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Genotoxic Effects of Urban Air Pollution Exposure among Adult Residents of Delhi, India: Evidence from Biomonitoring and Cytogenetic Assays

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Abstract

Chronic exposure to urban air pollution poses a serious threat to human health due to the presence of genotoxic and carcinogenic compounds. The present study evaluated benzene exposure and associated genotoxic effects among adult residents of Delhi, India, and compared them with age- and sex-matched rural controls. Urinary Trans,trans-muconic acid (t,t-MA), a biomarker of benzene exposure, was quantified by HPLC-UV. Genotoxic damage was assessed using the micronucleus (MN) assay in exfoliated buccal and airway epithelial cells and the alkaline single-cell gel electrophoresis (Comet) assay in peripheral blood lymphocytes. Urban residents, particularly individuals occupationally exposed to vehicular emissions, exhibited significantly elevated urinary t,t-MA levels compared to controls. The frequency of MN in buccal and airway epithelial cells was markedly higher among Delhi residents, even among never-smokers and non-chewers. Lymphocytes from urban subjects showed significantly increased DNA strand breaks, as indicated by higher Comet tail length and frequency of damaged cells. Spearman's correlation analysis revealed strong positive associations between ambient respirable suspended particulate matter (RSPM), benzene, and benzo(a)pyrene levels with MN formation and DNA damage. These findings demonstrate that chronic exposure to Delhi's urban air pollution, particularly benzene-rich vehicular emissions, induces measurable genetic damage in exposed populations, underscoring the urgent need for effective air quality control and public health interventions.

Keywords: Air pollution; Benzene exposure; Genotoxicity; Micronucleus assay; Comet assay; Urban health; Vehicular emissions; Delhi,

Introduction

Urban air pollution is mixture of thousands of organic and inorganic compounds, some of which may cause damage to the DNA. These compounds are therefore genotoxic, and are considered extremely harmful for human health. The most notable examples are benzene and benzo(a)pyrene, which are genotoxic as well as carcinogenic. In addition, free radicals present in air pollution or generated in respiratory or defense cells such as neutrophils following pollution exposure may cause DNA damage. In view of these, we were interested to assess the genotoxicity in exposed cells of person chronically exposed to Delhi's air pollution.

As a measure of genetic damage at the level of chromosomes, we have employed the micronucleus (MN) test. MN is defined as microscopically visible, round or oval cytoplasmic chromatin mass next to the nucleus (Schmid, 1975). They originate from aberrant mitoses and consist of acentric chromosomes, chromatid fragments or whole chromosomes that have failed to be incorporated into the daughter nuclei during mitosis. The MN test is the most frequent technique used to detect chromosome breakage or mitotic interference, events thought to be associated with increased risk for cancer (Stich et al., 1982, Tolbert et al., 1991). MN is useful because it can be studied directly in target cells of the buccal and airway epithelium. Considering these, we have analyzed in this study the MN frequency in exfoliated buccal epithelial cells of persons chronically exposed to high level of traffic-related emissions in Delhi. In addition, single cell gel electrophoresis or alkaline Comet assay in circulating lymphocytes was undertaken for detection of DNA damage.

Micronucleus formation and other nuclear anomalies:

Micronucleus (MN) formation is the accepted laboratory practice to detect chromosomal breaks in a cell. MN is defined as microscopically visible, round or oval chromatin masses in the cytoplasm next to the nucleus (Schmid, 1975). MN consists of a part of the chromosome or chromatid, or a whole chromosome that has not been incorporated in the spindle apparatus due to aberrant mitosis (Schmid, 1975). Assessment of the number of MN is widely used to identify the genotoxic damages and its formation is considered a simple biomarker of mutagenic effect of environmental pollutants (Stich et al., 1982; Belien et al., 1995). Since the air pollutants enter the body via nasal-oropharyngeal route, the epithelial cells of the buccal mucosa are in constant touch with the foreign particles. Therefore, genotoxic effects of air pollutants can best be evaluated by MN test in buccal epithelial cells.

