1.1 INTRODUCTION

The World Health Organization reports that 4.6 million people die each year from causes directly attributable to air pollution and in Europe alone 310,000 have been reported to air pollution than to automobile accidents (Sunyer 2001). Direct causes of air pollution related deaths include aggravated asthma, bronchitis, emphysema, lung and heart diseases, and respiratory allergies. The US EPA estimates that a proposed set of changes in diesel engine technology could result in 12,000 fewer premature mortalities, 15,000 fewer heart attacks, 6,000 fewer emergency room visits by children with asthma and 8,900 fewer respiratory-related hospital admissions each year in the United States.

The worst short term civilian pollution crisis in India was the 1984 Bhopal Disaster. Industrial vapours leaked from the Union Carbide factory, belonging to Union Carbide, Inc., U.S.A., killed more than 2,000 people outright and injured from 150,000 to 600,000 people, some 6,000 of whom would later die from their injuries (Bani et al., 1990; Goswami et al., 1984). The United Kingdom suffered its worst air pollution event when the December 4th Great Smog of 1952 formed over London. More than 4000 died in six days and 8,000 more died within the following months. An accidental leak of anthrax spores from a biological warfare laboratory in the
former USSR in 1979 near Sverdlovsk is believed to have been the cause of hundreds of civilian deaths. The worst single incident of air pollution to occur in the United States of America occurred in Donora, Pennsylvania in late October, 1948, when 20 people died and over 7,000 were injured.

The health effects caused by air pollutants may range from subtle biochemical and physiological changes to difficulty in breathing, wheezing, coughing and aggravation of existing respiratory and cardiac conditions. These effects can result in increased medication use, increased doctor or emergency room visits, more hospital admissions and premature death. The human health effects of poor air quality are far reaching but principally affect the body's respiratory system and the cardiovascular system (Soban et al., 2005). Individual reactions to air pollutants depend on the type of pollutant a person is exposed to, the degree of exposure, the individual's health status and genetics. People who exercise outdoors, for example, on hot, smoggy days increase their exposure to pollutants in the air.

1.2. AIR POLLUTION

Air Pollution is a chemical, physical (e.g. particulate matter) or biological agent that modifies the natural characteristics of the atmosphere. The atmosphere is a complex, dynamic and natural gaseous system that is essential to support life on planet Earth. Stratospheric ozone depletion due to air pollution has long been recognized as a threat to human health as well
as to the Earth's ecosystems. Worldwide air pollution is responsible for large number of deaths and cases of respiratory disease. Enforced air quality standards like the Clean Air Act in the United States have reduced the presence of some pollutants. While major stationary sources are often identified with air pollution, the greatest source of emissions is actually made up by mobile sources, mainly the automobiles. Gases such as carbon dioxide, which contribute to global warming, have recently gained recognition as pollutants, while some recognize this gas as being essential to life and therefore, incapable of being classed as a pollutant.

1.3. AIR POLLUTANTS

There are many substances in the air which may impair the health of plants and animals (including humans) or reduce visibility. These arise both from natural processes and human activity. Substances not naturally found in the air or at greater concentrations or in different locations from usual are referred to as 'pollutants'. Among the variety of air pollutants such as suspended particulate matter (SPM), sulphur dioxide (SO₂) and oxides of nitrogen (NOₓ) are considered to be major pollutants in India (Ravichandran et al., 1996).

Pollutants can be classified as either primary or secondary. Primary pollutants are substances directly produced by a process, such as ash from a volcanic eruption or the carbon monoxide gas from a motor vehicle exhaust.
Secondary pollutants are not emitted. Rather, they are created in the air when primary pollutants react or interact. An important example of a secondary pollutant is ground level ozone - one of the many secondary pollutants that make up photochemical smog.

While some pollutants may be both primary and secondary: that is, they are both emitted directly and formed from other primary pollutants.

Primary pollutants produced by human activity include:

- oxides of sulfur, nitrogen and carbon
- organic compounds, such as hydrocarbons (fuel vapours and solvents)
- particulate matter, such as smoke and dust
- metal oxides, especially those of lead, cadmium, copper and iron
- chlorofluorocarbons (CFCs)
- hazardous air pollutants (HAP)
- persistent organic pollutants (POPs)
- odours

Secondary pollutants include some particles formed from gaseous primary pollutants and compounds in photochemical smog, such as nitrogen dioxide, ground level ozone and peroxyacetyl nitrate (PAN).
1.4. SOURCES OF AIR POLLUTION

Anthropogenic sources (human activity) related to burning different kinds of fuel:

- Combustion-fired power plants
- Controlled burn practices used in agriculture and forestry management
- Motor vehicles generating air pollution emissions.
- Marine vessels, such as container ships or cruise ships and related port air pollution.
- Burning wood, fireplaces, stoves, furnaces and incinerators

Other anthropogenic sources

- Oil refining, power plant operation and industrial activity in general.
- Chemicals, dust and crop waste burning in farming.
- Fumes from paint, hair spray, varnish, aerosol sprays and other solvents.
- Waste deposition in landfills, which generate methane.
- Military uses such as nuclear weapons, toxic gases, germ warfare and rocketry.

