in the combined data set is related to fossil fuel combustion, as it has high loadings of most aromatic compounds and all the PAHs. This factor is consistent with suggestions in the literature (Lim et al. 1999; Ho and Lee 2002; Kim et al. 2002; Levy et al. 2002; Sakai et al. 2002). The second new factor is associated with the use of solvents, as it has high loadings of aromatic hydrocarbons but negative loadings for PAHs, as reported by others (Watson et al. 2001; Choi and Ehrman 2004; Guo et al. 2004b). Factor 3 is related to ETS, as it groups pyridine and 3-ethenylpyridine, whereas factor 4 appears to be linked with use of consumer products.

PERFORMANCE OF THE PERSONAL EXPOSURE MODEL

In this study, we obtained measurements of VOCs and PAHs for both personal exposures and in various micro-environments. We also gathered personal lifestyle information. This information has been incorporated into several models to predict the personal exposure of a non-occupationally exposed population to a selected group of VOCs and PAHs.

There are several considerations regarding the quantity or quality of the information provided by the subjects whose data have been used to develop the model:

- The factors covered by the storage questionnaire for subjects 1 through 27 were estimated from information available from photographs, other questionnaires, and researchers' knowledge of the subjects, because when those subjects were sampled, the storage questionnaire had not been prepared.
- No specific information on ETS as regards the number of cigarettes smoked or the relationship of the subject to the smoker is available for subjects 1 through 50, because for those subjects a separate ETS questionnaire was not used. This information therefore could not be entered into the model.
- The volunteer's perception of ETS is subjective. For example, some volunteers, in the company of a relative

or friend who might have smoked just one cigarette, describe the environment as being “slightly smoky—people are smoking occasionally” on the ETS questionnaire or location description sheet. This close ETS event may have created a greater ETS concentration in the area of the subject than if the smoker were not a person in their company.

- Some subjects were active smokers but did not declare this at the outset. Data from these subjects were considered outliers during model development and validation and have been removed.
- Information on some activities or characteristics that may have affected the measured concentrations was not collected. This information includes time spent photocopying, type of paint used, and the presence of new furniture in the home or office.
- Some subjects gave inadequate information about where they were or what they were doing, making it difficult to interpret some of the high concentrations recorded.
- Some of the high concentrations whose causes have been identified were difficult to enter into the model, because there were many variables that could have had an effect and that could not be controlled (e.g., location of the briefcase with reference to the pollutant source, ventilation in the building, activities performed by other dwellers or co-workers not recorded on the forms).
- Sampling periods that were < 1100 minutes and therefore did not include the nighttime were not entered into the model, as the resulting concentrations were not comparable with concentrations of samples collected over 1400 minutes (covering the daytime and nighttime).

The information extracted from the performance of model 1 (Table 22) gives valuable information in terms of evaluating the influence that the two microenvironments where people spent most of their time have on personal exposure. The model that correlates personal exposure with home microenvironment explains most of the variance in personal exposure, generating, in some cases, R^2 values of 0.7, such as for benzene, toluene, and the trimethylbenzenes. Other compounds for which home microenvironments are a good predictor of personal exposures are *p*-isopropyltoluene, naphthalene, and the xylenes, with R^2 around 0.5, and ethylbenzene (with $R^2 = 0.4$). These observations are confirmed by the scatterplots of the personal exposures predicted by the model versus the personal exposures measured (presented in Appendix 20). In several contexts—when subjects spent a large amount of time at home, there is little ventilation at home, there are additional

indoor sources (e.g., heating systems or cleaning products in use), or there are strong indoor sources (e.g., ETS at home or recent redecoration)—the home contribution is expected to impact greatly on the personal exposure, and hence home microenvironmental concentrations will be a good predictor of personal exposures.

On the other hand, the model correlating personal exposure with the workplace explains less of the variance in personal exposure, presenting correlation coefficients in the range of 0.15–0.28 for most of the compounds. From the performance of both models, it is clear that the home microenvironment affects personal exposure the most, with subjects spending an average of 62% of their time at home. Personal exposure to all compounds was therefore dominated by the contribution from the home (Kim et al. 2002; Adgate et al. 2004a).

The time-weighted model, which used specific subject-related information when available and pooled data otherwise (model 2), explained more variability in personal exposure than did the model using the home measurements alone, except for benzene and toluene. Similar results were reported for benzene by Adgate and colleagues (2004a) and for other compounds by Kim and associates (2002). Nevertheless, direct measurements in the subjects' own locations are required for this model.

On the other hand, model 3 uses generic stratified microenvironmental concentrations for all the microenvironments that the subjects visit, in conjunction with the information extracted from the time–activity diaries. Therefore, no direct measurements are required to predict personal exposure. The performance of this model is lesser than for the other tested models. This is a consequence of the difficulties in adequately stratifying home microenvironments, given the modest number of samples collected as well as the home-to-home variation in concentrations. Even if the number of samples is large ($N = 160$), the large number of strata (e.g., integral garage, ETS exposure, first-line property, or type of location) reduces considerably the sample size per stratum. The range of activities in which the subjects are engaged in their normal life was reflected in the specific home and workplace levels and therefore was accounted for well in models 1 and 2. This was accounted for less well in model 3, however, because the generic stratified microenvironmental concentrations used in the model do not contain specific information about each subject's microenvironment. Nevertheless, the scatterplots of the measured personal exposures versus the predicted exposures (Appendix 20) show that the model predicts the concentrations well, except in some cases where concentrations are typically under-predicted. Further study shows that these cases are linked to activities such as exposure to ETS, home repair and improvement, photocopying, and

use of solvents. Therefore, model 3 predicts the concentration well in most instances but does not perform well when the subjects engage in an activity that presumably results in a substantial increase in VOC exposure.

To address this difficulty, model 4 uses the concentrations as calculated in model 3 but includes a range of add-on variables that represent activities and home characteristics that could not be reflected in the stratified data and that lead to an increase in VOC levels. This approach better reflects the VOC concentrations, explaining higher levels of variance: 80% for ethylbenzene, xylenes, styrene, and trimethylbenzenes and around 50% for compounds such as benzene, toluene, and 1,3-butadiene. The amount of variance left unexplained by model 4 (e.g., 51% for 1,3-butadiene) must be due to the fact that the sources of such compounds were not well captured in the proposed microenvironmental concentrations or add-on variables.

Table 22 shows that the model that best predicts the personal exposure is model 4. The same comparison of predicted versus measured concentrations was performed using the \log_{10} -transformed database, showing in all cases correlation coefficients lower than for the nontransformed data (i.e., R^2 ranging from 0.25 for *p*-isopropyltoluene to 0.46 for *p*-xylene and *o*-xylene).

However, some compounds, such as benzene and toluene, are best predicted by model 1, in which personal exposures are predicted directly from home exposures. It appears that, for these two compounds, representation of other microenvironments visited (as in model 2 and model 3) or other activities performed during the day (as in model 4) does not improve the prediction of personal exposures; rather, it increases the uncertainty not accounted for by the model (e.g., for benzene, model 1 [home] explains 67% of the variability but models 2 and 4 explain only 44% and 47%, respectively). As observed, most of the variation for these two components arises from the home concentrations. On the other hand, the other microenvironments or activities that contribute to the personal exposure could not be captured by the other proposed models (models 2 through 5). This suggests that more detailed information is needed to fully understand the non-home sources contributing to benzene and toluene concentrations and that the estimates of home microenvironmental concentrations used in models 3 and 4 do not fully reflect the behavior of these two compounds, thus requiring further study.

Regarding the performance of model 1 for the PAH data set, home microenvironmental levels could explain 36% and 43% of the variance for chrysene and dibenz[*a,h*]-anthracene, respectively. Workplace concentrations could explain 50% and 38% of the variance for benzo[*b*]fluoranthene and benzo[*g,h,i*]perylene, respectively. Contrary to

the observations for the VOC data set, PAH concentrations at home do not always dominate personal exposures and hence, they are not good predictors of personal exposure, suggesting that activities or sources outside the home contribute largely to PAH personal exposures.

