

Levels of benzene and other volatile aromatic compounds in the blood of non-smokers and smokers

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Summary. The concentrations of benzene, toluene, ethylbenzene, and o-, m- and p-xylene were measured in venous blood samples collected from 13 non-smokers and 14 cigarette smokers. The blood samples were analysed by a purge and trap technique followed by gas chromatography/mass spectrometry/computer analysis. The above-mentioned volatile organic compounds (VOC) could be detected in measurable amounts in all blood samples. This finding seems to reflect the ubiquitous exposure of humans to these agents in the urban environment, in non-smokers as well as in smokers. Smokers were found to have significantly higher blood concentrations of benzene (median 547 ng/l) and toluene (median 2201 ng/l) than non-smokers (median 190 ng/l and 1141 ng/l, respectively). The concentrations of ethylbenzene and xylenes also tended to be higher in smokers when compared to non-smokers. The different concentrations of these compounds in the blood of non-smokers appear to reflect the common concentration pattern found in outdoor urban air as well as in indoor air and also seem to be influenced by the different blood/air partition coefficients of these compounds. The results indicate that smoking is associated with a significant additional exposure to VOC, in particular to benzene and toluene.

Key words: Benzene – Toluene – Volatile organic compounds

Introduction

It is well known that urban air contains a large number of volatile organic compounds (VOC) including aroma-

tic hydrocarbons such as benzene, toluene, ethylbenzene, and o-, m- and p-xylene (Bruckmann et al. 1983, 1988; Wallace et al. 1985, 1986; Fishbein 1988a, b, c; LIS 1988). The composition of aromatic pollutants in air is generally considered to be similar to that of gasoline, suggesting the latter as the major source. Due to the widespread occurrence of toluene, ethylbenzene, and mixed xylenes in a large variety of building materials and consumer products (Wallace et al. 1987) indoor air concentrations of these compounds are frequently found at levels exceeding those outdoors (Wallace et al. 1985, 1986; Seifert et al. 1986; Hartwell et al. 1987; Krause et al. 1987). Benzene is no longer used as an intentional ingredient in building materials and consumer products and it is believed that the contaminant levels remaining are unlikely to result in significant consumer exposure to benzene vapour. Nevertheless, indoor air concentrations of benzene are also frequently found at higher levels than outdoors, particularly in homes with smokers, suggesting that smoking is a significant source of exposure (Wallace et al. 1986, 1987; Krause et al. 1987).

In the general population, the respiratory route is considered as the major source of exposure to benzene and other VOC. Benzene has also been reported to occur in food and beverages and it has been estimated that the dietary intake of benzene may be as high 100 to 250 µg/d (WHO-Euro 1987; Fishbein 1988a).

Significant additional exposure to VOC results from tobacco smoking. It has been estimated that a smoker inhales 20 to 80 µg of benzene, 80 to 160 µg of toluene, about 10 µg of ethylbenzene and about 10 to 30 µg of the xylenes per cigarette (Elmenhorst and Schultz 1968; Berlin et al. 1980; IARC 1986; Wallace and Pelizzari 1986; WHO-Euro 1987). It can be expected, therefore, that cigarette smokers are significantly more exposed to

these agents than non-smokers. Applying highly sensitive analytical techniques Berlin et al. (1980), Wallace and Pelizzari (1986), and Hartwell et al. (1987) could demonstrate that exhaled breath of smokers contains significantly higher levels of benzene, ethylbenzene, and xylenes than exhaled breath of non-smokers. Perbellini et al. (1988) showed that smokers have significantly higher benzene concentrations in alveolar air and blood than non-smokers.

Passive smokers may also be exposed to increased levels of aromatic hydrocarbons since sidestream smoke contains these compounds in even greater amounts than mainstream smoke (IARC 1986). Homes of smokers were found to contain higher benzene concentrations in air than homes with no smokers (Wallace and Pelizzari 1986; Krause et al. 1987), and the daily benzene intake of passive smokers may be about 50% higher than that of unexposed non-smokers (Wallace and Pelizzari 1986).

