

Benzene and lead exposure assessment among occupational bus drivers in Bangkok traffic

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Abstract: Four environmental and biological monitoring sites were strategically established to evaluate benzene and lead exposure assessment at various traffic zones of Bangkok Metropolitan Region (BMR). Biological measurement of 48 non air-conditioned, male bus drivers was carried to study the relationship between individual exposure levels and exposure biomarkers. The study group was further subdivided into four age groups (16—25, 26—35, 36—45 and 46—55 years old) to monitor the age—related exposure effects. A total of 12 unexposed persons were deliberately chosen as the control group. Measurement of unmetabolized benzene in blood and analysis of urinary *tt*-Muconic acid urine and urinary creatinine are recommended as biomarkers of benzene exposure. Measurement of lead in blood and urine is also recommended for the biological monitoring of lead exposure.

During the monitoring period, benzene and lead levels at Yaowarat Road was C_6H_6 : $42.46 \pm 3.88 \mu\text{g}/\text{m}^3$, Pb: $0.29 \pm 0.03 \mu\text{g}/\text{m}^3$ and decreased to C_6H_6 : $33.5 \pm 1.35 \mu\text{g}/\text{m}^3$, Pb: $0.13 \pm 0.01 \mu\text{g}/\text{m}^3$ at Phahonyothin Road. Significant difference was established between the nonsmoking exposed group and nonsmoking control group for blood benzene concentrations ($P < 0.001$, two-tailed, Mann-Whitney U test). Strong correlations were also found between *trans-trans*-Muconic acid concentrations in post shift samples and atmospheric benzene concentrations. Similarly, good correlation between all of biomarkers and lead level in air is established from automobile emissions.

The analysis revealed that among the occupational population in the urban sites, the driver groups were found to have the highest risk of benzene and lead exposures derived from automobile emission.

Keywords: benzene and lead measurement; biomarkers; exposure assessment; human biomonitoring; unleaded gasoline

Introduction

Air pollution problems as a result of fast growth in the total number of vehicles in urban cities are on the increase. Preliminary epidemiological exposure assessment suggest that the detrimental effect of air pollution generated by vehicular traffic in developing cities like Bangkok and many other Asian cities are often much higher than in developed cities where most of the previous health research has been undertaken. In urban cities, environmental and biological monitoring of the occupational workplace can be used as an indicator for driver groups who are exposed occupationally to traffic pollutants such as benzene and lead content (Akhter, 1993).

Likewise, vehicles utilizing unleaded gasoline will emit great quantities of volatile organic compounds (VOC) such as benzene to street air and produces deterioration in air quality and health. Long-term inhalation exposure to benzene and toluene at a certain level may cause chromosomal damage, leukemia and other health effects (U. S. EPA, 1998). In the work place, exposure of the general population to benzene is mainly through inhalation. Once in the bloodstream, benzene is temporarily stored in the bone marrow and fat. The liver and kidney convert benzene into metabolites, which are excreted in the urine, primarily as the sulfate and glucuronide conjugates of phenol. Most of the unmetabolized benzene is eliminated by exhalation. Exposure studies show potential biomarkers of benzene exposure can be divided into three groups: (1) Unmetabolized benzene is eliminated by exhalation via air, blood and urine. (2) Urinary metabolites of benzene such as ring-hydroxylated compound (phenol), ring-open compounds, *trans*, *trans*-muconic acid (*ttMA*), and glutathione adduct (*S*-phenylmercapturic acid)

and (3) Adducts with DNA (*N*⁷-phenylguanine), haemoglobin or albumin (*N*-phenylvaline, *S*-phenylcysteine). For human biomonitoring proposes, only the first two groups have been applied to measure unmetabolized benzene in blood and urine as biomarkers of benzene exposure (Scherer, 1998). Analysis of urinary *tt*-Muconic acid in a post-shift urine specimen is also recommended for the biological monitoring of benzene exposure (Kok, 1997).

Although leaded gasoline was completely phased out in 1996, a maximum allowable lead content of 0.013 g/L is still allowed in Thai unleaded gasoline. The combustion of unleaded gasoline containing allowable alkyl lead compounds had been considered as the prominent source of lead compounds in the atmosphere near traffic zones. There have been recent studies of background blood lead levels on adults and children from many different types of activities in Thailand (Ruangkanchanasetr, 1994). When lead passes into the blood stream, it spreads to many organs, such as the brain, lungs, liver, spleen and bones. The half-life of lead in blood is approximately 36 d and a second half-life is generally considered to be approximately 4 years. The blood lead level is commonly used to assess indicative exposure. Research into the detrimental effects of lead poisoning on human health shows that blood lead levels of 50—80 $\mu\text{g}/\text{dl}$ can cause chronic kidney deterioration in adult and severe nerve damage in children (Waldren, 1974).