Natural sources

- Dust from natural sources, usually large areas of land with little or no vegetation.
- Methane, emitted by the digestion of food by animals, for example cattle.
- Radon gas from radioactive decay within the Earth's crust.
- Smoke and carbon monoxide from wildfires.
- Volcanic activity, which produce sulfur, chlorine, and ash particulates.

1.5. REDUCTION EFFORTS

There are many air pollution control technologies and urban planning strategies available to reduce air pollution enforced air quality standards, like the Clean Air Act in the United States, have reduced the presence of some pollutants. Many countries have programs for debating how to reduce dependence on fossil fuels for energy production and shift toward renewable energy technologies or nuclear power plants (Obe and Slacik-erben 1973).

Efforts to reduce pollution from mobile sources includes primary regulation (many developing countries have permissive regulations), expanding regulation to new sources (such as cruise and transport ships, farm equipment, and small gas-powered equipment such as lawn trimmers, chainsaws, and snowmobiles), increased fuel efficiency (such as through the use of hybrid vehicles), conversion to cleaner fuels (such as bioethanol, biodiesel), or conversion to electric vehicles with renewable energy sources (batteries or clean fuel such as hydrogen being used for transport and storage).
1.6. TRAFFIC POLICEMEN- WORLD SCENARIO

Automobiles are necessary evil. On one hand they have made living easy and convenient but on the other hand they have also made human life more complicated and vulnerable to both toxic emission and increased risk of accidents. Urban people are the most affected and among the worst suffers are traffic policemen who are particularly close to the automobile exhaust.

The automobiles are an important source of not only air pollution but also of a significant proportion of noise pollution. The traffic police engaged in controlling traffic, particularly at heavy traffic junctions, belong to the high-risk group to be affected by the health hazards of noise and air pollution (Sun et al., 1995., Albertini et al., 2000., Fenech 2002., Keshava and Ong 1999 ; Deknudt et al., 1973). Because the irritation of upper respiratory tract resulting in cough is a somewhat acute phenomenon, most of the traffic policemen use a mask to prevent the ill-effects of air pollution. However, a majority of them remain unaware about the health effects of noise on their hearing ability as this is an insidious process and takes long time to become overt.

Health effects of noise include both the auditory as well as non-auditory effects. Research has been carried out to study these effects in different categories of population exposed to high intensity and frequencies
of sound in their workplaces (Galloway et al., 1986, Klemans et al., 1995, Lakhanisky et al., 1993, Ingle et al., 2005; Shalinder Singh Kamboj and Vasudha Sambyal 2006). However, the auditory effects of noise generated by automobiles among the traffic policemen have never been explored, particularly in India. This may be one of the reasons for not providing hearing protection devices to this group of work force. However, the need should be felt by the traffic policemen themselves and this can happen only when they have adequate knowledge about the associated health hazards.

Despite the heavy pollution, many policemen prefer to work on the roadside rather than in the criminal sector. Duration of life, progressive disease, and increased susceptibility to acute effects are linked to air pollution. In several studies, investigators have evaluated associations between community ambient air particulate matter and chronic respiratory symptoms in adults.

1.7. TRAFFIC POLICEMEN - INDIAN SCENARIO

The effect of air pollution include breathing and respiratory problems, aggravation of existing respiratory and cardiovascular disease, alteration in body defense systems against foreign materials, damage to lung tissue, carcinogenesis and premature death (WHO, 1995a,b and Cotes, 1978). Of the three million premature deaths in the world due to outdoor and indoor air pollution, the highest number are assessed to occur in India.
The adverse health effects of air pollution depend on the level of exposure, nutritional status, population structure, genetic susceptibility and personal habits. Greater the exposure burden, including smoking and poorer the nutritional status, higher the health risks.

The number of vehicles in Delhi has jumped tenfold in the past two decades to reach 35 lakhs in 2000. Coupled with high density passenger and goods transportation, these vehicles spewing pollutants into Delhi's environment have given the Capital the dubious distinction of being one of the most polluted cities of the world. Vehicle emissions are estimated to account for 60 to 70 per cent of total air pollution. What makes this pollution worse is that it is generated at the ground or breathing level, affecting the commuter to an extent, but more harmfully the traffic policemen posted at intersections.

