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Investigation of benzo(a)pyrene in outdoor air, indoor air and personal inhalation air

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ABSTRACT

Title of Thesis: INVESTIGATION OF BENZO(A)PYRENE IN OUTDOOR AIR, INDOOR AIR AND PERSONAL INHALATION AIR

Hsiu-wen Chen, Master of Science in Env. Sci., 1989.

Thesis directed by: Dr. Arthur Greenberg

The assessment of human exposure to an environmental contaminant requires the measurement of the concentrations levels present in each pathway of possible contact.

This was an investigation of human exposure to an environmental pollutant, benzo(a)pyrene, in outdoor air, indoor air and personal inhalation air samples. The analytical procedure involved extraction by ultrasonic and separation by thin layer chromatography, and detection by fluorescence. The resulting data was used to develop a profile of indoor and outdoor human exposure to B(a)P during the year.

INVESTIGATION OF BENZO(A)PYRENE IN OUTDOOR AIR, INDOOR AIR AND PERSONAL INHALATION AIR

by

Hsiu-wen Chen

Thesis submitted to the faculty of the Graduate School of
the New Jersey Institute of Technology
in partial fulfillment of the requirements for
the degree of Master of Science in Environmental Science
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Chapter I INTRODUCTION

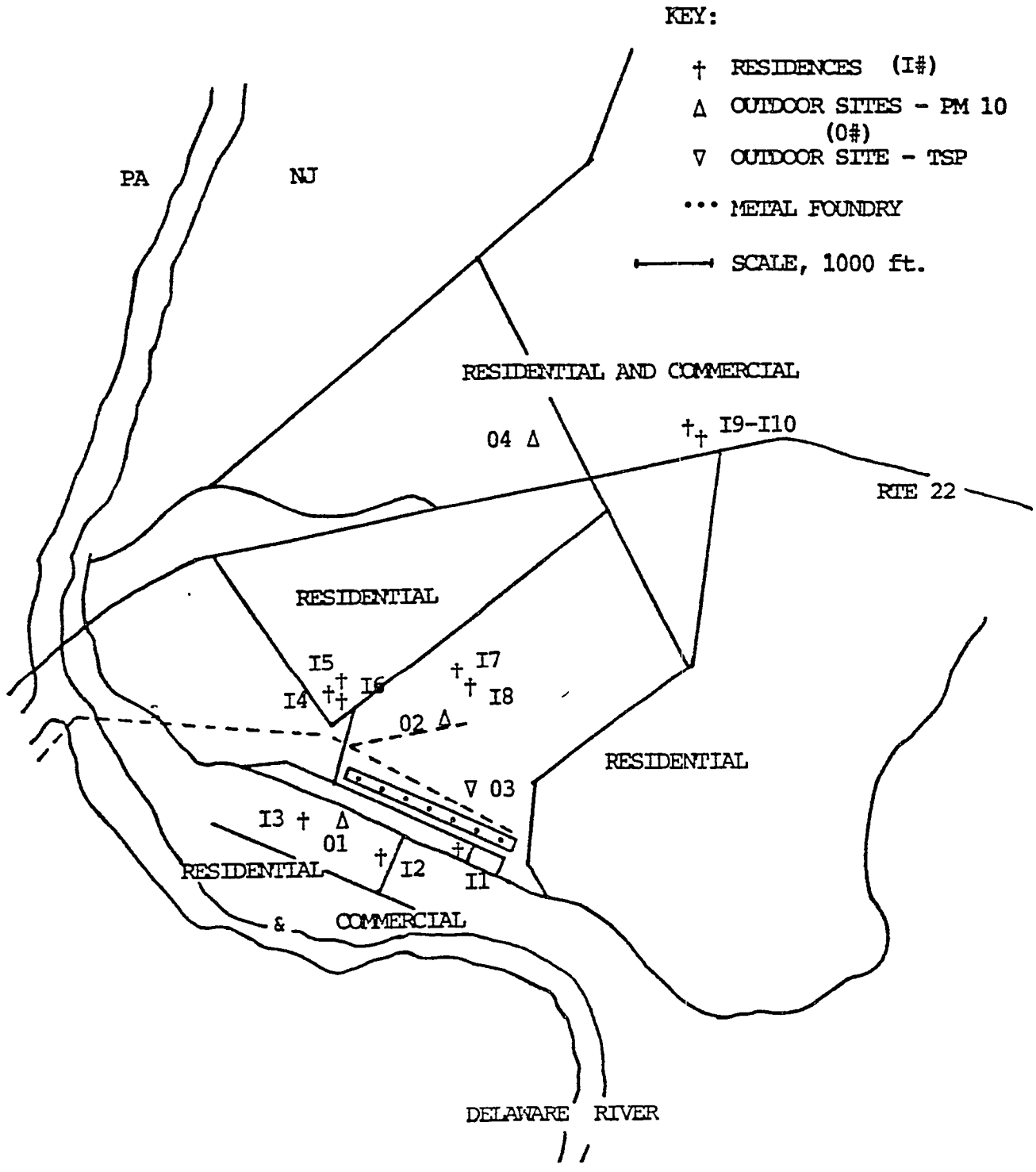
The assessment of human exposure to an environmental contaminant requires the measurement of the levels present in each pathway of possible contact. This thesis, the development of methods and determination of very low levels of airborne benzo(a)pyrene relates to the determination of human exposure via inhalation of this important ubiquitous carcinogen. This study focused on Phillipburg, New Jersey, a town having a metal foundry and some high previous B(a)P measurements. [1] Combustion sources used in the home and personal activites were recorded in a daily questionnaire and ingestion pathways were also explored. Inhalation and ingestion are the major suspected routine pathways of human exposure to benzo(a)pyrene.

The traditional approach to examining the concentration patterns of pollutants or the uptake by humans usually has been to focus on a single medium, e.g. air or water. Significant progress has been made in extending study designs to multimedia in recent years.[2-6] The exposure assessment guidelines developed by the EPA have illustrated

some of the important types of source, measurement and pathway information necessary to conduct a multimedia analysis for a pollutant or class of pollutants.[2-6] The need to continue the development of approaches for examining exposure led to the Total Human Environmental Exposure Study (THEES) in collaboration with Professors Paul Lioy and Jed Waldman at Robert Wood Johnson Medical School (RWJMS) of the University of Medicine and Dentistry of New Jersey (UMDNJ).

A study of total human exposure to benzo(a)pyrene was conducted in a foundry-impacted area in Phillipsburg, New Jersey along with appropriate control sites.(Figure I-1) A two-week microenvironmental field experiment was conducted in January and February, 1987. The project was formulated as a model study for methodology and as a prototype for investigations of human exposure to other environmental contaminants. The objectives are to quantify the different exposure pathways and to develop models for estimating total exposure to benzo(a)pyrene. The approach involves monitoring benzo(a)pyrene in all media containing significant benzo(a)pyrene levels that the study population would come in contact with and quantitatively assessing the contributions of each exposure route.

Figure I-1 THE PHILLIPSBURG AREA AND SITE LOCATIONS



Indoor air quality is a major determinant of personal exposure to pollutants in the environment. People spend much of their time in numerous indoor environments. Appropriate research has shown that the typical person in the U.S. spends 58%-78% of her/his time in the home.[7]

Extensive measurements have been and are being made of the presence and concentrations of many types of pollutants in the outdoor air. In contrast, considering the importance of the problem, very few data have been gathered on the presence, concentration, and generation of pollutants in indoor environments and on the penetraion of pollutants from the outdoor environment into buildings. Even though a large number of publications include some information applicable to the problem of indoor pollution, only recently have comprehensive investigations of the problem been initiated.

The outdoor concentrations of benzo(a)pyrene were similar to the values previously recorded in Phillipsburg [1] and are also within the range of values recorded in other urban areas, 0.5-2.9 ng/m^3.[8] Very few measurements of benzo(a)pyrene have been made indoors. In one instance, a sports arena, the maximum concentration of benzo(a)pyrene

was 21 ng/m^3.[9] An indoor-outdoor polycyclic aromatic hydrocarbon study by Butler and Crossley[10] in Birmingham U.K. during the late 1970's found mean concentrations of benzo(a)pyrene of 2.1 ng/m^3 with a peak of approximately 3 ng/m^3. More recently Wilson et al.[11] reported results for benzo(a)pyrene in two homes that ranged from 0.7 to 1.5 ng/m^3. The maximum levels found in the THEES study were 2-4 times higher than the maximum observed in the cited home studies. However, further studies in other seasons are necessary to obtain a clearer understanding of the nature of benzo(a)pyrene in the indoor environment.

Since we could not measure the concentration present in the potential air microenvironments other than the home and ambient air, an estimate of daily inhalation exposure needs to account for the hours not spent at home. By using the ambient benzo(a)pyrene as the minimum concentration which a person encounters, the remaining benzo(a)pyrene exposure can be estimated. The paired benzo(a)pyrene exposure for each home and each pathway indicated that the potential intake could be similar in each medium. Of the twenty exposure weeks, ten had higher food benzo(a)pyrene exposure, and ten had higher inhalation benzo(a)pyrene exposures. In the instances with greater benzo(a)pyrene exposures due to the

food pathway, the values were at least two times greater than those contributed by inhalation. In the other ten cases, the maximum values of B(a)P were lower, but the relative inhalation exposures were more than two times greater than the food exposure in most instance. However, the range of food exposures was much greater than the of air exposure.

Our Phase I study focussed on the examination of the benzo(a)pyrene present in the major pathways of exposure to man. Microenvironmental sampling was conducted for four media which included outdoor and indoor air, food, water and all of these four media in Phase I have been discussed previously. [12] The nondetectable level of B(a)P in tap water eliminated this as a pathway of interest for Phase II and III. The present study reported only Phase II and Phase III research. Besides outdoor and indoor air samples and food samples, [13] in Phase II and III, the monitoring protocals were expanded to include personal exposure of individuals to benzo(a)pyrene through personal air sampling for benzo(a)pyrene and personal biological monitoring for benzo(a)pyrene metabolites and adducts in urine and blood respectively.[14]

In the present study, the well-known USEPA technique for benzo(a)pyrene monitoring in outdoor air (typically 1600-1800 m^3/sample) has been extended to indoor air (typically 14 m^3/sample) and then to personal air (typically 4 m^3/ sample). In addition to extending the utility of this technique, running numerous environmental samples and controls, the technique was also examined for the determination of pyrene. The reason for the extension to pyrene will be discussed later.

The objective of quantitative risk assessment is to arrive at a value or range of values that describe the possible adverse effects on human health associated with exposure to a known or suspected toxic substance.

A major reason for selecting benzo(a)pyrene is that it is emitted during outdoor/ or indoor fossil fuel combustion (including coal and wood burning), smelting and is found in the emission from stoves and cigarettes. [15,16] A major focus of the Total Human Environmental Exposure Study (THEES) is linking personal as well as indoor and outdoor activites to the variation of total exposure to

benzo(a)pyrene. The primary purpose of this project has been to illustrate the process of health risk assessment in the context of indoor air pollution. The precise and sensitive measurements reported here have extended the range of utility of the benzo(a)pyrene determination.

REFERENCE

[1] R. Harkov and A. Greenberg, "Benzo(a)pyrene in New Jersey-Results from a Twenty-Seven site study." J. Air Poll. Control Assoc. 35, 238-243, 1985

[2] L. Wallace, R. Zweidinger, M. Erickson, S. Cooper, D. Whitaker and E. Pellizari. "Monitoring Individual Exposure Measurements of Volatile Organic Compounds In Breathing Zone Air, Drinking Water and Exhaled Breath"., Env. Internat. 8, 269-282, 1982.

[3] L. Wallace, E. Pellizari, L. Sheldon, T. Hartwell, C. Sparacino, and H. Zelon, 1986 "The Total Exposure Assessment Methodology (TEAM) Study" : Direct measurements of personal exposure through air and water for 600 residents of seversl U.S. cities; in Pollutants in a Multimedia Environment, Plenum Press, N.Y. 1986.

[4] W. Ott, "Total Human Exposure" Env. Sci. & Tech., 19, 880-891, 1985.

[5] O. Severn, "Exposure Assessment" Env. Sci. & Tech., 21, 1159-1163, 1987.

[6] Federal Register. EPA Proposed Guidelines for Exposure Assessment, Vol. 48. No. 227, 46305-46312, Nov. 23, 1984.

[7] P. Walsh, C. Dudney, E. Copenhaver, " Indoor Air Quality " CRC Press. Inc. Fourth Printing, 1986.

[8] J. Santodonato, P. Howard, D. Basu, " Health and Ecological Assessment of Polynuclear Aromatic Hydrocarbons."J. of Env. Path and Tox. 5,1-364, 1981.

[9] L. Elliott, D. Rowe, Air Quality During Public Gatherings, JAPCA, 25,635-636, 1975.

[10] J. Butler, P. Crossley, An Appraisal of Realtive Airbone Suburban Concentrations of Polycuclic Aromatic Hydrocarbons Monitored Indoor and Outdoors. Sci. of Tox. Environ., 11, 53-58, 1979.

[11] N.K. Wilson, R.G. Lewis, C.C. Chang, B.A. Peterson, Analytical and Sampling Methodology for Characterization of Polynuclear Aromatic Compounds in Indoor Air 85-30A, Proceeding of the 78th Annual meeting of APCA. Detroit MI, 1985.

[12] S. K. Liang, Master Dissertation. New Jersey Institute of Technology, October, 1988.

[13] C. H. Hsu, Master Dissertation. New Jersey Institute of Technology, December, 1989.

[14] Z. Ouyang, Master Dissertation New Jersey Institute of Technology, October, 1989.

[15] National Research Council. "Indoor Air Pollutants", National Academy Press, Washsington, D.C. 1985.

[16] A. Bjorseth and T. Ramdahl, Handbook of Polycyclic Hydrocarbons, Vol. 2, Emission Sources and Recent Advaces in Analytic Chemistry, Dekker, New York. 1981.

Chapter II. BACKGROUND INFORMATION

Benzo(a)pyrene was first prepared by Kennaway's group at the Institute of Cancer Research, London, in 1933,[1] in a series of experiments to determine the chemical composition of the carcinogenic constituents of coal tar.

The history of benzo(a)pyrene during the following fifty years was largely that of unravelling the mechanism of chemical carcinogenesis, and other work on the compound was incidental to that. The newly discovered carcinogen soon attracted interest. As shown below, the number of papers referring to benzo(a)pyrene published each year grew exponentially after the Would War II : [2]

Year	1940	1945	1950	1955	1960	1965	1970	1975	1980
Paper	32	21	24	39	70	130	160	380	800

As a model carcinogen, benzo(a)pyrene has the following advantages for an exposure study :

(i) It is widely distributed in the environment and therefore likely to be relevant to cancer in man.

(ii) It is invariably formed when organic matter is burned, and is therefore a useful indicator of industrial pollution.

(iii) It is readily detected and quantitated by its fluorescence. Its pharmacology was therefore readily studied before the introduction of radioactive tracers.

(iv) It has proved to be one of the most potent carcinogens known in practically every species tested.

Early experiments were concerned with improving the technique for induction of tumors with benzo(a)pyrene. During the 1950s and 1960s a number of advances were made towards the understanding of benzo(a)pyrene interactions with cellular components.

Benzo(a)pyrene is a chemical commonly found in the emission products from most types of fuel combustion [Table II-1] whether it occurs in the engine of an automobile,the fireplace of a home, or an industrial installation. It is known to be a human carcinogen. [2]

The U.S. Environmental Protection Agency asked the National Academy of Sciences National Research Council to assess the health risks of humans exposed to the compounds that can be identified and characterized in the atmosphere, to identify those persons most susceptible to the toxic effects of the compounds, and to characterize the other major sources of human exposure, with emphasis on emission from mobile sources. The NRC report "INDOOR POLLUTANTS" assesses some of the sources of pollutants indoors and their effect on air quality. A preliminary study [3] reports B(a)P concentrations both inside and outside a home with a wood burning stove and a residence with a fireplace. When wood stoves were in use, the benzo(a)pyrene concentration monitored over 24 hours indoors was five times higher than on days when stoves were not use.

