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Measurement of genotoxic air pollutant exposures in street vendors and school children in and near Bangkok

Mathuros Ruchirawat[a,b,*], Panida Navasumrit[a], Daam Settachan[a], Jantamas Tuntaviroon[a], Nantaporn Buthbumrung[a], Suman Sharma[a]

[a]*Laboratory of Environmental Toxicology, Chulabhorn Research Institute, Bangkok 10210, Thailand*
[b]*Department of Pharmacology, Faculty of Science, Mahidol University, Bangkok 10400, Thailand*

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Abstract

The effects of air pollution on human health are a great concern, particularly in big cities with severe traffic problems such as Bangkok, Thailand. In this study, exposure to genotoxic compounds in ambient air was studied by analysis of particle-associated polycyclic aromatic hydrocarbons (PAHs) and benzene through direct measurement of concentrations in air as well as through the use of different biomarkers of exposure: urinary 1-hydroxypyrene (1-OHP) for PAHs and urinary *t,t*-muconic acid (*t,t*-MA) for benzene.

The study was conducted in various susceptible groups of the population with different occupations in 5 traffic-congested areas of Bangkok, as well as in primary school children. The level of total PAHs on the main roads at various sites ranged from 7.10 to 83.04 ng/m^3, while benzene levels ranged from 16.35 to 49.25 ppb. In contrast, ambient levels in nearby temples, the control sites, ranged from 1.67 to 3.04 ng/m^3 total PAHs and 10.16 to 16.25 ppb benzene.

Street vendors selling clothes were exposed to 16.07 ± 1.64 ng/m^3 total PAHs and 21.97 ± 1.50 ppb benzene, levels higher than in monks and nuns residing in nearby temples (5.34 ± 0.65 ng/m^3 total PAHs and 13.69 ± 0.77 ppb benzene). Grilled-meat vendors in the same area were exposed to both total PAHs and benzene at even higher levels, possibly due to additional formation of PAHs during the grilling of meat (34.27 ± 7.02 ng/m^3 total PAHs; 27.49 ± 2.72 ppb benzene). At the end of the workday, urinary 1-OHP levels in street vendors (0.12 and 0.15 μmol/mol creatinine in clothes and grilled-meat vendors, respectively) were significantly higher than in controls (0.04 μmol/mol creatinine; $P < 0.01$). Afternoon urinary *t,t*-MA levels in both groups of street vendors (0.12 mg/g creatinine) were also significantly higher than in controls (0.08 mg/g creatinine; $P < 0.05$).

School children from two schools in Bangkok were exposed to total PAHs and benzene at levels of 6.70 ± 0.47 ng/m^3 and 4.71 ± 0.25 ppb, respectively, higher than those to which children living outside the city were exposed (1.25 ± 0.24 ng/m^3 total PAHs; 2.10 ± 0.16 ppb benzene). At the end of the school day, levels of urinary 1-OHP and *t,t*-MA were significantly higher ($P < 0.001$ and $P < 0.01$, respectively) in Bangkok school children (0.23 μmol/mol creatinine and 0.27 mg/g creatinine, respectively) than in school children from outside Bangkok (0.10 μmol/mol creatinine and 0.08 mg/g creatinine, respectively).

Keywords: 1-Hydroxypyrene; Air pollution; Bangkok; Benzene; Biomarker; Children; Genotoxic; Polycyclic aromatic hydrocarbons; *t,t*-Muconic acid

Introduction

Atmospheric pollution is an established health hazard in many cities around the world, especially busy metropolises like Bangkok. An important source of this pollution is incomplete fossil fuel combustion, with an increasing trend in numbers of fuel-driven vehicles and traffic congestion being observed globally. Studies have been conducted

* Corresponding author. Laboratory of Environmental Toxicology, Chulabhorn Research Institute, Vipavadee Rangsit Highway, Lak Si, Donmuang, Bangkok 10210, Thailand. Fax: +66 2 574 0616.
E-mail address: mathuros@tubtim.cri.or.th (M. Ruchirawat).

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which show that there is a link between exposure to contaminated city air and health effects (Janssen et al., 2003; Nakatsuka et al., 1991; Oftedal et al., 2003; van Vliet et al., 1997). Two of the main genotoxic substances found in urban atmospheric pollution are polycyclic aromatic hydrocarbons (PAHs) and benzene. Exposures to these compounds are a concern, especially in occupational exposures, where levels tend to be higher, and in children, where there is a potentially greater susceptibility to their toxic effects.

