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A study of human exposure to benzo(a)pyrene (BAP) through different pathways. Part A ; Assessment of benzo(a)pyrene (BAP) exposure through urine analysis with the hydriodic acid reduction reaction. Part B

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ABSTRACT

Title of Thesis: PART A. A STUDY OF HUMAN EXPOSURE TO BENZO(A)PYRENE (BAP) THROUGH DIFFERENT PATHWAYS

PART B. ASSESSMENT OF BENZO(A)PYRENE (BAP) EXPOSURE THROUGH URINE ANALYSIS WITH THE HYDRIODIC ACID REDUCTION REACTION

Shao-Keng Liang. Master of Science, 1988

Thesis directed by: Dr. Arthur Greenberg
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PART A: An investigation of total human exposure to an environmental pollutant is described. The study is being conducted in a community impacted by a foundry suspected to be a dominant local source of benzo(a)pyrene (BaP). The research aims to assess the relative contributions of indoor and outdoor sources and to measure BaP in media including indoor air, outdoor air, food and water.

PART B: A method for biological monitoring of exposure to benzo(a)pyrene, a representative polycyclic aromatic hydrocarbon (PAH), has been developed. The analytical procedure includes extraction of PAH and PAH metabolites from urine using commercial cartridges containing c_18-modified silica, reduction of metabolites to PAH ("reverse metabolism") by hydriodic acid, detection by thin layer chromatography (TLC) and plate-scanning spectrofluorometry. This method is derived from a published procedure in which all PAH derived from PAH-metabolites are analyzed by gas chromatography. The BaP metabolite urine analysis has been applied to:

1. A control group of people who voluntarily treated a psoriasis condition with dermal application of coal tar.
2. Other human subjects who were more typical of the Phillipsburg population.

Limited studies of the HI reduction of specific metabolites have been performed.

PART A. A STUDY OF HUMAN EXPOSURE TO BENZO(A)PYRENE (BAP) THROUGH DIFFERENT PATHWAYS

PART B. ASSESSMENT OF BENZO(A)PYRENE (BAP) EXPOSURE THROUGH URINE ANALYSIS WITH THE HYDRIODIC ACID REDUCTION REACTION

by

Shao-Keng Liang

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
1988

APPROVAL SHEET

Title of Thesis: PART A: A STUDY OF HUMAN EXPOSURE TO BENZO(A)PYRENE (BAP) THROUGH DIFFERENT PATHWAYS

PART B: ASSESSMENT OF BENZO(A)PYRENE (BAP) EXPOSURE THROUGH URINE ANALYSIS WITH THE HYDRIODIC ACID REDUCTION REACTION

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

PART A: A STUDY OF HUMAN EXPOSURE TO BENZO(A)PYRENE (BAP) THROUGH DIFFERENT PATHWAYS

Chapter I. INTRODUCTION

The assessment of human exposure to an environmental contaminant requires the measurement of the amount contributed by each pathway. The traditional approach is to examine the concentration patterns of a pollutant in a single medium, e.g. air or water, since either the pollutant is considered to occur predominantly in that one medium or the major pathway of human intake is through that medium. In recent years progress has been made in extending study designs to multiple media.[1] However, these studies have usually been limited in scope and the data available from each medium are not always compatible for estimating exposure.

A study of total human exposure to BaP is currently being conducted in a foundry-impacted area in Phillipsburg, New Jersey. A two-week microenvironmental field experiment was conducted in January and February, 1987. The project has been 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 BaP. The approach involves monitoring BaP in all media containing significant BaP levels that the study population would come in contact with and quantitatively assessing the contributions of each exposure route.

In this thesis, the experimental design is described and the indoor-outdoor air results of the winter, 1987 microenvironmental study, are presented.

Chapter II. PRESTUDY DESIGN SITES AND SAMPLE COLLECTION

Pollution Selection

Benzo(a)pyrene (BaP) has been selected as the subject for this exposure measurement and modeling investigation termed the Total Human Environmental Exposure Study (THEES). Two of the major reasons are that the compound is mostly anthropogenic and is emitted during outdoor and/or indoor fossil fuel combustion.[2] The PAH are mutagens and known human carcinogens[2] and BaP has long been studied as a representative mutagen and carcinogen in this class. There are, in general, numerous outdoor point and area air pollution sources of BaP, including oil, coal and wood burning, smelting and automobile emissions. Indoor BaP is emitted from combustion sources, such as fireplaces, wood stoves, ovens and cigarettes. In addition, BaP can be found in water and soil from atmospheric deposition as well as in waste tailing poles from smelters. BaP is also found in food, especially in cooked food generated by smoking, curing, or broiling over a direct flame via the pyrolysis of fats.

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 smoke stack 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 (Figure 1).[3]

A previous thirteen-month air sampling study for airborne BaP in New Jersey showed that, of twenty-seven monitoring sites, Phillipsburg had the maximum BaP concentration for a twenty-four hour period (7.93 ng/m^3) and the second highest mean annual concentration (0.81 ng/m^3)[4].

Population and Home Characteristics

There are ten homes participating in THEES in Phillipsburg. The population for this study was not meant to be a statistically selected subgroup or representative sample of the types of people living in the Phillipsburg area. The selection of a limited number of participants was based upon the desire to examine the contributions of indoor and local outdoor air pollution and other media to exposure and to obtain an idea of dominant pathways in a pilot-scale study. Eight homes out of the ten chosen are near the plant and the remaining function as two outdoor air controls. The location of each home is shown in Figure 1. Both the homes and the individuals studied had characteristics in common as well as other unique characteristics. One home used a coal stove, seven used oil and two used gas for central heating (Table 1 & 2). Home I_{10} was selected for the study as a control for the outdoor BaP sources due to further distance from the potential emission source.

For the winter, however the presence of a coal stove in I_{10} created an indoor microenvironment which could be used to explore the influence of indoor coal combustion on indoor air quality.

FIGURE 1: THE PHILLIPSBURG AREA AND SITE LOCATIONS

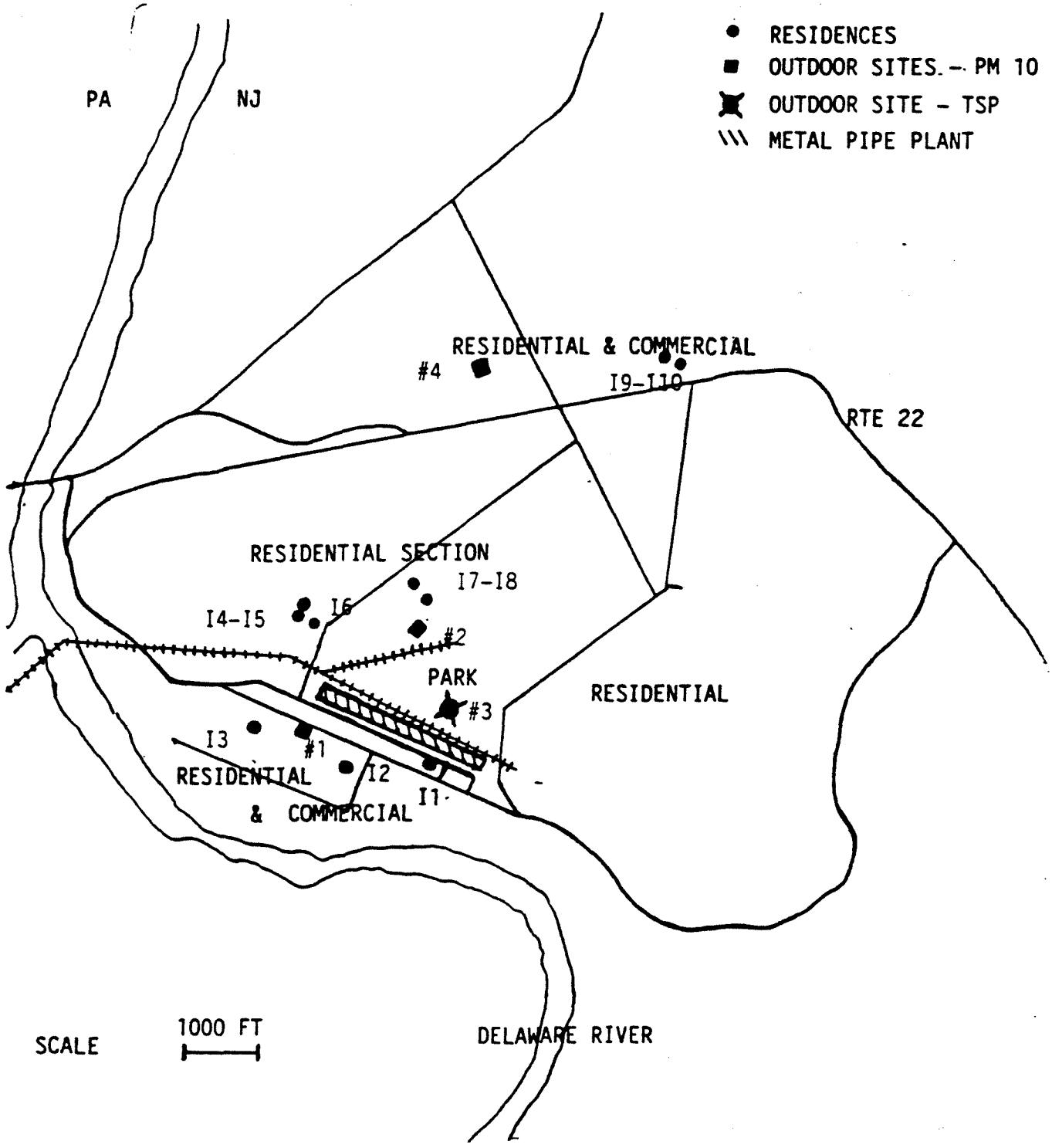


TABLE I
Indoor Combustion Sources located each THEES Home

Home ID	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 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 Central Heat (primarily & in living room Kerosene Heater (in kitchen) Gas Range & Oven Microwave Oven

Table II

Characteristics of Home Activities and Sources Over the Course of the Microenvironmental Study Conduct from January 20 through February 12, 1987

House#	Smokers	Room Space Heating	Open Windows	Hobby-Repairs Events*	#Occupant
I1	N	N	N		Y1
I2					
I4	N	K(9)	N		N2
I5	N	N	Y(14)		N2
I6	N	K(2)	Y(9)		Y4
I7	N	N	Y(1)		N3
I8	N	N	Y(1)		Y3
I9	Y(14)	N	N		N3
I10	N	C(14)	Y(1)		N3

(X) - Number of days in which activity occurred

N = NO, Y = YES, K = KEROSENE and C = COAL

* - Event such as gluing, hacksawing, painting, repairing furniture etc.

