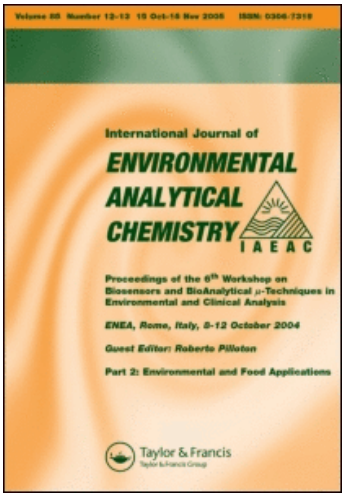


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# Czech-U.S. Epa Health Study: Assessment of Personal and Ambient Air Exposures to Pah and Organic Mutagens in the Teplice District of Northern Bohemia

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# CZECH-U.S. EPA HEALTH STUDY: ASSESSMENT OF PERSONAL AND AMBIENT AIR EXPOSURES TO PAH AND ORGANIC MUTAGENS IN THE TEPLICE DISTRICT OF NORTHERN BOHEMIA

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For over 40 years the Northern Bohemia area of the Czech Republic has been characterized by heavy industrialization, open pit mining, and utilization of high sulfur brown coal by industry and in residential home heating. These conditions have resulted in severe environmental pollution and concern over evidence of adverse health effects for residents in the region. Beginning in August 1991, Czech scientists from the Teplice District Institute of Hygiene and U.S. Environmental Protection Agency researchers from the Health Effects Research Laboratory and the Atmospheric Research and Exposure Assessment Laboratory cooperated in conducting pilot studies in the Teplice District, which is centrally located in the industrialized area. These studies determined ambient concentrations and personal exposures to airborne polycyclic aromatic hydrocarbons (PAHs) and organic mutagens. Active personal air samplers were used in personal exposure studies of coal miners, policemen and other workers in the Teplice district. Stationary medium-volume ($PM_{10}$) and high-volume ($PM_{2.5}$ and TSP) samplers were also used to collect ambient air samples. Personal sampler results from 24 h sampling in January 1992 of a group of Teplice policemen showed BaP exposures averaged 40 ng/m$^3$. Ambient high-volume (HiVol) air sampling results from 12 h nighttime samples collected in Teplice between February 17 and March 27, 1992 showed particle-associated BaP averaged 12 ng/m$^3$ and ranged from 2–34 ng/m$^3$. The sixteen PAHs that were quantified averaged 131 ng/m$^3$ for the same time periods. Approximately 50% of the particle-bound PAH concentrations in Teplice air resulted from

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compounds that are carcinogenic in animals. Mutagen concentrations and potency were determined by the Ames plate incorporation assay. The mutagenic potency of extractable organics from ambient air particles was higher than those for U.S. residential areas that are heavily impacted by wood smoke but similar to those from U.S. cities more heavily impacted by vehicle emissions.

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**KEYWORDS:** Czech Republic, air pollution, polycyclic aromatic hydrocarbons (PAH), mutagens.



## INTRODUCTION

The Northern Bohemia brown coal basin is comprised of four districts located in the northwestern portion of the Czech Republic. This brown coal or lignite contains 1–3% sulfur and in some localities as much as 5% sulfur.[1] The lignite is typically surface-mined from open pits which are sources of air pollutant emissions arising from pockets of continuous in-ground combustion. Coal mining, combined with heavy industrialization of these districts over the past few decades, has resulted in some of the worst environmental pollution in all of Europe.[1,2] Coal-fired power plants in these four areas produce 35% of the electricity utilized in the former Czechoslovakia.[1] This high-sulfur coal is also used by glassworks and the engineering, chemical and petrochemical industries. Coal is also used extensively for heating homes, large apartment complexes and business facilities. Over the past 25–30 years air pollution from these sources has caused extensive destruction of natural resources. Both hardwood and softwood forests have been heavily damaged. Conifers in the Krušné Hory (Ore Mountains) forming the northern border of this region have essentially been destroyed. Moldan and Schnoor[2] published an in-depth review of these environmental problems in the Czech Republic.

The health consequences of environmental pollution is, of course, a major concern of the Czech government and public. Early studies provided evidence of a high incidence of cancer, reproductive and behavioral effects.[1] New health studies have been initiated on human exposure and both short-term and long-term impact of air pollution on health of the population. One of these districts, Teplice, has been designated as a model district for study of the health effects of heavy pollution through improved environmental monitoring and health assessment studies. International cooperation has also been sought and, as a result, the U.S. Environmental Protection Agency (EPA) has joined the Czech Republic in developing air pollution monitoring, exposure assessments and health effects studies.

This is a report on the personal and ambient air sampling pilot studies that were conducted in the Teplice District during the summer of 1991 and winter of 1992. These experiments were jointly conducted by Czech and American collaborators to provide range estimates of personal exposures to PAH and organic mutagens. Wintertime sampling was particularly important because residential heating with coal and frequent temperature inversions in these industrial regions result in the highest pollution levels. Personal sampling was conducted for individuals that spend a significant portion of their work day in the outdoors, i.e., Teplice policemen. Teplice miners were also studied because of their occupational exposures to PAHs resulting from pockets of continuous in-ground combustion. These mine exposures

occur in open pits that are as much as 170–200 meters below the surface. The staff members of the Teplice District Institute of Hygiene were also included in personal sampling experiments to provide comparison data for workers that are predominantly inside buildings during the day. Some personal and ambient sampling pilot studies were also conducted in the Prachatice District located in Southern Bohemia. This latter district is a forested and mountainous region that is relatively free from industrial pollution and, therefore, was chosen to serve as a comparative district with presumed lower air pollutant concentrations. However, residential heating with coal is common to both districts and, indeed, common throughout the entire country.

The personal samples were analyzed for U.S. EPA "priority pollutant PAHs"[3] (see Table 1) that were extracted from the particles and separately extracted from the XAD resin. The amount of sample collected during 24 hour personal sampling was not sufficient to allow determination of particle mass concentrations.