On the other hand, contrary to previously reported results (Ohura et al. 2005), neither of the two proposed time-weighted models (model 2 and model 3), which consider only time spent in various microenvironments, could explain the variance for the PAH compounds. Model 4, however, was able to explain 25–66% of the variance for the PAH compounds (e.g., 35% of variance for benzo[*a*]pyrene). This improvement was a consequence of including add-on variables accounting for various activities in the proposed time-weighted model (see Appendix 21 for detailed add-on variables and model coefficients). Therefore, the evolution of model development for the PAH database suggests that the home microenvironment alone is not a good predictor for personal exposures, and therefore model 1 does not perform well. It also suggests that including information about other microenvironments visited during the day does not improve the prediction of personal exposures (model 2 and 3) and also highlights the strong influence of the add-on variables, which reflect activities, to predict the PAHs personal concentrations, as shown by the improvement of the R^2 values seen for model 4. Hence, the sources that mainly affect PAHs are not generally found at home or in any other particular microenvironment but are mostly a consequence of various activities that the subjects perform.

Although model 4 is the best-performing model as a consequence of the introduction of the add-on variables that account for various activities, such as exposure to ETS or commuting, it is not able to predict the personal exposures to PAH as well as the personal exposures to VOCs. This might suggest that the PAH database was not big enough to allow the model to be trained with a significant number of cases across the exposure range; either the PAH exposures in the database were very similar and therefore there was low variability in important predictors in the sample (e.g., commuting and ETS exposure), there was variability introduced by activities not recorded in the time–activity diaries, or the information captured in the time–activity diaries needed to be more exhaustive to enter more detailed information into the model.

A correlation analysis of the variables entered into model 4 for each VOC and PAH was performed to assess any collinearity of the variables selected (Appendix 22). Several variables were identified as correlating with one another with a Pearson $R > 0.8$. This was the case with “Time spent while others were painting” (Time_Indirect_Paint) and

“Time spent in contact with paint” (Time_Paint), which are included in the *o*-xylene and the *m*-xylene models. A similar situation occurs between the variables “Time exposed to Frequent ETS” and “Pyridine Modeled” in model 3, which affects the pyridine model; and also between the variables “Time spent in Frequent and Constant ETS” and “Time spent in contact with ETS,” which were entered as variables in the models of 3-ethenylpyridine and 1,3-butadiene. These findings imply that there is a degree of overlap within each of these pairs of variables. Nevertheless, one of the identified variables was dropped from the model if any of the Pearson *R* values exceeded 0.9 and the variance inflation factor for these variables in their respective models was greater than 10.

Some of the models presented in this study were driven by skewed distributions. A sensitivity analysis was performed, which consisted of performing the same comparisons of predicted versus measured data from the log₁₀-transformed database (Table 23 and Table 25 for VOCs and PAHs, respectively), showing in all cases *R*² values lower than were found for the nontransformed data (e.g., for model 4, 25% for *p*-isopropyltoluene to 46% for *p*-xylene and *o*-xylene in the VOC data set and 42% for chrysene to 13% for benzo[*k*]fluoranthene in the PAH data set). However, the sensitivity analysis also confirmed that model 4 is able to predict significant personal exposures for all the studied compounds except benzo[*g,h,i*]perylene. These conclusions are in contrast with the similar results found for the log₁₀-transformed and non-transformed data for the two best performing models: model 4, which predicts personal exposures using a stratified, independent concentrations database and a lifestyle questionnaire, and model 2, which depends on direct measurements from the subject's home and workplace. In the case of benzene, ethylbenzene, and the xylenes, model 4 performs similarly to model 2; for ETS compounds, trimethylbenzenes, and toluene, model 4 performs slightly worse than model 2; and for all the PAHs, model 4 performs considerably better.

The influences of several activities on the PAH and VOC levels can be assessed from the information listed in Table 26 (see Table A21.4 and Table A21.5 for the complete list of VOCs and PAHs). ETS exposure is important for 3-ethenylpyridine, pyridine, and 1,3-butadiene, as anticipated. Traffic is a good predictor for compounds such as benzene, toluene, 1,3-butadiene, and benzo[*a*]anthracene, as for all these compounds, model 4 contains traffic-related variables. However, toluene was the only compound for which levels were higher for personal exposures and home micro-environments at trafficked roadsides than those away from trafficked roadsides. The use of paints is an activity impacting the levels of ethylbenzene, the xylenes, and the trimethylbenzenes. Similarly, storing paints in an integral

garage increases the levels of *n*-hexane, benzene, toluene and ethylbenzene. In line with activities related to the integral garage, parking the car in the garage raises the levels of benzene, toluene, ethylbenzene, the xylenes, and the trimethylbenzenes. The use of fuels other than natural gas for heating increases the concentrations of *n*-hexane, benzene, ethylbenzene and *p*-xylene.

For the PAH database, the seasonal effect was considered in the model, as all PAHs except coronene were found at lower concentrations in summer than in winter. ETS was identified as an important contributor, increasing the levels of almost all the compounds, especially for subjects in close proximity to a smoker. Other situations identified as important contributors to PAH levels are the use of a gas cooker and the absence of a stovetop hood.

Detailed information about which activities and situations affect each compound are summarized in Table 26 (with complete information given in Table A21.4 for VOCs and in Table A21.5 for PAHs) is given below.

The *n*-hexane multivariate model explains the personal exposure levels by including variables such as storage of paints in the garage, ETS-related activities, use of additional heating other than by gas or electricity, occupationally related variables such as working in a laboratory or factory, and other activities such as visiting a hospital. This model accounts for 35% of the variance in personal exposure. Storage of paints in the garage was identified as the strongest predictor (standardized coefficient beta = 0.52), working in a laboratory was the second-strongest predictor (beta = 0.24), and other predictors had a similar influence on *n*-hexane concentrations (0.1 < beta < 0.18). The variables chosen are supported by the literature. *n*-Hexane is found in a wide range of household products such as adhesive-related products, oils, greases and lubricants, automobile products, paint-related products, and household cleaners and polishes (Edwards et al. 2005), as well as in carpet glues, wallpaper, chipboard, insulation foam, and newly painted surfaces (Zuraimi et al. 2006).

The benzene model explains 47% of the total variance in personal exposure. This model takes into account the existence of an integral garage where paints and the car are kept; traffic variables such as living in an urban area, use of trains, and time spent commuting by car; ETS exposure; variables related to heating; and other variables linked to activities such as working in a hospital. Storage of paints in the garage was again identified as the strongest predictor (beta = 0.41), followed by time exposed to constant and frequent ETS (beta = 0.28), and storage of car in the garage (beta = 0.22). Predictors related to traffic had standardized coefficients 0.15–0.08 and those related to heating had beta values around 0.17–0.15. Higher benzene levels have been related to the presence of integral garages (Fruin et al.

2001; Marshall et al. 2003; Batterman et al. 2006b; Jia et al. 2008), ETS (Heavner et al. 1995), and traffic (Edwards et al. 2005; Pérez Ballesta et al. 2006). A previous study that attempted to model benzene levels explained only 28% of the variance (Heavner et al. 1995). Apparently the total sum of all other unidentified benzene sources in the model of Heavner and coworkers (1995) exerted an overall greater effect on benzene concentrations than the variables applied in that model, which is consistent with our findings. Pérez Ballesta and colleagues (2008) modeled benzene from information in time–microenvironment–activity diaries, reporting an R^2 value of 0.47, which is similar to the value reported here, although those researchers did not verify their model results with an independent data set.