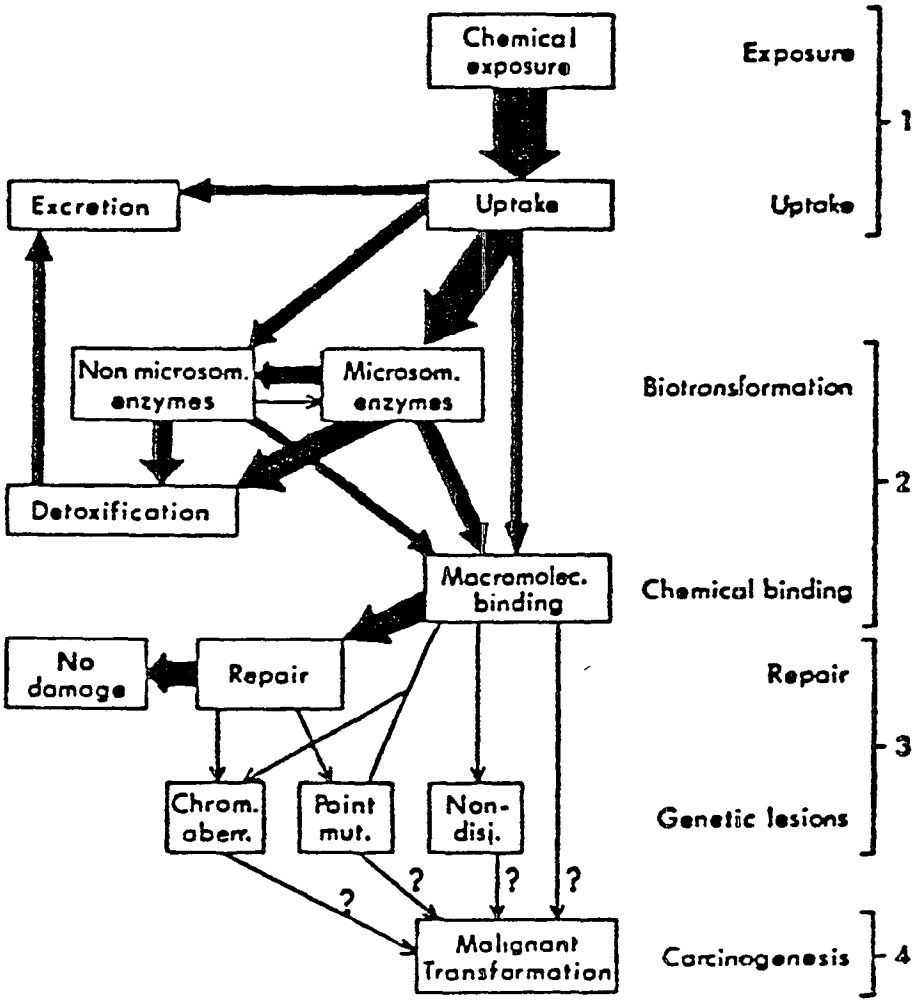
Table II-1

Estimated Total Annual Benzo(a)pyrene Emission for 1975 and 1985

Estimate	Benzo(a)pyrene Emission (metric tons) 1975	1985
Minimum	346	67
Intermediate	588	385
Maximum	1,676	885

* adapted from A. Bjorseth [14].

Figure II-1



Sequence of possible events from exposure to carcinogenesis.

Carcinogenesis

We use the term carcinogenic to mean 'capable of initiating the growth of tumors', whether these are malignant or not. This is a long-established usage of the word, though the terms oncogenic or tumorigenic are preferred by some authors as being more precise.

Benzo(a)pyrene has been tested for carcinogenicity many times, in several species of animal, by various routes of administration,[Figure II-1] alone and in combination with other substances. The mass of data thus accumulated is too large and varied to be listed and assessed in one volume; we shall cover only certain aspects of general interest.[1]

Inhalation or Intratracheal Instillation

Inhalation is one natural means of exposure, and animal studies of exposure involve introduction of microscopic particles of the solid into the lung which are readily absorbed into the bloodstream. However, this is difficult to carry out with a nonvolatile substance like

benzo(a)pyrene. More commonly benzo(a)pyrene, alone or mixed with an inorganic solid, is ground to a fine powder, suspended in saline and introduced into the trachea of anaesthetized animals with a long syringe. Rats and hamsters are generally used,as they have a lower spontaneous incidence of lung tumors than most laboratory mice. Benzo(a)pyrene induces both lung and stomach tumours in this way.[4]

The polycyclic aromatic hydrocarbons (PAH) have been reviewed previously as components of atmospheric pollution and as potential human health hazards. High atmospheric concentrations arise mostly from stationary combustion, especially that of coal, wood, and oil. Wood burning stoves and fireplaces, currently nonregulated sources of PAH, are ubiquitous and are important and increasing sources of atmospheric PAH in urban areas, as well as in rural areas with restricted air flow.

The toxicity of PAH including mutagenesis, carcinogenesis, and teratogenesis, result from multistage processes, and variations in any of the intermediate stages can influence susceptibility to the effects. Sensitivity to

PAH induced biologic effects is probably controlled at the level of uptake into particular cells, metabolic activation or inactivation of the parent PAH , capacity of cells to repair PAH metabolite DNA adducts, capacity of cells to express DNA damage and allow progression to the phenotype of a mutant or tumor cell, and immunocompetence of the host. Compilation of data from humans and animal model systems has demonstrated a degree of genetic regulation at each of these stages but the information is far too sketchy for specific conclusions to be drawn on the role of PAH.[2]

PAH in both human and animal systems are taken up and metabolized by microsomal monooxygenases that are under some sort of genetic regulation. In murine-model systems, susceptibility to carcinogenesis induced by PAH is genetically linked to the capacity to respond to and metabolize these chemicals. [Table II-2] In humans, development of cigarette smoke associated lung cancers also may be linked to the capacity to respond to and metabolize PAH.[5]

Human exposure to PAH is almost exclusively via the

Table II-2

Mutagenic Efficacy of PAH in Relation to B(a)P

Compound	Concentration (ug/ml)	Relative Mutagenic Potency*
2-Methylanthracene	15.4	0.15
9-Methylanthracene	14.4	0.08
1-Methylphenanthrene	15.4	0.50
2-Methylphenanthrene	7.7	0.30
Pyrene	28.3	0.07
1-Methylpyrene	17.3	0.05
Cyclopenta[cd]pyrene	13.9	1.51
Benz[a]anthracene	14.8	0.14
Chrysene	10.3	0.20
Fluoranthene	1.0	1.00
Benzo[a]pyrene	1.3	1.00
Benzo[e]pyrene	22.7	0.11
Perylene	2.8	6.00
Anthanthrene	12.1	0.08
Dibenz[ac]anthracene	3.6	0.77

* Relative to benzo[a]pyrene, set at 1.00; rate-limiting factor is concentration that produced too much cell death.

gastrointestinal and respiratory tracts and approximately 99% of these substances is ingested in the diet.[5] PAH are ubiquitous in food stuffs. The PAH content of most foods before preparation is quite low, but some have surprisingly high concentrations, presumably as a result of pollution from soils, irrigation waters, and atmospheric fallout and perhaps from the initial phases of food processing. The contaminants include 100 or even more PAH. [5] The mode of cooking, especially broiling, also affects the composition and quantity of PAH in foods. [Table II-3]

Pyrene

Our studies of B(a)P metabolites in urine indicated problems in recoveries and since pyrene produces high levels of 1-hydroxypyrene as a metabolite and since this was more easily analyzed, we developed an interest in human exposure to pyrene.[6]

Early studies on pyrene metabolism were in rats and showed increased urinary excretion of sulfuric acid esters

Table II-3

Estimated Daily Human Exposure to PAH from Air Water and Food

Source	Benzo(a)pyrene (ug)	Total PAH (ug)
Air	0.0095-0.0435	0.207
Water	0.0011	0.027
Food	0.16-1.6	1.6-16

* adapted from A. Bjorseth [14].

and glucuronic acid conjugates. Later, 1-hydroxypyrene and 1,6-dihydroxypyrene were identified. More definitive studies of pyrene metabolism were performed in rabbits and rats by analysis of urinary metabolites after intraperitoneal injection. While the PAH pyrene is noncarcinogenic and nonmutagenic, 1-nitropyrene, which is found in quantities comparable to benzo(a)pyrene, is a carcinogen in animals and a direct mutagen in bacterial bioassay.[7]

EXPERIMENTAL-ANIMAL STUDIES

Some data on cocarcinogenic activity of PAH with other chemicals are available, but this data base needs to be strengthened, and PAH other than benzo(a)pyrene need to be studied further. Specifically, data are needed to establish whether various PAH exhibit cocarcinogenic activity with other components of exhaust from mobile sources or emission from other combustion sources, especially wood smoke. The potential promoting activity of PAH (including BaP) needs to established. A model for promotion other than the mouse skin tumorigenesis system is needed. Of special interest

would be a promotion system using human cells.

Extrapolation of findings from animal studies to humans is tentative without additional biochemical and pharmacokinetic data. Sorting out the toxic chimicals in any complex mixture (such as automobile exhaust, wood smoke, or cigarette smoke) is always difficult. Animal models and compound-specific testing systems are needed to ascertain the toxic effects (if any) of long-term (chronic) exposure of animals to diesel exhaust and other complex kinds of emission. In this regard, it is important to stress that the animal model systems include introduction of the PAH (alone, in mixtures, and bound to particles) into the diets of animals in lifetime studies of carcinogenesis. Such dietary exposure is based on the data that indicate that ingestion contributes heavily to the body burden of the PAH. As results from these studies begin to distinguish the toxic components, biochemical and pharmacokinetic data on experimental primates (e.g., squirrel monkeys) will be particularly useful in confirming the findings in animal species and extrapolating to huamns. With improving characterization of the toxic components, studies should be conducted on lung depositon, uptake, and clearance of PAH. Studies on the relationships of carrier-particle size,

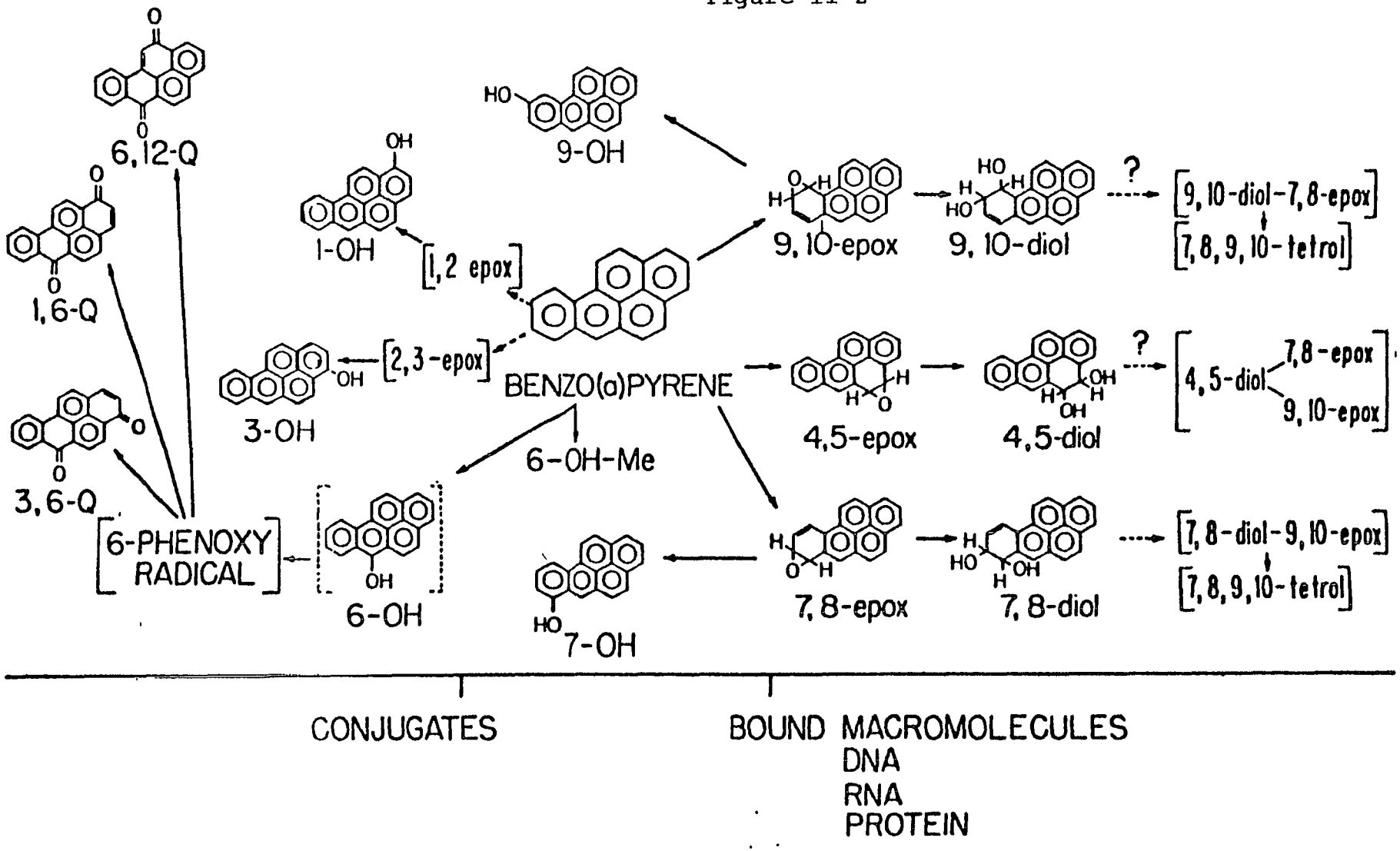
surface properties in the submicrometer range, and absorption and adsorption of individual PAHs should be continued and expanded with an eye to learning the source of the greatest exposure to the toxic chemicals.

Metabolism

The fate of benzo(a)pyrene in animals has been widely studied. [Figure II-2] After injection of a solution or suspension of benzo(a)pyrene under the skin, it is transported round the body in the blood and lymph vessels. However, benzo(a)pyrene reaches the bloodstream, it is there transported to all parts of the body and taken up rapidly into the tissues. Injected benzo(a)pyrene into a rat vein and found that it disappeared with a half-life of less then 5 min. [8]

The distribution of benzo(a)pyrene in the body can be studied by whole-body autoradiography of animals treated with labelled benzo(a)pyrene. An hour after administration, it is located chiefly in the liver,lungs and kidney, at the site of injection, and in fat deposits round the body. Peak concertrations in rat liver occurred at 0.5 hour, in the

Figure II-2



Composite of metabolic products of benzo[a]pyrene.

stomach and intestine at 0.5-1 hour, and in kidney 6 hours after exposure [9] The chief site of metabolism is the liver, and mainly the parenchymal cells there.

Benzo(a)pyrene and Human cancer

Does benzo(a)pyrene cause cancer in man? This unanswered question may be resolved into two components. Do environmental sources of benzo(a)pyrene cause cancer? How much does benzo(a)pyrene contribute to their total effect?

The incidence of cancer in workers exposed to soot, tar and various oils was reviewed by Kipling and Cooke[10] Increased incidences of cancer have been noted in chimney sweeps, users of pitch, gas workers(i.e. where coal is carbonized), coke-oven workers at steelworks, and spinners and metal workers exposed to lubricating oils. This is probably due to the PAH and related heterocyclic compounds that these people take into their lungs or skin. It is more difficult to assess the incidence of cancer in the general public which can be ascribed to the much lower concentrations of soot tar and smoke to which they are

exposed.