Stationary $PM_{10}$ (0–10 micron diameter particles) medium-volume (MedVol), PM2.5 (0–2.5 micron diameter particles) high-volume (HiVol) and total suspended particles (TSP) HiVol ambient samplers were also used during the 1992 winter sampling to collect larger samples that allowed determination of percent extractable organic mass (EOM) and ambient concentrations of particles and PAHs. Extracts from HiVol ambient samples were also analyzed by the Ames *Salmonella typhimurium* histidine reversion bioassay[4] with strain TA98 to allow assessment of the mutagenic potency of extractable organics associated with air particles and to allow determination of the concentration of mutagens per cubic meter of air. This assay was chosen because chemicals and mixtures recognized as human carcinogens are generally mutagenic in short-term tests.[5] It has also been reported that chemicals that are rodent carcinogens across several species and organ sites (trans-species carcinogens) and act by a genotoxic mechanism are generally also mutagenic in *Salmonella typhimurium.*[6] Both combustion emissions and urban particle extracts have been found to be mutagenic in this assay and carcinogenic in rodents.[7,8]



## EXPERIMENTAL

Active personal sampling was conducted for 24 h periods with six Teplice miners and five staff members at the Teplice Institute of Hygiene during August 1991. The two groups were sampled two days apart. Similarly, four Teplice miners and three Institute of Hygiene staff were again sampled in January of 1992. The January sampling experiments also included five policemen in Teplice and five policemen in the Prachatice control district. The Teplice police and Institute of Hygiene workers were sampled on the same day. The miners were sampled one day earlier and the Prachatice policemen were sampled five days later as indicated in Figures 2 and 3. The Teplice policemen sampling was repeated in March of 1992 with six participants.

The personal samplers utilized a battery operated pump to pull approximately 1.7 l/min and an inlet/impactor to exclude particles larger than 2.5 microns. Fine particles were collected on Teflon impregnated glass fiber (TIGF) filters and semi-volatile organics on XAD-2 resin. Vapor phase nicotine was also collected on double filters which had been

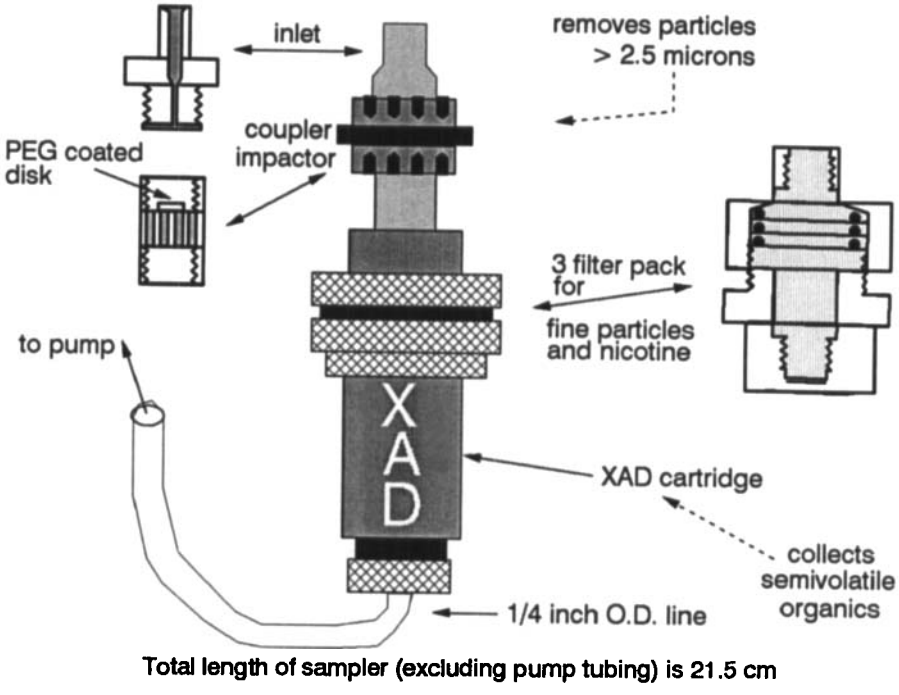


**Figure 1** Personal air sampler.

chemically treated with sodium bisulfate.[9,10] These two nicotine filters were positioned between the particle filter and the XAD resin (see Figure 1).[11]

Ambient air sampling was conducted using $PM_{10}$ MedVol samplers, $PM_{2.5}$ HiVol and TSP HiVol samplers. Two MedVol samplers, operated for 24 h periods at approximately 16 l/min, were run simultaneously to allow comparisons of pollutant concentrations at bottom/top of the Teplice mining pit, inside/outside of the Teplice Institute of Hygiene (located in the business district), and inside/outside a home in Prachatice. The $PM_{2.5}$ HiVol was operated on the roof of an apartment building centrally located in the town of Teplice and collected 12 hour nighttime samples from mid February to the end of March 1992. Samples from these same periods were also analyzed by other EPA laboratories to determine concentrations of metals/elements and various chemical species, i.e., $SO_x$ and $NO_x$. These latter results will be reported elsewhere. The TSP HiVol sampler, which was located on a first floor balcony of the Teplice Institute of Hygiene and less than 1 km from the $PM_{2.5}$ HiVol site, collected 24 hour ambient samples. The TSP filter samples from January 2 to January 13, 1992 were analyzed along with the above filter samplers because they covered the time periods when personal sampling was also being conducted in the Teplice district and because the $PM_{2.5}$ HiVol had not been placed in operation at the time these samples were collected.

## MATERIALS AND METHODS

### *Personal sampling*

The personal sampler was attached to the individual's shirt or blouse by means of a clip and was positioned in a vertical manner so that the inlet was at the top and near the individual's breathing zone. The battery operated pump was contained in a small pouch/belt assembly and worn around the waist. Tygon$^R$ tubing connected the pump to the exit end of the sampler assembly. The sampler (available from University Research Glassware Corp, 118 E. Main Street Carrboro, NC 27510 U.S.A.) components (Figure 1) and sampler validation experiments have been previously reported.[11] Briefly, the sampler consists of a Teflon$^R$ coated aluminum inlet, particle accelerator and elutriator (a borosilicate impaction disk coated with polyethyleneglycol) (PEG) to retain particles having an aerodynamic $dp_{50}$ of > 2.5 microns), a three stage filter pack (holds 1 to 3 filters, separated by O-rings and Teflon®-coated stainless steel screens), a 1 cm × 5 cm resin cartridge for collection of semi-volatile organics, and an adapter for connection of the plastic (Tygon$^R$ tubing to the sampler pump. The sampler was operated at 1.7 l/min to achieve the 2.5 micron particle size cut-point. The filter-pack assembly for these experiments contained three filters which are described as follows: first, a 2.5 cm TIGF filter (T60A20, from Pallflex, Putnam, Connecticut, USA); second and third filters were 2.5 cm TIGF coated with sodium bisulfate for collection of vapor phase nicotine.[9,10] The resin cartridge contained 2–2.5 g (dry mass) of XAD-2 (available from Rohm and Haas, Pittsburgh, PA USA). The pumps (Sibata, MP-CF15, Japan-available in the U.S. from Gilian Instrument Corporation, 35 Fairfield Place, West Caldwell, New Jersey 07006-6206) operated continuously for 24 h periods and were powered by eight AA batteries. Flow rates were adjusted to 1.7 l/min just prior to sampling and measured again immediately after sampling. Acceptable final flows were within 10% of initial flow and sampling flow rate was calculated as the mean of start and stop values. Samples were stored in a – 80°C freezer until analyses could be performed.