The aim of this study was to investigate the benzene and lead exposure among occupational bus drivers in Bangkok traffic through environmental and bio-monitoring and assess the health risk to individuals. In addition, the results of this research effort may be useful for decision-makers in deploying air pollution control strategies to reduce benzene and lead level further from transportation sector in Bangkok Metropolitan Area (BMA, 1999).

1 Materials and methods

1.1 Study sites

Four air sampling and bio-monitoring locations were established near some of the busiest road intersections in the Bangkok Metropolitan Area (BMA). The criterion for selection of monitoring locations was based on traffic density and flow condition in particular traffic zone, as well as other factors including the response of bus companies and drivers to participate in the study. The study was undertaken from June, 2001 to June, 2002. The descriptions for individual location of the monitoring stations are indicated in Table 1 and Fig. 1, respectively.

1.2 Study groups

A total of 48 non air-conditioned, male bus drivers at the four monitoring sites were selected for the sixteen study groups and represented 1% to 8% of the daily traffic exposed driver at each study

site. Their daily working time ranged from 8 to 14 h and had an average pollutant exposure dose of 83% to 94%. The working group was further classified into four different age groups (16–25, 26–35, 36–45 and 46–55 years old) to provide better indication of pollutant exposure due to aging. The eligible study groups were checked for medical history and socio-economic lifestyles, which are useful factors for the occupational exposure assessment. Wallace (Wallace, 1996) reported that the effect of cigarette smoke contributes about 89% of the overall source of individual benzene exposure and must be considered when evaluating suspected cases of exposure. In this study, cigarette smoking is considered as a potential confounding factor for benzene bio-monitoring, thus only exposed non-smoking drivers are recruited for the study group.

Four control groups with non-daily traffic exposure of 12 persons (25% of the study groups) were selected from those who were not exposed daily to traffic air pollutant and spend most of their time, work