It is not possible to assess the extent to which benzo(a)pyrene contributes to the total carcinogenicity of these pollutants in man, but it can be done for animals. Some 11% of the total carcinogenicity of flue gas condensate and about 7% of the carcinogenicity of automobile exhaust condensate could be attributed to its BaP content [11] Moreover, the combination of benzo(a)pyrene with just one cocarcinogen, cyclopenta[cd]purene may be sufficient to account for the whole of the carcinogenicity of automobile exhaust [12]

Ultimately, indoor air quality must be examined and understood on an individual pollutant basis. In some cases, the federal government or another national body has examined the health effects data in specific pollutants and established guidelines for limiting exposure concentrations in various human contact situations. The ultimate analysis of any indoor air quality situation requires that the biological effects of airborne concentrations of various pollutants be examined. Health risk analysis is a rapidly evolving methodology which deals with two major problem areas. One is how best to predict effects of chemicals at

low dose and dose rate when most data are collected under conditions at high dose and dose rate. The other is how to predict effects on humans when most data are collected in biological systems other than intact human beings.

HEALTH EFFECTS ASSESSMENT

Along with measurement or estimation of exposure, a health risk assessment must also include an estimation of the relationship between the exposure and an adverse response in human populations. Exposure-response relationships are ideally established using toxicological data from human studies; however, the lack of such studies may necessitate the use of toxicological data from subhuman systems and a model or methodology that enables these data to be used to estimate human risk.

Data relating exposure to complex mixtures of organic pollutants, and more specifically,estimated or measured B(a)P concentrations, and adverse human health effects come from a variety of sources. Recently, a number of investigators [13] have attempted to utilize the

available data to establish a unit risk measure for benzo(a)pyrene. A unit risk measure is defined here as the increased or excess risk associated with the continuous lifetime exposure to one unit (ng/m^3) of B(a)P. It may be expressed as either an absolute or a proportional increase in lung cancer deaths associated with each nanogram per cubic meter of Benzo(a)pyrene.

REFERENCE

[1] E. Kennaway, Further experiments on cancer-producing substances. Biochem. J., 24, 497-504, 1930.

[2] M. Osborne, N. Crosby, Benzopyrenes , Cambridge, London. 1987.

[3] D. Moschandreas, J. Zabransky and H. Rector, "The effects of woodburing on the indoor residential air quality. Env. Int., 4, 463, 1980.

[4] C. Jones, R. Santella, E. Huberman, J. Selkirk, and D. Grunberger, "Cell specific activation of B(a)P by fibroblasts and hepatocytes. Carcinogenesis. 4, 1351-7, 1983.

[5] G. Becher, " PAH in work atmospheres Occurrence and Determination ", Petroleum Research Center, Bergen, Norway. 1986.

[6] Z. Ouyang, Master Dissertation New Jersey Institute of Technology, October, 1989.

[7] Rosenkranz, 1982, 1984.

[8] E. Schlede, R. Kuntzman, S. Haber and A. Conney, " Effect of enzyme induction on the metabolism and tissue distribution of B(a)P ", Cancer Res., 30, 2893-7, 1970.

[9] C. Michell, " Distribution and retention of B(a)P in rats after inhalation.", Toxicol Lett., 11, 35-42, Toxicology, 28, 65-73, 1982.

[10] M. Kipling, M. Cooke, " Soot tars and oils as causes of occupational cancer." In Chemical Carcinogens, ed. C.E. Searle, pp. 165-74. Washington: American Chemical Society.

[11] G. Grimmer, H. Brune, R. Deutsch, G. Dettbarn, J. Misfeld, W. Abel and J. Timm, " Contribution of PAH fractions with different boiling ranges to the carcinogenic impact of emission condensate from coal-fired residential furnaces as evaluated by topical application to mice." Cancer Lett., 28, 203-11, 1985.

[12] E. Cavalieri, A. Munhall, E. Rogan, S. Salmasi and K. Patil, " Syncarcinogenic effect of the environmental pollutants cyclopenteno(cd)pyrene and B(a)P in mouse skin. Carcinogenesis., 4, 393-397, 1983.

[13] M. Pike, R. Gordon, B. Herderson, H. Menck, J. SooHoo, Air Pollution in Persons at High Risk of Cancer, Fraumeni, J.F., Ed., Academic Press, N.Y. 1975, 225.

[14] A. Bjorseth, T. Ramdahl, Handbook of Polycyclic Hydrocarbon. vol. 2, Emission Sources and Recent Advances in Analytic Chemistry. Dekker, N.Y. 1985.

Chapter III STUDY DESIGN

The Phase I of THEES (1/29-2/12/87) was a microenvironmental study of indoor air, outdoor air, food, water, and soil. Phase II (1/8-1/22/88) and Phase III (9/16-9/30/88) of THEES consisted of microenvironmental surveys of indoor air, outdoor air, and food, plus personal air measurements. During Phases II and III, each adult in the home carried a personal air PM-10 monitor with the inlet continuously in their breathing zone.[1]

The development of personal monitors for project use required a significant effort on the part of UMDNJ, with the specific responsibility resting with Tim Buckley,[2] to 1) evaluate the collection characteristics of the sampling device and 2) to achieve a sampler design or sampler holder design that will reduce the problems associated with excess noise (> 70 Db). A prototype model was demonstrated to the group which had approximately a 17-18 Db reduction in noise level with only the first layer of noise attenuation material added to the aluminum sample holder. The sampler holder was very well designed and looked very unobtrusive to the group. It was received very favorably by the study subjects.

The filter holder problems were resolved by designing a teflon ring holder instead of the normal ring holder provided by Mr. Turner of Air Diagnostics, who had designed and constructed the personal samplers. Intercomparison studies of the personal sampler with the indoor sampler have been quite successful.

A. Pollutant Selection

Benzo(a)pyrene has been selected as the subject for this exposure measurement and modeling study. A major reason for selecting benzo(a)pyrene is that it is emitted during outdoor and indoor fossil fuel combustion, including coal and wood burning, smelting, and automobiles. Benzo(a)pyrene is emitted indoors from stoves and cigarettes. Benzo(a)pyrene is also found in food, especially in cooked foods, generated by smoking, curing, or broiling over a direct flame (and the pyrolysis of fats). Therefore, it was selected as a model compound to monitor in multiple media and to use for quantitatively assessing the contribution of relevant exposure pathways.

B. Field Study - Location

This study is being conducted in Phillipsburg, New Jersey. It is a municipality with a population of approximately 16,500 in a rural section of the western part of the state. The area is advantageous to study because there is a major smokestack industry in the town, a cast iron pipe manufacturing company, which has been existence since 1856. The other major local outdoor air pollution sources are space heating and motor vehicle traffic. The northern portion of the city is situated adjacent to primary truck routes from Pennsylvania to New Jersey.

REFERENCE

[1] T.J. Buckley, J.M. Waldman, P. Lioy, " High-Flow, 24-Hour Personal Sampling : Problems and Solutions," paper no. 88-115.5, in Proceedings of the 81st APCA Annual Meeting, Air Pollution Control Association, Pittsburgh, PA, 1988.

[2] P. Lioy, J.M. Waldman, T. Buckley, J. Butler, C. Pietarinen, " The Personal, Indoor and Outdoor Concentrations of PM-10 Measured in an Industrial Community During the Winter," Atmos. Environ. (in Press)

Chapter IV. METHODOLOGY

A. SAMPLING METHODOLOGY

The environments for THEES were the local atmosphere for the outdoor air samples, and the activities associated with the ten individual homes for the indoor air, personal air and food samples. A proposed control study was abandoned in Phase III because it was not clear what constituted a proper control.

Air Samplers

For the outdoor air, 24 hours PM-10 samples were collected each day from approximately 4:00 PM to 4:00 PM. This sampling interval was chosen because it coincided with the period of time when access could be obtained to each home. For sites #1, 2 these were PM-10 samplers. The sampler at Site #3 was used for TSP mass and comparisons with historical data.(since the State has long monitored this site) Simultaneously, PM-10 mass was collected in each

of the homes. The use of PM-10 samplers for the collection of mass for benzo(a)pyrene analyses provided an opportunity to examine that portion of the airborne particles most likely to be deposited in the thoracic and gas gas exchange regions of the lung. [1]

The outdoor PM-10 was collected using a Wedding Inlet Hi-volume sampler which was operated at 40 cfm with a flow controller. The collection medium was Gelman AE glass fiber filter which had been prefired by NJDEP in a muffle oven at 400^{o}C for 48 hours,weighed and stored in aluminum foil prior to use in the field.[2]

Two homes were studied for approximately five days with one to three individuals per home. This practicum included the full complement of microenvironmental samples and personal chemical or biological techniques. These have been analyzed for either B(a)P or B(a)P metabolites. (No blood samples had been taken at that time) Therefore, analysis for B(a)P-DNA adducts were not included in the practicum.

B. CHEMICAL ANALYSIS

Outdoor air samples,indoor air samples and personal air samples are received wrapped in aluminum foil from UMDNJ. These are promptly stored in a freezer at -10^{o}C until analysis.

(i) Chemicals and Apparatus

* Standard solutions of benzo(a)pyrene (Aldrich Chemical Co.) were prepared by dissolving weighed amounts in cyclohexane and storing in the freezer in amber vials.

* The extraction solvents cyclohexane, ethanol, dichloromethane and methanol are HPLC-grade and purchased from J. T. Baker Company, Inc.

* A Sonic System, Inc ultrasonic instrument was used for extraction of benzo(a)pyrene from glass fiber filters by cyclohexane at a temperature just below its boiling point.

* Precoated thin layer chromatography (TLC) plates with 250 micron thickness 20% acetylated cellulose, were purchased from Analtech Co.

* An IBM personal computer with non-linear correction program was used to calculate B(a)P concentration.

* Analytical Instrument Specialties TLC multispotter was used for TLC plate spotting and florescence detection was performed with a Perkin-Elmer MFP-44B Fluorescence Spectrophotometer.

(ii) Analytical Procedure For Air Samples

a. Outdoor air samples:

A 1 inch by 9 inch strip from the center of each high volume filter was taken and ultrasonically extracted using 10 ml of cyclohexane at 73-78^{o}C for 30 minutes. These samples were then spotted on an acetylated cellulose (TLC) plate using a 100 ul aliquot. The TLC plate was developed using 2:1 ethanol/dichloromethane and analyzed spectrofluorimetrically via the plate scanner at 387 nm

excitation wavelength and 428.6 nm emission wavelength. [3,4]

b. Indoor air samples:

The filter (diameter 3.7 cm) was cut in half following sampling and each half was ultrasonically extracted using 2 ml of cyclohexane at 73-78^{o}C for 30 minutes. The samples were then spotted on an acetylated cellulose (TLC) plate using a 250 ul aliquot, and the same procedure followed as for the outdoor air samples.[Figure IV-1]

c. Personal inhalation air samples:

The filter (diameter 2.54 cm) was ultrasonically extracted using 1 ml of cyclohexane at 73-78^{o}C for 30 minutes, and the same procedure followed as for the indoor air samples.

d. For pyrene detection, we used the same procedures for each kind of air sample except to change the fluorescence scanner to 335.5 nm excitation wavelength and 390 nm emission wavelength which are the best detection conditions for pyrene. [5], [Figure IV-2,3]

