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A Study of Environmental Exposure to PAHs among Economically Underprivileged Population of Urban Areas of Uttar Pradesh

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A STUDY OF ENVIRONMENTAL EXPOSURE TO PAHs IN ECONOMICALLY UNDERPRIVILEGED POPULATION OF URBAN/RURAL AREAS OF UTTAR PRADESH

(Project #P129)

Final Report

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1.0 INTRODUCTION, BACKGROUND AND PROBLEM STATEMENT

Polycyclic Aromatic Hydrocarbons (PAHs) are one of the hazardous (carcinogenic) contaminants of the present ambient air environment. It is a group of 2-3 ring or >3 ring cyclic compounds and a well-known class of carcinogens.

Ambient air pollution due to PAHs, both in rural and urban areas, is an environmental health hazard and is largely due to emissions from autoexhaust or fuel biomass combustion. The gasoline/diesel exhaust emissions constitute the most important source of PAH pollution in urban India. Busy traffic channels are one of the worst polluted sites. A considerable number of subjects su0ch as road-side venders, traffic constables and controllers, cycle riksha or trolley pullers and daily commuters in these areas get exposure to PAHs everyday.

Emission of fuel biomass is the source of PAHs pollution in rural India. Biomass is a very common type of non-fossil fuel that is used frequently as an inexpensive source of energy for cooking food in rural parts of India. In winter months, it is also used as a bonfire to warm the house. Emissions from such practices form the single largest source of PAHs pollution in rural ambient/indoor air. The women folk get relatively more exposures to PAHs specially while cooking food for extended period of time particularly due to low energy value of the biomass fuel.

No study is available reporting concurrently the site-specific environmental load of pollutants and an exposure assessment of population dwelling specifically in PAHs polluted urban traffic channels, rural huts and the vicinal areas in India. In literature, only sporadic studies, reporting the indoor air PAHs profile in smoke emissions of fossil fuel or non-fossil biomass fuel, were spotted **(Raiyani et al., 1993, Pandit et al 2001, Venkataraman et al 2002, Kulkarni and Venkataraman 2002).** However, there is a complete paucity of information on exposure & health risk assessment of Indian urban/rural subjects exposed to PAHs in India.

Therefore, a need was visualized to (a) analyze total PAHs contents and its types vis-à-vis other criteria air pollutants in indoor/ambient air samples collected from sites around

the wood/refuse burning hearths and stoves in rural India or the busy traffic channel in urban India and (b) to undertake PAHs exposure assessments of subjects living/working near polluted sites both in urban & rural areas. The advent and availability of biological marker viz. urinary 1-hydroxypyrene provided a potential means of assessing environmental exposure specifically to PAHs.

Herein, we report (a) the analysis and quantification of total PAHs content and its profile in air samples from urban/rural hot spots of the city of Lucknow, (b) a spectrum of PAHs exposure assessment in subjects living/working near PAHs polluted sites and (c) assessment of prevalent morbidities in PAHs exposed population. There is a lack of human data on these aspects. The study was restricted to a total of 384 adult subjects dwelling specifically in PAHs polluted traffic channels in urban areas or the PAHs polluted huts in rural areas. The study was limited to a 100-km. radius of Lucknow. Till date, no body has attempted such kind of study on environment & health risk assessment with economical endpoints. A cross-sectional study design was chosen for its inherent benefits of (a) low cost, (b) quick results and (c) logistic feasibility. It provides valuable information for planning and execution of more detailed studies to answer specific policy problems.

1.0.1 OBJECTIVES

1. To determine PAHs concentrations in urban ambient/rural indoor air environment
2. To assess the potential risk of adverse health effects in the PAHs exposed subjects
3. Economic evaluation of health damages caused by exposure to PAHs

Environmental Exposure to PAHs

2.0 SITE SELECTION & MONITORING

Only the ambient/indoor air of High Traffic Density & Low Traffic Density sites of urban part and fuel biomass using site of the rural part of Lucknow were monitored. The sampling was done in two seasons, viz. summer & winter and as per plan shown in **Table 1&2**. For the study, summer was defined as the period of March through June and winter as period of November through February in this part of the country.

2.0.1 AMBIENT AIR MONITORING

The ambient air was sampled using respirable dust sampler. The monitoring plan and its characteristics are described in detail in **Table 1&2.** Sampling was done at three different locations of the city **(See Annexure III)** and in two seasons viz. summer (June 2001) & winter (December2001/January & February 2002). Two of the monitored sites were High-Traffic Density (HTD) site (site #1 Charbagh & site #2 Hussainganj) and one was Low-Traffic Density (LTD) site (viz. Janakipuram). The HTD sites were the busiest traffic channels of the city with idling or slow moving traffic. The LTD site was scarcely busy with relatively fast moving traffic. For ambient air monitoring, samples were collected 2X/wk for four weeks except at one site in winter where it has been done continuously (due to an inevitable local problem). The period of one month as a representative of the season was not the aim. A sum of twenty-four samples were collected and analyzed from all three sites in one season **(Table 1)** and a total of forty-eight in two seasons. The parameters monitored were SPM, RSPM, SO_2, NOx and PAHs **(Table 2)**. Levels of SPM, RSPM, SO_2 & NO_x were monitored to identify the levels of criteria air pollutants at the time of study of PAHs in the environment. Further the proposed study of health effects needed the information, as the health effects are the net result of a complete exposure not only to PAHs but also to criteria air pollutants plus the other toxic air pollutants.

2.0.2 INDOOR AIR MONITORING

The indoor air was sampled at locations in Village-Mall (Tehsil-Malihabad, District-

Lucknow)**,** approximately 40 km NW of Lucknow City **(See Annexure IV).** Rural huts with single room accommodation and of similar volume were selected. The monitoring plan and its characteristics are described in detail in **Table 1& 2**.

The types of fuel used in this area were Jalauni Lakadi (Wood), Cow Dung Cake (CDC) and Liquid Petroleum Gas (LPG). The kerosene and electric heaters were not prevalent in these areas. Agricultural waste was also not found in use during survey. The cooking areas and the smoke-less non-cooking areas were monitored. The sampling was done at the time of cooking and only for an hour. The concentration of PM collected could have got diluted if sampling was done for 24h period. Sampling was done using both Personal/Area Samplers, which is a standard practice. RSPM, SO_2, NO_x and PAHs were monitored in indoor air. Only RSPM & PAHs were monitored both for active or passive exposure. A sum of 80 samples was collected and analyzed in one season and a total of one hundred and sixty in two seasons **(Table 1&3)**.

3.0 MATERIAL & METHOD

The methods are summarized in **Table 4&5**. Filters with sampled particulate matrix were transported to laboratory for processing under the protection of light and in vacuum dessicator.

3.0.1 AMBIENT AIR QUALITY MONITORING

3.0.1.1 SPM & RSPM

A known volume of air was passed through pre-weighted glass fiber filter paper (GF/A) of size 8 x 10". Centrifugal force of cyclone acted on the dust particles and separated it into two parts.

(I) Particles <10μ collected on filter paper

(ii) Particles >10μ collected in cyclone cap.

The difference in initial and final weight of filter paper and cyclone cap represented RSPM (<10μ) and non-RSPM (>10μ) fraction respectively. Dividing gain of weight in filter paper by the total volume of air sample yielded RSPM content. Cyclone dust and filter dust together represented SPM. Details of used methods and their detection limits are described in **Table 4.**

3.0.1.2 Sulphur Dioxide (SO_2)

A known quantity of air was passed for 8 hours through an impinger containing known quantity of SO_2 absorbing solution; sodium tetrachloromercurate (TCM). The SO_2 absorbed solution formed stable colored complex of di-chloro-sulphito-mercurate with p-rosaniline hydrochloride. A_{560} was estimated at spectrophotometer for which a calibration curve had already been drawn **(IS: 5182/Part II, 1969 Reaffirmed 1989)**. Details of used methods and their detection limit are described in **Table 4**.

3.0.1.3 Oxides of Nitrogen (NO_x)

A known quantity of air was bubbled for 8 hours through an impinger containing known quantity of NO_x absorbing solution (sodium hydroxide). It formed a stable solution of sodium nitrite. The nitrite ion thus produced was allowed to react with phosphoric acid, sulphanilamide and NEDA (N-1 Naphthyl-ethylene-di-amine di-hydro-chloridele) to yield a colored complex. A_{540} was determined colorimetrically **(IS: 5182/Part VI; 1975 Reaffirmed 1992)**. Details of used methods and their detection limit are described in **Table 4**.

3.0.1.4 PAHs

Extraction

The dried filter paper containing the particulate air sample was put in extraction thimble (filter paper Whatman No. 1) and placed in a Soxhlet extractor. Samples were extracted in methylene chloride for 16 h at the rate of 4 cycles per hr under protection from light. The extract was concentrated upto 1 ml in rot evaporator under reduced pressure.

Clean-up

The extract was solvent exchanged to cyclohexane before the silica gel column cleanup. This was done by nitrogen flushing in warm water. The slurry of 10g activated silica gel in methylene chloride was prepared and placed into a chromatographic column (I.D. 10mm). The column was tapped to settle the silica gel and 1g of anhydrous sodium sulphate was then added at the top of silica gel. The methylene chloride was eluted from the column. The column was pre eluted with 25 ml of n- pentane at an elution rate of about 2 ml per min. The eluate was discarded. Just prior to being eluted dry, 1ml of sample extract in cyclohexane was loaded on the column. An additional 3 ml cyclohexane was used to complete the transfer. Again just prior to being eluted dry, the column was washed with 25 ml of n-pentane without disturbing the upper layer. The pentane elute was discarded. The PAHs adsorbed on the column were eluted with 25 ml of methylene chloride/n-pentane mixture (2:3 v/v) in a conical flask **(EPA, 1986)**. The

eluate was flash-evaporated to 1ml and solvent exchanged with 1 ml Acetonitrile. The sample was stored at 5°C and under the protection from light till analysis.

Analysis by HPLC

A reverse phase HPLC setup with gradient elution technique was used. A non-polar absorbent octadecyl silicate coupled with a non-polar solvent was used to separate the non-polar compounds. The solvent composition of Acetonitrile and water in the ratio of 40:60 up to 100 % of Acetonitrile was used. The gradient solvent flow was 1 ml/min with the run time of 55 min. A 10µl volume of the sample was injected and the column was kept at 27°C. The ultraviolet (UV) detector of HPLC was set at 254 nm (William et al., 1988). The retention time, percent recovery and minimum detection limit of the analyzed PAHs are presented in **Table 6&7**. The detection limit ranged from 0.002ng (anthracene) to 0.5 ng (acenaphthylene). The percent recovery of all the investigated types of PAHs ranged from 61.5 to 83% with the exception of acenaphthylene and acenapthene registering a lower recovery value. Low levels of recovery in case of the two may be attributed to their volatile nature. The chromatograms of reference standards of PAHs, rural indoor air and urban ambient air samples are presented in **Figure 1–3** respectively. The peak area of each PAH type was recorded from HPLC chromatograms and quantified as described below.

PAHs specific to autoexhaust/biomass smoke emissions (as listed in **Table 6**) were analyzed. It included volatile PAHs (acenaphthylene, acenaphthene), particle bound PAHs (chrysene, benzo(a)pyrene, benzo(ghi)perylene) plus PAHs occurring in gas as well as in particle bound form (phenanthrene, anthracene, fluoranthene, pyrene).

Calculations

The PAHs concentration was calculated as following:

$$\text{Conc. } (ng/m^3) = \frac{C_{std.} \times A_1}{R_f \times A_{std.} \times V_s}$$

Where,

C_{std} = Concentration of a compound in the standard mixture

A_1 = Area of sample peak

R_f = Recovery factor

A_{std} = Area of standard peak

V_s = Volume of air sample (in m^3) at STP.

3.0.2 INDOOR AIR QUALITY MONITORING

3.0.2.1 RSPM

Both personal and area samplers were employed to collect the respirable dust. The aim was to monitor both active & passive exposure. Samples were analyzed according to NIOSH protocol 0600 **(Table 5).** Personal sampler was attached to the chief cook with a belt and samples collected to monitor the active exposure. Low volume area sampler was placed at an obstruction free place in the cooking area and in the breathing zone. It monitored the passive exposure. Samples were collected using 10mm nylon cyclone equipped with 37mm diameter and 25 mm diameter glass micro fiber (GF/A) filters. The flow rates for each were 1.9 liter/minute. Air was drawn through the cyclone pre-selectors using battery operated constant flow pumps of the Make Casella, Millipore and Rotheroe and Mitchell Ltd. All pumps were calibrated using an electronic flow meter on the field that was in turn calibrated using a mini buck soap bubble meter in laboratory before and after sampling. All filters were conditioned for 24 hours before weighing. Respirable dust concentrations expressed in terms of mg/m^3 and were calculated by dividing the blank corrected filter mass increase by the total volume sampled according to following formula.

$$C = \frac{(W2-W1)}{V} \times 10^6$$

Where,

W1 = Initial weight in g

W2 = Final weight in g

V = Sample volume in liter

C = Concentration in mg/ m^3

3.0.2.2 Sulphur Dioxide (SO_2) and Oxides of Nitrogen (NO_x)

Only area samplers were used for gas monitoring. Samples were collected at the time of cooking. Details of used methods and their detection limit are described in **Table 5**.

3.0.2.3 PAHs

The samples were collected & processed as described in section **3.0.1.4**

4.0 RESULTS

4.0.1 URBAN AMBIENT AIR POLLUTION

4.0.1.1 SPM

In summer

The SPM levels at HTD site #1 & #2 ranged from 110.75-224.00 μg/m^3 and from 100.20 to 182.91μg/m^3 respectively. At LTD site, the concentration ranged from 94.40–186.76 μg/m^3. The average concentration of SPM at all the three location was found to be 159.60 μg/m^3, 153.35 μg/m^3 and 137.54 μg/m^3 respectively **(Table 8-10 and Figure 4)**. The results were compared with National Ambient Air Quality Standards (NAAQS) values. It was found that the SPM levels at all the monitored locations were close to or within the permissible limit for residential area **(Annexure-I)**.

In winter

The SPM levels at HTD site #1 & #2 ranged from 273.22-447.50 μg/m^3 and from 189.29-465.32 μg/m^3 respectively. At LTD site, the concentration ranged from 113.98-306.03μg/m^3. The average concentration of SPM at all the three location was found to be 361.86 μg/m^3, 332.82 μg/m^3 and 222.61 μg/m^3 respectively **(Table 11-13 and Figure 4).** The results were compared with NAAQS values. Unlike summer, the SPM levels at all the monitored locations exceeded the permissible limit for residential area **(Annexure-I).**

4.0.1.2 RSPM

In summer

The RSPM levels at HTD site #1 & #2 ranged from 86.60-114.27 μg/m^3 and from 82.23

to 116.91 μg/m^3 respectively. At LTD site, the concentration ranged from 67.29 –77.58 μg/m^3. The average concentration of RSPM at all the three location was found to be 100.37 μg/m^3, 96.37 μg/m^3 and 72.98 μg/m^3 respectively **(Table 8-10 and Figure 4).** The results were compared with NAAQS values. It was found that the RSPM levels at all the monitored locations were close to or within the permissible limit for residential area **(Annexure-I, II)**.

In winter

The RSPM levels at HTD site #1 & #2 ranged from 237.36-364.60 μg/m^3 and from 130.45-349.49 μg/m3 respectively. At LTD site, the concentration ranged from 83.61-258.69 μg/m3. The average concentration of RSPM all the three location was found to be 288.67 μg/m^3, 264.32 μg/m^3 and 165.22 μg/m^3 respectively (**Table 11-13 and Figure 4)**. The results were compared with NAAQS values. Unlike summer, the RSPM levels at all the monitored locations exceeded the permissible limit for residential area (**Annexure-I, II**).

4.0.1.3 Sulphur Dioxide (SO_2)

In summer

The SO_2 levels at HTD site #1 & #2 ranged from 15.53 -17.00 μg/m^3 and from 16.48 to 16.96 μg/m^3 respectively. At LTD site, the concentration ranged from 14.59 – 15.94 μg/m^3. The average concentration of SO_2 at all the three location was found to be 16.12 μg/m^3, 16.71 μg/m^3 and 15.21 μg/m^3 respectively **(Table 8-10 and Figure 4).** The results were compared with NAAQS values. It was found that SO_2 levels at all the monitored locations were within the permissible limit for residential area **(Annexure-I).**

In winter

The SO_2 levels at HTD site #1 & #2 ranged from 16.68-17.99 μg/m^3 and from 16.37-19.59 μg/m^3 respectively. At LTD site, the concentration ranged from 14.36-15.89

$\mu g/m^3$. The average concentration of SO_2 at all the three location was found to be 17.33 $\mu g/m^3$, 18.46 $\mu g/m^3$ and 15.10 $\mu g/m^3$ respectively **(Table 11-13 and Figure 4)**. The results were compared with NAAQS values. Like summer, SO_2 levels at all the monitored locations were within the permissible limit for residential area **(Annexure-I).**

4.0.1.4 Oxides of Nitrogen (NO_x)

In summer

The NO_x levels at HTD site #1 & #2 ranged from 20.60-25.45 $\mu g/m^3$ and from 20.97 - 21.99 $\mu g/m^3$ respectively. At LTD site, the concentration ranged from 17.00-17.97 $\mu g/m^3$. The average concentration of NOx at all the three location was found to be 23.63 $\mu g/m^3$, 21.54 $\mu g/m^3$ and 17.34 $\mu g/m^3$ respectively in **(Table 8-10 and Figure 4).** The results were compared with NAAQS values. It was found that the NOx levels at all the monitored locations were within the permissible limit for residential area **(Annexure-I)**.

In winter

The NO_x levels at HTD site #1 & #2 ranged from 24.32- 27.81$\mu g/m^3$ and from 26.73-28.38 $\mu g/m^3$ respectively. At LTD site, the concentration ranged from 19.46-22.78 $\mu g/m^3$. The average concentration of NOx at all the three location was found to be 23.36 $\mu g/m^3$, 24.45 $\mu g/m^3$ and 20.61 $\mu g/m^3$ respectively (**Table 11-13 and Figure 4)**. The results were compared with NAAQS values. Like summer, NO_x levels at all the monitored locations were within the permissible limit for residential area **(Annexure-I)**.

4.0.1.5 PAHs

In summer

The analysis of PAHs profile revealed that all the examined varieties of PAHs were present in sampled air **(Table 14)**. The ΣPAHs levels at HTD site #1 and #2 ranged from 2.73 to 95.40 ng/m3 and from 9.11 to 57.11 ng/m3 respectively. At LTD site, it ranged from 1.82 to 31.47 ng/m3. The mean concentrations at HTD site #1 and #2 were 20.97

& 24.76 ng/m3 respectively **(Table 14).** At LTD site it was 9.44 ng/m3. Range of ΣPAHs (92.67, 48.00), observed at HTD site, was more than the same (29.65) observed at the LTD site. The mean values at HTD sites were more than the LTD site value. These increased by 2X. These values were far more than the guide value of 1ng/m^3 set by WHO for ambient air benzo(a)pyrene, that has highest carcinogenic potential compared to other PAHs **(WHO-Euro1987)**. NAAQS values for PAHs are not available in India.

The concentration & type of PAHs registering minimum or maximum were 0.36 ng/m3 of anthracene & 9.60 ng/m3 of benzo (ghi) pyrene at HTD Site #1, 0.61 ng/m3 of anthracene & 4.50 ng/m3 of chrysene at HTD site #2 and 0.20 of anthracene & 1.88 ng/m3 of acenaphthylene at LTD site. Anthracene registered minimum level at all the locations. Nevertheless, three different varieties viz. benzo (ghi) perylene, chrysene and acenaphthylene registered maximum at three different monitored sites. At HTD locations, each type of PAH registered an excess over its LTD site value. The increase was 1-2 times. At LTD site, three PAHs that registered lowest levels were anthracene, pyrene and acenapthene. The types that registered highest at LTD site were benzo (ghi) perylene, benzo (a) pyrene, chrysene, acenaphthylene, acenapthene, phenanthrene and fluoranthene. PAHs occur generally in the atmosphere in both the particle phase and the vapor phase. The three-ring PAH compounds (Acenaphthylene, acenapthene, phenanthrene, anthracene) are found in the atmosphere primarily in the gaseous phase whereas five and six ring PAHs (benzo (a) pyrene, benzo (ghi) perylene) are found mainly in the particle phase. The four ring PAHs (Fluoranthene, pyrene and chrysene) are found in both phases.

HTD sites when compared together showed a dissimilar pattern in their PAHs profile. Acenaphthylene, acenapthene, anthracene, fluoranthene and chrysene were higher at site #2 whereas benzo (ghi) perylene was higher at site #1. Surprisingly, the levels of benzo (a) pyrene at HTD site #2 were <1ng/m3 and were similar to that of LTD site. The unusually low levels of these PAHs at HTD site #2 may be related to meteorological & topographical conditions at the monitored sites in summer.

A percent composition of the studied PAHs profile at the investigated sites is described

in **Figure 5-7**. Benzo (ghi) perylene alone constituted 50% of all the investigated PAHs at HTD site #1 **(Figure 5)**. At other monitored sites the percent composition was similar and evenly distributed among the studied components. A study of the correlation of RSPM with benzo (a) pyrene levels revealed that there existed a moderate but statistically insignificant ($p<0.05$) correlation between the two in summer (**Figure 8)**.

In winter

All the examined varieties of PAHs were present in sampled air **(Table 15)**. The ΣPAHs levels at HTD site #1 and #2 ranged from 77.72 to 156.59 ng/m^3 and from 28.99 to 249.80 ng/m^3 respectively **(Table-15)**. At LTD site, it ranged from 5.10 to 85.30 ng/m^3. The mean concentrations at HTD site #1 and #2 were 106.08 and 100.33 ng/m^3 respectively **(Table 15)**. At LTD site, it was 26.64 ng/m^3. Range (78.87, 220.81), observed at HTD site, was more than the same (80.20) observed at the LTD site. Mean PAHs concentrations hiked in winter. It increased by 100-300% over its LTD site value. These values were far more than the guide value of 1ng/m^3 set by WHO for ambient air PAH **(WHO-Euro1987)**. NAAQS values for PAHs are not available in India.

Study of the PAHs profile revealed that all the examined varieties of PAHs were present in the sampled air **(Table 15).** The concentration & type of PAHs registering minimum or maximum were 0.47 ng/m^3 anthracene & 37.34 ng/m^3 chrysene at HTD Site #1, 0.32 ng/m^3 anthracene & 42.85 ng/m^3 chrysene at HTD site #2 and 0.05 ng/m^3 anthracene & 7.21 ng/m^3 benzo (ghi) perylene at LTD site. Like summer, anthracene registered a minimum and chrysene and benzo (ghi) perylene registered a maximum level at all locations. But unlike summer, benzo (a) pyrene registered maximum in place of acenaphthylene at one of the HTD locations. Benzo (a) pyrene, chrysene and benzo (ghi) perylene are auto-exhaust specific and particle bound. High levels of these types is not unexpected and appears related to the idling auto-engines, slow traffic clearance of the diesel/petrol operated vehicles at the monitored sites or the diesel engine driven power generator sets in adjacent markets. In winter, very few types of PAHs registered <1ng/m^3 levels at LTD site. At HTD locations each of these hiked by a factor of 2-10X. Unlike summer, the two high traffic density sites showed a similar trend in PAHs profile.

The concentration of acenaphthylene, phenanthrene, anthracene, chrysene, benzo (a) pyrene and benzo (ghi) perylene were higher but similar at both the sites.