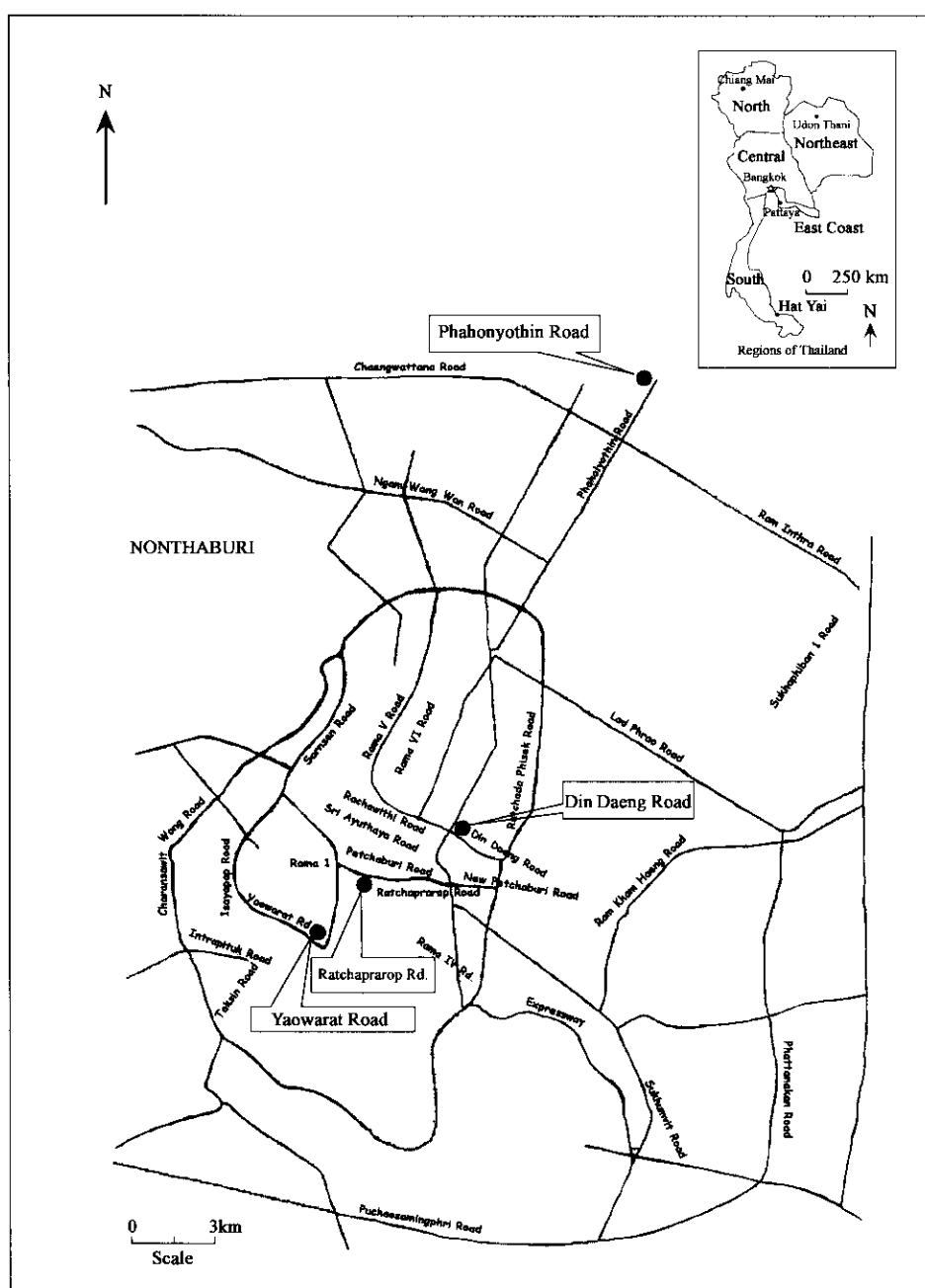


Fig. 1 Locations of sampling stations at the study area

and live primarily in air-conditioned indoors. They were expected to have lower pollutant exposure because indoor air pollution level was much

different from outdoor level.

Table 1 Description of monitoring sites for Bangkok Metropolitan Area

Site	Location, traffic zones	Road layout	Vehicle/(h·lane), at peak h	Benzene conc., $\mu\text{g}/\text{m}^3$	Lead conc., $\mu\text{g}/\text{m}^3$
A ₁	Yaowarat Rd. (Inner Core)	6 lanes	1212	42.46 ± 3.88	0.29 ± 0.09
A ₂	Din Daeng Rd. (Inner, section I)	Near T-junction 6 lanes	1132	39.95 ± 2.40	0.16 ± 0.07
A ₃	Ratchaprarop Rd. (Inner, section II)	Near T-junction 6 lanes	1104	35.91 ± 1.80	0.15 ± 0.03
A ₄	Phabonyothin Rd. (Middle)	8 lanes	920	33.50 ± 1.35	0.13 ± 0.01

Source: Land Transportation Department, Bangkok, Thailand, 2001

1.3 Benzene

Sampling for atmospheric benzene can be accomplished by simple charcoal sampling traps (SKC No. 226-09). The sampling rate was maintained at 0.2 L/min using a portable air-sampling pump (SKC model 224-PCXR8). The procedure is according to NIOSH method (P&CAM127). Individual exposure to benzene during the work shift (8 h) was measured by using a passive organic vapor monitor (3M, model 3500). The air pollutant was adsorbed by and active adsorbent medium (charcoal tube). After sampling, all sampling charcoal tubes were brought to the laboratory for analysis, within 8 h. The trapped benzene in the charcoal tube was desorbed in 3 ml of carbon disulfide (CS₂), shaken for 30 min in ultrasonic bath and analyzed by gas chromatography (GC). In this study, the gas chromatograph was Hewlett Packard HP 6890 Series with a flame ionisation detector (FID). The column was a 5% phenyl methyl siloxane (HP-5), 0.32 mm i. d. × 30 m and 0.25 μm film thickness. The column temperature was 110°C, injector and detector temperature at 130°C with a nitrogen flow rate of 45 ml/min. Peaks for benzene is eluted at 2.2 min. The linearity of the system was demonstrated by injecting a series of standards for each analyst in the range of about 10 to 1000 ppbv. The detection limit of benzene by considering S/N > 3 is 0.05 mg/L.