A local survey has indicated that the incidence of respiratory diseases in Delhi is 12 times the national average and 30 per cent of Delhi's population suffers from respiratory disorders due to air pollution. However, the city's environment has started showing some improvement after the Supreme Court ordered closure, shifting of polluting industries and introduction of CNG- run public transport.

Recognizing the gravity of air pollution and its impact on traffic policemen, Delhi Traffic Police had pioneered three studies in the early
'90s. The first, in collaboration with the Central Road Research Institute (CRRI) and the All India Institute of Medical Sciences (AIIMS), studied the effects of pollution on traffic policemen performing duties at traffic intersections.

The second followed up in association with the Patel Chest Institute of Delhi University with a continual programme till 1997 for diagnosis and treatment of traffic policemen's lungs and other respiratory diseases. Then in the third, the local Majidia Hospital in 1999 volunteered to provide free treatment to traffic policemen affected with such ailments.

All the three studies revealed that traffic policemen working in adverse environmental conditions are affected; both in the short and long run their health and efficiency are impacted. Eye irritations, throat infections, respiratory discomfort, skin ailments, impaired hearing, chest diseases, excessive Carboxy haemoglobin and annoyance with noise are some of the ailments they suffer from (Soban et al., 2005).

The CRRI-AIIMS study recommended better and special medical care for traffic policemen on active duty; various duty places for them need to be scientifically evaluated for their exposure risks; duty hours at critical points be curtailed and a case for appropriate hazard allowance to traffic policemen be considered. On the basis of this study, pollution masks were given to traffic police on duty at higher polluted junctions. An awareness
programme was also conducted to sensitize them about pollution effects on their health. Simultaneously, a research programme was undertaken at the Patel Chest Institute to provide free treatment and continually check the health of traffic police. Toxic allowance for traffic policemen from the rank of constable to inspector was recommended, but did not find favour with the Fifth Pay Commission. To decrease their exposure to high levels of pollution, a posting policy was evolved on the basis of three categories: Highly congested places in Delhi and New Delhi; places less congested and outer areas which are least congested. Traffic personnel are constantly rotated in these areas every six months so that they do not remain exposed to pollution for long periods. Similarly, the point of duty within his circle is also changed every fortnight. In addition eco-friendly solar traffic booths are installed at traffic intersections in Delhi and New Delhi areas. These booths contain ionizers which suppress the suspended particulate matter and provide a healthy environment for a policeman seated inside.

The outcome of the studies by AIIMS and CRRI and the remedial measures taken by doctors of the Patel Chest Institute in the '90s, the Delhi Traffic Police and a group of physicians of BBCI is educate traffic police in better self-management of chest ailments. The policemen were addressed on respiratory system and lung function tests and given a demonstration on emergency care, first aid, etc., while on the road. The various studies among Indian population cohorts include policemen working in Jalagon city along
NH-6 in North India, Amristar city in Punjab and several other related studies. The main outcome of these studies were that these people had several respiratory problems, that were mild, moderate to severe levels. Thus safety protection equipments for such field works were recommended (Shalinder Singh Kamboj and Vasudha Sambyal 2006., Joshi et al., 1975., Mishra and Retherford, 2007., IAEA 1999., Chandrasekaran et al., 1996; Moorhead et al., 1960).

1.8. AIR POLLUTION AND BIOMARKERS

Since the late 1960s, there has been a formal interest and research in the development and use of biomarkers and bioindicators of air pollution. The development and use of markers and indicators resulted from a political prohibition of being able to study the effects of air pollutants on human health. Central Europe from the 1950s through the 1980s was the scene of acute and chronic air pollution from industrialization. Researchers from different institutes and universities conducted extensive studies on biomarkers and bioindicators of air pollution such as chromosome aberration, sister chromatid exchange and micronuclei (Holland et al., 2000). Biomarkers included such responses as changes in the chemical composition of lens and changes in the blood chemistry of wild hares.

Urban air pollution consists of a complex mixture of organic and inorganic compounds, many of which are genotoxic and potentially
carcinogenic. Hemminki and Pershagen (1994) suggested an association between high levels of urban air pollution and increased risk of lung cancer and the yearly excess of lung cancer due to air pollution in Western industrialized countries has been estimated to range between 30 and 150 cases per million people.