Figure IV-1 TLC Scanner Graphic for Benzo(a)pyrene Detective

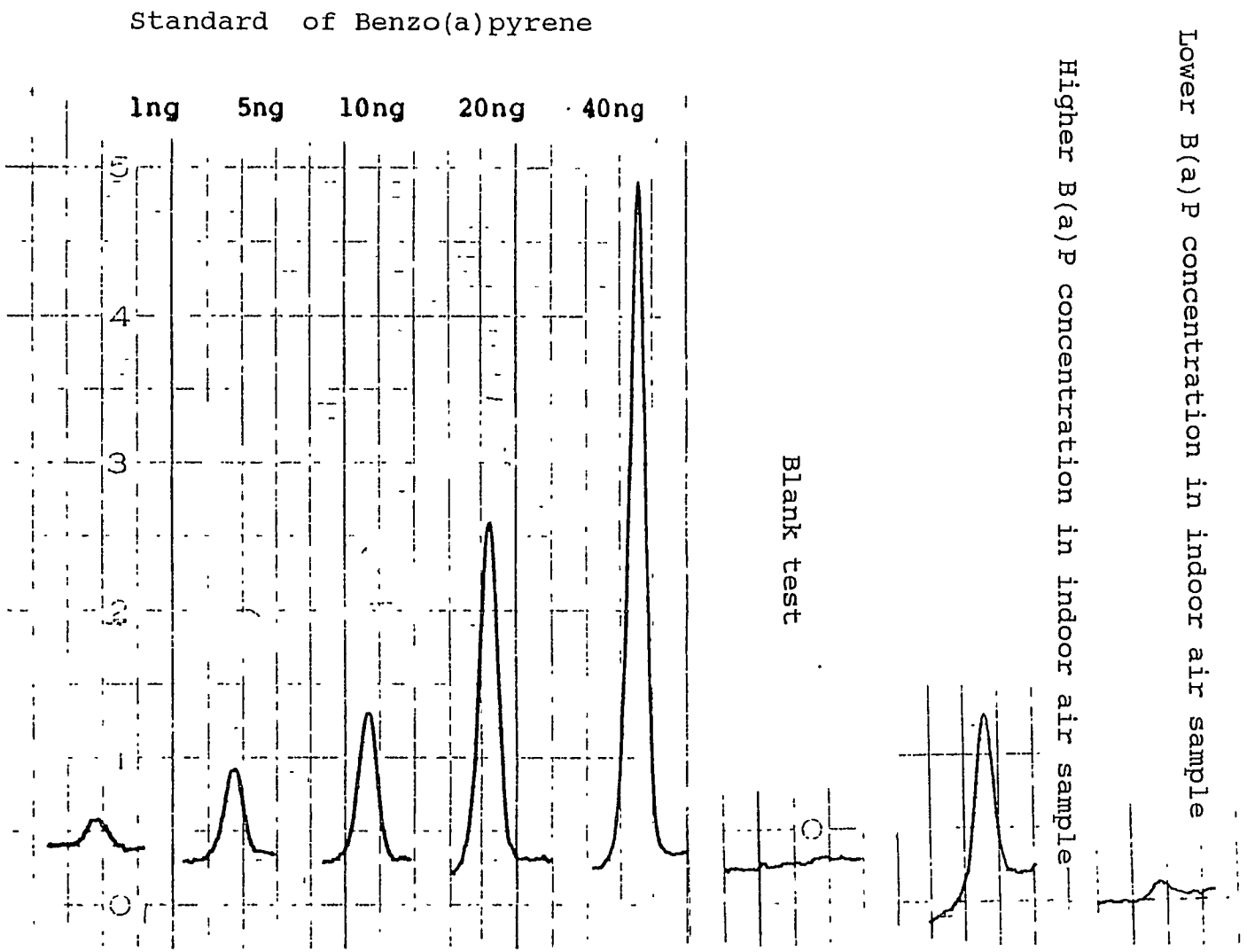


Figure IV-2

TLC Scanner Graphic for Pyrene Detective

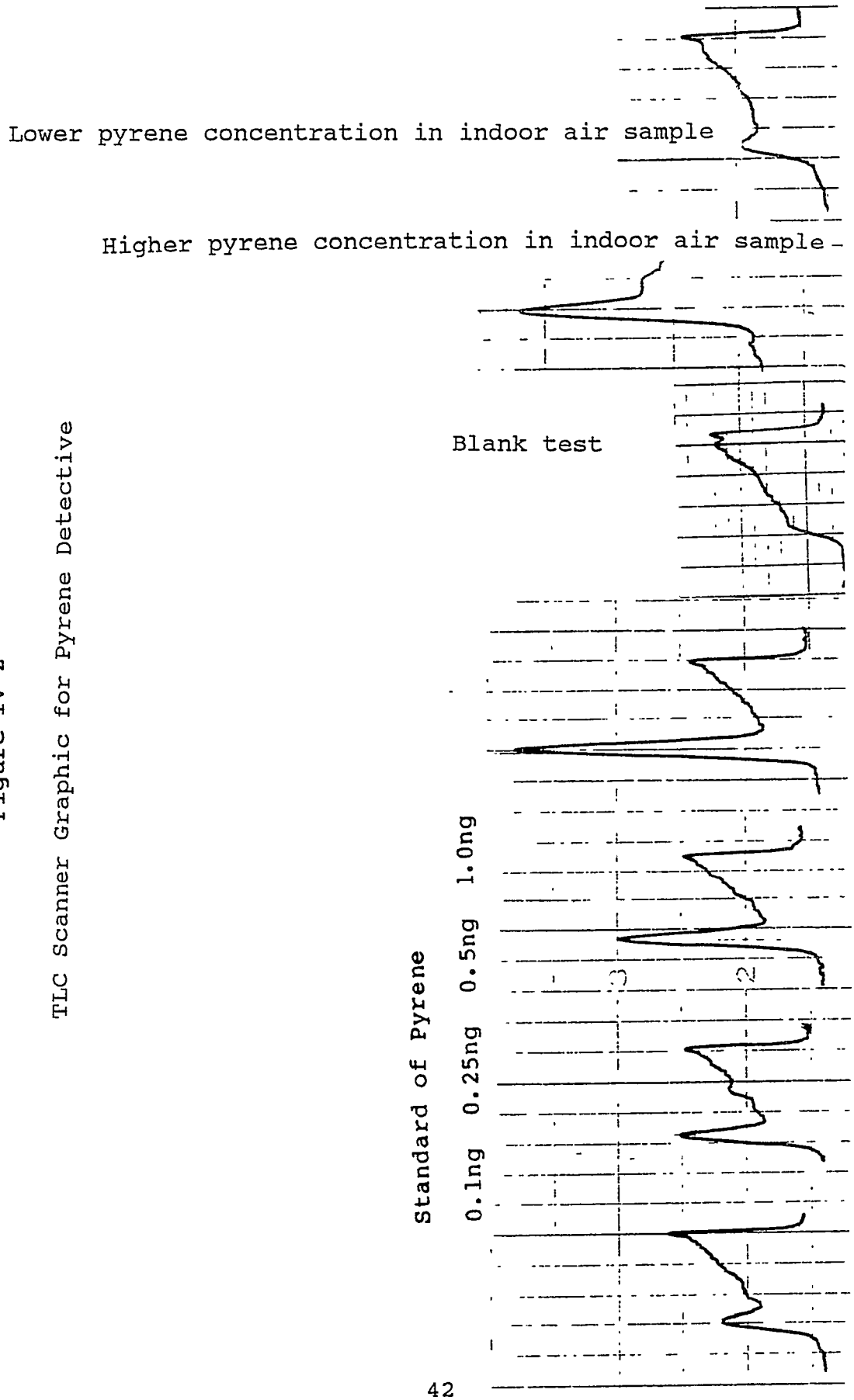
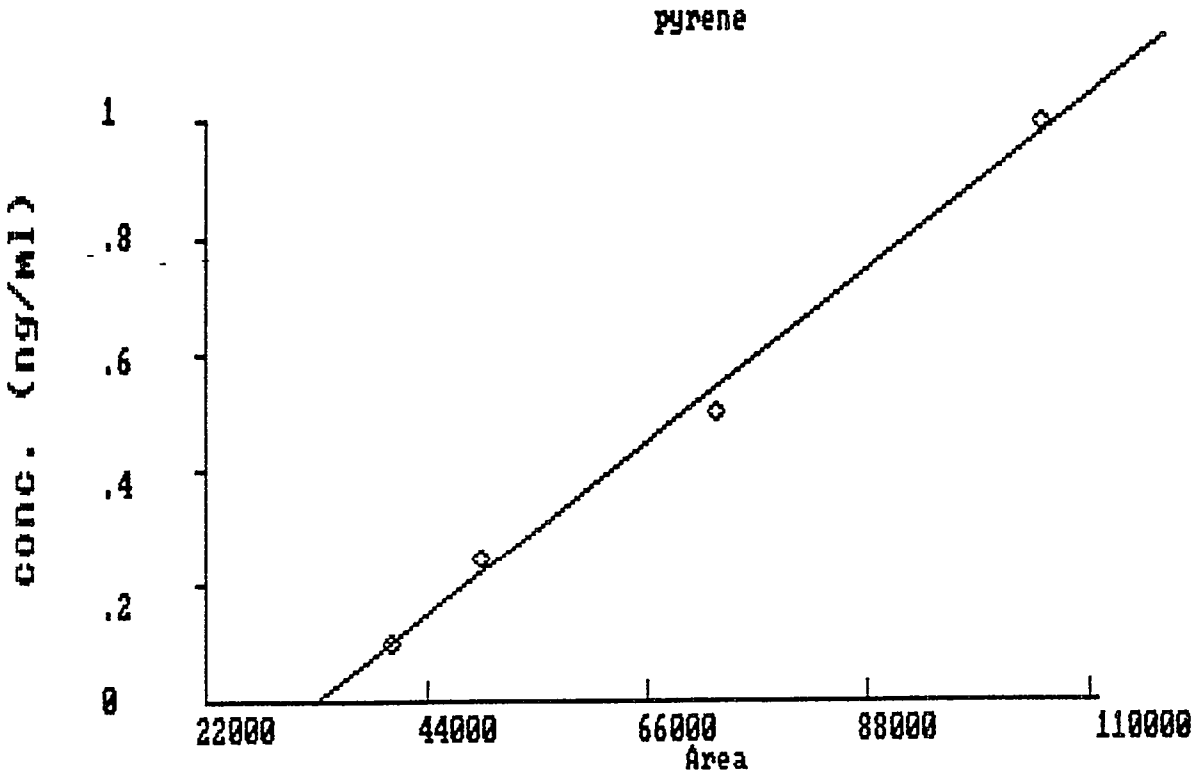


Figure IV-3

Calibration curve for Pyrene Standard



pyrene

POINT	X	Y
1	40506.5	.1
2	49360.5	.25
3	72856.5	.5
4	105136.5	1

Slope = 1.361861E-05 +/- 7.593618E-07
Intercept = -.4494702 +/- 3.798133E-02
Correlation = .9969054
Calculated on points 1 TO 4

C. QUALITY ASSURANCE/QUALITY CONTROL REGIME FOR BENZO(A)PYRENE

This QA/QC plan concerns itself with the storage,analysis and determinations of precision, accuracy and limits of detection for benzo(a)pyrene in airborne particulate samples on both low volume and high volume filters soil samples, etc. [6]

(i) Blank Runs, cleanliness of glassware, syringes

For the extraction of airborne particulate filters, HPLC-grade solvents (cyclohexane or methanol) were employed with previously unused vials. Syringes for spotting new TLC plates were washed with hot methanol and cyclohexane.

For each two-week campaign period, three blank runs were made with cyclohexane mainly for the purpose of assessing impurity buildup in the syringes as well as TLC plate and instrument responses.

(ii) Normal calibration and replication procedures

Two separate one inch strips of high volume filters were

extracted and analyzed simultaneously. Filter strips were taken from the center to minimize edge effects and the unused parts of the filter were stored in aluminum foil at -10^{o}C. For the low volume samples the entire filter were extracted. Of the 2 ml sample, two 250 ul aliquots will be analyzed simultaneously and the remaining extract stored in the dark at -10^{o}C. If the two results did not agree to within +/-25% then a third sample will be run. Calibration is done at the start of each analysis day.

(iii) Analysis of accuracy and precision of analyses

For air particulates, accuracy and precision were assessed through extraction of triplicate samples of U.S. National Bureau of Standards SRM 1649 urban particulate which had been certified at 2.9 +/-0.5 ug/g during each campaign. Our previous studies included interlaboratory comparison with the USEPA laboratory that developed the Benzo(a)pyrene TLC/Fluorescence procedure. Our results were in good agreement with theirs and our +/- 20% precision corresponded with their results.

(iv) Limit of detection and limit of Quantitation

If the blanks for benzo(a)pyrene were blank, then we used the minimun integration ares as our measure of random noise and our limit of detection was three times this value. [7] All blank filters were below the limit of detection while all run filters were above the limit of detection. For outdoor air samples, the limit of detection is 0.06 ng/m^3. Indoor air samples, 0.15 ng/m^3 as limit detection, and 0.13 ng/m^3 as personal inhalation samples' limit detection.

Daily Questionnaire

An individual in each participating household filled out a daily activity questionnaire. They recorded information about the time spent in the home, personal activities, indoor combustion source use, smoking, and ventilation. Other informations on hobbies, home repairs, and personal product use was also requested in the questionnaire. Details on the content of each meal was obtained for the food eaten at home and at other locations.

REFERENCES

[1] R. Phalen, W. Hinds, W. Jones, P. Lioy, M. Lippmann, M. McCawley, O. Raabe, S. Soderholm, and B. Stuart, " Rationale and Recommendations for Particle Size Selective Sampling in the Workplace. " Appl. Ind. Hyg., 1, 3-14, 1986.

[2] J. Elston, Personal Communication, Bureau of Air Pollution Control, New Jersey, DEP. 1987.

[3] R. Harkov, A. Greenberg, Benzo(a)pyrene in New Jersey - Results from a twenty - seven site study, JAPCA, 35, 238-243, 1985.

[4] R. Faoro, A. Manning, Trends in Benzo(a)pyrene JAPCA, 32, 62-64, 1966-77.

[5] W. Karcher, R.J. Fordham, J.J. Dubois, P.G.J.M. Glaude, J.A.M. Ligthart, SPECTRAL ATLAS OF POLYCYCLIC AROMATIC COMPOUNDS, D Reidel Pub. 1985.

[6] A. Greenberg, " Quality Accurance / Quality Control Regime for Phillipsburg, NJIT, THEES STUDY 1986.

[7] D. MacDougall, Guidelines for Data Acquisition and Data Quality Evaluation in Environmental Chemistry. Anal. Chem. 52, 2242-2249, 1980.

Chapter V. EXPERIMENTAL RESULTS AND DISCUSSION

RESULTS

INDOOR AIR - PERSONAL AND OTHER SOURCES ACTIVITIES

A major focus of the THEES project is linking personal as well as indoor and outdoor environmental activities to the variation of benzo(a)pyrene found in the home. The mean numbers of hours spent at home for all individuals participating in the study was 16 hours (67%) [1] for the day. This included the time spent in the house by children, and adults. [Table V-1] lists the common combustion sources present in each home.

Each of the participating homes had activities during the two week study which affected the indoor B(a)P levels found in the PM-10 samples. These were related either to an indoor source, or the penetration of outdoor air.

* All the outdoor air samples data Phase II & Phase III site 101-104 are listed in Appendix Table V 1-6

* Indoor air samples data Phase II & Phase III site 10-90

Table V-1

Common Indoor Combustion Sources located in each THEES Home

Home #	Types of Sources
1	Gas Cooking Range and Oven Toaster Oil Central Heat
2	Oil Central Heat Electric Range & Oven Microwave Oven 1 Smoker in residence
3	Oil Central Heat Kerosene heater (in living room or kitchen) Electric Range & Oven 2 Smokers in residence
4	Oil Central Heat Kerosene heater (in dining room or hall) Electric heater Gas range & Oven Microwave Oven
5	Electric Range & Oven Microwave Oven Oil Central Heat
6	Gas Central Heat Kerosene Space Heater (in attic) Electric Range & Oven Toaster
7	Gas Central Heat Electric Range & Oven Microwave
8	Oil Central Heat Gas Range & Oven Toaster Oven Microwave Oven
9	Oil Central Heat Gas Range & Oven 1 Smoker in residence
10	Oil Central Heat (rarely used) Coal Space Heat (primarily & in living room) Kerosene Heater (in kitchen) Gas Range & Oven Microwave Oven

are listed Appendix Table VI 1-8, Table VII 1-10

* Personal inhalation air samples data Phase II site 10 - 90 are listed Appendix Table VIII 1-8

* Compare indoor air B(a)P and Pyrene data are listed Appendix Table IX-1 and Appendix Figure 1-4

* Compare personal inhalation air B(a)P and Pyrene data are listed Appendix Table X-1 and Appendix Figure 5-6

DISCUSSION

[Figure I-1] depicts the fact that two of the outdoor sites for sampling benzo(a)pyrene were situated within a quarter mile of the foundry. One was located to the east (site 102) and the other to the west (site 101). Outdoor site 103 was located in Walters Park, which is situated just above the plant. This was selected primarily for historical perspective since it has been used to collect TSP data for a number of years.[2]

From experimental data, the highest B(a)P concentration is site 103 which is the nearest to foundry. This results

supports the view that the foundry is a source of B(a)P in local outdoor environments

There are eight indoor sites near the foundry. The largest variations appear to be due to activities as smoking or painting in the house. Home 1 is the site right in front of the foundry with the closest distance. The B(a)P concentration comparison between home 1 and outdoor site 101 seem to have the same trend but when we examine the correlation between them it does not show a signifcant relationship. This is probably due to personal activity which affected the penetration.

For site 30, there are peaks of B(a)P concentration at relatively high levels. The main reason appears to be a coal heating unit in the living room. Buring of coal appears to be a predominant source in this microenvironment. Site 0 shows the lowest concentration among the eight residences. The average B(a)P concentrations of houses with smokers and without smoker are 1.5 ng/m^3 and 1.1 $ng/^3$ respectively. This means that smoking is an important B(a)P source in indoor environments.

In comparing the indoor B(a)P levels, we can find indoor B(a)P concentrations appeared to be affected by outdoor B(a)P concentrations through penetration. If it is assumed that on all days when the B(a)P ratio of outdoor air to indoor air is less than 1 there are non indoor source concentrations, then a household penetration factor can be estimated for these homes. This factor could include natural draft penetration and the effects of opening doors and windows.

There are some proportional relationships between the indoor air samples concentrations and personal inhalation air samples at the same site. These data show that personal inhalation air is directly related to indoor air quality. [Fig V-1]

During Phase II, in contrast, the 24-hours personal air sampling data reflect all microenvironments, to which the participant was exposed, and does not rely in estimates of time spent in each microenvironment or on estimates of B(a)P levels in microenvironments that were not monitored. The exposure values derived from these data, therefore, would be expected to provide a more accurate reflection of the

particpant's actual exposure. [Fig V-2]

A significant benefit of this research is that by investigating all routes of exposure, the most important exposure pathways resulting in potential health impacts will be indentified. These findings will demonstrate where to concentrate efforts in exposure reduction for the carcinogen B(a)P. Personal activities, lifestyle,and diet can strongly influence individual exposure to B(a)P.

EXPOSURE TO B(A)P THROUGH INDOOR AIR

In order to have an idea of human doses of B(a)P through different pathways and the percentage contribution of each pathway to the total dose, we take the indoor samples concentrations as a measure of contribution to total human exposure. These results only provide a first level-approximation of human exposure and a relatively simplistic calculation for quantitative risk assessment.