Levels of benzo (ghi) perylene were surprisingly similar at all the locations. Levels of most of the PAHs at HTD sites were at least 4-10X of the corresponding LTD site value **(Table 15)**. In summer, this value did not increase more than 1.5 times. The unusually high ambient air levels of PAHs in winter may be related to meteorological & topographical conditions viz. low wind speeds and relatively shorter cold-air columns of SPM/RSPM at the monitored sites.

The PAHs profile and its percent composition are shown in **Figure 9-11.** Chrysene, benzo (a) pyrene & benzo (ghi) perylene together constituted 60-70% of the sum PAHs levels. Chrysene alone constituted 20-40% of all the examined ones. A study of the correlation of RSPM with benzo (a) pyrene levels revealed a moderate but insignificant ($p<0.05$) correlation between the two in winter **Figure 12**.

4.0.2 RURAL INDOOR AIR POLLUTION

4.0.2.1 RSPM during Passive Exposure

In summer

The indoor air levels of RSPM were measured at the cooking sites using fuel wood, CDC, LPG and at the non-cooking site. The values ranged from 3.20 to 8.33 mg/m^3, 7.61 to 11.11 mg/m^3, below detectable limits (BDL), and BDL to 1.11 mg/m^3 respectively **(Table 16-19 and Figure 13)**. The mean concentration was found to be 6.35 mg/m^3, 9.53 mg/m^3, BDL and 0.44 mg/m^3 respectively**.** RSPM levels were 5-10X higher at the fuel biomass using-site. The non-cooking site is basically the biomass combustion site but during the non-cooking and the smokeless period.

In winter

The indoor air levels of RSPM were measured at the cooking sites using fuel wood,

CDC, LPG and at the non-cooking site. The values ranged from 4.17 to 16.67 mg/m^3, 5.00 to 16.63 mg/m^3, BDL to 1.11 mg/m^3 and BDL to 1.86 mg/m^3 respectively **(Table 20-23 and Figure 13)**. The mean concentration was found to be 9.71 mg/m^3, 9.91 mg/m^3, 0.86 mg/m^3 and 0.89 mg/m^3 respectively. RSPM levels were 10X higher at fuel biomass using site.

4.0.2.2 RSPM during Active Exposure

In summer

The indoor air levels of RSPM were measured at the cooking sites using fuel wood, CDC, LPG and at the non-cooking site. The values ranged from 8.77 to 16.59 mg/m^3 10.96 to 17.11 mg/m^3, 0.50 to 1.04 mg/m^3 and 0.77 to 1.75 mg/m^3 respectively (**Table 16-19** and **Figure 13)**. The mean concentrations were found to be 12.86 mg/m^3, 13.85 mg/m^3, 0.95 mg/m^3 and 1.01 mg/m^3 respectively. RSPM levels were 10X higher at fuel biomass using site.

In winter

The indoor air levels of RSPM were measured at the cooking sites using fuel wood, CDC, LPG and at the non-cooking site. The values ranged from 7.02 to 21.05 mg/m^3, 7.89 to 20.18 mg/m^3, BDL –1.17 mg/m^3 and BDL to 2.00 mg/m^3 respectively (**Table 20-23** and **Figure 13**). The mean concentrations were found to be 15.79 mg/m^3, 15.18 mg/m^3, 1.02 mg/m^3 and o.81 mg/m^3 respectively. RSPM levels were 5X higher at fuel biomass using site.

4.0.2.3 Sulphur Dioxide (SO_2)

In summer

The indoor air levels of SO_2 were measured at the cooking sites using fuel wood, CDC, LPG and at the non-cooking site. The values ranged from 28.57 to 50.00 μg/m^3, 39.47 to 62.50 μg/m^3, 7.02 to 8.73 μg/m^3 and 6.33 to 7.73 μg/m^3 respectively (**Table 16-19** and **Figure 13)**. The mean concentrations were found to be 39.58 μg/m^3, 49.07 μg/m^3,

7.97 μg/m^3 and 7.11 μg/m^3 respectively. SO_2 levels were 5X higher at fuel biomass using site.

In winter

The indoor air levels of SO_2 were measured at the cooking sites using fuel wood, CDC, LPG and at the non-cooking site. The values ranged from 36.17 to 83.67 μg/m^3, 43.00 to 94.83 μg/m^3, 8.33 to 9.33 μg/m^3 and 8.17 to 10.67 μg/m^3 respectively (**Table 20-23** and **Figure 13).** The mean concentrations were found to be 64.98 μg/m^3, 69.18 μg/m^3, 8.84 μg/m^3 and 9.53 μg/m^3 respectively. SO_2 levels were >5X higher at fuel biomass using site.

The levels of SO_2 in winter and summer were different for wood and CDC (Fig 13). It could be so because of the prolonged burning of the firewood for house heating purpose. Besides use of the moist wood was noted that might be the additional cause.

4.0.2.4 Oxides of Nitrogen (NO_x)

In summer

The indoor air levels of NO_x were measured at the cooking sites using fuel wood, CDC, LPG and at the non-cooking site. The values ranged from 32.67 to 71.46 μg/m^3, 50.14 to 71.46 μg/m^3, 10.91 to 12.18 μg/m^3 and 9.91 to 11.18 μg/m^3 respectively **(Table 16-19** and **Figure 13)**. The mean concentrations were found to be 51.08 μg/m^3, 61.28 μg/m^3, 11.47 μg/m^3 and 10.47 μg/m^3 respectively. NO_x levels were 5X higher at fuel biomass using site.

In winter

The indoor air levels of NO_x were measured at the cooking sites using fuel wood, CDC, LPG and at the non-cooking site. The values ranged from 50.69 to 116.87 μg/m^3, 59.83 to 122.97 μg/m^3, 11.20 to 13.70 μg/m^3 and 10.59 to 12.95 μg/m^3 respectively **(Table 20-23** and **Figure 13).** The mean concentrations were found to be 88.80 μg/m^3, 91.44

$\mu g/m^3$, 12.04 $\mu g/m^3$ and 11.85 $\mu g/m^3$ respectively. NO_x levels were >5X higher at fuel biomass using site.

The levels of NO_x in winter and summer were different for wood and CDC (Fig 13). It could be so because of the prolonged burning of the firewood for house heating purpose. Besides use of the moist wood was noted that might be the additional cause.

4.0.2.5 PAHs during Passive exposure

In summer

The analysis of PAHs profile revealed that all the examined varieties of PAHs were present in sampled air **(Table 24)**. The ΣPAHs levels at the cooking sites using fuel wood, CDC, LPG and at the non-cooking site ranged from 4.79 to 49.28 $\mu g/m^3$, 11.99 to 66.0 $\mu g/m^3$, 3.21 to 15.94 $\mu g/m^3$ and 5.70 to 9.41 $\mu g/m^3$ respectively **(Table 24)**. The mean concentrations were found to be 19.98$\mu g/m^3$, 27.83$\mu g/m^3$, 7.42$\mu g/m^3$ and 6.18 $\mu g/m^3$ respectively. Range (44.99, 54.01, 12.73) at biomass/fossil fuel using site was more than the same (3.71) at the non-cooking site (Table 24). These values were much greater than the guide value of 1ng/m^3 set by WHO for ambient air PAH **(WHO-Euro1987)**. But comparing with occupational exposure standard of 0.2mg/m3 (TLV), recommended by NIOSH **(ACGIH 1986)**, the obtained mean values were quite low. It is important to note that the current TLV of 0.2 mg/m3 is being commented as unacceptable for after 40 years, a relative risk of 1.2-1.4 for lung and 2.2 for bladder cancer is involved **(Mastrangelo et al 1996)**. NAAQS values for PAHs are not available in India.

The non-cooking site is basically the biomass combustion site but during the non-cooking and the smokeless period. The levels of PAHs at the non-cooking site and at the LPG combustion sites are almost alike. It is inferred that LPG is not adding to the minimum detectable levels of indoor air PAHs at the non-cooking sites.

Mean ΣPAHs values at non-cooking site and at the site using LPG fuel type were

similar. All PAHs levels at these sites ranged in between 0.06 to ~5 $\mu g/m^3$ **(Table 24)**. At sites using bio-fuels, mean level of ΣPAH registered a hike by a factor of 2-4X. The types of PAHs & their levels registering minimum and maximum at investigated sites were anthracene (0.08 $\mu g/m^3$) and acenaphthylene (6.77 $\mu g/m^3$) at biomass wood using site, anthracene (0.14 $\mu g/m^3$) and acenaphthylene (10.47 $\mu g/m^3$) at biomass CDC using site, anthracene (0.04 $\mu g/m^3$) and acenaphthylene (5.30 $\mu g/m^3$) at LPG using site and anthracene (0.06 $\mu g/m^3$) and acenaphthylene (5.04 $\mu g/m^3$) at non-cooking site.

The types of PAHs that registered lowest three at all the monitored sites in rural areas included anthracene, benzo (a) pyrene and benzo (ghi) perylene. The types that registered highest five at all the sites using different fuel type included acenaphthylene, phenanthrene, fluoranthene, acenapthene & chrysene and were surprisingly very similar. PAHs occur in atmosphere generally both in particle phase as well as in vapor phase.

The percent composition of studied PAHs profile at monitored sites is shown in **Figure 14-17**. At the cooking sites using fuel wood or CDC, acenaphthylene, acenapthene and fluoranthene were the major components and constituted >75% of all the investigated PAHs. Acenaphthylene and acenapthene, the volatile PAHs, alone constituted a total of 60% at the site using biomass fuel. Acenaphthylene, pyrene, chrysene and benzo(ghi)perylene were the major components at sites using LPG. At non-cooking site, only acenaphthylene was the major component. A high level of acenaphthylene and acenapthene in indoor air environment is associated with use of fuel biomass. Source for the presence of chrysene, pyrene and fluoranthene in indoor air is not known but may be related to occasional use of the refuge burning, use of diesel driven electric generator sets in surrounding areas for irrigation purposes or indoor smoking habits of the cooks.

In winter

All the examined varieties of PAHs were present in sampled air **(Table 25)**. The ΣPAHs levels at the cooking sites using fuel wood, CDC, LPG and at the non-cooking site

ranged from 13.21 to 112.79 μg/m^3, 24.00 to 63.44 μg/m^3, 4.26 to 32.20 μg/m^3 and 2.52 to 23.14 μg/m^3 respectively **(Table 25)**. The mean concentrations were found to be 37.46μg/m^3, 42.31μg/m^3, 20.27μg/m^3 and 19.06 μg/m^3 respectively. Range (99.58, 39.44, 27.94) at biomass/fossil fuel using site was more than the same (20.62) at the non-cooking site **(Table 25)** and greater than in summer. These values were much greater than the guide value of 1ng/m^3 set by W.H.O. for ambient air PAH **(WHO-Euro1987)**. But comparing with occupational exposure standard of 0.2mg/m3 (TLV), recommended by NIOSH **(ACGIH 1986)**, the obtained mean values were quite low. NAAQS values for PAHs are not available in India. It is important to note that the current TLV of 0.2 mg/m3 is being commented as unacceptable for after 40 years, a relative risk of 1.2-1.4 for lung and 2.2 for bladder cancer is involved **(Mastrangelo et al 1996)**. NAAQS values for PAHs are not available in India.

The non-cooking site is basically the biomass combustion site but during the non-cooking and the smokeless period. The levels of PAHs at the non-cooking site and at the LPG combustion sites are almost alike. It is inferred that LPG is not adding to the minimum detectable indoor air levels of PAHs at the non-cooking sites.

Mean ΣPAHs values were similar at non-cooking site and at the site using LPG fuel type All PAHs levels at these sites ranged in between 0.12 to ~16 μg/m^3 **(Table 25)**. At sites using bio-fuels, mean level of ΣPAH registered a hike by a factor of only 2X in winter. The types of PAHs & their levels registering minimum and maximum at investigated sites were anthracene (0.15 μg/m^3) and acenaphthylene (15.25 μg/m^3) at biomass wood using site, anthracene (0.18 μg/m^3) and acenaphthylene (16.39 μg/m^3) at biomass CDC using site, anthracene (0.18 μg/m^3) and acenaphthylene (16.61 μg/m^3) at LPG using site and anthracene (0.12 μg/m^3) and acenaphthylene (16.81 μg/m^3) at non-cooking site. The types of PAHs that registered lowest three at all the monitored sites in winter included anthracene, benzo (a) pyrene and benzo (ghi) perylene. Acenaphthylene, acenapthene, phenanthrene, fluoranthene, pyrene & chrysene registered highest at the sites using fuel wood & biomass. Only acenaphthylene registered highest at the site using LPG or at non-cooking site.

The percent composition of studied PAHs profile at monitored sites is shown in **Figure 18-21**. At the cooking sites using fuel wood or CDC, acenaphthylene, acenapthene, fluoranthene pyrene and chrysene were the major components of PAHs and constituted >90% of all the investigated PAHs. Like summer, acenaphthylene and acenapthene, the volatile PAHs, alone constituted a total of 60% at the site using biomass fuel. Acenaphthylene and fluoranthene were the major components at sites using fuel LPG. At non-cooking site, only acenaphthylene was the major component. A high level of acenaphthylene and acenapthene in indoor air environment is associated with use of fuel biomass. Source for the presence of chrysene, pyrene and fluoranthene in indoor air is not known but may be related to use of the refuge burning, or indoor smoking habits of the cooks.

4.0.2.6 PAHs during Active Exposure

In summer

The ΣPAHs levels at the cooking sites using fuel wood, CDC, LPG and at the non-cooking site ranged from 14.42 to 182.13μg/m^3, 19.75 to 226.19 μg/m^3, 4.58 to 18.3 μg/m^3 and 6.07 to 20.52 μg/m^3 respectively **(Table 26).** The mean concentrations were found to be 40.83 μg/m^3, 77.01 μg/m^3, 11.10 μg/m^3 and 11.36 μg/m^3 respectively. These values were much greater than the guide value of 1ng/m^3 set by W.H.O. for ambient air PAH **(WHO-Euro1987)**. But comparing with occupational exposure standard of 0.2mg/m3 (TLV), recommended by NIOSH **(ACGIH 1986)**, the obtained mean values were quite low. NAAQS values for PAHs are not available in India. It is important to note that the current TLV of 0.2 mg/m3 is being commented as unacceptable for after 40 years, a relative risk of 1.2-1.4 for lung and 2.2 for bladder cancer is involved **(Mastrangelo et al 1996)**. NAAQS values for PAHs are not available in India.

The non-cooking site is basically the biomass combustion site but during the non-cooking and the smokeless period. The levels of PAHs at the non-cooking site and at

the LPG combustion sites are almost alike. It is inferred that LPG is not adding to the minimum detectable indoor air levels of PAHs at the non-cooking sites.

At non-cooking site or at site using LPG fuel type, the PAHs contents were similar and ranged in between 0.01 to 17.5 $\mu g/m^3$ **(Table 26)**. At sites using bio-fuels, mean level registered a hike by a factor of 2-20X. The types of PAHs & their levels registering minimum and maximum at investigated sites were anthracene (0.23 $\mu g/m^3$) and acenaphthylene (11.08 $\mu g/m^3$) at biomass wood using site, anthracene (0.54 $\mu g/m^3$) and acenapthene (13.72 $\mu g/m^3$) at biomass CDC using site, benzo (ghi) perylene (0.11 $\mu g/m^3$) and acenaphthylene (7.55 $\mu g/m^3$) at LPG using site and benzo (ghi) perylene (0.07 $\mu g/m^3$) and acenaphthylene (7.57 $\mu g/m^3$) at non-cooking site. The types of PAHs that registered lowest three and highest five at all the sites were very similar to that observed in passive exposure.

The percent composition of studied PAHs profile at monitored sites is shown in **Figure 22-25**. Acenaphthylene, acenapthene, fluoranthene and pyrene were the major components at the cooking sites using fuel wood or CDC. Together these constituted >75% of all the investigated PAHs. Acenaphthylene and acenapthene, the volatile PAHs, alone constituted a total of 50% at the site using biomass fuel. The percent profile changed at non-cooking site or at sites using fuel LPG. Here only the acenaphthylene and phenanthrene were the major components.

In winter

The ΣPAHs levels at the cooking sites using fuel wood, CDC, LPG and at the non-cooking site ranged from 19.73 to 170.51 $\mu g/m^3$, 68.76 to 178.74 $\mu g/m^3$, 12.68 to 33.73 $\mu g/m^3$ and 6.41 to 38.5 $\mu g/m^3$ respectively **(Table 27)**. The mean concentrations were found to be 69.23 $\mu g/m^3$, 90.5 $\mu g/m^3$, 26.78 $\mu g/m^3$ and 23.73 $\mu g/m^3$ respectively. These values were much greater than the guide value of 1ng/m^3 set by W.H.O. for ambient air PAH **(WHO-Euro1987)**. But comparing with occupational exposure standard of 0.2mg/m3 (TLV), recommended by NIOSH **(ACGIH 1986)**, the obtained mean values were quite low. NAAQS values for PAHs are not available in India. It is important to note

that the current TLV of 0.2 mg/m3 is being commented as unacceptable for after 40 years, a relative risk of 1.2-1.4 for lung and 2.2 for bladder cancer is involved **(Mastrangelo et al 1996)**. NAAQS values for PAHs are not available in India.

The non-cooking site is basically the biomass combustion site but during the non-cooking and the smokeless period. The levels of PAHs at the non-cooking site and at the LPG combustion sites are almost alike. It is inferred that LPG is not adding to the minimum detectable indoor air levels of PAHs at the non-cooking sites.

At non-cooking site or at site using LPG fuel type, the contents were similar and ranged in between 0.25 to ~19 $\mu g/m^3$ **(Table 27)**. At sites using bio-fuels, mean level registered a hike by a factor of 3-4X. The types of PAHs & their levels registering minimum and maximum at investigated sites were anthracene (0.36 $\mu g/m^3$) and acenaphthylene (23.16 $\mu g/m^3$) at biomass wood using site, anthracene (0.67 $\mu g/m^3$) and acenapthene (27.29 $\mu g/m^3$) at biomass CDC using site, anthracene/benzo (ghi) perylene (0.3 $\mu g/m^3$) and acenaphthylene (18.95 $\mu g/m^3$) at biomass LPG using site and anthracene/benzo (ghi) perylene (0.25 $\mu g/m^3$) and acenaphthylene (17.06 $\mu g/m^3$) at non-cooking site. The types of PAHs that registered lowest three and highest five at all the sites were very similar to that observed in passive exposure.

The percent composition of studied PAHs profile at monitored sites is shown in **Figure 26-29**. Acenaphthylene, acenapthene, fluoranthene pyrene and chrysene were the major components at the cooking sites using fuel wood or CDC. Together these constituted >90% of all the investigated PAHs. Acenaphthylene and acenapthene, the volatile PAHs, alone constituted a total of 50% at the site using biomass fuel. The percent profile changed at non-cooking site or at sites using fuel LPG. Here unlike summer, only the acenaphthylene, acenapthene and fluoranthene were the major components.

Adverse Health Effects & Exposure Estimates in PAHs Exposed Subjects

5.0 MATERIAL & METHOD

5.0.1 Study design

A total of 384 subjects from urban and rural areas of Lucknow were studied. As there is little information regarding early health impacts and economic valuation of health damage caused by PAHs exposure, a cross-sectional study design was opted to study the problem and get a preliminary picture.

5.0.2 Selection of samples

2001 census reported a rural population of 33.3 per cent in Lucknow. Approximately 40 per cent of the rural subjects were included in our study. Rural sample was drawn from an area located about 45 km North West of Lucknow. Attempt was made to divide equally the urban sample between high traffic density area (congested) in the center of the city and a low traffic density area (non-congested) at the periphery of the city. A random sample considering household as sampling unit was used for selection of subjects. Representation of economically under privileged, users of biomass fuel (especially in rural areas) and other demographic variable that may have a bearing on PAHs exposure like smoking etc was ensured. Informed consent was taken from each study subject before inclusion in of the study.

5.0.3 History and clinical examination

The study subjects were interviewed for details about personal, occupational and clinical history, which were noted on a pre-tested schedule. A complete clinical examination (viz. general health physical examination, respiratory, cardiovascular, gastro-intestinal, musculo-skeletal and central nervous system) of each subject was done and recorded by a medical doctor.

5.0.4 Biological monitoring

Urine samples were collected from consented volunteers and analyzed for exposure marker 1-hydroxypyrene. In brief, urine samples were treated with beta-glucuronidase/ sulfatase for enzymatic hydrolysis of glucuronide-hydroxypyrene conjugate at pH 5. After sample clean up and enrichment by solid-phase extraction over Sep-pak C18 cartridge, 1-pyrenol was isolated & quantified over HPLC with fluorescence detector. Standard 1-OH-pyrene eluted after a retention time of 27min. Spiked samples showed a recovery of 70%. The results were corrected for the percent recovery and expressed as μmol 1-OH pyrene/mol urinary creatinine. The urine sample was collected from each subject in sterilized containers and transported at 4C to the laboratory for analysis of 1-hydroxy pyrene.

We monitored the total exposure essentially by uhp analysis as proposed. Uhp analysis is an exposure biomarker specific to PAHs and studied usually for exposure to PAHs from all routes. The uhp is the index for total exposure. This has been validated in a number of studies. There is no other exposure biomarker available although there are several effect biomarkers. The total exposure to PAHs nevertheless meant the exposure of subjects from all the three possible routes (i.e. inhalation, ingestion and skin absorption). Reason for measuring PAHs in air samples only was basically the fact that dermal exposure does not apply here as it is mostly for the occupational environment. Only the ingestion could be the confounding factor. However since ingestion of PAHs in Indian subjects is very little compared to western counterparts where the food style is pivotally the charcoal broiled/deep fried meat, monitoring of PAHs in food was not considered in the original plan also. Further, the health damages (associated with PAHs exposure) are related to only respiratory health, where the inhaled PAHs are deposited in lungs for relatively longer periods and (known for local injury) are feared to cause health damages locally in lungs only. The metabolic milieu in lungs, being relatively sluggish, allows more time to the static deposits of PAHs (mixed with RSPM) to cause local injury and thus the biological effects, unlike GI tract where due to gastric motility RSPM mixed PAHs fail to establish contact with the tissue.

Besides, PAHs are metabolized fast by the bile juices and excreted quickly through feces giving little time for the local injury.

5.0.5 Economic component

The purpose was to make a rough estimate of the total magnitude of economic cost associated with health damage due to PAHs exposure in Lucknow. These costs measured economics of health impacts. In valuing the costs of morbidity, three types of costs were considered: (a) medical expenses; (b) lost wages and (c) individual disutility (discomfort, suffering, and the opportunity cost of time). The questions pertaining to sickness during last one month and one year for each subject were recorded. Total expenditure incurred on medical treatment was recorded. Loss of earnings in rupees due to absence from work (days with symptoms) and due to poor quality / less work (restricted study with days) due to illness were also recorded.

5.0.6 Economic valuation

Rough estimate of the total magnitude of economic costs associated with PAH exposure was calculated. These costs were calculated only in terms of health damage. The calculation is as follows:

Total cost = (Incidence of deaths x value of life) + Incidence of sickness due to PAH exposure x (cost of treatment + cost wages)

Considering that sources of PAH exposure include transport, industry, energy, smoking, exposure to bio-mass fuel burning and food, etc. no attempt was made to differentiate the health impacts by source of PAH exposure. The cost due to death in the family was excluded as the design of the study was cross-sectional and hence death could not be attributed to PAH exposures. The overall limitations of this “back-of-the envelope”

approach to value environmental costs were very clear. The methodologies, data and estimates of 'average' costs and values are all subject to extensive refinement

5.0.7 Data processing and statistical analysis

The data generated was compiled and transferred to the personal computer through use of Lotus, MS Excel and EPI INFO. Analysis was done using software EPI INFO and Systat 9.0. The significance of mean value of different parameter in different exposure groups were analyzed using analysis of variance techniques after ascertaining the assumptions of normality and homogeneity of variance. Significance of prevalence of different signs and symptoms in different exposed and control groups were analyzed using Chi square or Fisher exact test depending upon the expected cell frequencies. Odds ratio for exposed (Urinary pyrene excretion µmol/mol >0.5) in comparison to non-exposed (Urinary pyrene excretion µmol/mol <=0.5) for different explanatory variables were calculated and significance tested using Mental Hanzel's procedure.