Sampling

A. Outdoor Sample

Outdoor samples were collected using a Wedding Inlet Hi-Volume sampler, which was operated at 40 cfm with a flow controller. The collection medium was a Gelman AE glass fiber filter which had been prefired in a muffle oven at 400°C for 48 hours weighted and stored in aluminum foil prior to use in the field. Before and after collection, filters were conditioned for 18-24 hours in the dark at room temperature and relative humidity < 20%. Subsequently, the filter was extracted for BaP analysis.

B. Indoor Sample

The indoor samples were collected using a Marple-Turner-Spengler impactor[5] with a 50% 10 um particle cut size, which operated at a flow of 10 liter/min. The flow through the impactor head was maintained to within ± 2% by a flow controller. The collection medium was a 37/mm Gelman AE filter that had been prefired in a manner similar to the use for the outdoor filters.

C. Water Sample

Water was taken from the kitchen tap in each home once a week during the primary study (two-week period), using polypropylene containers for sampling and laboratory storage. Subsequently, the sample water was drawn through a C_{18} Sep-Pak cartridge that had been pretreated with methanol to extract dissolved and particle borne organics. The Sep-Pak cartridge was then stored in a freezer until subjected to BaP analysis.

D. Food Sample

The food samples were collected from the in-home meals eaten by a single individual from each household. Each collected meal represented approximately one third of an adult portion. These were stored in labelled aluminum containers and frozen. The samples from each household were separated into weekly composite sets. The individual sets of food samples were weighed and then homogenized by Mr. Peter Creighton of UMDNJ-Robert Wood Johnson Medical School in a commercial blender prior to extraction.

Food samples were analyzed for BaP using a technique developed by Ms. Suyi Luo who was aided by Ms. Xueying Sun and Ms. Aijin Shen. The food sampling focussed on the eating habits of an identified individual in the home. Samples of that individual's breakfast, lunch and dinner were taken for analysis. The only restriction to the protocol was that we could only sample foods eaten at home and a complete accounting of meals taken in and out-of-the-home was documented by a questionnaire to allow for adjustment of in home food measurements.

E. Snow Sample

Snow samples were taken near each outdoor site with the same day as water sample taken, and represented one week's accumulation (during the study) of dry deposition of freshly fallen snow. Snow samples were kept at room temperature until melted. A known amount of distilled water was used to wash the container wall and the entire solution was filtered through a pre-wetted C_{18} cartridge as used for water samples.

F. Questionnaire

Each day during the study period, each participating family filled out an activity questionnaire designed by UMDNJ. They recorded information about the time spent in the home, indoor combustion sources used and information concerning ventilation. Other information in the questionnaire including details of foods eaten at home and at other locations.

Chapter III. METHODOLOGY

A. Samples Received and Storage

Indoor air samples and outdoor air samples are received wrapped in aluminum foil; water samples are received in C_{18} Sep-Pak cartridges in aluminum foil folders, and food composites are received in aluminum boxes covered with cardboard. These are promptly stored in a freezer at -10°C until analysis.

B. Chemicals and Apparatus

The extraction solvents cyclohexane, ethanol, dichloromethane and methanol are HPLC-grade and purchased from J.T. Baker Company, Inc. Standard solutions of benzo(a)pyrene (BaP) were prepared by dissolving weighed amounts, which were purchased from Aldrich Chemical Co., in cyclohexane and stored in the freezer in amber vials. A Sonic System, Inc ultrasonic instrument was used for extraction of BaP from glass fiber filters by cyclohexane at a temperature just below its boiling point.[4] Precoated thin layer chromatography (TLC) plates with 250 micron thickness 20% acetylated cellulose, were purchased from Analtech Co. An Analytical Instrument Specialties TLC multispotter was used for TLC plate spotting and florescence detection was performed with a Perkin-Elmer MFP-44B Florescence Spectrophotometer. An IBM personal computer with non-linear correction[6] program (which fit a least squares parabola having the form $Y=A_0+A_1X+A_2X^2$) was used to calculate BaP concentration.[4]

C. Analytical Procedure For Indoor/Outdoor Air Samples

Each indoor air filter (diameter 3.7 cm) was cut in half following sampling and each half was ultrasonically extracted using 2

ml of cyclohexane at 78-80°C for 30 minutes. The samples were then spotted on an acetylated cellulose (TLC) plate using a 250 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.

For outdoor air samples, a 1 inch by 8 inch strip from the center of each high volume filter was taken and ultrasonically extracted using 10 ml of cyclohexane at 78-80°C for 30 minutes following the same procedure as the indoor air samples. The TLC plate was similarly spotted and this was developed and examined by fluorescence detection.

D. Analytical Procedure For Food Samples

This is a brief outline of the procedure developed by Ms. Suyi Luo and detailed in her thesis.[7] A 40 gram sample of food composite and 7 grams KOH were refluxed with 100 ml absolute ethanol for 2 hours at 95°C. The sample solution was extracted with 50 ml isooctane three times. All isooctane solutions were washed with warm distilled water. The isooctane solution is passed through a column containing fluorisil and BaP subsequently eluted with 60 ml benzene. The volume of benzene is reduced under nitrogen to near dryness and the residue taken up in cyclohexane and a 100 ul aliquot analyzed as above by TLC/Fluorescence.

E. Analytical Procedure for Water and Snow Samples

Sample cartridges of Water and snow were eluted with 5 ml of methanol. The eluted solution was concentrated to 1 ml for TLC separation and fluorescence detection.

For control water samples, BaP purchased from Aldrich Chemical Co. was used. BaP was dissolved in acetonitrile as the stock solution This was mixed with different volumes of deionized distilled water to yield different concentrations. The control samples were eluted with the same kind of C_{18} cartridge following the same analytical procedures as the real samples.

Blank samples were prepared by using deionized distilled water as eluant, following the procedure described above.

Chapter IV RESULTS AND DISCUSSION

The effort focused on a detailed analysis of the roles of microenvironments and human exposure to B(a)P in the Phillipsburg area:

1. The role of each pathway.
2. The relation between each pathway.
3. The outdoor environment immediately surrounding the grey iron foundry, an important B(a)P source.
4. Inside homes in neighborhoods immediately surrounding the foundry.
5. estimate the human exposure B(a)P.

The B(a)P concentrations contributed by each pathway are listed as follows:

* All the indoor air sample 10 homes data are listed in Table III-1 to Table III-10 in home sequence.
* Outdoor sample data location numbers 101 to 104 are listed in Table IV-1 to Table IV-4.
* Food sample data for the 10 homes in the study are listed in Table V-1 and Table V-2.
* Snow sample results are presented in Table VI.
* The total B(a)P concentration by site will be shown on figure II.
* Since all the water samples readings of B(a)P are below the limits of quantification (Appendix I) the results are reported as no-detection.

Table III-1

Home 1 Indoor Air Sample Data

No.	Time	Hour	Air Vol (m^3)	Mass (ug)	Mass Conc (ug/m^3)	BaP (ng)	BaP Conc (ng/m^3)
1	1/29-1/30	23.3	14.0	434	31	67.1	4.80
2	1/30-1/31	23.4	14.0	302	21.5	8.90	0.63
3	1/31-2/01	24.3	14.6	360	24.7	1.72	0.12
5	2/02-2/03	24.0	14.4	728	50.6	21.7	1.51
6	2/03-2/04	23.8	14.3	391	27.4	1.83	0.13
7	2/04-2/05	24.7	14.8	306	20.6	1.12	0.08
8	2/05-2/06	22.8	13.7	385	28.2	19.3	1.41
9	2/06-2/07	24.0	14.4	500	34.7	12.4	0.86
10	2/07-2/08	23.5	14.1	366	25.9	6.14	0.43
11	2/08-2/09	23.8	14.3	569	39.8	5.90	0.41
12	2/09-2/10	25.0	15.0	310	20.7	3.98	0.27
13	2/10-2/11	24.0	14.4	1372	95.3	1.87	0.13
14	2/11-2/12	23.6	14.2	1087	76.8	18.88	1.33
average		23.9	14.3	546.9	38.2	13.1	0.9
std. dev.		0.6	0.3	317.8	22.2	17.1	1.2
geo. mean					33.7		0.5

Table III-2

Home 2 Indoor Air Sample Data

No.	Time	Hour	Air Vol (m^3)	Mass (ug)	Mass Conc (ug/m^3)	BaP (ng)	BaP Conc (ng/m^3)
3	1/31-2/01	23.9	2.9	317	111	12.6	4.40
4	2/01-2/02	22.4	2.7	151	56.3	0.03	0.01
5	2/02-2/03	24.9	14.9	1704	114	59.1	3.96
6	2/03-2/04	23.8	14.3	1300	91.2	28.4	1.99
7	2/04-2/05	24.3	14.6	1125	77.2	13.4	0.92
8	2/05-2/06	23.3	14.0	1318	94.5	25.9	1.86
10	2/07-2/08	23.3	14.0	1920	137	16.9	1.20
11	2/08-2/09	23.8	14.3	1277	89.4	16.2	1.13
12	2/09-2/10	24.4	14.6	1300	89.0	13.7	0.93
13	2/10-2/11	24.5	14.7	1404	95.5	13.7	0.93
14	2/11-2/12	23.8	14.3	1569	110	26.5	1.86
average		23.8	12.3	1216	96.8	20.6	1.70
std. dev.		0.7	4.5	511	20.2	14.4	1.30
geo. mean					94.6		1.00

