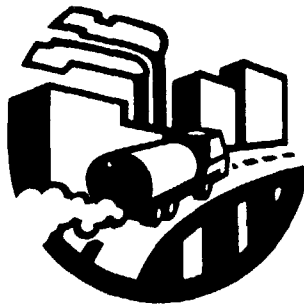


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Chemical and Biological Characterization of Urban Particulate Matter



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Göran Löfroth

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Summary

Airborne particulate matter has been collected on glass fiber filter by high volume sampling in the Gothenburg urban area. The samples were, after extraction with respect to organic components, tested for biological effects in the Salmonella mutagenicity assay, affinity to the cytosol TCDD receptor and toxicity towards a mammalian cell system and analyzed chemically for selected polycyclic aromatic compounds. Additional analyses were performed on selected elements, ionic species and gas phase simple aromatic compounds.

A series of samples collected simultaneously at a street level location and a rooftop site showed that most parameters associated with the organic compounds adsorbed to airborne particulate matter had similar concentrations at the two levels. The differences observed for the mutagenic effects in different strains and conditions showed that the rooftop samples had a different composition compared to the street samples indicating that atmospheric transformations have occurred.

The main project comprised parallel sampling at two different and variable street locations having different traffic patterns. There were only small differences between the parallelly measured parameters indicating that air pollutants are spread efficiently over a large area.

Chemical fractionation of representative samples showed that the distribution of mutagenic activity among different fractions is dissimilar to the distribution obtained in the fractionation of both gasoline and diesel engine exhaust particles. The results indicate either that polar mutagens have been formed or that non-polar mutagens have been destroyed in atmospheric reactions.

Partial least squares regression analysis showed qualitatively that diesel exhaust is a major source of airborne particulate mutagenic activity and source apportionment with chemical mass balance and multilinear regression corroborated this quantitatively. The multilinear regression analysis gave the result that the airborne activity in Salmonella TA98-S9 originated to 54 ± 4 % from diesel exhaust and to 26 ± 3 % from gasoline exhaust. The contribution is more equal for the activity measured with TA98+S9. The relative magnitude of the contributions is in agreement with experimental emission data and the abundance of gasoline and diesel powered vehicles.

The usefulness of short-term bioassays as an addition to chemical analysis of airborne particulate matter depends on whether only polycyclic aromatic hydrocarbons (PAH) are major carcinogens, as has been suggested in the literature, or whether also other polycyclic aromatic compounds (PAC) are of importance.

Sammanfattning

Svävande luftburet stoft har insamlats på glasfiberfilter med högvolym-provtagning på olika platser i Göteborgsområdet. Efter extraktion av organiska ämnen har proverna undersökts med avseende på mutagen effekt i Ames Salmonella-test, bindning till TCDD-receptor och toxicitet i ett mammaliecellsystem samt förekomst av polycykliska kolväten. Vid provtagningarna analyserades även enkla aromatiska kolväten samt partiklarnas grundämnesammansättning och halt av vissa joner.

En serie prover erhållna genom samtidig provtagning på två närliggande platser, en i gatunivå och en i taknivå, visade att de flesta av de parametrar som är förbundna med luftburna partiklar hade liknande värden och koncentrationer på de två provtagningsplatserna. En skillnad i den mutagena aktiviteten i olika bakteriestammar och testbetingelser visade att sammansättningen av mutagena komponenter var olika på de två provtagningsplatserna vilket antyder att atmosfärskemiska reaktioner skett.

Huvudstudien omfattade provtagning i gatunivå på flera provtagningsplatser med olika trafikbelastning och utfördes med samtidig provtagning på två olika platser. Resultaten visade att det endast förekom mindre skillnader i de uppmätta parametrarna mellan de samtida provtagningarna vilket antyder att luftföroreningarna sprider sig över ett större område.

Kemisk fraktionering av utvalda prover visade att fördelningen av den mutagena aktiviteten i de olika fraktionerna är skild från den fördelning som erhålles vid samma fraktionering av partikelbundna ämnen i både bensin- och dieselavgaser. Resultaten indikerar att antingen har polära mutagena ämnen bildats eller har icke-polära ämnen förstörts i atmosfärskemiska reaktioner.

Analys med partiell minstakvadratregression visade kvalitativt att dieselavgaser är en väsentlig källa för partikelbunden mutagen aktivitet. Källbidragsberäkningar med kemisk massbalans och multilinjär regression bestyrkte detta kvantitativt. Analysen med multilinjär regression gav resultatet att 54±4 % av den mutagena aktiviteten i Salmonella TA98-S9 kom från dieselavgaser och 26±3 % från bensinavgaser. Mätt med TA98+S9 är bidraget från dessa två källor mer lika. Dessa relativa bidrag står i överensstämmelse med experimentella emissionsdata och med förekomsten av bensin- och dieselfordon.

Användbarheten av biologiska korttidstester som ett tillägg till kemisk analys av luftburna partiklar beror i hög grad på om endast polycykliska aromatiska kolväten (PAH) är de huvusakliga carcinogenerna, vilket framförts i litteraturen, eller om även andra polycykliska ämnen (PAC) är av betydelse.

Introduction

Much of the history of urban air pollution is associated with the urban community. The London smog episode in 1952 is well-known and may be regarded as a starting point for modern air pollution abatement work. Much of the early work was focused on acute effects (Goldsmith 1962) rather than long-term effects. This may partly be explained by the lack of risk assessment methods for these types of phenomena.

The first bioassays of urban air pollutants were commenced in 1936 by Siegel and Shear (see Leiter et al. 1942) who extracted fall-out soot and tested the extracts with negative results for the production of tumors after subcutaneous injection in mice. Subsequent studies (Leiter et al. 1942, Leiter and Shear 1942) with extracts of filter-collected atmospheric dusts showed that the extracts were tumorigenic at the site of injection. Kotin et al. (1954a) continued the studies and collected, extracted and performed carcinogenicity tests of particulate organic matter. The same authors also tested motor vehicle exhaust in the same manner (Kotin et al. 1954b; 1955). The origin of these early studies were partly reflected in the hypothesis that urban air pollution was responsible for the increase of lung cancer observed in both America and Europe. Although this hypothesis was untenable, as attention was being directed to the emerging knowledge that tobacco smoke is the most important causative agent for lung cancer (Wynder and Graham 1950), studies on the carcinogenicity of urban pollutants continued (Epstein et al. 1966; Asahina et al. 1972).

Chemical analysis of the particulate organic matter was first mainly limited to a single or some polycyclic aromatic hydrocarbons (PAH) (Waller 1952, Sawicki et al. 1960) and later extended to many different PAH (Pierce and Katz 1975, Gordon 1976). The first Swedish studies on PAH, which were also limited to benzo(a)pyrene (BaP), were conducted between 1961 and 1963 by Frank and Gerhardsson (1962) and Gerhardsson (1966). An average daytime concentration of about 6 ng BaP per m³ in the center of Stockholm has been calculated from their data (Löfroth 1979).

The first short-term bioassays of extracts of urban particulate matter were published by Freeman et al. (1971) and Gordon et al. (1973) who utilized a transformation assay with embryo cells. With the advent of

bacterial assays for mutagenicity, e.g. the Ames Salmonella-microsome test (Ames et al. 1975), a renewed interest for the organic components of airborne particulate matter emerged. Within one year, four independent groups published studies on the bacterial mutagenicity of extracts from urban particulate matter. They all concluded that PAH were not the only mutagenic compounds in this complex mixture of components (Dehnen et al. 1977; Pitts et al. 1977; Talcott and Wei 1977; Tokiwa et al. 1977).

In 1979 the National Swedish Environment Protection Board started a research program in order to improve the understanding of urban air pollution. The research was soon focused on genotoxic effects and included both ambient air as well as motor vehicle emissions. This report summarizes the work performed on ambient air since 1983. Earlier work, largely consisting of method developments, has been reported by Gustafsson (1983), in the form of separate conference articles (Lewtas et al. 1985) and elsewhere.

When the work was started some knowledge existed about the presence of common air pollutants in Swedish urban areas, such as sulfur dioxide (SO_2), soot and carbon monoxide (CO). Some data about total suspended particulate matter (TSP) and lead were also available. Only a few measurements had been made with respect to elemental composition, PAH and volatile organic compounds. Assays for mutagenicity of urban particulate matter had commenced in 1978 (Löfroth 1981). Trend analysis for some common air pollutants have been possible from data collected in Gothenburg since 1960 (Göteborgs kommun 1989; and previous reports). These data show decreasing concentrations of SO_2 and soot since 1960 and somewhat lower concentrations of nitrogen oxides in the later part of the 1980s compared to the 1970s (cf. Fig. 1). Air pollution from space heating appears to have decreased, while air pollution from motor vehicles seems to have changed in a more complex pattern. Modern vehicle engines with a more efficient combustion emit less CO and soot than earlier engines, but NO_x -emissions have not changed in a similar way.

The main purpose of this work has been to increase the knowledge of genotoxic components in urban ambient particulate matter in order to improve risk assessment and abatement strategies. In order to achieve this, the chemical composition and biological activity of ambient air has been determined, followed by an estimation of contributing sources.

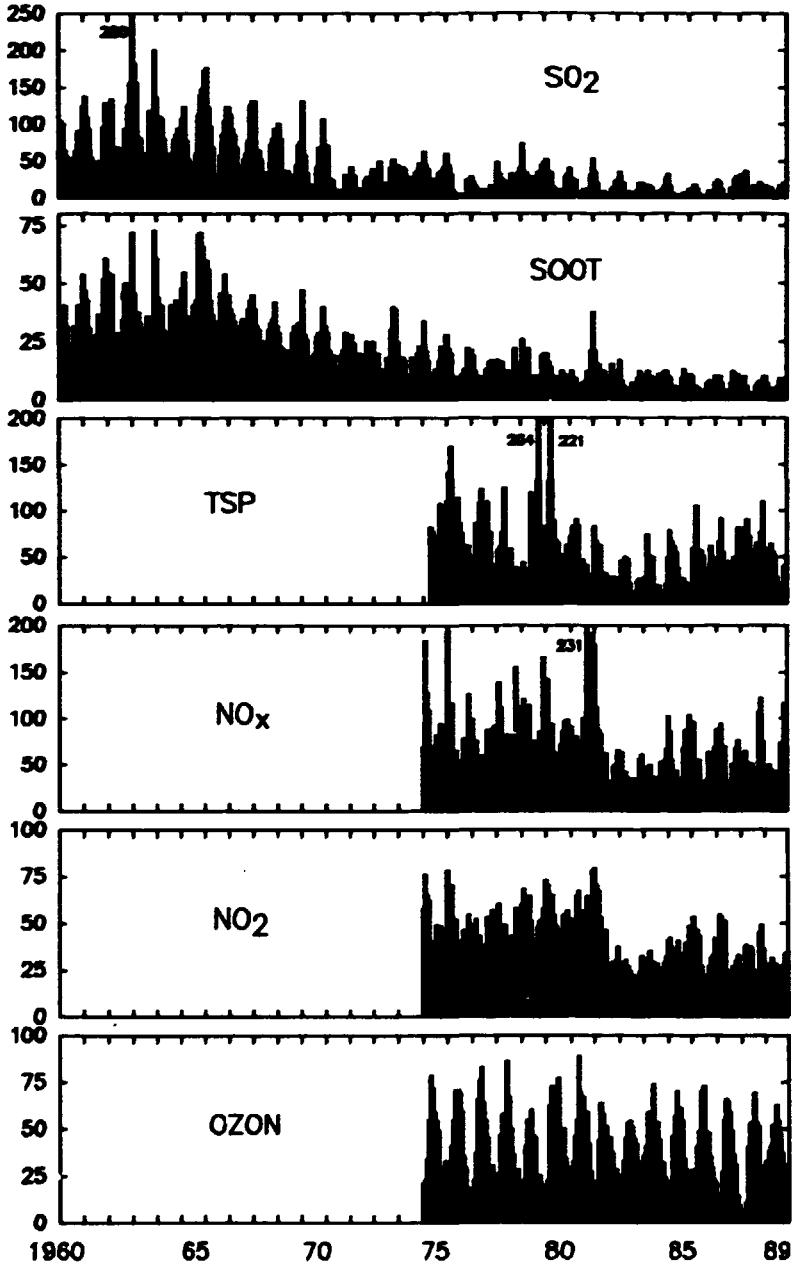


Fig. 1. Monthly average concentrations in $\mu\text{g per m}^3$ of sulfur dioxide, soot, total suspended particles, nitrogen oxides and ozone in central Gothenburg in the period 1960/1975-1989.

Materials and methods

Sampling program

Samples have been collected at several locations in the Gothenburg urban area. The sampling sites are shown in Fig. 2 and 3 and further described below. The locations were chosen in order to represent various types of local urban environments apparent within the wider urban region. When comparing different sites, samples were collected simultaneously at two sites, in order to compensate for the relatively low number of samples in each of the series.