PAHs constitute a large class of compounds that are generated through the incomplete burning of fossil fuels, tobacco, and meat, as well as natural-occurring processes such as volcanic eruptions and forest fires. Less than 20 of these compounds, out of more than a hundred, have been well-studied and characterized, and many of those, including benzo[a]pyrene (BaP), have been classified as probable human carcinogens. Exposures to PAHs can occur in the home, or occupationally, and may be through the inhalation of contaminated air or the consumption of contaminated food and water. Biomarkers have been developed to assess human exposure to PAHs (Grimmer et al., 1991; Jongeneelen et al., 1987). Urinary 1-hydroxy-pyrene (1-OHP) has served as a marker of occupational exposure to volatile PAHs where levels are generally low in people exposed only to ambient air pollution (Øvrebø et al., 1995; Pastorelli et al., 1999).

Benzene is also found in the environment from both natural and anthropogenic sources. It is found in crude oil and is released during volcanic eruptions and forest fires. Significantly, benzene is also released into the atmosphere from burning of coal, tobacco smoke, and gasoline. The primary route of exposure to benzene is through inhalation of contaminated air. Benzene is classified as a known human carcinogen, with the main target tissue being the hematopoietic system. Apart from measuring ambient and blood levels of the parent compound, urinary *trans,trans*-muconic acid (*t,t*-MA) is a good indicator for assessing exposure to low levels of benzene (Ducos et al., 1990).

Environmental monitoring of air pollutants such as benzene and PAHs is important, because the information obtained can help to ascertain if there is a potential health problem and who the high-risk groups are. Due to the nature and sources of these two classes of compounds of interest, much research has been done on traffic-related exposures. Traffic-related occupational exposure to PAHs in ambient air pollution has been studied in bus drivers (Nielsen et al., 1996), workers in bus garages (Hou et al., 1995), highway toll station booth attendants (Tsai et al., 2004), and traffic police officers (Merlo et al., 1998; Ruchirawat et al., 2002; Tomei et al., 2001). Benzene exposures have been monitored in the general population and in people exposed occupationally (Brugnone et al., 1989; Inoue et al., 1989; Karacic et al., 1987). It is of course the main hypothesis of such studies that occupations that necessitate increased exposure to traffic and traffic-related elements would increase exposure to traffic-related pollutants such as PAHs and benzene, and that this increased exposure is measurable, both in terms of ambient levels, as well as levels of biological markers. It may also be inferred that the greater the exposure, the greater the potential for health-related problems.

With a clearer understanding as to the patterns of exposure to these genotoxic compounds and who are at risk, it may be possible to initiate preventive or corrective measures so that the health risks from exposure to traffic-related pollution can be reduced.

Methods

Study location. Five heavily congested areas of Bangkok were chosen as study locations for roadside levels of PAHs and benzene, namely the Pratunam, Banglampu, Chakrawad, Pratumwan, and Anusawaree areas. One temple from each of 3 of these areas (Pratunam, Banglampu, and Anusawaree), located approximately 500 m from the main roads, was chosen in which to study ambient air levels, personal exposure levels, as well as biomarkers of exposure for all test subjects. Three schools were chosen in which to study exposures of school children to benzene and PAHs. Two of these schools are situated in Bangkok (Chakrawad and Pratumwan) and the other is situated in Bangphra district, Chonburi province.

Study population. All test subjects, apart from school-children, were non-smoking, healthy volunteers between the ages of 18 and 40. Schoolchildren were healthy 10 to 12 year-old boys. Street vendors set up their stalls directly at the roadside. The 2 control groups were monks/nuns who spent most of their time in the temples and primary school children who attended a rural school outside Bangkok.

Sample collection for PAH analysis. Air particulates were collected on glass fiber filters (37 mm) using personal air samplers attached to a battery-operated SKC air check sampler (model 224). Air samples were collected in the breathing zone of study subjects at a flow rate of 2 L/min. Sampling time was 8 h after which filters were wrapped in aluminum foil and kept in a plastic bag at −20 °C until analysis. Urine samples were collected in the morning (prior to the start of the work or school day) and afternoon (at the end of the work or school day), and stored frozen until analysis.

Quantification of PAHs. Filters were extracted by ultrasonication with dichloromethane (10 ml). The extracts were concentrated under a gentle nitrogen stream. All processes were carried out without direct exposure to light. After a solvent exchange step to acetonitrile, the extract was filtered through a 0.45-μm nylon syringe filter and analyzed by HPLC equipped with diode-array UV and fluorescent detectors. The PAHs were separated on a Lichrospher PAH (250 × 3 mm) column at 18 °C using a 60–100% acetonitrile gradient and a flow rate of 0.56 ml/min.