The proposed multivariate model for toluene has the highest number of input variables. A total of 51% of the variance in toluene levels is explained by variables such as home characteristics (e.g., open-plan kitchen, location in London), activities performed in the house (e.g., incense burning, drawing or painting, having a manicure, wrapping presents, spraying artificial snow) or outside (e.g., working in a hospital or factory), and finally by the storage of paints and a car in the integral garage. The main predictor of toluene was the duration of exposure to solvents (e.g., glues, manicure products, garden products), with a standardized coefficient of 0.48. Working in a hospital ($\beta = 0.25$) and wrapping presents and using artificial snow ($\beta = 0.23$) were identified as the second and third main predictors. Predictors with less influence on toluene concentrations were the use of additional gas heating ($\beta = 0.06$), working in a factory ($\beta = 0.06$), and burning incense ($\beta = 0.03$). Toluene has been associated with emissions from carpet or vinyl flooring (Zuraimi et al. 2006) and with wood boards and wood-based furniture (Saarela 1999), which might explain the fact that an open-plan kitchen was associated with higher concentrations of toluene than traditional kitchens. Toluene is also emitted from transport sources (Hinwood et al. 2006; Na and Kim 2007; Song et al. 2007), evaporation of gasoline (Song et al. 2007), paints (Na et al. 2004; Song et al. 2007), newspapers and adhesive sprays (Saarela 1999), integral garages (Jia et al. 2008), and solvents (Choi and Ehrman 2004; Brown et al. 2007; Na and Kim 2007; Song et al. 2007). Other authors who attempted to model toluene on the basis of information collected in time–activity diaries reported R^2 values lower than those in our study (Pérez Ballesta et al. 2008).

Ethylbenzene and the xylenes are estimated with four models that take into account the same variables: carpet fumigation, activities performed indoors such as painting or outside the house such as visiting a hospital or working in a factory, and parking the car in the integral garage. The

proposed multivariate model explains an average of 80% of the total variance of each compound. The *p*-xylene and *o*-xylene models also include as an explanatory variable the storage of a gasoline lawnmower in the garage and the time spent in place while others were painting, respectively. In the case of ethylbenzene, two unique variables are storage of paints in the garage and having a new carpet. Time spent in contact with fresh paint has been identified as the main predictor for ethylbenzene and the xylenes, with standardized coefficients (β) ranging from 0.68 for ethylbenzene to 1.30 for *o*-xylene. In the case of ethylbenzene, another important predictor is having the carpet fumigated ($\beta = 0.55$); the remaining predictors have standardized coefficients ranging from 0.07 to 1.52. For *m*-xylene and *o*-xylene, the second-strongest predictor is time spent in a place while others were painting ($\beta = -0.53$ and -0.60 , respectively). These negative coefficients appear to be an artifact of the model. Storing the car in the garage ($\beta = 0.22$) was also identified as a main predictor for *m*-xylene. The remaining predictors for all the xylenes had similar standardized coefficient values (0.07–0.18). Evaporation of solvents represents another important source for C₆-aromatic hydrocarbons and may explain why these compounds are found in particularly high concentrations in the indoor air of homes (Ilgen et al. 2001c). Ethylbenzene and xylenes are reported to be emitted from traffic (Mukund et al. 1996), insecticides (Yang et al. 2002; Scorecard 2006), and paints, industrial solvents, natural gas, and ETS (Song et al. 2007). *o*-Xylene is also emitted from dry-process photocopiers (Zuraimi et al. 2006), and *m*-xylene from printers (Watson et al. 2001). Pérez Ballesta and associates (2008) modeled ethylbenzene and *m,p*-xylene exposures using variables derived from time–microenvironment–activity diaries and reported R^2 values of 0.27 and 0.56, respectively, which are lower than those reported in the present study.

Both trimethylbenzenes were correlated in multivariate models with the same explanatory variables. The model developed explains 79% of the variance for 1,3,5-trimethylbenzene and 87% for 1,2,4-trimethylbenzene. The explanatory variables were parking the car in the integral garage, time spent painting, and house characteristics such as location of the door connecting the garage to the kitchen, use of a gas cooker, and working in a factory. The main predictor was time spent painting, with standardized coefficients of 0.78 for 1,3,5-trimethylbenzene and 0.80 for 1,2,4-trimethylbenzene. The remaining predictors had, in both cases, coefficients between 0.03 and 0.16. Trimethylbenzene levels have previously been significantly correlated with increasing duration of painting (Lai et al. 2004). Other sources include traffic, use of solvents (Watson et al. 2001), ETS (Hinwood et al. 2006), heating systems (Kim

et al. 2002), integral garages (Jia et al. 2008) and printers (Watson et al. 2001).

The model adopted to predict styrene concentrations explained 87% of the variance with just 3 variables, which were the personal exposure concentration calculated in model 3 ($\beta = 0.06$), burning wood ($\beta = 0.10$) and having the carpet fumigated ($\beta = 0.93$) which was the strongest predictor. These results are consistent with the literature, where styrene was associated with carpet or vinyl flooring emissions (Edwards et al. 2001a; Zuraimi et al. 2006), traffic (Hinwood et al. 2006), industrial solvents (Brown et al. 2007), wood burning (Austin et al. 2001) and ETS (Hinwood et al. 2006).

A total of 48% of the variance in *p*-isopropyltoluene personal exposure levels is explained by the model we developed. Ten variables accounted for the variability: incense burning, redecoration of the home, time spent painting, use of chiropody solvents, drawing, painting, time spent in the gym, use of additional gas heating, cooking without a stovetop hood, and removal of mold from within the house. The two main predictors were time spent painting ($\beta = 0.45$) and use of chiropody solvents ($\beta = 0.35$). The third main predictor was the inverse of the time since removal of mold from within the house ($\beta = 0.26$). The remaining predictors had standardized coefficients between 0.05 and 0.16. Sources of *p*-isopropyltoluene include paints, wood office furniture, and consumer products (Scorecard 2006).

Both pyridine and 3-ethenylpyridine, the vapor-phase ETS marker, had a significant correlation with all five smoking-activity variables. The variability explained by the model for pyridine and 3-ethenylpyridine is 70% and 75%, respectively. The main predictors in both cases was time spent in constant and frequent ETS microenvironments ($\beta = 0.65$ for 3-ethenylpyridine and $\beta = 0.54$ for pyridine). Time spent in frequent ETS microenvironments was also a strong predictor for pyridine ($\beta = 0.54$). The other predictors had β values of -0.32 to 0.22 for 3-ethenylpyridine and -0.29 to 0.21 for pyridine, with the first variable being identified as the compound modeled in model 3 and the second the variable representing the total time spent in ETS environments. This result is partially supported by the work of Heavner and coworkers (1995), who found a significant correlation for 3-ethenylpyridine only.

The multiple regression analysis for naphthalene was able to account for 42% of the variance in personal exposures, consistent with other studies (Edwards et al. 2005). The strongest predictor was the use of naphthalene as a moth repellent ($\beta = 0.60$). The second-highest standardized coefficient was related to burning incense ($\beta = 0.17$).

Other coefficients were related to the use of gas heating ($\beta = 0.11$) (Edwards et al. 2005), the inverse of the time since new carpet was installed ($\beta = 0.06$), and the presence of photocopiers and printers within the home ($\beta = 0.10$), which could be due to the ink used. Naphthalene appears to be present in solvents used in domestic paints and dyes. Crystalline naphthalene is used as a moth repellent and as a solid block deodorizer for toilets (Edwards et al. 2005). Naphthalene has been also reported to be emitted from dry-process photocopiers (Zuraimi et al. 2006).