Criteria for identifying micronucleus:

MN are round or oval in shape, consist of nuclear material that is fully separated from the parent nucleus and covering a total area $<1/5^{th}$ of the parent nucleus (Tolbert et al., (1991). Both structural and numerical chromosomal aberrations have been observed fairly consistent in the lymphocytes and bone marrow cells of the individuals exposed to benzene at workplace. It is now generally accepted that benzene is a human clastogen (IARC, 1987). Increases in the number of both unstable and stable chromosomal aberrations were observed in men, even 2 years after cessation of workplace exposure (Tough and Court Brown, 1965). Somatic mutations as an endpoint of benzene-induced genotoxic effects in heavily exposed workers were studied recently by Rothman et al., 1995). They used the glycophorin A (GPA) mutation assay. The results suggested that benzene induces gene-duplicating mutations, presumably through recombination mechanisms, but not gene-inactivating mutations due to point mutations or deletions.

Tolbert and his associates (1991) found nuclear anomalies in buccal epithelial cells of snuff users. In another study they proposed that the nuclear anomalies were as common as micronucleus (Tolbert et al., 1992). The induction of karyorrhexis and 'nuclear anomalies' in colonic crypt cells has been correlated positively with the

induction of colonic tumors by chemical treatment (Duncan et al., 1985). Formation of nuclear anomalies like pyknotic and karyorrhectic nuclei in intestinal epithelial cells has been reported in rats intra-peritoneally injected with benzidine (Percy et al., 1989) and aromatic hydrocarbons (Blakey et al., 1985). Further, Subrahmanyam (1991) and his colleagues showed that benzene exposure in human and animals result in structural and numerical chromosomal aberration in lymphocytes and bone marrow cells. Analyses of micronucleus (MN) frequencies in peripheral lymphocytes by use of the cytokinesis-block technique among 49 traffic police revealed that MN frequency was significantly higher among the traffic police and frequency was found to increase with age, but no influence was observed for gender or smoking (Maffei, 2005).

DNA damage, assay technique:

Induction of DNA damage is an important initial event in the pathway of carcinogenesis. A considerable battery of assays exists for the detection of different genotoxic effects of compounds in experimental systems. Among these, single cell gel electrophoresis (SCGE) or Comet assay is technically simple, relatively fast, cheap, and sensitive method for detection of DNA strands breaks. The assay can be undertaken in virtually all mammalian cell types without requirement for cell culture. Detection of programmed cell death by apoptosis is also done by this method, as degradation of nuclear DNA by endonuclease enzymes is the hallmark of apoptosis.

In this method the cells are embedded in agarose and lysed, generating nucleus-like structures in the gel (referred to as nucleoids). Following alkaline electrophoresis, the DNA strands migrate toward the anode, and the extent of migration depends on the number of strand breaks. The migration is visualized and scored in a fluorescence microscope after staining. Although not all types of genotoxic exposures should be expected to result in DNA damage in mononuclear blood cells, the Comet assay seems to be a valuable tool for detection of genotoxic exposure in humans. The Comet assay has been successfully applied for detection of DNA damage in mammalian cells elicited by environmental exposures, including diet, exercise, hypoxia, and sunlight (Moller, 2005). Five of the metabolites of benzene *viz.* muconic acid, hydroquinone, catechol, p-benzoquinone and benzenetriol caused DNA damage *in vitro* in lymphocyte Comet assay (Anderson et al., 1995; Gaskell et al., 2005). Increase in DNA damage by cumulative benzene exposure has been confirmed *in vivo* in laboratory animals (Plappert et al., 1994; Tuo et al., 1996) and in human subjects employed in printing press (Joo et al., 2004, Sul et al., 2005). DNA strands break in liver cells has been recorded in benzene-exposed mice (Plappert et al., 1994; Vestergaard et al., 2002).

Materials and Methods

Measurement of t,t-MA in urine:

The concentration of t,t-MA, a benzene metabolite and a biomarker of benzene exposure, was measured in urine by HPLC-UV. Altogether, 54 samples from non-chewing and never-smoking males were measured: 24 samples from control subjects, 30 from urban subjects, who were occupationally exposed to city's vehicular pollution.