5.1 RESULTS

5.1.1 Adverse Health Effects & Exposure Estimates

The general profile & 1-hydroxypyrene excretion pattern of the study population is shown in **Table 28 & 29**. Normal upper limit value of 1-hydroxypyrene is described to be 0.66umol/mol creatinine **(Jongeneelen 1997)** in residents from the Netherlands**.** In our study we selected ≤ 0.5umol/mol creatinine as the normal upper limit value for the cut-off limit towards coveted computations. In a study of traffic policemen and traffic drivers exposed to urban air the overall mean value of 1-hydroxypyrene excretions of control subjects have been found to be 0.57umol/mol **(Burgaz et al., 2002)**. Background-values vary a little from country to country due to variations in the environmental background and or dietary intake of PAH. A higher percentage (82.8 %) of population excreting more than normal levels of urinary hydroxypyrene (uhp) was observed **(Table 29)** in

congested areas. However, mean pyrene levels in different PAHs exposed population groups were not statistically significant. Factors affecting uhp excretion were found to be gender, vehicular emission exposure, non-vegetarian diet & smoking. Females excreted 1-hydroxypyrebne more than males in both the rural areas and in urban non-congested area. However the differences were not statistically significant **(Table 30)**. On the contrary, in congested areas males excreted more compared to females but the differences were again statistically insignificant. Mean uhp levels among non-vegetarians **(Table 31)** were higher ($p < 0.1$), Smokers, living in congested areas **(Table 32)**, excreted more of uhp. Mean pyrene levels were more than the means in non-smokers but the differences were again statistically insignificant. Tobacco smoking affects the uhp concentration **(Jongeneelen 1997)**. Proportion of subjects excreting more than normal levels of uhp (> 0.5 µmol/mol) was significantly higher in group exposed to vehicular emission (OR = 2.32, $p < 0.001$) compared to proportion of subjects in group less exposed to vehicular emission in low traffic density area **(Table 33)**.

Uhp is known to be a good exposure marker for PAH exposure. PAHs carcinogenic potential is often reported but the short-term clinical effects are hardly known. In our study we detected a group of symptoms related to eye, ear, cardiovascular and respiratory systems that were significantly more in groups excreting more than normal levels **(Table 34)**. Subjects having these symptoms-cluster and excreting more than normal levels of uhp were observed to be 29.4 percent compared to 17.3 percent who had the symptom cluster but excreted normal levels of uhp. These differences were statistically significant **(Table 35)**. A number of these symptoms are linked with exposure to other criteria air pollutants like PM, SO_2, NOx, ozone, lead etc. **(Schwela 2000)**. PM is also known to be carriers for PAHs **(Lioy and Greenberg 1990)**. As such this cluster of symptoms are effects of exposure to different air pollutants. But in subjects with high uhp excretion, exposure to PAHs could be an attributing factor for a proportion of these symptoms.

No statistical association could be observed in subjects with specific morbidity or affected organ system and high uhp excretion **(Table 36)**. Mortality as shown in **Table**

37 was recorded on basis of verbal autopsy report from family members of the study subjects. As evident, no links with uhp could be detected. Average YLL (00) for deaths in the family of both the groups were found to be 15.9 years (normal uhp) and 21.3 years (high uhp) considering 63 years life expectancy at 0 age. Subjects excreting more uhp experienced more days of sickness and lost more money in the form of lost earnings **(Table 38)**. More than 20% of subjects both in low & high uhp group spent money on treatment **(Table 39)**. Total money spent on treatment and that spent on consultation and drugs was more in subjects with more uhp excretion **(Table 40 & 41)**. This is also true for lost earnings due to sickness and poor quality work/less time devoted to work **(Tables 42 & 43)**. A population of 16.4 lakh persons was estimated to be excreting higher than normal levels of uhp in Lucknow and hence is exposed to PAHs. A sizeable population of 3.84 lakh of these suffers from a symptom cluster that is attributable to PAHs **(Table 44)**. The cost of PAHs exposure related symptom cluster is estimated to range between Rs. 20.0 -163.3 crore for Lucknow **(Table 45)** with an average estimated cost to be 91.9 crore **(Table 46)**.

6.0 Discussion

We studied the total human environmental exposure to PAHs in impoverished subjects of Lucknow by monitoring concurrently the ambient/indoor air PAHs and the criteria air pollutants in urban/rural parts of the district alongwith the exposure assessment of the subjects from the monitored sites. The ambient/indoor air monitoring has most commonly been used to evaluate exposure to airborne PAHs **(Chuang et al., 90, Risner and Conner 91, Shuguang et al., 1994)**. This method however allows an estimate of respiratory uptake only and does not take into account other exposure routes. An increasingly popular approach on the other hand has been the analysis of urinary hydroxy pyrene **(uhp)** as a biological indicator of overall PAH exposure **(Jongeneelen et al., 88, Jongeneelen 94, Jongeneelen 97, Zhao et al., 92)**. Exposure to PAHs can occur through inhalation, ingestion or skin contact. Therefore a measurement of absorbed dose allows taking into account exposures through different routes **(Lauwerys & Hoet 1993, Bowman et al., 97, Sithisarankul et al., 1997, Van Rouij et al., 1994, Zhao et al., 1995).** The primary major source of ambient-air-PAHs in urban, suburban or rural is the incomplete combustion of wood and fuel **(Perwak et al., 1982)**. Automobiles, truck engines, various two stroke or four stroke gasoline or diesel powered engines, the gas-burning engines, wood burning hearths, coke ovens, stoves, and furnaces or industrial smoke **(IARC, 1983**; **Nikolaov et al., 1984)** are the major contributors. In ambient air, PAHs are distributed between suspended particulate matter and the gases according to their vapor pressure and according to ambient temperature **(Valerio and Pala, 1991)**. Tobacco smoke and the charcoal-broiled food constitute another common source of PAHs intake.

Lucknow, the studied site, is the capital of Uttar Pradesh, India. It is a fairly large city with a population of 22,07,340. About 2/3rd area of Lucknow has urbanized leaving only 1/3rd area as the rural part of the district **(Census 2001)**. According to conservative estimates, the city has approximately 4,50,000 vehicles on roads. Two or three wheelers (scooters, motor bikes etc) constitute about 78% of the vehicular density and car, taxi or heavy-duty vehicles constitute the rest. The number of vehicles in the city is growing by a factor of 10% each year. Petrol and diesel consumption of the city is >70,000 kiloliter

and >1,30,000 kiloliter per annum respectively **(Pandey 2001)** and is increasing. As a consequence, the ambient air burden of the city due to the autoexhaust emission is increasing and so also the load of the hazardous air pollutants (HAP) in the city. Besides this, the city has added petrol/diesel driven electricity generator sets to cater to the increasing demand of the electricity, which is further escalating the environmental load of hazardous air pollutants. To best of our knowledge, ambient/indoor air PAHs levels in Lucknow have not been studied yet. There is no published information on levels of PAHs pollution, the PAHs burden in the exposed subjects and the associated illnesses in Lucknow. This is the first report on the subject.

The urban air monitoring has been done in urban areas of the underprivileged class of the society living adjacent to the heavy traffic areas. Respirable dust samplers were used for ambient air monitoring. The basis for using the fixed type samplers is the BIS approved method. The urban indoors have not been monitored because the penetration of the ambient air pollutants from outdoor into indoor environment is described to be 70% **(Lioy & Greenberg 1990)**. Therefore, the indoor air levels will be predominantly similar as the ambient air during non-cooking hours. Further, the aim was not to compare urban indoor air values with the rural indoor values. On the contrary, the aim was to reveal the levels of the ambient air pollutants especially PAHs in urban areas and to examine subsequently its affiliation with traffic density.

Similarly in rural areas the aim was to examine the affiliation of levels of indoor air Toxic Air Pollutants with the biomass combustion. Both fixed-type low volume sampler and the mobile type personal sampler were employed for monitoring the levels of Toxic Air Pollutants, especially PAHs, during passive and active exposure respectively. Then the objective was the exposure assessments both in subjects from urban and rural areas of the underprivileged class in order to identify the PAHs exposed population for assessment of the health risk specifically in subjects excreting more than the normal levels of the exposure marker uhp. The types of samplers used for ambient/indoor air monitoring of urban or rural areas are different. The cut off size of the Respirable Dust Sampler is 10 micron and that of the Personal Sampler is 5 micron. We have not compared the data obtained from RDS with that from PS.

The auto-exhaust and diesel exhaust emission contains particulate matter that contributes to air pollution in general and to ambient particulate pollution in particular by >50%. The autoexhaust derived particulate matter (i.e. SPM) carries PAHs adsorbed on it and is the major ambient air pollutant of the present day urban society **(Lioy and Greenberg 1990, Health Effects Institute, USA 1995, WHO 1996, Dubowsky et al 1999, Grillies and Gertler 2000, Zhu and Liu 2001)**. Ambient air monitoring of urban Lucknow revealed status of PAHs vis-à-vis status of criteria air pollutants in ambient air. An increase in levels of particulate matter at all the monitored sites was seen. Hike in particulate matter content showed a seasonal trend and exceeded the NAAQS limit in winter at all the monitored locations. Levels of SO_2 & NOx, analyzed concurrently at all locations, were found to be within the permissible limits both in summer as well as in winter. Analysis of PAHs revealed presence of both autoexhaust/biomass-smoke specific varieties of PAHs in ambient air. Their contents increased 5X in winter. Mean PAHs levels at HTD sites were more compared to LTD sites in both the seasons. This increase was 1-2X in summer and 1-3X in winter. This observation showed the relation of ambient air PAHs content with traffic density. The content of each type of PAHs at HTD site was more compared to its level at LTD site. A difference in spectrum of PAHs was noticed at two HTD monitored sites. The site rich with idling diesel-powered-locomotive/bus engines yielded more of the 5&6 ring PAHs, the particulate bound type. The site rich with fast moving diesel-powered bus engines showed a uniform distribution of 3-6 ring PAHs. Higher content of benzo(ghi)perylene & chrysene were noted in ambient air during both the seasons. The particle bound PAHs were found in our studies to be the major pollutant in urban Lucknow. In our studies, a moderate correlation of ambient air benzo(a)pyrene with ambient air RSPM was also noticed and in both the seasons. These observations (for details see Results) are in consonance with the published reports.

Several air-monitoring studies indicate that concentration of PAHs higher than NAAQS values are present in urban air. In a 120 city based study in USA, benzo(a)pyrene concentrations ranged between 0.2 and 19.3 ng/m^3. Ambient benzo(a)pyrene concentration in non-urban areas ranged between 0.1 and 1.2 ng/m^3 **(Pucknat 1981)**. Atmospheric concentrations of particulate phase PAHs in urban or rural areas have also

been evaluated in summer and winter seasons. Urban PAH concentrations have been found to be 3-5 times higher than the rural site in the same season. Concentrations in winter were 5-10 times higher than in summer. Geometric mean concentrations of 10 PAHs (including benzo(a)pyrene, chrysene, benzo(b,j,k)fluoranthene, benz(a)anthracene, benzo(g,h,i)perylene and pyrene) ranged from 0.03 to 0.62 ng/m^3 in urban areas and from 0.01 to 0.12 ng/m^3 in rural areas during summer seasons. During the winter seasons, geometric mean concentrations of these PAHs ranged from 0.40 to 11.5 ng/m^3 in urban areas and from 0.08 to 1.32 ng/m^3 in rural areas. Geometric mean concentrations of specifically benzo (a) pyrene ranged from 0.11 to 0.23 ng/m^3 (urban) and 0.04 to 0.06 ng/m^3 (rural) during the summer season and from 0.69 to 1.63 ng/m^3 (urban) and 0.17 to 0.32 (rural) during the winter season **(Greenberg et al., 1985)**. There are more of such data on atmospheric PAHs for many places & cities in the world **(Alsberg et al., 1985, Menchini 1992, APARG 1995, Ciccioli et al., 1996, IPCS 1998, Anh et al., 1999, Menchini et al., 1999, Panther et al., 1999, Pereira Netto et al., 2002)**. Seasonal trends have also been observed in temperate & cold climates **(Rocha & Duarte 1997, Menchini et al., 1999)**. For tropical cities, only scanty reports are available on the subject **(Panther et al., 1999, Pereira Nato et al., 2002)**. In India in a study at Kanpur, the ambient average PAHs and benzo(a)pyrene concentration has been reported to be 282 ng/m^3 and 126 ng/m^3 respectively **(Thakre et al., 1997)**. These values are very high as compared to our studies and other published reports on the subject. In another study of the PAHs levels in the atmosphere of Bhilai, an urbanized industrial site, in India, higher yearly average values (4.66 ug/m3) than most of the concentrations previously reported in the literature were noted. Benzo(a)pyrene levels were found to be 6.27 and 3.83ng/m^3 at two different monitored locations **(Pandey et al., 1999)**.

Auto-emission is reported to contain more benzo (ghi) perylene and pyrene **(Santodonato et al., 1981, Rogge et al., 1993, Bostrom et al., 2002)**. PAHs in diesel exhaust particulates are dominated by fluoranthene, phenanthrene and pyrene **(Kelley et al., 1993, Rogge et al., 1993, Westerholm and Li, 1994)**. This may explain the high levels of uhp in vehicular-emission exposed population observed in our studies. The three ring PAHs compounds (Acenaphthylene, acenapthene, phenanthrene,

anthracene) exist in atmosphere primarily in gaseous phase whereas five and six ring PAHs (benzo (a) pyrene, benzo (ghi) perylene) exist in particle phase. Benzo(a)pyrene is the main indicator of carcinogenic PAHs. Fluoranthene is an important volatile PAH and is suggested as a complementary indicator to benzo(a)pyrene **(Bostrom et al. 2002)**. The four-ring PAHs (Fluoranthene, pyrene and chrysene) are found in both phases. Chrysene, triphenylene, pyrene and fluoranthene are dominant among the PAHs found in fine particle emissions from natural gas home appliances **(Rogge et al., 1993).** Acenaphthylene, fluorene and phenanthrene have been found to be predominant in total diesel emissions **(Lowenthal 1994)**. Benzo (ghi) perylene is the most abundant and frequently detected PAH also in fly ash samples collected from municipal solid waste incinerators **(Shane et al., 1990)**.

Wood or biomass burning is a major source of energy for indoor cooking & heating in rural parts of India. People use wood, sticks, twigs, unprocessed cow-dung biomass, agricultural crop waste etc for the biomass fuel. Wood or biomass combustion has been recognized as an important source of airborne PAHs **(Knight & Humphrey 85, Lioy et al., 88, Lioy and Greenberg 90, Wilson and Chuang 91)**, particulate matter and CO **(Chen et al., 1990).** Indoor air monitoring of rural Lucknow in our studies revealed the status of PAHs vis-à-vis status of criteria air pollutants in indoor air of the rural part of district. Use of biomass or the fossil fuel by rural subjects was found in our studies to affect the indoor air quality. Levels of particulate matter & PAHs were more at biomass fuel using sites compared to non-cooking sites. Volatile PAHs e.g. acenaphthylene were prevalent at the monitoring site more during active exposure in winter. These are the characteristic of biomass combustion site. It reflected the biomass burning to be the major source of particulate matter and thus the major indoor air pollutant. Use of LPG was comparatively safe. These observations (for details see Results) are in agreement with the published reports. High levels of combustion by-products during the cooking & heating activities are reported **(Ligman et al., 1997, Shaughnessy et al., 1997).** Emissions from residential wood combustion contain more acenaphthylene than other PAHs **(Perwak et al., 1982)**. Acenaphthylene, phenanthrene, fluoranthene, pyrene, benzo (a) anthracene, chrysene, benzo(a,e)pyrene, benzo(b,k)fluoranthene, dibenz(a,h)anthracene, benzo(g,h,i)perylene, indeno(1,2,3-cd)pyrene have been

detected during cooking hours in houses using different type of cooking fuels e.g. cattle-dung cakes, wood or fossil fuel **(Raiyani, et al., 1993**). Wood/biomass combustion is the largest source of atmospheric PAHs releases **(Ramdhal et al., 1982)** and is primarily the result of inefficient combustion and uncontrolled emissions **(Freeman and Cattell, 1990, Tan et al., 1992)**. Combustion of wood, biomass or fossil fuel for energy makes the inhabitants and users prone to be exposed to larger doses of PAHs. In particular women are exposed more than men to the smoke emitted from the fuel burning and cooking itself **(He et al., 1991, Liu et al., 1993, Mumford et al., 1993)**. This partly explains the higher levels of uhp excreted by women in our studies. Indoor air pollution resulting from the incomplete combustion of coal and unprocessed biomass (crop residues, wood, sticks and twigs) to heat homes or to cook food is a major public health problem in developing countries **(McMichael and Smith 1999, Bruce et al., 2000, Smith and Liu 1994)**. Environmental Tobacco Smoke further adds to the problem **(Kuller et al., 1986 and Wang et al., 2000)**. The constituents generated from incomplete combustion of these fuels contain chemicals that have been identified as known or suspected carcinogens in studies of lung cancer in China & other countries **(Chen et al., 1990)**. Long-term exposure to combustion related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality **(Arden Pope et al., 2002)**. In rural China, indoor exposure to coal increases the risk for lung cancer **(Kleinerman et al., 2002)**. Indoor air pollution is postulated as a potential risk factor for head & neck neoplasm **(Kodama & Dollar 1983, Dietz et al., 1995, Pintos et al., 1998)** and lung cancer **(Smith & Liu, 1994, Kleinerman et al., 2002).** In a study of indoor exposure to PAH in traditional houses in Burundi, Africa, substantially high levels of the studied pollutants in indoor air and higher uhp contents in exposed subjects are reported, which is feared to constitute a potential health hazard to rural population of Burundi **(Viau et al., 2000)**

In present study, the exposure assessment of subjects, studied in both HTD/LTD sites of the urban area and biomass fuel using sites of the rural area, was done. In humans, 1-hydroxypyrene is excreted in urine as a glucuronide conjugate and is the index biomarker for assessing the total exposure to PAHs **(Jongeneelen et al., 1986)**. It reflects the recent exposures to mixed PAHs **(Zhao et al., 1990, Jongeneelen et al.,**

1994, Buckley et al., 1995, Kang et al., 1995a, Merlo et al., 1998). The urinary 1-hydroxypyrene is a stable metabolite **(Spierto et al., 1997, Bouchard and Viau 1999)**. It accounts for 81-100% of total pyrene metabolite excreted in urine **(Singh et al., 1995)**. The detection limit of uhp is very low viz. 0.01umol/mol creatinine comparable to approximately to 0.1nM **(Jongeneelen 1997)**. Background median uhp from people throughout the world has been reported in the range from 0.03 to 0.76umol/mol creatinine **(Levin 1995)**. The mean levels of uhp detected in our study in urban/rural subjects were higher than the reported value 2.06pmol/ml (comparable to 0.2umol/mol creatinine) and 0.33pmol/ml (comparable to 0.03umol/mol creatinine) recorded in Chinese & Korean subjects **(Roth et al., 2001)**. The highest uhp value (50.14 and 62.49umol/mol creatinine) was detected in both urban/rural subjects albeit only in population exposed to vehicular/biomass emissions. In US individuals eating charbroiled beef **(Kang et al., 1995b)**, an uhp concentration of 10-83pmol/ml (comparable to 1-8umol/mol creatinine) has been recorded. Normal upper limit value of 1-hydroxypyrene is described to be 0.66umol/mol creatinine in residents from the Netherlands **(Jongeneelen 1997).** We selected ≤ 0.5umol/mol creatinine as the upper cut-off limit value for the computations in our study. In a study of traffic policemen and traffic drivers exposed to urban air the overall mean value of 1-hydroxypyrene excretions of control subjects have been found to be 0.57umol/mol **(Burgaz et al., 2002)**. The background-values vary a little from country to country due to variations in the environmental background and or dietary intake of PAH. Exposure to PAHs due to domestic heating and cooking with coal burning stoves is associated with significantly increased levels of uhp and ingestion of food contaminated with PAHs, e.g. grilled food preparations, is among the major sources of inter-individual variability in 1-OHPG excretion **(Van Rouij et al., 1994, Siwinska et al., 1999)**. In our studies, a higher percentage (82.8%) of population living in HTD areas of Lucknow district excreted more than the cut-off levels (>0.5 µmol/mol) of uhp **(Table 29)**. Factors affecting uhp excretion were found to be gender, vehicular emission exposure, biomass emission exposure, non-vegetarian diet & smoking. In our studies, proportion of urban subjects excreting more than the cut-off levels of uhp (> 0.5 µmol/mol) was significantly higher in group exposed to vehicular emission **(OR = 2.32, p < 0.001)** compared to proportion of

subjects in group less exposed to vehicular emission in low traffic density area **(Table 33)**. Similarly, proportion of subjects excreting more than normal levels of uhp was higher also in the group exposed to biomass (cow dung cake/wood/agricultural refuse/ emissions compared **(OR =1.62, p=0.0525)** to the group not exposed to biomass emissions **(Table 33b,c).**

Risk of cancers (e.g. cancer of head & neck, lung and upper aero-digestive tract) following chronic exposure to smoke from indoor fires, to traffic related air pollution or to high PAHs in airborne dust surrounding aluminum or gas plants or coke works is often described **(Kawahata et al., 1983, Kodama and Dollar 1983, IARC 1987, WHO 1987, IPCS 1998, Pintos et al., 1998, Kleinerman et al., 2002)** but short-term clinical effects following chronic exposure to PAHs in ambient/indoor air as observed in our study are hardly reported. Whereas no statistical association could be observed in subjects with specific morbidity or specific affected organ system and high uhp excretion, a group of symptoms related to eye, ear, cardiovascular and respiratory systems were significantly more in groups excreting higher than the cut-off limit. Subjects suffering with the symptom-cluster and excreting more than the cut-off levels of uhp were observed to be 29.4 percent compared to 17.3 percent who had the symptom cluster but excreted the cut-off or less than the cut-off levels of uhp. These differences were statistically significant **(Table 35)**. As a number of these symptoms are linked with exposure to criteria air pollutants like PM, SO_2, NOx, ozone, lead etc. also **(Schwela 2000)**, the prevalence of symptom cluster in the uhp-excreting population can not be causally linked solely to PAHs exposure. As such this cluster of symptoms are understood to be effects of exposure to different air pollutants. But in subjects with high uhp excretion, exposure to PAHs could be an attributing factor for a proportion of these symptoms. A relation between ambient air pollution and respiratory symptom complex in preschool children at Lucknow has been reported **(Awasthi et al., 1996)**. Road traffic exposure increases several adverse effects on respiratory health of children **(Ciccone et al., 1998)**. An association between diesel-exhaust exposure and chronic lung disorders is reported among adolescents of Harlem, New York **(Northridge et al., 1999)**. Increased cardiopulmonary morbidity are reported to be associated with elevated levels of particulate matter of <10mm diameter (PM_{10}) **(Archer 1990, Pope et al., 1992, 1995,**

Nitta et al., 1993, Schwarz 1993, Magari et al., 2001 and Peters et al., 2001). Inhalation of fine particulate matter air pollution and ozone at concentrations that occur in the urban environment causes acute conduit artery vasoconstriction in healthy adults **(Brook et al., 2002)** and the effect is at least partly mediated through increased susceptibility to myocardial ischemia **(Pekkanen et al., 2002)**. The eye and the ear problem related to PAHs exposure could not be found in the available literature. The observed ocular symptoms e.g. defective near and distance vision, lachrymation, pain, swelling and the redness of eye in high uhp excreting population suggested its relation to PAHs and other associated pollutants like acrolein, particulate matter (especially fine & ultra-fine) and volatile organic compounds in ambient environment. The symptoms pertaining to ear e.g. pain, ear discharge, tinnitus, decrease hearing acuity in high uhp excreting group could be indirectly related to vehicular PAH, more noise levels due to pressure horns in high traffic density area and upper respiratory tract infections. As the purpose of the study was to describe the health effects associated with exposure to PAHs, a cross-sectional design was followed where the study population is contacted only once. Hence subjects with diseases long incubation period e.g. cancer are not likely to be included in the study sample.