### *Ambient sampling*

The Andersen $PM_{10}$ MedVol sampler was equipped with a 7.5 cm diameter TIGF Pallflex filter (T60A20) and operated at approximately 16 l/min for 24 hr sampling periods. The pump was a Gilian-Aircon 520 ACT. Flow rates were calibrated before and after each field trial. The Ströhlein TSP sampler utilized a 25.4 cm diameter circular TIGF Pallflex filter (T60A20) and was operated for 24 hr sampling periods. Flow rate was 0.83 $m^3$/min. The Sierra-Andersen $PM_{2.5}$ HiVol was equipped with an 8 × 10 inch (20.3 cm × 25.4cm) TIGF Pallflex filter (T60A20) and operated for 12 hour nighttime periods at approximately 1.1 $m^3$/min.

*Extraction and chemical analysis*

The 2.5 cm TIGF particle filters from personal samplers were individually extracted by sonication (10 minutes) with 20 ml of dichloromethane (DCM). The extraction was repeated two additional times. Extracts were combined and concentrated by evaporation under a stream of nitrogen to a 10 ml volume. Aliquots were removed for determination of EOM, PAHs and mutagenicity. The personal sampler primary and secondary nicotine filters were combined, extracted and analyzed by the method of Hammond[9,10] with modifications described by Williams.[12] Semi-volatile organics were extracted from the personal sampler XAD-2 resin without removing the resin from the chamber. Teflon tubing was attached to the exit end of the chamber and 25 ml of DCM was eluted through the vertically positioned chamber in a flow pattern reversed from that used to collect the sample. The eluate was concentrated under nitrogen flow for subsequent PAH analysis.

MedVol and HiVol filters were individually triply extracted in 250 ml Teflon bottles by 10 minute sonications with 100 ml DCM used for each extract. Extracts were combined, concentrated by rotary vacuum evaporation at 35°C, volume adjusted and aliquoted for determinations as described above.



*PAH analysis*

PAH analyses were performed on extracts from particle filters and from XAD-2 resin samples using a modification of the U.S. EPA Method 610.[13] Modification of the HPLC method consisted of using a PAH-specific column (25 cm × 4.6 mm i.d., 5-micron Supelcosil LC-PAH, #5-8229, Supelco, Inc. Bellefonte, PA 16823-0048 USA) and a time-programmable fluorescence detector. This column enabled nearly complete resolution between all 16 priority PAHs (Table 1) while the programmable detector gave increased specificity and sensitivity for each species.

*Bioassay analysis*

The Ames *Salmonella typhimurium* plate incorporation assay[4] with strain TA98 was used to determine mutagenic potency of the EOM (rev/μg EOM) and mutagenicity concentrations

**Table 1** PAH compounds included in the analyses.

| | |
|---|---|
| naphthalene | *benzo(a)anthracene |
| acenaphthene | *chrysene |
| acenaphthylene | *benzo(b)fluoranthene |
| fluorene | *benzo(k)fluoranthene |
| phenanthrene | *benzo(a)pyrene |
| anthracene | *dibenzo(a,h)anthracene |
| fluoranthene | *indeno(1,2,3-cd)pyrene |
| pyrene | |
| benzo(ghi)perylene | |

*IARC determined to be an animal carcinogen.

(rev/$m^3$). Bioassay experiments were conducted on individual sample extracts using a minimum of five doses of EOM and duplicate plates with and without Aroclor induced rat liver S9 metabolic activation ( + S9 and – S9) for each dose. Only HiVol samples were bioassayed.

## RESULTS AND DISCUSSION

Table 1 is a list of 16 PAH compounds that were quantified in the filter and XAD extracts. This table also indicates compounds that have been determined to be animal carcinogens by the International Agency for Research on Cancer (IARC).[14] PAH results are reported in three ways; "total PAHs" is a summation of all 16 compounds, "carcinogenic PAHs" is a summation of the 7 carcinogens listed in Table 1, and individual BaP results are reported to allow comparisons with other studies found in the literature.

Table 2 gives the range of total PAH results from personal sampling pilot studies that were conducted for 24 h periods with Teplice miners and Teplice Institute of Hygiene workers during August 1991 and for the same groups plus Teplice policemen during January 1992. Personal sampling was also conducted for policemen in the Prachatice District during the January 1992 sampling trials. Teplice police sampling was repeated in March of 1992. This table indicates significantly higher individual exposures (approximately a factor of 5) to PAHs in the winter months. This was expected, of course, because of winter meteorological inversion conditions and greater pollutant concentrations resulting from residential heating with the "brown" coal and wintertime vehicle emissions.

Figure 2 shows the particle sampling total PAH results for the January 1992 personal sampling filters, MedVol filters and the TSP HiVol filters. Each data point is from analysis of one sample. Results from the March 1992 repeat personal sampling of Teplice policemen are also shown. All Table 1 compounds are included in the totals. The MedVol results from January 8 indicate the coal mine is a significant source of PAHs. These 24 hour stationary samplers at the mine showed total PAH concentrations of 1070 ng/$m^3$ immediately adjacent to the pit and 1300 ng/$m^3$ at the bottom of the mining pit. It should be noted, however, that the mine area is located near a coal-fired power plant and other industrial sources as previously mentioned. The miners personal sampler results are lower and ranged from 150 to 580 ng/$m^3$. This is due to the fact that the samplers were either worn or kept with the miner for a 24 h period and mine exposures were during an approximate 8 h work shift. The January 9 results for Teplice police also indicates similar levels of personal exposure with total PAHs

**Table 2** Personal sampler comparisons of PAH exposures

| | *Particle Filter Total PAHs*[1] *(ng/$m^3$)* | | |
|---|---|---|---|
| *Group Sampled* | *August* | *January* | *March* |
| Teplice Miners | 30–130(70)[2] | 150–580(390) | |
| Teplice Inst. Hyg. Staff | 20–70(30) | 100–180(150) | |
| Teplice Police | | 200–460(320) | 60–180(110) |
| Prachatice Police | | 50–120(80) | |