Measurement protocol

Urine (25-50 ml) was collected into 100- ml plastic screw-cap vials for analysis of t,t-MA. Samples were analyzed following the procedure of Ducos et al., (1990). In brief, the samples were protected from light, brought to the laboratory at Kolkata in ice buckets and stored at –20^{o}C until analysis. The samples were thawed and a 1- ml aliquot of each was passed through a Bond elute extraction cartridge filled with 500 mg of SAX sorbent preconditioned with 3 ml of methanol and 3 ml of distilled water. The cartridge was washed with 3 ml of 1% acetic acid solution. Then the t,t-MA was eluted with 3 ml of a 10% aqueous acetic acid solution, and measured in high performance liquid chromatography (HPLC, Waters, USA).

Ten microliter fractions of the elute were used per injection in column filled with LiChrosorb C18, 5µM (Waters), and the detector was set at 259 nm. The eluent was a solution of 1% aqueous acetic acid/methanol (90/10). With a flow rate of 1.2 ml /min, the retention time of t,t-MA was 10 min and the duration of an analytical run was 20 min. A stock solution of t,t-MA (100 mg/l, Sigma Chem, USA) was prepared in 10% acetic acid.

Micronucleus (MN) assay:

Collection of buccal mucosal and airway epithelial cells:

The subject was asked to wash his/her mouth with 0.9% saline (NaCl) water. Then the inner side of the cheeks was scrapped using a sterile spatula to obtain the buccal mucosa, which was then smeared on clean glass slides. Airway epithelial cells were obtained from spontaneously expectorated sputum samples. For each subject, at least 3 slides each for buccal and airway cells were made. Feulgen staining for DNA was done following the procedure of Pearse (1991). Parallel slides were fixed in Carnoy's fixative for 10 min (ethanol: chloroform: glacial acetic acid in the ratio 6: 3: 1, v/v).

Feulgen staining for DNA:

The fixed slides were rinsed 2-3 times in distilled water, incubated in 1N HCl at 37°C for 20 min and at 60°C for 12 min. Then the slides are stained in Schiff's reagent for 2 hours, rinsed 3 times in bisulfite solution, washed in distilled water, dehydrated in graded ethanol, cleared in xylene and mounted in DPX. DNA stained bright reddish purple or magenta. Using a light microscope, at least 1000 cells were counted at 400x and 1000x magnification and the total number of MN per 1000 cells was recorded.

Single cell gel electrophoresis (Comet assay) in peripheral blood lymphocytes:

Analysis of DNA damage in peripheral blood lymphocytes was done following the procedure of Singh et al., (1988) with the modification of Zhu et al., (1999).

Lymphocyte separation:

EDTA-anticoagulated whole blood was centrifuged for 10 min at 400 g at 20°C with Ficoll-Paque (Sigma Chem, USA) following the instruction of the manufacturer. This procedure separates a buffy coat layer of lymphocyte and platelet by the density gradient centrifugation technique. The buffy coat was pipetted out, collected in Eppendorf tubes and stored at –20^{o}C till further use.

Comet Assay:

Frosted microscopic slides were covered with 100 µl of 0.1% high melting agarose, immediately covered with a cover glass and kept at 4^{o}C for 5 min to solidify. Next, the cover glass was removed and 70 µl of 0.6% low melting agarose was added to the slide, covered with a cover glass and kept at 4^{o}C for another 5 min to solidify. Thereafter, 10 µl of the thawed lymphocytes was mixed with 90 µl of 0.8% low melting agarose to form a cell suspension. After gently removing the cover glass, 50 µl of this cell suspension was rapidly added onto the agarose layer, spread using a cover slip, and again kept cold for 10 min to solidify. Thereafter the cover glass was removed and 60 µl of 0.8% low melting agarose was added and kept at 4^{o}C for 10 min to solidify. Then, the cover glass was removed and the slides were immersed in freshly prepared lysing solution at 4°C for 1 hr. After lysis, the slides were washed with electrophoretic buffer and then placed on a horizontal electrophoresis tank filled with fresh electrophoresis solution to a level approximately 0.25 cm above the slides for 20 min to allow the unwinding of the DNA and expression of alkali labile damage before electrophoresis. To electrophorese the DNA an electric current of 25 V and 300 mA was applied for 30 min. All of these steps were conducted under dimmed light to prevent additional DNA damage. After electrophoresis, the slides were gently washed to neutralize excess alkali by placing the slides horizontally and flooding them with neutralization

buffer. After 5 min the cells were stained with 25 µl of ethidium bromide (Sigma Chem, USA), covered with cover slip, kept in a humidified box and analyzed within 48 hr using a fluorescence microscope (Nikon, Japan).