Each sample collection consisted mostly of two consecutive 24-hour periods but some samples were collected during one 24-hour period and some during four 24-hour periods.

E series

This study was performed in 1984/85 with simultaneous sample collection at the rooftop (ER) and street (ES) levels at a city center location (Fig. 2 and 3). The rooftop site was a regular air pollution monitoring station. Assessed parameters included mutagenicity and cell toxicity for all samples, selected elemental composition for most samples and TCDD receptor affinity and PAH for selected samples in addition to the regular evaluation of soot, SO_2 , NO_x and O_3 at the rooftop site.

C, D, T and A and B series

These studies comprised a selection of sites designated as:

C series, central (+) and non-central (-) location

D series, high diesel (+) and low diesel (-) location

T series, high traffic (+) and low traffic (-) flow location.



Fig. 2. Roof and street sampling locations for the E series.

The A series are individual samples within the C, D and T series which were preselected to be sufficiently large to permit chemical fractionation. The samples were thus collected during four 24-hour periods instead of the regular two 24-hour periods. One sample became sufficiently large after only one 24-hour period due to the presence of a long-range transported aerosol.

The B series was specially designed to study the impact of an open air bus station (+) in comparison to a nearby backyard (-). The series contains only five samples collected within a month.

Site description

Bräckevägen - D/B and T/B

The sampling point was located on a parallel local street 6 m south of the edge of the main four-lane road. The average traffic flow is about 29,000

vehicles per day of which 14 % are heavy duty trucks. Bräckevägen is located close to an industrial district with refineries, container and oil harbours and car factories. The location was considered a "high diesel" and "high traffic" site when compared to other sites.

EPR (rooftop) and EPS (street)

Samples were collected on the roof of a building located close to the railway station and on the neighbouring street (Fig. 2). The traffic flow is about 16,000 vehicles per day. The site is representative of the urban center of Gothenburg.

Kastellgatan - TPK

The sampling point was located on a street with low traffic intensity in a residential area near the city center. Green park areas were situated a few blocks away. The location was considered as a "low traffic" site.

Mannheimers väg - C/M

The sampling point was located on a hill in a central area, some distance from streets with high traffic intensity. The site was considered as a "central" and a "low traffic" location.

Norska gatan - T/N

The sampling point was close to that of Bräckevägen, but it was located in a residential area with little or no heavy vehicle traffic. The site was considered as a "low traffic" location.

Nya Allén - D/M

Samples were collected NW of the centrally located four-lane street with about 23,500 vehicles per day of which about 3 % were heavy duty vehicles. The street is avenue-like with trees and narrow green verges on both sides. Sampling was carried out 4 m from the edge of the street. The site was considered as a "low diesel", "central" and "high traffic" location.

Övre Husargatan - D/P and T/P

Sampling was undertaken 5 m from the two-lane street which carried a traffic flow of about 25,000 vehicles per day with about 3 % heavy duty vehicles. The area is mainly residential and is close to the Kastellgatan site. The site was considered as a "high traffic" location.

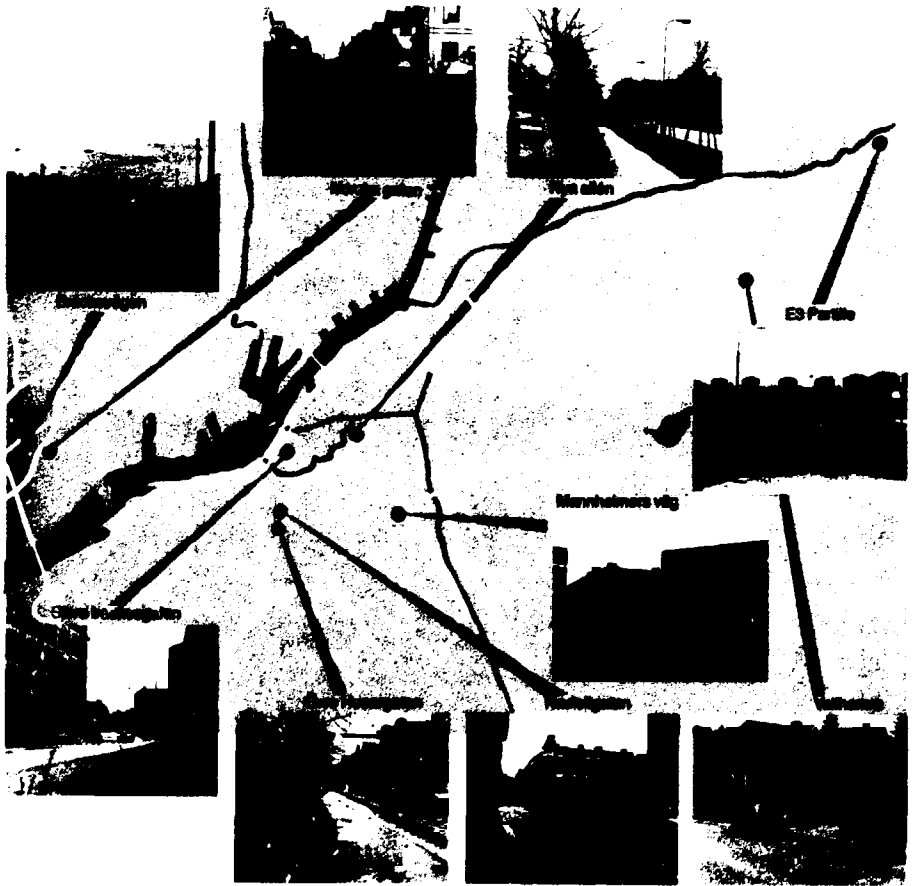


Fig. 3. Sampling sites for the C, D and T series.

Partille - T#P

Sampling was undertaken 15 m NNW of the E3 highway in the Partille district. The traffic intensity was about 33,000 vehicles per day and 8 % of this was due to heavy duty vehicles. A coal-fired district heating plant and a waste incinerator are located 3-4 km W of the sampling site. The site represented a "non-central" area with "high traffic".

Stora Badhusgatan - D#S

The sampling point was located 1 m off the roadway on a four-lane canyon-type street. The traffic flow was approximately 26,000 vehicles per day of which about 10 % was heavy duty vehicles. Less than 50 m from the site was an oil-fired power plant. Due to its tall stack it was assumed that there was no significant influence from combustion products at the sampling point. As the plant used bunker C oil, its presence would not influence the concentration of volatile hydrocarbons. The area is of a mixed commercial and residential type with an intensive through-traffic. The site was considered as a "high diesel", "high traffic" and "central" location.

Vågmästarplatsen - B#V and B#G

Samples in the B series were collected at the Vågmästarplatsen bus terminal (BV) and in a back-yard among the neighbouring apartment houses (BG). Vågmästarplatsen is an open place with several intersecting roads and many traffic lights.

Vallhamra - C#V and T#V

The sampling point was located 10 m W of a moderately busy road in the outskirts of Gothenburg. The area is mainly residential with a high portion of single-family houses. The site was considered a "non-central" and a "low traffic" location.

Sampling equipment

Sampling was performed from two identically equipped trailers, each having two high-volume particle samplers (ASTM 1962) with glass fiber filters (Munktell MG 160NH), one low-volume particle sampler with membrane filter

(Millipore AAMP), one soot sampler (OECD 1965) and one sampler for hydrocarbons on charcoal tubes. All samplers were operated over consecutive 24-hours periods between which filters and tubes were changed. The high and low volume samplers were run at the same face velocity, 70 cm/s, in order to decrease dissimilarities in cut-off characteristics due to different intake geometries. The hydrocarbon sampling was undertaken for one minute each hour in order to avoid column breakthrough.

Sample analysis

TSP

The total weight of the collected high volume particle samples were determined by weighing the filters before and after sampling after an equilibration for 6 hours at room temperature. The filters were stored in aluminum foil at -20°C until they underwent extraction for chemical and biological analysis.

Elemental composition and ionic species

The particle mass collected on the membrane filters was determined by weighing filters after equilibration at constant temperature and humidity. X-ray fluorescence was used to determine particle bound elements on the membrane filters. The analysis was initially made with a wave-length dispersive method (Grennfelt et al. 1971). A breakdown of the X-ray tube brought on a change to an energy dispersive method (Öblad et al. 1982) starting from samples B1, C4, D7 and T6 in the different series. Analysis was primarily made with respect to the elements S, Pb, Ti, Ni, V and Cl, but data on other elements were also obtained when the energy dispersive method was used.

In the determination of ionic species, sulfate, nitrate and chloride, the membrane filters were extracted in 10 ml mQ water for 24 hours. The water extract was then analysed on a Dionex 10 ion-chromatograph equipped with an

anionic column and conductive detector. Identification and quantification was made with external standard solutions.

Soot

Soot was determined according to OECD (1965). Air was drawn through a Whatman cellulose filter for 24 hours, at a rate of 1.4 l/min, after which the reflectance of the darkened area was determined.

Aromatic hydrocarbons

The activated carbon was added to a screw-capped vial containing 4 ml carbon disulfide and the vial was shaken for about 10 min. A 2 μ l portion of the solvent was then analyzed by gas chromatography on a Varian 3700 instrument with flame ionization detection and a 30 m CP Wax 52CB capillary column. A 4 ml standard solution containing 1.25 μ g/ml of benzene, toluene and xylene isomers was also shaken with activated carbon in the same way as the samples and this solution was used for quantification.

Parameters determined at the main monitoring station

This station was a regular air pollution monitoring station located on the roof of a city center building (Fig. 2).

Sulfur dioxide was determined with a Bendix 8300 using a flame photometric detector based on the chemiluminescence of sulfur compounds in a hydrogen flame. Nitric oxide and nitrogen dioxide were determined with a Bendix 8100 instrument based on the chemiluminescent reaction of NO with ozone. Ozone was determined with a Meloy OA 330 instrument using the chemiluminescent reaction between ozone and ethylene.

Wind velocity and direction were determined by a cup anemometer and a wind vane of Lambrecht type. The temperature was measured by resistance thermometer and the humidity by a LiCl cell. Data on cloudiness and precipitation were obtained from the Swedish Meteorological and Hydrological Institute weather station at Säve airport, located 9 km NW of the city center.

Particle extraction and extract analysis

Extraction

All high volume filter samples were Soxhlet-extracted with acetone (E. Merck, FRG) using about 250 ml solvent and a 100 ml thimble extractor with about four cycles per hour. The E series samples were extracted at the Swedish Environment Research Institute, Gothenburg, for 24 hours and the extract was concentrated to a small volume by rotary evaporation. Aliquots of the concentrated extracts were then shipped in a -20°C freezer container to the Department of Radiobiology, University of Stockholm for further processing which took place within one or two days after extraction. All other high volume filter samples were extracted at the Department of Radiobiology for 16 hours and the extracts were concentrated and processed directly after the end of the extraction. The concentrated extracts were subdivided in aliquots for chemical analysis and assays of mutagenicity, cell toxicity and affinity to the dioxin receptor.

Fractionation

The crude extract was fractionated with respect to polarity on a 15 mm x 120 mm silica column (Merck Kieselgel 60, 70-230 mesh). The silica was heated to 500°C for 16 hours prior to deactivation with 10 % (w/w) distilled water. The column was conditioned with 112 ml hexane (Rathburn Chemical Ltd., GB) before sample application. The extract was adsorbed on silica before introduction on the column. Five fractions were collected: fraction I, 22 ml hexane; II, 90 ml hexane; III, 112 ml 25 % dichloromethane (DCM) (E. Merck, FRG) in hexane; IV, 112 ml DCM and V, 112 ml methanol (E. Merck, FRG).

Portions (1/3) of each fraction were combined to a reconstituted sample. The fractions and the reconstituted sample were evaporated to near dryness and then solvent-exchanged to acetone. Aliquots were prepared for chemical analysis and bioassays.

Analysis of polycyclic aromatic compounds (PAC)

Prior to further clean-up, B,B'-binaphthyl was added as an internal standard. Crude unfractionated samples were fractionated on 6 mm x 120 mm silica column as described above with correspondingly smaller fractions. In order to check recovery, a dimethyl-formamide clean-up according to Stenberg et al. (1981) was made on selected samples beside fractionation.

Fraction II and III were analysed by gas chromatography flame ionization detection (GC-FID), gas chromatography mass spectrometry (GC-MS) and liquid chromatography (HPLC). Fraction II was analysed with respect to 14 PAHs and to sulfur-containing PAHs, dibenzothiophene and benzo(b)naphtho(2,1-d)thiophene. The analysis was performed on a Hewlett Packard 5790 gas chromatograph with a 10 m SE-54 fused silica capillary column, temperature programmed from 70 to 300 °C with 7 °C/min. The time for splitless injection was one minute. Identification and quantification were made by relative retention times and relative response factors. In order to confirm the identification, GC-MS was used. The mass spectrometer was a JEOL JMS-D300 instrument with a Finnigan INCOS system. The gas chromatograph connected to the mass spectrometer was also a HP 5790 equipped and temperature programmed as above.