[1]total of the 16 PAH listed in Table 1
[2]() mean value

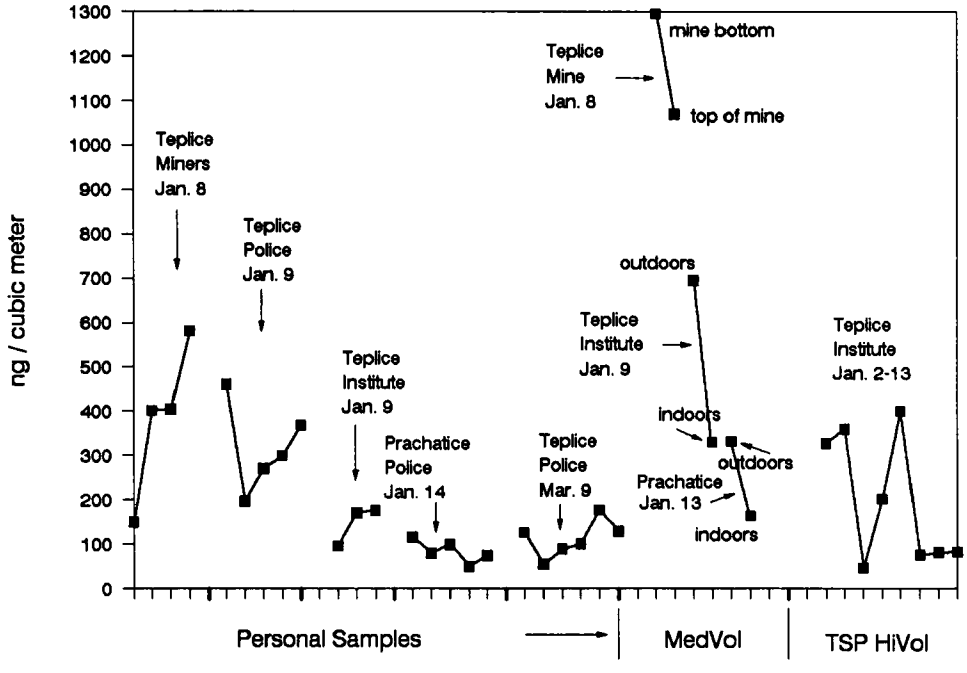


**Figure 2** Teplice and Prachatice total PAH results from 24 h personal and ambient particle sampling.

ranging from 200 to 460 ng/m$^3$. The lower exposure levels for the Teplice Institute of Hygiene workers (98–177 ng/m$^3$) was likely due to their greater time indoors. Figure 2 $PM_{10}$ MedVol results show a factor of two greater concentration of PAH pollutants outdoors compared to indoors for the Teplice Institute of Hygiene building. A similar factor of 2 difference was observed for a residence in Prachatice. Future sampling should focus on determination of indoor/outdoor pollutant ratios for additional residences, workplaces and schools in order to better characterize personal exposure levels. The TSP HiVol filters for January 2–13 showed total PAH levels in ambient air that were consistent with the personal and MedVol sampler results.

Figure 3 shows only BaP results from the above data set and demonstrates patterns similar to those for total PAHs. Again, there is a similarity between miner and police personal exposures. The BaP exposures for these two groups ranged from 13 to 60 ng/m$^3$. The indoor/outdoor paired samplers also showed the same factor of two differences as previously noted.

Figures 4 and 5 show paired personal sampler PAH results for particle filters and XAD cartridges from the same sampler. Each data pair represents results from one individual. Figure 4 compares total PAH results from the particle filter and XAD combinations. This graph indicates that the total PAH mass associated with particles is quite small compared to the amount of semi-volatile PAHs collected in the XAD cartridge. The XAD values were dominated by naphthalene. Acenaphthene and phenanthrene, respectively, represented the second and third largest contributors to total PAHs in the XAD samples. The higher