Statistical analysis:

All data are expressed as mean± standard deviation. The collected data were processed and analyzed in EPI info 6.0 and SPSS software.

Results

Concentration of t,t-MA in urine:

The concentration of t,t-MA, a benzene metabolite and a biomarker of benzene exposure, was measured in urine by HPLC-UV. The control male subjects (n=24) had a mean of 102± 38 µg/g creatinine of t,t-MA . In contrast, the male residents of Delhi with office jobs (n=16) had 218±102 µg/g creatinine t,t-MA in urine, indicating 2-times more benzene metabolites in their urine ($p<0.001$). Occupationally exposed subjects to vehicular emissions of Delhi such as auto rickshaw and taxi drivers (n=14) had 326± 117 µg/g creatinine of t,t-MA in urine, which was 1.5-times more than the office employees of Delhi and 3.2-times more than that of rural controls ($p<0.001$). Therefore, the citizens of Delhi in general, and the occupationally exposed subjects to Delhi's vehicular pollution in particular, were several times more exposed to benzene than the age-and sex-matched rural controls.

Elevated MN level in buccal epithelium among urban subjects of Delhi:

MN is rare under normal physiological conditions. In control subjects, a mean number of 1.34 ± 0.56 (SD) MN-containing cells per 1000 exfoliated buccal epithelial cells was found in non-smokers. Current smokers among controls had a significantly higher MN count (3.17 ± 1.74 micronucleated cells/1000 cells, $p<0.01$) than never smokers.

In Delhi, even the never-smokers had 3.03 ± 1.46 MN-containing cells per 1000 exfoliated buccal epithelial cells. The smokers had 4.17 ± 1.83 MN per 1000 cells. Thus, the residents of Delhi who had never smoked in their life had MN count closer to that of current smokers in control group. In general, Delhi's never-smokers had 2.3-times more MN than their control counterparts (3.03 vs. 1.34 MN /1000 cells, $p<0.001$, Plate 25).

Like smoking, tobacco chewing could enhance MN number, because genotoxic materials are present in chewing tobacco. To eliminate the effect of that confounder, the MN number of non-smoking, non-chewing individuals from control and urban group was compared. Non-smokers and non-chewers in control group had a mean MN count of 1.15 ± 0.43 per 1000 cells. In contrast, non-smokers and non-chewers residing in Delhi had 2.46 ±0.84 per 1000 cells, indicating more than 2-fold rise in MN number in the latter group. Smokers plus chewers of Delhi had 32% more MN than only smokers (Table 1) while only chewing increased the MN count by 19% (from 2.46 to 2.93/1000 cells), suggesting synergistic effect of chewing on MN formation in smokers.

Table 1. Micronucleated cell per 1000 exfoliated buccal epithelial cells.

Group	Control	Delhi	Change over control (%)
Smoker	3.17 ± 1.74	4.17 ± 1.83*	32
Chewer	1.76±0.69	2.93±1.68*	66
Smoker plus chewer	3.82±1.54	5.49±2.32*	44
Non-smoker plus non-chewer	1.15 ± 0.43	2.46 ±0.84*	114

**, $p<0.05$ compared with respective control value*

Increased MN count in exfoliated airway epithelial cells in sputum of the urban residents:

We have simultaneously examined the presence of MN in airway epithelial cells exfoliated in sputum. In control subjects who were non-smokers and non-chewers, 1.35 ± 0.73 (SD) MN-containing cells per 1000 exfoliated airway epithelial cells in sputum were recorded. Smokers had significant, 2.6-times more MN-containing airway cells, but chewers showed only modest 15% rise in MN count, showing a weak relationship between tobacco chewing and MN formation in airway epithelial cells, although the association was strong for MN development in the oral cavity where the lining cells are in direct contact with the tobacco products. In Delhi, the non-smokers and non-chewers had 2.92 ±1.24 MN-containing cells per 1000 exfoliated airway epithelial cells, which was 2.2-times higher ($p<0.05$) than their control counterparts (Table 2). Delhi's smokers had 1.9-times more MN in airway cells, while chewers showed 21% rise in MN per number. Smokers plus chewers had 13% more MN than only smokers, suggesting a small additive effect of chewing on MN formation in airway epithelial cells of smokers.