The auto exhaust/biomass emission contributes particulate matter, which carries the products of incomplete combustion (PIC). PIC contributes by ~37% to air pollution related cancer cases. PAHs are the major constituent of PIC. These are an important class of environmental contaminants because of their suspected carcinogenicity **(IPCS 1998)**. These in fact have been classified as human carcinogen by IARC, Lyon France **(IARC 1987, 1999)**. Exhaust from diesel-powered engines has been classified as a Group 2A carcinogen according to the IARC whereas gasoline engine exhaust has been classified as a Group 2B carcinogen **(IARC 1989, IPCS 1996).** The health damaging and environment polluting effects of PAHs compounds are widely known **(Hammond et al., 1976, Grimmer 1979, IPCS 1998)**. Annually thousands of tons of PAHs enter the biosphere due to fossil fuel or biomass combustion. **US EPA (1990)** has short-listed PIC as one of the 12 major air pollutants that are responsible for more than 90% of the total cancer incidences. The World Health Organization has included PAHs into the list of the priority pollutant in both air & water **(WHO-Euro 1987).** As a result

several developed nations have prescribed their PAHs emission limits **(OECD 1995)** with an objective not only to keep a watch over the ambient air PAHs levels but also to reduce the environmental load of HAP for better environment & health. It is a common consensus there that the priority pollutant PAHs pose a serious health hazard and as such they require maximum emission reduction or zero levels regardless of the cost **(OECD 1995)**. In rapidly developing countries like India, ambient air quality guidelines for hazardous air pollutants are yet to be prescribed. In India, the levels of PAHs in urban ambient/rural indoor air or even at the work place environment are not being monitored routinely and could be dangerously high levels, e.g. coke oven emissions. The significance of the ambient air PAHs monitoring program & health risk assessment is beginning to be realized **(Pandey et al. 1999).** The carcinogenic potential of the particulates in India may be more than that from the developed countries due to the different living style, environment, body constitution, nutritional status and most important of all the destitute status of the exposed subjects.

Economic evaluation of health damages caused by exposure to PAHs in impoverished subjects has revealed substantial financial losses in terms of YLL, loss of earnings due to sickness, more expenditure on treatments, medical consultation and drugs (for details please see Results). A sizeable population (~3.84 lakh) suffers from PAHs-attributable symptom cluster. The whopping cost of the PAHs exposure related symptom cluster is estimated to range between Rs. 20-163.3 crore annually for Lucknow with a mean estimated cost to be 91.1 crore. The cost due to death in the family was excluded, as death could not be attributed to PAHs exposure owing to the cross-sectional study design. The overall limitations of this “back-of-the envelope” approach to evaluate environmental costs were very clear. The methodologies, data and estimates of ‘average’ costs and values are all subject to extensive refinement

7.0 CONCLUSION

7.0.1 Ambient/indoor air PAHs analysis

(a) PAHs are present in ambient/indoor air environment at higher than the WHO prescribed limit or guide values.

(b) PAHs concentrations change with traffic density in urban areas and with biomass combustion in rural areas. Seasonal effects are also visible. Levels increase in winter by several orders of magnitude.

(c) Ambient air benzo (a) pyrene levels show a modest correlation with RSPM levels

(d) A detailed study of individual PAH shows an abundance of 2-3 ring (volatile) PAHs in rural areas at the biomass combustion sites and an abundance of 3-6 ring (particle bound) PAHs in urban areas at the high-density traffic sites

(e) PAHs concentrations were greater in indoor air during the active exposure than the passive exposure

7.0.2 Uhp Levels

(a) A substantially higher percentage (82%) of population in traffic-congested areas excreted more than the cutoff levels of 0.5umol/mol creatinine. Proportion of subjects excreting more than the cutoff levels was significantly higher in group exposed to vehicular emission (OR = 2.32, $p<0.001$) compared to proportion of subjects in group less exposed to vehicular emission in low traffic density areas.

(b) Factors affecting uhp excretion were gender, non-vegetarian diet and smoking.

(c) It is a first report on the uhp levels in INDIAN subjects

7.0.3 Adverse health effects

(a) PAHs carcinogenic potential is reported but the short-term clinical effects are hardly known

(b) We detected a group of symptoms related to eye, ear, cardiovascular and respiratory systems that were significantly more in groups excreting more than normal levels.

(c) Subjects having these symptoms-clusters and excreting more than the normal levels of uhp were observed to be 29.4% compared to 17.3 % who had the symptom cluster but excreted normal levels of uhp. These differences were statistically significant.

(d) As such this cluster of symptoms are effects of exposure to different air pollutants. Burt in subjects with high uhp excretion, exposure to PAHs could be an attributing factor for a proportion of these symptoms.

(e) A population of 16.4 lakh persons was estimated to be excreting higher than normal levels of uhp in Lucknow and hence is exposed to PAHs. A sizeable population of 3.84 lakh of these suffers from a symptom cluster that is attributable to PAHs.

(f) No statistical association could be observed in subjects with specific morbidity or affected organ system and high uhp excretion.

(g) No links of mortality with uhp excretion could be detected. Mortality was recorded on basis of verbal autopsy report from family members of the studied subjects.

7.0.4 Health Economics

(a) Average Year Life Loss (YLL) for deaths in the family of both the groups were found to be 15.9 years (normal uhp) and 21.3 years (high uhp) considering 63 years life expectancy at 0 age.

(b) Subjects excreting more uhp experienced more days of sickness and lost more money in the form of lost earnings.

(c) More than 20% of subjects both in low & high uhp group spent money on treatment. Total money spent on treatment and that spent on consultation and drugs was more in subjects with more uhp excretion. This is also true for lost earnings due to sickness and poor quality work/less time devoted to work.

(d) The cost of PAHs exposure related symptom cluster is estimated to range between Rs. 20.0-163.3 crore for Lucknow with an average estimated cost to be 91.9 crore.

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Policy Recommendations

Existing Scenario

PAHs levels in ambient/indoor air of the urban/rural areas of Lucknow are substantially higher than the WHO-prescribed limits or guide values for other countries. A great majority of the studied (destitute) subjects (>80%) are exposed to PAHs as ascertained by the urinary 1-OH-pyrene analysis, the biological marker for assessing exposure to PAHs. Exposure to PAHs is associated with the prevalence of symptoms-cluster related to ear, eye, respiratory and cardiopulmonary systems and the association is statistically significant. The sources of these hazardous air pollutants are the autoexhaust & biomass combustion emissions. Economic evaluation of health damages caused by exposure to PAHs in (impoverished) subjects has revealed substantial financial losses in terms of Year Life Loss, loss of earnings due to sickness, and more expenditure on treatments, medical consultation and drugs. We have found that a sizeable population (~3.84 lakh) is threatened to suffer from PAHs-attributable symptoms-cluster in a study population of 22 lakh only at Lucknow. The cost of the PAHs exposure related adverse health effects could range between Rs. 20-163.3 crore annually with a mean estimated cost to be 91.1 crore only for Lucknow. The main issue is that the National Ambient Air Quality Standards (NAAQS) for PAHs are yet to be prescribed in India.

Studies on a larger scale (e.g. National Coordinated Program) are needed in India to characterize more PAHs polluted areas and PAHs exposed subjects especially for the health risk assessment plus health-damage cost evaluations vis-à-vis PAHs pollution. It will establish a large database not only to link the PAHs emissions exposure with prevalence of cardiovascular & respiratory system specific ailments but also to compute a dose response relationship. A site investigation plan should be developed in the country that addresses the issues of potential sources, local hot spots or the geographical spreads of PAHs contamination and its affiliation with the prevalence rate of PAHs exposure related health hazards. The PAHs exposure assessment of urban/rural Indian subjects (by monitoring urinary exposure marker level as

done in our project for the first time in the country) should be a priority, as it shall help define areas where exposure point concentrations exceeded the levels of health concern. The site investigation plan should include environmental monitoring, exposure assessment, residential history information and health survey. The work-plan should be validated in various areas of the country to distinguish between the least and the most polluted areas and for comparison of the environment & health in India. These endeavors will help to study the size and geographical spread of the population at risk and will also reveal the prevalent types of morbidities or cancer risk in PAHs exposed Indian subjects. There is a paucity of such data in India. This is also the high time that National Ambient Air Quality Standard for PAHs should be prescribed.

Last but not the least, a wide publicity on environmental levels of toxic air pollutants (e.g. web notice boards) and the associated health risk for increased public awareness & political sensitivity to curb the PAHs levels, exposures and related health effects is recommended.

To contain the PAHs pollution in ambient/indoor air in urban/rural areas in India, following aspects should be considered on priority:

TECHNOLOGY

Low PAH emitting vehicles

Change in fuel policy to limit the environmental levels of PAHs

Phasing out of the combustion engines with outdated designs

Replacement of gasoline/diesel-powered transport by suitable alternatives of mass transport

Biomass combustion replacement with safer fuels e.g. LPG

Ventilated housing or work environment

Raise the current speed limits on well-protected highways

Increase the number of high-speed roads and the high-speed fly-over in the PAHs polluted city

ENVIRONMENT

Geographical spread and intensification of Green Belts

Regular monitoring of the ambient/indoor air PAHs levels to contain it within in the limit or the guide value

MEDICAL

Exposure assessment of PAHs exposed subjects to study the pattern and the change in prevalent rate of associated morbidities by environmental or technological interventions

Lung cancer (target tissue) risk assessment particularly in view of extraordinarily high levels of benzo(a)pyrene & RSPM content of ambient air environment

Study mortality rate association with ambient air PAHs/RSPM content

Study PAHs burden in body tissues

Main objective is to bring down the PAHs environmental levels, exposure levels and the frequency of related adverse health effects to normal values.