Certain occupational groups, such as professional drivers working in urban areas are exposed to a high level of ambient air pollution (Guillemin et al., 1992). An increased risk of several types of cancer, including that of the lungs and bladder, was observed among men working in urban, suburban, and interurban passenger transport and in trucking freight (Silverman et al., 1986., Balarajan and McDowall 1988., Hayes et al., 1989., Guberan et al., 1992., Steenland et al., 1992., Barbone et al., 1995., Jakobsson et al., 1997; Soll-Johanning et al., 1998). In addition, truck drivers and railroad workers showed increased mortality due to cerebrovascular diseases and arteriosclerosis, and it was suggested that exposure to diesel exhaust played a contributing role (Hansen 1990).

In epidemiological studies, detailed information on exposure is usually scarce, and because several confounding factors exist, the actual contribution of ambient air pollution as a risk factor of cancer is difficult to assess. Biomarkers such as adducts in DNA and proteins and cytogenetic alterations may help in identifying the exposure and revealing its early effects (Perera et al., 1992., Autrup et al., 1995; Nielsen et al., 1996). In this
context, structural chromosomal aberrations are of special interest, because high chromosomal aberration levels in peripheral lymphocytes have been associated with an increased risk of cancer (Hagmar et al., 1994; Bonassi et al., 1995). The exhaust from diesel engines is a major contributor to the mutagenicity of urban air (Stevens et al., 1990) and induces cytogenetic damage in both mammalian cells in vitro and rodents in vivo (IARC 1989b), exposure to urban air polluted with diesel exhaust may also increase the level of chromosome damage in humans. Egyptian traffic policemen showed an increased frequency of chromosomal aberrations and SCEs in their peripheral lymphocytes (Anwar and Kamal 1988; Hayes et al., 1989). Policemen from the city of Genoa demonstrated exposure to benzo(a)pyrene, but no effect on SCEs or micronuclei in peripheral lymphocytes was observed (Bolognesi et al., 1997a; Bolognesi et al., 1997b). No association between occupational exposure to diesel exhaust and chromosomal aberration frequencies could be seen in two Swedish studies (Nordensson et al., 1981; Fredga et al., 1982) or in a Welsh study (Parry et al., 1997).

Several studies have found associations between increased cancer risk and impaired DNA repair (Bohr et al., 1989; Pero et al., 1983). Therefore, individual differences in DNA repair capacity may generally influence the rate of genotoxic damage and cancer susceptibility from exposure to urban air pollution. Another potential source of individual
susceptibility is the polymorphism of xenobiotic-metabolizing enzymes. A large number of chemical carcinogens present in ambient air are metabolized to the ultimate carcinogenic form or detoxified to less toxic or nontoxic metabolites that are excreted in the urine. These reactions are primarily mediated by members of the cytochrome P-450 family of enzymes and by conjugating enzymes such as glutathione S-transferases and N-acetyltransferases. High rates of metabolic activation and defective detoxification have been associated with an increased risk for certain types of cancer, with one example being lung cancer in individuals who lack GSTM1 activity due to a homozygous allelic loss of the Glutathione S-transferase M1 (GSTM1) gene (McWilliams et al., 1995).

In a Danish National research program to evaluate genetic biomarkers to assess exposure to the genotoxic compounds present in urban air, bus drivers and postal workers in Copenhagen were selected as the study population. To exclude contribution by active smoking, only nonsmokers were chosen. City bus drivers, in comparison with rural and suburban bus drivers were observed to have elevated levels of bulky DNA adducts in their mononuclear WBCs (Nielsen et al., 1996). An analysis of air pollution from personal air samplers demonstrated that bus drivers were exposed up to three times higher levels of naphthalene than mail carriers (Hansen et al., 1998; Hemminki et al., 1990; Sram et al., 1996). A number of different biomarkers of genotoxicity have been applied to assess human
exposure to the genotoxic compounds present in ambient air. Increased levels of chromosomal aberrations and SCEs have been demonstrated in studies of populations exposed to heavy air pollution, e.g., in Poland and in the Czech Republic (Nielsen et al., 1996., Anwar et al., 1988., Chandrasekaran et al., 1996., Bolognesi et al., 1997a., Bolognesi et al., 1997b; Nordensson et al., 1981).

In the present study, chromosomal aberrations were used to assess genotoxic effects in bus drivers exposed to a high level of urban air pollution. Besides bus drivers, we also studied postal workers who are expected to be exposed to lower air pollution levels than bus drivers. All subjects were nonsmokers to exclude the effects of this common confounder that is known to affect chromosome aberration rates. Some of the individuals studied were ex-smokers, but no significant effects of previous smoking was observed although some of the previous data have suggested that the chromosome-damaging effect of smoking can persist for several years (Valjus et al., 1994).