The blood collection (5 ml/sample) is performed using Venoject glass vacuum tubes containing 500 μl of 10% ethylenediaminetetra-acetic acid (EDTA) solution. After sampling, blood samples were stored at 4°C and analyzed within 48 h. Blood sample was pretreated by adding 0.2 mg Antifoam B emulsion and spiked 10 μl deuterated benzene as internal standard. A purge and trap sampling device (Tekmar model LSC 2000, Tenax trap 27 cm) was used to sparging blood sample to the gas chromatography-mass spectrometry (GC 3400; Varian, Finnigan Mat ITS40, ion trap mass spectrometry). The column was a 5% phenyl methyl siloxane (DB1), 0.25 mm i. d. × 30 m and 0.25 μm film thickness. The injection condition was split 1:50 at 250°C with a helium flow rate of 40 ml/min at pressure 13 psi. The condition of mass spectrometry is mass range: 50–100 amu, filament delay: 150 seconds, electron impact: 70 eV and ion source: 210°C. The linearity of the system was demonstrated by injecting a series of standards for each analyst in the range of about 10 to 1000 ppbv.

The urine samples were collected in polycarbonate bottles, during the work shift (8 h) and immediately transferred to the laboratory for analysis. Sample was pretreated by preconditioned strong anion-exchange (SAX) column. An aliquot of 1 ml of alkalized urinary sample (pH 7

to 10) was applied to the SAX column, which was subsequently washed with 2-ml H₂O, 2-ml of 0.1 mg/L NaCl and 2-ml 1% (v/v) acetic acid. The trans, trans-muconic acid (tt-MA) was eluted with 1 ml 10% (v/v) aqueous acetic acid in glass vials and made up the volume to 5 ml with millipore water. The trans, trans-muconic acid (tt-MA) was determined by a Hitachi 7000 series high performance liquid chromatography (HPLC) with a C18 ODS2 column (4.6 × 150 mm, 5 μ , Prodigy, Phenomenex, USA) and UV detector, 262 nm. The mobile phase flow rate was set at 1.2 L/min with a (90/10, v/v) 1% aqueous acetic acid-methanol/methanol mixture. Standards of known trans, trans-muconic acid (tt-MA) derivatives in the range of about 10 to 1000 ng/ml were used for daily calibration. The detection limit of trans, trans-muconic acid (tt-MA) is 0.07 ng/ml.

Urinary creatinine was measured by using Sigma Diagnostic Creatinine Kit (Cat No. 555-A) at the absorbance of 500 nm. Briefly, 15 μl of water, creatinine standard (3 mg/dl) or urine sample is mixed with 150 μl of alkaline picrate solution at room temperature for 10 min. The initial absorbance of either standard or sample are measured against water (blank) at the wavelength of 500 nm. Subsequently, 5 μl of acid reagent is added into each tube, mixed thoroughly and allow standing for 5 min at room temperature. The final absorbance of standard and samples are read against blank at 500 nm. The concentration of urinary creatinine was calculated as follows:

$$\frac{\text{Initial absorbance (sample)} - \text{final absorbance (sample)} \times 3 \text{ mg/dl}}{\text{Initial absorbance (standard)} - \text{final absorbance (standard)}}$$

1.4 Lead

Particulate lead is collected by 0.8 μm membrane filter with 1 to 4 L/min flowrate. The particulate sample is digested with mixture of 3:1 (v/v) HNO₃:H₂O₂ and diluted with distilled water (P&CAM7105; NIOSH, 1990). Measurements of lead in blood and urine are considered the most useful tool for screening and assessing exposure. The blood sample is digested by mixture of 3:1:1 (v/v/v) HNO₃:HClO₄:H₂SO₄ (P&CAM8005; NIOSH, 1985a). The urinary samples are extracted with polydithiocarbamate resin, filtered on cellulose ester membrane, neutralized by sodium hydroxide solution, followed by ashing, dissolution, heating and diluting with distilled water (P&CAM8310; NIOSH, 1984). Particulate lead is collected by 0.8 μm membrane filter with 1 to 4 L/min flowrate.

All analytical measurements for heavy metals were followed to the NIOSH's Manual of Analytical Methods. The most common method currently used for the analysis of lead in biological samples is graphite

furnace atomic absorption spectrometry (Hitachi Z-8230) with Zeeman background correction that can minimize the impact of the absorbency of molecular species. Limits of detection for atmospheric particulate lead using graphite furnace atomic absorption spectrometry are generally in the 0.1–20 $\mu\text{g}/\text{m}^3$ range. Likewise, the detection limit for blood and urinary samples are 0.01 $\mu\text{g}/\text{dl}$ and 0.005 mg/L , respectively. Calibration was performed using external standards prepared from a set of corresponding standard solutions.