Fraction III, containing mononitro PAHs, was analysed for 1-nitropyrene by means of a reversed phase, two dimensional HPLC. The analysis was carried out using "heart-cutting" technique and on-line reduction of nitropyrene to aminopyrene by a platinum/rhodium catalyst according to Tejada et al. (1982). The aminopyrene was detected by fluorescence.

The detection limit for the PAH analysis was typically 0.01 ng/m³. For the analysis of 1-nitropyrene the limit of detection was 1 pg/m³

Mutagenicity assays

The extracts were solvent-exchanged to dimethyl sulfoxide (DMSO) and then stored at -20°C until the samples were tested. The methods used were the plate incorporation test of the Ames Salmonella mutagenicity assay (Maron and Ames 1983) and a modified version (Löfroth et al. 1988) of the micro-suspension test (Kado et al. 1983). Mammalian metabolic activation was obtained with liver-S9 from Aroclor 1254-induced male Sprague-Dawley rats supplemented with necessary co-factors. The amount of S9 added was $20\ \mu\text{l}$ per plate in the plate incorporation test and $10\ \mu\text{l}$ per tube in the microsuspension test.

The response, expressed as revertants per mg particulate matter or per m^3 air, was obtained from the linear part of the dose-response curve. Each sample was tested with at least four doses on three independent test occasions with one plate per dose or on two independent test occasions with two plates per dose.

Cell toxicity

Toxicity was evaluated on ascites sarcoma BP 8 cells which were grown in suspension culture, in Hams F10 medium complemented with 10 % fetal calf serum, penicillin (96 IU/ml) and streptomycin (83 $\mu\text{g}/\text{ml}$), in a CO_2 -incubator with 5 % CO_2 in air at 37°C and 80 % relative humidity according to Curvall et al. (1984). The samples to be tested were dissolved in $20\ \mu\text{l}$ DMSO and added to 2-ml aliquots of cell suspension containing 30,000 cells/ml. The tests were in triplicate for at least three different concentrations in each experiment. After an incubation for 24 h, the cell density was determined utilizing an electronic particle counter. Inhibition of cell growth is expressed as the ratio between the growth rate of the treated cell culture and the solvent blank (DMSO). The growth rate was calculated from $\ln(N/N_0)/(t \times \ln 2)$ where N_0 is the cell density at the start of the experiment and N is the density at time t (24 h). The 50 % inhibitory concentration was determined by linear estimation of the dose-response curve and a mean value for each sample was calculated based on several experiments. Evaluation of the difference in toxicity between the

roof and the street samples was performed using the t-test of two means and the combined probability analysis based on the p-values of these tests (Fisher 1950).

Assay for TCDD receptor binding

Compounds competing with ^3H -2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) for binding to the TCDD receptor were assayed as described by Poellinger et al. (1985). Rat liver cytosol was labelled with either 1.5 nM ^3H -TCDD, to estimate total TCDD binding in the cytosol, or 1.5 nM ^3H -TCDD + 300 nM non-radiolabelled 2,3,7,8-tetrachlorodibenzofuran (TCDF), to estimate the binding of ^3H -TCDD to non-receptor components. The difference in ^3H -TCDD binding in the absence and presence of TCDF is thus considered as receptor-bound ^3H -TCDD. To assay for compounds in air samples that compete with ^3H -TCDD for binding to the receptor, rat liver cytosol was incubated with 1.5 nM ^3H -TCDD in the presence of increasing concentrations of extract from air samples. The incubations were then adsorbed to hydroxylapatite in a batch assay. The use of hydroxylapatite permits the separation of TCDD receptor from non-receptor TCDD binders in the cytosol, and also from the free ligand. The binding of ^3H -TCDD to the receptor in the presence of air samples was expressed as a percentage of the binding to the receptor seen in the control samples. The relative binding affinities for the individual air samples were calculated from log-logit plots of the competition for TCDD-binding were $\text{logit (receptor binding)} = \ln (\text{receptor binding} / (1 - \text{receptor binding}))$. The log-logit plots were calculated using linear regression analysis. The results were expressed as EC_{50} -values, EC_{50} meaning the concentration of sample inhibiting 50 % of ^3H -TCDD binding to the receptor seen in control samples.

Computations

Partial least squares (PLS) regression

PLS is a multivariate data analysis method useful for establishing relationships between different sets of variables (Wold et al. 1984). Here, PLS is used as a means of predicting the mutagenicity (TA98+S9) from a number of chemical variables, e.g. PAH, sulfur-PAH (S-PAH), simple aromatics and lead. The SIMCA software package (Wold and Sjöström 1977) was used for this. A more extensive multivariate analysis of the data presented here, is given elsewhere (Alsberg et al. 1989).

Source apportionment

In principle, two alternative ways exist when determining source contribution. One uses meteorological dispersion models and the other uses the ratios between different substances and their covariation, i.e. receptor models. The latter types were chosen in this project, as dispersion models cannot account for background contributions and require data which were not available.

The first step in receptor modelling is to inquire about number and types of sources that influenced the samples which were collected. This has been done partly by factor analysis and partly by just estimating the magnitude of known sources in the urban area.

Initial principal component factor analysis (Johnson and Wichern 1988) was used to find the number of factors having communalities above one and these then underwent further analysis with varimax rotation. The variables having a strong or moderate factor loadings were then used to identify the factors.

Two methods are used for the determination of source contributions:

- weighted least square chemical mass balance, CMB (Henry et al. 1984)
- multilinear regression, MLR (Morandi et al. 1987)

The CMB method requires knowledge of the ratio of the measured parameters both in ambient air and contributing sources. The method is based on solving the overdetermined system:

$$C_i = \sum a_{ij} S_j$$

where C_i is the relative concentration of substance i in ambient air, a_{ij} is the relative concentration of the substance i in the source j and S_j is the contribution from source j . Relative source concentrations were obtained from Egeback and Bertilsson (1983). The parameters used in the calculations are given in Table I together with estimates of their errors.

Table I. Measured average ambient concentrations and source signatures used in the chemical mass balance calculation.

Component		Ambient concn. /m ³	Gasoline exhaust /km	Diesel exhaust /km	Road dust	Oil heating
TA98-S9	rv./m ³ or km	9.33 (1.25) ^a	40000 (2)	400000 (2)	1 (2)	10000 (4)
Ti	μg/m ³ or km	0.335 (1.10)	10 (6)	250 (6)	13000 (1.5)	50 (4)
V	ng/m ³ or km	42.6 (1.20)	1000 (6)	5000 (6)	20000 (2)	300000 (2)
Pb	μg/m ³ or km	0.350 (1.10)	3300 (2)	150 (2)	30 (2)	75 (4)
Benzene	μg/m ³ or km	9.36 (1.10)	90000 (1.5)	25000 (1.2)	0 (-)	0 (-)

a) an estimate of the error expressed as a factor of the given value.

The MLR method requires no knowledge of the relative concentrations of the substances in the sources, but needs more or less unique tracers for the major contributors. Ideally this method requires that tracers do not covary. This is, however, not the case with the present data as high concen-

trations of diesel exhaust caused by high traffic intensity occur at the same time as high concentrations of gasoline exhaust. A small variation, however, in the intensity of diesel traffic between the sites and between business and rush hours may introduce the necessary divergence between parameters. Loadings which are high only for one factor indicate tracers for the multiple linear regression analysis. In the case where a factor has no unique tracer, a multiple linear regression analysis is performed with a candidate tracer. The candidate tracer acts as an independent variable and is used to find the unique part of this tracer for the factor in question. The unique part is assumed to be the part of the tracer candidate not being significantly explained by other factor tracer substances. The new modified tracer can now be used together with the others in a multiple linear regression with the formula:

$$P = \sum K_i C_i + R$$

where P is the variable for which the source contribution is sought, K_i is the regression coefficient of the element i, C_i is the concentration of the element and R is a residual term.

The source contribution S_j is obtained by:

$$S_j = \frac{K_i C_i}{p}$$

where i is the tracer for the source j.

Results and discussion

Primary data

Measurement data are given in the Appendix Tables 1-3 in which most primary data are listed. Not included are, however, some inorganic elemental components and some PAH.

E series, street and rooftop

In this study, comprising 20 parallel samples collected over a period of about one year, a comparison was made between a street location and a nearby rooftop site.

Although there was a considerable difference in the particulate concentrations between the two sites (Table II), the overall average mutagenic activity was about the same. This is also the case for the toxicity to the mammalian cell system and for the limited number of samples analyzed for affinity to the TCDD receptor. The biological effects are caused by a complex mixture of organic compounds of which the PAH are a part. The similar average concentration of PAH at the two sites is an additional indication that the long-term average concentration of organic compounds adsorbed to particulate matter is of about the same magnitude at the two sampling sites. In contrast, the concentration of lead is less at the rooftop location than at the street level. This may, partly at least, be due to the resuspension of larger street dust particles containing deposited lead, which causes an apparent increase in airborne lead concentration at the street sampling site.

The range of the EC_{50} values in the TCDD receptor test, 0.0006-0.12 m^3/m cytosol, is similar to those found in an earlier study with samples from the Stockholm area (Toftgård et al. 1983), with the exception of a very low value for sample E12S (0.0006 m^3/m) collected during an inversion. Also in similarity to the previous study, the determined concentrations of various PAH which bind to the TCDD receptor, cannot account for the observed competition.

Table II. Average and range concentrations measured at the street and rooftop sites; from Appendix Table 1.

Component	Unit	Street location		Rooftop location	
		Average	Range	Average	Range
TSP	$\mu\text{g}/\text{m}^3$	81	29-210	51	34-120
Soot	"	- ^a	-	11	1.5-22
SO ₂	"	-	-	15	3-52
NO _x	"	-	-	83	27-320
NO ₂	"	-	-	34	18-53
O ₃	"	-	-	33	8-89
Lead	ng/m^3	215	38-570	112	20-400
PAH ^b	"	29	2.4-100	25	1.3-98
Mutagenicity					
TA98-S9	$\text{rev.}/\text{m}^3$	8.7	1.5-20	9.0	0.9-24
TA98+S9	"	13	2.0-51	11	1.2-41
TA100-S9	"	7.5	1.2-21	6.9	0.9-19
TA100+S9	"	16	2.7-61	13	1.6-51
Cell toxicity ^c	m^3/ml	5.3	16-2.4	5.1	20-2.0
TCDD affinity ^d	"	0.003	0.07-0.0006	0.010	0.12-0.0043

a) - not measured; b) 5 periods; c) 17 periods; d) 6 periods.

The mutagenicity was determined with two *Salmonella* strains in the absence and presence of S9 metabolic activation. It is therefore feasible to look for variation in the pattern of activity between the two sample sites. It can be noted (Table III) that when compared with the rooftop samples, the street samples have a higher average increase of activity in the presence of S9, i.e. the ratio of +S9/-S9 is larger. As samples were assayed at different times causing an inherent variation, these ratios are not significantly different. Normalizing each of the twenty sample sets by forming the ratio street/roof results in average ratios which are statistically different from one. Although the mutagenic activity is about the same for both sites, this indicates that the mutagens contributing to the activity

Table III. Average ratios of the mutagenic activity of E series.

	Average ratio \pm S.E.			
	TA98 +S9/-S9	Street/Roof	TA100 +S9/-S9	Street/Roof
E Street samples	1.47		2.12	
E Rooftop samples	1.21	1.28 \pm 0.08	1.85	1.16 \pm 0.07

also are different to some extent. This difference is probably due to atmospheric transformation, which has occurred during the particle transport to the rooftop level from street levels upwind to the rooftop site.

C, D, T and B series

The results from the analysis of the C, D, T and B series, as given in Appendix Tables 2 and 3, are condensed in Table IV.

Among major parameters, only the TSP concentrations are consistently higher at the +locations than at the -location for all series. A comparison between the + and -location for other parameters is more variable with components having similar average concentrations in some series and lower concentrations in other series.

A relatively large difference was observed for the + and - locations in the B series showing that a bus terminal is a substantial local source for air pollutants.

Table IV. Average concentrations and ranges for D, T, C & B series.