The group of bus drivers studied had been previously examined for aromatic DNA adducts in peripheral mononuclear cells; the results showed a clear exposure, with a 15-fold increase in the level of DNA adducts in comparison with a rural control group (Nielsen et al., 1996). The findings suggest that the bus drivers have experienced genotoxic exposure, compared with the postal workers.
During the last 20 years, several non-positive and positive studies on the association between exposure to diesel exhaust and cytogenetic damage have been reported (Anwar et al., 1988, Chandrasekaran et al., 1996, Bolognesi et al., 1997a, Bolognesi et al., 1997b, Nordensson et al., 1981, Fredga et al., 1982; Parry et al., 1997), all of which included both smokers and nonsmokers and relatively small numbers of subjects.

1.9. CYTOGENETICS

Cytogenetics is the study of the structure of chromosome material. With the advent of harvest procedures which allowed easy enumeration of chromosomes, discoveries were quickly made in abnormalities arising from nondysjunction events which cause cells with aneusomy (additions or deletions of entire chromosomes).

Initially the work on cytogenetics have started sometime during early 1940’s with the studies of Karl Sax on the induction of chromosomal aberration in Tradescantia microspores by X-rays and later it has been applied in the study of human chromosomes. Generally, the cytogenetic techniques are useful to estimate radiation doses received due to external radiation exposure. It is difficult to estimate radiation doses except for certain radio-nuclides like tritium. The entire techniques are referred in IAEA publication (Perry et al., 1974).
Dicentric (DC) chromosome assay is widely used to measure the accidental radiation exposure. In this method the blood lymphocytes are stimulated to enter the cell cycle by PHA-M (mitogen) and then the cell cycle is arrested when the cells are at metaphase stage by the addition of colcemid, a spindle poison at 44 hours. This stage helps to visualize the chromosome as distinct X-shaped entities. The number of dicentric chromosomes formed due to pollution exposure are scored using the microscope. Since the frequency of DC chromosome follow Poisson distribution this technique can be used to differentiate whole and partial body exposures.

Micronucleus (MN) technique is gaining importance in the field of biodosimetry in recent time. Acentric fragments of chromosomes formed during exposure to radiation fail to get incorporated into daughter nuclei during mitosis, due to lack of centromere, develop into MN. The technique, developed by Fenech and Morley (1985), allows scoring of MN in cells that have completed their first division, by blocking the cells at cytokinesis stage. The procedure is similar to that followed for DC chromosome assay except that in place of colcemid, Cytochalasin – B is added at 44 hours of culture to arrest cells at cytokinesis stage. The presence of MN in the cytokinesis-blocked cells is scored under microscope. From the frequency of MN the dose is estimated using a dose-response curve. The advantage of this technique is scoring of MN is easy and fast compared to that of DC.
Since the distribution of MN follows overdispersion in whole and partial body exposure, it cannot be used to differentiate whole and partial body exposure.

1.10. DNA LESIONS AND REPAIR

The damage at the DNA level manifests to gross structural changes at the chromosomal level it is important to understand the various DNA lesions induced by radiation. The lesions induced in DNA are single- (ssb) and double- (dsb) breaks, base damage (bd) of different types like DNA-DNA and DNA– protein cross links, alkylation, phosphotriesters, radical formations, intercalations, formation of bulky adducts, pyrimidine dimers, apurinic and apyrimidinic sites. Though DNA lesions lead to form structural chromosomal aberrations, most of them get repaired and do not lead to gross structural changes in chromosomes.

It has been shown that the number of single strand and double strand breaks are nearly 1000 and 50 times higher than chromosomal aberrations induced by a given dose respectively. It has also been shown that with low Linear Energy Transfur (LET) gamma radiation, the induction of dsbs, ssbs and bds follow a ratio of 40:1500:3000 (Brogger, 1982). Although it has been said that DNA lesions lead to chromosomal aberrations, it is the dsbs that play directly in the formation of chromosomal aberration. Two such lesions are necessary for the formation of an exchange aberration. However,
DNA damage can be repaired to give an apparently normal chromosome. Alternatively, it can be misrepaired to form an exchange or remain unrepaired, resulting in a deletion.