1.5 Statistical analysis

All analytical measurements are performed in triplication to give mean values with standard deviation. In all statistical analysis, every effort is ensured that benzene and lead concentrations were kept below the minimum detection limit of the laboratory methods. In order to find out the biological monitoring effects on benzene/lead concentration in assessing exposure, statistical analysis was carried out. One-way analysis of variance (ANOVA) was used to determine the statistically significant difference between each assessing exposure with respect to a control treatment. In addition, statistical program (SPSS version 10.0) and the non-parametric test (Mann-Whitney U test) were used to analyze the differences between exposed and control groups.

2 Results

2.1 Benzene and lead in environmental samples

Table 1 shows an indicative result between atmospheric benzene and lead levels and traffic density at each study site. It is revealed that the average airborne benzene and lead levels at station A_1 was C_6H_6 : $42.46 \pm 3.88 \mu\text{g}/\text{m}^3$, Pb: $0.29 \pm 0.03 \mu\text{g}/\text{m}^3$ and decreased to C_6H_6 : $33.5 \pm 1.35 \mu\text{g}/\text{m}^3$, Pb: $0.13 \pm 0.01 \mu\text{g}/\text{m}^3$ at station A_4 . The monitoring result indicated that benzene and lead levels in Bangkok were generally below established standards (Fig. 2). Elevated benzene and lead levels were found in those areas which were characterized by poor traffic flow particularly at station A_1 , Yaowarat Road (1212 vehicle/(h·lane)). This is an one way street with frequent traffic jams, low speed, braking, accelerating and so on. Hence, if traffic flow is “stop and go driving”, air pollutant level will also be maximum. Similarly, Din Daeng and Ratchaprarop Roads (sites A_2 and A_3) with frequent decelerating mode at intersection had also shown higher traffic pollutant level. On the other hand, lower automobile emission level is expected on Phahonyothin Road (site A_4) which is a 8 lanes-two ways highway providing stable traffic movement. In general, the highest average atmospheric benzene and lead levels were found among busy main roads with higher traffic density while lower levels were found among roadside stations with lower traffic density (Table 1). The sequence of atmospheric benzene and lead levels at various traffic zones is $A_1 > A_2 > A_3 > A_4$. The median level of benzene at control site was $9.49 \mu\text{g}/\text{m}^3$. Significant difference was statistically obtained between study and control sites for benzene exposure level ($P < 0.001$, two tailed Mann-Whitney U test). From this result, it is shown that bus drivers have a higher risk of benzene exposure than general population. In this study, all the benzene exposure levels were found to be below the ACGIH-TWA limit of 500 ppb or $1.6 \text{ mg}/\text{m}^3$ (ACGIH, 1991) to cause any adverse health effect from the occupational work place (Fig. 2). Likewise, the Pollution Control Department of Thailand proposed TWA of lead level to be $1.5 \mu\text{g}/\text{m}^3$ (PCD, 1997).

Although lead level in ambient air of Bangkok is not high, the

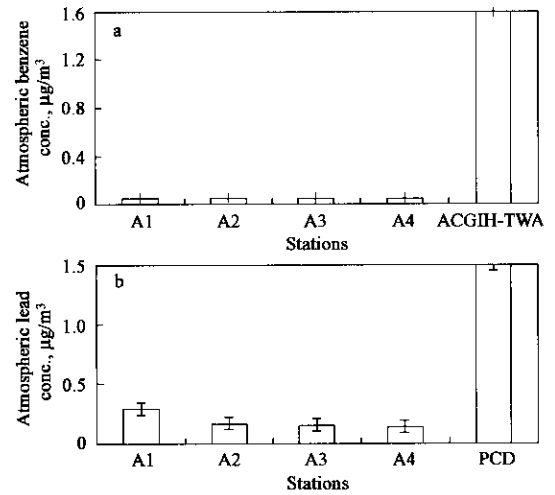


Fig. 2 Ambient air concentrations for (a) benzene and (b) lead at various monitoring sites

Notes: A_1 = Yaowarat Rd.; A_2 = Din Daeng Rd.; A_3 = Ratchaprarop Rd.; A_4 = Phahonyothin Rd.; ACGIH = American Conference of Governmental Industrial Hygienists, 1991; PCD = Pollution Control Department, Ministry of Science, Technology and Environment, Thailand, 1997

government's policy is to reduce lead level further by introducing unleaded gasoline. Consequently, annual benzene levels are increasing over the past 6 years. The air quality problem in Bangkok is sufficiently serious that the government is considering gasohol for substitution of gasoline (Leong, 2002).