Component	Unit	+ Location		- Location	
		Average	Range	Average	Range
C series, 10 periods					
TSP	$\mu\text{g}/\text{m}^3$	58	22-200	46	16-160
Soot	"	17	3.5-48	17	2.3-48
Lead	ng/m^3	160	12-640	140	27-400
Mutag. TA98-S9	$\text{rev.}/\text{m}^3$	11	1.3-47	9.6	1.0-38
" TA98+S9	"	8.4	0.8-34	8.7	0.6-37
ΣPAH	ng/m^3	9.7	0.6-42	9.9	1.2-33
1-Nitropyrene	pg/m^3	8.6	<1-36	3.8	<1-11
Benzene	$\mu\text{g}/\text{m}^3$	3.4	<0.3-7.9	2.9	0.3-7.0
D series, 12 periods					
TSP	$\mu\text{g}/\text{m}^3$	140	29-350	86	33-190
Soot	"	59	6.6-190	26	7.3-43
Lead	ng/m^3	730	49-1900	700	110-1600
Mutag. TA98-S9	$\text{rev.}/\text{m}^3$	13	1.1-35	12	2.0-30
" TA98+S9	"	13	1.4-39	12	2.0-28
ΣPAH	ng/m^3	24	1.0-57	17	2.8-38
1-Nitropyrene	pg/m^3	20	1-68	12	2-35
Benzene	$\mu\text{g}/\text{m}^3$	17	0.7-50	14	4.2-26
T series, 16 periods					
TSP	$\mu\text{g}/\text{m}^3$	74	20-280	51	15-200
Soot	"	28	7.6-70	17	5.4-55
Lead	ng/m^3	250	60-930	140	21-380
Mutag. TA98-S9	$\text{rev.}/\text{m}^3$	8.3	2.1-18	6.3	0.5-18
" TA98+S9	"	8.5	2.0-17	6.2	0.3-19
ΣPAH	ng/m^3	18	3.3-56	9.5	0.7-33
1-Nitropyrene	pg/m^3	14	1-42	6.8	<1-32
Benzene	$\mu\text{g}/\text{m}^3$	9.6	1.0-30	6.7	0.3-24
B series, 5 periods					
TSP	$\mu\text{g}/\text{m}^3$	140	40-230	66	28-120
Soot	"	47	14-88	29	2.7-59
Lead	ng/m^3	380	200-700	170	33-300
Mutag. TA98-S9	$\text{rev.}/\text{m}^3$	11	5.2-16	7.3	2.9-13
" TA98+S9	"	15	4.8-22	7.3	2.1-15
ΣPAH	ng/m^3	38	8.8-79	14	2.9-25
1-Nitropyrene	pg/m^3	17	6-32	12	3-24
BNT	ng/m^3	0.59	0.06-1.2	0.16	0.06-0.27
Benzene	$\mu\text{g}/\text{m}^3$	12	6.3-17	7.3	4.2-11

Mutagenic activity

The mutagenic activity in the *Salmonella* strain TA98 varied from a low response of about 1 rev./m³ to 47 rev./m³. As the samples were mainly collected over 48 h, the variation between shorter periods, e.g. 24 h, can be higher. The mammalian metabolic activation sometimes decreased and sometimes increased the activity.

Although the samples were collected at different locations, the similarity between parallelly sampled locations may permit the use of the entire set for an inquiry about the seasonal variation. All 76 samples from the C, D and T series are plotted against time of the year in Fig. 4 and compared with similar data from Stockholm (Löfroth 1981; Alfheim et al. 1983 and a few additional samples). The seasonal variation shows that a high response is most likely to occur during the winter months. This has also been noted in other studies (Daisey et al. 1980; Reali et al. 1984; Wullenweber et al. 1982). Higher response during winter is most probably due to a combination of factors including more combustion, colder vehicle engines, less windy climate, a higher adsorption of organic compounds to particles, etc.

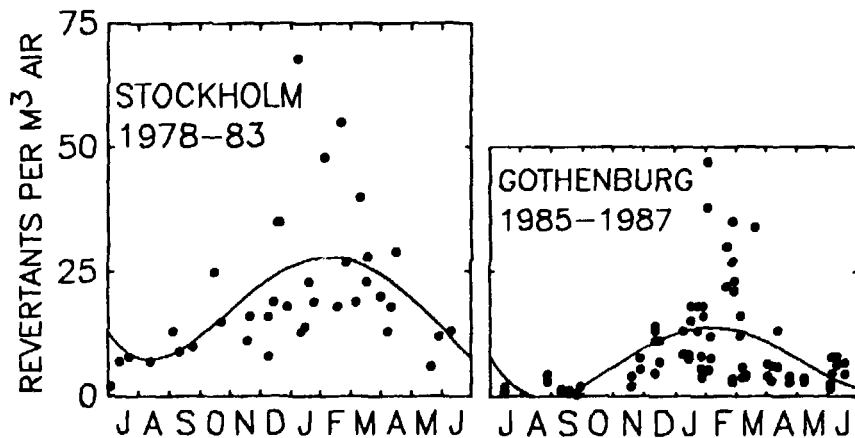


Fig. 4. The annual variation of mutagenic response in TA98-S9 of airborne particulate matter collected at a rooftop site in central Stockholm and several street locations in Gothenburg. Each point is the average of two (Gothenburg) and two-four (Stockholm) consecutive 24 h samples. The response per m³ air is plotted against the month of collection from July to June and a moving average has been fitted to the data. It can be estimated that the results from Stockholm corresponds to an annual average of 18 rev./m³ and those from Gothenburg to 8 rev./m³.

Polycyclic aromatic compounds (PAC)

Selected PAC concentrations are given in the Appendix Tables 1-3. A more detailed PAH analysis was made on the A samples included in the C, D and T series and the results are given in Appendix Table 4.

Samples collected during the winter showed markedly higher levels of PAH which is in accordance with earlier results (Prahl et al. 1984). This is illustrated in Fig. 5. It could be explained by meteorological conditions, i.e. inversions, by cold motors and impact from local heating. Greater adsorption of semivolatile PAH (with three to four rings) on particles at lower temperatures is also important.

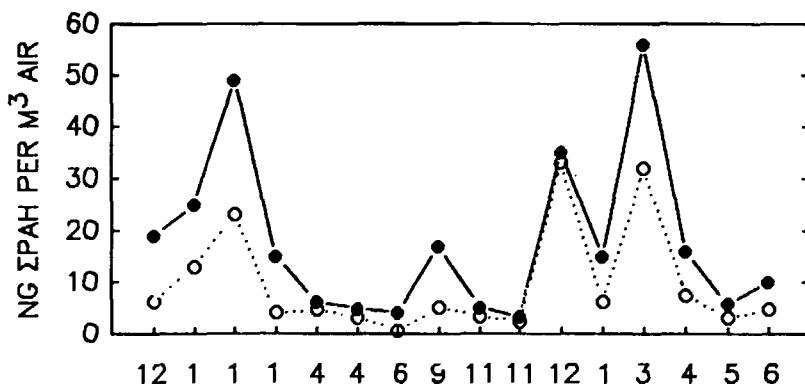


Fig. 5. The variation of the PAH concentration over 1.5 years within the T+ (●) and T- (○) series.

1-Nitropyrene was analyzed in all samples. No obvious trend or covariance with other measured parameters was, however, seen for this compound.

From emission data (Alsberg et al. 1989), a higher proportion of 3-4 ring PAH and sulfur containing PAH was expected in connection with a high load of diesel vehicles. Special efforts were thus made to analyse sulfur containing PAH like dibenzothiophene and benzo-(b)naphtho(2,1-d)thiophene (BNT). BNT was preferred as an indicator for diesel traffic due to its higher particle to gas phase distribution ratio.

The influence from diesel vehicle exhaust can be seen within the D series. In Fig. 6 the ratios of BNT/BeP for the D+ samples are compared to those for the D- samples. For the majority of samples the relative BNT level is higher in samples with an expected large influence from diesel traffic.

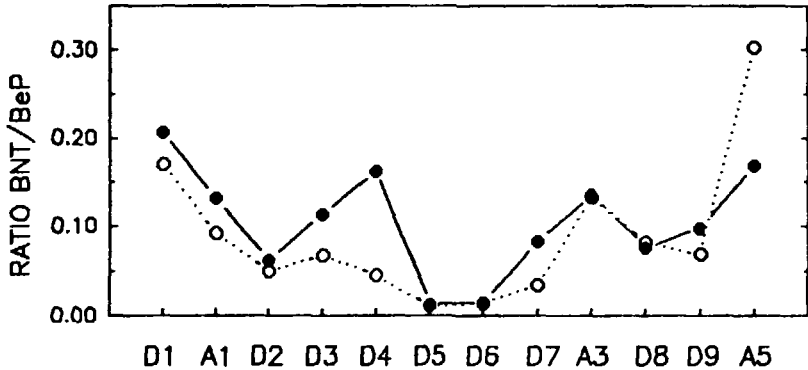


Fig. 6. The ratio of BNT/BeP in the D+ (●) and D- (○) series.

The samples within the C series showed small differences between the + and - locations and generally low concentrations of PAH. Special attention was paid to the analysis of retene (1-methyl-7-isopropylphenanthrene) in these samples, since retene has been proposed as a marker compound for wood combustion (Ramdahl et al. 1983). No trend was, however, seen for retene concentrations with regard to sample site or meteorology. In fact, this was not to be expected since wood is not a predominant fuel in areas where the sampling took place.

The levels of sulfur-containing BNT are somewhat higher in the B+ samples supporting the hypothesis that this compound is useful as an indicator for diesel vehicle traffic.

Fractionation

The five sampling periods comprising ten samples, selected for fractionation, are representative for the sampling series and locations. The selection, however, also considered the potency of the samples. No meaningful results would have come from the fractionation of samples with very low activity, as the amount of each sample was limited.

The fractionated samples were subjected to a more detailed PAC analysis (Appendix Table 4), mutagenicity (Appendix Table 5) and TCDD receptor binding (see below). The results on these parameters for the crude unfractionated samples are given in Table V.

Table V. PAH concentrations and biological activities of the unfractionated samples selected for fractionation.

Sample	Σ PAH, ng/m ³		Receptor binding		TA98 revertants/m ³			
	+location	-location	EC ₅₀ , m ³ /ml		+location		-location	
	a / b	a / b	+loc.	-loc.	-S9	+S9	-S9	+S9
A 1	12 /14	24 /28	0.12	0.065	7.7	5.6	12	11
A 2	6.1 /6.4	4.7 /5.0	0.11	0.025	6.1	4.1	3.1	3.2
A 3	2.5 /2.7	3.8 /4.2	- ^c	-	3.8	2.4	4.2	3.9
A 4	42 /47	33 /37	0.042	0.060	47	34	38	37
A 5	41 /47	9.6 /11	0.015	0.057	13	13	5.9	6.4

a) from Appendix Table 2 and 3; b) from Appendix Table 4; c) - not tested.

The total calculated PAH concentration does not change appreciably with the inclusion of additional compounds detected in the more detailed analysis. There is a gross correlation between the PAH concentration and the biological activities.

The EC₅₀ values for TCDD receptor binding was determined on fractions from three of the A samples (Table VI).

Table VI. Competition for TCDD receptor binding by fractionated extracts of urban particulate matter. The results are given as EC_{50} values with m^3/ml cytosol^a.

Fraction/Sample	A 1B	A 1N	A 2P	A 2V	A 4M	A 4V	A 5S	A 5N
Crude extract	0.12	0.065	0.11	0.025	0.042	0.060	0.015	0.057
Fraction I	^b	-	6.2	0.53	>23	>23	>6.9	>15
Fraction II	-	-	0.13	0.39	0.067	0.056	0.049	0.30
Fraction III	-	-	0.95	1.0	0.17	0.10	0.16	0.84
Fraction IV	-	-	0.63	0.20	0.14	0.074	0.30	1.3
Fraction V	-	-	0.35	2.7	0.064	0.43	0.97	0.92
Reconstituted sample of fraction I-V	0.54	0.17	0.16	0.049	0.045	0.064	0.022	0.18
(% of crude)	(22)	(38)	(69)	(51)	(93)	(94)	(68)	(32)
Sum of fraction I-V	-	-	0.075	0.092	0.023	0.023	0.032	0.16
(% of crude)	-	-	(147)	(27)	(183)	(262)	(47)	(36)

a) the reciprocal of EC_{50} values are additive; b) - not tested.

The recovery of the receptor binding activity in the fractionation is variable as shown by the large differences between the crude and reconstituted samples as well as the calculated sum of their activities.

The results show that most of the compounds competing with TCDD for receptor binding elute in fraction II. This fraction also contains most of the PAH and has previously been shown to be the most potent when an extract of gasoline exhaust particles was fractionated in the same manner (Alsberg et al. 1985). Considerable activity was, however, also found in fractions III-V which contain more polar compounds. With one exception, sample A2V, very low levels of competition for receptor binding was

observed in fraction I, containing highly non-polar compounds. There is presently no explanation to the exceptional fraction I of sample A2V. The exact nature of the compounds competing for TCDD receptor binding is not known although for fraction II, PAH with high receptor affinity are likely to be major contributors.

The recovery of the mutagenic activity from the fractionated samples (Appendix Table 5) was about 80 %. There was no trend towards a recovery significantly above 100 % indicating that components in the different fractions did not inhibit the mutagenic activity in other fractions.