ANNEXURE-I and II
Figures and Graphs

Figure 1: Chromatogram of Reference Standards of PAHs

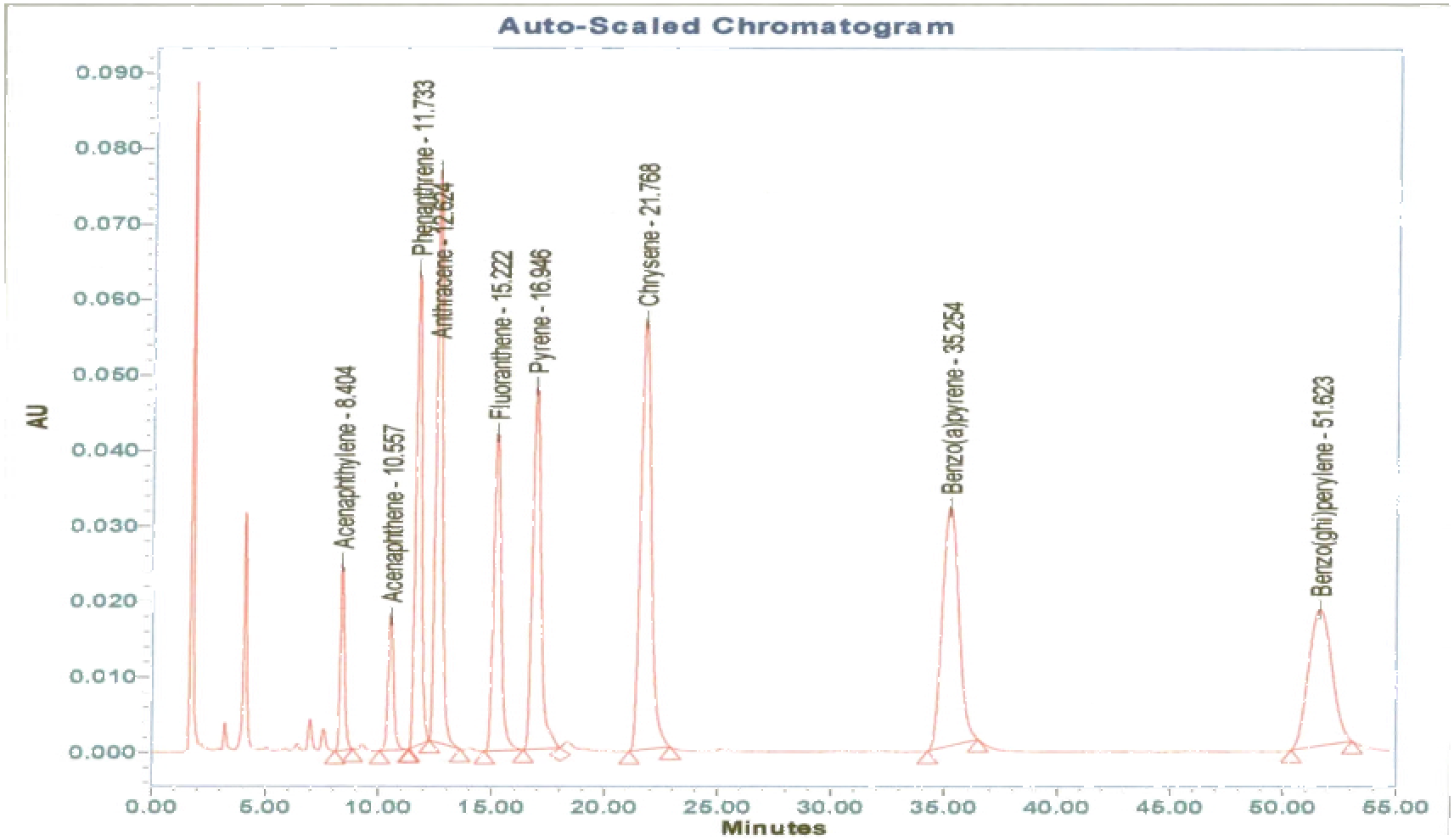


Figure 2: Chromatogram of Indoor air PAHs Samples

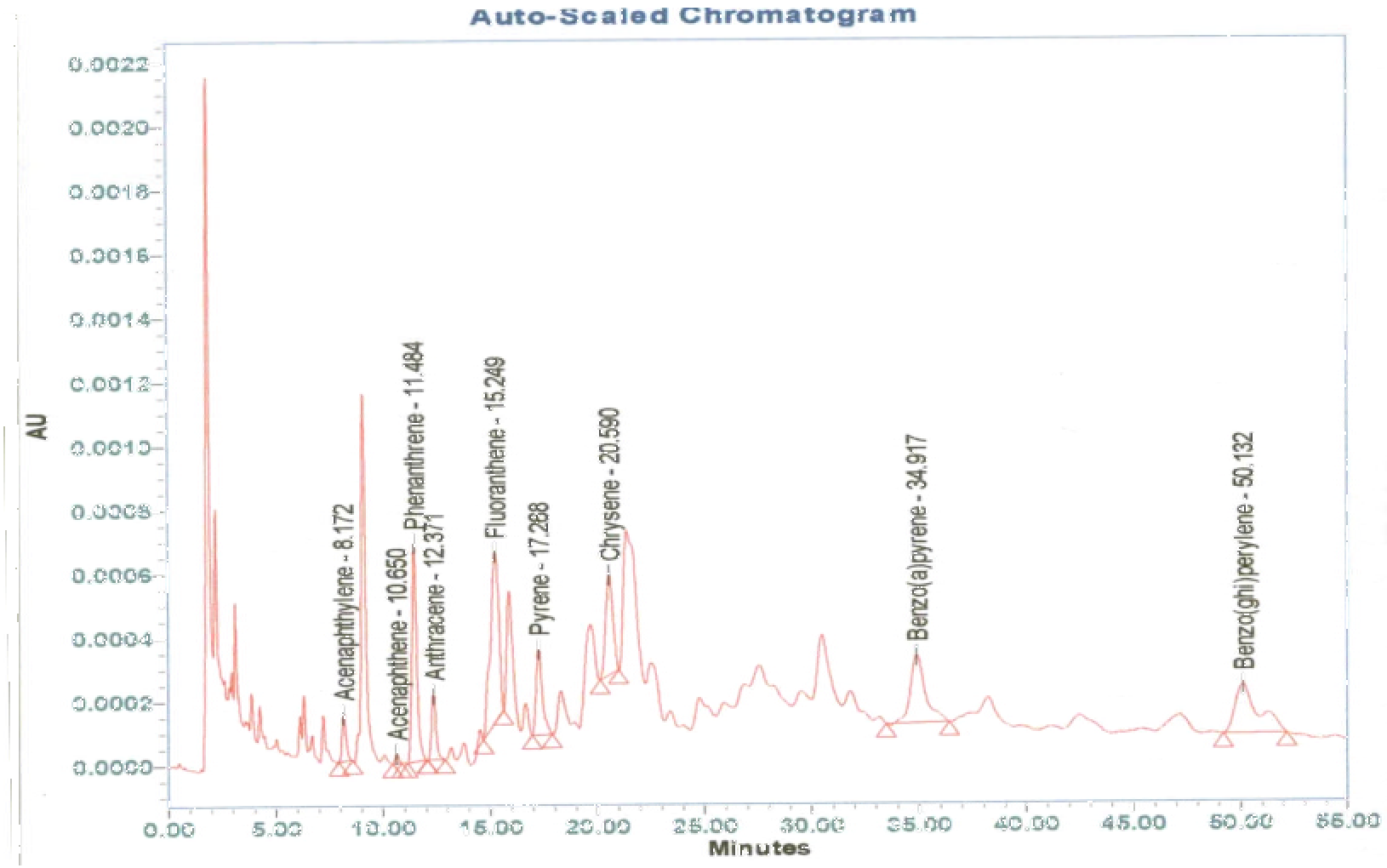


Figure 3: Chromatogram of Ambient Air PAHs Samples

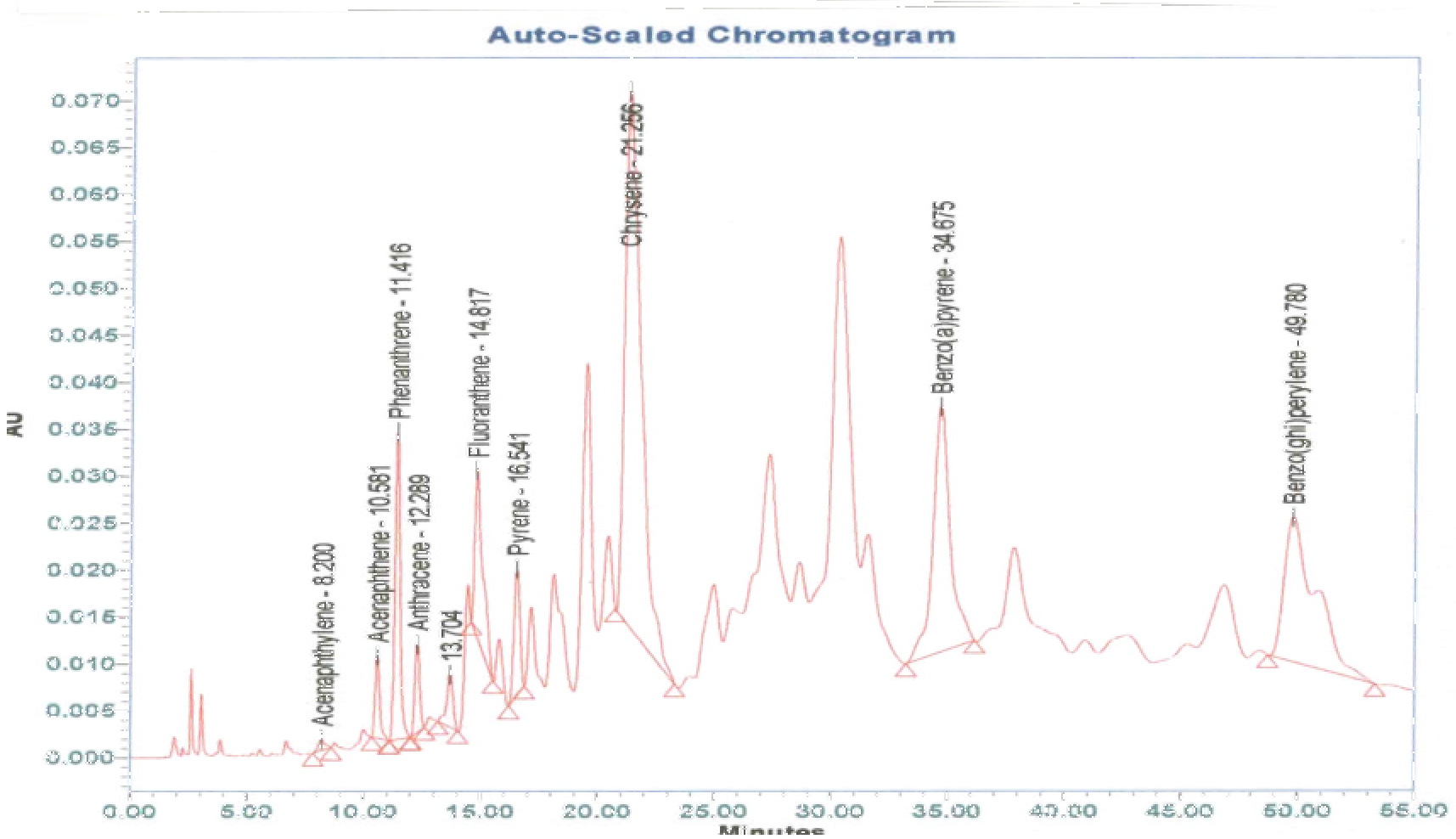


Figure-4: Comparison Of Ambient Air RSPM, SPM, SO_2 & NO_X Levels in Two Seasons

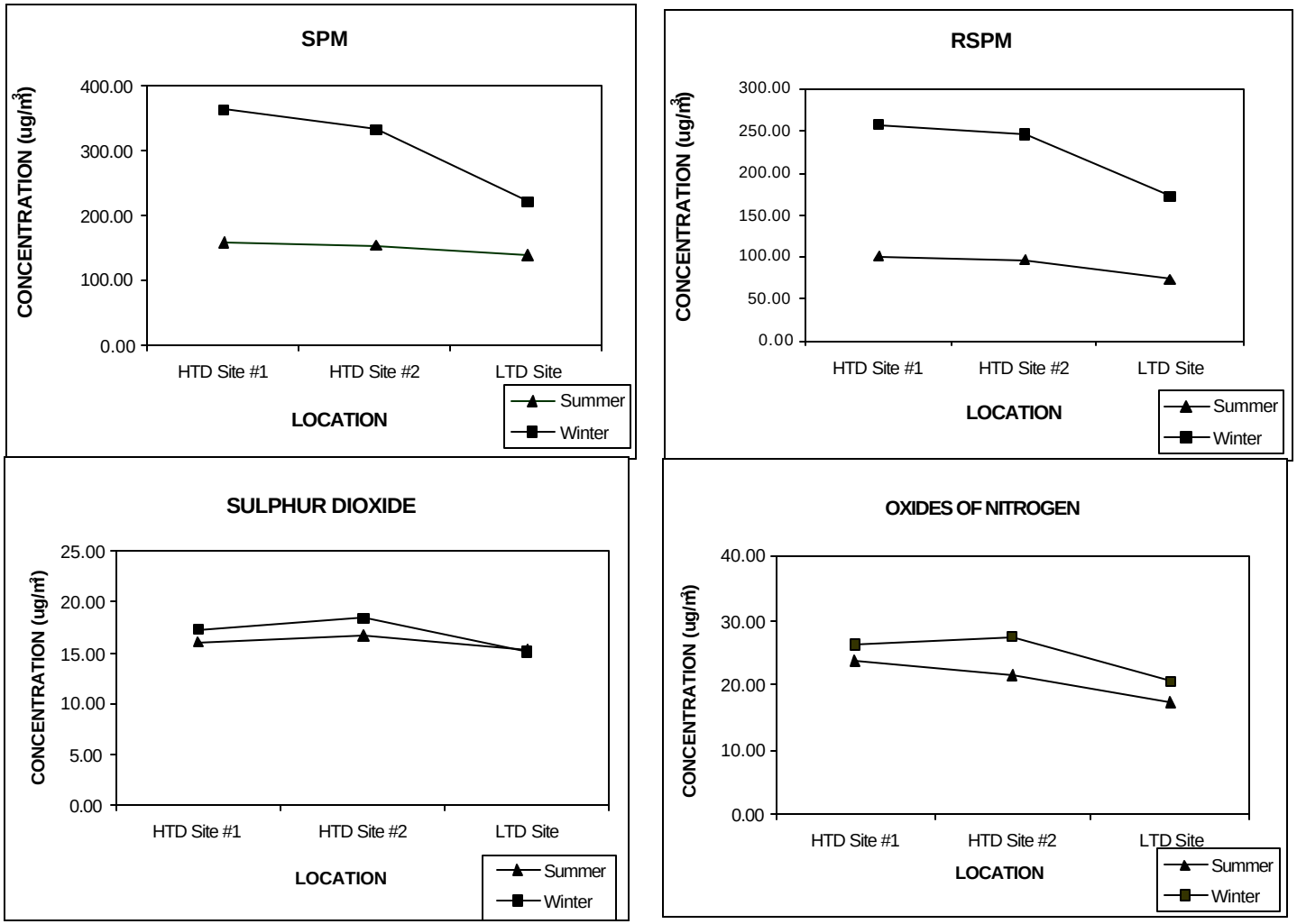


Figure 5: Profile of Ambient Air PAHs at HTD site #1 in summer

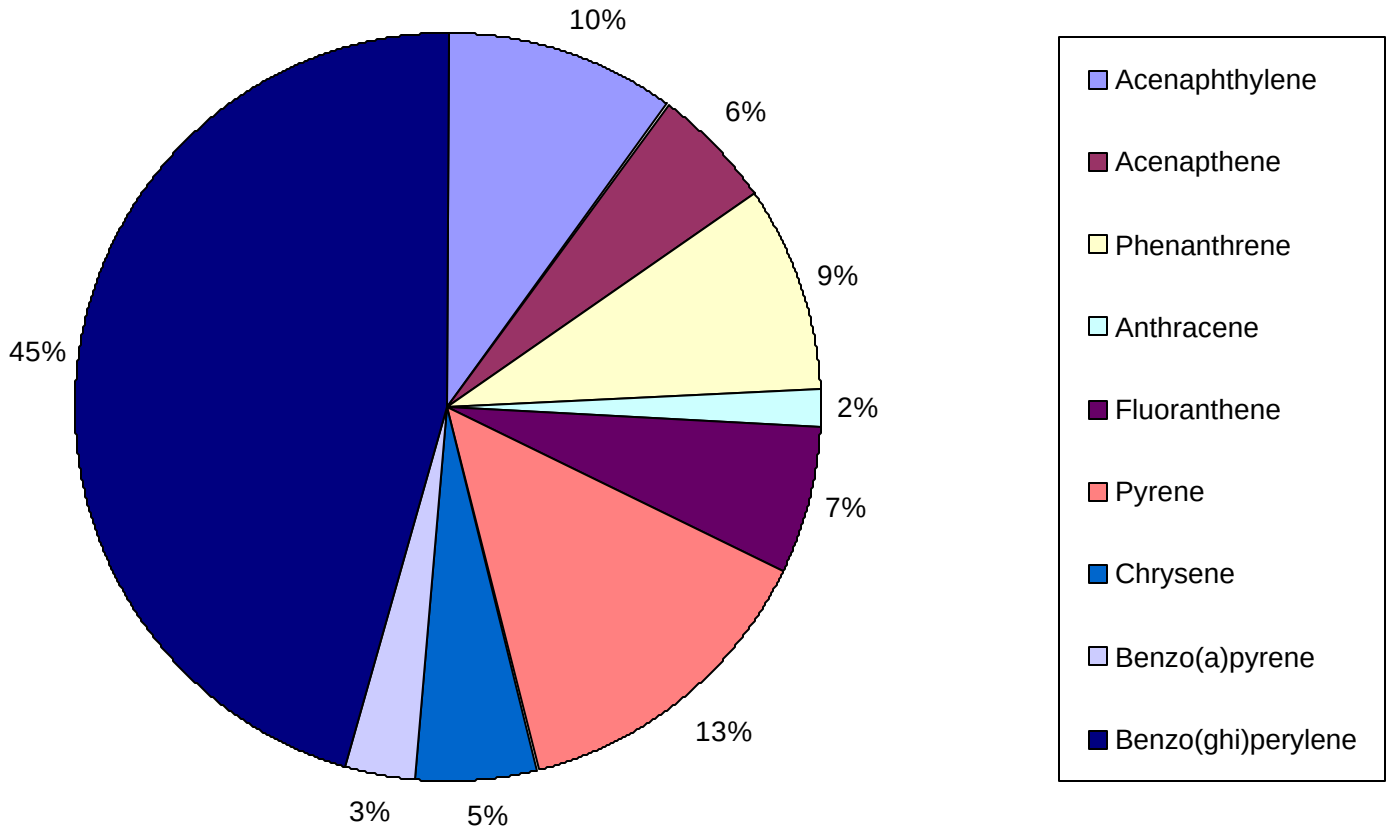


Figure 6: Profile of Ambient Air PAHs at HTD site #2 in summer

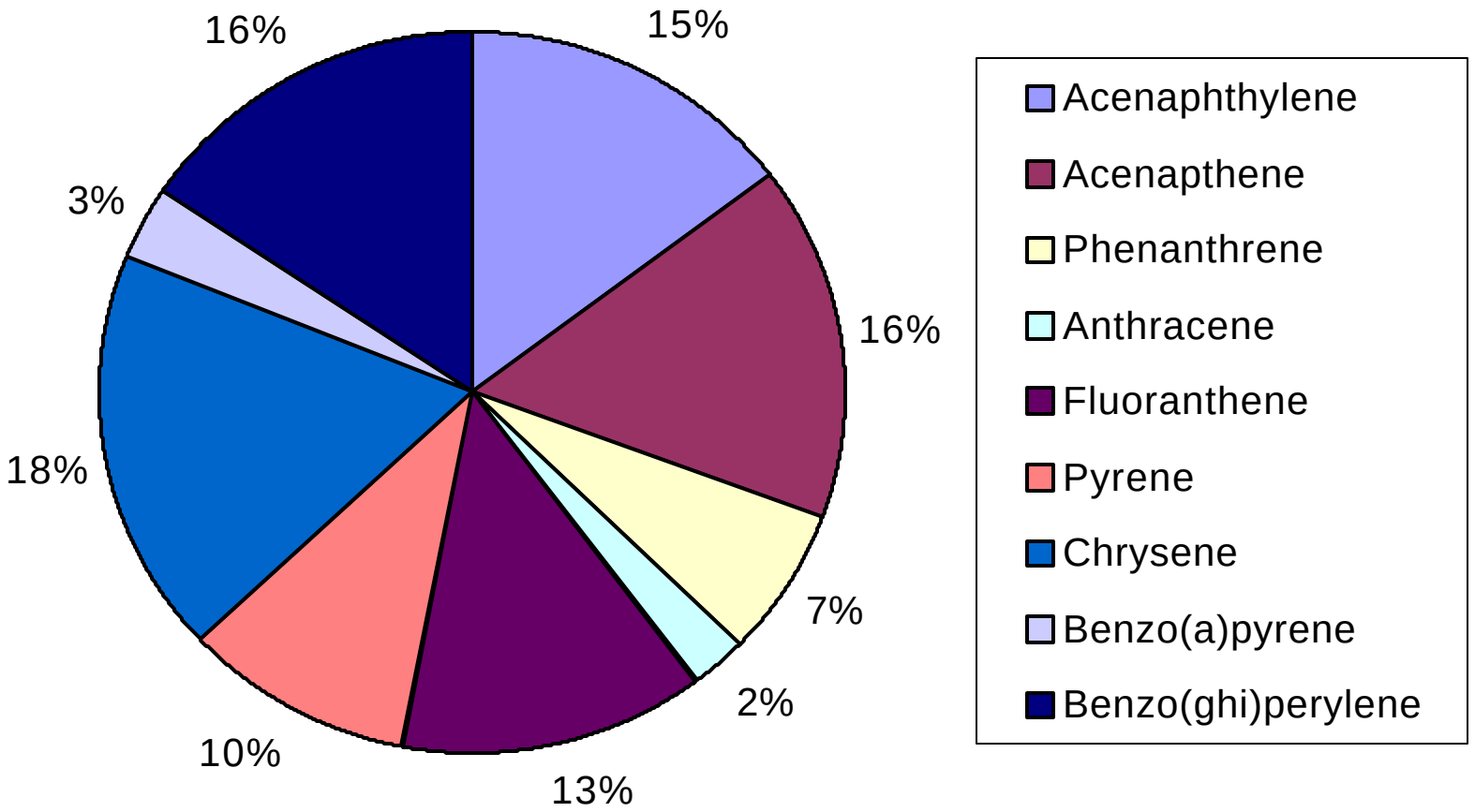
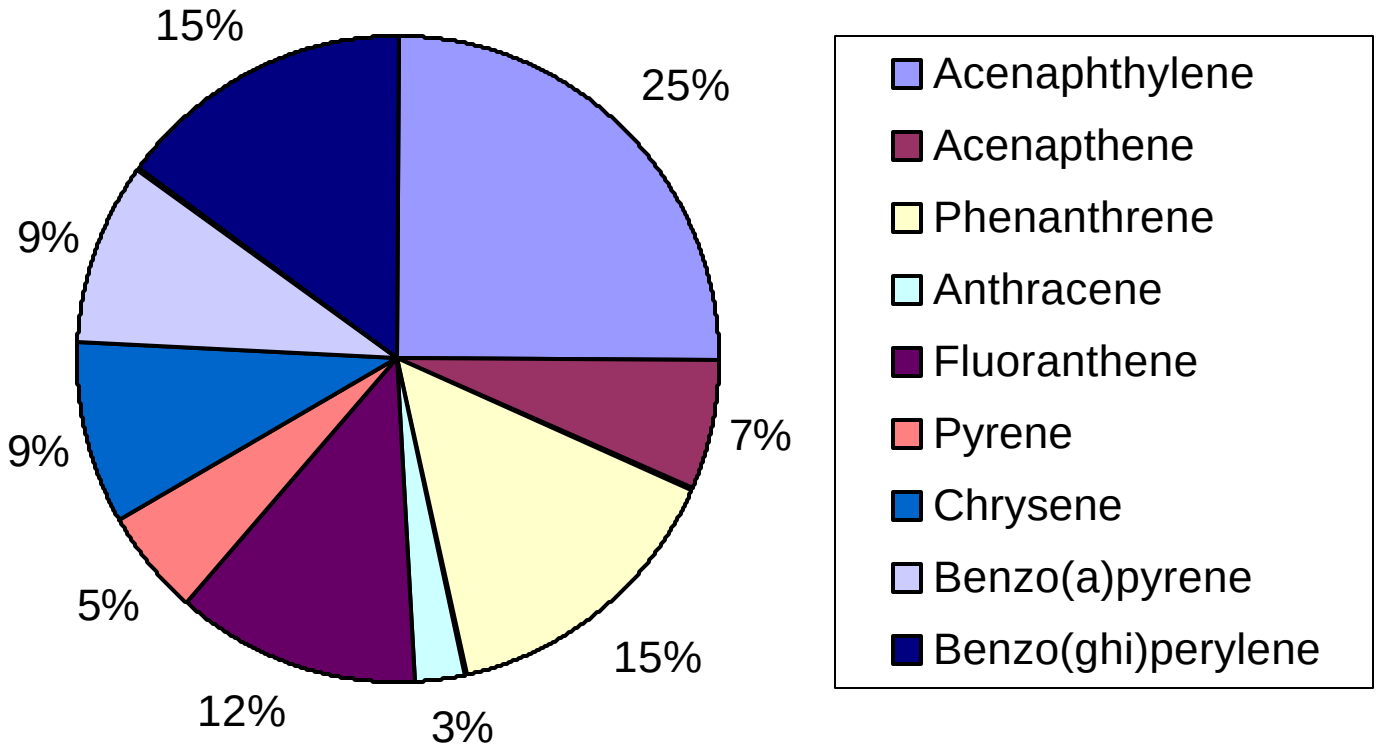


Figure 7: Profile of Ambient Air PAHs at LTD site in summer



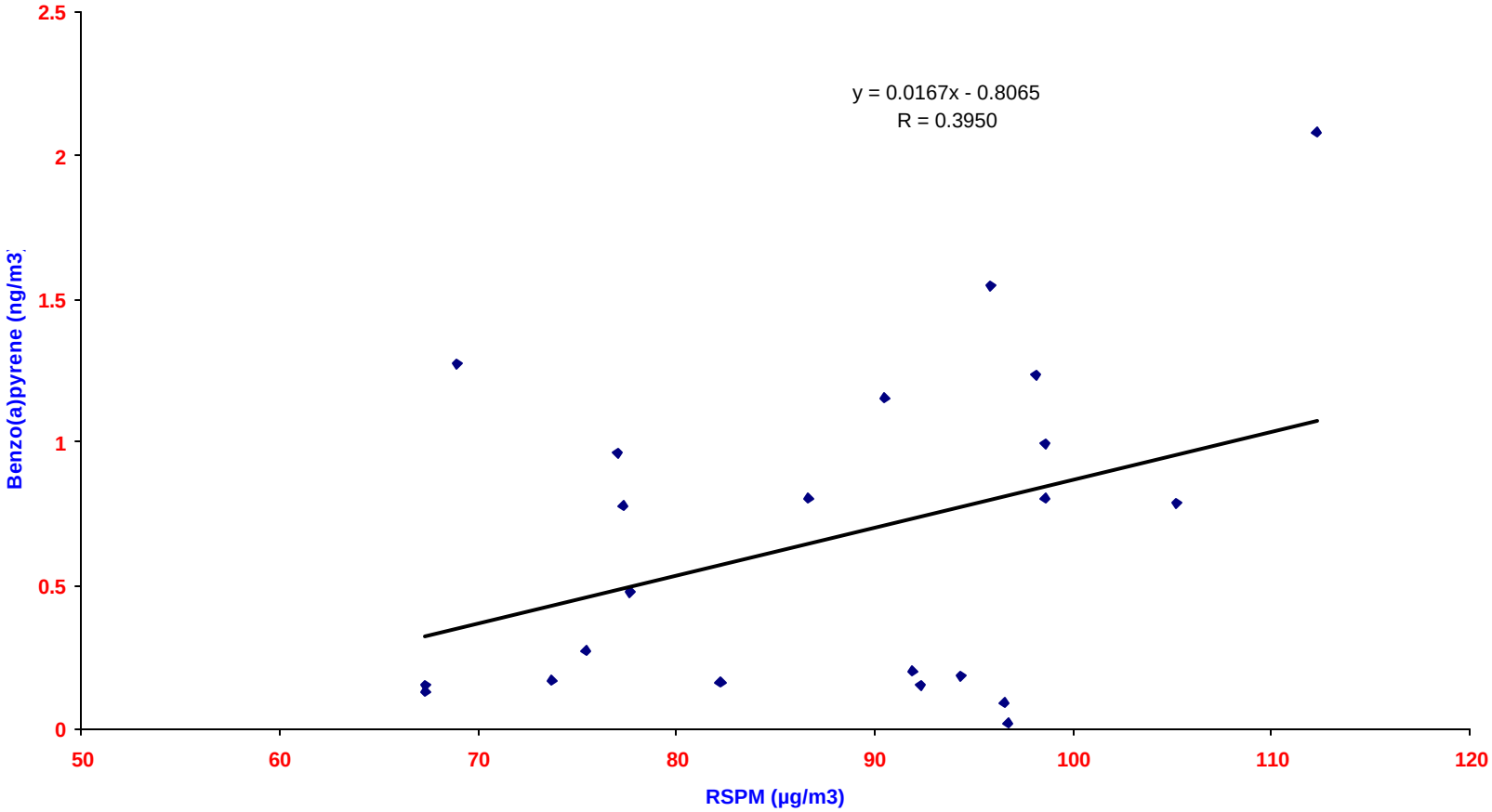
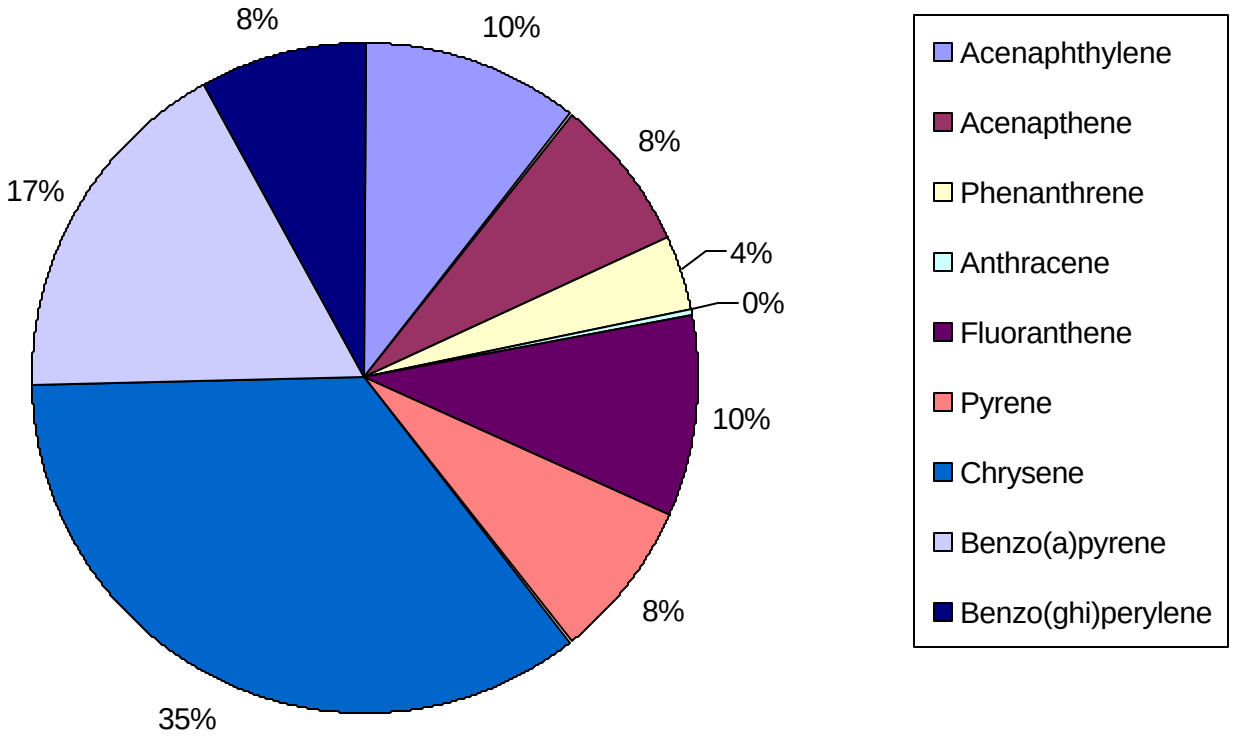


Figure 8: Correlation plot of Ambient Air RSPM and Benzo (a) Pyrene In summer. Regression line with intercept – 0.81, equation y = 0.0167x- 0.8065, r =0.395*, p 0.05 (Pearson's correlation), significant (2 tailed) correlation, n=22