Figure V-1

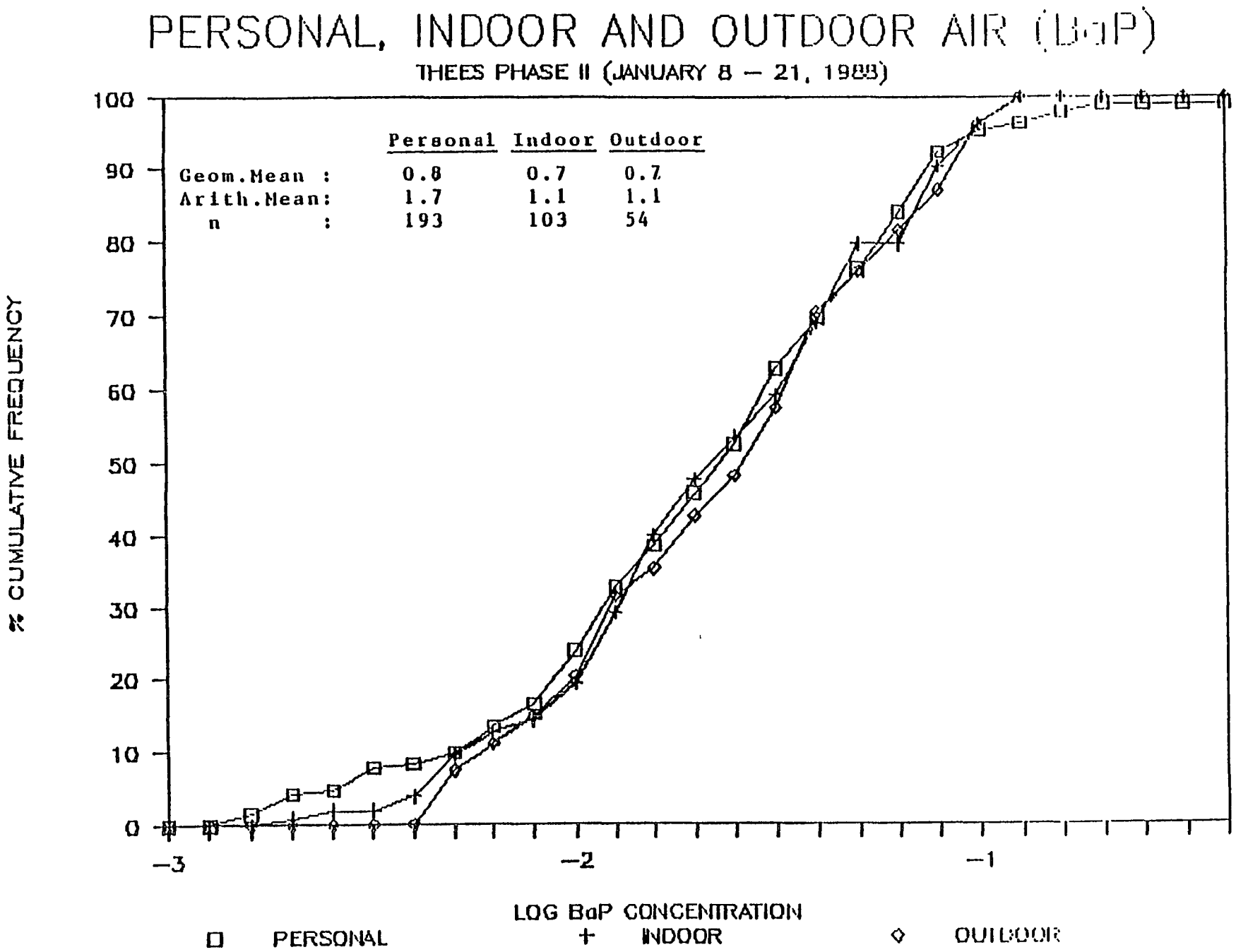
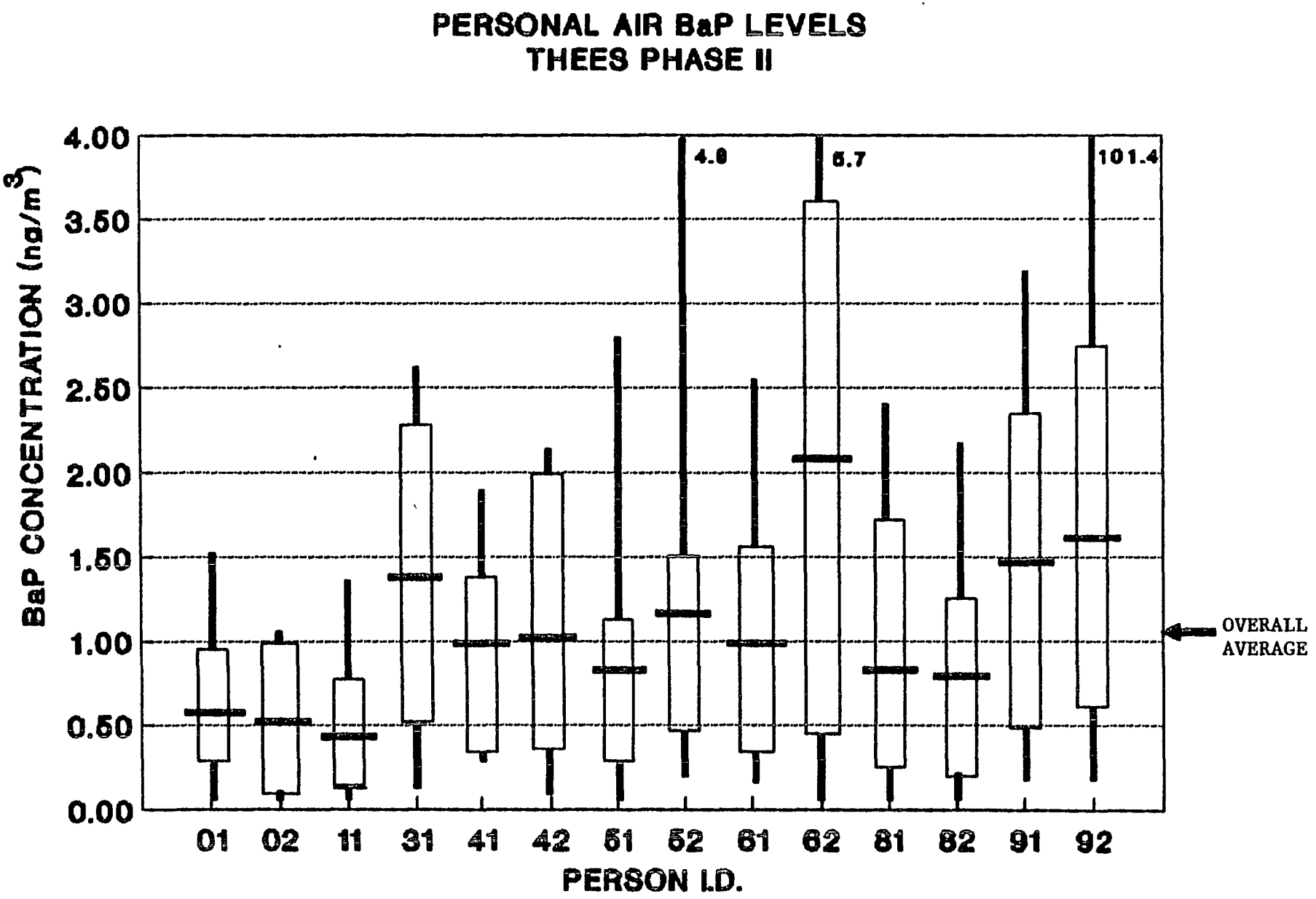

PERSONAL, INDOOR AND OUTDOOR AIR (BaP)
THEES PHASE II (JANUARY 8 – 21, 1988)
Personal Indoor Outdoor
Geom.Mean : 0.8 0.7 0.7
Arith.Mean: 1.7 1.1 1.1
n : 193 103 54
% CUMULATIVE FREQUENCY
100
90
80
70
60
50
40
30
20
10
0
−3
−2
−1
LOG BaP CONCENTRATION
PERSONAL
INDOOR
OUTDOOR

Figure V-2


PERSONAL AIR BaP LEVELS
THEES PHASE II
BaP CONCENTRATION (ng/m3)
4.00
3.50
3.00
2.50
2.00
1.50
1.00
0.50
0.00
4.8
6.7
101.4
OVERALL AVERAGE
01
02
11
31
41
42
51
52
61
62
81
82
91
92
PERSON I.D.

REFERENCE

[1] P. Walsh, C. Dudney, E. Copenhaver, " Indoor Air Quality " CRC Press. Inc., Fourth Printing, 1986.

[2] J. M. Waldman, J. P. Lioy, R. Harkov, A. Greenburg Field Study of Human Exposure to Benzo(a)pyrene in a Community Directly Impacted by a Foundry, Preprint for 4th International Indoor Air Conference Berlin, GR. August, 1987.

Chapter VI. CONCLUSION

Generalization of the first phase THEES results will require the continued analysis of data from the remaining phases of the study. Important questions to be addressed will be the times when grills are used for cooking food, and the times personal inhalation exposures will be affected more by the Benzo(a)pyrene in the local outdoor environment. Approximately 25-50% of individual average weekly B(a)P inhalation exposures were greater than dietary exposures in Phase II .

The levels observed for each medium were within those ranges previously established in separate studies; although, an advantage of the present study has been the simultaneous sampling of Benzo(a)pyrene in each medium. The infiltration of outdoor air to the indoors was the major source of indoor exposures in some cases.

Although outdoors, indoors and personal inhalations pollution samples measurements data have been presented in a large number of references, examination of Appendix Table V - Table X reveals that the amount of reliable and readily comparable data must still be considered highly limited.

Thus, the conclusions resulting from this review must be regarded as merely tentative.

Limit of detection was discussed in chapter IV. It was found that all blank filters were below the limit of detection while all run filters were above the limit of detection.

Indoor air quality is a major determinant of personal exposure to pollutants in the environment. People spend much of their time in numerous indoor environments. Appropriate research has shown that the typical person in the United State spends 70% of their time in their homes. [1]

Sources of indoor air pollution include influx of polluted outdoor air, geologic materials around the building, cooking, cleaning, smoking, wood combustion for heat, hobbies, etc. Indoor air quality is determined by a complex set of interacting parameters and can be viewed at different levels of detail. One of these levels is specific analysis of sources levels and likely health effects.

The population for this exposure study was not meant to be a statistically selected subgroup or representative sample of the types of people living in the Phillipsburg area.

Under normal circumstances, [2] the best available estimate for indoor concentrations of particulates can be obtained by presuming them equal to outdoor concentrations. It is possible that indoor concentrations of these pollutants are lower than outdoor levels when outdoor concentrations are high, but the available data do not definitely establish this relationship.

Indoor concentrations and outdoor concentrations and indoor/outdoor ratios have been found to vary on seasonal bases. According the experimental data, we can see that B(a)P concentration is higher in winter than in summer. The one of many reasons is that many families use the woodburning combustion for heating in winter.

However, it is not easy to measure the concentrations present in potential air microenvironments other than the home and ambient air. To estimate daily inhalation exposure, one needs to take account of the hours not spent at home. By using ambient B(a)P as the minimum concentration which a person encounters, the remaining B(a)P exposure can be estimated.

With these assumptions, comparison of the two weeks of data for 24 hour inhalation exposure each day and food exposure was made. The paired B(a)P exposures for each home and each pathway indicated that the potential intake could be similar in each medium.

The aerosol particle size we collected is larger than 10 um, but from human health point of view, the most damaging particle sizes are smaller than 0.1 um [3,4]. These particles penetrate the nasopharyngeal and bronchiolar regions and deposit in the alveolar region. This maybe one of the reasons bioavailable concentrations are different from the concentration we detected in the air. And it also can explain why we just can detect a small volume of Pyrene in indoor air concentration. It is because 56% of the pyrene exists in the vapor phase and can not be collected on the filter.

There had done in two homes in the Piscataway area and mimic the procedures that are used in the Phase II study. Logistics in the Phase II study design is a bit more cumbersome than Phase I study design.

This report is thus only a desiging in total human

exposure studies. We have tried to measure environmental concentrations of pollutants available through different pathways in order to characterize the role of each pathway also to develop the methodology of total human exposure study. Further study will be done to look at the pollutant concentration in blood and in urine. This study can give a picture of human biological dose which is more directly related to human exposure.

REFERENCE

[1] P. Walsh, C. Dudney, E. Copenhaver, " Indoor Air Quality " CRC Press. Inc. Fourth Printing, 1986.

[2] F. Benson, J. Henderson, D. Caldwell, " Indoor-Outdoor Air Pollution Relationships " A Literature Review EPA Pub. No. AP-112

[3] F. Sperizer, " Overview of the Risk of Respiratory Cancer from Airborne Contaminations." Envirnomental Health Perpectives vol. 70. pp.9-15, 1986.

[4] G. Matanoski, L. Fishbein, C. Redmond, " Contribution of Organic Particulates to Respiratory Cancer. " Environmental Health Perpectives vol. 70. pp. 37-49, 1986.

Appendix A: Table V-1

B(a)P OUTDOOR AIR SAMPLE ANALYSIS

PHASE II

Site 101

Sample No.	Ng B(a)P/Filter	Average
101-1	602.82	579.33
101-1	555.84	
101-2	1863.13	1863.08
101-2	1863.02	
101-3	4998.71	5182.02
101-3	5365.32	
101-4	3350.27	3523.15
101-4	3696.02	
101-5	2365.99	2390.36
101-5	2414.73	
101-6	265.38	263.17
101-6	260.96	
101-7	1691.29	1696.96
101-7	1702.63	
101-8	3040.93	3036.27
101-8	3031.64	

(continue) Table V-1

101-9	1332.83	1362.16
101-9	1391.49	
101-10	1228.06	1218.0
101-10	1207.94	
101-11	470.65	468.31
101-11	466.97	
101-12	913.12	910.51
101-12	907.9	
101-13	645.6	651.45
101-13	657.3	
101-14	220.2	225.90
101-14	231.59	
101-15	ND	
101-15	ND	
101-16	ND	
101-16	ND	

Appendix A: Table V-2

B(a)P OUTDOOR AIR SAMPLE ANALYSIS

PHASE II

Site 102

Sample No.	Ng B(a)P /Filter	Average
102-1	296.06	299.61
102-1	303.16	
102-2	518.34	529.20
102-2	540.05	
102-3	204.89	205.89
102-3	206.89	
102-4	4856.75	4852.58
102-4	4848.4	
102-5	3086.99	3071.03
102-5	3055.07	
102-6	307.36	304.04
102-6	300.72	
102-7	1862.24	1860.54
102-7	1858.83	
102-8	3761.16	3720.95
102-8	3680.73	

(continue) Table V-2

102-9	1849.37	1850.87
102-9	1852.36	
102-10	1745.55	1736.54
102-10	1727.61	
102-11	478.43	486.85
102-11	495.27	
102-12	1432.41	1410.46
102-12	1388.5	
102-13	904.99	903.24
102-13	901.49	
102-14	446.02	446.20
102-14	446.37	
102-15	ND	
102-15	ND	
102-16	ND	
102-16	ND	

Appendix A: Table V-3

B(a)P OUTDOOR AIR SAMPLE ANALYSIS

PHASE II

Site 103

Sample No.	Ng B(a)P / Filter	Average
103-1	567.37	548.37
103-1	529.36	
103-2	1679.8	1734.71
103-2	1789.61	
103-3	4249.27	4368.30
103-3	4487.32	
103-4	5654.71	5673.86
103-4	5693.02	
103-5	2404.98	2401.53
103-5	2398.08	
103-6	217.75	212.42
103-6	207.78	
103-7	1468.13	1478.32
103-7	1488.54	

(continue) Table V-3

103-8	5190.64	5278.93
103-8	5357.22	
103-9	1280.1	1265.46
103-9	1250.81	
103-10	1008.15	1016.94
103-10	1025.72	
103-11	389.77	389.53
103-11	389.29	
103-12	1112.84	1106.78
103-12	1100.72	
103-13	863.89	870.62
103-13	877.34	
103-14	212.69	208.63
103-14	204.56	
103-15	ND	
103-15	ND	
103-16	ND	
103-16	ND	

Appendix A: Table V-4

B(a)P OUTDOOR AIR SAMPLE ANALYSIS

PHASE III

Site 101

Sample No.	Ng B(a)P / Filter	Average
101-01	83.32	78.07
101-01	72.82	
101-02	84.81	83.84
101-02	82.87	
101-03	ND	
101-03	ND	
101-04	63.72	61.39
101-04	59.06	
101-05	81.96	81.52
101-05	81.07	
101-06	76.52	74.11
101-06	71.69	
101-07	250.71	244.33
101-07	237.95	
101-08	52.41	51.74
101-08	51.06	

(continue) Table V-4

101-09	394.13	
		400.93
101-09	407.72	
101-10	267.07	
		292.35
101-10	317.63	
101-11	265.52	
		254.32
101-11	243.11	
101-12	581.55	
		608.75
101-12	635.95	
101-13	195.39	
		190.47
101-13	185.55	
101-14	293.76	
		321.45
101-14	349.14	

Appendix A: Table V-5

B(a)P OUTDOOR AIR SAMPLE ANALYSIS

PHASE III

Site 102

SAMPLE NO.	Ng B(a)P /Filter	Average
102-01	111.07	110.87
102-01	110.67	
102-02	143	152.26
102-02	161.51	
102-03	243.39	246.35
102-03	249.3	
102-04	107.14	106.64
102-04	106.14	
102-05	256.63	258.74
102-05	260.85	
102-06	271.17	266.87
102-06	262.56	
102-07	745.95	762.43
102-07	778.91	