2.2 Benzene and lead in biological samples

Biological measurements were conducted to monitor the biomarker of benzene and lead levels in Bangkok bus drivers. Graphs were plotted to show the variation of biomarkers with different traffic condition of the monitoring station.

2.2.1 Biomarker of blood benzene

The result demonstrated that there are variation in blood benzene level with traffic density and flow condition at different traffic zone (Yaowarat Road (A_1): 189.90 ± 7.10 – 253.68 ± 12.16 ppt, Din Daeng Road (A_2): 167.12 ± 2.60 – 237.79 ± 8.30 ppt, Ratchaprarop Roads (A_3): 155.65 ± 1.60 – 188.33 ± 3.56 ppt and Phahonyothin Road (A_4): 128.10 ± 1.45 – 146.93 ± 5.82 ppt). The overall median level of blood benzene in bus driver was 184.58 ppt. For control group, the median level of blood benzene concentration is 80.09 ppt. Significant difference was established between the nonsmoking exposed group and nonsmoking control group for blood benzene concentrations ($P < 0.001$, two-tailed, Mann-Whitney U test). The results were compared with previous study conducted by Kok *et al.* (Kok, 1997) who reported that the truck driver of refinery plant has blood benzene level of 3399 ppt at the exposure level of 1.98 ppm.

2.2.2 Biomarker of tt-muonic acid (tt-MA)

Similar to blood benzene level, it can be seen that tt-muonic acid (tt-MA) in urinary samples at site A_1 , Yaowarat Road (43.16 ± 0.46 – 63.35 ± 1.05 ng/ml) and A_2 , Din Daeng Road (38.30 ± 3.37 – 57.83 ± 3.00 ng/ml) were higher than site A_3 , Ratchaprarop Road (34.11 ± 3.70 – 54.66 ± 3.10 ng/ml) which was in turn higher than site A_4 , Phahonyothin Road (13.08 ± 0.06 – 46.87 ± 0.79 ng/ml). During work-shift (8 h), the median level of urinary trans, trans-muonic acid

(t-MA) among study and control groups were 47.04 ng/ml and 17.74 ng/ml, respectively. This may be due to the fact that subjects were exposed to benzene before the time of sampling, such as oral and dermal exposures. In most cases, the finding showed that bus driver working nearer to denser traffic site are usually exposed to more benzene accumulations than those in less traffic site. A significant difference was statistically established between exposed and control groups for t-MA concentrations ($P < 0.01$, two tailed, Mann-Whitney U test). In addition, the airborne benzene concentrations were plotted against t-MA concentrations in logarithmic presentation. Strong correlations were found between trans, trans-muconic acid concentrations in post shift samples and atmospheric benzene concentrations (Table 2).

Table 2 Correlations between biomarkers and airborne benzene/lead level for exposed drivers

Relationship	Correlated equation
$\log(\text{benzene exposure})$ vs $\log(\text{benzene in air})$	$y = 0.5715x + 0.6768, R^2 = 0.9698$
$\log(\text{blood benzene})$ vs $\log(\text{benzene in air})$	$y = 0.4316x + 0.5894, R^2 = 0.9877$
$\log(\text{t-MA})$ vs $\log(\text{benzene in air})$	$y = 0.7766x + 0.2445, R^2 = 0.9767$
$\log(\text{urinary creatinine})$ vs $\log(\text{benzene in air})$	$y = 0.594x + 2.3427, R^2 = 0.9076$
Blood lead vs lead in air	$y = 0.0153x - 0.1175, R^2 = 0.5404$
Urinary lead vs lead in air	$y = 0.275x - 0.2508, R^2 = 0.6939$

2.2.3 Biomarker of urinary creatinine

Analytical results in Fig. 3 indicate that urinary creatinine concentrations in bus drivers fall in the range 0.01–0.08 mg/g creatinine. The median level of urinary creatinine during work shift (8 h) was 0.06 mg/g creatinine. A variety of factors can affect the level of creatinine excretion. These factors include body mass, age, gender as well as dietary intake. The biological exposure indices (BEI) of urinary creatinine for a healthy worker at the end of work shift was 0.5 mg/g creatinine as set by the American Conference of Governmental Industrial Hygienists (ACGIH, 1998). Compare with the BEI indices, all of the driver groups were found to have urinary creatinine concentrations below 0.5 mg/g creatinine.