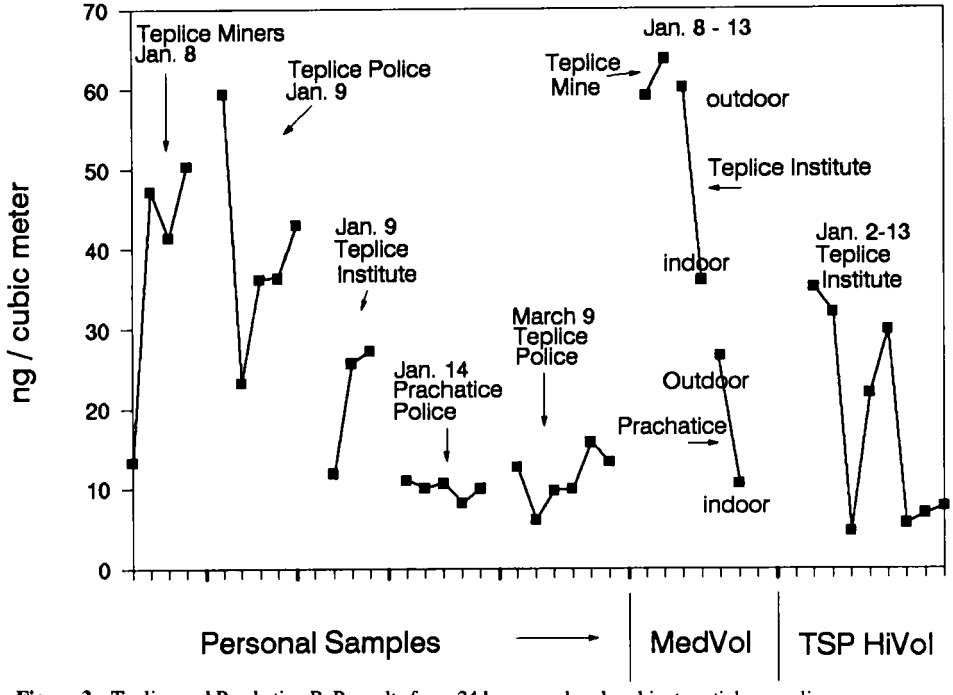


**Figure 3** Teplice and Prachatice BaP results from 24 h personal and ambient particle sampling.

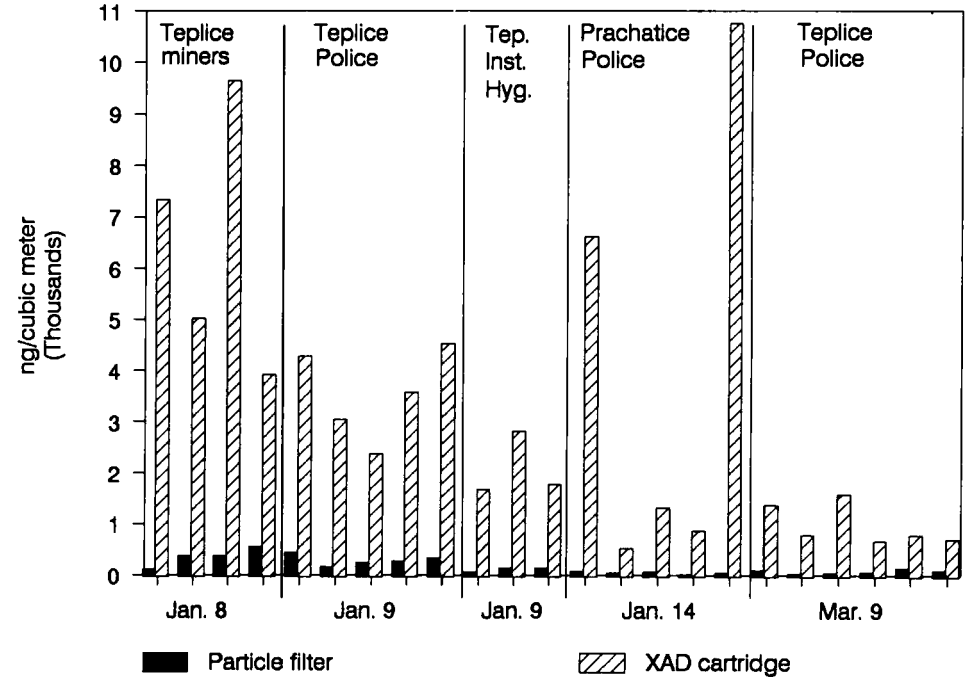


**Figure 4** Personal sampler total PAHs: particle filter compared to XAD.

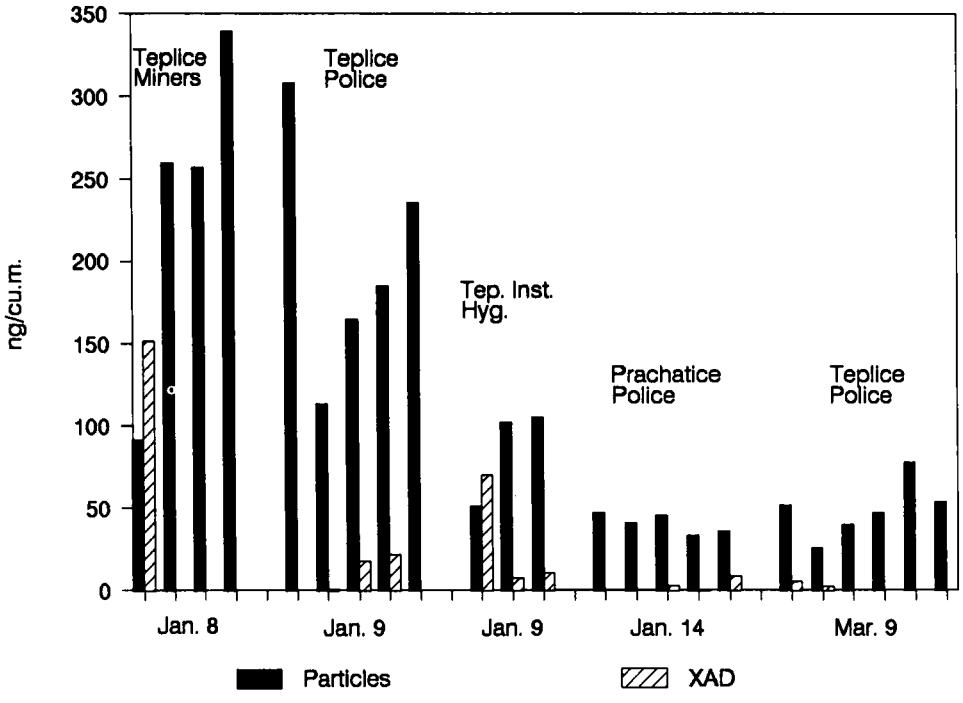


**Figure 5** Personal sampler carcinogenic PAHs: particle filter compared to XAD.

molecular weight PAHs associated with the particles, however, consist of several mutagens and carcinogens. Figure 5 is a re-analysis of the same data but shows only total concentrations of the carcinogenic PAHs that are listed in Table 1. This graph demonstrates that carcinogenic PAHs are primarily associated with particles. Most personal samples showed no (Table 1)carcinogens in the XAD extracts. Only 2 out of 23 XAD samples were found to contain significant amounts of carcinogens when compared to the particle sample. Benz(a)anthracene was the primary carcinogenic PAH found in these two XAD samples.

Figure 6 compares four personal sampler results for each individual. These results allow comparisons of personal sampler nicotine concentrations with total particle related PAH, total particle related carcinogenic PAHs, and total particle PAHs plus semi-volatile PAH found in the XAD cartridge. It is evident from this graph that these personal exposures to PAH are not significantly affected by passive cigarette smoke. A regression analysis of nicotine and total PAHs (particle filter plus XAD cartridge) showed no correlation. Cigarette smoke contains PAHs, however, the amount of contribution to the existing ambient levels was not significant. It should be noted, however, that active or passive cigarette smoke may have a profound effect on other biomarkers of exposure, i.e., urine mutagenicity and DNA adducts.