In the present study we determined the concentrations of six volatile aromatic hydrocarbons (benzene, toluene, ethylbenzene, o-, m- and p-xylene) in whole blood samples of some non-smokers and smokers without known occupational exposures to these agents. The objective of this study was to establish baseline concentration data for these compounds in human blood and to evaluate the effect of cigarette smoking, which could be a significant confounding variable when assessing exposures to these agents at low environmental levels.

Materials and methods

Subjects. Blood samples were taken by venipuncture from 27 subjects without any known occupational or hobby-related exposure to volatile organic hydrocarbons. The subjects were living in a large urban agglomeration in the Federal Republic of Germany (Düsseldorf and surrounding areas). The subjects were divided according to their smoking habits into non-smokers ($n = 13$) and smokers ($n = 14$).

Blood sampling. Twenty millilitres of venous blood were collected from each subject using syringes and canules, which previously had been examined to be free of contamination by the target compounds mentioned above. The blood was immediately transferred into a glass vial containing 400 µl of an aqueous EDTA- K_2 solution (8.75%). The glass vials as well as all other glass tubes used for blood analyses were kept in an oven constantly maintained at 100°C. The water used for the preparation of the EDTA solution previously was purged from traces of organics by UV irradiation. The blood samples were taken in the morning hours (usually between 9.00 and 11.00 h) at least 30 min after smoking the last cigarette.

Blood analysis. The blood samples were analysed within 24 h by a procedure similar to that described by Antoine et al. (1986). The volatile organic compounds (VOC) were purged from the blood by a stream of helium and collected on a Tenax/Silicagel adsorption tube. After thermal desorption and subse-

quent cryofocussing the VOC were analysed by capillary gas chromatography/mass spectrometry/computer analysis (GC/MS/COMP). Briefly, the analytical procedure was as follows:

A 2-ml sample of whole blood containing EDTA as an anticoagulant was transferred by a glass pipette to a 20-ml purging vessel containing 50 µl Antifoam B Emulsion (Sigma Chemicals Co.; 1% in aqueous solution). The blood sample was then purged for 15 min with helium (quality grade 5; Messer-Griesheim, FRG) at a flow rate of 30 ml/min. The VOC were transferred by this procedure to a Tenax/Silicagel adsorption tube (0.5 × 8 cm glass column packed with 2/3 Tenax TA (60/80 mesh; Macharey & Nagel, Düren, FRG) and 1/3 silicagel (60/80 mesh, Alltech, Unterhaing, FRG). During this time the purging vessel was placed in a warm water bath maintained at 40°C. The adsorption tube was arranged so that the He/VOC stream first passed the Silicagel zone and then the Tenax zone. In order to reduce the water content of the He/VOC stream a cold trap maintained exactly at -15°C was placed between the purging vessel and the Tenax/Silicagel adsorption tube. The adsorbed VOC were then thermally desorbed at 250°C and separated by capillary gas chromatography (Carlo Erba, HRGC 5300, Mega Series) using an ion trap detector (Finnegan, ITD 700) for peak identification and quantitation. In detail, the desorption procedure and GC/MS analysis was carried out as follows: The Tenax/Silicagel column is placed into the thermo-desorption unit (Carlo Erba, TDAS 5000 Thermal Desorption Auto Sampler) and purged with helium for 30 s at room temperature (purge). The helium gas stream is then stopped and the column heated to 250°C (pre-heating). After 180 s the valves are opened again and the desorbed VOC are transferred with the helium stream to a cryotrap (Carlo Erba, Cold Capillary Trap) maintained at -120°C by liquid nitrogen for the duration of the desorption period. The interface transfer line used consists of a fused silica capillary maintained at 60°C. The cryotrap is rapidly heated then to 250°C and the VOC are separated on a 30 m × 0.25 mm fused-silica DB-5 column (J & W Scientific; film thickness 0.25 µm; carrier gas: helium, 0.9 bar). Simultaneously, the GC temperature programme was started, which was as follows: 60°C isotherm for 1 min after the cryotrap has reached 250°C; 60 to 150°C at 10°C/min; 150°C isotherm for 3 min. The ITD scan mode usually was: full scan, 50 to 110 µ; 1 scan/s.