1.11. REPAIR MECHANISMS

It has been estimated that every hour, human and other mammalian cells undergo at least 50 to 100 times as much spontaneous DNA damage as would result from exposure to 10 mGy of ionising radiation, that occurs in the natural background of the earth. The fact that normal human being who are invariably exposed to spontaneous DNA damage do not suffer from its effects suggest that all organisms are equipped with a wonderful network of DNA repair mechanisms which faithfully and promptly repair and restore the damage of DNA to its original form (Preston et al., 1989). However such repair mechanism could not be seen in repair deficient diseases like Xeroderma pigmentosum, Ataxia telangiectasia and Retinoblastoma (Scott et al., 1983., IAEA 1986 ; Carrano and Natarajan 1988). Based on the nature of DNA damage different repair mechanisms such as direct, excision and recombinational appear to be activated.

1.12. CHROMOSOMAL ABERRATIONS AND CANCER

Chromosome rearrangements occurring over a period of time due to the exposure to various mutagens from environment and life style are known to cause cancer. The relationship between specific chromosomal
rearrangements and cancer has been shown in a number of neoplastic tissues using cytogenetic analysis. These studies show translocation, deletion, gain or loss of chromosomes in tumour cells. The karyotype of various tumours has been reported in recent years. However, in patients with Bloom’s syndrome, Fanconi’s anaemia and Ataxia telangiectasia, the frequency of spontaneous chromosomal aberrations such as chromatid breaks (ChB), gaps, dicentrics (DC) and sister chromatid exchanges (SCE) were shown to be higher than those of normal healthy person in peripheral blood lymphocytes. Similarly, higher incidence of breaks and SCE were reported in childhood leukaemia and in chronic myeloid leukaemia (CML) patients. Antoine et al., (1981) have reported an increased DC frequency in the peripheral blood lymphocytes of cervical cancer patients. Honeycomb (1981) shown excess acentrics and chromatid breaks in patients with chronic myeloid leukaemia. Aronson et al. (1982) also observed excess acentrics and chromatid breaks in blood of children with leukaemia. Venkatachalam et al., (1998) have shown an increased frequency of DC chromosomes and MN in blood samples of cancer patients compared to those obtained in normal subjects. On the contrary Diener et al., (1988) and Kleinerman et al., (1989) did not find any increase in chromosomal aberrations in peripheral blood of cervical cancer patients and in Morbus Hodgkin’s disease patients.
1.13. AIR QUALITY ASSESSMENT IN INDIA

Central Pollution Control Board is executing a Nation-wide National Air Quality Monitoring Programme (NAMP). The network consist of 332 operating stations covering 121 cities/towns in 25 States and 4 Union Territories of the country. The objectives of the NAMP are to determine status and trends of ambient air quality; to ascertain whether the prescribed ambient air quality standards are violated; to Identify Nonattainment Cities; to obtain the knowledge and understanding necessary for developing preventive and corrective measures; to understand the natural cleansing process undergoing in the environment through pollution dilution, dispersion, wind based movement, dry deposition, precipitation and chemical transformation of pollutants generated.

Under NAMP, four air pollutants viz., sulphur dioxide (SO$_2$), oxides of nitrogen as NO$_2$ Suspended Particulate Matter (SPM) and Respirable Suspended Particulate Matter (RSPM/PM10) have been identified for regular monitoring at all the locations (Table 1.3). The monitoring of meteorological parameters such as wind speed and direction, relative humidity and temperature have also been integrated with the monitoring of air quality. The monitoring of pollutants are carried out for 24 hours (4-hourly sampling for gaseous pollutants and 8-hourly sampling for particulate matter) with frequency of twice a week, to have 104 observations in a year. The monitoring is being carried out with the help of
Central Pollution Control Board; Zonal Office; State Pollution Control Boards: Pollution Control Committees and National Environmental Engineering Research Institute (NEERI), Nagpur.

The air quality of different cities/towns has been compared with the respective National Ambient Air Quality Standard (NAAQS). The air quality has been categorized into four broad categories based on an Exceedence Factor (the ratio of annual mean concentration of a pollutant with that of a respective standard).

The four air quality categories are:

- Critical pollution (C) when Exceedance Factor is more than 1.5;
- High pollution (H) when the Exceedance Factor is between 1.0 - 1.5;
- Moderate pollution (M) when the Exceedance Factor is between 0.5 - 1.0; and
- Low pollution (L) when the Exceedance Factor is less than 0.5.