Figure 7 results from TSP HiVol particle filters collected during January 2–13 at the Teplice Institute of Hygiene and from $SO_2$ monitors at the same location. This graph, which

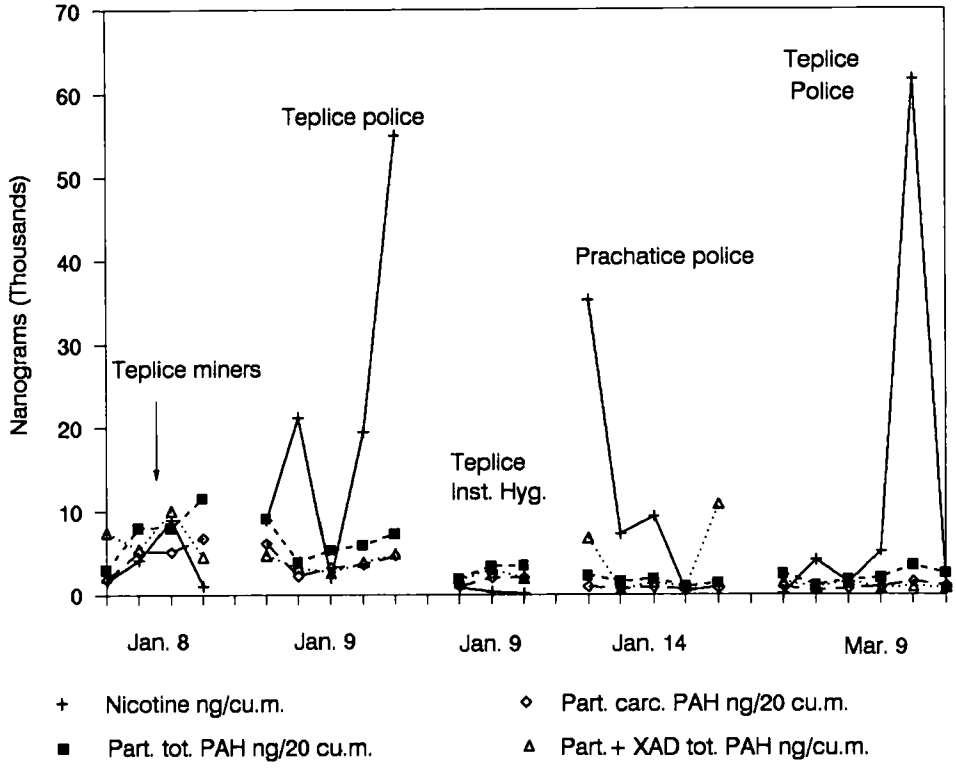


**Figure 6** Personal sampler comparison of nicotine and PAH results.



compares concentrations of particles, $SO_2$, EOM, mutagenicity and total PAHs, indicates a high degree of correlation for all of these measurements. The $SO_2$ concentrations correlated strongly with the other four parameters with all r values ≥0.85. These correlation coefficients (r values) are shown in the graph. Linear regression analysis for mutagenicity concentrations and carcinogenic PAH concentrations (data not shown) gave a correlation coefficient of 0.98 and a slope of 0.97. Table 3 shows correlation coefficients for a larger data set derived from HiVol filters collected in February and March of the same year (see the following discussions).

Figures 8 and 9 show results from analysis of $PM_{2.5}$ HiVol filters that were collected during 12 h night sampling periods between February 17 and March 27, 1992. The sampler was located on the roof of an apartment building that was centrally located in the town of Teplice. Figure 8 shows the fine particle concentrations during this period and allows comparisons with concentrations of total PAHs, carcinogenic PAHs and BaP. Four of these filters (15% of the total number) had a portion removed and were separately extracted and analyzed for a quality assurance verification of the PAH analyses. These duplicate results were in good agreement with a mean PAH difference of 12% + / – S.D. of 8. Figure 9 is a plot of particle, EOM and indirect-acting ( + S9) mutagen concentrations for the same sample

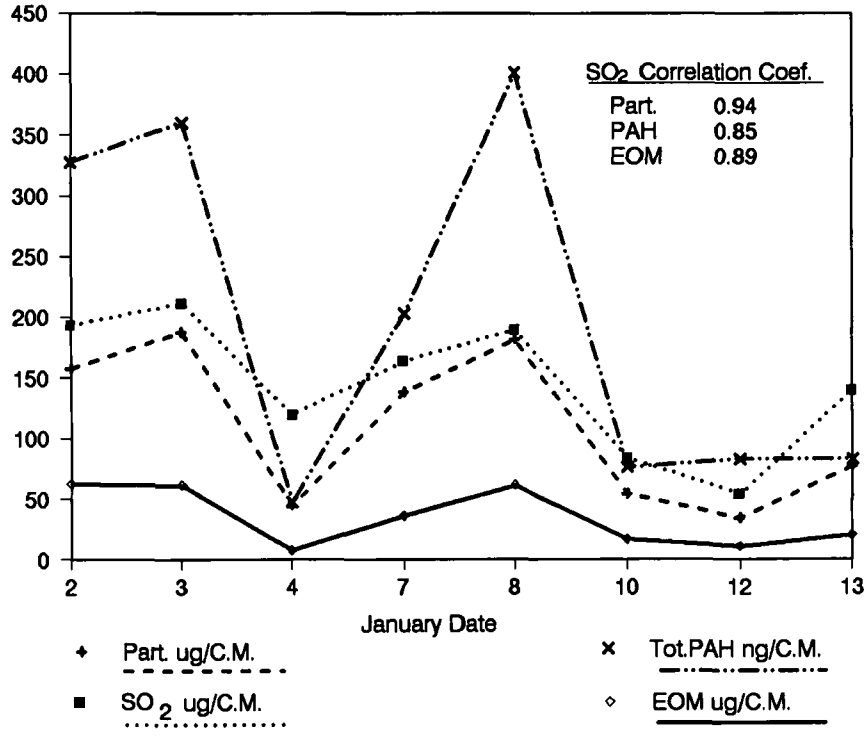


**Figure 7** $SO_2$ compared to particle, PAH, EOM and mutagenicity concentrations from Teplice TSP HiVol particle filters collected in January 1992.

set. Mutagenicity is expressed as revertant colonies/$m^3$ (rev/$m^3$ of sampled air. The mean + S9 potency value was 3.9 rev/μg EOM. The mean direct-acting ( – S9) potency was 1.38 rev/μg EOM or about one-third of the indirect-acting potency. Table 3 shows correlation coefficients (r values) from linear regression analyses that were performed on the $PM_{2.5}$ HiVol data collected in Teplice during February and March 1992. All measurement parameters showed moderate to strong correlations. The concentration of organic mutagens in ambient air was strongly correlated with concentrations of EOM, total particle PAHs and carcinogenic PAHs with r values ≥ 0.90. Particle concentrations were moderately correlated with mutagenicity (r = 0.64).