(continue) Table V-5

102-08	92.84	
		97.42
102-08	102	
102-09	477.74	
		499.29
102-09	520.83	
102-10	156.54	
		158.38
102-10	160.22	
102-11	265.94	
		274.68
102-11	283.42	
102-12	600.03	
		546.25
102-12	492.46	
102-13	123.77	
		123.18
102-13	122.59	
102-14	109	
		113.08
102-14	117.15	

Appendix A: Table V-6

B(a)P OUTDOOR AIR SAMPLE ANALYSIS

PHASE III

Site 103

Sample no.	ng B(a)P /Filter	Average
103-01	89.48	89.74
103-01	90.0	
103-02	95.55	93.25
103-02	90.94	
103-03	180.89	177.62
103-03	174.34	
103-04	69.02	68.57
103-04	68.12	
103-05	67.01	66.38
103-05	65.74	
103-06	46.97	49.7
103-06	52.43	
103-07	311.37	300.48
103-07	289.58	

(continue) Table V-6

103-08	43.63	44.27
103-08	44.9	
103-09	439.1	440.7
103-09	442.3	
103-10	210.12	217.76
103-10	225.41	
103-11	168.68	157.8
103-11	146.92	
103-12	690.61	727.2
103-12	763.79	
103-13	70.75	70.82
103-13	70.88	
103-14	208.13	207.63
103-14	207.12	

Appendix A: Table VI-1

B(a)P INDOOR AIR SAMPLES ANALYSIS

PHASE II

Site 10

Sample No.	ng B(a)P /filter	Average
001	2.35	2.30
001	2.25	
002	8.93	9.05
002	9.17	
003	22.42	22.22
003	22.02	
004	21.58	21.83
004	22.07	
005	12.65	12.58
005	12.51	
006	3.26	3.28
006	3.29	
007	9.15	8.94
007	8.73	
008	32.9	32.93
008	32.95	

(continue) Table VI-1

009	10.38	10.57
009	10.76	
010	5.95	6.14
010	6.33	
011	0.95	0.99
011	1.03	
012	5.32	5.39
012	5.46	
013	4.58	4.68
013	4.77	
014	ND	
014	ND	
015	0.76	0.75
015	0.74	
016	ND	
016	ND	

Appendix A: Table VI-2

B(a)P INDOOR AIR SAMPLES ANALYSIS

PHASE II

Site 20

Sample No.	Ng B(a)P/Filter	Average
017	4.56	4.95
017	5.33	
018	11.49	11.72
018	11.94	
019	37.63	37.98
019	38.32	
020	40.03	42.40
020	44.77	
021	25.86	27.06
021	28.25	
022	3.14	3.17
022	3.2	
023	20.59	20.06
023	19.53	
024	39.68	39.34
024	38.99	

(continue) Table VI-2

025	13.19	13.21
025	13.23	
026	14.23	14.50
026	14.77	
027	5.44	5.32
027	5.2	
028	6.32	6.34
028	6.35	
029	6.15	6.22
029	6.29	
030	4.54	4.52
030	4.5	
031	ND	
031	ND	
032	ND	
032	ND	

Appendix A: Table VI-3

B(a)P INDOOR AIR SAMPLES ANALYSIS RESULT

PHASE II

Site 30

Sample No.	Ng B(a)P / Filter	Average
033	5.94	5.86
033	5.77	
033	5.79	5.85
033	5.91	
034	17.21	17.72
034	18.22	
034	19.45	19.53
034	19.61	
035	50.93	51.45
035	51.97	
035	48.50	47.21
035	45.91	
036	44.63	44.59
036	44.55	
036	47.06	47.22
036	47.37	

(continue) Table VI-3

037	24.90	25.26
037	25.61	
037	26.41	26.68
037	26.95	
038	7.87	7.72
038	7.57	
038	7.68	7.55
038	7.41	
039	19.46	19.02
039	18.57	
039	18.45	18.34
039	18.22	
040	46.56	47.58
040	48.59	
040	46.99	46.26
040	45.52	
041	20.95	20.96
041	20.96	
041	20.26	20.13
041	20.0	
042	32.87	32.82
042	32.77	
042	34.48	34.59
042	34.79	

(continue) Table VI-3

043	16.0	15.86
043	15.72	
043	15.60	15.80
043	15.99	
044	19.82	19.89
044	19.95	
044	20.25	20.07
044	19.88	
045	9.19	9.25
045	9.31	
045	10.45	10.48
045	10.50	
046	5.12	5.11
046	5.10	
046	4.82	4.81
046	4.79	
047	ND	
047	ND	
047	ND	
047	ND	
048	ND	
048	ND	
048	ND	
048	ND	

Appendix A: Table VI-4

B(a)P INDOOR AIR SAMPLES ANALYSIS

PHASE II

Site 40

SAMPLE No.	NG B(a)P/FILTER	AVERAGE
049	7.13	6.57
049	6.01	
049	5.84	5.76
049	5.68	
050	20.65	20.93
050	21.20	
050	21.76	22.01
050	22.25	
051	42.78	43.77
051	44.76	
051	46.52	46.64
051	46.75	
052	39.60	38.17
052	36.73	
052	36.95	36.54
052	36.12	

(continue) Table VI-4

053	11.63	11.86
053	12.08	
053	12.51	12.59
053	12.66	
054	1.77	1.87
054	1.96	
054	1.64	1.70
054	1.75	
055	10.48	10.30
055	10.11	
055	9.83	9.69
055	9.55	
056	1.80	1.80
056	1.79	
056	1.58	1.63
056	1.68	
057	0.18	----
057	ND	
057	0.12	----
057	ND	
058	12.23	12.57
058	12.90	
058	14.66	14.59
058	14.52	

(continue) Table VI-4

059	1.60	1.53
059	1.45	
059	1.28	1.36
059	1.43	
060	0.90	0.90
060	0.89	
060	0.92	0.91
060	0.91	
061	24.0	23.7
061	23.40	
061	23.15	22.78
061	22.4	
062	ND	
062	ND	
062	ND	
062	ND	
063	ND	
063	ND	
063	ND	
063	ND	
064	ND	
064	ND	
064	ND	
064	ND	

Appendix A: Table VI-5

B(a)P INDOOR AIR SAMPLES ANALYSIS

PHASE II

Site 50

Sample No.	Ng B(a)P / Filter	Average
065	5.15	5.08
065	5.01	
065	5.09	5.14
065	5.19	
066	14.90	14.99
066	15.08	
066	15.88	15.80
066	15.71	
067	33.88	34.39
067	34.90	
067	37.50	37.30
067	37.09	
068	37.61	37.57
068	37.53	
068	37.80	37.98
068	38.15	

(continue) Table VI-5

069	17.79	17.61
069	17.43	
069	18.23	18.18
069	18.12	
070	2.13	2.12
070	2.10	
070	2.08	2.10
070	2.11	
071	11.86	12.0
071	12.13	
071	12.01	12.18
071	12.35	
072	32.07	32.13
072	32.18	
072	31.84	31.83
072	31.82	
073	8.29	8.36
073	8.43	
073	8.10	8.01
073	7.92	
074	ND	
074	ND	
074	ND	
074	ND	

(continue) Table VI-5

075	4.74	4.74
075	4.73	
075	4.81	4.79
075	4.77	
076	7.68	7.52
076	7.35	
076	7.67	7.54
076	7.41	
077	6.55	6.54
077	6.53	
077	6.26	6.49
077	6.72	
078	2.62	2.61
078	2.59	
078	2.57	2.58
078	2.59	
079	ND	
079	ND	
079	ND	
079	ND	
080	ND	
080	ND	
080	ND	
080	ND	

Appendix A: Table VI-6

B(a)P INDOOR AIR SAMPLES ANALYSIS

PHASE II

Site 60

Sample No.	Ng B(a)P /Filter	Average
081	5.07	5.07
081	5.06	
081	5.03	5.07
081	5.10	
082	11.0	11.02
082	11.04	
082	11.06	11.06
082	11.05	
083	37.10	36.46
083	35.81	
083	35.96	36.05
083	36.14	
084	38.04	38.03
084	38.01	
084	37.68	37.47
084	37.25	

(continue) Table VI-6

085	17.40	17.25
085	17.09	
085	16.27	16.24
085	16.21	
086	1.99	2.00
086	2.00	
086	1.93	1.97
086	2.00	
087	16.52	16.72
087	16.92	
087	14.92	15.29
087	15.65	
088	33.18	32.95
088	32.71	
088	33.01	33.42
088	33.83	
089	8.69	8.74
089	8.78	
089	9.03	9.05
089	9.06	
090	6.75	6.60
090	6.34	
090	7.09	7.11
090	7.22	

(continue) Table VI-6

091	8.99	8.99
091	8.98	
091	8.59	8.67
091	8.75	
092	ND	
092	ND	
092	ND	
092	ND	
093	3.41	3.48
093	3.55	
093	3.86	3.91
093	3.95	
094	7.30	6.88
094	6.46	
094	6.53	6.42
094	6.31	
095	1.54	1.51
095	1.48	
095	1.51	1.51
095	1.50	
096	ND	
096	ND	
096	ND	
096	ND	

Appendix A: Table VI-7

B(a)P INDOOR AIR SAMPLES ANALYSIS

PHASE II

Site 80

Sample No.	ng B(a)P/Filter	Average
097	2.47	2.45
097	2.43	
097	2.48	2.47
097	2.45	
098	18.69	18.52
098	18.34	
098	18.90	18.71
098	18.52	
099	33.28	33.09
099	32.90	
099	32.95	33.06
099	33.16	
100	2.94	2.91
100	2.88	
100	2.76	2.72
100	2.68	

(continue) Table VI-7

101	19.01	18.85
101	18.68	
101	19.18	18.84
101	18.49	
102	2.21	2.22
102	2.23	
102	2.27	2.27
102	2.27	
103	10.99	11.0
103	11.00	
103	10.49	10.88
103	11.27	
104	8.52	8.22
104	7.92	
104	8.33	8.54
104	8.74	
105	5.52	5.66
105	5.79	
105	5.52	5.66
105	5.78	
106	6.29	6.30
106	6.31	
106	6.13	6.24
106	6.34	

(continue) Table VI-7

107	5.40	5.30
107	5.19	
107	5.58	5.58
107	5.57	
108	6.49	6.71
108	6.93	
108	7.33	7.17
108	7.04	
109	6.85	7.20
109	7.55	
109	6.91	6.97
109	7.03	
110	2.91	2.92
110	2.93	
110	2.97	2.90
110	2.82	
111	ND	
111	ND	
111	ND	
111	ND	
112	ND	
112	ND	
112	ND	
112	ND	

Appendix A: Table VI-8

B(a)P INDOOR AIR SAMPLES ANALYSIS

PHASE II

Site 0

Sample No.	ng B(a)P/Filter	Average
129	1.83	1.80
129	1.76	
130	6.16	6.34
130	6.51	
131	11.35	11.45
131	11.54	
132	15.65	15.49
132	15.32	
133	4.43	4.27
133	4.10	
134	1.98	1.95
134	1.91	
135	6.04	6.06
135	6.08	

(continue) Table VI-8

136	8.08	8.00
136	7.91	
137	6.01	6.25
137	6.48	
138	5.55	5.40
138	5.25	
139	4.94	4.82
139	4.7	
140	3.7	3.69
140	3.67	
141	1.83	1.92
141	1.99	
142	ND	
142	ND	
143	11.52	11.76
143	12.02	
144	ND	
144	ND	

Appendix A: Table VII-1

B(a)P INDOOR AIR SAMPLES ANALYSIS

PHASE III

Sample No.	Ng B(a)P /Filter	Average
2001	ND	
2001	ND	
2004	0.62	0.62
2004	0.62	
2005	3.09	3.12
2005	3.15	
2008	2.33	2.46
2008	2.58	
2041	0.48	0.49
2041	0.5	
2042	5.57	5.66
2042	5.74	
2049	2.1	2.21
2049	2.32	

(continue) Table VII-1

2050	0.24	0.33
2050	0.41	
2052	1.1	1.12
2052	1.14	
2053	3.64	3.66
2053	3.67	
2055	2.16	2.14
2055	2.11	
2057	2.2	2.20
2057	2.19	
2059	3.44	3.37
2059	3.3	
2063	5.97	5.99
2063	6.0	
2067	0.67	0.69
2067	0.71	
2091	1.97	1.99
2091	2.0	
2093	1.91	1.96
2093	2.0	

Appendix A: Table VII-2

B(a)P INDOOR AIR SAMPLES ANALYSIS

PHASE III

SAMPLE NO.	Ng B(a)P /Filter	Average
2002	0.49	0.56
2002	0.62	
2051	0.41	0.42
2051	0.43	
2065	0.74	0.74
2065	0.73	
2066	0.67	0.66
2066	0.64	
2068	3.94	3.96
2068	3.97	
2069	1.02	1.11
2069	1.19	
2092	1.02	1.13
2092	1.23	
2095	0.36	0.36
2095	0.36	

Appendix A: Table VII-3

B(a)P INDOOR AIR SAMPLES ANALYSIS

PHASE III

Sample No.	ng B(a)P /filter	Average
2003	0.97	0.99
2003	1.0	
2014	1.99	1.94
2014	1.89	
2018	1.79	1.79
2018	1.79	
2033	2.77	2.86
2033	2.95	
2045	1.93	1.93
2045	1.92	
2070	0.69	0.72
2070	0.75	
2094	1.51	1.54
2094	1.56	
2108	1.32	1.33
2108	1.33	

(continue) Table VII-3

2123	2.93	3.02
2123	3.11	
2132	1.58	1.40
2132	1.21	
2140	1.42	1.47
2140	1.51	
2143	1.99	1.98
2143	1.97	
2145	0.62	0.61
2145	0.6	
2147	1.6	1.58
2147	1.56	
2148	2.0	1.96
2148	1.92	
2171	1.19	1.18
2171	1.16	

Appendix A: Table VII-4

B(a)P INDOOR AIR SAMPLES ANALYSIS

PHASE III

SAMPLE NO.	ng B(a)P /Filter	Average
2006	3.74	3.92
2006	4.1	
2062	0.78	0.77
2062	0.75	
2078	0.32	0.31
2078	0.29	
2102	0.54	0.54
2102	0.53	
2106	1.05	1.08
2106	1.11	
2141	0.94	0.99
2141	1.04	
2144	0.91	0.90
2144	0.89	
2161	3.75	3.68
2161	3.61	
2163	1.71	1.71
2163	1.70	
2173	2.33	2.34
2173	2.34	

Appendix A: Table VII-5

B(a)P INDOOR AIR SAMPLES ANALYSIS

PHASE III

SAMPLE No.	Ng B(a)P /Filter	Average
2007	2.87	2.64
2007	2.4	
2043	0.54	0.54
2043	0.53	
2046	0.49	0.48
2046	0.46	
2060	4.01	4.15
2060	4.29	
2064	1.69	1.68
2064	1.67	
2073	5.84	5.83
2073	5.81	
2076	2.66	2.57
2076	2.47	
2105	0.53	0.52
2105	0.51	

(continue) Table VII-5

2162	0.30	0.25
2162	0.19	
2172	0.83	0.79
2172	0.75	
2176	3.99	4.05
2176	4.10	
2178	0.60	0.61
2178	0.62	

Appendix A: Table VII-6

B(a)P INDOOR AIR SAMPLES ANALYSIS

PHASE III

Sample No.	Ng B(a)P /filter	Average
2011	1.11	1.12
2011	1.12	
2015	0.94	0.95
2015	0.95	
2016	2.69	2.71
2016	2.72	
2017	2.07	2.06
2017	2.05	
2019	0.41	0.40
2019	0.39	
2020	2.79	2.75
2020	2.71	
2079	3.04	2.88
2079	2.71	
2097	11.15	10.80
2097	10.44	
2103	3.0	2.96
2103	2.91	