After each analytical run the Tenax/Silicagel column was purged with helium (20 ml/min) at 250°C for 15 min (cleaning) and then kept sealed until the next analytical run. The blanks of the Tenax/Silicagel columns were controlled routinely before each analytical series.

Calibration was obtained as follows: A whole blood sample collected from a non-smoker was purged several times as described above until constant, very low levels of the target compounds were obtained. The counts obtained from this blood sample ("zero blood" because it does not contain measurable amounts of VOC) were considered the "blanks" of the system and were used to determine the lower detection limit for the target compounds. Samples of "zero blood" were then spiked with known amounts of the target compounds. The counts obtained for these spiked blood samples were used to calculate the calibration curve.

The detection limits in whole blood were: benzene, 21 ng/l; toluene, 80 ng/l; ethylbenzene, 28 ng/l; m-/p-xylene, 152 ng/l; o-xylene, 50 ng/l. m- and p-xylene were coeluted with the same retention time. Therefore, only the sum of m- and p-xylene concentrations could be measured.

The reproducibility of the procedure was tested by replicate analyses of blood samples with different levels of the target compounds (blood from smokers and non-smokers). The coefficients of variation usually were in the range of 3 to 10%, a gen-

Table 1. Concentrations of volatile aromatic hydrocarbons (ng/l) in blood of non-smokers and smokers

Compound	Non-smokers (<i>n</i> = 13)						Smokers (<i>n</i> = 14)					
	Md	Mn	SD	GM	GSD	Range	Md	Mn	SD	GM	GSD	Range
Benzene	190	218	96	202	1.5	112–455	493*	547	195	517	1.4	287–947
Toluene	1141	1630	1371	1284	2.0	495–4614	2001**	2133	771	2019	1.4	1316–3804
Ethylbenzene	431	651	633	460	2.3	175–2284	533	837	640	695	1.8	378–2697
m-/p-xylene	1094	1580	1364	1261	1.9	548–5602	1490	1705	663	1597	1.4	916–3008
o-xylene	324	409	375	316	2.0	129–1472	352	463	262	412	1.6	236–1130

Abbreviations: MD, median; Mn, mean; SD, standard deviation; GM, geometric mean; GSD, geometric standard deviation

* Significantly different from non-smokers ($Z = 4.05$; $P < 0.0001$)

** Significantly different from non-smokers ($Z = 2.40$; $P < 0.05$)

Table 2. Results of linear regression between the concentrations of various volatile aromatic hydrocarbons in blood of non-smokers and smokers^a

Group	<i>n</i>	Parameters correlated		<i>r</i>	<i>P</i>	Slope	Intercept
Total	27	log B	vs log T	0.46	< 0.05	0.505	0.896
		log B	vs log EB	0.44	< 0.05	0.365	1.512
		log B	vs log mpX	0.45	< 0.05	0.521	0.872
		log B	vs log oX	0.41	< 0.05	0.421	1.439
		log T	vs log EB	0.49	< 0.05	0.377	2.173
		log T	vs log oX	0.55	< 0.01	0.421	1.439
		log EB	vs log mpX	0.86	< 0.001	1.205	1.863
		log EB	vs log oX	0.95	< 0.001	1.174	−0.249
		log mpX	vs log oX	0.89	< 0.001	0.786	1.143
Non-smokers	13	log B	vs log EB	0.65	< 0.05	0.314	1.468
		log B	vs log mpX	0.77	< 0.01	0.482	0.809
		log B	vs log oX	0.72	< 0.01	0.421	1.252
		log EB	vs log mpX	0.89	< 0.001	1.147	−0.894
		log EB	vs log oX	0.95	< 0.001	1.148	−0.207
		log mpX	vs log oX	0.91	< 0.001	0.856	0.960
Smokers	14	log T	vs log EB	0.56	< 0.05	0.325	+2.381
		log EB	vs log mpX	0.79	< 0.01	1.253	−1.172
		log EB	vs log oX	0.95	< 0.001	1.162	−0.197
		log mpX	vs log oX	0.81	< 0.01	0.633	+1.549

Abbreviations: B = benzene; T = toluene; EB = ethylbenzene; mpX = m- and p-xylene; oX = o-xylene

^a Data are given only for correlations with *r* significantly different from zero

erally considered acceptable level. The recovery rates were found to be on average; benzene, 112%; toluene 119%; ethylbenzene, 91%; m-/p-xylene, 89%; o-xylene, 98%.