Table III-3

Home 3 Indoor Air Sample Data

No.	Time	Hour	Air Vol (m^3)	Mass (ug)	Mass Conc (ug/m^3)	BaP (ng)	BaP Conc (ng/m^3)
1	1/29-1/30	17.9	10.7	501	46.6	12.3	1.15
2	1/30-1/31	21.1	12.7	571	45.1	10.9	0.86
3	1/31-2/01	27.7	16.6	628	37.8	7.32	0.44
4	2/01-2/02	23.7	14.2	816	57.4	17.6	1.23
5	2/02-2/03	24.0	14.4	851	59.1	30.8	2.14
6	2/03-2/04	23.8	14.3	485	34.0	14.1	0.99
7	2/04-2/05	24.4	14.6	263	18.0	7.55	0.52
9	2/06-2/07	23.8	14.3	731	51.2	24.8	1.74
10	2/07-2/08	24.0	14.4	687	47.7	13.4	0.93
11	2/08-2/09	23.8	14.3	328	23.0	4.49	0.31
12	2/09-2/10	24.7	14.8	636	42.9	11.8	0.80
13	2/10-2/11	24.4	14.6	364	24.9	12.6	0.86
average		23.6	14.2	572	40.6	14.0	1.00
std. dev.		2.20	1.30	181	12.8	7.10	0.50
geo. mean					38.3		0.90

Table III-4

Home 4 Indoor Air Sample Data

No.	Time	Hour	Air Vol (m^3)	Mass (ug)	Mass Conc (ug/m^3)	BaP (ng)	BaP Conc (ng/m^3)
1	1/29-1/30	23.6	14.2	550	38.8	19.7	1.39
2	1/30-1/31	23.4	14.0	561	40.0	9.70	0.69
3	1/31-2/01	23.8	14.3	424	29.6	4.02	0.28
4	2/01-2/02	24.2	14.5	349	24.1	14.3	0.99
5	2/02-2/03	20.1	12.0	710	59.0	22.8	1.90
6	2/03-2/04	25.6	15.4	268	17.4	2.42	0.16
7	2/04-2/05	23.0	13.8	191	13.8	5.09	0.37
8	2/05-2/06	23.3	14.0	488	34.9	13.4	0.96
9	2/06-2/07	23.7	14.2	771	54.2	22.9	1.61
10	2/07-2/08	23.4	14.0	461	32.9	9.54	0.68
11	2/08-2/09	23.9	14.4	123	8.60	2.76	0.19
12	2/09-2/10	24.6	14.8	119	8.10	6.20	0.42
13	2/10-2/11	24.6	14.8	188	12.7	7.98	0.54
14	2/11-2/12	24.2	14.5	359	24.7	17.4	1.20
average		23.7	14.2	397	28.5	11.3	0.80
std. dev.		1.20	0.70	203	15.7	6.80	0.50
geo. mean					24.0		0.60

Table III-5

Home 5 Indoor Air Sample Data

No.	Time	Hour	Air Vol (m^3)	Mass (ug)	Mass Conc (ug/m^3)	BaP (ng)	BaP Conc (ng/m^3)
1	1/29-1/30	23.7	14.2	401	28.3	8.91	0.63
2	1/30-1/31	23.3	14.0	247	17.6	8.26	0.59
3	1/31-2/01	24.1	14.5	106	7.3	0.03	0.00
4	2/01-2/02	24.0	14.4	383	26.6	13.2	0.92
5	2/02-2/03	23.9	14.3	443	30.9	16.0	1.12
6	2/03-2/04	23.8	14.3	171	12.0	0.03	0.00
7	2/04-2/05	24.6	14.8	7.00	0.50	0.03	0.00
8	2/05-2/06	21.2	12.7	215	16.9	7.85	0.62
9	2/06-2/07	26.0	15.6	328	21.0	14.7	0.94
10	2/07-2/08	23.8	14.3	67.0	4.70	6.22	0.44
11	2/08-2/09	23.3	14.0	39.0	2.80	1.15	0.08
12	2/09-2/10	24.8	14.9	116	7.80	1.26	0.08
13	2/10-2/11	24.1	14.5	163	11.3	2.80	0.19
14	2/11-2/12	26.6	10.0	182	18.3	7.68	0.77
average		23.4	14.0	205	14.7	6.30	0.50
std. dev.		2.10	1.30	134	9.30	5.40	0.40
geo. mean					10.2		0.10

Table III-6

Home 6 Indoor Air Sample Data

No.	Time	Hour	Air Vol (m^3)	Mass (ug)	Mass Conc (ug/m^3)	BaP (ng)	BaP Conc (ng/m^3)
1	1/29-1/30	23.4	14.0	733	52.2	16.0	1.14
2	1/30-1/31	23.4	14.0	376	26.8	7.94	0.57
3	1/31-2/01	25.8	15.5	975	63.0	0.03	0.00
4	2/01-2/02	22.3	13.4	436	32.6	9.54	0.71
5	2/02-2/03	23.9	14.3	668	46.6	19.9	1.39
6	2/03-2/04	23.7	14.2	374	26.4	0.48	0.03
7	2/04-2/05	25.0	15.0	296	19.7	1.52	0.10
8	2/05-2/06	22.8	13.7	433	31.7	14.6	1.07
9	2/06-2/07	47.7	28.6	1356	47.4	32.9	1.15
10	2/07-2/08	23.9	14.3	389	27.1	0.03	0.00
11	2/08-2/09	24.6	14.7	204	13.8	4.98	0.34
12	2/09-2/10	24.1	14.5	277	19.2	1.60	0.11
13	2/10-2/11	23.6	14.2	396	28.0	17.7	1.25
average		25.7	15.4	532	33.4	9.80	0.60
std. dev.		6.40	3.80	313	14.0	9.60	0.50
geo. mean					30.6		0.20

Table III-7

Home 7 Indoor Air Sample Data

No.	Time	Hour	Air Vol (m^3)	Mass (ug)	Mass Conc (ug/m^3)	BaP (ng)	BaP Conc (ng/m^3)
1	1/29-1/30	23.6	14.2	420	29.7	6.64	0.47
2	1/30-1/31	23.5	14.1	360	25.5	5.24	0.37
3	1/31-2/01	25.8	15.5	248	16.0	2.57	0.17
4	2/01-2/02	22.1	13.3	302	22.8	14.4	1.09
5	2/02-2/03	24.1	14.5	720	49.8	21.7	1.50
6	2/03-2/04	23.7	14.2	175	12.3	4.05	0.28
7	2/04-2/05	24.5	14.7	33.0	2.20	6.26	0.43
8	2/05-2/06	23.1	13.9	386	27.8	21.2	1.53
9	2/06-2/07	24.5	14.7	605	41.2	18.0	1.23
10	2/07-2/08	25.1	15.1	646	42.8	9.18	0.61
11	2/08-2/09	21.9	13.1	156	11.9	3.40	0.26
12	2/09-2/10	24.4	14.7	156	10.6	2.98	0.20
13	2/10-2/11	24.3	14.6	241	16.5	1.20	0.08
14	2/11-2/12	24.2	14.5	297	20.5	14.2	0.98
average		23.9	14.4	339	23.5	9.40	0.70
std. dev.		1.00	0.60	194	13.2	6.90	0.50
geo. mean					19.0		0.50

Table III-8

Home 8 Indoor Air Sample Data

No.	Time	Hour	Air Vol (m^3)	Mass (ug)	Mass Conc (ug/m^3)	BaP (ng)	BaP Conc (ng/m^3)
1	1/29-1/30	23.3	14.0	773	55.2	91.9	6.56
2	1/30-1/31	23.4	14.0	3983	284	19.3	1.37
3	1/31-2/01	24.2	14.5	3134	216	5.00	0.35
4	2/01-2/02	23.8	14.3	505	35.4	9.79	0.69
5	2/02-2/03	24.0	14.4	926	64.3	24.8	1.72
6	2/03-2/04	23.7	14.2	138	9.70	1.12	0.08
7	2/04-2/05	24.9	14.9	973	65.1	28.8	1.93
8	2/05-2/06	23.8	14.3	610	42.8	31.3	2.20
9	2/06-2/07	23.3	14.0	523	37.4	22.7	1.62
10	2/07-2/08	23.3	14.0	127	9.10	7.46	0.53
11	2/08-2/09	23.8	14.3	1244	87.1	33.9	2.38
12	2/09-2/10	24.7	14.8	553	37.3	39.1	2.64
13	2/10-2/11	24.3	14.6	749	51.4	52.2	3.58
14	2/11-2/12	24.7	14.8	337	22.7	10.6	0.71
average		23.9	14.4	1041	72.7	27.0	1.90
std. dev.		0.50	0.30	1081	76.3	22.8	1.60
geo. mean					47.2		1.20

Table III-9

Home 9 Indoor Air Sample Data

No.	Time	Hour	Air Vol (m^3)	Mass (ug)	Mass Conc (ug/m^3)	BaP (ng)	BaP Conc (ng/m^3)
3	1/30-1/31	23.0	13.8	872	63.2	13.4	0.97
4	1/31-2/01	23.7	14.2	834	58.6	12.9	0.90
5	2/01-2/02	23.6	14.2	1052	74.3	28.3	2.00
6	2/02-2/03	24.1	14.5	1301	90.0	29.7	2.06
7	2/03-2/04	23.6	14.2	598	42.1	21.4	1.51
8	2/04-2/05	24.9	14.9	564	37.8	34.0	2.27
9	2/05-2/06	23.1	13.9	686	49.5	24.6	1.78
10	2/06-2/07	23.7	14.2	1218	85.7	25.9	1.82
11	2/07-2/08	23.9	14.4	1014	70.6	25.5	1.77
12	2/08-2/09	23.5	14.1	326	23.1	17.4	1.24
13	2/09-2/10	24.4	14.6	454	31.1	30.2	2.07
14	2/10-2/11	24.0	14.4	486	33.7	23.1	1.60
15	2/11-2/12	23.9	14.3	454	31.7	33.2	2.32
average		23.8	14.3	758	53.2	24.6	1.70
std. dev.		0.50	0.30	303	21.3	6.60	0.40
geo. mean					48.9		1.60

