

# Influence of PAHs in ambient air on chromosomal aberrations in exposed subjects: International study – EXPAH

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## Abstract

The aim of this study was to determine the influence of carcinogenic polycyclic aromatic hydrocarbons (c-PAHs) in complex mixtures in ambient air on DNA damage (chromosomal aberrations) in occupationally exposed subjects measured as percent of aberrant cells (% AB.C.).

There were in total 203 exposed subjects and 150 respective controls in the whole project, allocated in three different European cities – Kosice (Slovakia), Prague (Czech Republic) and Sofia (Bulgaria). The studied population from Kosice (Slovakia) consisted of 106 subjects. From these 51 were exposed policemen and 55 were controls. The Czech population comprised 52 exposed policemen and 50 controls. In Bulgaria, there were two equally numerous exposed groups: 50 policemen and 50 professional bus drivers together with 45 controls. According to personal monitoring, policemen and bus drivers in the Bulgarian capital Sofia were exposed to the highest levels of c-PAHs amongst the exposed subject groups in the cities ( $45.3 \pm 25.9$  ng/m<sup>3</sup> in policemen resp.  $36.1 \pm 31.6$  ng/m<sup>3</sup> in bus drivers in Sofia,  $26.8 \pm 39.8$  ng/m<sup>3</sup> for policemen in Kosice and  $11.9 \pm 11.2$  ng/m<sup>3</sup> for policemen in Prague), compared to the respective controls ( $24.9 \pm 17.7$  ng/m<sup>3</sup> for controls in Sofia,  $7.9 \pm 3.8$  ng/m<sup>3</sup> for controls in Kosice and  $6.2 \pm 3.6$  ng/m<sup>3</sup> for controls in Prague).

We observed the following frequency of % AB.C. scored by conventional method:  $2.60 \pm 2.64$  in exposed policemen and  $2.14 \pm 1.61$  in controls in Kosice ( $p = \text{n.s.}$ );  $2.33 \pm 1.53$  in exposed policemen and  $1.94 \pm 1.28$  in controls in Prague ( $p = \text{n.s.}$ );  $3.04 \pm 1.64$  in exposed policemen, respectively,  $3.60 \pm 1.63$  in exposed bus drivers and  $1.79 \pm 0.77$  in the control group in Sofia ( $p < 0.05$ , respectively,  $p < 0.05$ ).

According to data from multiple regression analysis, and group comparison of smokers versus nonsmokers in Sofia also cigarette smoking ( $p = 0.055$ ) and the age ( $p = 0.020$ ) seem to play an important role within the aberrant cell formation in addition to the occupational c-PAHs exposure ( $p = 0.000$ ). Smoking status was the modifying factor for % AB.C. in Kosice ( $p = 0.020$ ) after multiple regression approach was employed.

In summary, we can say that subjects occupationally exposed to higher levels of c-PAHs in ambient air in Sofia are at greater genotoxic risk compared to those working indoors.

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**Keywords:** Air pollution; Personal monitoring; Carcinogenic PAHs; Chromosomal aberrations

**Abbreviations:** % AB.C., percentage of aberrant cells; CAs, chromosomal aberrations; c-PAHs, carcinogenic polycyclic aromatic hydrocarbons; PBLs, peripheral blood lymphocytes; PM, particulate matter

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## 1. Introduction

Ambient air contains a diversity of chemical compounds, some of which have genotoxic effects. One of the most important sources of genotoxins in air pollution is incomplete combustion of fossil fuels coming from car exhaust, industrial emissions and to some extent from residential heating [1,2]. Genotoxic airborne compounds often occur in complex mixtures and are absorbed on respirable particles (PM<sub>2.5</sub>). Exposure to ambient air pollution is a well-known environmental risk factor with various adverse health effects. Several air pollutants are considered to be carcinogenic. For example, exhaust from diesel engines has been classified as a Group 2A carcinogen according to the International Agency for Research on Cancer (IARC), whereas gasoline-driven engine exhaust has been classified as a Group 2B carcinogen [3,4]. Polycyclic aromatic hydrocarbons (PAHs) are the major group of carcinogens in ambient air, and most recent studies have focused on studying the PAH exposure in local residents and/or occupationally exposed subjects [2,5–8].