Figure 9: Profile of Ambient Air PAHs at HTD site #1 in winter



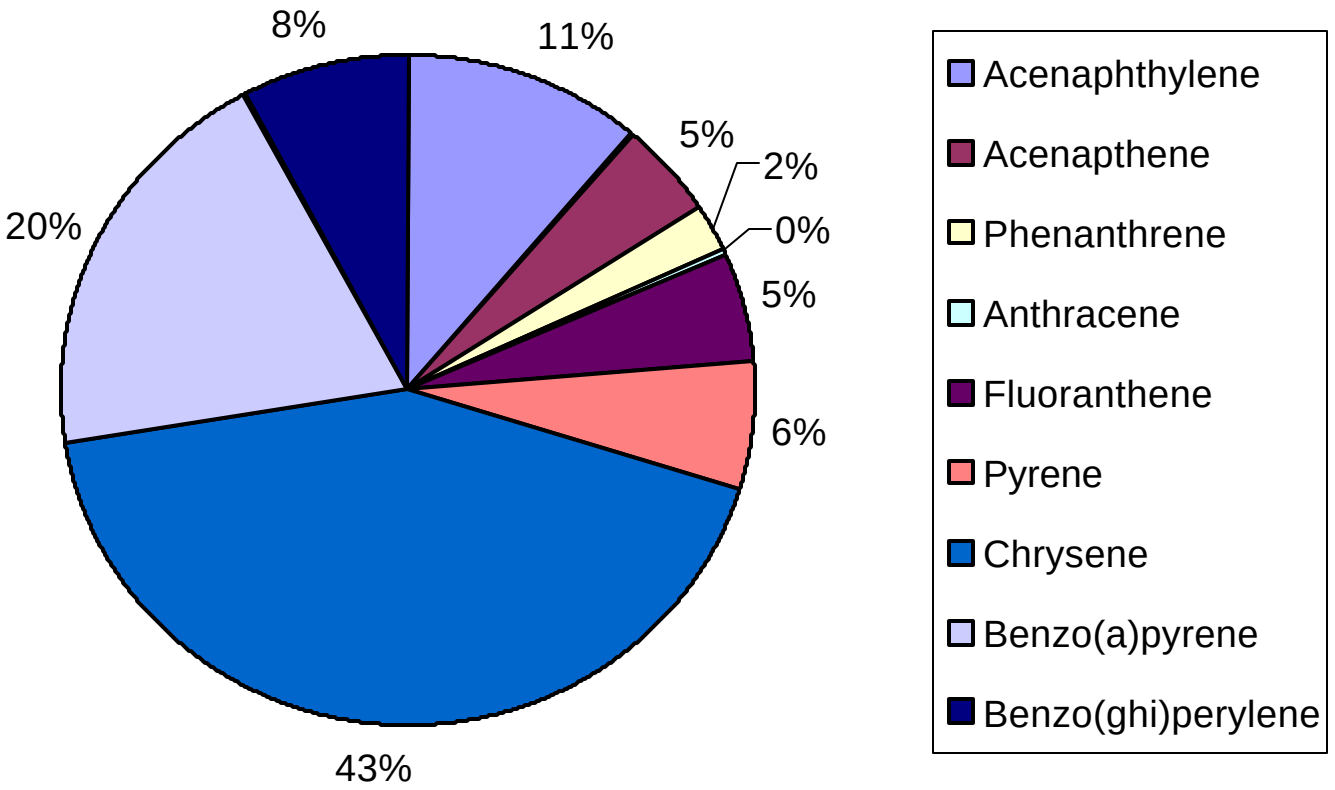


Figure 10: Profile of Ambient Air PAHs at HTD site #2 in winter

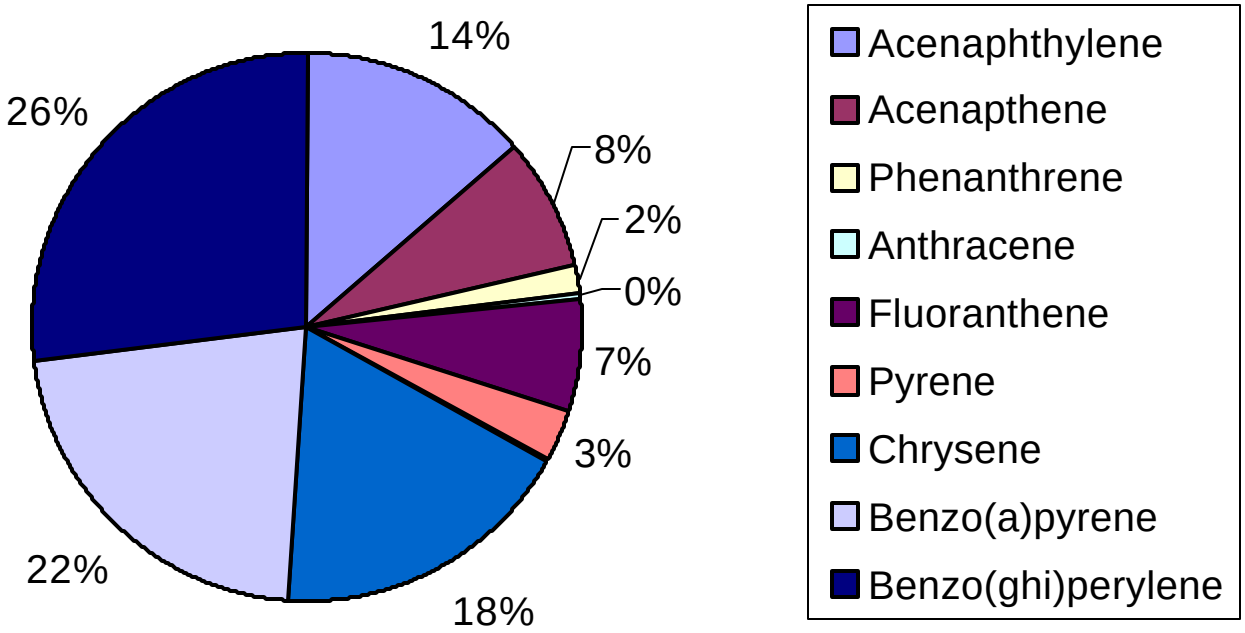


Figure 11: Profile of Ambient Air PAHs at LTD site in winter

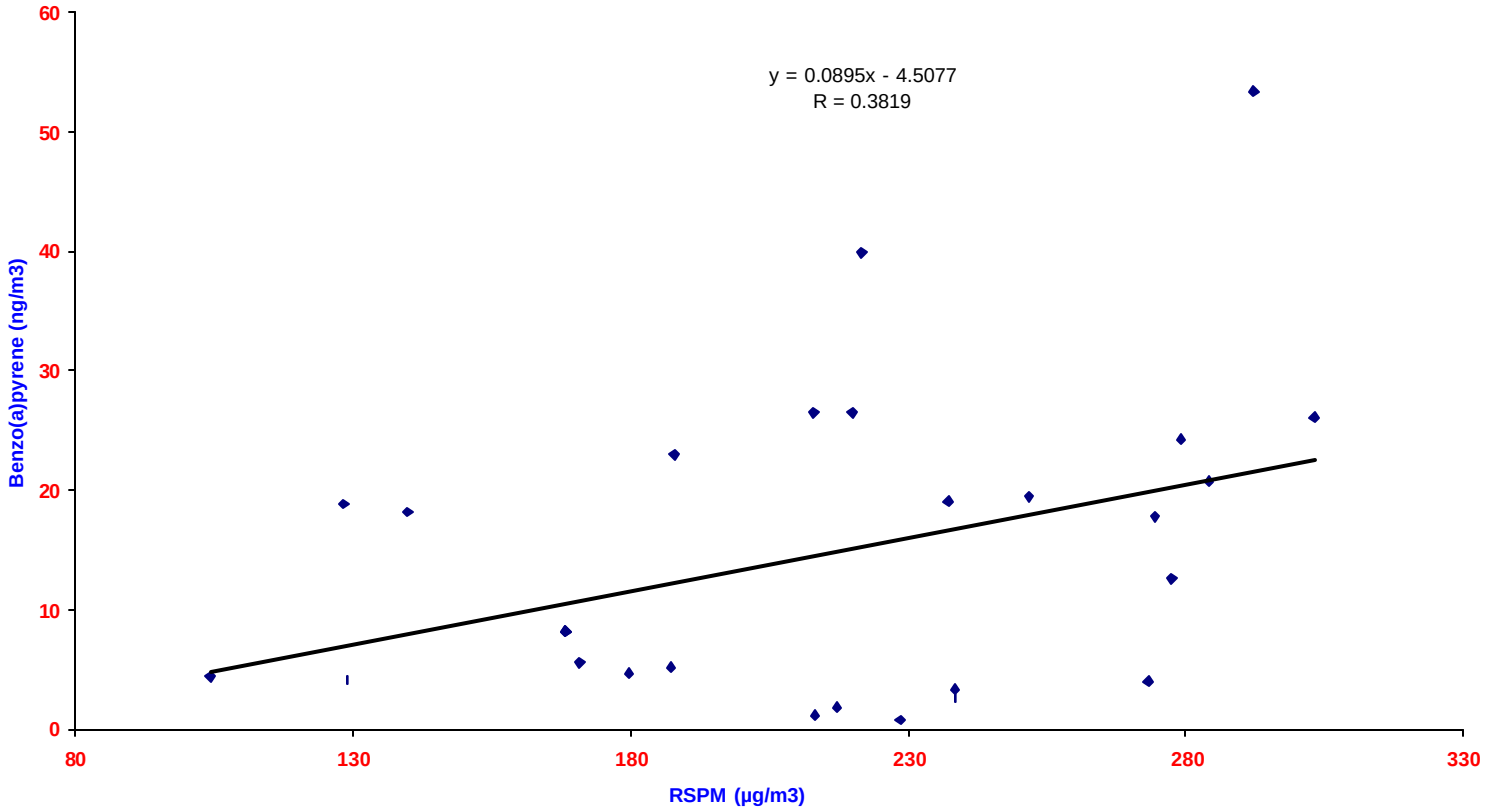
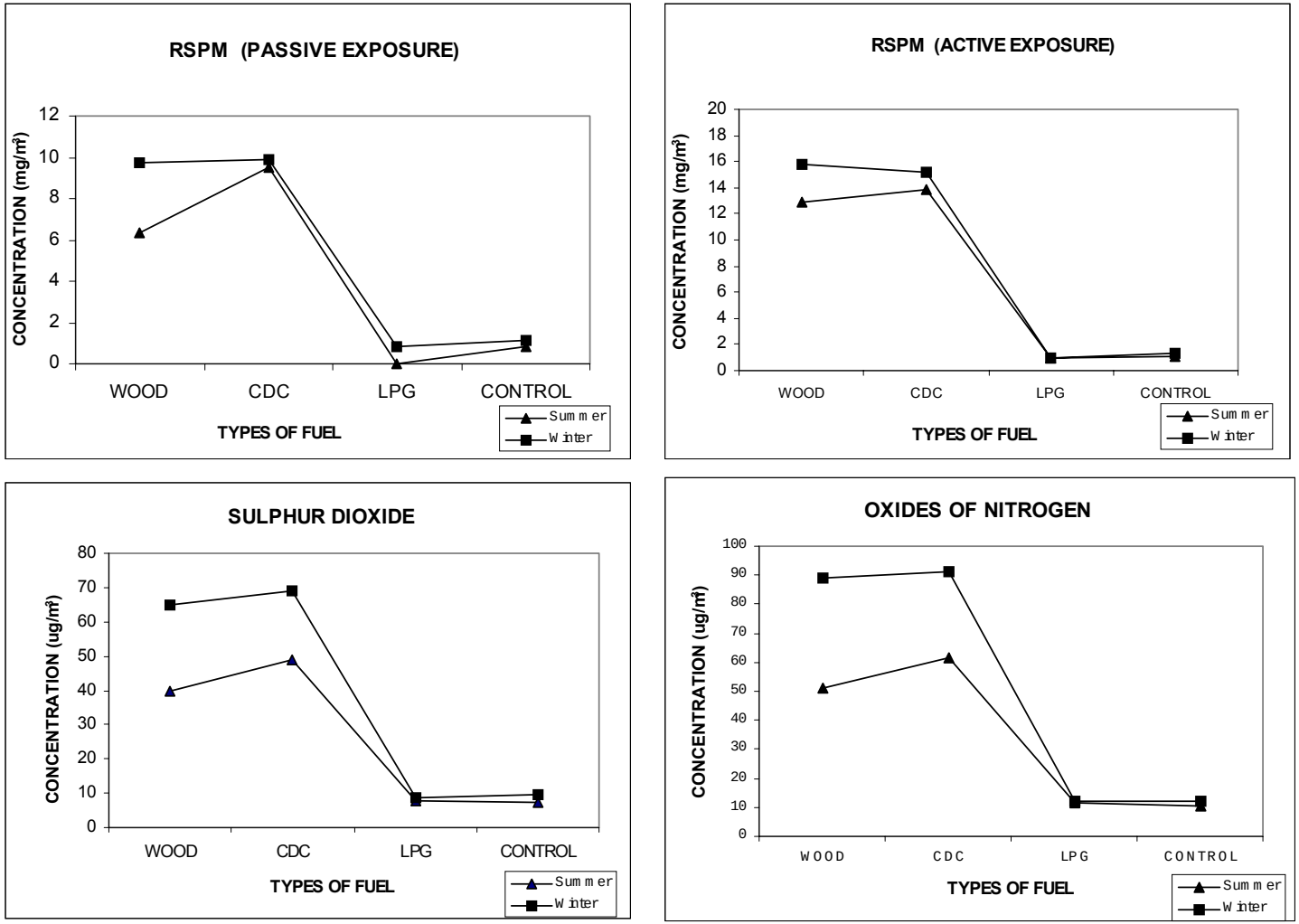


Figure 12: Correlation plot of Ambient Air RSPM and Benzo (a) Pyrene In winter. Regression line with intercept –4.5, equation y=0.0895x-4.5007, r=0.382, p 0.05 (Pearson's correlation), significant (2 tailed) correlation, n=24

Figure-13: COMPARISON OF RURAL INDOOR AIR RSPM, SO_2 AND NOx LEVELS IN TWO SEASONS



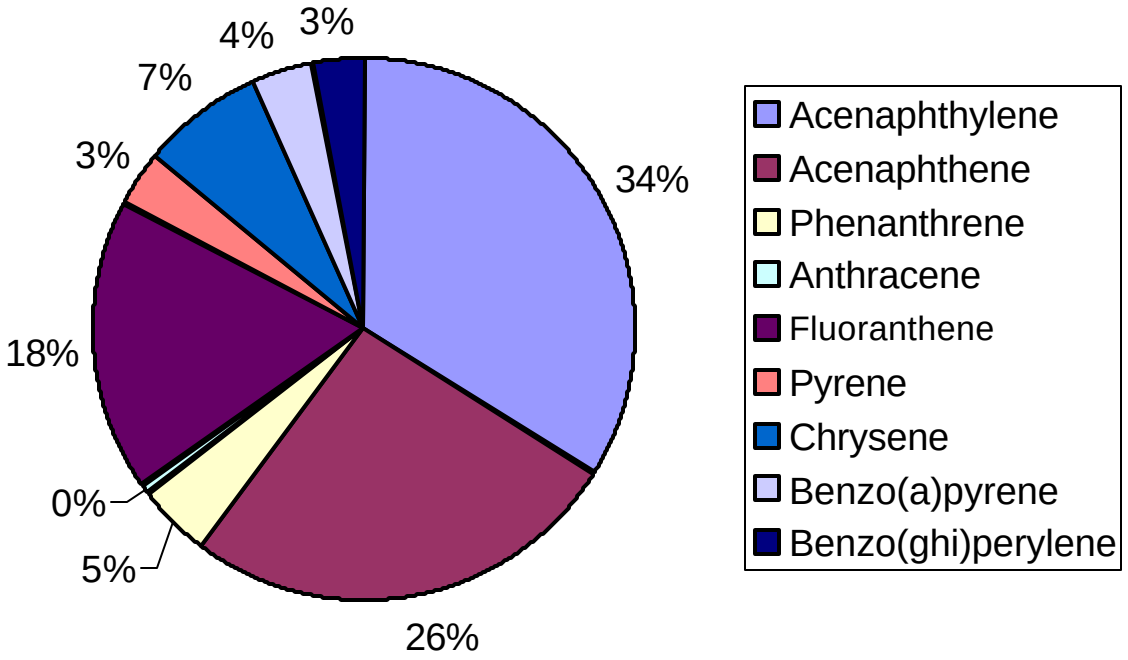


Figure 14: Profile of Indoor Air PAHs during Passive Exposure at Fuel Wood using site in summer

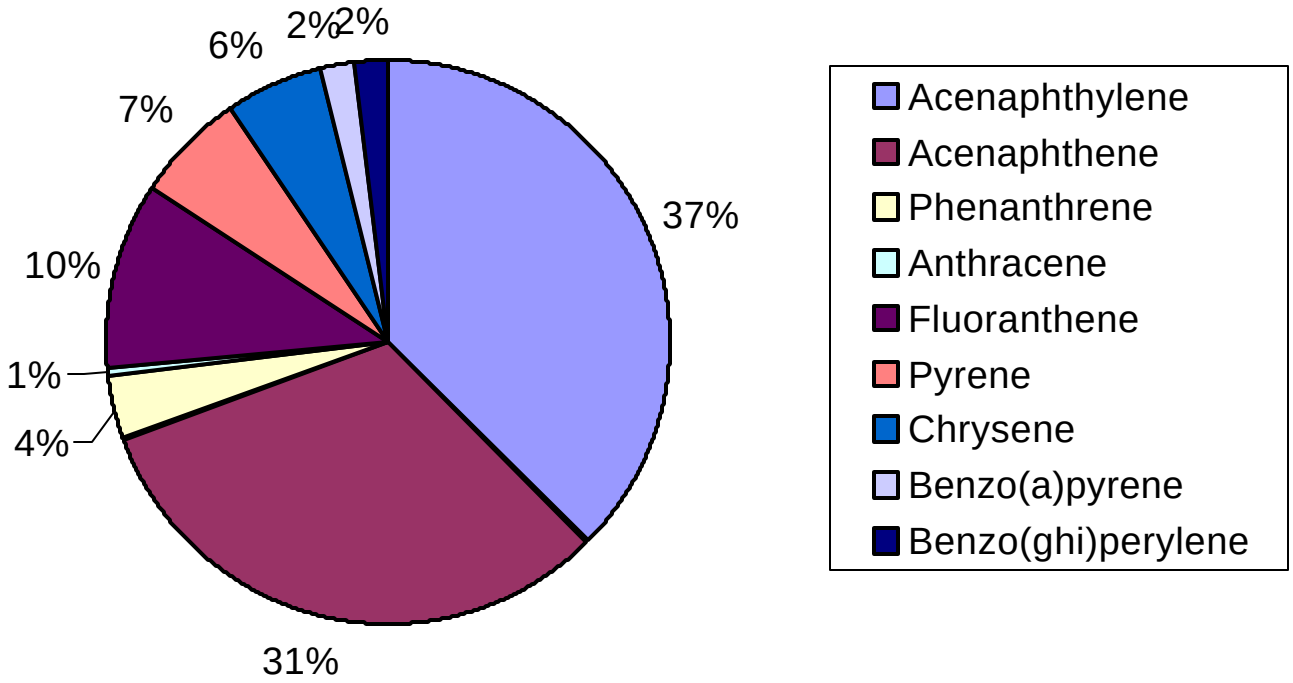


Figure-15: Profile of Indoor Air PAHs during Passive Exposure at Fuel CDC using site in summer

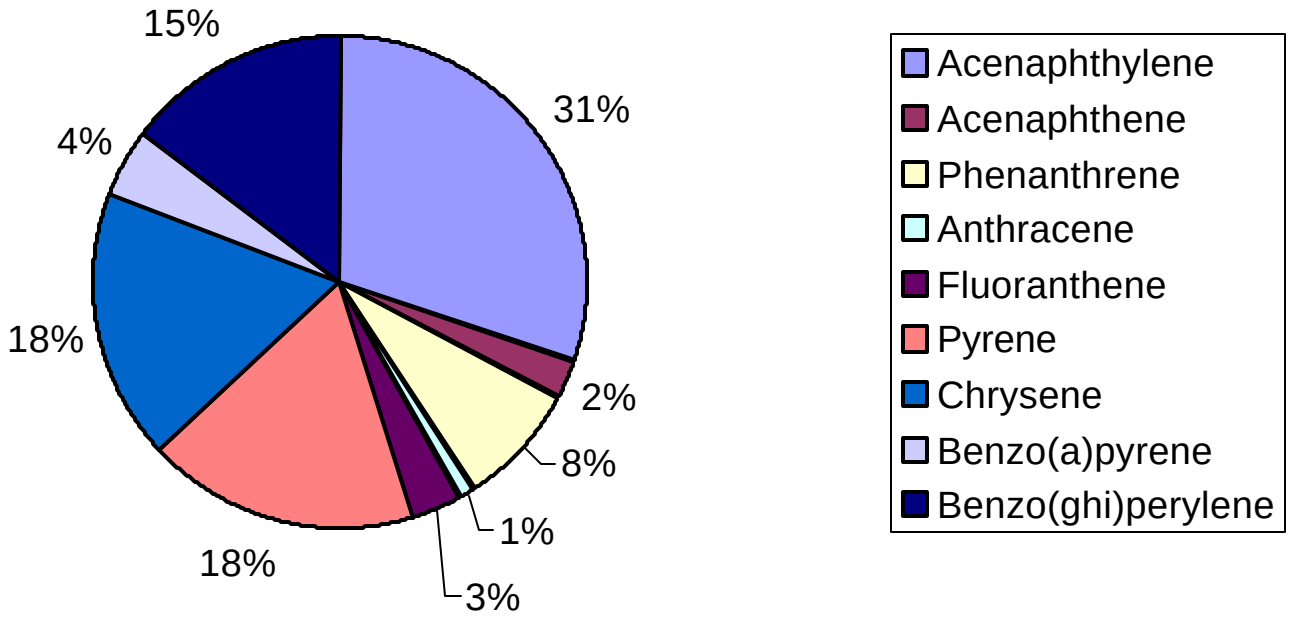


Figure-16: Profile of Indoor Air PAHs during Passive Exposure at Fuel LPG using site in summer

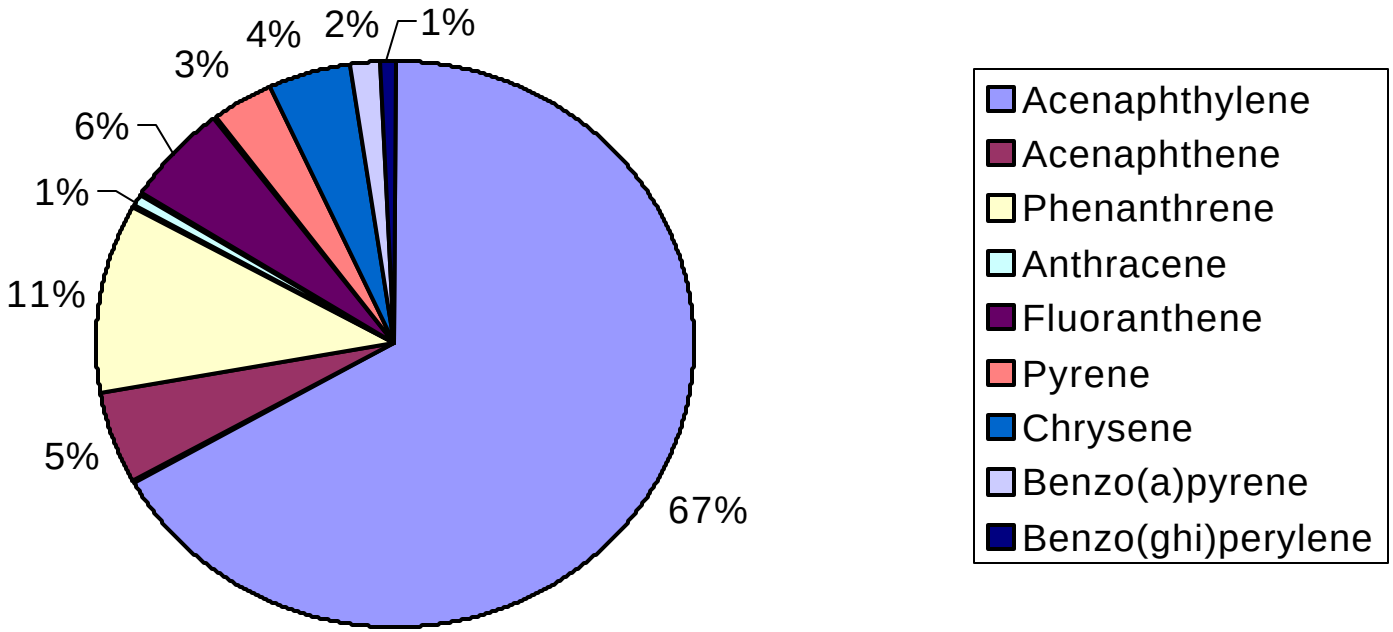


Figure-17: Profile of Indoor Air PAHs during Passive Exposure at the Non-cooking site in summer