Table III-10

Home 10 Indoor Air Sample Data

No.	Time	Hour	Air Vol (m^3)	Mass (ug)	Mass Conc (ug/m^3)	BaP (ng)	BaP Conc (ng/m^3)
1	1/29-1/30	23.7	14.2	867	61.1	15.5	1.09
2	1/30-1/31	22.9	13.7	3723	272	3.94	0.29
4	2/01-2/02	23.6	14.2	1282	90.5	0.03	0.00
5	2/02-2/03	17.9	10.7	1486	138	23.7	2.21
6	2/03-2/04	23.5	14.1	280	19.9	21.9	1.55
7	2/04-2/05	25.0	15.0	381	25.4	23.1	1.54
9	2/06-2/07	23.7	14.2	1093	76.9	45.5	3.20
10	2/07-2/08	23.8	14.3	1558	109	116	8.11
11	2/08-2/09	23.8	14.3	534	37.4	2.14	0.15
12	2/09-2/10	24.3	14.6	375	25.7	2.82	0.19
13	2/10-2/11	23.8	14.3	644	45.1	17.0	1.19
14	2/11-2/12	23.8	14.3	950	66.5	62.5	4.37
average		23.3	14.0	1097.	80.6	27.8	2.00
std. dev.		1.70	1.00	894	67.2	31.9	2.20
geo. mean					60.8		0.70

Table IV-1

Out Door Air Sample Data Site 101

No.	Time	Hour	Air Vol (m^3)	BaP (ng/fil)	Mass (ug/fil)	BaP Conc (ng/m^3)	Mass Conc (ug/m^3)
1	1/29-1/30	22.8	1551	1171	71000	0.80	45.8
2	1/30-1/31	22.9	1557	820	5800	0.50	3.70
3	1/31-2/01	23.9	1621	216	2300	0.10	1.40
4	2/01-2/02	23.9	1622	2184	146400	1.30	90.3
5	2/02-2/03	23.8	1618	3348	130300	2.10	80.6
6	2/03-2/04	23.9	1624	474	42300	0.30	26.0
7	2/04-2/05	24.7	1637	631	32600	0.40	19.4
8	2/05-2/06	23.4	1589	2666	91300	1.70	57.5
9	2/06-2/07	24.2	1644	3308	106100	2.00	64.6
10	2/07-2/08	23.5	1594	1750	55400	1.10	34.8
11	2/08-2/09	23.6	1605	620	39500	0.40	24.6
12	2/09-2/10	24.8	1682	838	40200	0.50	23.9
13	2/10-2/11	23.4	1589	683	39700	0.40	25.0
14	2/11-2/12	25.2	1715	2216	73500	1.30	42.9
average		23.8	1621	1495	62600	0.90	38.6
std. dev.		0.70	45.2	1035	41659	0.60	25.7
geo. mean						0.70	26.2

Table IV-2

Out Door Air Sample Data Site 102

No.	Time	Hour	Air Vol (m^3)	BaP (ng/fil)	Mass (ug/fil)	BaP Conc (ng/m^3)	Mass Conc (ug/m^3)
1	1/29-1/30	22.8	1552	1982	76100	1.30	49.0
2	1/30-1/31	23.9	1624	850	8500	0.50	5.20
3	1/31-2/01	23.8	1614	262	41100	0.20	25.5
4	2/01-2/02	24.0	1632	1722	79800	1.10	48.9
5	2/02-2/03	23.7	1607	4226	117700	2.60	73.2
6	2/03-2/04	23.9	1626	646	41800	0.40	25.7
7	2/04-2/05	25.0	169	284	30400	0.20	17.9
8	2/05-2/06	23.1	1567	2526	80200	1.60	51.2
9	2/06-2/07	24.2	1643	3464	102100	2.10	62.1
10	2/07-2/08	23.5	1595	383	54900	0.20	34.4
11	2/08-2/09	23.7	1608	533	36300	0.30	22.6
12	2/09-2/10	24.2	1646	808	48700	0.50	29.6
13	2/10-2/11	23.4	1588	609	38400	0.40	24.2
14	2/11-2/12	25.2	1710	2408	672	1.40	39.3
average		23.9	1622	1479	58800	0.90	36.3
std. dev.		0.60	42.5	1226	28774	0.80	17.9
geo. mean						0.60	31.0

Table IV-3

Out Door Air Sample Data Site 103

No.	Time	Hour	Air Vol (m^3)	BaP (ng/fil)	Mass (ug/fil)	BaP Conc (ng/m^3)	Mass Conc (ug/m^3)
1	1/29-1/30	23.9	1621	1051	116200	0.60	71.7
2	1/30-1/31	24.0	1628	1361	41900	0.80	15.7
3	1/31-2/01	23.7	1613	262	53100	0.20	323
4	2/01-2/02	24.0	1631	2039	107200	1.30	65.7
5	2/02-2/03	23.7	1607	6470	193200	4.00	120
6	2/03-2/04	24.0	1629	1121	101100	0.70	62.1
7	2/04-2/05	24.4	1666	972	77200	0.60	46.5
8	2/05-2/06	23.8	1619	2829	149700	1.70	92.4
9	2/06-2/07	24.0	1633	3804	146300	2.30	89.6
10	2/07-2/08	23.4	1591	2357	68800	1.50	43.2
11	2/08-2/09	23.7	1608	1016	65900	0.60	41.0
12	2/09-2/10	24.4	1660	1166	106300	0.70	64.0
13	2/10-2/11	23.4	1589	736	116500	0.50	73.3
14	2/11-2/12	/	/	182	27500	/	/
average		23.9	1622	1937	103338	1.20	63.7
std. dev.		0.30	21.0	1603	41151	1.00	25.5
geo. mean						0.90	58.6

/ : no data available

Table IV-4

Out Door Air Sample Data Site 104

No.	Time	Hour	Air Vol (m^3)	BaP (ng/fil)	Mass (ug/fil)	BaP Conc (ng/m^3)	Mass Conc (ug/m^3)
1	1/29-1/30	24.8	1685	1280	82600	0.80	49.0
2	1/30-1/31	23.9	1624	645	57900	0.40	35.6
3	1/31-2/01	23.7	1607	262	77700	0.20	48.3
4	2/01-2/02	24.1	1638	2009	82700	1.20	50.5
5	2/02-2/03	23.6	1605	2613	118500	1.60	73.8
6	2/03-2/04	24.0	1629	208	35400	0.10	21.7
7	2/04-2/05	23.5	1600	458	24800	0.30	15.5
8	2/05-2/06	24.7	1681	2317	92700	1.40	55.1
9	2/06-2/07	24.0	1628	3846	107800	2.40	66.2
10	2/07-2/08	23.5	1596	1805	60400	1.10	37.8
11	2/08-2/09	23.6	1606	506	35900	0.30	22.4
12	2/09-2/10	24.4	1658	751	37300	0.50	22.5
13	2/10-2/11	23.9	1623	469	34900	0.30	21.5
14	2/11-2/12	24.9	1689	2102	70200	1.20	41.6
average		24.0	1634	1377	65629	0.80	40.1
std. dev.		0.50	31.0	1055	28474	0.60	17.4
geo. mean						0.60	36.2

Table V-1

The BaP Concentration in Weekly Food Samples and Weekly Food Ingestion Exposure

Home #	Week 1 BaP ng/g Wet Food Weight	# meals	BaP Week 1 ng	Week 2 BaP ng/g Wet Food Weight	# meals	BaP Week 2 ng
1	0.180	19	1435	0.053	21	456
2	0.208	21	1240	0.069	21	442
3	0.017	16	61	0.021	16	69
4	0.004	8	14	0.027	9	118
5	0.004	20	10	0.148	16	385
6	0.015	19	47	0.181	18	839
7	0.005	13	21	0.027	11	103
8	0.021	19	144	0.128	15	626
9	0.373	18	1685	1.173	18	4005
10	0.024	19	178	0.033	17	250

Table V-2

Intercomparison of the weekly Food Ingestion and Inhalation Exposure to Benzo(a)pyrene for each THESS Home

Home #	Week #	Food BaP(ng)	Inhaled BaP(ng)	Ratio Food/inhaled
1	1	1435	165	8.7
1	2	456	116	3.9
2	1	1240	260	4.7
2	2	442	174	2.5
3	1	61	156	0.4
3	2	69	134	0.5
4	1	14	140	0.1
4	2	118	140	0.8
5	1	10	93	0.1
5	2	385	78	4.9
6	1	47	107	0.4
6	2	839	94	8.9
7	1	21	115	0.2
7	2	103	126	0.8
8	1	144	310	0.5
8	2	626	297	2.1
9	1	1685	202	8.3
9	2	4005	268	14.9
10	1	178	146	1.2
10	2	250	385	0.7

Note: Inhaled BaP is over a 24 h period each day which includes the Total Indoor Exposure and an estimate of the BaP exposure for the hours not in the home. The non-home-hours estimate uses the average ambient concentration for this microenvironment, i.e. no occupational or occasional indoor source exposure while away from the home.

Table VI

Snow Sample Data

Sample	Volume(ml)	ng/injection	ng/filter
101	260	27.8	107
102	670	14.0	21.0
103	/	4.25	/
104	800	1.99	2.49
Blank	800	ND	ND

/ : no data available
ND: below detection limit
note:sample was taken at the same time with water sample and beside outdoor sampler 101, 102, 103, 104, respectively.