The model proposed to estimate 1,3-butadiene levels in personal exposures explains 49% of the total variance, using 10 variables. These variables are use of solvents, ETS-related variables, visiting a gas station, time since carpet was installed in the living room, time spent commuting, location of the door connecting the integral garage to the kitchen, use of additional heating other than by natural gas or electricity, time spent using artificial snow, time spent wrapping presents, and time since mold was removed from the house. The main predictor was time spent wrapping presents and spraying artificial snow ($\beta = 0.43$). Other important predictors were time spent in constant and frequent ETS environments, using gas for additional heating, and time since mold was removed from the house ($\beta = 0.24$ for all three). The remaining predictors had β values ranging from 0.03 to 1.5. Area sources of 1,3-butadiene include prescribed burning, residential and commercial space heating, fuel and gasoline distribution, and the burning of other materials (e.g., cigarettes). Mobile sources include on- and off-road motor vehicles, aircraft, rail vehicles, and marine vessels (Curren et al. 2006). 1,3-Butadiene has previously been linked to ETS exposure (Kim et al. 2001b; Kim et al. 2002; Hinwood et al. 2006).

The models proposed to estimate PAH concentrations involved add-on variables related to ETS (e.g., number of cigarettes smoked within 2 meters of the subject), combustion (e.g., incense burning) and traffic (e.g., use of bus). The same sources have been largely attributed to PAHs in the literature (Chuang et al. 1991; Harrison et al. 1996; Dubowsky et al. 1999; Lim et al. 1999; Levy et al. 2001; Wu et al. 2005). Georgiadis and colleagues (2001) designed a multiple linear regression model in which total PAH levels in winter were correlated at the $P < 0.05$ level only with the declared time of exposure to ETS.

Model 5, which attempts to predict personal exposures on the basis of selected key determinants such as living in a first-line property, living in a house with integral garage, being exposed to ETS, and living in an urban, suburban, or rural location was the model that explained the least variance. Model 5 was a good predictor of compounds that are ETS related, such as 3-ethenylpyridine and pyridine (for

which 27% and 21% of the variance is explained). Among the PAHs, model 5 explained only 9% of the variation for dibenz[*a,h*]anthracene, which is associated with living in rural places.

VALIDATION OF THE PERSONAL EXPOSURE MODEL

Direct measurement of human exposure to VOCs via personal monitoring is the most accurate method of exposure assessment currently available. However, wide-scale application of this method to evaluate exposures at the population level is prohibitive both in terms of cost and time. Consequently, indirect measurements via a combination of microenvironmental monitoring and use of personal-activity diaries represent a potentially useful alternative. If indirect exposure estimates are to be routinely used, then it is important that they are evaluated by means of comparison with direct measurements.

Such a comparison was conducted in the present study, with the conclusion that, with some exceptions (naphthalene and 1,3-butadiene), there is good agreement between the indirect and direct methods for the VOCs. This conclusion is supported by four main findings: (1) the correlation coefficients for the plots of the direct and indirect exposure estimates tested in a separate and independent validation data set, which range from 0.4 for benzene to 0.9 for styrene; (2) the absolute value of the normalized mean bias is less than 25% for most compounds; (3) the mean fractional bias is below 25% for most compounds; and (4) 60% of the concentrations are predicted within a factor of two, and this percentage increases to approximately 80% for prediction within a factor of three, for most of the VOCs (Table 27).

In the case of the PAHs, the models developed, although explaining some of the variance in the independent data set and being good indicators of the sources affecting PAH concentrations, could not be validated statistically, probably because the PAH validation data set is too small. The correlation coefficient was significant only for pyrene, the normalized bias mean was above 50% for most PAHs, the mean fractional bias was greater than 100% for most, and around 20% of PAHs were predicted within a factor of two and 35% were predicted within a factor of three (Table 28).

The correlation coefficients (R^2) were similar in both the training and validation data set for compounds such as *n*-hexane, benzene, toluene, pyridine, and the trimethylbenzenes, and the coefficient for styrene was better in the validation set than in the training set. Nevertheless, other compounds performed worse in the validation data set, such as ethylbenzene, the xylenes, *p*-isopropyltoluene, and 3-ethenylpyridine. For naphthalene and 1,3-butadiene, no correlation was found between the predicted exposures

and the measured exposures in the validation data set. In the case of the PAH models, the correlation coefficients are generally lower in the validation data set than in the training data set, except for pyrene, for which it is considerably higher in the validation data set.

The normalized mean bias in the VOC data set shows that most of the compounds (except *o*-xylene, styrene, and naphthalene) are under-predicted by the model by < 25%. This observation is confirmed by the negative mean fractional bias for compounds such as benzene and the trimethylbenzenes. As regards the PAH data set, some compounds (e.g., pyrene) are over-estimated by the proposed model, whereas others (e.g., benzo[*a*]pyrene) are under-predicted. Although the normalized bias of the model is generally above 50%, compounds such as benzo[*a*]pyrene, dibenz[*a,h*]anthracene, and coronene are predicted with a normalized bias of < 30%. The mean fractional bias for the PAH validation data set is generally above 100% for most of the compounds.

The percentage of predicted values within a factor of two is around 60% in the VOC validation data set. This percentage increases to 80% if a factor of three is considered. Nevertheless, this is not the case for 1,3-butadiene, for which the factor-of-two value is 31% and the factor-of-three value is 49%. For the PAH data set, 20% of the values are predicted within a factor of two for most of the compounds, and this percentage increases to around 40% when considering a factor of three. The same trend of high percentages of predicted values within a factor of two for VOCs and low percentages for PAHs is visible in Figure 10 and Figure 11, respectively.

CATEGORIZATION OF LOW AND HIGH PERSONAL EXPOSURES

As regards the performance of the model in the categorization of personal exposures according to the proposed threshold values in the validation data set, the model is successful in correctly classifying cases of low exposure, with 95% to 100% of cases classified correctly (Table 29) for VOCs and 83% to 100% for PAHs (Table 30). Nevertheless, the success rate decreases for high exposures, with 11% to 60% of cases classified correctly (for benzene and pyridine, respectively) in the VOC data set and 33% to 67% (for dibenz[*a,h*]anthracene and pyrene, respectively) in the PAH data set. This result is not unexpected, as all the models show a tendency to underestimate personal exposure levels. For three VOCs (i.e., *p*-isopropyltoluene, naphthalene, and 1,3-butadiene) and most PAHs, none of the high personal exposures were classified correctly. Again, this is not surprising, as these data have higher slopes and intercepts and these are the compounds for which the model could not be validated.

The models developed with the training data set for VOC compounds have been validated using the independent data. The models developed for the PAHs could not be validated statistically for most of the compounds. Nevertheless, the models proposed explain some of the variance in the independent data set and are good indicators of the sources affecting PAH concentrations. Differences between the absolute values of directly and indirectly obtained estimates of exposure were not unexpected and were consistent with previous reports (Heavner et al. 1995; Leung and Harrison 1998; Edwards et al. 2001b; Kim et al. 2002). In essence, the differences we observed are a consequence of the dynamic nature of the source–receptor relationship, which implies that the sampling of air at stationary monitoring locations may not accurately reflect the contaminant composition of the air inhaled by individuals, except if the level of a contaminant is spatially uniform or the individuals are relatively stationary (Kim et al. 2002).

PERCENT CONTRIBUTIONS OF VARIOUS MICROENVIRONMENTS TO OVERALL PERSONAL EXPOSURES TO VOCs

The assessment of percent contributions of each microenvironment to personal exposure to VOCs (Figure 12) gives us very useful information for assessing the most influential microenvironments in personal exposures. The average contribution of the home microenvironment to personal exposures varies from 80% for *p*-isopropyltoluene to 50% for *m*-xylene or 1,3-butadiene. In the case of ETS-related compounds, the home contribution is lower (e.g., 35% for 3-ethenylpyridine), as other microenvironments such as pubs are important contributors (e.g., 38% for 3-ethenylpyridine). In the general population, the contribution of the workplace is less important than that of the home for all the VOC compounds; the percent contribution of the workplace averages 15% for ethylbenzene, the xylenes, and the trimethylbenzenes and around 8% for the others. The contribution of commuting is the third in importance, although the contribution is small (5–9%) compared with the home contribution. The percentage of personal exposures not attributable to the home, workplace, or commuting is around 18%, which can be accounted for by pubs, restaurants, other indoor environments, and the outdoors.