(continue) Table VII-6

2104	0.46	0.48
2104	0.49	
2107	1.66	1.65
2107	1.64	
2109	1.74	1.73
2109	1.71	
2131	0.31	0.33
2131	0.35	
2133	0.53	0.55
2133	0.57	
2135	0.29	0.30
2135	0.3	
2151	0.38	0.40
2151	0.41	
2152	ND	
2152	ND	
2156	0.67	0.66
2156	0.65	
2157	0.51	0.51
2157	0.5	
2159	1.36	1.39
2159	1.41	
2160	0.36	0.35
2160	0.33	

Appendix A: Table VII-7

B(a)P INDOOR AIR SAMPLES ANALYSIS

PHASE III

Sample no.	ng B(a)P /Filter	Average
2013	1.89	1.90
2013	1.91	
2030	2.55	2.45
2030	2.34	
2034	1.37	1.38
2034	1.39	
2044	1.96	1.89
2044	1.82	
2048	1.51	1.51
2048	1.5	
2054	1.69	1.65
2054	1.61	
2056	0.56	0.55
2056	0.54	
2058	2.73	2.75
2058	2.77	

(continue) Table VII-7

2134	1.35	1.35
2134	1.35	
2154	1.8	1.71
2154	1.62	
2155	18.74	19.43
2155	20.12	
2158	3.29	3.47
2158	3.65	

Appendix A: Table VII-8

B(a)P INDOOR AIR SAMPLES ANALYSIS

PHASE III

Sample no.	ng B(a)P /Filter	Average
2021	2.93	3.06
2021	3.18	
2022	1.51	1.80
2022	2.09	
2024	0.19	0.19
2024	0.18	
2025	2.06	1.90
2025	1.73	
2026	ND	
2026	ND	
2027	4.69	4.84
2027	4.98	
2028	2.84	2.94
2028	3.04	
2029	0.55	0.54
2029	0.52	

(continue) Table VII-8

2096	0.57	0.59
2096	0.61	
2099	1.56	1.71
2099	1.86	
2121	1.24	1.22
2121	1.19	
2122	1.57	1.55
2122	1.53	
2125	0.75	0.75
2125	0.74	
2127	3.06	2.98
2127	2.90	
2129	1.56	1.56
2129	1.56	
2130	0.63	0.63
2130	0.62	
2136	3.98	3.70
2136	3.42	
2146	0.97	1.04
2146	1.10	
2149	0.62	0.63
2149	0.63	
2150	0.47	0.46
2150	0.45	

Appendix A: Table VII-9

B(a)P INDOOR AIR SAMPLES ANALYSIS

PHASE III

Sample No.	Ng B(a)P /Filter	Average
2023	0.61	0.65
2023	0.69	
2031	5.04	4.97
2031	4.89	
2032	3.99	3.88
2032	3.77	
2035	4.08	4.06
2035	4.04	
2036	0.33	0.31
2036	0.29	
2037	0.97	1.00
2037	1.03	
2038	4.09	4.10
2038	4.10	
2039	5.13	5.19
2039	5.25	

(continue) Table VII-9

2040	8.06	8.09
2040	8.12	
2100	0.79	0.76
2100	0.72	
2124	5.05	5.01
2124	4.97	
2128	1.3	1.3
2128	1.3	
2138	1.05	1.05
2138	1.05	
2139	4.05	4.12
2139	4.18	

Appendix A: Table VII-10

B(a)P INDOOR AIR SAMPLES ANALYSIS

PHASE III

Sample no.	ng B(a)P/Filter	Average
2061	3.07	3.25
2061	3.42	
2071	ND	
2071	ND	
2072	3.69	3.55
2072	3.4	
2075	2.87	2.80
2075	2.73	
2080	0.73	0.84
2080	0.95	
2085	0.39	0.39
2085	0.38	
2087	0.26	0.26
2087	0.25	
2088	0.17	----
2088	ND	

(continue) Table VII-10

2090	ND	----
2090	ND	
2115	0.18	0.15
2115	0.11	
2117	ND	
2117	ND	
2126	ND	
2126	ND	
2164	2.05	2.17
2164	2.28	
2179	4.91	4.71
2179	4.51	
2180	1.04	0.99
2180	0.93	

Appendix A: Table VIII-1

B(a)P PERSONAL INHALATION AIR SAMPLES ANALYSIS

PHASE II

Site 0

Sample No.	ng B(a)P /Filter (Average)
001	1.8
002	1.81
003	2.96
004	3.75
032	6.79
034	7.58
051	0.26
053	8.43
056	0.49
057	5.87
071	4.31
083	1.44
091	6.18
103	6.01

(continue) Table VIII-1

115	1.02
127	3.26
140	1.44
141	3.52
142	3.62
143	1.89
155	ND
159	ND
160	0.27
161	2.1
189	2.59

Appendix A: Table VIII-2

B(a)P PERSONAL INHALATION AIR SAMPLES ANALYSIS

PHASE II

Site 10

Sample No.	Ng B(a)P /Filter (Average)
005	0.28
006	1.72
040	7.72
052	5.65
063	3.12
081	0.57
087	1.53
114	5.37
125	2.22
135	2.27
144	0.67
163	2.1
182	1.59
190	0.29

Appendix A: Table VIII-3

B(a)P PERSONAL INHALATION AIR SAMPLES ANALYSIS

PHASE II

Site 30

Sample No.	B(a)P ng/Filter	Average
007	7.41	7.37
007	7.33	
008	2.42	2.51
008	2.60	
036	14.31	13.92
036	13.53	
049	13.14	13.21
049	13.27	
065	9.70	9.53
065	9.36	
075	0.66	0.68
075	0.69	
101	14.17	14.03
101	13.88	

(continue) Table VIII-3

105	11.78	11.78
105	11.78	
118	7.18	7.30
118	7.42	
133	4.53	4.57
133	4.61	
156	2.37	2.38
156	2.38	
178	10.83	10.92
178	11.01	
184	7.15	7.10
184	7.05	
191	2.82	2.84
191	2.86	

Appendix A: Table VIII-4

B(a)P PERSONAL INHALATION AIR SAMPLES ANALYSIS

PHASE II

Site 40

Sample No.	ng B(a)P/Filter	Average
009	11.75	11.72
009	11.68	
011	1.69	1.73
011	1.77	
012	5.16	5.09
012	5.02	
029	11.76	11.73
029	11.69	
044	7.22	7.39
044	7.56	
055	12.15	12.19
055	12.22	
059	5.85	5.82
059	5.78	

(continue) Table VIII-4

061	5.00	4.98
061	4.95	
073	0.22	0.29
073	0.35	
074	2.11	2.13
074	2.14	
092	8.97	9.11
092	9.25	
095	4.48	4.45
095	4.41	
099	3.68	3.64
099	3.59	
106	12.64	12.58
106	12.51	
107	5.59	5.73
107	5.87	
126	4.83	4.88
126	4.93	
136	8.11	8.08
136	8.04	
137	3.76	3.76
137	3.76	
149	1.82	1.80
149	1.78	

(continue) Table VIII-4

151	3.15	3.14
151	3.13	
165	4.48	4.50
165	4.52	
169	2.15	2.06
169	1.97	
170	3.09	3.03
170	2.96	
185	2.25	2.33
185	2.41	
196	1.47	1.53
196	1.59	
197	0.96	0.97
197	0.97	

Appendix A: Table VIII-5

B(a)P PERSONAL INHALATION AIR SAMPLES ANALYSIS

PHASE II

Site 50

Sample No.	ng B(a)P/Filter	Average
013	1.51	1.50
013	1.49	
014	4.09	4.07
014	4.05	
015	1.11	1.12
015	1.13	
016	6.20	6.23
016	6.25	
030	5.84	5.84
030	5.84	
041	6.96	6.89
041	6.82	
043	11.76	11.82
043	11.87	

(continue) Table VIII-5

050	27.06	27.24
050	27.42	
062	4.80	4.78
062	4.76	
068	3.14	3.11
068	3.07	
076	5.38	5.36
076	5.34	
082	0.22	0.23
082	0.24	
088	5.12	5.14
088	5.16	
093	12.03	12.05
093	12.06	
100	6.20	6.17
100	6.14	
112	10.28	10.20
112	10.11	
117	2.45	2.47
117	2.49	
120	2.00	1.96
120	1.91	
128	1.71	1.73
128	1.75	

(continue) Table VIII-5

130	3.17	3.12
130	3.06	
152	1.54	1.57
152	1.60	
153	8.76	8.85
153	8.93	
172	2.58	2.61
172	2.65	
176	3.32	3.25
176	3.18	
177	2.51	2.50
177	2.49	
186	2.10	2.14
186	2.18	
192	1.34	1.32
192	1.29	
193	1.22	1.19
193	1.16	

Appendix A: Table VIII-6

B(a)P PERSONAL INHALATION AIR SAMPLES ANALYSIS

PHASE II

Site 60

Sample No.	Ng B(a)P/Filter	Average
010	30.14	30.18
010	30.21	
017	2.02	2.04
017	2.06	
018	4.24	4.19
018	4.14	
019	1.26	1.27
019	1.28	
020	3.40	3.41
020	3.42	
031	10.84	10.84
031	10.84	
037	25.05	25.15
037	25.24	

(continue) Table VIII-6

048	14.44	14.45
048	14.45	
054	7.40	7.45
054	7.50	
067	5.58	5.65
067	5.72	
069	19.03	19.18
069	19.33	
072	1.28	1.28
072	1.27	
084	0.43	0.45
084	0.47	
096	24.14	24.08
096	24.02	
097	3.51	3.52
097	3.52	
104	11.02	11.03
104	11.03	
110	6.08	6.04
110	5.99	
119	4.03	4.04
119	4.04	
129	6.91	6.92
129	6.92	

(continue) Table VIII-6

138	5.02	5.05
138	5.08	
148	21.65	21.60
148	21.55	
154	0.75	0.72
154	0.68	
157	7.06	7.06
157	7.06	
166	0.29	0.29
166	0.29	
183	4.67	4.72
183	4.76	
187	3.60	3.64
187	3.67	
198	1.31	1.34
198	1.37	

Appendix A: Table VIII-7

B(a)P PERSONAL INHALATION AIR SAMPLES ANALYSIS

PHASE II

Site 80

Sample No.	Ng B(a)P /Filter (Average)
021	0.73
022	3.11
023	0.64
024	2.11
033	7.01
039	12.02
046	8.19
047	12.4
066	2.18
070	0.26
078	0.21
080	0.48
085	7.07
086	6.57

(continue) Table VIII-7

102	1.33
108	8.13
109	13.46
116	2.72
121	2.2
124	2.8
134	2.77
139	3.08
146	0.93
147	1.43
162	2.57
164	1.52
174	0.99
180	2.46

Appendix A: Table VIII-8

B(a)P PERSONAL INHALATION AIR SAMPLES ANSLYSIS

PHASE II

Site 90

Sample No.	Ng B(a)P /Filter (Average)
025	1.6
026	5.54
027	0.94
028	4.65
035	10.37
038	18.22
042	11.89
045	12.18
064	4.78
077	4.44
079	9.88
089	11.44
098	15.43
111	14.29

(continue) Table VIII-8

113	59.51
122	132.09
123	15.5
131	2.9
132	3.38
145	4.82
150	2.54
158	1.91
167	25.62
168	3.87
173	12.88
194	0.72
195	3.44

Appendix A: Table IX-1

INDOOR SAMPLE ANALYSIS RESULT

COMPARE OF CONCENTRATION BETWEEN B(a)P and PYRENE

Sample No.	Ng B(a)P /Filter	Ng Pyrene /Filter
2002	0.56	0.14
2051	0.42	0.181
2065	0.74	0.197
2066	0.66	0.168
2068	3.96	0.173
2069	1.11	0.261
2092	1.13	0.107
2095	0.36	ND
2006	3.92	0.236
2062	0.77	ND
2078	0.31	0.075
2102	0.54	0.165
2106	1.08	0.19
2141	0.99	0.195
2144	0.9	0.095
2161	3.68	0.712
2163	1.71	0.74
2173	2.34	0.277

(continue) Table IX-1

2007	2.64	0.244
2043	0.54	ND
2046	0.48	ND
2060	4.15	0.563
2064	1.68	0.51
2073	5.83	0.65
2076	2.57	0.271
2105	0.52	0.154
2162	0.25	0.064
2172	0.79	0.29
2176	4.05	0.47
2178	0.61	0.12
2013	1.9	0.31
2030	2.45	0.377
2034	1.38	0.221
2044	1.89	0.06
2048	1.51	0.224
2054	1.65	0.442
2056	0.55	0.227
2058	2.75	0.42
2134	1.35	ND
2154	1.71	0.214
2155	19.43	ND
2158	3.47	0.455

Appendix A: Table X-1

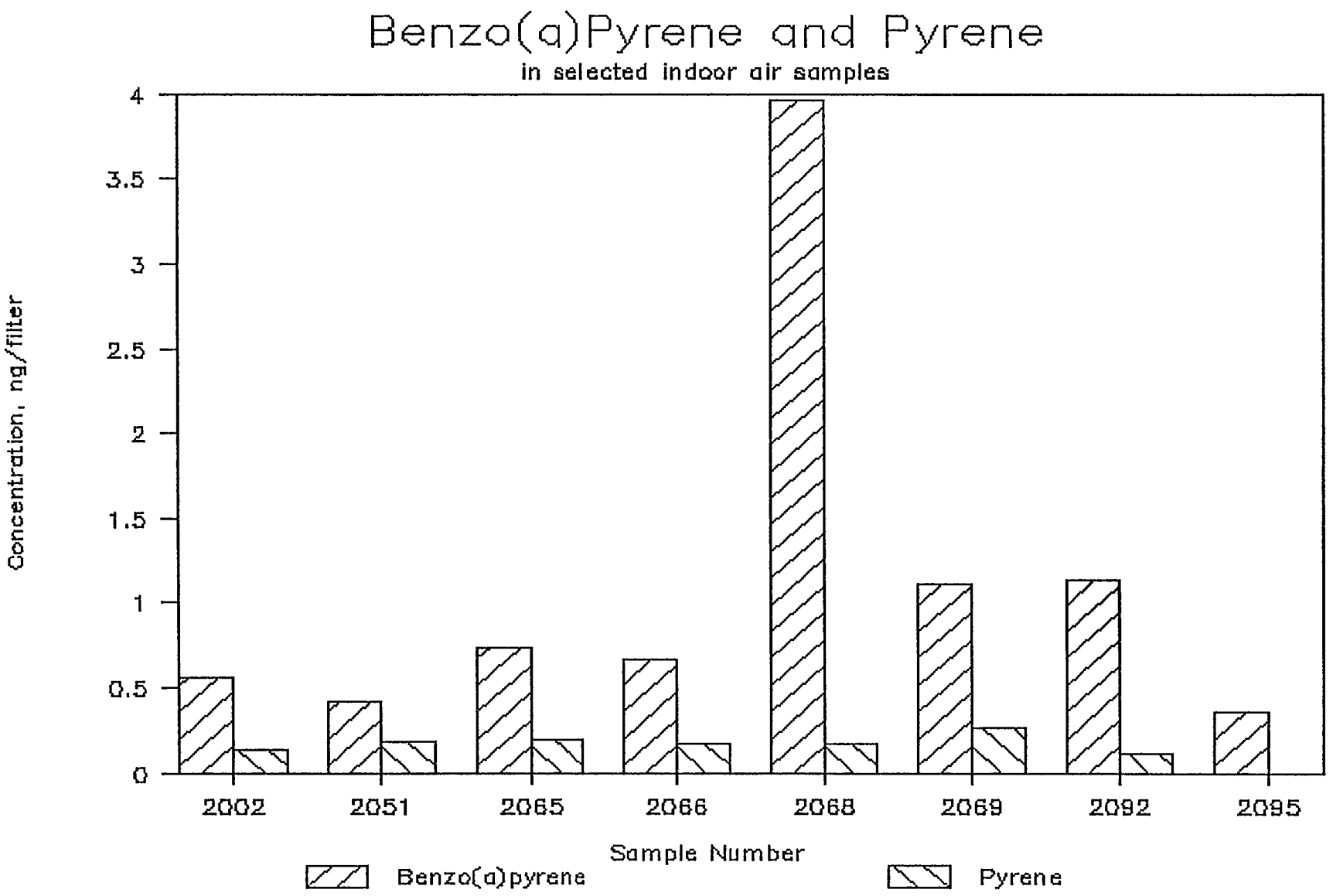
PERSONAL INHALATION AIR SAMPLES ANALYSIS