BaP Concentrations by Site

THEES, January 29 – February 12, 1987

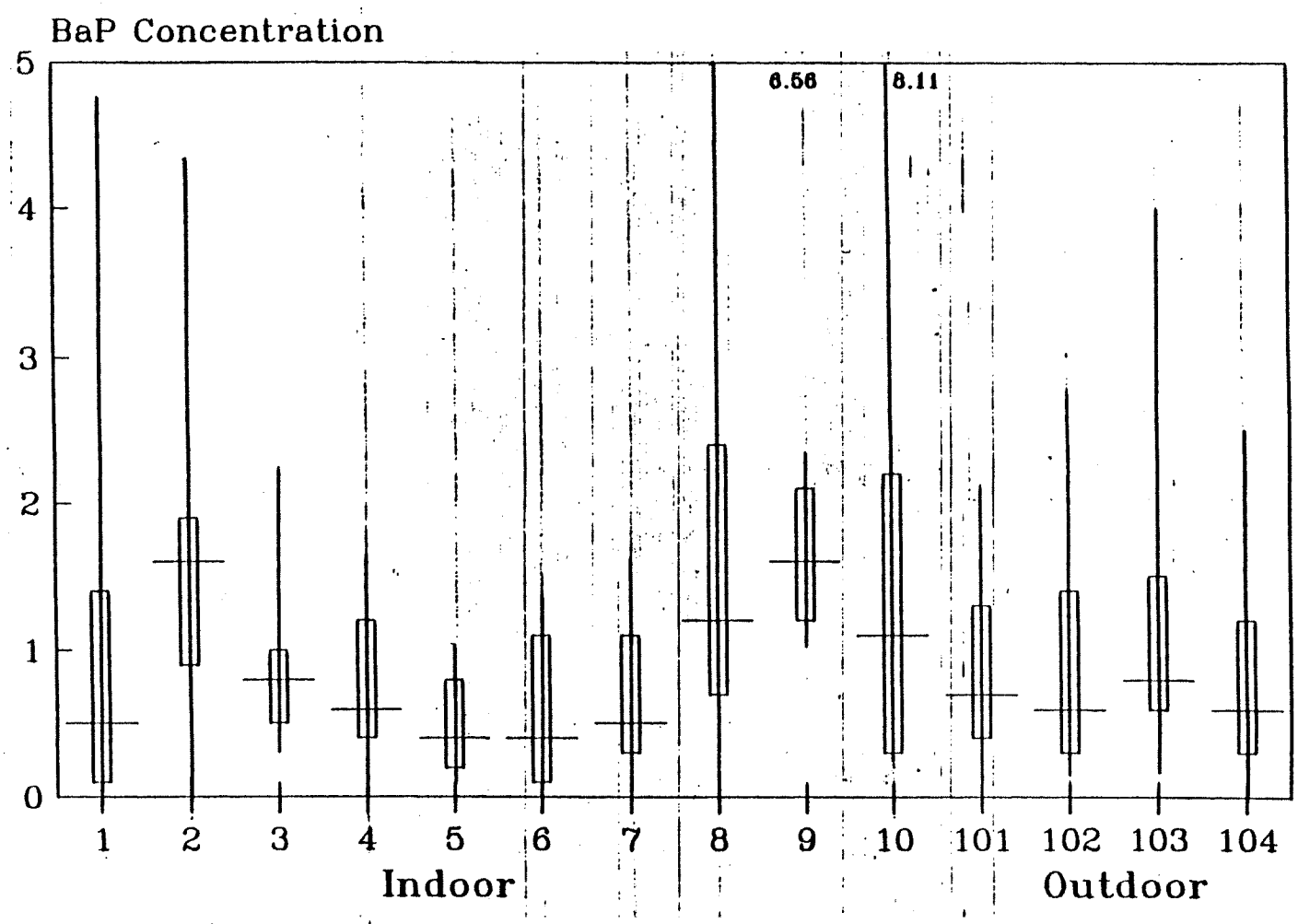


Figure II

Concentration in ng/ cubic meter
Horizontal line is geometric mean

Figure 1 depicts the fact that two of the outdoor sites for sampling BaP were situated within a quarter mile of the foundry. One was located to the east (#102) and the other to the west (#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 (total suspension particle) data for a number of years[3].

The mean outdoor concentration of BaP was 0.9 ng/m^3, the highest BaP concentration (1.2 ng/m^3) is site #103 which is the nearest to foundry. Site #104, the control outdoor sampling site, is approximately one mile away from the foundry and closest to route 22 and has the lowest concentration (0.8 ng/m^3). This supports the view that the foundry is a source of BaP in local outdoor environments.

There are eight indoor sites (I1 to I8) near the foundry and the other two sites (I9,I10) as outdoor air control. The locations are depicted in Figure I. The indoor concentrations ranged from 0.1 to 8.1 ng/m^3 during the study. The largest variations appear to be due to activities as smoking or painting in the house.

The average indoor BaP concentration for all sites is 1.18 ng/m^3 and six houses out of ten have indoor BaP concentrations higher than the average outdoor BaP concentration which is 0.9 ng/m^3. This means that not only is the foundry a BaP source, but there are also other sources in each individual house. According to questionnaire The average hours of participants spending indoor is about 15.

Figure 2 shows the BaP concentration by site through the study period. The range in concentrations was narrow for Homes 4,5,6,7 and

9. These either had no major indoor sources or seemingly in the case of Home 9, were affected by relatively constant emissions, i.e. the same number of cigarettes smoked per day.

For Home 1, there is one lady occupant who is a non-smoker average BaP concentration is low (0.9 ng/m^3). Nevertheless, it shows large variation in BaP levels. The reason is suggested in Figure 1: Home 1 is the site right in front of the foundry with the closest distance. The BaP concentration comparison between home 1 and outdoor site 101 (which is the outdoor sampler on the same side with home 1 and nearest) seem to have the same trend but while we examine the correlation between each other it dose not show a signifcant relationship. This probaly due to personal activity which affected the penetration.

For Home 10, there are high peaks of BaP concentrations at relatively high levels. The main reason appears to be a coal heating unit in the living room. Burning of coal appears to be a predominant source in this microenvironment.

For House 5, there is only an elderly couple who seldom cook or cook with a microwave oven. This seems to explain the observation that the concentrations are the lowest among the ten residences.

The average BaP concentrations of houses with smoker(s) and without smoker(s) are 1.5 ng/m^3 and 1.1 ng/mrespectively. This means that smoking is an important BaP source in indoor environments. If the concentration of 1.1 ng/m^3 (which is the average of six houses surrounding the foundry and one control house) can be taken as the baseline for an average house without a smoker and 1.5 ng/m^3 (which is

the average of two houses surrounding the foundry and one control house) can represent a average house with smokers, smoking contributes 27% of the BaP concentration to the indoor environment.

In comparing the indoor BaP levels in Homes 4,5,6 and 7 with the outdoor BaP levels we find indoor BaP concentrations appeared to be affected by outdoor BaP concentrations through penetration. If it is assumed that on all days when the BaP ratio of outdoor air to indoor air is less than 1 there are no 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. The data of the 2/2-2/3/87 sample days when an outdoor pollution episode affected the area is noteworthy. The BaP levels were 2.6 ng/m^3 at outdoor site 2. This levels was compared to the indoor levels for Homes 4,5,6 and 7. This outdoor site was within two blocks of each of these homes and all were located on the ridge above the foundry. The apparent penetration was 70,43,53,58% for Homes 4,5,6 and 7 respectively.

Food samples were acquired from family meals each day. Due to practical problems in the collection of food, some participants could only provide as little as 8 meals (of a possible table of 21) a week. The food sample represented a one-third portion of each meal eaten at home. The range of BaP per gram of wet weight of food was between 0.004 to 1.2 ng/g Table V-1. The results depend on particular foods and how they were cooked.

The BaP ratios of food ingested to inhaled are listed in Table V-2. Residents of House numbers 1,2 and 9 have ratios larger than 1

within the two weeks study period. Food ingestion is the predominant BaP source these houses. In house numbers 3,4 and 7, the ratios are smaller than 1. This means that inhalation of BaP is the predominant pathway in these houses. The remaining houses have one week in which the ratio is larger than 1 and the other week where it is smaller than 1. The differences between the ten houses depend on activities and life styles.

The BaP dose through food ingestion on a per meal basis is the BaP concentration in weekly food sample divided by meals. These data are listed in Table V-3.

Table V-3

The BaP concentration per meal

Home #	total BaP in two weeks period (ng)	# of meal	ng BaP/meal
1	1891	40	47.3
2	1682	42	40.1
3	130	32	4.1
4	132	17	7.8
5	395	36	11.0
6	886	37	24.0
7	124	24	5.2
8	770	34	22.7
9	5690	36	158.1
10	428	36	11.9

Appendix I indicates that BaP concentrations contributed through drinking water are negligible possible due to clean up through chlorination and filtration. When considering human intake of BaP through drinking water we still need further information concerning whether people take well water as a drinking water source or not and what BaP levels are in well water.

Inhalation Exposure Calculations for BaP in Indoor and Outdoor Air

Calculations of personal inhalation exposure to concentrations of BaP present indoors are based upon concentrations in that environment over a given 24 hour-sampling period and the amount of time a selected individual spent in the home during the same period. Since continuous data is not available, the BaP concentration is assumed to be constant in the home microenvironment over the entire 24 hours. In some cases this may bias the calculation, but it should be minimal since indoor sources are maximized while an

individual is in the home, e.g. smoking, cooking or stoking a coal or wood stove.

The time individual spent in the home was obtained from the activity questionnaire. From this information, the person who spent the most time in the home was determined and this was the person from whom food samples were accumulated for analysis and furthermore this person was selected for the following exposure calculations.

Calculation of indoor microenvironmental inhalation exposure was done as follows:

$$E_j = c_{ij} \times t_{ij}$$

where E_j = the indoor exposure for home j (ng/m^3-h)

c_{ij} = the microenvironmental indoor concentration on day i in home j

t_{ij} = the time spent on day i in home j

The value of E_j was then multiplied by an inhalation (ventilation) rate of 1 m^3/hr[8] to obtain an inhalation exposure E_j' where

$$E_j' = E_j \times v$$

v = ventilation rate

The results for each home are shown in Table VII

From the results of the household penetration for home 4,5,6 and 7, it is apparent that outdoor air affects indoor levels. Using the percentage penetration for these homes and an estimated 50% penetration for the remaining homes, and the concentration of BaP in PM-10 at the outdoor site nearest the home, an estimated inhalation exposure due to penetration of outdoor air can be calculated for each

day.