Thus, for the general population, the home is on average the dominant microenvironment affecting personal exposure levels (average contribution, 64%), followed by the workplace (13%) and commuting by car (6%). However, caution should be exercised in interpreting these data, as there is error associated with the calculated percentages, indicated by the amount of unexplained variance in model 2. The contributions of the three main microenvironments

to personal exposures are highly correlated with the fraction of time that subjects spent in them (e.g., around 60% at home, 18% at work, and 6% commuting; Table 8). Although in-vehicle concentrations are normally high compared with other microenvironmental concentrations (Pérez Ballesta et al. 2006; HEI Air Toxics Review Panel 2007), in this study, in-vehicle concentrations were generally similar to the concentrations found at home. Because time spent in-vehicle is tenfold lower than the time spent at home, the contribution of commuting to personal exposures is proportionally reduced. This finding of a large contribution of the home microenvironment to personal exposures is consistent, however, with the results obtained in model 1, where personal exposures were well predicted from home concentrations, as well as being consistent with previous studies that reported daily exposures to VOCs that were almost completely determined by indoor exposure at home and in the office, with a minor contribution during transport (Leung and Harrison 1998; Carrer et al. 2000; Ilgen et al. 2001c; Kim et al. 2002). In the case of ETS-related compounds such as 3-ethenylpyridine, pyridine, and 1,3-butadiene, the contribution of the home microenvironment to the exposure of the entire population decreases to 43%, and pubs become one of the largest contributors (30%). (Sampling occurred before July 1, 2007, when the smoking ban came into effect.) However, because the variability explained by model 2 for these three compounds is very low, the findings should be interpreted with caution.

For the subjects that were exposed to ETS, the contribution of ETS-polluted environments, such as pubs and the homes of friends or relatives who smoke, gain importance. Figure 13 shows that, in this subgroup, the relative contribution of the home microenvironment for the VOC compounds (60%) is lower than for the general population, ranging from 70% for *p*-isopropyltoluene, *n*-hexane, and 1,3,5-trimethylbenzene to 50% for *m*-xylene. In turn, the contribution of the pub microenvironment is increased for nearly all the compounds, especially the ETS-associated substances, for which the contribution is approximately 42%. Another microenvironment that gains importance for the ETS-exposed population is friends' or relatives' houses (5%), where ETS generation is more frequent than in the overall population. A study performed in southern California between 1989 and 1997 reported that passive smoking accounted for one fourth of all the exposure to benzene among adult non-smokers, whereas in-transit microenvironments accounted for 15% (Fruin et al. 2001). This result for the ETS contribution is consistent with our result of 22% for benzene, which is the value resulting from the addition of places identified as the main ETS-influenced microenvironments as reported by ETS subjects (i.e., pubs, restaurants, and relatives' or friends' homes). The value of

in-transit contribution in our study is half that reported by Fruin and associates (2001), which may reflect a reduction in traffic emissions or the different location of sampling.

Figure 12 attributes a significant proportion of exposure to certain VOCs to the laboratory environment. Our study subjects included a number of laboratory workers. Consequently, this result is unlikely to extrapolate well to the wider community. Similarly, as most of our subjects were office workers (purposefully recruited as such), their data may also not be representative of the general population.

SUMMARY

BEHAVIORAL INFORMATION

- Subjects participating in this study spent 91% of time indoors, 4% outdoors and 6% in transit.
- Most of the time spent indoors was at home (62%).
- Activities such as candle or incense burning, fireplace use, and home repair and improvement activities performed by the subjects influence personal exposures, as demonstrated by the developed model.
- Patterns of exposure to ETS were identified. A total of 67% of subjects exposed to ETS were exposed indoors in places such as pubs (before the U.K. smoking ban), the home, and a friend or relative's homes (in decreasing order of importance).

PERSONAL EXPOSURES

- Personal exposure levels in this study are generally lower than those reported in similar studies conducted previously in the United States and Europe.
- The personal exposure concentrations presented in this study do not generally show significant differences according to geographic location or type of location (urban, suburban, or rural) for either VOCs or PAHs.
- Personal exposure levels were compared with levels measured in microenvironments, showing that personal exposures exceed in-home and in-vehicle concentrations, with outdoor concentrations being the lowest. An exception to this trend was ETS-related compounds, which were at highest concentrations in pubs.
- Subjects living in houses located on trafficked roadsides do not have significantly different personal exposures than those living away from traffic, except for toluene and the high-molecular-weight PAH compounds.
- Subjects living in houses with integral garages had higher personal exposures to almost all VOCs but not PAHs.

- Subjects living in houses with ETS had higher concentrations for almost all the VOCs and PAHs.
- The highest PAH levels were measured for subjects who used a fireplace at home.
- The senior subpopulation (≥ 66 years old) had a significant pattern of reduced exposure to PAHs.

MICROENVIRONMENTAL CONCENTRATIONS

Subjects' Homes

- Levels measured at home in this study were generally lower than those previously reported in Europe and the United States.
- The home microenvironmental concentrations do not generally show significant differences according to geographic location or type of location (urban, suburban, or rural) for VOCs.
- However, the concentrations of high-molecular-weight PAHs were highest in rural homes, followed by suburban and urban homes, probably because of a seasonal effect as well as fireplace and fuel usage.
- Similarly, homes located in Wales (rural homes) had the highest PAH concentrations, followed by homes in West Midlands (rural, suburban, and urban) and homes in London (urban). This trend might also be affected by season and use of fireplaces for space heating.
- Homes located on trafficked roadsides and homes located away from traffic had similar concentrations for almost all VOCs and for the high-molecular-weight PAHs, except toluene, which was significantly higher in first-line homes.
- Concentrations measured in houses with integral garages showed higher concentrations for almost all the VOCs but not the PAHs.
- Homes where ETS was present had higher concentrations of almost all VOCs and PAHs than did homes without ETS.
- No significant difference between daytime and nighttime concentrations was found.

Homes Other Than the Subjects'

- Within the home, VOC concentrations were highest in the garage and PAH concentrations were highest in the living room.
- Samples collected in winter had higher concentrations of both VOC and PAH compounds than those collected in summer.
- Samples collected concurrently indoors and outdoors (i.e., in the living room and backyard) in homes

located away from traffic showed higher levels indoors, and samples collected concurrently in the living room and kitchen showed lower concentrations in the kitchen.

- No significant difference between daytime and nighttime concentrations was found for either VOCs or PAHs.

Workplaces

- The effect of traffic was assessed by comparison of offices located close to and those located far from trafficked roadsides. First-line offices showed higher concentrations for some VOCs (not significantly higher) and for some high-molecular-weight PAHs ($P < 0.05$).
- The influence of office location within the city was also assessed, with urban and suburban offices having similar concentrations, for both VOCs and PAHs.

Streets

- VOC and PAH levels measured in this study are generally lower than those reported by others elsewhere.
- The various street microenvironmental samples showed higher concentrations in association with higher traffic loads (trafficked roadsides > background streets > pedestrian streets > parks).
- Similarly, samples collected during rush hour showed higher VOC and PAH concentrations than those sampled during the afternoon.
- Samples collected during winter had higher concentrations than those collected in summer.