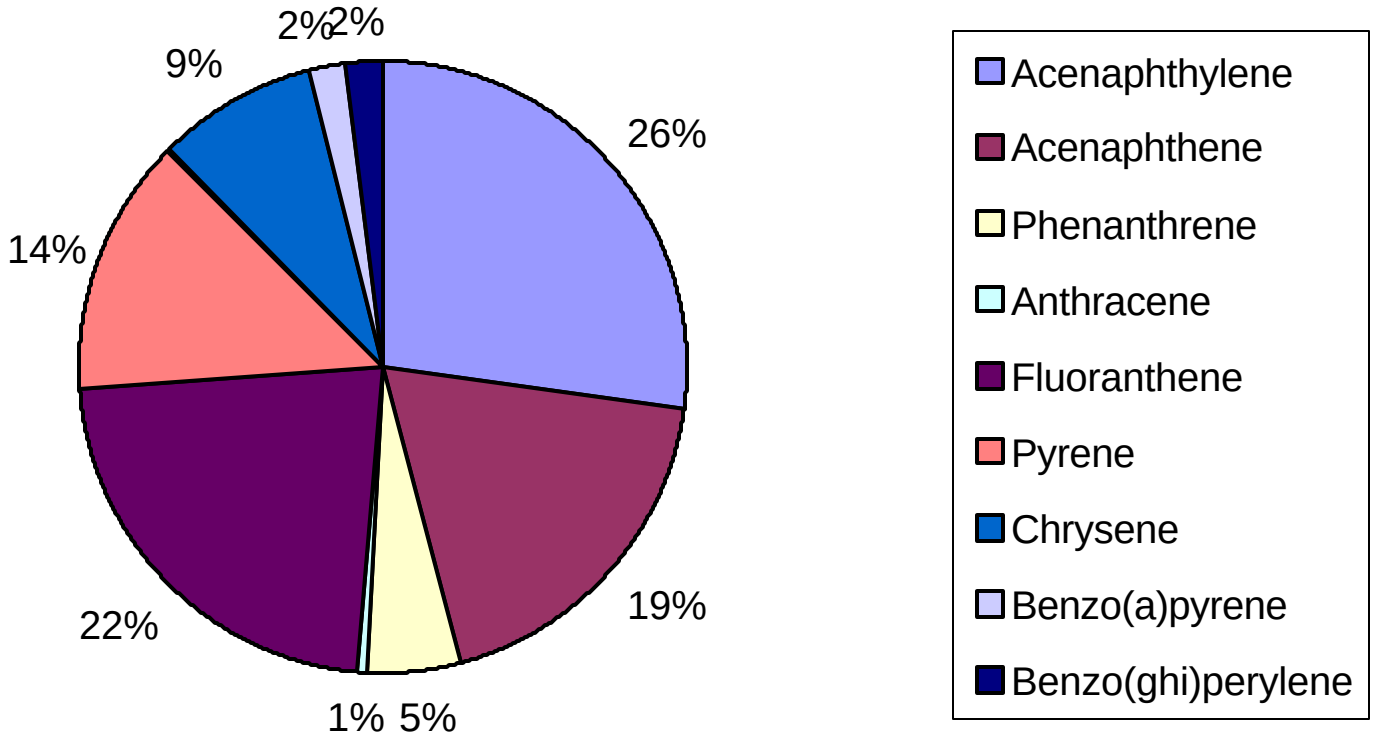


Figure-22: Profile of Indoor Air PAHs during Active Exposure at Fuel Wood using site in summer

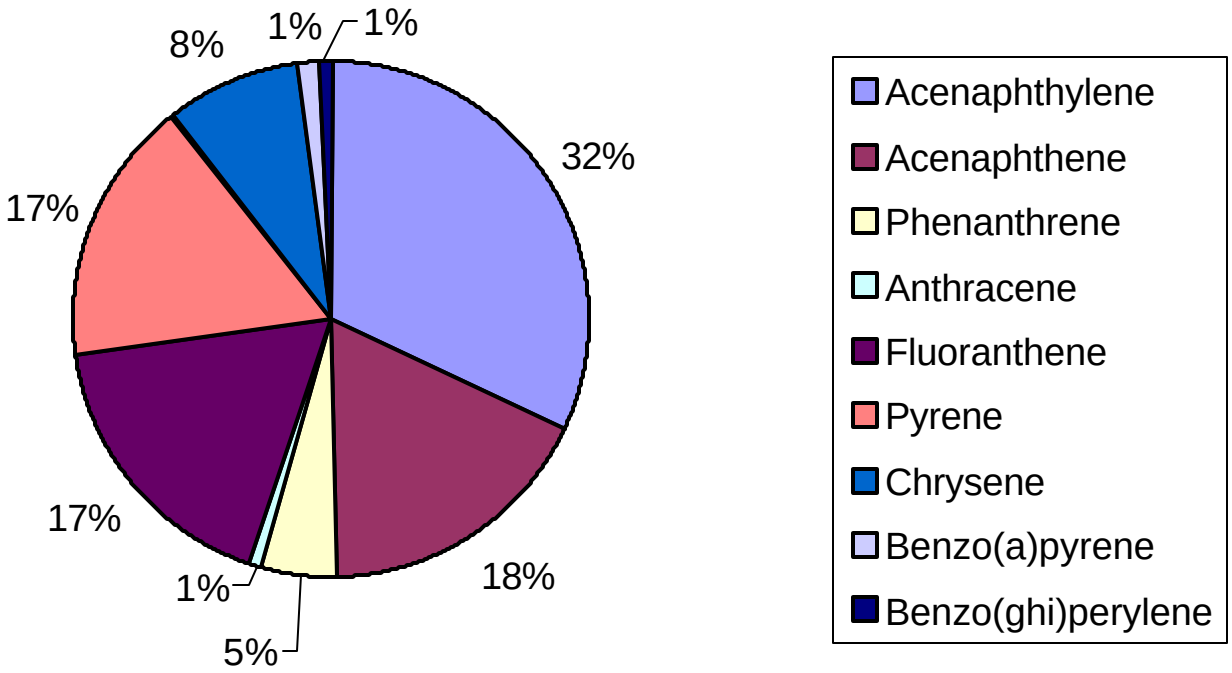


Figure-23: Profile of Indoor Air PAHs during Active Exposure at Fuel CDC using site in summer

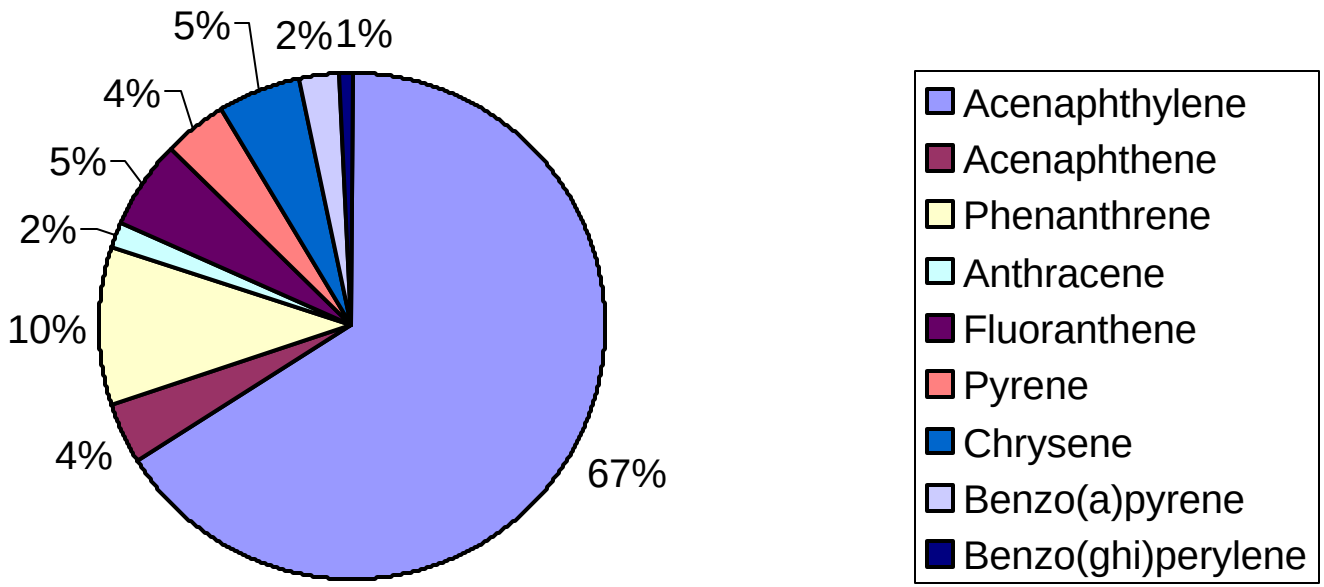


Figure-24: Profile of Indoor Air PAHs during Active Exposure at Fuel LPG using site in summer

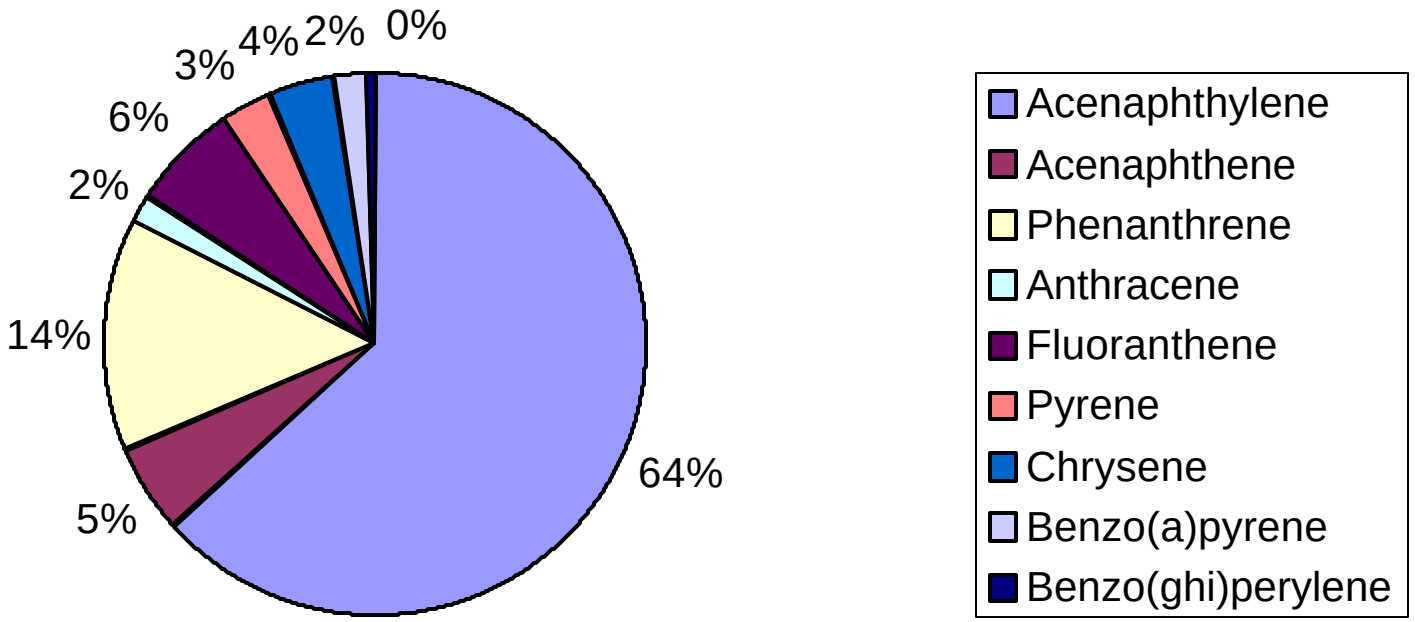


Figure-25: Profile of Indoor Air PAHs during Active Exposure at the Non-cooking site in summer

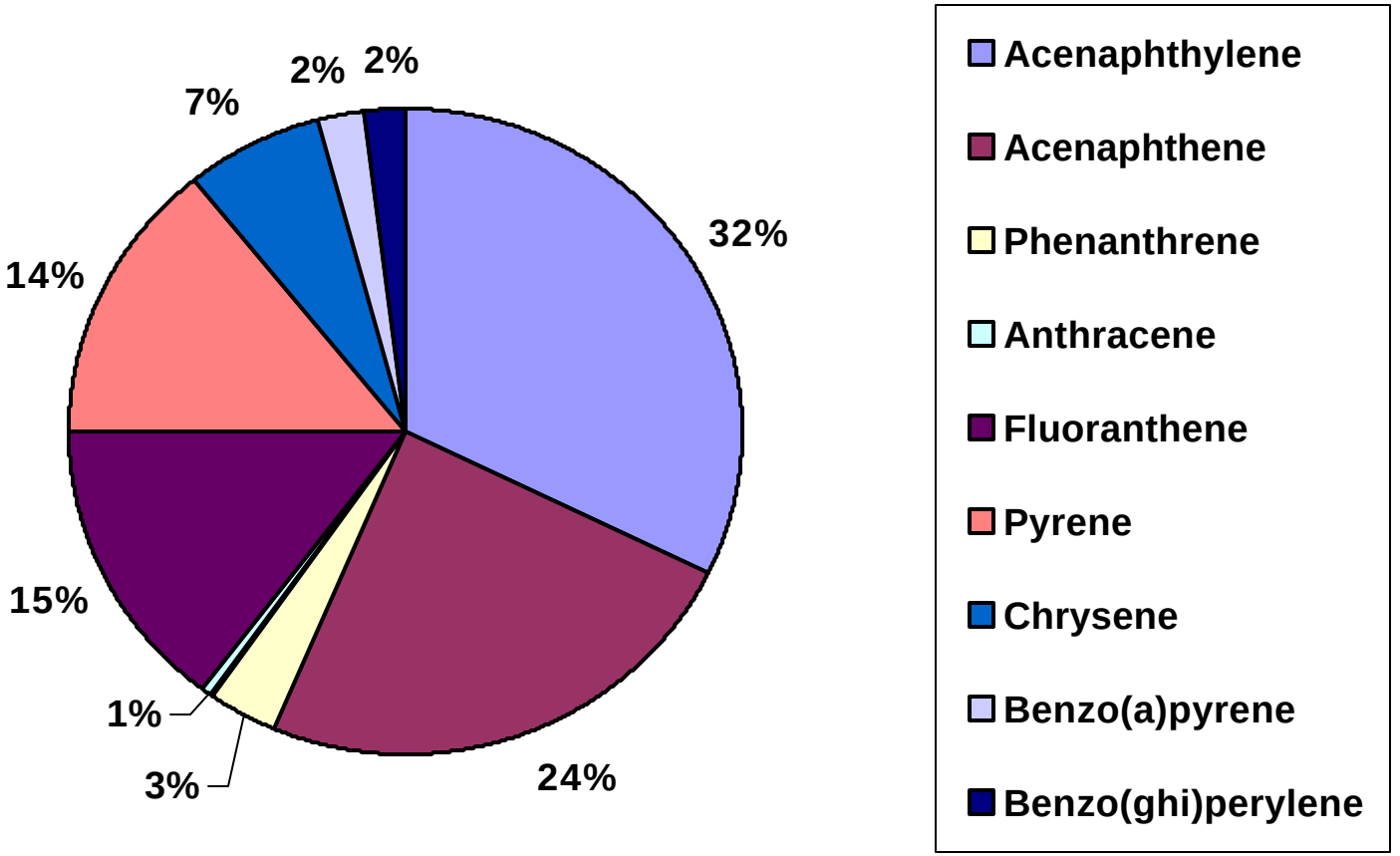


Figure-26: Profile of Indoor Air PAHs during Active Exposure at Fuel Wood using site in winter

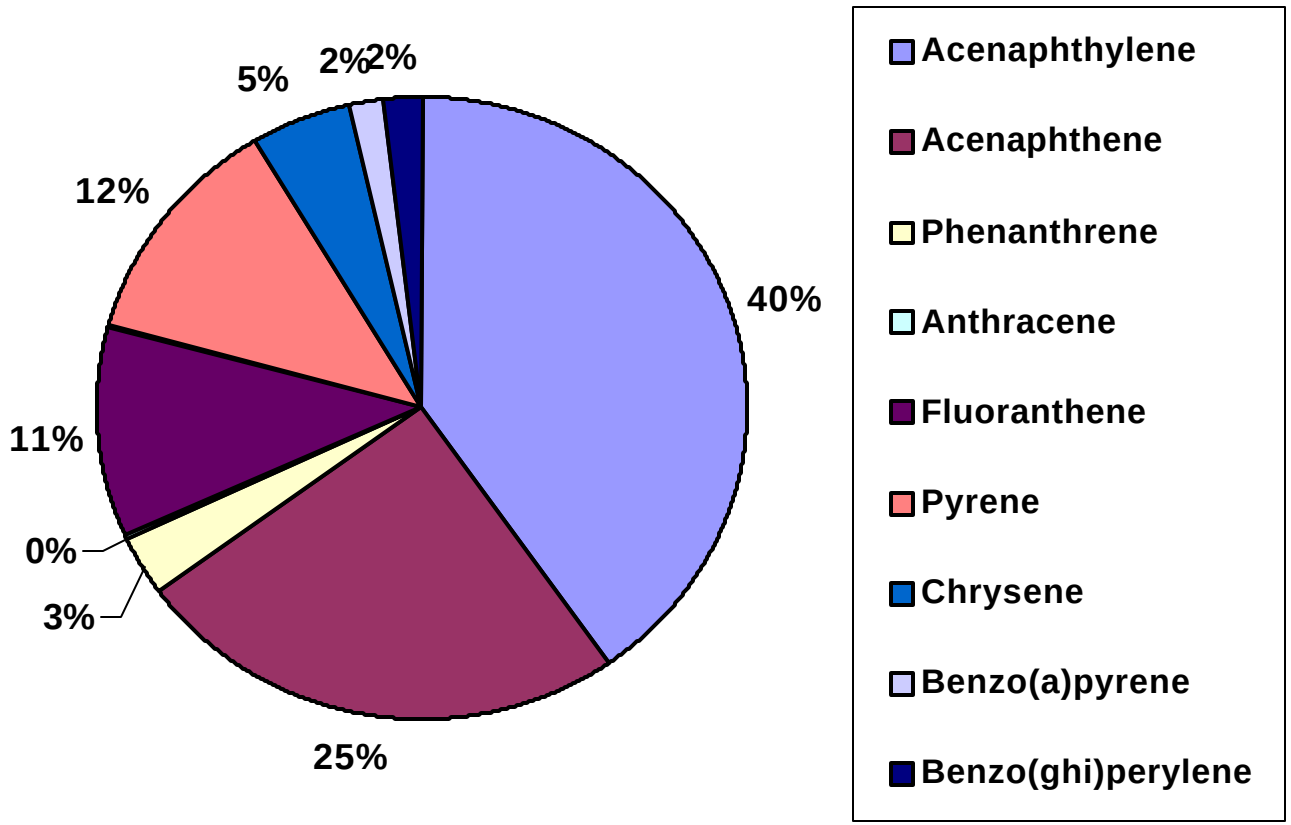


Figure 18: Profile of Indoor Air PAHs during Passive Exposure at Fuel Wood using site in winter

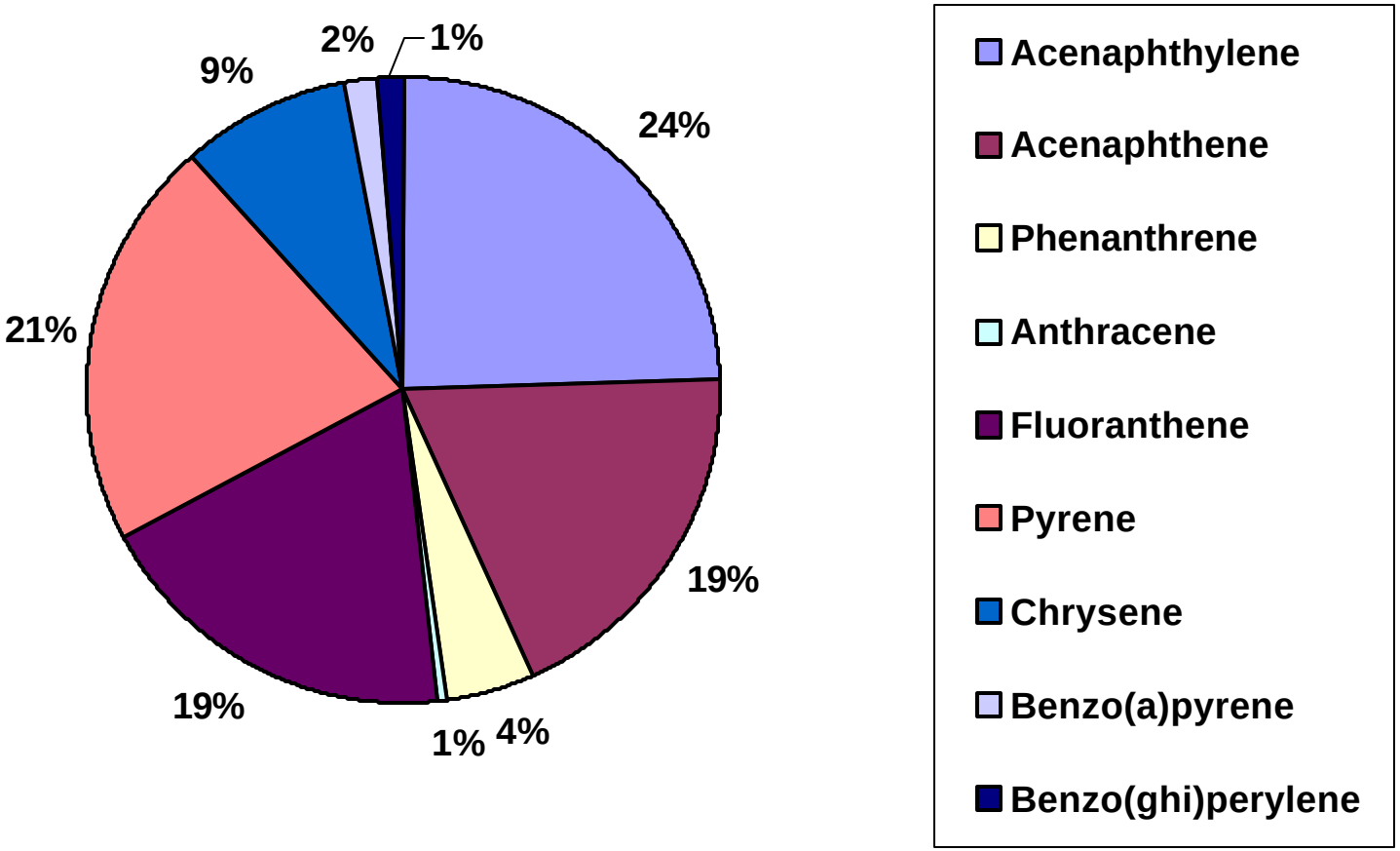


Figure-27: Profile of Indoor Air PAHs during Active Exposure at Fuel CDC using site in winter

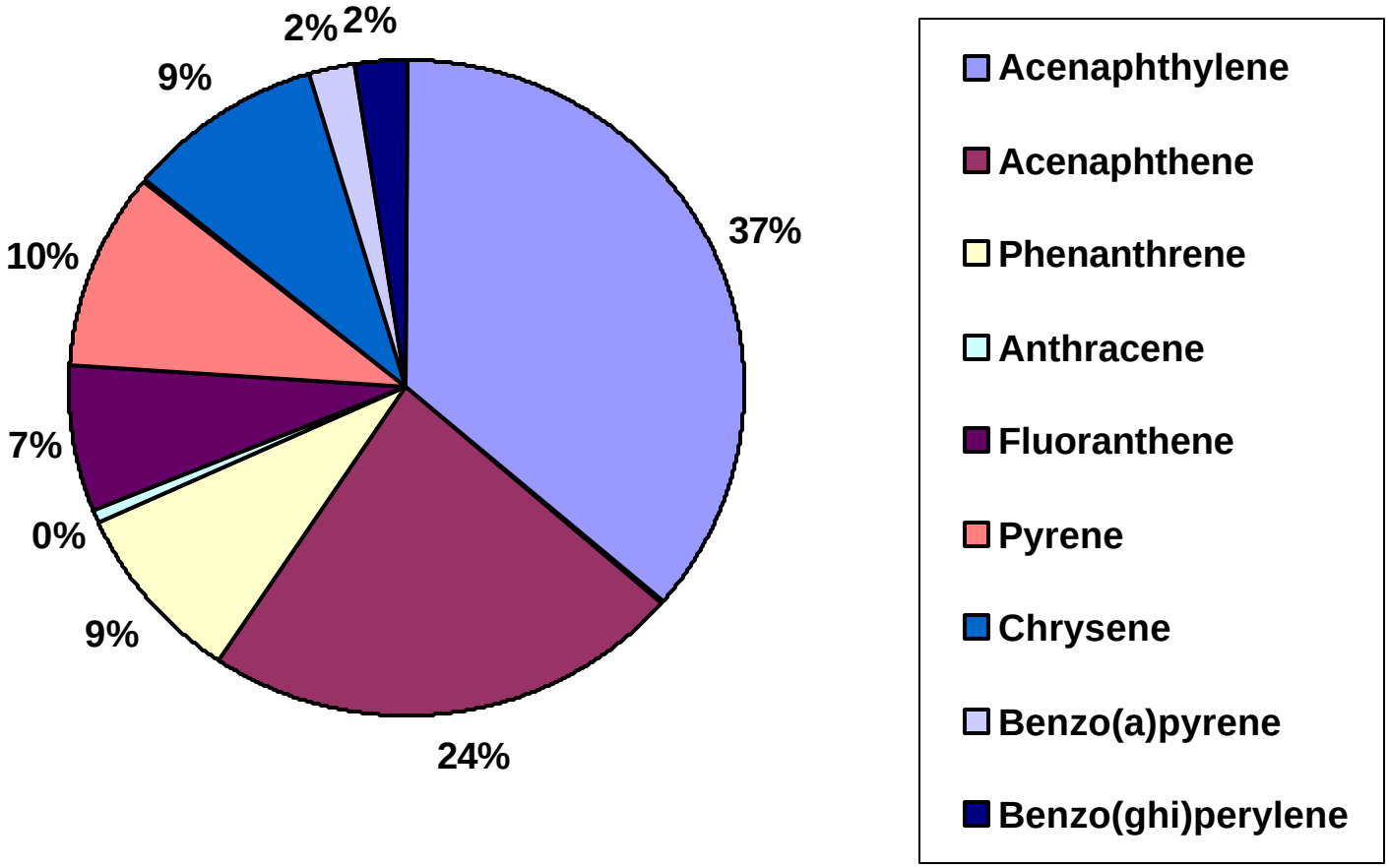


Figure-19: Profile of Indoor Air PAHs during Passive Exposure at Fuel CDC using site in winter