2.2.4 Biomarker of blood lead

The blood lead concentrations of bus drivers collected from the various sites are given in Fig. 4. Blood samples from Yaowarat Road had the highest lead content (12.07 ± 0.06 – 29.47 ± 0.83 biomarker of urinary creatinine $\mu\text{g}/\text{dl}$) indicating relatively higher automobile emission at this site. Similarly, Din Daeng Road (10.15 ± 0.17 – 26.72 ± 0.18 $\mu\text{g}/\text{dl}$) and Ratchaprarop Roads (9.57 ± 0.18 – 23.45 ± 0.31 $\mu\text{g}/\text{dl}$) with high automobile emission at intersection had also shown high blood lead level while the phahonyothin site having the lower blood lead level (8.92 ± 0.09 – 19.45 ± 1.90 $\mu\text{g}/\text{dl}$). The overall blood lead concentrations in the study areas were found to be relatively low, when compare to blood lead concentrations of 68 $\mu\text{g}/\text{dl}$ in an Egyptian traffic policeman conducted by Ahmed *et al.* (Ahmed, 1987). Throughout the monitoring period, most of the bus drivers have blood lead level below the national standard of 25 $\mu\text{g}/\text{dl}$. However, many studies reported that even at low blood lead level of 10 mg/dl, it could affect central nervous system in children.

2.2.5 Biomarker of urinary lead

In this study, urinary lead concentrations in Bangkok bus drivers were found to be in the range 1.00 ± 0.08 – 1.81 ± 0.01 mg/L. Fig. 4a shows that driver groups working in monitoring site, A₁ had higher

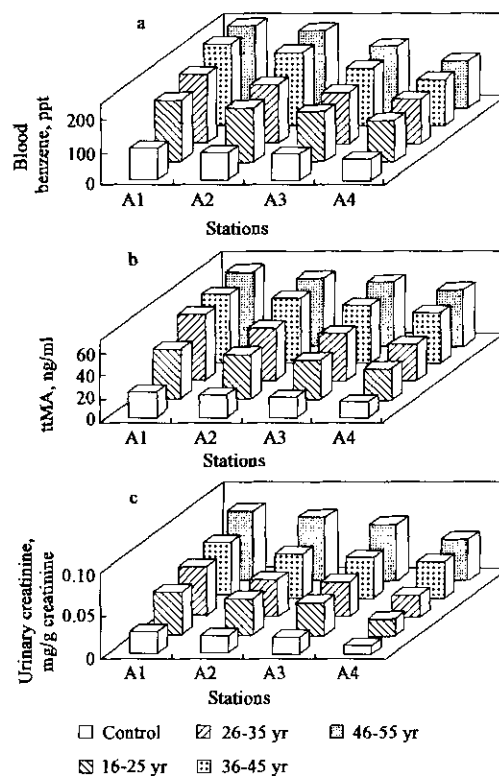


Fig. 3 Biomonitoring of benzene-exposed drivers with respect to: (a) unmetabolized benzene in blood; (b) t-MA in urine and (c) urinary creatinine for different age groups and monitoring sites

Notes: A₁ = Yaowarat Road; A₂ = Din Daeng Road; A₃ = Ratchaprarop Road and A₄ = Phahonyothin Road

urinary lead level than that of the driver groups working in other monitoring sites. The results of statistical analysis showed that higher dose of exposure to traffic pollutant could cause higher lead excretion in the driver's urine. Adverse health effect can happen to the driver group if the daily pollutant exposures were recurrented over several hours per day for months or years. In any case, to conform our study, the mean urinary lead concentrations of different driver groups were compared with that of control groups (non-exposed groups). The results revealed that the urinary lead levels in the control group were remarkably lower than that of the different age groups at all monitoring sites.