Transport Microenvironments

- Among all the mobile-transport microenvironmental samples, those collected in London buses and the London Underground (subway) showed the highest VOC and PAH concentrations, respectively, with London trains having lower concentrations.
- High VOC concentrations were measured in car parks and high PAH concentrations were generally found in main train stations.
- Samples collected in mobile-transport microenvironments during rush hour had higher VOC and PAH concentrations than those collected during the afternoon.
- In contrast, samples collected in transport stations had similar concentrations throughout the day.
- Samples collected during winter had higher concentrations than those collected in summer.

Indoor Areas

- Various indoor microenvironments were sampled, with the highest VOC and PAH levels recorded in pubs where ETS was present.

- VOC and PAH levels sampled in other indoor environments (e.g., libraries and museums) were generally lower than those in pubs and restaurants.
- The influence of ETS was further assessed in pubs and restaurants, showing higher VOC and PAH concentrations in pubs and restaurants where ETS was present than in those in which it was absent.
- Samples collected during winter had higher concentrations than those collected in summer.

URINARY BIOMARKERS

- ETS urinary biomarkers were significantly correlated with ETS-related VOCs such as 3-ethenylpyridine and 1,3-butadiene.
- ETS urinary biomarkers were significantly correlated with high-molecular-weight PAH compounds.
- PAH urinary biomarkers did not show significant correlations with the respective PAH parent compounds, possibly owing to a sampling artifact for the low-molecular-weight PAHs or the ETS-related VOC compounds.
- Urinary 3-naphthol did not show a significant correlation with naphthalene in the gas phase, which may be a consequence of the different pathways of naphthalene intake.

CORRELATIONS

- Strong correlations were observed between ethylbenzene and the xylenes, the two trimethylbenzenes, the two ETS markers, and all the high-molecular-weight PAHs in the personal exposure, home, and workplace databases.
- An increased number of VOC compounds correlated with one another for the street and transport microenvironments, suggesting a common source, which was identified to be traffic.
- This was not the case for PAH compounds, which may be a consequence of the influence of regional atmospheric transport and dilution. However, the combined VOC and PAH data collected in street and transport microenvironments showed stronger correlations than for personal exposures or home and workplace microenvironments.
- Correlations found in ETS-influenced environments clearly showed the influence of tobacco smoke on VOC and PAH levels.

SOURCE APPORTIONMENT BY FACTOR ANALYSIS

- Factor analysis performed on PAH personal exposure data showed that two factors explained most of the

personal variance, with the first factor being associated with gasoline emission and combustion sources (explaining 54% of the variance) and the second with cooking or diesel vehicle emissions (explaining 13% of the variance).

- Factor analysis performed on VOC personal exposure data also revealed two factors: the first, a mix of solvent use and combustion sources (48%), and the second associated with ETS (20%).
- Combining the VOC and PAH databases allowed for the separation of the two sources previously reported as factor 1 in the VOC data set, with 34% of the variance now allocated to fossil fuel combustion and 30% to solvent use. The third factor was identified as ETS (10%) and a fourth factor emerged, related to consumer products (9%).

VOC AND PAH MODEL DEVELOPMENT

- Five different models were developed to predict personal exposures.
- The modeling suggested that individual-level activity and microenvironmental data are needed for developing good predictive models (model 4).
- In the case of benzene and toluene, personal exposures were better predicted from concentrations measured at home (model 1.1).
- Personal exposures modeled on the basis of housing characteristics (i.e., first-line properties, ETS exposure at home, having an integral garage, and geographic and urban, suburban, or rural location) were not very accurate, giving the worst predictions among all the models proposed (model 5).
- The influences of several activities on VOC levels were assessed from information in the models:
 - ETS exposure is important for predicting 3-ethenylpyridine, pyridine, and 1,3-butadiene exposures, as anticipated.
 - Traffic is a good predictor of exposure to compounds such as benzene, toluene, 1,3-butadiene, and benzo[*a*]anthracene.
 - The use of paints is an activity impacting the levels of ethylbenzene, the xylenes, and the trimethylbenzenes; storing paints in an integral garage increases the levels of *n*-hexane, benzene, toluene, and ethylbenzene.
 - Activities related to the integral garage, such as parking a car in it, increases the levels of benzene, toluene, ethylbenzene, the xylenes, and the trimethylbenzenes.

- The use of fuels other than natural gas for heating increases the concentrations of *n*-hexane, benzene, ethylbenzene, and *p*-xylene.
- Activities that affect personal exposures to PAHs have also been identified during model development:
 - Season affects all the PAHs (except coronene), with lower concentrations in summer than winter.
 - ETS is an important contributor to PAH personal exposure levels.
 - Other situations influencing PAH exposures are the use of a gas cooker and the absence of a stovetop hood.
 - The use of a fireplace is a source of PAHs in personal exposures, but this variable could not be included in the model development, owing to an insufficient number of cases.
- Recommendations for further model development include:
 - Use of a larger data set for personal exposures, especially for the PAH model.
 - Use of a larger data set for microenvironmental concentrations, to perform accurate and detailed stratification of the microenvironments entered into the model.
 - Increased variability among the personal exposures and that captured by the important predictors.
 - Recording of more detailed information in the questionnaires about activities performed and microenvironments visited by the subjects.
 - Inclusion of meteorologic variables.

PERSONAL EXPOSURE VALIDATION

- The model developed from activity information and microenvironmental concentrations (model 4), which was the best-performing model, was tested in a validation data set (an independent set of 25% of the data).
- There is good agreement between the values predicted by model 4 and the measured values in the validation data set for most of the VOC compounds, except naphthalene and 1,3-butadiene.
- For the PAHs, although the model could explain some of the variance, it could only be validated for pyrene. This implies that further study is required to provide predictive capability for PAH concentrations in the general population.

PERSONAL EXPOSURE CATEGORIZATION

- A model for categorizing lower and higher exposures, defined on the basis of exposure thresholds, has been proposed.

- The model correctly classifies the lower exposures to VOCs and PAHs, but the success rate is decreased for higher exposures.

ASSESSING MICROENVIRONMENTAL CONTRIBUTIONS TO PERSONAL EXPOSURES

- The home microenvironment was the microenvironment that contributed most to personal exposure, with contributions ranging from 50–80%.
- The second-largest contribution was from the workplace (8–15%), followed by commuting by car (5–9%).
- For subjects exposed to ETS, the contribution of microenvironments where ETS is present gained importance for exposures to all compounds, at the expense of the contribution of the home microenvironment, especially for the ETS-related compounds.

CONCLUSIONS

Both the environmental concentrations and personal exposure concentrations measured in this study are lower than those reported in the majority of earlier published work. This is consistent with the application of abatement measures to control air toxics emissions, which in independent data sets have been shown to have been very successful in reducing ambient concentrations.

Outdoor, indoor, and transport concentrations were found to be higher in the winter than the summer, and outdoor and in-vehicle concentrations were higher during the morning rush hour than in the afternoon, demonstrating the impact of traffic sources.

Personal exposure concentrations exceeded home indoor concentrations, which in turn exceeded outdoor concentrations. This pattern of concentration ranking, together with the pattern of time spent in each of those microenvironments, allows for calculation of the contribution of individual microenvironments to personal exposures. The home microenvironment is the dominant individual contributor to personal exposure, followed by the workplace and, to a lesser extent, commuting by car. However, for subjects exposed to ETS, microenvironments in which ETS is present gain importance as personal exposure contributors, in some cases becoming similar to home contributions for ETS-related compounds.

A number of generic factors have been shown to be associated with personal exposure concentrations. The presence of an integral garage within the home is associated with both home indoor and personal exposure concentrations for VOCs but not for PAHs. When the home is a first-line property, there is, on average, a small positive increment

in VOC concentrations within the home and a clearer association with the high-molecular-weight PAH concentrations. There are marked differences in both personal exposures and home concentrations between situations influenced by ETS and those free of ETS. The highest PAH levels in personal exposures and home microenvironments were found when fireplaces had been used. Additionally, source apportionment identified a number of sources that contribute to personal exposures. These are fossil fuel combustion, use of solvents, ETS exposure, and use of consumer products.