Table 2. Micronucleated cell per 1000 exfoliated airway epithelial cells.

Group	Control	Delhi	Change (%)
Smoker	3.57±1.59	5.47±2.20*	53
Chewer	1.56±0.76	3.53±1.78*	126
Smoker plus chewer	3.77±1.29	6.19±2.32*	64
Non-smoker plus non-chewer	1.35±0.73	2.92±1.24*	116

**, $p<0.05$ compared with respective control value*

DNA damage in lymphocytes:

The frequency and extent of damage in DNA of peripheral blood lymphocytes was examined by Comet assay in 12 control and 15 Delhi's residents who were non-chewer and never smokers. Compared to control subjects, significantly higher ($p<0.05$) frequency of DNA damage was recorded in lymphocytes of the residents of Delhi. In citizens of Delhi 33.8% lymphocytes had damaged DNA against 18.5% of controls ($p<0.05$; Table 48). The tail length of Comet, an indicator of the extent of DNA damage, was 2.8±0.2 (SD) µm in Delhi compared with 1.7±0.2 µm in control subjects ($p<0.05$); (Table 3). Therefore, MN and Comet assays showed significantly increased genotoxicity among the residents of Delhi, as compared with rural controls.

Table 3. Assessment of DNA damage by COMET Assay in peripheral blood lymphocytes.

	n	Cells with damaged DNA (%)	Average tail length (µm)
Delhi	15	33.8±7.3	2.8 ± 0.6
Control	12	18.5±4.3*	1.7± 0.3*

*Results are mean± SD; *, $p<0.05$ compared with rural*

Association between air pollution exposure and MN formation:

Association with RSPM:

In order to investigate whether the observed high MN count in buccal and airway epithelial cells among the residents of Delhi were due to higher level of air pollution in city, we have analyzed the data by Spearman's rank correlation study. RSPM level of the city was positively correlated with MN formation in buccal (rho=0.40, $p<0.01$) as well as airway epithelial cells (rho=0.44,

p<0.01, Table 4), and DNA damage in lymphocytes (rho= 0.37, p<0.05).

Correlation of MN formation with ambient benzene and benzo(a)pyrene [B(a)P] levels:

A strong correlation (rho=0.77, p<0.001) was found between benzene level in ambient air and MN frequency in buccal and airway epithelial cells, cells on direct line of contact with inhaled pollutants. B(a)P concentration in breathing air of Delhi also showed a significant, positive correlation with MN in buccal and especially airway cells. But the strength of this correlation was weaker than elicited by benzene (rho=0.33 and 0.41 for buccal and airway cells, p<0.05; Table 4).

Table 4. Spearman's rank correlation between air pollution levels and micronucleus formation.

Variable	Correlation (rho value)	P value
RSPM and MN in buccal cells	0.40	<0.01
RSPM and MN in airway cell	0.44	<0.01
Benzene and MN in buccal cell	0.77	<0.001
Benzene and MN in airway cell	0.77	<0.001
B(a)P and MN in buccal cell	0.33	<0.05
B(a)P and MN in airway cell	0.41	<0.05

In essence, it is apparent that the greater prevalence of genotoxicity in buccal and airway epithelial cells, and peripheral blood lymphocytes of the residents of Delhi could be attributed, at least in part, to city's air pollution level with special reference to benzene, benzo(a)pyrene, which usually enters the body being adsorbed on the surface of RSPM.

Discussion

The present study provides compelling evidence that chronic exposure to urban air pollution in Delhi is associated with significantly elevated genotoxic damage in exposed individuals. Using a combination of exposure biomarkers, cytogenetic endpoints, and DNA damage assays, we demonstrate that residents of Delhi especially those occupationally exposed to vehicular emissions experience substantially higher genetic insult compared to age- and sex-matched rural controls. These findings reinforce concerns regarding the long-term health implications of sustained exposure to complex urban air pollutant mixtures.