2.3 Age-related exposure

Fig. 3 shows the relationship between different age groups of bus driver and biomarkers with respect to blood benzene, t-MA and urinary creatinine levels at various monitoring sites. In general, exposure biomarkers were normally increasing with age when the subjects were constantly exposed to excessive pollutant exposure. The finding showed that the younger adult group (16–25 years old) perceives lower pollutant exposure than other older age groups in the same monitoring site. Throughout the monitoring period, it was found that the tested group of 46–55 years old living in monitoring site, A₁ (Yaowarat Road) had the highest blood benzene level (253.68 ± 8.83 ppt) than the 16–25 year groups (189.90 ± 3.43 ppt). The analytical finding revealed that different t-MA and urinary creatinine concentrations were obtained when urinary samples of same age groups were collected from different traffic density sites. Similarly, it can be seen from the graphs (Fig. 4a) that blood lead data collected on older adults (46–55 years old) showed

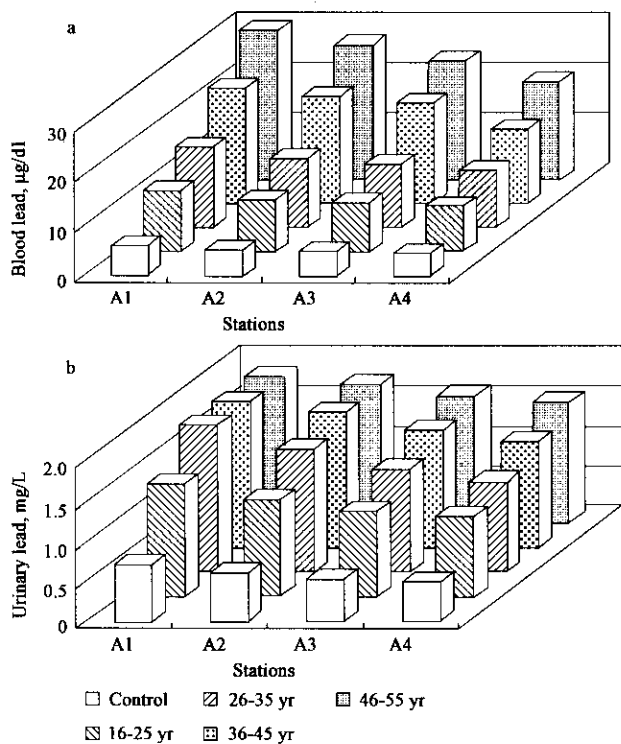


Fig. 4 Biomonitoring of lead-exposed drivers with respect to (a) unmetabolized lead in blood; (b) unmetabolized lead in urine for different age groups and monitoring sites

Notes: A₁ = Yaowarat Road; A₂ = Din Daeng Road; A₃ = Ratchaprarop and A₄ = Phahonyothin Road

higher blood lead level than other younger age groups of the same monitoring site. Comparing the urinary lead data collected for the age group at site A₁ (Fig. 4b), they showed to have higher urinary lead content relative to the same age groups of drivers in other monitoring sites who were not so exposed to excessive lead pollution.

2.4 Correlation analysis

In this study, regression analysis was used to show correlation between benzene/lead in air and the biomarker fractions resulting from benzene/lead exposure in the workplace (Table 2). It would help to understand the relationship between personal exposure and environmental media, in various biomarkers.

The correlation between blood benzene and urinary benzene with benzene in air is very high. The correlation coefficients R^2 shown in Table 1 are greater than 0.9 which are even greater than those obtained by the works carried out by Kok *et al.* (Kok, 1997) ($R = 0.38$ – 0.64). Similarly good correlation between all of biomarkers and lead level in air is established and implies that the major source of lead in the atmosphere is probably from automobile emissions.

3 Conclusions

The analytical results showed that benzene and lead in environmental and biological samples can be used to assess occupational exposures derived from automobile emission. Good correlation is found between individual exposure levels and exposure biomarkers. This implies that benzene and lead exposure in automotive occupations could develop higher risk of adverse health effects than the general population. Ashley *et al.* (Ashley, 1996) conducted measurement of volatile organic

compounds in human blood and concluded that age, sex and lifestyle are the important factors for accuracy of measurement. However, in our study we do not take into consideration other controlling factors such as gender, service years, smoking habit and exposure patterns. These may be the work for future research.

In this study, blood benzene exposure for bus drivers were found to be below the ACGIH-TWA limit of 500 ppb or 1.6 mg/m^3 . Likewise, most of the bus drivers were found to have blood lead level below the national standard of 25 µg/dl however, there was a slight increase in blood lead levels with years of exposure, and is a health concern as well.

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