COMPARE OF BENZO(A)PYRENE & PYRENE

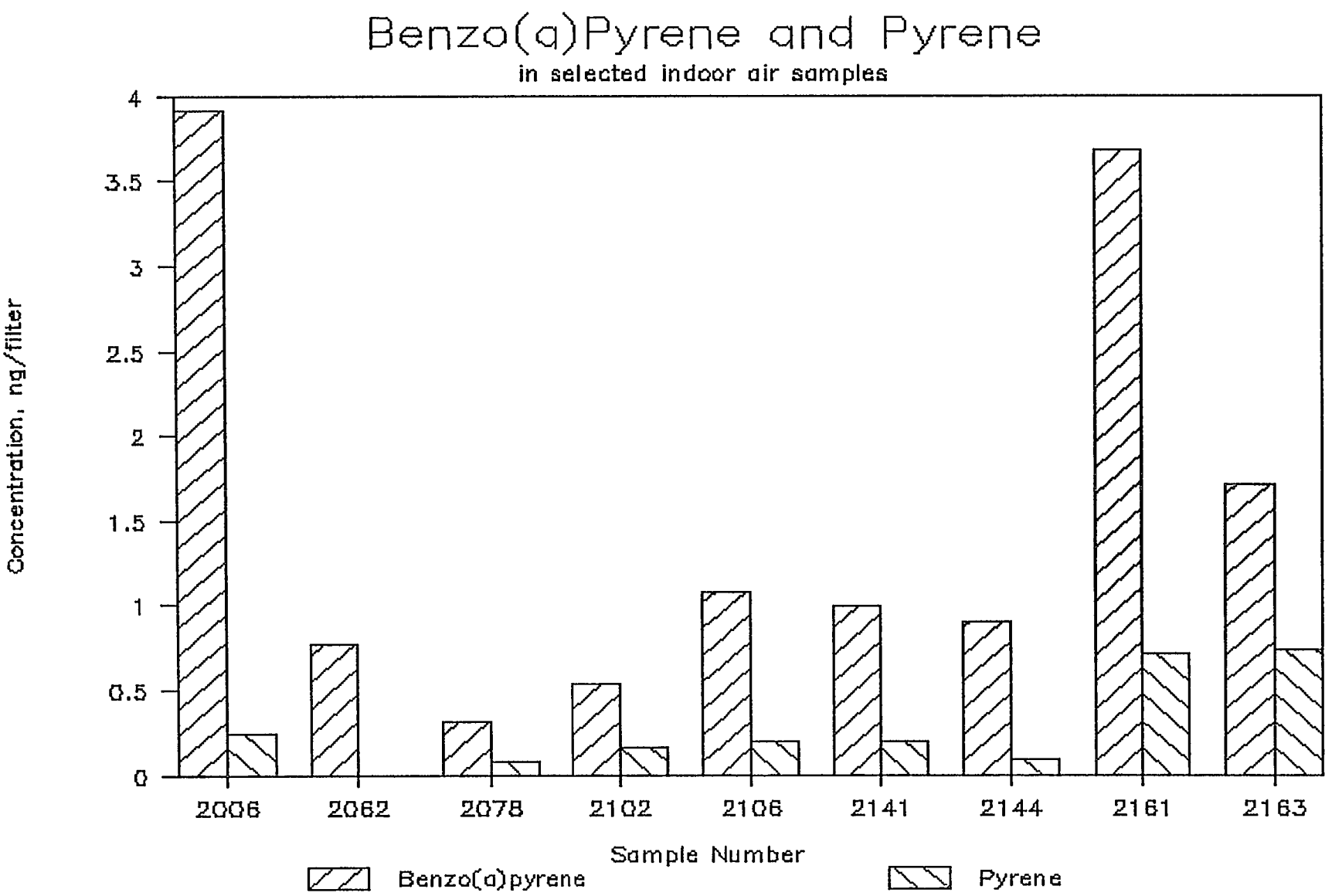
PHASE III

Sample NO.	ng B(a)P /Filter	ng Pyrene /Filter
1011	1.56	0.07
1030	2.51	ND
1036	0.19	0.053
1132	1.53	ND
1149	0.37	0.042
1151	0.67	0.05
1158	1.87	0.103
1172	0.78	0.062
1207	0.73	0.062
1221	1.34	0.079
1240	0.48	0.062
1254	0.40	0.047
1261	0.95	0.065
1266	0.31	ND

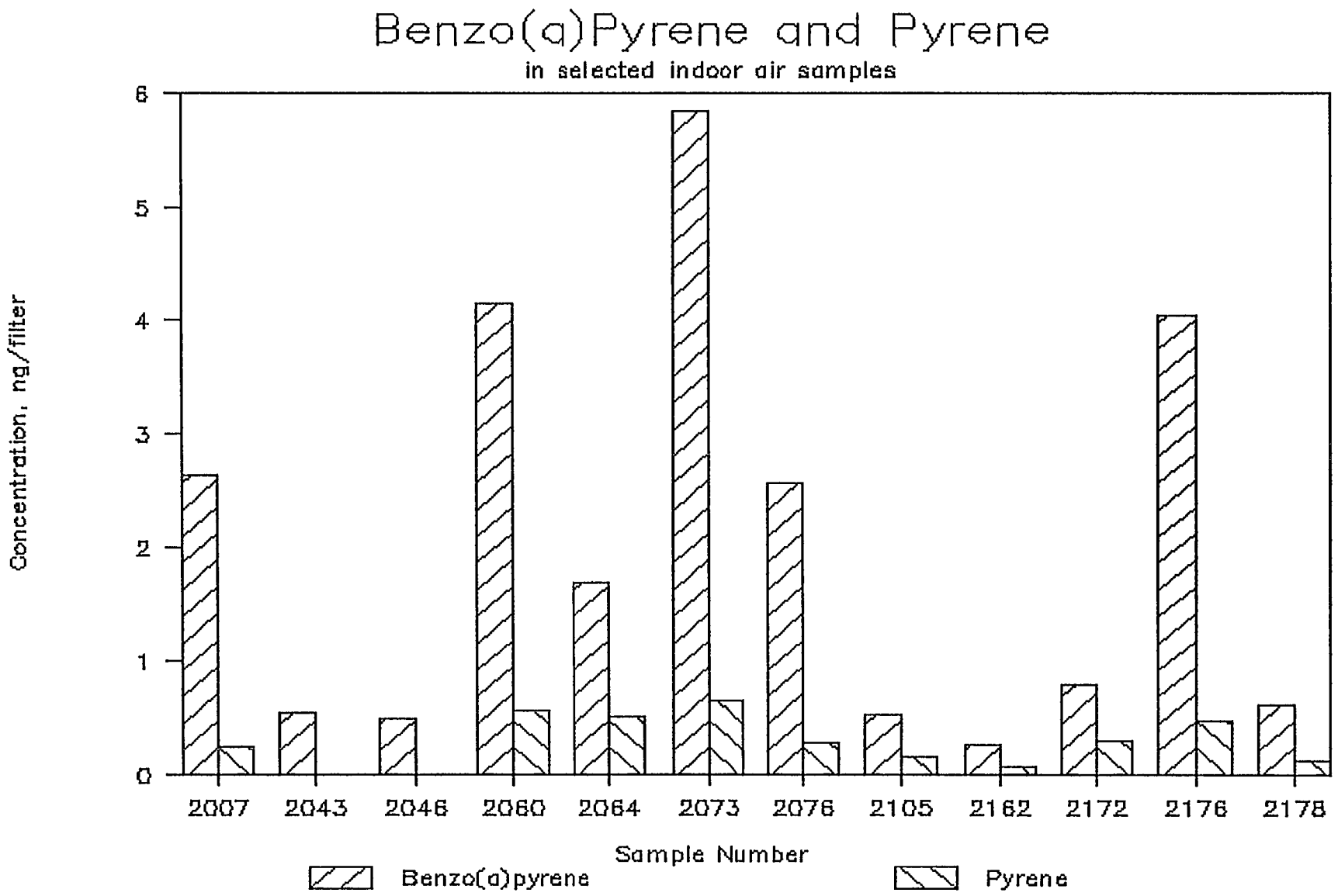
Appendix B: Figure 1



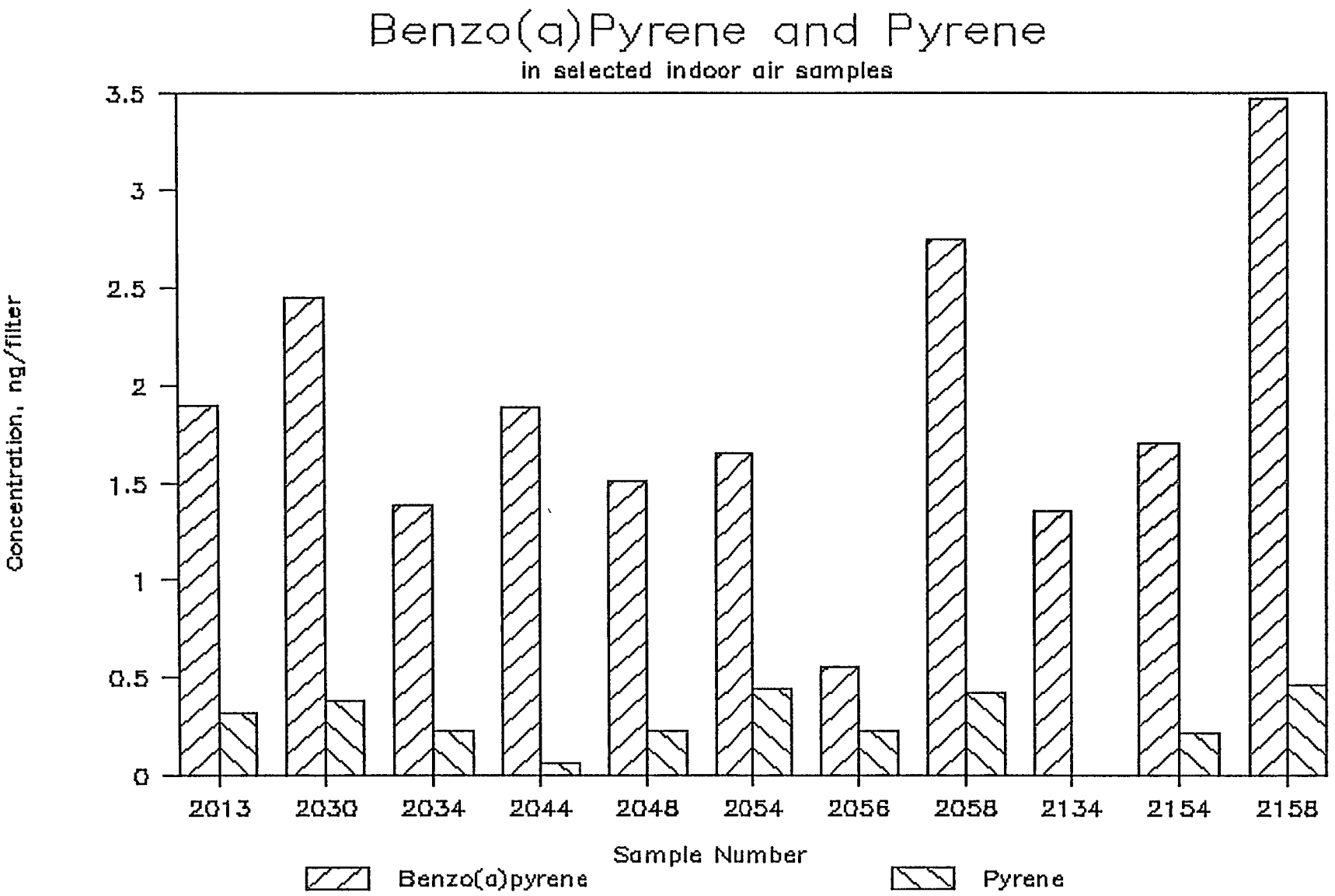
Appendix B: Figure 2



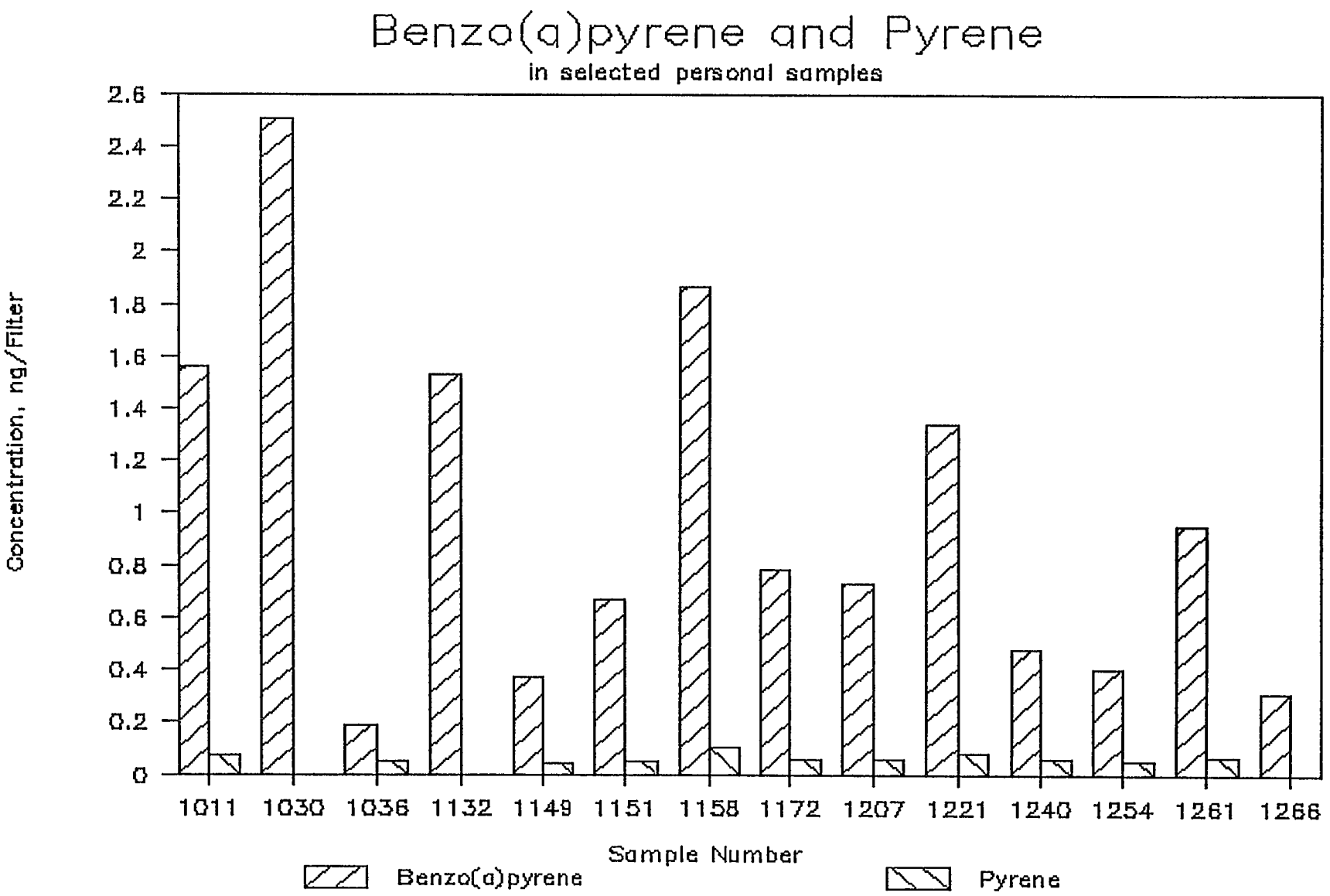
Appendix B: Figure 3



Appendix B: Figure 4



Appendix B: Figure 5



Appendix B: Figure 6