Benzene is a well-established human carcinogen and clastogen, and vehicular emissions represent a major source of ambient benzene in urban environments. The significantly elevated levels of urinary t,t-MA observed among Delhi residents indicate increased internal benzene burden. Occupationally exposed individuals such as auto-rickshaw and taxi drivers exhibited the highest t,t-MA concentrations, reflecting cumulative exposure from prolonged time spent in traffic-dense environments. These results are consistent with earlier reports demonstrating elevated benzene metabolites among traffic-exposed populations and urban residents.

Genotoxic effects were evaluated at the chromosomal and DNA levels using the micronucleus assay and Comet assay, respectively. The MN assay in exfoliated buccal epithelial cells revealed more than a two-fold increase in MN frequency among Delhi residents, even in never-smokers and non-chewers. This finding is particularly important, as it indicates that air pollution alone—independent of tobacco-related confounders—can induce chromosomal damage in epithelial cells directly exposed to inhaled pollutants. Buccal cells serve as a relevant target tissue because they represent the first line of contact for airborne contaminants entering through the nasal–oropharyngeal route.

A similar pattern was observed in exfoliated airway epithelial cells obtained from sputum samples. The elevated MN frequency in airway cells among urban residents suggests sustained genotoxic stress within the lower respiratory tract. Unlike buccal cells, airway epithelial cells showed a weaker association with tobacco chewing, reinforcing the role of inhaled pollutants rather than oral exposure pathways. This distinction highlights tissue-specific vulnerability and supports the use of airway epithelial cells as sensitive indicators of inhalation-mediated genotoxicity.

The Comet assay further confirmed systemic DNA damage in peripheral blood lymphocytes of Delhi residents. Both the proportion of lymphocytes with damaged DNA and the average tail length were significantly higher among urban subjects compared to controls. Since lymphocytes circulate throughout the body, DNA damage in these cells reflects systemic exposure and potential long-term carcinogenic risk. The observed increase in DNA strand breaks may result from oxidative stress generated by reactive oxygen species present in polluted air or produced endogenously following pollutant metabolism.

Importantly, correlation analyses revealed strong positive associations between ambient air pollution indices and genotoxic endpoints. Respirable suspended particulate matter (RSPM) showed significant correlations with MN formation in both buccal and airway epithelial cells, as well as DNA damage in lymphocytes. Particulate matter acts as a carrier for toxic organic compounds, including benzene and benzo(a)pyrene, facilitating their deposition in the respiratory tract. The particularly strong correlation between ambient benzene levels and MN frequency underscores the central role of benzene in mediating genotoxic effects in urban environments.

Benzo(a)pyrene, a polycyclic aromatic hydrocarbon, also exhibited significant but comparatively weaker correlations with MN formation. This difference may reflect variations in exposure levels, metabolic activation pathways, or cellular uptake mechanisms. Nevertheless, the combined presence of benzene, polycyclic aromatic hydrocarbons, and particulate matter likely contributes synergistically to the overall genotoxic burden experienced by urban populations.

The findings of this study are consistent with previous reports documenting increased chromosomal aberrations, micronuclei formation, and DNA damage among individuals exposed to benzene and traffic-related air pollution. Notably, the persistence of chromosomal damage long after cessation of benzene exposure reported in earlier studies suggests that the observed genetic alterations may have long-term health consequences. Elevated MN frequency is widely recognized as a predictive biomarker for cancer risk, lending further significance to the present results.

While smoking and tobacco chewing are known genotoxic factors, careful stratification of subjects in this study allowed us to minimize their confounding effects. The persistence of significantly elevated genotoxic markers among non-smokers and non-chewers residing in Delhi strengthens the conclusion that ambient air pollution is a primary contributor to the observed genetic damage.

In conclusion, this study demonstrates that chronic exposure to Delhi's urban air pollution—particularly benzene-rich vehicular emissions—results in significant genotoxic damage at cellular and molecular levels. The integration of exposure biomarkers with cytogenetic and DNA damage assays provides robust evidence of pollution-induced genetic injury. These findings emphasize the urgent need for stricter emission controls, improved urban air quality management, and regular bio-monitoring of at-risk populations to mitigate long-term health risks.

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