Results

All blood samples that were analysed in this study were found to contain measurable quantities of benzene, toluene, ethylbenzene, and xylenes. The mean and median concentrations and concentration ranges are presented in Table 1. The highest concentrations were generally found for toluene, followed by m-/p-xylene, ethylbenzene, o-xylene and benzene. Smokers have significantly higher concentrations of ben-

zene and toluene in blood than non-smokers. Other volatile aromatic compounds, such as ethylbenzene and xylenes, also tended to occur in higher concentrations in the blood of smokers when compared to non-smokers (differences not significant). As shown in Table 2 there are a number of significant correlations between the blood levels of the VOC analysed in this study.

Discussion

The benzene concentrations found in this study are similar to those reported by Perbellini et al. (1988) for non-smokers and smokers without occupational

Table 3. Blood/air partition coefficients of some volatile aromatic hydrocarbons

Compound	Reference		
	(1)	(2)	(3)
Benzene	6.5	7.8	6.4
Toluene	15.6	15.6	10.0
Ethylbenzene	—	28.4	
o-xylene		31.1	
m-xylene	42.1	26.4	
p-xylene		37.6	

References: (1) Sherwood (1976)

(2) Sato and Nakajima (1979)

(3) Fiserova-Bergerova and Diaz (1986)

benzene exposure. The median values found by these authors were 299 and 465 ng/l, and the geometric mean values were 303 and 442 ng/l. Antoine et al. (1986) analysed blood samples from 250 patients from the New Orleans area (USA). The mean concentrations of benzene, toluene, ethylbenzene, and xylenes reported by these authors were 0.8 µg/l, 1.5 µg/l, 1.0 µg/l and 5.2 µg/l, respectively. Apart from toluene, these mean values are higher than those found in our study. Significantly higher toluene levels in environmentally exposed subjects (average 53 µg/l; $n = 30$) were reported in an earlier study by Szadkowski et al. (1973), but it may be difficult to compare these results due to shortcomings in sampling and analysis. Apostoli et al. (1982) were not able to detect toluene in the blood of occupationally non-exposed subjects.

Since it is not known that benzene, toluene, ethylbenzene, and xylenes occur in human blood as a result of the normal physiological metabolism, the finding of measurable concentrations of these compounds in blood should reflect human exposure to these agents, mainly by inhalation and possibly also by food. It is well known that the above named aromatic compounds are ubiquitously found in urban air with indoor concentrations frequently exceeding those outdoors. The concentration pattern in blood as found in this study seems to parallel largely the concentration pattern found in indoor and outdoor air. The highest air concentrations are generally found for toluene, which consistently occurs at higher concentrations in blood than the other aromatic compounds. The air concentrations of m-/p-xylene are usually much lower than that of toluene. However, due to the higher blood/air partition coefficient (Table 3), which decreases elimination by exhalation, the blood concentrations are relatively high and similar to that of toluene. The average air concentrations of ethylbenzene and o-xylene are frequently similar to that of benzene but due to the much higher blood/air partition coefficients their con-

centrations in blood are higher than that of benzene. In addition to the different rates of elimination via exhalation, the different rates of metabolic conversion must also be taken into account.

It is interesting to note that there are a number of significant correlations between the blood concentrations of different VOC analysed in this study (Table 2). This finding indicates a relatively uniform inhalation exposure pattern originating from a common source. In fact, it is well known that automobile exhausts represent the main source of VOC in urban air. The degree of human exposure depends on the local VOC concentrations in air (which is related to traffic density) as well as on individual circumstances such as indoor air contaminants at home or at the working place and smoking habits. The large variation of the individual blood concentrations seems to reflect these rather variable exposure conditions.