The largest mutagenic response resided in the most polar fraction, V. This distribution of activity is dissimilar to the distribution obtained from gasoline car exhaust and heavy duty diesel exhaust as illustrated in Fig. 7. The observed shift to more polar mutagens is an additional indication that atmospheric reactions have changed the exhaust emissions.

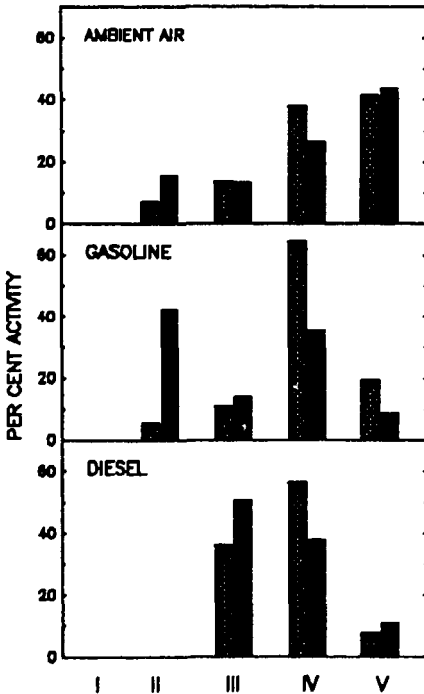


Fig. 7.

The relative distribution of the mutagenic activity without (-S9, hatched) and with (+S9, filled) metabolic activation in five fractions of increasing polarity. The activity is given as per cent of the total recovered activity in extracts from ambient air particles (this study, average of all ten A samples), gasoline exhaust particles (Löfroth et al. 1985) and diesel exhaust particles (Westerholm et al. 1990).

Mutagenic activity with microsuspension

The plate incorporation mutagenicity assay requires samples from relatively large volumes of ambient air to give a detectable response. A more sensitive system was developed by Kado et al. (1983) primarily for testing of urine samples and later utilized for air samples (Kado et al. 1986) in which a high concentration of bacteria is incubated with the sample in a small volume. This microsuspension system is a variation of the pre-incubation test method (Yahigi et al. 1977) in which also the bacteria are incubated with the sample in suspension, in contrast to the plate incorporation assay in which the incubation occurs on the agar plates.

When utilized by other investigators, the microsuspension method gave toxicity problems which were solved by changing the buffer system (Löfroth et al. 1988) or by using a diluted buffer (DeMarini et al. 1989).

In order to obtain a comprehensive comparison between the microsuspension and the regular plate incorporation assays, all 86 samples in the C, D, T and B series have been tested in both assays with the strain TA98. The complete results are given in Appendix Table 6 and a summary with response ratios is given in Table VII.

Table VII. Ratio of the mutagenic response in the microsuspension assay to that in the plate incorporation assay.

Sample		Ratio \pm SE	
		TA98-S9	TA98+S9
Air particles	(n=86)	3.81 \pm 0.13	8.18 \pm 0.48
Quercetin ^a	(n=33)	4.67 \pm 0.34	not tested
Benzo(a)pyrene ^a	(n=33)	not tested	8.97 \pm 0.43

a) the ratio is calculated from the response per unit weight obtained with 5 μ g quercetin and 0.5 μ g benzo(a)pyrene in the microsuspension assay and 25 μ g quercetin and 2,5 μ g benzo(a)pyrene in the plate incorporation assay.

For the air samples, the microsuspension gave about four times higher response in the absence of S9 and eight times higher in the presence of S9 compared to the plate incorporation method. The concurrent positive control compounds, quercetin and benzo(a)pyrene, gave about similar ratios, but this may be coincidental as the ratio is dependent on compound as well as the amount of S9 used in the two tests.

The distribution of the responses is illustrated in Fig. 8 with a linear regression slope of about 3 for the TA98-S9 condition and about 5 for the TA98+S9 condition. The differences between the average ratio (Table VI) and the linear regression slope (Fig. 8) are mainly due to the fact that a number of samples with a low response in the plate incorporation assay have a substantially higher enhancement with the microsuspension method than other samples. This may indicate that the composition with respect to mutagenic components are somewhat dependent on the level of pollution.

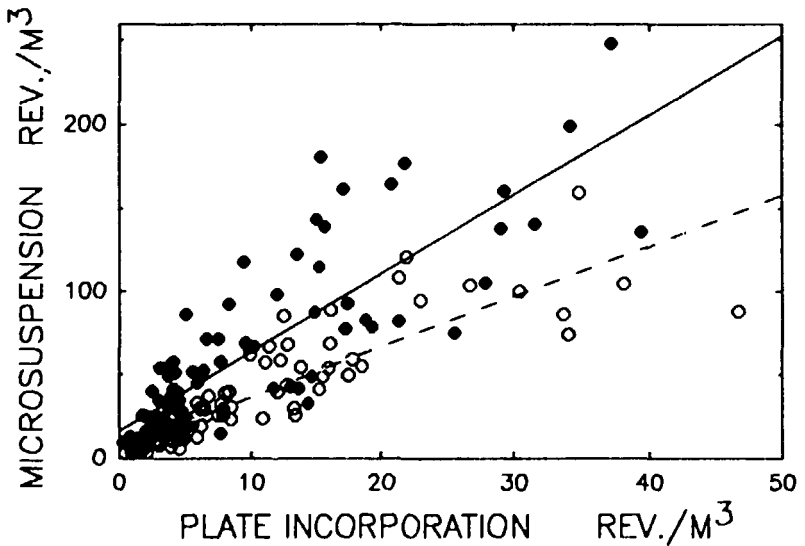


Fig. 8. The relationship between the mutagenic response with the plate incorporation assay and the microsuspension assay as found with 86 samples of airborne particulate matter;
-S9 o - - - o, +S9 ●——●.

Secondary data

Partial least squares (PLS) regression

In PLS, a "training set" is used for the construction of a prediction model, while a "test set" is used for testing the model. In other words, the measured values of the test objects are compared with the values predicted by the model. The predictive strength of a model is evaluated by plotting the predicted and measured values. Ideally, the plot should be a straight line with the slope 1. A high predictive strength is an indication of a strong, direct and indirect, relationship between the independent and the dependent variables.

In the present case, the D series with 24 samples was used as a training set and the remaining 52 samples from the C and T series comprised the test set. The use of simple aromatics, individual PAH, dibenzothiophene, BNT and lead as independent variables gave the best prediction of mutagenic response (Alsberg et al. 1989). Using the sum of PAH instead of individual PAH or omitting the simple aromatics and lead, yielded poorer regression statistics than that showed in Fig. 9.

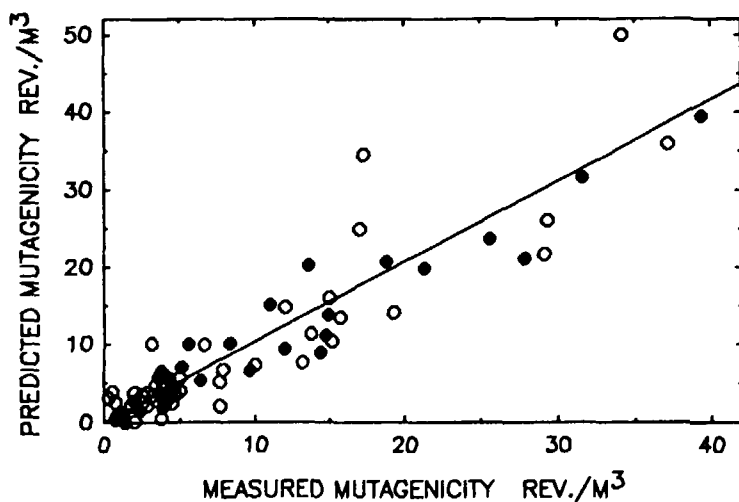


Fig. 9. PLS prediction of the mutagenic response with TA98+S9 based on simple aromatics, individual PAH and S-PAH and lead. Training set, $n=24$ from the D series: ● Test set, $n=52$ from the C and T series: ○ — ○ The slope is 0.99, $r^2=0.85$ and the standard error of prediction 4 rev./m^3 .

The conclusion from the PLS regression study is that the variables used describe the samples reasonably well in terms of mutagenic response. The result also indicates that the contribution to the mutagenicity from diesel vehicles is described by low molecular weight PAH and BNT and that high molecular weight PAH, simple aromatics and lead describe the mutagenic contribution from gasoline fueled vehicles. It should be emphasized that compounds used as predictors, need not necessarily be responsible for the mutagenic effects. This fact is evident in the case of simple aromatics, which are in the gas phase, and thus not present in the particle extracts from which the mutagenicity tests were performed.

Source apportionment

The chemical mass balance (CMB) apportionment was performed with the mutagenic activity in TA98-S9 using 76 samples from the C, D, T and B series and with input data given in Table I. The resulting shares for the mutagenic activity from the assumed sources were found to be:

Gasoline exhaust	44 ± 7 %
Diesel exhaust	41 ± 37 %
Road dust	0 ± 0 %
Oil, heating	15 ± 15 %

Errors reflected are assumed in the assignment of source signatures (Table I). The relatively large error for diesel exhaust is due to the fact that none of the tracers originated predominantly from diesel exhaust.

The factor analysis (Farahmand and Steen 1990) resulted in the identification of seven factors which had moderate to high loadings (Appendix Table 7). The assignment of the factors to known sources is, however, a matter of judgement. The factors 4, 5 and 6 assigned to long-range transport, oil combustion for heating and road-dust may not be real entities. The MLR calculation (Farahmand and Steen 1990) was first performed with 66 samples in the C, D and T series omitting samples from D1 and T1-T4 for which several elements were lacking. Singular missing values were sub-

stituted with the average concentration of the parameter and the calculation was then performed with normalized values, obtained by subtracting the average value and dividing with its standard deviation. Six outliers were identified, C3, D2 and D3, and the final regression was then performed with 60 samples with the result which is given in Table VIII.

Table VIII. Contribution of mutagenic response from the assigned factor-sources as obtained with MLR calculation on 60 samples from the C, D and T series.

Factor	Assignment	Per cent contribution \pm SE	
		TA98-S9	TA98+S9
1	Gasoline exhaust	26 \pm 3	42 \pm 3
2	Diesel exhaust	54 \pm 4	47 \pm 3
4	Long-range transport	11 \pm 3	12 \pm 3
6	Road dust	10 \pm 3	6 \pm 3
3,5,7	Other	-1 \pm 5	-7 \pm 5

About half of the total mutagenic response is accounted for by diesel exhaust. The relative higher contribution from gasoline exhaust in the presence of S9, compared to that in the absence of S9, is in agreement with studies on motor vehicle exhaust (Alsberg et al. 1985, Westerholm et al. 1988, 1990) in which addition of S9 often decreases the activity associated with diesel exhaust in contrast to the increase in activity associated with gasoline exhaust.

The two source apportionment calculations agree with each other concerning the contribution from major sources, *i.e.* diesel and gasoline exhaust, and the MLR calculation indicates a difference between absence and presence of S9, which corresponds to the mutagenic behavior of the vehicle exhausts.

The MLR calculation also gives a good prediction of the mutagenic response as can be seen in Fig. 10, where measured and predicted responses are compared graphically.

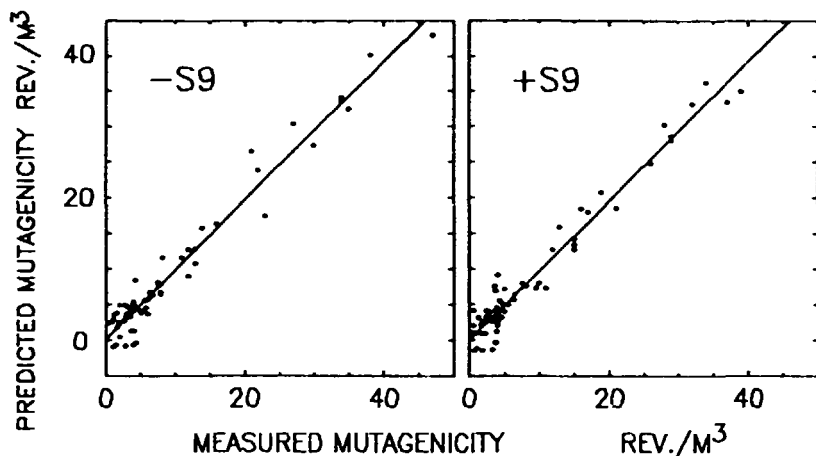


Fig. 10. The relationship between the measured and predicted mutagenic response in the MLR calculation using 60 samples. The six outliers are also incorporated after adjusting missing single values to more appropriate concentrations which reflect the time of sampling. For both -S9 and +S9 the slope is 0.98 and $r^2=0.96$ and these values are not largely dependent on the added and adjusted six outliers.