The calculation has the following form:

$$E_{jop}' = (c_{ik}) \times (t_{ij}) \times (v) \times (p_j)$$

where E_{jop}' = inhalation exposure from outdoor air penetration indoors

C_{ij} = outdoor BaP at site k nearest a home

P_j = penetration factor for home j

These results are also shown in Table VII. Subtraction of E_{jop}' from E_j indicated that, as would be expected from the activity questionnaire, Homes 2,8,9 and 10 were dominated by contributions from indoor sources, and homes 4,5,6 and 7 were influenced primarily by the infiltration of BaP from the outdoors. Spearman rank correlations for indoor BaP concentrations with outdoor BaP concentrations showed that for homes 4,5,6 and 7 the coefficients were >0.9 ($p=0.01$)[9].

Table VII Indoor Air Exposure to BaP during the Winter, 1987 THEES

Home #	Average Indoor Est. Inhaled BaP *ng/day	σ ng/day	Max. Exp. Day & Est. Inhaled BaP ng	Mean Hours Indoors	Week 1 Total Indoor BaP_T ng n = (samples)	Week 1 Est. from Outdoor Penetration **BaP_{OP} ng	Week 1 Est. Indoor Source $BaP_T - BaP_{OP}$ ng	Week 2 Total Indoor BaP	Week 2 Est. from Outdoor Penetration **BaP_{OP} ng	Week 2 Est. Indoor Source BaP ng
1	13.2	12.0	96	22.5	154.5(6)	47.2	107.3	106.8(7)	58.8	48
2	32.7	29.8	105	18.1	226.6(5)	36.2	186.4	136.7(6)	47.2	89.5
3	16.2	7.9	30	16.5	108.3(7)	66.3	42	86.1(5)	41.2	44.9
4	13.1	8.6	30	16.4	91.5(7)	75.5	16	92 (7)	82.8	9.2
5	9.9	8.0	25	20.7	72.4(7)	58.5	13.9	56.6(7)	48.9	7.7
6	12.3	10.1	27	19.8	81.4(7)	77	4.4	68.3(6)	47.	21.3
7	13.6	10.9	33	20.0	89.6(7)	87	2.6	101.2(7)	90.	10.2
8	39.3	37.7	144	19.6	282 (7)	73.2	208.8	269.4(7)	59.5	209.9
9	30.0	9.0	44	17.6	162 (6)	32.0	130.1	228.4(7)	61.6	166.8
10	34.9	53.5	190	15.1	89.9(6)	30.4	59.5	328.4(6)	46.9	281.5

* Assuming rate of $1 m^3/hr$ BaP ng = Exp x V = $\sum_i V_i \times C_i \times t_i$

** $BaP_{OP} = \sum C_{ij} \times t_i \times V_i \times P$

C_{ij} = Concentration at site j on day i,

t_i = time indoors on day i

v = ventilation

p = penetration coefficient

Exposure To BaP Through Outdoor Air

The arithmetic mean concentrations of BaP measured at all three outdoor PM-10 sites are shown in Table VIII. The values can be used to estimate the mean outdoor inhalation exposure for the residents in Phillipsburg. During the winter a reasonable estimation of average time spent outdoors is 1 h. According to questionnaire we estimate that the upper limit time of an individual exposed to outdoor air is one half of the non-home hours, which was about 4.5 hours. This upper limit would assume that an individual probably had participated in an outdoor winter activity. Since the time spent outdoors was not specifically measured in Phase 1, only the mean outdoor inhalation exposure per day can be calculated. The results showed an exposure ranging from 0.9 to 4.1 ng/day of BaP. In either case, the amount of direct indoor inhalation exposure is from 2 to 40 times the outdoor source exposure, or the indirect outdoor air exposure resulting from the penetration of BaP outdoors.

Table VIII Outdoor Air Exposures

Outdoor Site	Aver. Concentration ng/m^3	GM ng/m^3	Maximum Outdoor BaP ng/m^3	*Range of Outdoor Exposure ng/day
1	0.9	0.6	2.1	0.9 - 4.1
2	0.9	0.8	2.6	0.9 - 4.1
4	0.8	0.6	2.6	0.8 - 3.6

*Assume Inhalation of $1m^3/h$

Outdoor Exposure Range/day - Low estimation of 1 h outdoors

$$BaP = C_i t_i V = 0.9 \times 1 \times 1 = 0.9$$

High estimation 1/2 the highest non-home hours (average) for an individual

$$t_i = 1/2\ (24.-15.1) = 4.5 \text{ h outdoors}$$

GM = Geometric Mean

Exposure To BaP Through Indoor Air

In order to have an idea of human doses of BaP through different pathways and the percentage contribution of each pathway to the total dose, we take the average of the ten homes indoor air sample concentrations as a measure of contribution to total human exposure. The average is 1.18 ng/m^3. Of course this is not satisfactory for a statistical study since the sample size and the representativeness of the population is not sufficient. As mentioned earlier, these results only provide a first level-approximation of human exposure and a relatively simplistic calculation for quantitative risk assessment. This work needs much more high quality data before statistical support can be reliably employed.

Exposure To BaP In Food

The one-third adult portions of meals acquired each day from each home were composited as weekly sample for BaP analysis. In most instances at least 15 of the possible 21 meals per week were available for blending and compositing. The number of samples that were available per home are shown in Table V-3. The ng of BaP per g of wet weight food are listed in the same Table. The values ranged from 0.01 to approximately 1 ng per g of BaP. Examination of the questionnaire information did not clearly define the reasons for the variation in BaP found in the weekly composited food samples. This included the information from range and oven use, types of cooking and the types of food.

The number of grams of food sampled for each home were then multiplied by the appropriate BaP value (ng/g) and scaled up to a

complete adult portion. Adjustments were then made to account for water added to a sample prior to being blended as composites. The BaP measured in food for each week was then calculated using:

$$BaP_j = 3\ (BaP/g\ wet\ food)_j \times (wet\ g\ of\ food - added\ g\ H_2O)_j$$

Where: BaP_j = grams of BaP in food in home j

The actual exposure assumes that the BaP is fully absorbed in the digestive tract. Therefore, the ng of BaP for each home given in Table V-1 represents an estimated ingestion exposure to BaP over the course of the study.

There are big variations in ng of BaP per meal due to family eating habits and the way food is cooked. Thus, we take the median value as representative of the food contribution.

Median = (11.9 + 22.7)/2 = 17.3 ng

Since 17.27 ng is only a one-third portion of each meal and one assumes three meals per day, the total BaP dose through food ingestion is 3x3x17.3 ng = 155 ng of BaP/day

Human Dose Of BaP And Calculation Of Risk

For outdoor/indoor dose calculation:

dose(ng) = [concentration(ng/m^3) x inhalation rate(m^3/hr) x exposure time(hr)

*Inhalation rate for adult is 1 m^3/hr[8].

*Take 16 hr as indoor exposure duration according questionnaire from household.

* Take 4 hr as the outdoor exposure duration and assume there is no other BaP exposure for the rest 4 hour.

For food dose calculation, we take the average value as BaP ingestion and assume 100% absorption.

Result as follow:

Pathway	concentration	duration	dose(ng)	percentage
outdoor	0.90 ng/m^3	4 hr	3.60	2.03%
indoor	1.18 ng/m^3	16 hr	18.9	10.6%
food	155 ng/day	/	155	87.4%
			total: 178	

The percentage of BaP contributed through each pathway is shown in Figure IV.

The BaP intake through food pathway is 87% for average person. The overwhelming percentage due to the food pathway raises a very interesting question: "Why are we so concerned about air quality if we do not understand the importance of dietary contributions?" Additionally, "What kinds of food and what kinds of cooking processes contribute the major parts of BaP or BaP related compound?

Risk Calculation

In cancer risk calculation the most important factor is dose determination. In this case the residents lived in Phillipsburg have the daily dose of 2.54×10^{-6} mg/kg/day with the assumption average body weight of male is 70 kg (178ng/70kg/day = 2.54×10^{-6} mg/kg/day). The lifetime cancer risk is calculated as follow:

$$\text{Cancer Risk} = \underset{(\text{mg/kg/day})}{q^*} \times \underset{(\text{mg/kg/day})}{\text{dose}}$$

q^*: carcinogenic potency which is the slope of dose response relationship for specific carcinogen[10]

for BaP $q^* = 11.5$ (mg/kg/day)

The cancer risk turn out to be 29.2×10^{-6}. The number (29.2×10^{-6}) means that a person who is exposed to a BaP dose concentration at 2.54×10^{-6} mg/kg/day for 70 years, the chance to develop cancer is 29.2 out of one million. This number depends on many facts like the experimental process and design, exposure assumptions and toxicological information. It also clearly ignores synergistic factors. Even with so many uncertainties in the process of risk determination still can be as an index number identify and rank the potency for a pollutant if no accurate epidemiology information is available. Comparing the risk with Table IX BaP and PAH represented by BaP did reach a concerning risk level in exposure. The cancer risk of exposure to BaP is higher than the risk of home accidents which is 1/1190 lifetime risk. As for the creditability of the cancer risk we estimated today only the pathological study in Phillipsburg can verify and also modify the exposure model.

Figure III.