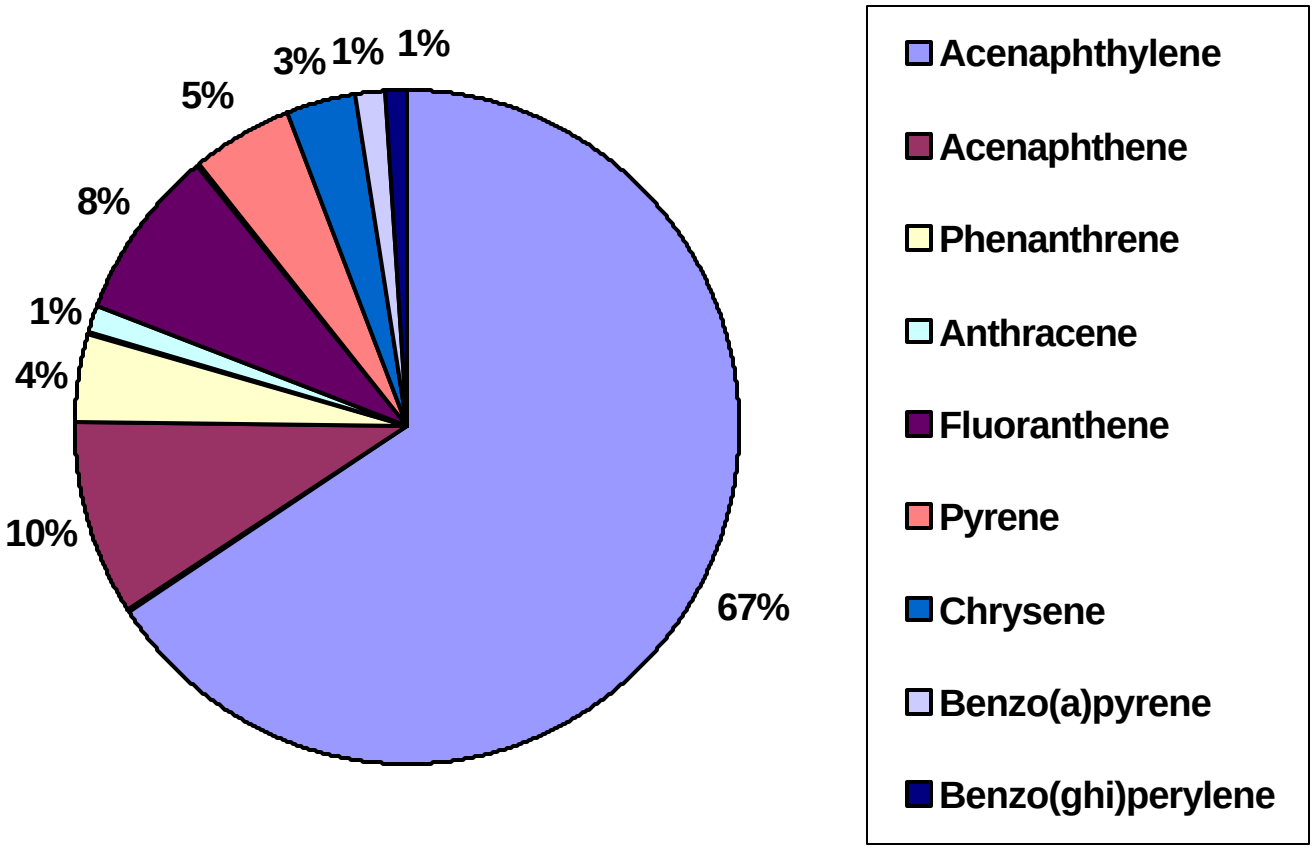


Figure-28: Profile of Indoor Air PAHs during Active Exposure at Fuel LPG using site in winter

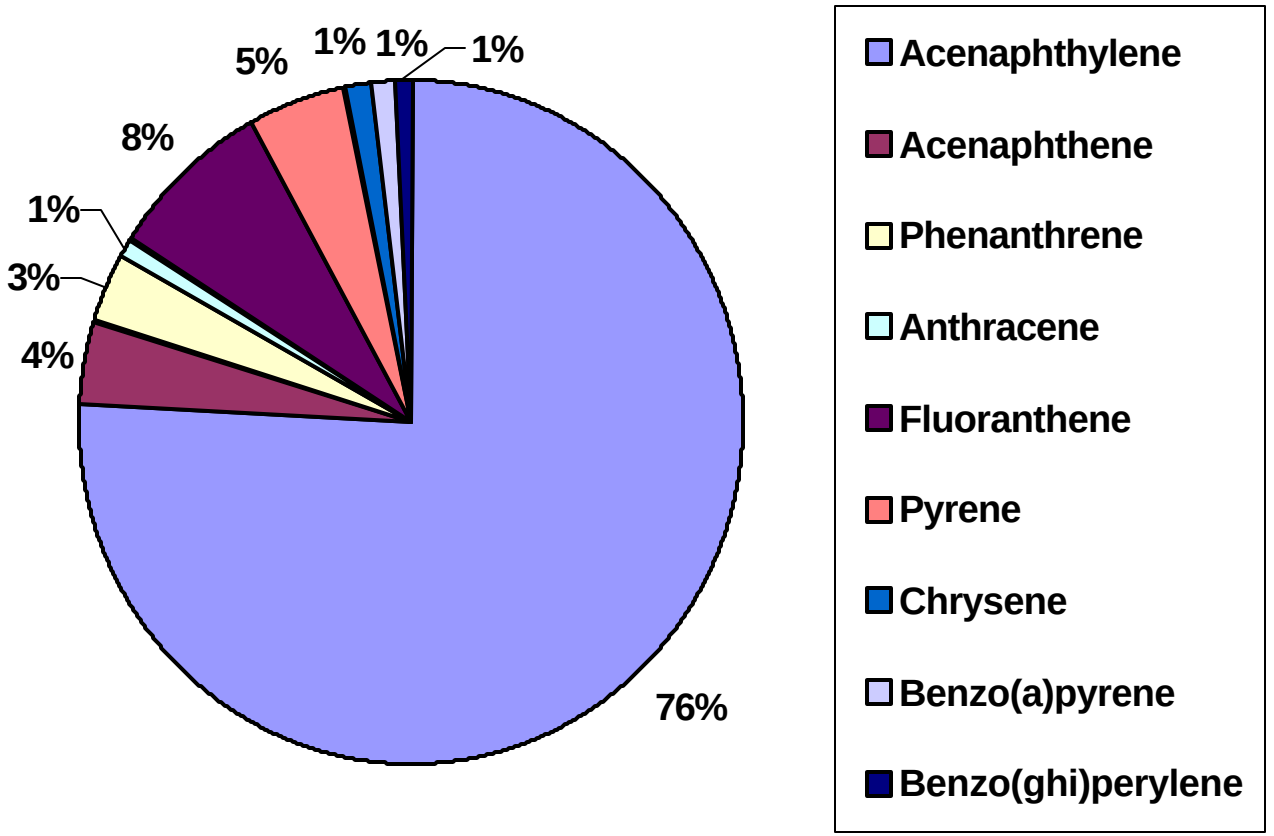


Figure-20: Profile of Indoor Air PAHs during Passive Exposure at Fuel LPG using site in winter

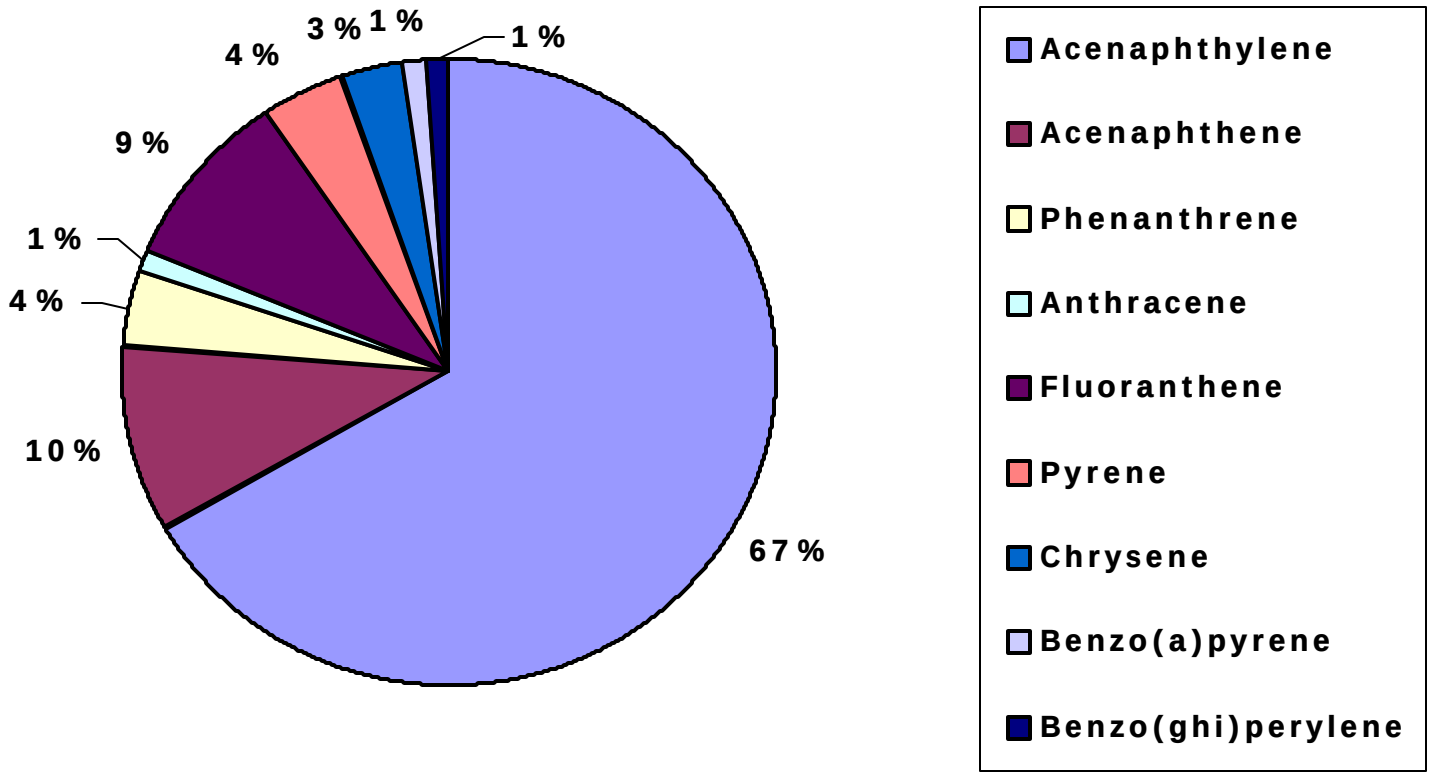


Figure-29: Profile of Indoor Air PAHs during Active Exposure at Non-Cooking site in winter

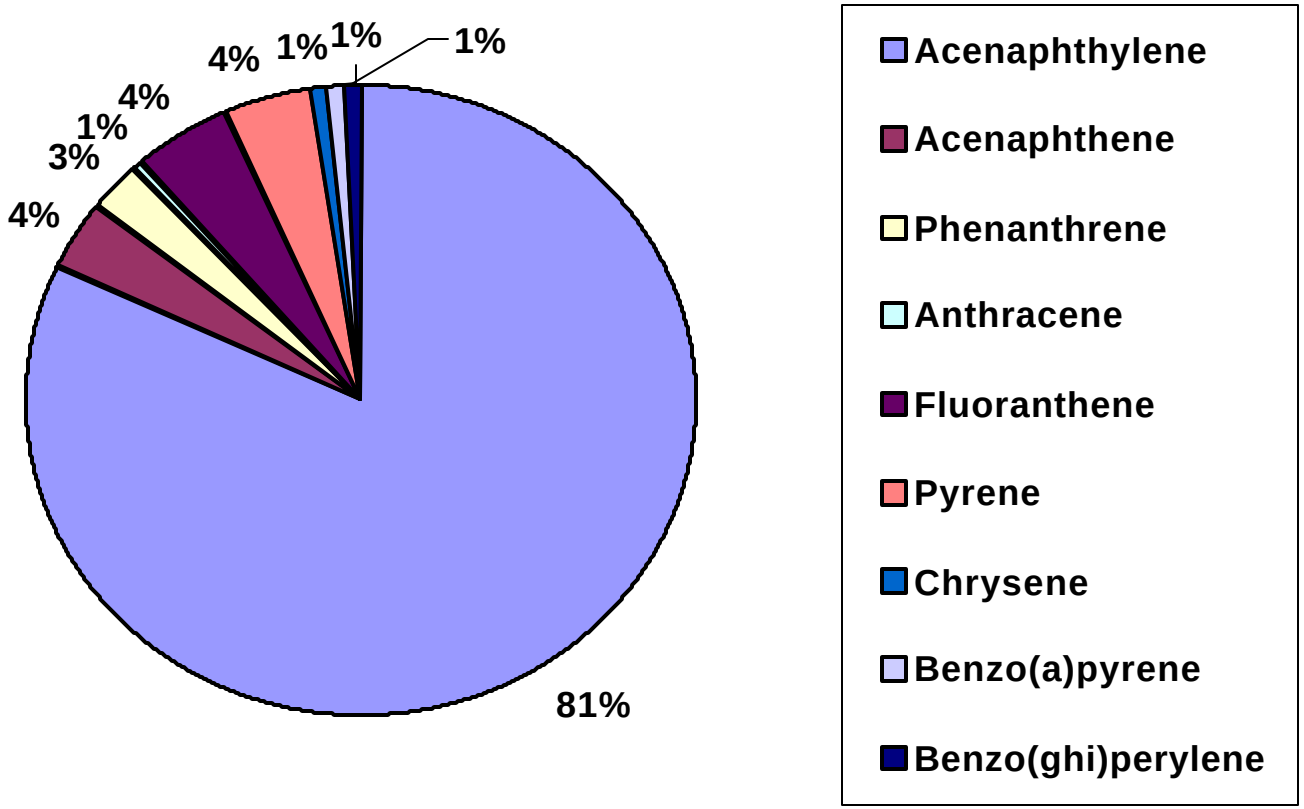
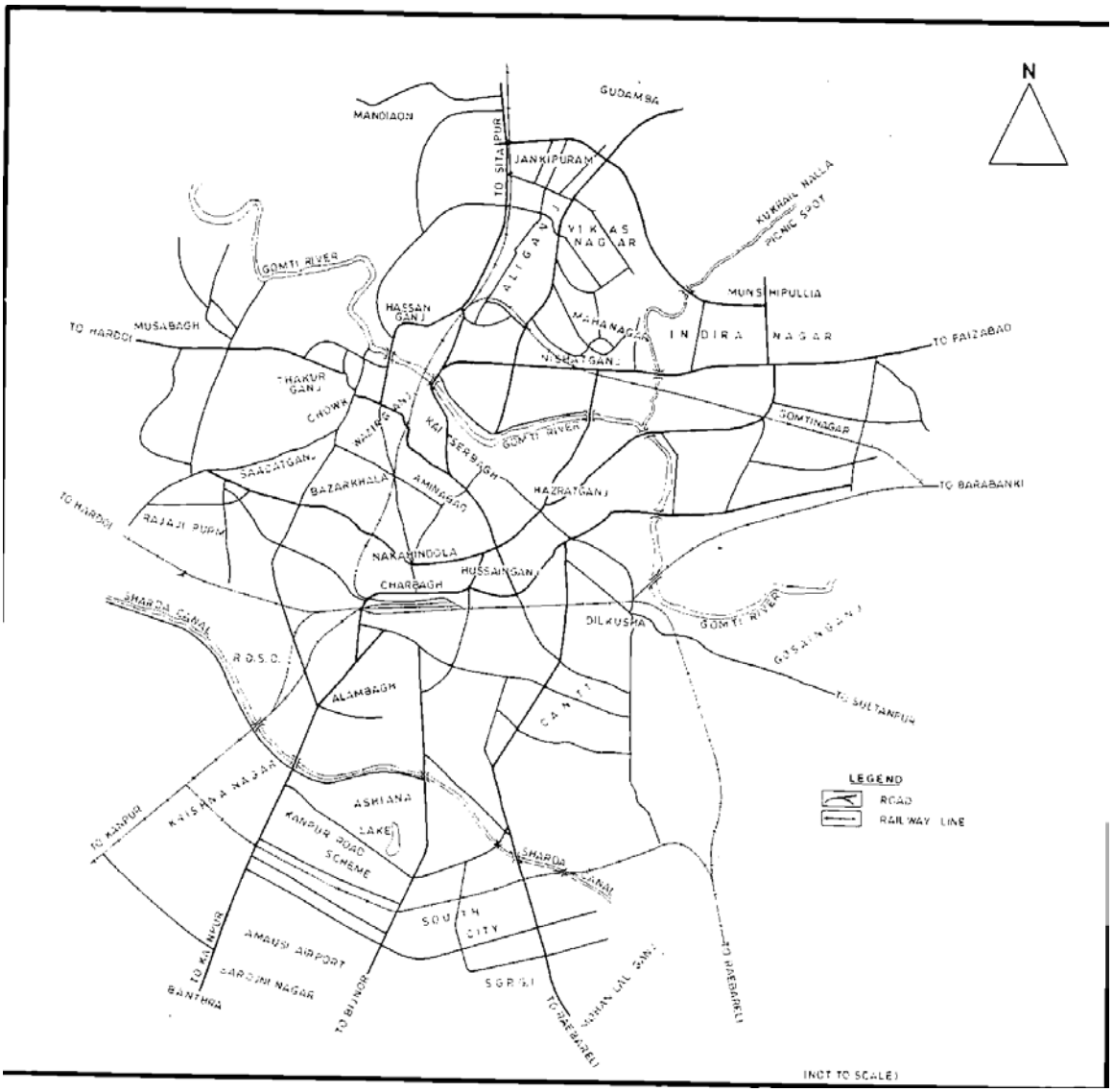


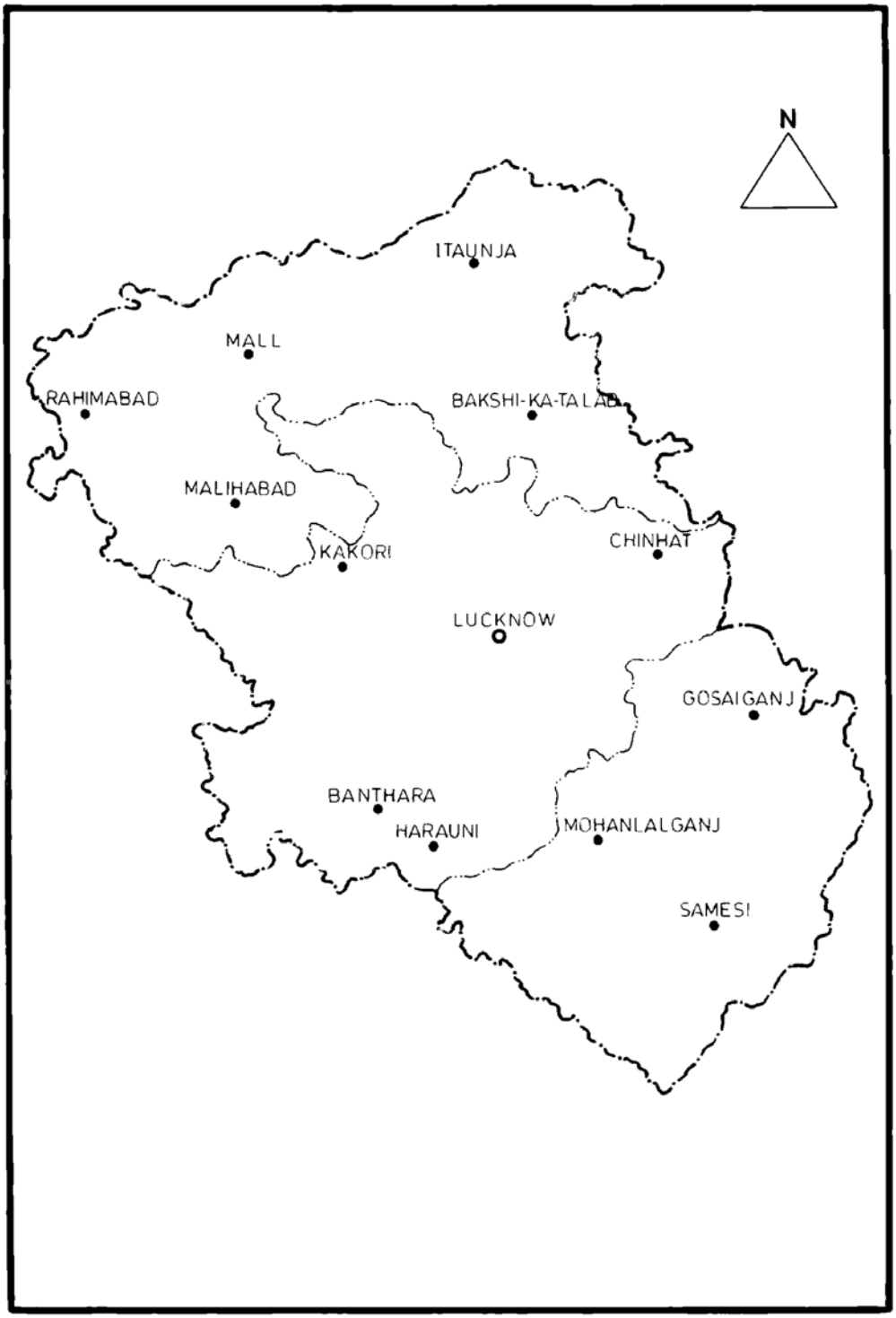
Figure-21: Profile of Indoor Air PAHs during Passive Exposure at Non-Cooking site in winter

ANNEXURE-III



Map Lucknow City

ANNEXURE-IV



Map Lucknow District