The question can be posed whether the calculated contribution from gasoline and diesel vehicles are realistic. Although there is presently no means to check the absolute contributions, the significance of their relative magnitude, 26 and 54 % in TA98-S9, *i.e.* a ratio of 1:2.1, can be verified either from the fuel usage of gasoline and diesel or from the abundance of gasoline and diesel powered vehicles and compared to the known emissions.

The use of gasoline and diesel fuel for transport in Sweden 1985 has been reported to be 5.1 and 1.8 Mm^3 , respectively, *i.e.* a ratio of 1:0.35 (Bilstatistik 1989). Assuming that this ratio also is representative for the urban environment, the relative contribution to mutagenicity per fuel volume can be calculated to be 1:5.9. Multiplication with the ratio of fuel consumption, being in the range of 1:2-5 for gasoline cars and the mix of diesel vehicles in use, yields a ratio of 1:12-30, *i.e.* the emission per unit distance from diesel vehicles is 12-30 times more

mutagenic than the emission from gasoline powered cars. This ratio in the range of 1:12-30 is not inconsistent with experimentally determined values (Alsberg et al. 1985, Westerholm et al. 1990) and is in agreement with a ratio of 27 reported by Larssen (1990) for Norwegian vehicles on the road but is slightly higher than those which can be calculated from data given by Pierson et al. (1983) for vehicles on the road in the United States.

An alternative approach can be made by using the statistics available for the distribution of vehicle traffic in representative urban areas (Statens Naturvårdsverk 1984). The pattern that emerged for major streets was 13 % diesel vehicles including light duty passenger cars (8 %) and heavy duty trucks of <10 ton (3 %), of 10-20 ton (2 %) and of >20 ton (1 %) and for secondary streets, 8 % diesel vehicles with 4 % of light duty passenger cars, 2 % of <10 ton trucks and 2 % of 10-20 ton trucks. Assuming an average of 10 % diesel vehicles the estimated ratio of emission becomes 1:19, i.e. the average diesel vehicle causes 19 times more airborne particulate mutagenic activity than the average gasoline powered car. This ratio is in the same range as was estimated from fuel usage. It can thus be concluded that the relative contribution from gasoline and diesel exhaust as calculated by MLR is compatible with emission data.

Concluding discussion

Mutagenic activity and polycyclic compounds

Studies on airborne particulate matter with respect to mutagenic activity in the Salmonella test with or without the use of other biological short-term tests and chemical analysis is an active research area. Some of the more recent international investigations have been performed in Alaska (Watts et al. 1988), Korea (Jang et al. 1989), Germany (Schleibinger et al. 1989), Italy (Barale et al. 1989), Poland (Motykiewicz et al. 1989) and Brazil (Miguel et al. 1990). Recent studies in other Nordic countries have been performed in Finland (Tuominen et al. 1988), Norway (Larssen 1988) and Denmark (Ostenfeldt 1989).

Over the years it has become evident that the mutagenic activity of the organic extracts from airborne particulate matter cannot be ascribed to a few compounds, but that the activity is the joint response of many different components. This complex mixture of compounds is generally described as polycyclic aromatic compounds (PAC) or polycyclic organic matter (POM) referring to the fact that it is much more than just polycyclic aromatic hydrocarbons (PAH). In exceptional instances it has been possible to ascribe a substantial part of the mutagenic activity to one or a few components as e.g. found by Salmeen et al. (1987) for some diesel exhaust samples in which 1-nitropyrene (INP) could be shown to cause about 20 % of the response in TA98-S9.

In contrast, it can be calculated that INP, which has a response of about 2 rev./ng in TA98-S9 (Löfroth et al. 1985), gives an average contribution of about 0.25 % for the average sample in the C, D, T, and B series; averages are 11.4 pg INP/m³ and 9.7 rev./m³. The sample with the highest contribution, about 1 %, is A5S. Arey et al. (1988) has also found that INP contributes <1 % of the total TA98-S9 activity for airborne particulate samples from southern California.

A similar consideration can be applied to benzo(a)pyrene (BaP). Its response has been about 140 rev./µg in TA98+S9 in the present study and the average BaP concentration of 0.68 ng/m³ would contribute about 1 % to the average mutagenic activity of 9.5 rev./m³. The sample with the highest contribution, about 1.8 %, is T120 with 2.2 ng BaP/m³.

In view of the fact that a limited number of chemical compounds cannot describe the toxicological features of a complex mixture, biological tests as the Salmonella mutagenicity assay and the assay for the TCDD receptor binding as well as other tests can thus add to or supplant the information obtained through chemical analysis of selected organic compounds.

The present investigation and other studies have found indications that the composition of mutagenic components is not the same in airborne particulate matter and exhaust particles from major sources, gasoline and diesel exhaust. The observed difference suggests the formation of more polar mutagens or loss of non-polar mutagens during the residence time in

the atmosphere. Chemical transformations within the various PAC classes have been presented (Gibson et al. 1986, Ramdahl et al. 1986). Experimental studies with emission particles from wood burning have shown that the mutagenic response of the particle extracts is drastically changed after exposure to elevated concentrations of ozone and nitrogen dioxide (Kamens et al. 1984, 1985). Similar studies by Kleindienst et al. (1986) have corroborated that particle-associated mutagenic activity is changed by atmospheric reactions but have also focused on the formation of gas phase mutagenic compounds from photo-chemical reactions between unsaturated aliphatic hydrocarbons and nitrogen oxides. The latter phenomenon, which is not a part of the present study, has been further studied experimentally by Kleindienst et al. (1985), Victorin and Ståhlberg (1988, 1989) and Löfroth et al. (1990) and seems to require substantial further studies before it can be judged whether such mutagens are formed in ambient air and to what extent.

Source apportionment

Source apportionment using trace metals has been successfully applied to particulate mass (Kleinman et al. 1980). The increasing interest for organic pollutants revealed the difficulties to apportion such compounds to specific sources which have very similar emission characteristics (Daisey et al. 1986).

De Raat and de Meijere (1988) tried to discriminate between local and more distant sources of mutagenic activity by means of factor and multivariate analyses with sulfur dioxide, ozone and nitrogen oxides and concluded that a large part of the mutagenic response at the sample location in the Netherlands was due to long-range transport.

In a Danish study on the airborne mutagenicity in and around Copenhagen (Ostenfeldt 1989) K. Kemp attempted factor analysis and reported that the mutagenic activity ($TA_{98\pm S9}$) originated to about equal extent, 20-30 % each, from four sources, motor vehicles, local sources except motor vehicles, long-range transport and residual sources.

Using ^{14}C -analysis of soot carbon, Ramdahl et al. (1984) were able to show that, in a small Norwegian town, a major part of the mutagenic activity and PAH associated with airborne particulate matter originated from residential wood combustion.

Also focusing on wood smoke and in addition on motor vehicle exhaust, Lewis et al. (1988) and Stevens et al. (1990) were able to apportion the *Salmonella* mutagenic response from airborne particulate matter collected in two residential areas with heavy impact of wood smoke. The studies were performed, using MLR, with soil-corrected potassium as a tracer for wood smoke and lead for motor vehicle exhaust. The calculated apportionment was also consistent when compared with ^{14}C -analysis. The same group within the Integrated Air Cancer Project (IACP) at the U.S. EPA is presently trying to discriminate between diesel and gasoline exhaust emissions and oil combustion using organic derivatives of samarium isotopes added as tracers to diesel fuel and heating oil (R. Stevens, personal communication).

Using an organic derivative of dysprosium, Horvath et al. (1988) were able to show that diesel exhaust caused a substantial part of the airborne particulate matter. An average background of $11 \mu\text{g}/\text{m}^3$ of suspended particles was traced to diesel emissions in summertime Vienna.

The present study is the first attempt to apportion the mutagenic activity of diesel and gasoline exhaust in an urban area, where both sources are judged to be of major concern. The results show that the direct mutagenic activity (TA98-S9) from diesel exhaust is about twice that from gasoline exhaust. A more equal contribution is found for the indirect activity (TA98+S9). The results are in agreement with experimental studies, fuel usage and motor vehicle distribution. The study was performed during 1985-87 prior to the introduction of three-way catalysts for gasoline powered passenger cars which became optional in Sweden for the 1988 models and compulsory for the 1989 models. It is therefore likely that the present or future situation will increase the diesel part of the total mutagenic activity.

Risk considerations

Risks associated with air pollutants having potential carcinogenic effects have been the subject of deliberations for a long time. Prior to studies on the genotoxicity with in vitro tests of airborne particulate matter, Doll (1978) evaluated the potential hazard of then and earlier prevailing pollution levels and concluded that such atmospheric agents cannot be responsible for more than about five cases of lung cancer per 100,000 persons per year in European populations. A committee (Cederlöf et al. 1978) came to the same conclusion. The same order of magnitude is in agreement with the conclusion reached for a particular source, light duty diesel exhaust, using a number of in vitro tests and epidemiological data from exposure to coke oven and roofing tar fumes and cigarette smoke (Albert et al. 1983).

With the exception of tobacco smoking, there exist few human situations for which both exposure to combustion emissions and associated cancer statistics are available. Information from one such instance is, however, emerging and may become a checkpoint for the theoretical and experimental risk estimations. The study comprises populations with exposure to high indoor concentrations of emissions from different domestic fuels. These populations show significantly increased lung cancer rates, including women who rarely smoke tobacco (Mumford et al. 1987, Chapman et al. 1988). There may exist other populations that can be studied epidemiologically and that are exposed to high ambient or indoor concentrations of air pollutants from various combustion processes.

Grimmer et al. (1983, 1984, 1985 and 1987) have, in a series of studies on gasoline and diesel exhaust and coal combustion emissions, reported that the carcinogenic effect, as detected with topical application on mouse skin and lung implantation in the rat, mainly resides in a fraction containing PAH with more than three rings. The only comparison with Salmonella mutagenicity can be found in a report from the same group (Norpoth et al. 1985), in which the mutagenic response of a gasoline exhaust sample and its different fractions are given for the Salmonella strain TA100. The crude condensate gave a very small response both in the absence and presence of the complete mammalian activation system (S9)

amounting to <1,000 rev./km. The fraction containing 4-7 ring PAH had a response equivalent to about 20,000 rev./km in the presence of S9 and a much lower response without mammalian activation, <3,000 rev./km. None of the other fractions containing hydrophilic compounds, paraffinic compounds, 2-3 ring PAH and polar hydrophobic compounds gave a detectable response, i.e. <1,000 rev./km.

In view of the fact that it has been shown that exhaust particles from gasoline powered passenger cars have a mutagenic activity in the order of about 20,000 rev./km or more in TA100 (Pierson et al. 1983, Alsberg et al. 1985, Westerholm et al. 1988) and that additional activity is present with semi-volatile compounds, the very low response reported by Norpoth et al. may indicate that the sample handling or fractionation system used by Grimmer and co-workers destroy labile mutagenic components, as e.g. those that are mutagenic in the absence of S9. Until it can be shown that the scheme used by Grimmer and co-workers recover the known mutagenic response from representative samples, the suggestion by the same group that 4-7 ring PAH are almost entirely responsible for the carcinogenic effect must be treated with caution. If, however, their suggestion is valid, air pollution measurements and abatements with respect to carcinogenic risks can be performed with a limited number of indicator PAH leaving short-term bioassays to the history of environmental science.

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Appendix

Legend to Appendix Tables 1–3
Appendix Tables 1–7

Legend to Appendix Tables 1-3

All concentrations are given as indicated units per m^3 .
-, denotes not available or not measured.

SAMPLE

No., sample identification: series, number and location.
Date, sampling date, generally comprising two 24-h periods.

METEOROLOGY

T, temperature; wind, wind speed; dir., wind direction; averages over the sampling periods. CS, clear sky, % time with sunshine as recorded at a nearby air field.

MAIN STATION PARAMETERS

Dust, TSP; Soot; SO_2 , sulfur dioxide; NO_x , nitrogen oxides; NO_2 , nitrogen dioxide; O_3 , ozone; ²average concentrations during the sampling periods calculated from hourly recordings.

SAMPLE PARAMETERS

Part. & Particles Grav., gravimetric determination; Soot.

Mutagen. & Mutagenicity

Revertants in Salmonella TA98 and TA100 in the absence (-S9) and presence (+S9) of metabolic activation.

Toxicity

Concentration causing 50 % cell growth inhibition in ascites BP 8 cells; note that the response is expressed as an inverted value.

Affinity

Concentration causing 50 % inhibition of TCDD binding to the TCDD receptor; note that the response is expressed as an inverted value.

Polycyclic hydrocarbons

PAH, sum of selected polycyclic aromatic hydrocarbons: phenanthrene, anthracene, fluoranthene (Flu), pyrene, benzo(ghi)-fluoranthene, cyclopenta(cd)pyrene, benz(a)anthracene, chrysene/ triphenylene, benzo(b,j&k)fluoranthenes, benzo(e)pyrene (BeP), benzo(a)pyrene (BaP), indeno(cd)pyrene, benzo(ghi)perylene (BgP), coronene (Cor). The E series (Appendix Table 1) did not include coronene.