1.14 AMBIENT AIR QUALITY STATUS IN TAMIL NADU

Air quality is being monitored by the Board to assess the concentration of air pollutants arising out of emissions from industries as well as increasing vehicular population. In Chennai alone, ambient air quality is being monitored at three locations under National Air Quality Monitoring Programme (NAMP) and at five locations under Chennai Ambient Air Quality Monitoring Programme (CAAQM). Apart from Chennai, monitoring of ambient air quality is carried out at Thoothukudi,
Coimbatore, Madurai, Salem and Tiruchirapalli. Besides, six continuous ambient air quality monitoring stations are established at Cuddalore, Tuticorin, Ranipet, Manali-Chennai, Royapuram- Chennai, Kottivakkam-Chennai to evaluate the levels of pollution (Table 1.1 and 1.2). There has been a rapid increase in the number of vehicles, as a result of urbanization, economic growth and easy availability of finance. Apart from new vehicles, old vehicles also exist often with outdated technology and nonobservance of emission norms. The quality of fuel supplied has also compounded the problem of vehicular pollution. In this regard, high octane and unleaded fuels are to be used by the vehicle owners. The European Union emission norms are a bench mark to measure and regulate vehicular pollution. The Board is monitoring vehicular emission since 1992. In Chennai city, three vehicular monitoring stations located at Alandur, Ambattur and Vyasarpadi conduct monitoring of the vehicular emission from goods and transport vehicles on a continuous basis. In addition to this, vehicular emission is being monitored at Dindigul, Palani, Ooty, Chengalpattu and Katteri. The Board tested 51398 vehicles in 2003-04. It is found that 13,206 vehicles exceeded the threshold limit during the first test. After rectification of defects, 1666 vehicles did not satisfy the emission standards. In order to reduce emission from vehicles cleaner fuels like unleaded petrol, 3 per cent benzene level and low sulphur fuel have been introduced in Chennai. Auto manufacturers are also incorporating technological changes towards this end.
### Table -1.1 Summary of Ambient Air Quality in Tamil Nadu

<table>
<thead>
<tr>
<th>NATURE OF POLLUTANT</th>
<th>SO2</th>
<th>NO2</th>
<th>RSPM</th>
<th>SPM</th>
</tr>
</thead>
<tbody>
<tr>
<td>NATURE OF THE AREA</td>
<td>I</td>
<td>R</td>
<td>I</td>
<td>R</td>
</tr>
<tr>
<td>CHENNAI</td>
<td>L</td>
<td>L</td>
<td>L</td>
<td>M</td>
</tr>
<tr>
<td>COIMBATORE</td>
<td>L</td>
<td>L</td>
<td>M</td>
<td>M</td>
</tr>
<tr>
<td>MUDURAIRE</td>
<td>L</td>
<td>L</td>
<td>M</td>
<td>L</td>
</tr>
<tr>
<td>SALEM</td>
<td>-</td>
<td>L</td>
<td>-</td>
<td>M</td>
</tr>
<tr>
<td>TUTICORIN</td>
<td>L</td>
<td>L</td>
<td>L</td>
<td>M</td>
</tr>
<tr>
<td>TRICHY</td>
<td>L</td>
<td>L</td>
<td>L</td>
<td>M</td>
</tr>
</tbody>
</table>

H = High pollution  
C = Critical pollution  
M = Moderate pollution  
L = Low pollution
Table - 1.2 List of cities in Tamil Nadu where National Ambient Air Quality Standard (NAAQS) was exceeded

<table>
<thead>
<tr>
<th>City</th>
<th>Major Sources of Pollution</th>
<th>Air Pollutants Violating NAAQS (Annual Average)</th>
<th>Air Pollutants Violating NAAQS (24 hourly average)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHENNAI</td>
<td>Vehicles, Industries</td>
<td>SPM</td>
<td>SPM</td>
</tr>
<tr>
<td>MUDURAI</td>
<td>Vehicles</td>
<td>RSPM, SPM</td>
<td>RSPM, SPM</td>
</tr>
<tr>
<td>SALEM</td>
<td>Vehicles</td>
<td>SPM</td>
<td>SPM</td>
</tr>
<tr>
<td>TUTICORIN</td>
<td>Vehicles</td>
<td>RSPM</td>
<td>RSPM, SPM</td>
</tr>
<tr>
<td>TRICHY</td>
<td>Vehicles, Industries</td>
<td>RSPM</td>
<td>RSPM, SPM</td>
</tr>
</tbody>
</table>
Table -1.3 Air Quality Assessments on SO₂, NOx, RSPM and SPM in different localities of Tiruchirappalli.