Several subjects with particularly high personal exposures were identified in this study. The high exposures were attributable to activities within the home and exposures to ETS, which play a major role in determining exposure. For this high-exposure subgroup, the results clearly indicate that outdoor pollution sources make only a modest contribution to personal exposure. Therefore, efforts to reduce the high personal exposures should focus on regulating high-emission household products and exposure to tobacco smoke. On the other hand, the majority of the subjects have lower personal exposures, deriving largely from indoor concentrations, which in turn are related to outdoor sources via infiltration and air exchange. For the general population, abatement measures relating to outdoor sources will have a large relative impact. Since most of the general population is not in the high-exposure category, the population-wide health benefits, especially for non-threshold levels of air toxics, deriving from abatement of outdoor sources may be appreciable. It should also be noted that a reduction in the VOC content of products used in the home and in ETS exposures would have a major benefit in reducing exposures in the general and high-exposure groups.

Urinary biomarkers of ETS exposure correlated strongly with the gas-phase markers of ETS (3-ethenylpyridine and pyridine) and 1,3-butadiene. The urinary ETS biomarkers also correlated strongly with high-molecular-weight PAHs in the personal exposure samples.

Several models have been developed for predicting personal exposures, and validation of the models with an independent data set suggests that individual-level activity and microenvironmental data are needed for good model performance. In contrast, personal exposures modeled using housing characteristics (e.g., having an integral garage) are not very accurate and had the worst performance of the models tested.

The data extracted from the models are indicative of a number of sources making important contributions to the concentrations of air toxics. In the case of VOCs, road traffic, solvents, and ETS are important contributors, whereas for medium- and high-molecular-weight PAHs, traffic and

ETS contributions are important. ETS present in any environment causes increases in the exposure concentrations of a wide range of VOCs and PAHs. Specific activities of the subjects and those around them can lead to elevated exposures, many of which have been identified. However, even when these are included in models of personal exposure, they do not account for all of the exposure to some compounds; hence, some exposure sources appear not to have been recognized.

On the other hand, although the proposed models identify the most important non-weather-related variables for VOCs and some of the factors that affect PAH personal concentrations, the use of such models in various geographic regions, countries, climates, and locations with markedly different sources of pollutants will require caution. Nevertheless, the models presented in this study will serve as a base and will guide the design of studies to develop specific models for a variety of locations.

This study has focused mainly on active adults. Other members of the public who may have greater susceptibilities were not studied. For accurate assessment of the personal exposures of these other populations, it would be necessary to collect information related to lifestyle and activity. Although the models are anticipated to be broadly applicable, this would need to be tested in practice.

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APPENDICES AVAILABLE ON THE WEB

The following materials are available on the HEI Web site <http://pubs.healtheffects.org>. They may also be requested by contacting the Health Effects Institute at 101 Federal Street, Suite 500, Boston, MA 02110, +1-617-488-2300, fax +1-488-2335, or e-mail (pubs@healtheffects.org). Please give (1) the first author, full title, and number of the Research Report and (2) the title of the appendix requested.

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OTHER PUBLICATIONS RESULTING FROM THIS RESEARCH

Brennan M, Vardoulakis S, Harrison RM, Harrad S, Delgado JM, Aquilina N, Baker S, Meddings C. 2006. Indoor air pollution: Developing the evidence base for risk assessment. In: Proceedings of the 9th World Congress on Environmental Health. June 19–23, Dublin, Ireland.

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ABBREVIATIONS AND OTHER TERMS

AER	air exchange rate	ME sampler	microenvironmental sampler (for subject-related microenvironments)
EPA	Environment Protection Agency	MS	mass spectrometer
ETS	environmental tobacco smoke	OME sampler	other microenvironmental sampler (for non–subject-related microenvironments)
EXPOLIS	Air Pollution Exposure Distributions of Adult Urban Populations in Europe	PAH	polycyclic aromatic hydrocarbon
GC	gas chromatograph	PCA	principal components analysis
GM	geometric mean	QA	quality assurance
GSD	geometric standard deviation	QC	quality control
HPLC	High-performance liquid chromatography	<i>R</i>	Pearson correlation coefficient
LC–MS–MS	liquid chromatography–tandem mass spectrometry	<i>R</i> ²	correlation coefficient
<i>m/z</i>	mass-to-charge ratio	RFA	Request for Application
MATCH	Measurement and Modelling of Exposure to Air Toxic Concentrations for Health Effect Studies	SD	standard deviation
		S-PMA	S-phenylmercapturic acid
		VOC	volatile organic compound

Research Report 143, *Measurement and Modeling of Exposure to Selected Air Toxics for Health Effects Studies and Verification by Biomarkers*, R.M. Harrison et al.

INTRODUCTION

Air toxics are a diverse group of air pollutants that are known or suspected, with sufficient exposure, to cause adverse health effects including cancer, damage to the immune, neurologic, reproductive, developmental, or respiratory systems, or other health problems. Monitoring has been performed by some state and local agencies (Health Effects Institute 2000), but substantial uncertainty regarding exposure to air toxics remains, largely because of their presence in the ambient environment at low concentrations. Although environmental exposures to air toxics are generally low, the potential for widespread chronic exposure and the large number of people who are exposed have led to concerns regarding their impact on public health. Estimation of the health risks of exposure to air toxics is complicated by the fact that there are multiple sources of air toxics. These may be outdoor and indoor (e.g., environmental tobacco smoke [ETS*], building materials, consumer products, and cooking).

One strategy for understanding potential health effects from exposure to toxic pollutants is to study populations living in areas with high concentrations of these pollutants (areas often referred to as hot spots). Because hot spots have levels higher than those to which the general public is exposed, these areas offer the opportunity to assess exposure and potential health effects in smaller populations. In 2003, HEI issued Request for Applications (RFA) 03-01, "Assessing Exposure to Air Toxics," which sought studies to assess ambient concentrations and personal exposure in areas likely to have elevated concentrations of air toxics. The specific pollutants focused on in the RFA were

Dr. Harrison's 3-year study, "Measuring and Modeling of Exposure to Air Toxics and Verification by Biomarker," began in December 2004. Total expenditures were \$978,382. The draft Investigators' Report from Harrison and colleagues was received for review in April 2008. A revised report, received in September 2008, was accepted for publication in October 2008. During the review process, the HEI Health Review Committee and the investigators had the opportunity to exchange comments and to clarify issues in both the Investigators' Report and in the Review Committee's Critique.

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* A list of abbreviations and other terms appears at the end of the Investigators' Report.

derived from the priority lists of mobile-source and urban air toxics developed by the U.S. Environmental Protection Agency (1999, 2001b); all are produced by mobile sources, and many are also produced by other sources, including indoor sources (Health Effects Institute 2003).

In response to RFA 03-1, Dr. Roy Harrison of the University of Birmingham submitted an application for a three-year study, "Measuring and Modeling of Exposure to Air Toxics and Verification by Biomarker." He proposed to investigate personal exposures to a broad group of air toxics, with the goal of developing detailed personal exposure models that take various microenvironments into account. The HEI Research Committee recommended Dr. Harrison's application for funding because the study had the potential to capture adequate variation in exposure concentrations and thus provide important information on personal exposures to air toxics.

APPROACH

The specific aims of the study were to:

1. Use personal monitoring and measurement of urinary biomarkers to assess daily exposures to a selection of air toxics—volatile organic compounds (VOCs), including 1,3-butadiene, and polycyclic aromatic hydrocarbons (PAHs)—among 100 healthy adult non-smokers with a range of residential locations and exposures to non-traffic sources.
2. Determine microenvironmental concentrations of a group of air toxics, with an emphasis on spatial and temporal variations in concentrations.
3. Develop models to predict personal exposures on the basis of microenvironmental concentrations and data from time–activity diaries and compare measured personal exposures with modeled estimates of exposure.
4. Produce a scheme for categorizing exposure (by compound) according to the location of residence and other lifestyle and exposure factors, including ETS, for use in the design of future case–control and ecologic studies of cancer incidence.