BNT, benzo(b)naphtho(2,1-d)thiophene.

NPy, 1-nitropyrene.

Elemental compounds

S, sulfur, Cl, chlorine; Ti, titanium; V, vanadium; Pb, lead.

Ionic species NO_3^- , nitrate; SO_4^{2-} , sulfate-

Hydrocarbons Bz, benzene; To, toluene; Xy, xylene.

Appendix Table 1. Summary table of E-series measurements on street (S) and rooftop (R) samples. All concentrations are given per m³.

SAMPLE	METEOROLOGY			MAIN STATION PARAMETERS					SAMPLE PARAMETERS														
									Part. Mutagenicity				Toxicity Affinity Polycyclic hydrocarbons				Elemental compounds						
									Grav.	-S9	+S9	-S9	+S9	PAH	Flu	BeP	BgP	S	Cl	Pb			
No.	Date	T	Wind	Soot	SO ₂	NO _x	NO ₂	O ₃	ug	ug	ug	ug	ug	ug	ug	ug	ug	ug	ug	ug	ug	ug	ug
		°C	m/s dir.	ug	ug	ug	ug	ug	ug	ug	TA98 Rev.	TA100 Rev.	ml ⁻¹	ml ⁻¹	ng	ng	ng	ng	ug	ug	ug	ug	ug
E 1S	840823	23	3	S	21	18	64	45	53	130	11	11	9.1	13	3.3	0.070	4.2	0.72	0.56	<0.10	-	-	-
1R	-24									120	14	13	12	13	3.6	0.12	4.3	0.88	0.72	<0.10	-	-	-
E 2S	840827	18	5	W-	8	4	-	-	57	85	5.7	4.4	4.1	4.2	9.4	-	2.4	0.38	0.33	<0.04	6.1	1.0	140
2R	-29			-WSW						56	3.2	2.3	2.3	3.3	8.9	-	1.3	0.20	0.23	<0.03	3.0	2.9	61
E 3S	840904	11	4	N	10	13	100	28	21	65	5.5	11	4.2	9.0	15	0.051	6.1	0.85	0.78	<0.07	3.1	5.7	230
3R	-06									46	4.9	8.7	4.1	7.3	13	0.024	4.9	0.55	0.73	<0.05	2.4	1.6	150
E 4S	840917	13	4	ENE-	6.4	7	27	18	27	63	7.1	8.8	5.9	6.7	10	-	-	-	-	-	3.5	1.4	160
4R	-19			-ESE						46	6.8	5.2	4.9	5.4	13	-	-	-	-	-	5.1	1.6	55
E 5S	841002	13	3	SSW-	12	10	58	24	16	98	13	15	16	17	4.1	0.006	-	-	-	-	6.0	2.0	340
5R	-05			-SSE						64	16	11	13	10	5.5	0.005	-	-	-	-	1.4	2.0	180
E 6S	841024	11	6	SSW-	5.8	6	-	-	28	63	15	22	10	20	4.9	-	-	-	-	-	4.2	3.8	210
6R	-26			-SW						45	12	15	9.4	14	8.7	-	-	-	-	-	1.8	3.9	100
E 7S	841107	8	3	SE-	17	5	42	23	10	94	18	27	12	34	2.2	0.013	-	-	-	-	2.8	0.44	400
7R	-09			-ESE						52	13	15	10	23	2.8	0.011	-	-	-	-	16	0.17	130
E 8S	841119	4	3	SE-	19	22	87	26	8	53	14	23	12	47	3.3	-	-	-	-	-	4.5	0.53	400
8R				-SSE						40	22	36	14	45	4.2	-	-	-	-	-	3.4	0.61	130
E 9S	841127	7	9	SSW	5.1	9	50	31	18	59	12	12	11	14	8.7	-	-	-	-	-	2.1	4.1	120
9R	-29									37	8.4	7.7	7.4	11	13	-	-	-	-	-	2.4	7.0	85

C 9W 870602	11	-	-	22	12	-	60	30	-	35	8.3	1.9	1.3	1.4	0.11	0.15	0.01	0.44	0.02	<1	4.2	2.0	69	28	30	3.5	4.7	1.8	10	23	
9W -04										33	8.3	1.7	1.3	2.0	0.08	0.17	0.28	0.46	0.01	<1	3.5	1.5	80	18	50	3.2	4.8	1.9	14	33	
D 18 860128	1	6	SSE	<1	11	16	40	18	26	31	-	18	14	12	1.2	1.4	0.88	0.64	0.29	6	-	-	-	-	-	-	-	-	-	-	
1N -30			-E							47	-	16	15	17	1.4	1.4	2.6	2.0	0.24	8	-	-	-	-	-	-	-	-	-	-	
A 18 860204	-6	6	ENE	20	14	10	89	46	31	70	26	7.7	5.6	12	1.5	0.68	1.2	1.1	0.09	5	1.9	4.8	360	140	560	4.5	6.1	0.7	14	16	
1N -07			-NE							75	32	12	11	24	2.9	1.2	3.2	2.4	0.11	1	1.8	3.7	510	110	730	1.6	3.9	4.7	32	32	
D 28 860219	-10	4	ENE	30	110	-	93	56	25	190	52	22	19	28	4.0	1.3	2.7	2.4	0.08	6	1.4	5.0	1000	80	750	2.8	4.3	8.8	22	27	
2N -21										110	42	30	26	30	4.0	1.8	3.5	3.2	0.09	15	1.4	1.8	700	120	1400	2.3	3.3	15	34	30	
D 30 860224	-7	2	E	26	170	-	310	83	17	290	44	35	39	57	4.6	4.7	4.6	0.22	0.53	-	1.6	4.3	2400	28	1800	5.7	3.7	40	94	83	
3N -26			-SE							190	-	27	28	30	2.4	2.4	3.4	4.6	0.16	15	1.5	3.0	1900	38	1200	1.9	4.4	26	66	60	
D 4S 860226	-2	3	SSW	33	80	-	310	82	26	290	190	21	32	64	6.8	3.5	7.5	6.2	0.57	48	1.6	16	1600	86	1900	18	4.1	50	140	140	
4N -28			-E							100	43	23	21	38	1.8	2.2	6.2	6.0	0.10	35	1.3	4.8	800	160	1600	4.5	3.0	25	82	94	
D 50 860609	17	7	S	49	91	3	27	24	110	97	40	6.2	3.8	5.9	0.38	0.76	0.89	0.26	<0.01	6	7.9	0.09	96	23	750	8.6	17	13	40	41	
5N -11										130	15	7.9	4.1	8.1	0.51	0.87	1.1	1.3	0.01	14	2.2	0.09	74	17	410	3.2	7.0	17	46	46	
D 6S 860616	25	3	SW	59	-	3	39	30	44	130	60	6.6	5.1	9.0	0.39	1.3	0.99	0.09	0.02	30	2.3	0.22	170	60	900	2.9	6.8	13	47	46	
6N -18			-SSW							91	13	4.4	3.6	7.8	0.28	0.72	1.7	0.32	0.01	14	2.1	0	23	880	160	580	3.0	6.5	11	34	37
D 78 860714	19	4	SSW	40	30	3	24	20	47	38	14	1.1	1.4	1.0	0.05	0.12	0.16	0.13	0.01	3	11	12	130	43	49	3.2	5.4	8.9	26	36	
7N -16										63	20	2.0	2.0	2.8	0.06	0.29	0.65	0.54	0.01	3	6.5	6.8	180	33	250	2.4	4.6	6.7	32	32	
A 38 861027	10	7	S	4	24	3	44	28	-	29	6.6	3.8	2.4	2.5	0.17	0.22	0.42	0.27	0.03	1	6.2	27	37	25	42	2.2	1.8	3.8	19	27	
3N -31			-SW							33	7.3	4.2	3.9	3.8	0.22	0.30	0.86	0.76	0.04	2	4.4	13	51	14	110	2.5	1.8	4.2	27	37	
D 8S 861216	3	5	W	0	7.3	7	100	45	5	9 ¹	68	11	15	36	2.2	1.7	3.8	5.0	0.13	25	4.2	1.3	270	50	450	1.8	2.6	23	56	55	
8N -18			-SSE							38	20	6.8	8.3	17	1.2	1.1	2.4	2.4	0.09	11	4.8	1.7	83	27	370	2.8	4.4	15	39	33	
D 98 870114	-6	4	NE	0	16	16	65	43	18	33	21	7.5	9.7	13	1.4	0.92	1.4	0.95	0.09	18	5.2	2.3	75	25	140	1.8	6.6	5.9	20	40	
9N -16			-E							35	32	8.3	12	16	1.6	1.6	1.9	1.2	0.11	18	8.0	1.1	99	37	760	1.9	7.0	17	51	53	
A 5S 870406	5	3	-	11	61	-	58	44	-	350	130	13	13	41	5.1	2.6	4.2	3.6	0.44	68	13	9.4	1900	20	690	6.5	8.4	22	54	59	
5N -8+13-15										120	35	5.9	6.4	9.6	1.1	0.56	1.3	1.1	0.17	9	6.7	5.0	540	47	250	5.7	7.3	8.9	23	32	

T 98 861126	9 6	SM-	8	-	12	61	40	-	41	7.6	3.6	4.5	3.3	0.26	0.28	0.40	0.32	0.04	4	8.0	36	68	34	50	4.4	3.4	5.2	21	18
9N	-28	-SSW							36	5.4	3.1	4.0	2.4	0.16	0.22	0.26	0.20	0.04	3	9.0	43	49	50	21	5.9	4.3	2.3	13	12
T100 861210	5 3	S	7	36	12	210	61	-	120	70	14	16	35	1.9	2.0	4.4	3.9	0.30	10	6.5	4.6	950	110	930	4.1	4.4	30	75	75
10K	-12								120	43	13	15	33	1.5	1.8	4.8	4.0	0.12	13	4.8	6.2	680	74	380	4.9	4.9	24	62	62
T11P 870126	-6 4	SM-	8	71	-	140	62	14	40	21	8.0	10	15	1.2	1.2	2.0	1.6	0.09	16	3.7	12	160	40	220	0.92	1.3	11	36	36
11V	-28	-S							22	10	5.7	4.7	6.1	0.40	0.39	0.94	0.57	0.07	5	3.6	7.3	72	31	130	1.2	1.3	6.3	20	19
T120 870304	-4 4	ESE	27	150	10	81	56	20	280	57	16	17	56	7.1	1.7	5.1	4.1	0.53	36	5.1	3.0	780	50	330	4.9	7.4	16	43	43
12K	-06								200	55	12	15	32	4.3	1.4	3.0	2.7	0.26	32	4.3	2.7	990	50	280	4.6	9.8	12	32	33
T138 870401	-	-	-	18	-	-	-	-	120	45	6.4	6.6	16	1.6	1.1	1.5	1.8	0.14	18	7.9	4.6	330	50	140	4.3	9.7	8.1	24	30
13N	-03								65	22	4.0	4.3	7.4	0.96	0.62	0.59	0.64	0.06	21	7.8	2.0	220	57	77	4.5	9.6	5.0	15	18
T14P 870506	10 4	-	51	57	-	99	63	-	54	14	3.6	3.8	5.8	0.24	0.41	1.1	0.91	0.03	8	2.0	2.3	98	6.8	84	1.3	0.77	9.2	32	38
14V	-08								39	6.2	2.8	2.5	3.1	0.20	0.40	0.47	0.43	0.01	2	2.0	3.3	92	7.7	40	1.4	0.71	8.3	34	38
T150 870604	15 -	-	11	-	-	34	26	-	71	-	7.9	7.7	10	0.24	0.85	1.9	2.8	0.04	10	7.3	0.66	190	47	300	3.6	8.0	16	45	53
15K	-06								75	17	4.7	4.3	4.7	0.17	0.39	0.93	0.87	0.03	<1	6.0	0.78	150	28	97	4.7	7.7	12	33	40
B 1V 870302	-10 3	SM-	33	150	17	170	62	16	230	57	16	22	79	18	3.7	1.7	1.9	1.2	32	3.8	4.3	520	21	220	2.2	4.7	13	39	38
1C	-04	-S							85	37	10	9.4	24	4.4	1.2	0.60	1.7	0.27	20	4.7	4.0	500	42	190	2.8	5.6	7.4	22	25
B 2V 870310	-1 -	-	27	70	39	220	86	8	200	88	16	21	51	8.7	2.4	5.0	0.71	0.64	29	6.9	4.9	850	85	700	5.8	7.3	17	45	43
2C	-12								120	59	13	15	25	3.3	1.6	2.1	1.7	0.26	24	6.2	3.7	570	58	300	5.1	6.0	11	29	34
B 3V 870325	-	-	2	-	-	-	-	-	67	19	8.5	7.5	11	1.2	0.90	1.4	0.27	0.27	5.6	4.5	1.2	130	30	270	6.4	9.5	8.3	24	26
3C	-27								42	16	4.6	3.9	8.3	1.3	0.53	0.54	0.65	0.13	5.7	3.8	0.91	54	32	82	4.7	7.0	4.9	15	19
B 4V 870327	-	-	<1	-	-	-	-	-	40	14	5.4	4.8	8.8	0.76	1.0	1.6	1.1	0.06	11	5.6	22	110	35	200	3.1	3.5	6.3	51	80
4C	-29								28	2.7	2.9	2.1	2.9	0.30	0.21	0.45	0.06	0.06	2.5	4.8	17	23	19	33	3.0	3.2	4.2	13	15
B 5V 870329	-	-	48	-	-	-	-	-	170	55	11	17	38	3.9	3.0	5.6	3.1	0.78	9.1	5.9	7.9	600	50	490	2.5	3.2	17	46	44
5C	-31								51	29	5.9	6.0	8.0	0.58	0.76	0.74	1.9	0.10	7.8	5.6	6.0	540	62	260	2.3	2.3	9.1	29	33

Appendix Table 4. Detailed PAC analysis of fractionated A samples.