The average percentage of BaP

contributed through each pathway

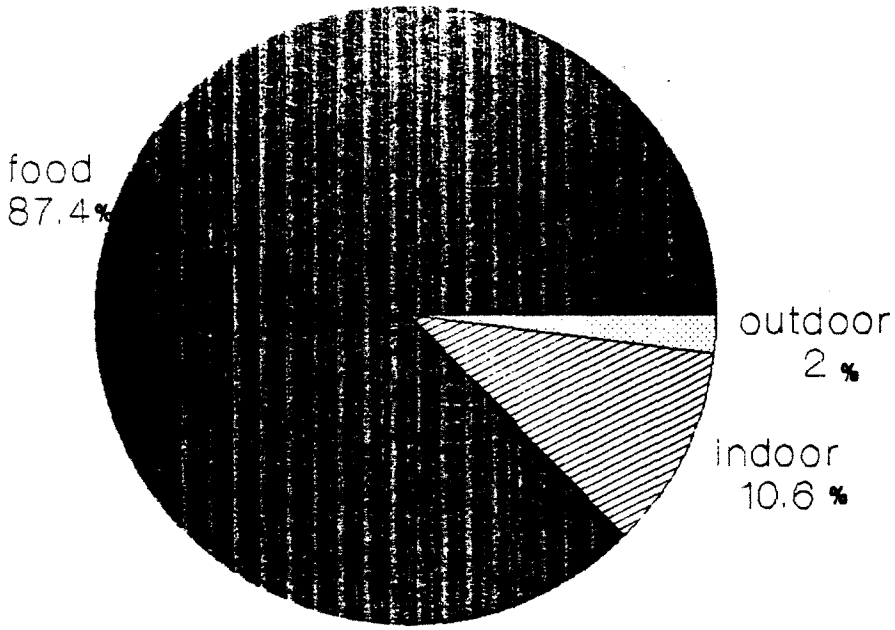


Table IX

Annual Risk of Death from Selected Common Human Activities[1]

	Number of Deaths in Representative Year	Individual Risk/Year[2]		Lifetime Risk[3]
Coal Mining:				
Accident	180	1.30×10^{-3}	or 1/770	1/17
Black Lung Disease	1,135	8×10^{-3}	or 1/125	1/3
Motor Vehicle Accident	46,000	2.2×10^{-4}	or 1/4,500	1/65
Truck Driving Accident	400	1×10^{-4}	or 1/10,000	1/222
Falls	16,339	7.7×10^{-5}	or 1/13,000	1/186
Home Accidents	25,000	1.2×10^{-5}	or 1/83,000	1/1190

1 Selected from Hutt (1978). Food. Drug. Comestic Law J. 33:558-589.

2 Risk is often represented as a negative exponent such as 2×10^{-5}; that expression in scientific units is easily converted to a more conventional expression by replacing the "x" multiplication sign by "per" or a division sign and changing the exponent to the appropriate number of integers. For example. 2×10^{-5} is equivalent to 2 per 100,000 or 2'100,000.

3 Estimated based upon 70-year lifetime and 45-year work exposure.

Taken from: Elements of toxicology and Chemical Risk Assessment. Environ Corporation. 1986.

Conclusion

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.

A major focus of this study is linking personal as well as indoor and outdoor environmental activities to the variations of BaP found in the home. The mean numbers of hours spent at home for all individual participating in the study was 16 hour (66%) for the day.

Each of the participating homes had activities during the two weeks study which affected the indoor BaP levels found in the PM-10 samples. These are related either to an indoor source, or the penetration of outdoor air. Table I and Table II summarizes the main sources and activities that occurred in the homes during the two weeks of study. Besides major sources, there are cases where individual high indoor levels were will above the outdoor ambient concentrations due to specific indoor activities (e.g. scraping paint from walls or burning food). In Home 8, paint scraping took place on three days during the study. A scatter diagram of the PM-10 concentrations Figure IV, showed very high excursion indoors on three days because of this activity(point A and B indicated in Figure IV). Another difference observed in the study was that Home 3, which had smokers, was highly correlated with the median outdoor PM-10 (ranked Spearman coefficient of 0.73).[6] From the daily questionnaire, it was disclosed that was a very active family with many people coming to and leaving the home during the day. Therefore, substantial penetration of outdoor air was possible.

Further studies in other seasons are necessary to obtain a clearer understanding of the nature of BaP in the indoor environment.

Since we could not measure the concentrations present in potential air microenvironments other than the home and ambient air, an estimate of daily inhalation of exposure needs to account for the hours not spent at home. By using ambient BaP as the minimum concentration which a person encounters, the remaining BaP exposure can be estimated. With this assumption comparison of the two weeks of data for 24 hours inhalation exposure each day and food exposure, shown in Table V-2 present an interesting result. The paired BaP exposures 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 BaP exposure, and ten had higher inhalation BaP exposure. In the instances with greater BaP exposures due to the food pathway, the values were at least two times greater than contributed by inhalation. In the other ten cases, the maximum value of BaP were lower, but the relative inhalation exposures were more greater than two times the food exposure in most instances.

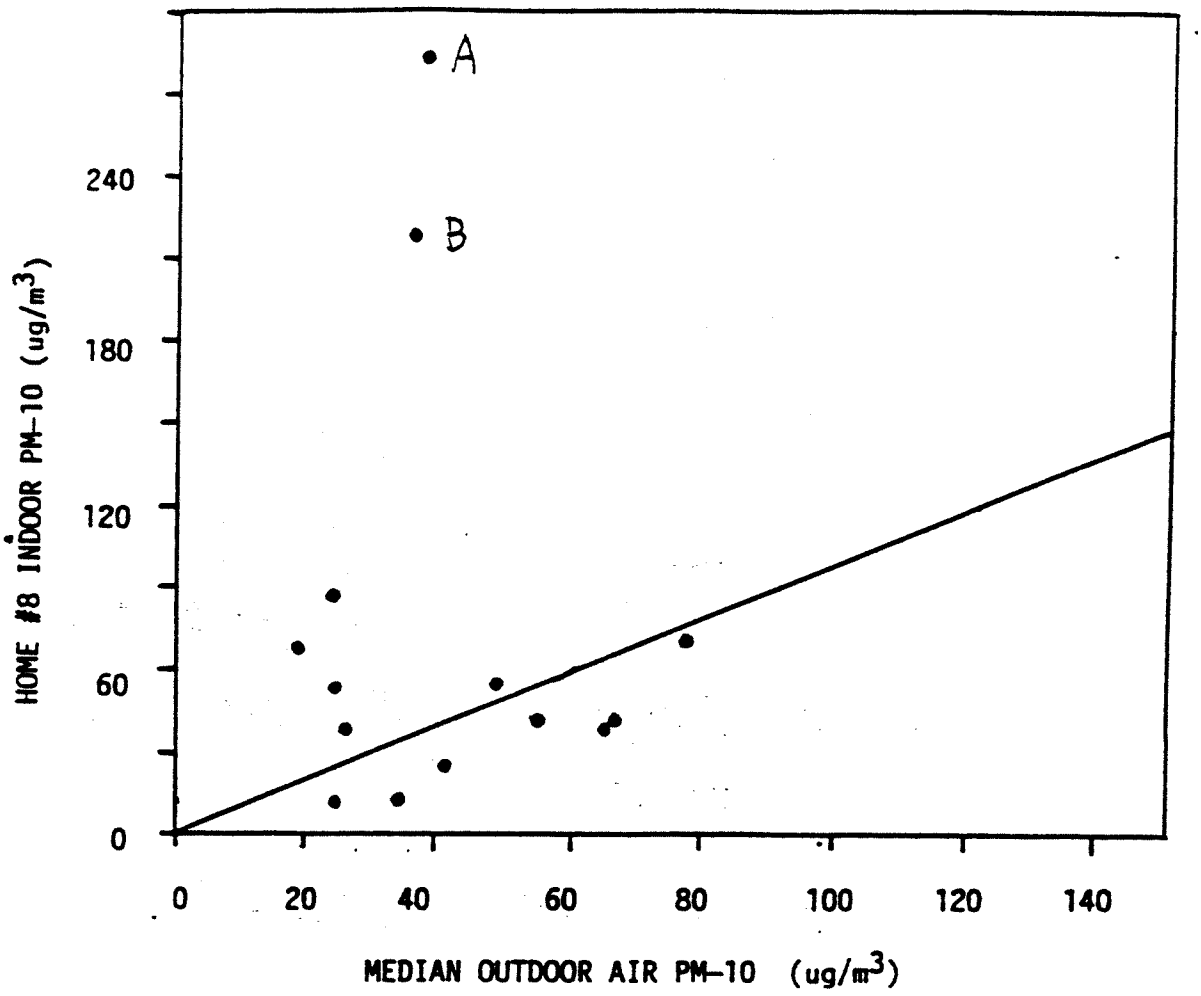
The individual differences appear to depend upon personal eating habits, and the personal life styles for use of the indoor combustion products. The infiltration of outdoor air to the indoors led to significant exposure in some cases.

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[11,12]. These particles penetration the nasopharyngeal and bronchiolar regions and deposit in the alveolar

region. This may be one of the reasons bioavailable concentrations are different from the concentration we detected in the air.

This report is thus only a designing in total human exposure studies. We have tried to measure environmental concentrations of pollutants available through different pathway 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 direct to human exposure. Afterward we can compare biological dose with statistic epidemiological data in the area to modify the model we approach and to assess the risk of human exposure. Maybe someday we can quantitatively answer the question like "What is the risk of person live in that environment? or will the foundry produce a problem to human health?".

FIGURE IV Scatter Diagram Of Outdoor PM-10 with Indoor PM-10 For Home #8 During the Winter, 1987 THEES



Appendix I

I. Limit of detection and limit of quantification

If the blank for BaP do appear blank as we find from past experiment, then we will use the minimum integration area our measure of random noise and our limit of detection will be three this value and our limit of quantification, ten times this value (Anal. Chem. 1980, 52, 2242-2249). The limit of detection under the instrument specific before is 1 ng/10 ul of BaP which generate 1/3 signal to noice ratio.

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PART B: ASSESSMENT OF BENZO(A)PYRENE (BAP) EXPOSURE THROUGH URINE ANALYSIS WITH THE HYDRIODIC ACID REDUCTION REACTION

PART B

Chapter V INTRODUCTION

In the first part of this thesis, the assessment of human exposure to BaP through different pathways including air, food and water was described. The nature of BaP sources in Phillipsburg, New Jersey and the roles of different exposure pathways are discussed. The first section summarizes the environmental concentrations of BaP determined in our study. This part of the thesis describes our efforts to develop and assess an analytical method to determine the concentration of BaP metabolites in human urine of selected residents of Phillipsburg.