The PAH results from personal sampling were examined along with similar results from $PM_{2.5}$ HiVol samples for the purpose of determining the relative amounts of carcinogenic PAHs found associated with particles. The personal particle filter and HiVol filters gave essentially the same result. Approximately one-half of the particle PAH concentrations resulted from carcinogens identified in Table 1. The personal sampler ratio of carcinogenic PAHs/total PAHs was 0.55 + / – 0.08 (mean + / – S.D.) and the comparable ratio from the HiVol particle samples was 0.57 + / – 0.05.

Table 4 shows Teplice PAH and bioassay data compared to study results from other high

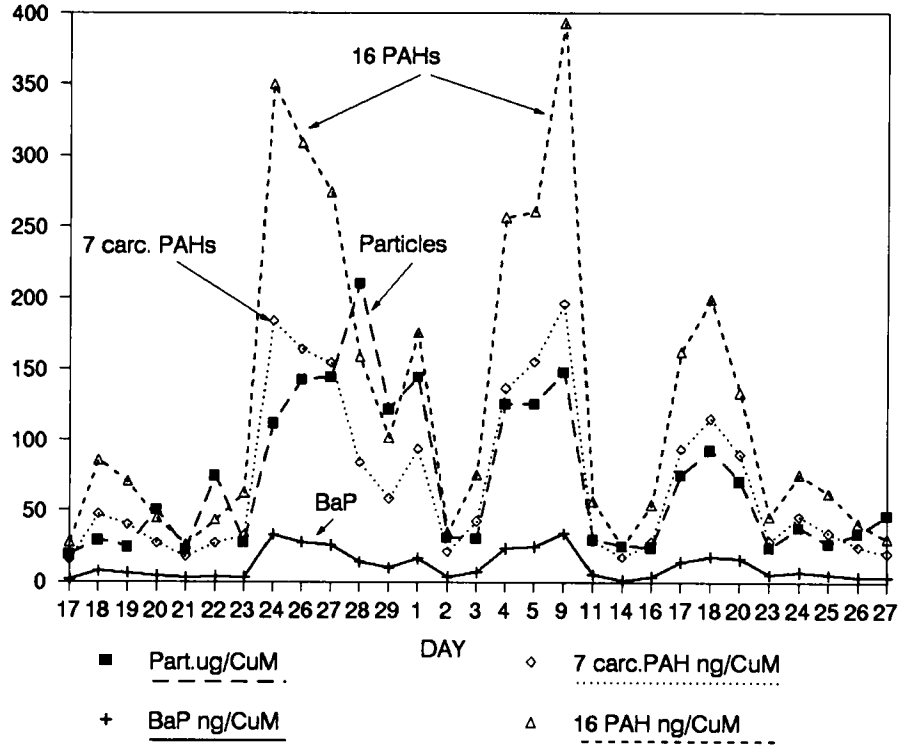


**Figure 8** Particle and PAH results from $PM_{2.5}$ HiVol particle samples collected from the roof of a Teplice apartment during February and March of 1992.

pollution areas. The first four cities listed were sampled by the U.S. EPA as part of residential wood smoke studies.[15-18] A comparison of data shows that % EOM values from Teplice are less than those from the wood smoke impacted areas. However, the Teplice data shows higher mutagenic potency (rev/μg EOM). The average potency of 3.9 rev/μg of EOM is about a factor of 2 greater than that measured for Albuquerque, New Mexico and Boise, Idaho during EPA's Integrated Air Cancer Project (IACP) studies and is approximately a factor of 4 greater than similar values from Juneau, Alaska and Raleigh, North Carolina. These U.S. urban air sheds were primarily impacted by a mixture of wood smoke and vehicle emissions. The average "particle" mutagenic potency (revertants/μg of particles) of 1.65 for the Teplice winter is higher than those found in Albuquerque, NM and Boise, ID during the IACP studies (0.82 and 0.96 respectfully). This particle potency is also higher than the January value of 1.11 for samples collected by the TSP sampler operated on the balcony of the Teplice Institute of Hygiene. This is expected because the February-March samples were collected by a $PM_{2.5}$ HiVol and past studies of Watts, *et al.*[19] have shown that airborne condensable organics are primarily associated with fine particles (less than 2.5 microns). EOM and mutagenicity data from non-winter studies is also included to allow comparisons of Teplice with two large urban areas in the U.S. that are primarily impacted by vehicle

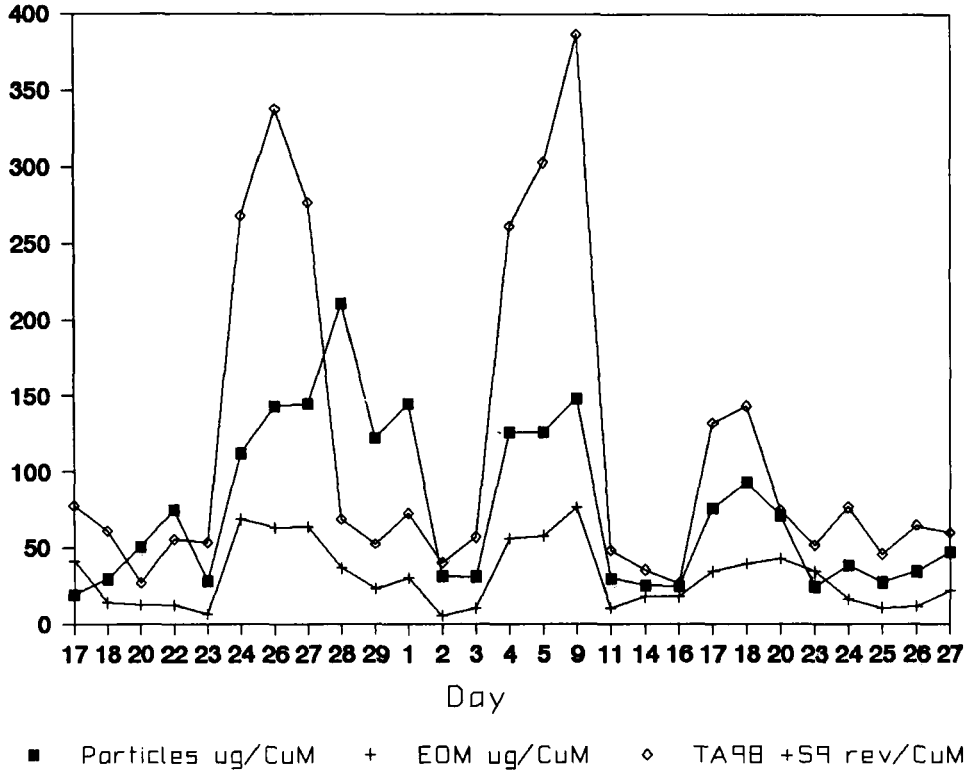


**Figure 9** Particle, EOM and mutagenicity concentrations from $PM_{2.5}$ HiVol particle samples collected from the roof of a Teplice apartment during February and March of 1992.



emissions. The mutagenic potency values of 4.1 and 5.8 rev/μg EOM, respectively from Washington, D.C.[20] and Philadelphia, PA,[21] are similar to those from Teplice. However, the approximately 5 percent organics associated with particles in these U.S. areas is quite small when compared to the 40 percent for Teplice air particles. This accounts for particle potency (rev/μg of particles) in Teplice being approximately six times greater than for the U.S. urban areas.