PAC	A 1B	A 1N	A 2P	A 2V	A 3B	A 3N	A 4M	A 4V	A 5S	A 5N
PAH, ng/m ³										
Phenanthrene	0.54	0.88	0.11	0.10	0.05	0.07	4.4	3.2	1.5	0.25
Anthracene	0.03	0.09	0.01	0.01	0.01	0.01	0.21	0.11	0.33	0.04
3-Methylphenanthrene	0.23	0.37	0.03	0.01	0.01	0.02	0.48	0.39	0.67	0.09
2-Methylphenanthrene	0.28	0.47	0.04	0.02	0.01	0.02	0.67	0.55	0.70	0.12
1-Methylanthracene	0.18	0.21	0.07	0.01	0.01	0.01	0.27	0.31	0.50	0.07
1-Methylphenanthrene	0.18	0.27	0.02	0.01	0.01	0.01	0.43	0.48	0.50	0.06
3,6-Methylphenanthrene	0.26	0.39	0.02	0.01	0.01	0.03	0.15	0.12	0.73	0.10
Fluoranthene	1.5	2.9	0.35	0.29	0.17	0.22	6.7	5.0	5.1	1.1
Pyrene	1.7	3.7	0.33	0.29	0.12	0.15	4.2	3.4	6.1	1.2
Benzo(a)fluorene	0.33	0.74	0.02	0.01	0.02	0.03	0.41	0.39	0.72	0.14
Benzo(b)fluorene	0.25	0.58	0.01	0.03	0.01	0.03	0.39	0.26	0.72	0.15
1-Methylpyrene	0.22	0.49	0.01	0.03	0.02	0.02	0.19	0.16	0.57	0.08
Retene	0.13	0.14	0.01	0.04	0.01	0.01	0.08	0.08	0.14	0.03
Benzo(ghi)fluoranthene	0.86	1.9	0.22	0.19	0.11	0.13	1.8	1.9	2.7	0.73
Cyclopenta(cd)pyrene	0.22	0.54	0.14	0.16	0.02	0.04	0.82	0.53	1.3	0.24
Benz(a)anthracene	0.53	1.0	0.22	0.18	0.08	0.09	1.5	1.2	2.8	0.56
Chrysene/Triphenylene	1.0	2.0	0.65	0.43	0.21	0.19	3.3	2.8	2.0	0.52
Benzo(b,k&j)fluoranthene	1.5	2.3	1.0	0.79	0.45	0.48	5.8	4.9	4.4	1.0
Benzo(e)pyrene	0.68	1.2	0.47	0.33	0.22	0.30	1.8	2.2	2.6	0.56
Benzo(a)pyrene	0.47	0.78	0.24	0.18	0.12	0.17	1.6	1.6	1.6	0.31
Indeno(cd)fluoranthene	0.14	0.29	0.12	0.10	0.07	0.12	1.3	0.84	0.48	0.13
Indeno(cd)pyrene	0.60	1.4	0.52	0.38	0.23	0.35	2.7	1.9	2.9	0.68
Benzo(ghi)perylene	1.2	3.2	0.98	0.78	0.42	0.86	4.8	2.1	4.2	1.3
Anthanthrene	0.02	0.11	0.01	0.03	0.02	0.03	0.63	0.39	0.29	0.06
Coronene	1.1	2.4	0.81	0.56	0.27	0.76	2.3	1.7	3.6	1.1
Sum of PAH	14	28	6.4	5.0	2.7	4.2	47	37	47	11
S-PAH, ng/m ³										
Dibenzothiophene	0.02	0.02	- ^a	-	0.01	0.01	0.49	0.26	0.04	0.01
Benzonaphthothiophene	0.09	0.11	0.09	0.01	0.03	0.04	0.33	0.29	0.44	0.17
NO ₂ -PAH, pg/m ³										
1-Nitropyrene	5	1	7	6	1	2	36	11	68	9

a) - not analyzed.

Appendix Table 5. The mutagenic response of the fractionated A samples expressed as TA98 revertants per m³ air.

Sample	A 1B		A 1N		A 2P		A 2V		A 3B		A 3N		A 4M		A 4V		A 5S		A 5N	
	-S9	+S9	-S9	+S9	-S9	+S9	-S9	+S9	-S9	+S9	-S9	+S9	-S9	+S9	-S9	+S9	-S9	+S9	-S9	+S9
Crude extract	7.7	5.6	12	10	6.1	4.1	3.1	3.2	3.8	2.4	4.2	3.9	47	34	38	37	13	13	5.9	6.4
Fraction I	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Fraction II	0.29	0.47	0.81	1.0	0.30	0.61	0.23	0.66	0.25	0.25	0.25	0.50	3.4	5.5	3.6	5.2	0.79	1.3	0.21	0.77
Fraction III	1.1	0.85	2.1	1.4	<0.2	0.24	0.32	0.23	0.52	0.32	0.50	0.44	4.9	3.8	4.6	3.5	1.7	2.0	0.53	0.59
Fraction IV	2.4	1.4	4.4	1.9	1.3	0.86	1.1	0.75	1.8	0.54	1.8	0.88	13	9.1	12	8.6	3.3	2.8	1.6	1.0
Fraction V	2.3	1.7	3.9	3.0	1.4	1.6	1.2	1.3	1.4	0.88	1.7	1.4	22	16	19	12	3.3	4.9	1.9	2.1
Reconstituted sample of fraction I-V	4.8	4.1	9.2	8.3	2.5	2.7	2.0	3.2	- ^a	-	-	-	34	37	28	28	7.0	13	3.2	5.0
(% of crude)	(62)	(75)	(77)	(83)	(41)	(66)	(65)	(100)	-	-	-	-	(72)	(109)	(74)	(76)	(54)	(100)	(54)	(78)
Sum of fraction I-V	6.1	4.4	11	7.3	3.0	3.3	2.9	2.9	4.0	2.0	4.3	3.2	43	34	39	29	9.1	11	4.2	4.5
(% of crude) ^b	(79)	(79)	(93)	(73)	(49)	(80)	(94)	(90)	(105)	(83)	(102)	(82)	(91)	(100)	(103)	(78)	(70)	(85)	(71)	(70)

a) - not tested; b) average +SE is 86+6 for -S9 and 82+3 for +S9.

Appendix Table 6. Comparison between the microsuspension and plate incorporation with the Salmonella mutagenicity assay for urban air particulate extracts.

SAMPLE No.	PARTICLES ug/m ³	TA98 REVERTANTS PER MG PARTICLES (+SE)				RATIO		SAMPLE No.	PARTICLES ug/m ³	TA98 REVERTANTS PER MG PARTICLES (+SE)				RATIO	
		Microsuspension		Plate incorporation		Micro/Plate				Microsuspension		Plate incorporation		Micro/Plate	
		-S9	+S9	-S9	+S9	-S9	+S9			-S9	+S9	-S9	+S9	-S9	+S9
B 1V	231.8	381+11	763+27	70+4	94+4	5.44	8.13	A 1B	69.9	462+13	744+33	111+6	80+5	4.17	9.30
1G	85.2	729+24	1380+33	116+6	110+7	6.28	12.51	1N	75.0	784+21	922+30	164+9	139+9	4.77	6.64
B 2V	196.3	348+11	838+37	82+7	106+8	4.22	7.92	A 2P	93.1	319+15	448+21	66+3	44+3	4.87	10.11
2G	123.9	550+14	1460+47	103+6	124+7	5.33	11.78	2V	48.7	298+18	708+34	64+3	65+3	4.63	10.91
B 3V	67.4	451+17	1058+26	126+6	112+4	3.58	9.49	A 3B	28.7	409+19	826+38	132+5	84+6	3.10	9.80
3G	42.3	330+13	1324+74	109+5	93+7	3.02	14.27	3N	32.7	513+47	1119+47	129+10	118+7	3.99	9.48
B 4V	40.2	518+16	692+35	130+7	120+4	3.99	5.79	A 4M	109.4	804+36	1820+100	427+17	313+8	1.88	5.81
4G	27.8	333+35	610+32	104+5	76+3	3.20	8.02	4V	86.2	1214+61	2881+131	442+17	431+13	2.75	6.68
B 5V	174.0	328+13	533+17	64+3	100+3	5.09	5.34	A 5S	348.5	244+29	351+27	36+2	39+2	6.74	9.07
5G	27.8	498+13	873+35	114+5	117+6	4.36	7.49	5N	116.7	281+22	451+25	51+3	55+3	5.55	8.20
C 1M	31.5	405+31	453+34	187+7	107+5	2.16	4.24	T 1P	57.8	417+6	722+38	189+8	237+11	2.21	3.05
1V	30.5	243+14	528+30	128+5	110+7	1.90	4.80	1V	27.7	220+16	426+27	165+7	180+9	1.33	2.37
C 2M	31.9	475+30	457+33	125+7	123+6	3.80	3.72	T 2P	24.5	1211+44	1702+54	541+20	480+21	2.24	3.55
2V	28.6	691+40	743+24	152+7	175+6	4.55	4.25	2V	16.6	1385+102	1780+112	511+23	475+26	2.71	3.75
C 3M	196.8	376+25	701+21	173+12	148+8	2.17	4.74	T 3B	40.5	1016+58	1908+93	377+10	428+17	2.69	4.46
3V	163.0	529+24	987+55	206+10	180+4	2.57	5.47	3N	33.2	1648+52	2370+108	566+18	581+41	2.96	4.08
C 4M	31.0	368+26	633+45	146+5	122+6	2.52	5.18	T 4O	56.5	1044+51	756+26	316+12	232+13	3.30	3.26
4V	21.2	371+32	486+43	140+7	93+8	2.65	5.23	4K	26.2	970+82	573+73	510+14	293+23	1.90	1.95
C 5V	26.9	196+10	180+13	44+2	33+3	4.47	5.52	T 5B	54.5	422+27	732+58	73+3	47+4	5.78	15.71
5V	20.1	215+9	266+13	76+5	58+4	2.81	4.62	5N	36.7	416+24	707+54	80+4	49+3	5.20	14.37

Appendix Table 7. Factor assignments to sources and components with high and moderate loadings obtained with factor analysis of 66 samples in the C, D and T series. ^a

Assignment	Factor Loading	1	2	3	4	5	6	7
Gasoline exhaust								
Diesel exhaust								
Photochemical activity								
Long-range transport								
Heating, oil combustion								
Road dust								
Sea salt								
High	Soot		SO ₂	O ₃	-	S	V	-
	Ti							
	Pb							
	Bz							
	To							
	Xy							
	BeP							
	BaP							
Moderate	-S9	-S9	T	Ant	T	Cor	Cl	
	+S9	+S9	CS	SO ₄ ²⁻	SO ₂			
	NO _x	Grav.		NO ₃				
	NO ₂	DBT						
	PAH _{2b}	SO ₄ ²⁻						
		Flu						
		Phe						

a) abbreviation are those used in Appendix Tables 1-3 (see Legend) and in addition benzo(a)pyrene (BaP), anthracene (Ant), phenanthrene (Phe) and dibenzothiophene (DBT);

b) all PAH except BaP and BeP having high loadings and Phe.

This report presents an investigation performed in Göteborg and concerns the contribution from different sources to certain chemical components in and mutagenicity of ambient particulate matter. A source receptor analysis has been performed using a chemical mass balance method and multiple regression analysis.

The investigation shows the importance of traffic for the occurrence of particulate mutagenic substances in ambient air (about 80 %) and that diesel cars, which is only 10 % of total traffic, contribute more than petrol fueled cars.