Epidemiological studies have shown that high exposure is not necessarily reflected in occupational cancer in industrial settings as those found in the aluminum industry[1-3]. Hence, high environmental concentrations of pollutants need not translate into high bioavailable concentrations in human. It is therefore necessary to monitor real exposure to and uptake of pollutants. The ideal biological monitoring method would permit direct measurement of a toxic agent in the target organ, but the problem of "invasive" sampling on a routine basis makes this approach impractical. Therefore, feces and body fluids, mainly urine and blood, are generally used for biological monitoring. The study by Becher et al. show that the PAH metabolite levels in feces are almost 10 times higher than in urine.[4] However, feces collection is much more "invasive".

Through the study of total human exposure we can better understand the characteristics of each pathway that contributes to the

bioconcentration. An analytical method was developed and limited experimentation has been done to evaluate this method.

Chapter VI SAMPLE PREPARATION AND METHODOLOGY

An analytical procedure based on the reduction of excreted oxidized metabolites of BaP back to the parent hydrocarbons was used[4]. The analytical procedure included extraction of BaP and BaP metabolites from urine using C_{18} Sep-Pak cartridges[5], reduction of metabolites to PAH by refluxing hydriodic acid ("reversed metabolism") and subsequent analysis by thin layer chromatography (TLC) using selective and sensitive fluorescence detection. This method was modified from the report by Becher and Bjorseth[4], based on the reduction reaction using hydriodic acid, developed by Konieczny and Harvey.[6] Whereas, Becher and Bjorseth determined by GC a series of PAH ("reducing" a problem of ca. 1000-2000 metabolites to 100-200 PAH)[7], we are further simplifying the analysis to metabolites of BaP (and thus BaP analysis only). Becher and Bjorseth[1] did not investigate the ability of different classes of PAH metabolites to reduce to the parent hydrocarbon with HI. Konieczny and Harvey[6] only investigated hydroxy-and methoxy-PAH and PAH-quinones. We have undertaken limited investigation of specific classes of oxygenated PAH.

Sample

Urine samples were provided by Professor Regina Santella, Columbia University School of Public Health, from patients with high dermal exposure under Goeckerman therapy[8] (psoriasis cure with coal tar: known to contain high concentrations of BaP); control samples were provided by NJIT students without exposure to coal tar therapy. Confounding factors such as smoking, education, and age were not

considered.

Procedure of Urine Analysis

A urine sample (100 ml) adjusted to pH=3 was filtered through a 1.2 um membrane filter and passed through the activated (methanol drawn through prior to use) C_{18} Sep-Pak cartridge at approximately 10 ml/min in order to separate PAH metabolites from the urine sample. Metabolites were eluted by pumping 5 ml methanol through the cartridge. The solvent was evaporated and the residue refluxed with 1 ml 57% hydriodic and 15 ml glacial acetic acid for 15 hours[6]. The hot solution was poured into 80 ml of 1% aqueous $K_2S_2O_7$ solution and extracted twice with 40 ml and 20 ml cyclohexane. The organic phase was washed with water and dried with Na_2SO_4. The extract was concentrated to 2 ml under nitrogen; a 250 ul aliquot was then spotted on an acetylated cellulose thin layer chromatography (TLC) plate. The TLC plate was developed using 2:1 ethanol/dichloromethane and analyzed spectrofluorometrically via plate scanner at 387 nm (excitation), 428.6 nm (emission).

Chemical and Apparatus

C_{18} Sep-Paks were purchased from Waters Associates (millipore Corp), Milford, MA. The plate-scanning spectrofluorometer was described in Part A. Acetic acid (Gold Label) was purchased from Aldrich Chemical Co, Inc. U.S.A. Cyclohexane and Hydriodic Acid 57% were purchased from J.T. Baker Chemical Co, Phillipsburg, New Jersey. Potassium disulfate was purchased from Fluka Chemical Corp., Ronkonkoma, New York. All solvents are HPLC grade.

Chapter VII RESULT AND DISCUSSION

RECOVERY

The procedure included three parts basically: cartridge separation, reflux (reduction reaction) and extraction. The recoveries for three different tests are:

C_{18} cartridge -------- 44.0% recovery

reflux and extraction -------- 49.1% recovery

whole procedure -------- 22.7% recovery

* Recovery test was done by using BaP standard as sample and detecting on each substep.

Our C_{18} Sep-Pak cartridge recovery is 44.0% of a BaP standard. According to the literature the recovery is 41%.[1,7] The recovery of the steps involving reflux and extraction by cyclohexane totals 49.1%. This gives us a picture of total recovery of the BaP standard ideally (based on separate steps): it is 44.0% x 49.1% = 21.6%. When using the BaP standard run through the entire procedure the recovery is actually 22.7%, slightly higher than 21.6%. The numbers are identical within the error limits. We find future a 98.0% recovery when solvent exchange methanol to cyclohexane is done in the procedure. From the data above, there is no significant loss of BaP in other steps. However, we have not yet assessed the efficiency of a) hydrolysis of conjugates and b) the efficiency of the reduction with HI. This is also a shortcoming of the Becher and Bjorseth investigation. These factors undoubtedly account for the relatively low total recoveries for the standards reported below.

Another recovery experiment was done by using urine from a

mouse injected with C^{14}-BaP. The average of duplicate analyses is 8.02% recovery based on radioassey of the urine (The mouse urine and radioassay were supplied by Professor Santella). Note that using our previous results, the % recovery of the hydrolysis and reduction steps can be calculated as follows, X = 0.0802/0.227, indicating a 35% recovery. This also infer the fact that probably there are only 35% of the BaP metabolites can be reduced to parent compounds by the HI reaction.

Table 8.1 indicates that Goeckerman therapy did expose the patient to high absorption of BaP. Relative BaP concentrations are deduced from comparison of levels in samples. In the analytical method itself, reduction reactions by hydriodic usually increase mean BaP values by about four to ten-fold. The ideal recovery of the method as mentioned above is 21.6% without including recovery from hydrolysis and HI reduction. The low (ca. 8%) recovery of the radiolabelled metabolites has two main origins. First, not all of the metabolites of BaP reduce with HI to BaP. We will address this topic below. Second, the percent recovery of hydrolysis and HI reduction for those compounds undergoing reduction have not yet been measured. The following steps may be applicable to improve recovery:

1. Use Beta-glucuronidase instead of HCl to break down conjugates of BaP metabolites.
2. Try other cartridges which can retain BaP and BaP metabolites with higher recovery than C_{18} (41%).
3. The light sensitivity of BaP is cause for rigorou protection from light throughout the whole process; low flow rate for

cartridge and enough time to reach equilibrium during extraction may also help.

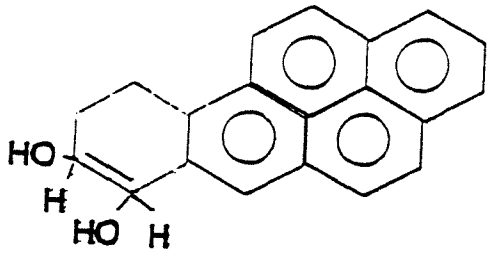
Human Urine Sample

Sample	Description	BaP in urine ug/l	BaP in urine with reduction ug/l
Control 1	NJIT student without significant expose to BaP	0.021	0.085
		0.019	0.047*
Control 2	as above	0.016	0.102
CT6	Patient under Goeckerman	0.146	0.740
CT5	as above	0.164	0.853
Literature[7]	exposed worker in aluminum factory	0.010	0.130

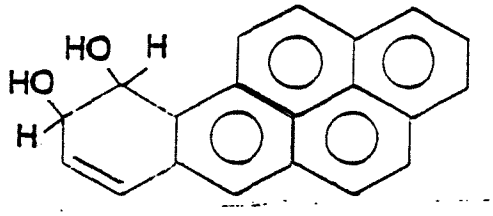
* same student two trials

In order to investigate the recovery of specific metabolites using the HI reaction, we have investigated the recovery of 7,8-dihydrobenzo(a)pyrene 7,8-diol (1) and 9,10-dihydrobenzo(a)pyrene 9,10-diol (2). For the 7,8-dihydrodiol subjected to different duration of hydrolysis experiment recoveries following 5 min and 30 min hydrolyses were, respectively, 0.64% and 0.71%. The recovery of 9,10-dihydrodiol is also very low (0.3%) even with longer duration of hydrolysis. This means that there is no difference in recovery whether the duration is 5 min or 30 min and HI cannot reduce these dihydrodiols to BaP. The study of Konieczny and Harvey investigated only reduction of polycyclic quinones, and hydroquinones, phenols and alkoxy-PAH to their parent compounds. There is no claim for compounds in the dihydrodiol class. It is interesting that dihydrodiols are estimated to comprise almost 50% of human BaP

metabolites. This gives recovery at the reaction stop to under 50%. Furthermore, 1-hydroxy benzo(a)pyrene has been estimated at about 35% consistent with our recovery at this stage.[9]



(1)



(2)

The actual measurement of PAH metabolites in human excretion offers advantages over conventional exposure estimation, which merely determines the amount of BaP in the individual's immediate environment:

1. It may provide better understanding of the degree of inter-individual variation that in uptake and metabolism of BaP in humans.
2. It may provide a measure of the individual dose or biologically effective dose of BaP in the organism and thus allow a better prediction of human risks associated with exposure to BaP in different environments.
3. The articular procedure we have introduced is highly specific for BaP and much more rapid than GC determination.

Since BaP or BaP metabolites can be excreted in feces, urine and from lung[10-12] mere monitoring of the concentration of BaP in urine cannot provide a measure of absolute bioconcentration in the human body. Due to practical obstacles in sample collection of feces

and exhalation samples, we limited ourselves to samples which are easier to obtain. BaP studies on animals indicate that there is a connection between exposure to BaP and excretion of BaP metabolites through urine after one to six hour metabolism lifetime[10,11]. Hence, the BaP levels in human urine can provide a better understanding of real exposure in the human body and help to characterize each exposure pathway.

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