Different biomarkers have been used to assess the exposure to genotoxic compounds in general and occupational environments, ranging from mutagenic activity in urine to the formation of DNA-adducts and induction of chromosomal damage in target and surrogate tissue. The chromosomal aberration (CA) assay is the most extensively used and best validated biomarker of early biological effect in population studies since its availability for human biomonitoring and toxicology studies in the late 1960s [9]. Exposure to clastogenic chemicals usually induces chromatid-type aberrations such as breaks and exchanges. Chromatid-type exchanges in cells can be converted to chromosome translocations and these subsequent cells are predicted to survive for a long period [10]. Cells with chromosomal abnormalities can transform to cancerous cells as indicated by the presence of chromosome deletions and translocation in the majority of cancer cells [11]. An association between high CA frequency and cancer incidence was originally detected in a collaborative project of 10 Nordic cytogenetic laboratories [12]. An independent study among 10 laboratories in Italy, based on cancer mortality data, arrived at the same conclusion [13]. Also Czech studies showed that higher frequency of CAs in peripheral blood lymphocytes is predictive for increased risk of cancer [14,15]. The findings from 45 studies reviewed by Brandt and Watson [16] indicated that levels of CA show a good correlation with exposure to PAHs.

Although it has been clearly demonstrated that tobacco smoking induces CA, high CA level is predictive

for cancer also in nonsmokers and the subjects with no identifiable occupational exposure to carcinogens [17]. The observed association between CA level and cancer risk in unexposed subjects could be accounted to unidentified carcinogens in environment, diet or deficiency of micronutrients, such as folate. Dietary exposure to PAHs can be significant in nonsmokers. Corroborative data indicating that PAH exposure from diet is important are the findings that intake of charcoal-broiled meat is more correlated to blood PAH adducts than smoking [18,19]. Also genetic polymorphisms in genes responsible for xenobiotic metabolism or DNA repair genes may affect the individual ability to respond to genotoxins and as a result the risk of cancer development [20,21].

In the present study, the genotoxic effect of c-PAHs in airborne organic mixtures (ambient air) was assessed. As a study group, city policemen from three European cities with various levels of air pollution were chosen (Kosice – Slovakia, Prague – Czech Republic and Sofia – Bulgaria). In Sofia (Bulgaria) there were two different exposed groups: one composed of policemen and another composed of bus drivers. CAs in PBLs were used as a biomarker for genotoxic effect.

The current study is a part of a more complex international study concerning the influence of c-PAHs on the health of occupationally exposed subjects: EXPAH.

## 2. Materials and methods

### 2.1. Subjects and samples

In each country, the local ethical committee approved the study. An informed written consent was obtained from subjects from the three cities that took part in the study. All subjects completed a questionnaire in order to determine general health, life style, years of employment, smoking status, alcohol drinking habits, diet (smoked and grilled meat), and use of medications.

To avoid effects caused by differences in sex, all investigated subjects were males. Generally the exposed subjects were recruited from among city policemen, usually on duty in busy streets in 8 h shifts. The subjects enrolled as controls were employees and clerks working indoors during whole 8 h shifts and without identifiable occupational exposure. There were a total number of 203 exposed subjects and 150 respective controls involved in this study.

The population studied in Kosice (Slovakia) consisted of 106 subjects. From these 51 were exposed policemen (mean age = 32.2 years, ranging from 24 to 45 years) and 55 were controls (mean age = 33.6 years, ranging from 19 to 58 years). The Czech population comprised 52 exposed policemen (mean age = 31.7 years, ranging from 22 to 50 years) and 50 controls (mean age = 29.7 years, ranging from 20 to 51 years). In Bulgaria, there were two equally numerous exposed groups:

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