Motykiewicz, *et al.*[22] reported a 1988 study of upper Silesia where January-March BaP values ranged from 29–61 ng/m$^3$. These average levels in Table 4 appear to be approximately twice those observed in Teplice. Watts, *et al.* [18] sampled a residential area of Juneau, Alaska that was heavily impacted by residential wood smoke. PAH concentrations for the same 16 PAHs included in this Teplice study ranged from 20–500 ng/m$^3$ for daytime sampling periods. Juneau night concentrations were higher with a total PAH average of approximately 150 ng/m$^3$. This total PAH nighttime concentration is similar to the 131 and 198 ng/m$^3$ averages for the Teplice HiVols. Cretney, *et al.*[23] reported winter PAH concentrations for residential areas of Christchurch, New Zealand. This urban population of approximately 300,000 experiences severe winter pollution with major contributions from residential

**Table 3** Correlation coefficients for Teplice winter $PM_{2.5}$ HiVol data.

| | *EOM* | *Total PAHs* | *Carc.PAHs* | *Mutagenicity* |
|---|---|---|---|---|
| Part. Conc. | 0.70 | 0.78 | 0.78 | 0.64 |
| EOM | | 0.90 | 0.91 | 0.90 |
| Total PAHs | | | 0.99 | 0.94 |
| Carc. PAHs | | | | 0.93 |

**Table 4** Teplice pollution compared with other study areas.

| | *HiVol averages from winter sampling periods* | | | | |
|---|---|---|---|---|---|
| *City* | *Particle (μg/m³)* | *EOM %* | *TA98 + S9 rev/μg EOM* | *TA98 + S9 rev/μg Part.* | *BaP/PAHs (ng/m³)* |
| Juneau, AK[1] | 57 | 59 | 0.73 | 0.42 | —/150 |
| Raleigh, NC[1] | 36 | 50 | 1.07 | 0.53 | |
| Albuquerque, NM[1] | 45 | 47 | 1.89 | 0.82 | |
| Boise, ID[1] | 40 | 61 | 1.60 | 0.96 | 7/—[2] |
| Teplice, Czech[3] | 110 | 30 | 3.87 | 1.11 | 18/198 |
| Teplice, Czech[4] | 72 | 40 | 3.86 | 1.65 | 12/131 |
| Washington, D.C.[5] | | 6 | 4.10 | 0.25 | |
| Philadelphia, PA[5] | | 3.9 | 5.82 | 0.23 | |
| Christchurch, NZ[6] | 215 | | | | 45/— |
| Silesia, Pol[7] | | | | | 46/— |

1) U.S. EPA wood smoke studies, $PM_{2.5}$ HiVol[15–18]
2) U.S. EPA, Boise wood smoke study, NIST data[17]
3) January 1992 TSP HiVol, Institute of Hygiene
4) February-March 1992 $PM_{2.5}$ HiVol
5) Non-winter studies of U.S. urban air sheds[20,21]
6) Winter TSP 24 h samples[23]
7) Ruda Slaska site: mean of January, February and March 1988[22]



fireplaces burning coal, coke, and wood. A suburban sampling site with particle concentrations of 107–373 μg/m³ gave BaP values of 19–71 ng/m³. Both particle and BaP concentrations appear to be approximately twice the levels found in Teplice.

## CONCLUSIONS

A series of personal and ambient sampling pilot studies were conducted in the Northern Bohemia area of the Czech Republic during the period of August 1991 through March 1992 for the purpose of providing some range estimates of personal exposures to PAH and organic mutagens. Conclusions resulting from these pilot studies are as follows:

1. Teplice winter season respirable ambient air particles contained approximately 40% by weight of extractable organic chemicals. These extractable organics were, as would be expected in any urban air shed, mutagenic in the Ames Salmonella typhimurium histidine reversion bioassay. However, the mutagenic potency of Teplice air particle organics appears to be approximately a factor of two greater than potency from areas heavily

impacted by residential wood smoke and is similar to the potency of organics from U.S. urban areas (Washington, D.C. and Philadelphia, PA) that are impacted primarily by vehicle emissions. The mutagenicity per particle potency for Teplice is significantly higher than for these vehicle impacted urban areas of the U.S..

2. Teplice wintertime mean concentration for 16 PAH compounds associated with $PM_{2.5}$ particles was 131 ng/m$^3$. Carcinogenic PAHs accounted for approximately 50% of the total PAHs associated with fine particles. Personal sampling results from these short-term studies indicated wintertime exposures were up to 5 times greater than summer exposures.
3. Wintertime PAH concentrations in the Teplice area are similar to those from other highly polluted areas that are impacted by vehicle exhaust plus residential heating with wood and/or coal.
4. There was insufficient personal and ambient data from the control district of Prachatice to characterize exposure differences, however, the limited data presented indicates that PAH exposures may be similar.
5. Teplice PAH exposures resulting from inhalable particles are greater during outdoor activities. Two experiments indicated approximately a factor of 2 difference in outdoor compared to indoor concentrations for the 16 PAH compounds included in the analyses. Additional sampling experiments are needed to better characterize these differences.
6. The pollution parameters determined for Teplice air; particles/m$^3$, total PAHs/m$^3$, EOM/m$^3$ and mutagenicity/m$^3$ all showed moderate to strong correlations with each other. The concentration of organic mutagens in ambient air was strongly correlated with concentrations of extractable organics and PAHs ($r \geq 0.90$). The January $SO_2$ concentrations also correlated with particle, PAH, EOM and mutagenicity concentrations ($r \geq 0.85$).
7. Personal sampler nicotine concentrations did not correlate with PAH levels, thereby indicating that cigarette smoking did not confound the PAH personal exposures determined from these pilot studies.



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