The finding that smokers have higher concentrations of benzene, toluene, ethylbenzene, and xylenes in blood than non-smokers may be explained by the well known fact that cigarette smoke contains significant amounts of these compounds (Elmenhorst and Schultz 1968; Berlin 1985; IARC 1986). It is interesting to note that the concentration differences in blood are most pronounced for benzene and toluene whereas the concentrations of the other compounds were only slightly increased in smokers compared to non-smokers. In smokers the benzene concentrations are increased, on average, by about 300 ng/l and the toluene concentrations by about 800 ng/l. This relationship is plausible if one considers the fact that mainstream smoke contains higher amounts of toluene compared to benzene and also considering the higher blood/air partition coefficient of toluene when compared to benzene (see Table 3). The average amount of ethylbenzene and xylene isomers inhaled per cigarette has been estimated to be about 5 to 20 µg/cigarette, which is significantly less than the amount of benzene and toluene. Therefore, it is plausible that the blood levels of these compounds are much less increased in smokers when compared to non-smokers. It should be noted that the blood/air partition coefficients of ethylbenzene and xylenes are significantly higher than that of benzene and toluene (Table 3). Pulmonary retention of these compounds should be very effective, whereas exhalation should be less pronounced than in the case of benzene and toluene. The exhalation data reported by Wallace et al. (1985), Wallace and Pelizarri (1986), and Hartwell et al. (1987) are consistent with this conclusion.

In the present study the blood samples were collected at different times of the day (predominantly in the morning hours) in order to assess the "normal", every-day exposure to these substances. It can be ex-

pected that under special conditions, e.g. smoking many cigarettes within a short period of time, heavy exposure to passive smoking or longer residence time in rooms with high VOC concentrations in air, much higher blood levels can be recorded. As mentioned above sidestream smoke of cigarettes contains even higher concentrations of VOC than mainstream smoke (IARC 1986). It has been shown that the indoor air concentrations of these smoking-related chemicals is higher in homes with smokers than in homes without smokers (Wallace and Pelizarri 1986; Krause et al. 1987). Unfortunately, the individual exposure conditions of the subjects were not evaluated in this study to relate the blood levels to personal air levels. This should be done in further studies.

With regard to the toxicokinetic behaviour of benzene and other VOC in the human body blood samples from smokers should be collected at least 30 min after the last cigarette. According to Sato (1988) the distribution of benzene, toluene and xylenes in the human body can be described by a three-compartment model: (1) a compartment (essentially the circulating blood and vessel-rich tissues), from which benzene is rapidly eliminated, either by exhalation or metabolic conversion, (2) a compartment (mainly muscles and skin) with low tissue/blood partition coefficients (1–2), and (3) a compartment (fat tissues, including white bone marrow), which has a very high affinity for these compounds. The biological half-life of lipid-soluble organic solvents in the latter compartment may be rather long. For benzene, a biological half-life of 20 to 30 h has been determined (Sherwood 1972; Berlin 1985). It can be assumed that under conditions of current environmental exposure the blood concentrations reflect, in part, the current exposure level and, in part, the human load of benzene resulting from long-term exposure. During and shortly after smoking or other kinds of acute exposure the blood concentration predominantly reflects the acute exposure situation. In order to evaluate in the human body burden resulting from long-term exposure the blood samples should be drawn about 1 to 3 h after exposure from smoking or other kinds of acute exposures.

The present study shows that the measurement of volatile aromatic hydrocarbons in blood is a useful monitoring method for detecting low-level exposures to these agents. Due to their relatively long biological half-life in the above-mentioned third compartment it should be possible to detect increased exposures even a few days after termination of exposure. The determination of phenolic benzene metabolites lacks specificity and sensitivity in assessing low-level environmental benzene exposure since phenol and its conjugates are excreted in the urine in relatively high amounts as products of protein catabolism and after exposure to

other substances as well (Lauwerys 1983). Likewise, the urinary excretion of hippuric acid is not appropriate for assessing low-level toluene exposure, whereas the determination of o-cresol might be more successful (Angerer 1979; Apostoli et al. 1982; WHO 1985). In order to assess the carcinogenic risk of benzene exposure the measurement of nucleic acid adducts in urine has been proposed (Norporth et al. 1988). Since bone marrow retains about 20 times as much benzene as blood (Braier et al. 1981; Sato 1988), the finding of even low levels of benzene in blood may be of toxicological significance.

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Received April 16 / Accepted July 14, 1989