Participants were 100 healthy adult non-smokers residing in the United Kingdom in an urban area (London or Birmingham, and the urban center of West Midlands), a

suburban area (suburban West Midlands), and a rural area (rural West Midlands and South Wales) that were expected to have different traffic exposures: high traffic, intermediate traffic, and a gradient between light and heavy traffic, respectively. The strategy of subject selection incorporated information on urban, suburban, or rural location; ETS exposure; presence or absence of an integral garage at the residence (generally referred to as an "attached" garage, in the United States); and proximity to a major road.

Repeated measurements of exposure to selected air toxics were made for each participant and also for major microenvironments, including the home and workplace. Measurements included five repeated 24-hour measurements of personal exposure to VOCs (including 1,3-butadiene) per participant, five urine samples collected to test for urinary biomarkers (PAH metabolites, cotinine, and *trans*-3'-hydroxycotinine) per participant, and one 24-hour measurement of particle-phase PAHs per participant, plus concurrent measurement of microenvironmental exposures at participants' homes and workplaces, for a total of 200 VOC, 190 1,3-butadiene, and 168 PAH samples, as well as measurements in other major microenvironments.

BRIEF SUMMARY OF RESULTS

Measured environmental and personal concentrations were on the lower end of those in the previously published literature (Wallace 1989 a,b; Brown et al. 1994; Leung and Harrison 1998; Edwards et al. 2001; HEI Air Toxics Review Panel 2007). Environmental concentrations were influenced by traffic and season (with higher concentrations in the winter). The presence of an integral garage resulted in higher indoor concentrations in the home and higher personal exposures to VOCs but not PAHs.

Consistent with other research, personal exposures generally exceeded indoor concentrations, which in turn exceeded outdoor concentrations (Turpin et al. 2007). Personal exposures were most heavily influenced by the home microenvironment and were higher in the presence of fossil fuel combustion, exposure to ETS, solvent use, use of selected consumer products, and commuting. After the home microenvironment, the workplace and commuting were the largest contributors to personal exposure. Urinary biomarkers were strongly correlated with gas-phase markers of ETS and 1,3-butadiene, as well as high-molecular-weight PAHs, in personal samples.

Statistical models based on microenvironmental factors and lifestyle were able to explain a fair amount of the variance in personal exposures for selected VOCs (approximately 50% of the variance in benzene exposures and 75% of that in 3-ethenylpyridine exposures) but were less

predictive of PAH exposures (with the best model for benzo[*a*]pyrene explaining only 35% of the variance). Models could be statistically validated for nearly all VOCs but, with the exception of pyrene, could not be validated for PAHs.

CRITIQUE

This study serves as a rich source of recent information on personal exposures to selected air toxics across a range of residential locations and exposures to non-traffic sources, with attention to spatial variation and areas in which air toxics exposures were likely to be elevated. Harrison et al. collected an impressive amount of data, which were analyzed carefully and subjected to stringent quality-assurance and quality-control procedures. Using appropriate methods of microenvironmental and personal monitoring and of measuring urinary biomarkers, they assessed daily exposures to over 30 air toxics, several of which are known to be hazardous to human health, with a focus on VOCs and PAHs. Overall, measured concentrations of the selected air toxics and personal exposures to them were somewhat lower than those reported in previous studies (HEI Air Toxics Review Panel 2007).

The sampling frame was designed to capture a range of exposures among healthy residents of urban, suburban, and rural populations. Challenges in the recruitment process resulted in a somewhat unbalanced sample, however, and a very small number of participants in some subgroups. Thus, although the authors reported all results that were based on four or more data points, some of these findings should be interpreted with caution. For example, few subjects in urban and suburban homes, and none in rural homes, were exposed to ETS at home. In addition, integral garages were present in 25% of both rural and suburban homes but were an uncommon source of exposure for urban residents; all but one of the London residents lived in flats.

Most of the participants were young adults (26–35 years of age) who reported spending little time commuting to work each day (the vast majority, < 15 minutes) but a substantial amount of time (mean, 86 minutes each day) in transit overall. The authors did find differences in the levels of personal exposure on the basis of age (18–65 years vs. ≥ 66 years) and professional status (retired vs. unemployed, or housewife vs. student vs. office workers), but again, definitive conclusions cannot be made, as the data are based on very small numbers of participants in some of the subgroups. The large proportion of young adults in the study also limits the ability to generalize results to more susceptible populations, such as children and the elderly.

Information on key determinants of exposure, including proximity to traffic, presence or absence of an integral garage, exposure to ETS, and extent of urbanization were identified by the authors largely on the basis of prior research. Harrison et al. did a thorough job of assessing the impact of nonresidential exposures, including proximity to known sources and specific activities, on personal exposures. For example, they hypothesized that those living within 20 meters of a heavily trafficked highway and those exposed to household ETS would be exposed to elevated levels of air toxics; indeed, living close to a heavily trafficked highway contributed to elevated PAH and toluene concentrations and ETS did affect VOC and PAH exposures. Nevertheless, with participants spending more than half of their time indoors at home, the home environment remained the biggest contributor to personal exposures (responsible for 50–80%); the second-largest contributor, the workplace, contributed only 8–15%.

Home air exchange rate (AER), a major determinant of exposure with large seasonal variability, was not explicitly evaluated, however. The authors chose to focus their efforts on the collection of additional microenvironmental samples, rather than direct measurements of home AER, as they felt microenvironmental data would better inform the prediction of personal exposures. However, they did collect data on qualitative indicators of AER, such as the degree of ventilation, the use or nonuse of air conditioning, and the presence or absence of open windows.

Harrison et al. endeavored to develop models to predict personal exposures on the basis of microenvironmental concentration data and time–activity diaries, with the idea that these models could inform the design of future health studies. They used an innovative approach to modeling, in which models were fitted using 75% of the data and were then validated on a test set comprising the remaining 25% of the data. Yet the model developed to explain the variance in personal exposures on the basis of a priori determinants of exposure did not yield very predictive results. Indeed, the most predictive statistical models did only a fair-to-moderate job of predicting personal exposures. Part of the inability to effectively model exposures using housing characteristics may be due to the lack of measured characteristics of home ventilation, particularly AER in the home; the addition of qualitative indicators of AER did not improve model predictions.

Harrison et al. initially intended to evaluate the exposure–response relationships of urinary biomarkers and corresponding air toxics in environmental exposures. Urine samples collected concurrently with air toxics samples were analyzed for urinary metabolites of ETS and PAHs, and the biomarker data were plotted against concentrations of

selected VOCs and PAHs in personal exposures. Because several potentially confounding factors (such as sex, age, and dietary exposures) were not taken into account, the analysis as presented is more descriptive than a full characterization of potential exposure–response relationships.

In summary, this report underscores the challenges of accurately predicting personal exposures. Harrison et al. collected an impressive amount of high-quality data on personal exposures, microenvironmental concentrations, residential characteristics, and time–activity data. They applied appropriate approaches to predicting personal exposures with these data, and yet the resulting models had only limited accuracy. This is most likely due to a combination of factors, including the lack of data on AER, as well as the inherent variability in personal activities and resulting exposures. Moreover, although the models identified the most important non–weather-related variables predicting exposure, particularly to VOCs, the transferability of these models to other settings, particularly in the absence of data on meteorologic characteristics, is somewhat limited. These limitations suggest that even though personal exposure monitoring requires extensive time and equipment, the science is not yet at a point at which exposures to VOCs and PAHs can be reliably predicted from time–activity patterns and microenvironmental concentrations alone.

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