<table>
<thead>
<tr>
<th>S. No</th>
<th>Localities</th>
<th>Sample Size (n)</th>
<th>Air Quality (in mg/m³)</th>
<th>Std Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>SO₂</td>
<td>NOx</td>
</tr>
<tr>
<td>1</td>
<td>KARUMANDAPPAM</td>
<td>25</td>
<td>-</td>
<td>21</td>
</tr>
<tr>
<td>2</td>
<td>BISHOP HEBER COLLEGE</td>
<td>35</td>
<td>1</td>
<td>28</td>
</tr>
<tr>
<td>3</td>
<td>THILLAI NAGAR</td>
<td>29</td>
<td>24</td>
<td>35</td>
</tr>
<tr>
<td>4</td>
<td>K. K. NAGAR</td>
<td>34</td>
<td>9</td>
<td>22</td>
</tr>
</tbody>
</table>

Source: Report on Status of Environment in Tamil Nadu, TNPCB, Chennai- 32

R - Residential and other areas, I - Industrial area, Std deviation - Standard deviation,

n - number of days monitored for 16 and more hours a day

Humans are exposed to a large number of physical or chemical genotoxic agents. Exposure to these agents can cause a variety of health hazards. Some of them are expressed immediately; others take years. The latter type of abnormalities include the induction of cancer or genetic diseases (Anwar et al., 1989; Vainio and Hemminki 1988). The exposure patterns are complicated with respect to exposure to single agent or
complex mixtures. These agents may act synergistically or with inhibitory effects. Therefore, continuing efforts are being made to identify hazardous agents, to recognize hazardous exposure conditions and to monitor populations for signs of excessive exposure to prevent adverse health consequences in the population. It has been shown in experimental systems that many environmental agents can be mutagenic or carcinogenic or both. Identification of mechanisms of cancer development has involved consideration of the somatic mutation hypothesis, on the basis of the widespread occurrence of chromosomal abnormalities in cancer cells. Subsequent correlations between the mutagenicity and the carcinogenicity of radiation and chemicals have provided considerable support for this hypothesis (Knudson 1989). Recently, increasing attention has been paid to the development of monitoring methods by which human exposure to mutagens and carcinogens can be detected and several biomarkers were developed for this purpose (Hulka et al.,1990). Epidemiologic studies on cancer development in humans are necessary for risk assessment approaches. However, the epidemiologic approach is limited for two main reasons: first, only relatively high risks can be detected, and second, the observations on end-effects are the consequence of exposures that may have occurred several decades earlier. Improved epidemiology, ideally needs direct and accurate estimates of individual exposures. Biomonitoring has become an essential part in the exposure assessment, its special objective defining biologically relevant doses. It may be relevant to look for early
effects directly in the exposed individuals or groups, especially from high exposure occupations. Presently, there is a need for multidisciplinary studies to evaluate the effect of different genotoxicants. The recommended methodology for risk assessment of environmental genotoxicants is to determine the external exposure by environmental monitoring, the internal absorption by biological monitoring and the biological effect by genetic monitoring. In urban areas, exposures to low levels and short term peak levels of engine exhausts are ubiquitous (IARC 1989a). Higher exposures to engine exhausts may occur in some occupations such as traffic policemen. Many studies have been carried out, using several animal species, to evaluate the potential carcinogenicity of exposure to whole exhaust and to components of exhaust from diesel- and gasoline-fueled internal combustion engines (Heinrich et al., 1986.; Ishinishi et al., 1986., Mauderly et al., 1986; Mauderly et al., 1987).

Urban air thus contains diverse chemical compounds, some of which are genotoxins. An increased risk of cancer has also been reported in occupations with heavy exposure to traffic-related pollution (Silverman et al., 1986; Balarajan and McDowall 1988). The aim of this study was to assess the cytogenetic effects of urban air pollution in the city of Tiruchirappalli located in the state of Tamil Nadu, India.
1.15 STUDY AREA

Tiruchirappalli, is a city in the Indian state of Tamil Nadu it is a municipal corporation and the fourth largest city and urban agglomeration in Tamil Nadu in terms of population. The estimated population of the urban agglomeration is 1,139,534 (as of 2009). It is situated in the centre of Tamil Nadu. It is the administrative headquarters of Tiruchirapalli District. The topology of Tiruchirappalli is flat. It lies at an altitude of 78 m above sea level. The area of the city is 146.90 sq.kms while the urban agglomeration is spread over an area of 180 sq.kms. The river Kaveri (also called Cauvery) and the river Coleroon (also called Kolli dam) flow through Trichy, the latter forms the northern boundary of the city (Fig - 1).

The city is divided into three parts, the Cantonment area to the south, the temples to the north and the bazaar in the centre of the city. Most of Tiruchirapalli's hotels and Government post office is situated in the cantonment, while most of Tiruchirapalli's temples are located in the north. The Rockfort and its temple are situated in the centre of the city and surrounded by a bazaar. There are few hills located within the city, the prominent among them are Golden Rock, Rock Fort, Kajamalai and in Thiruverumbur. There are few reserve forests along the river Cauvery, located at the west and the north-west of the city. The southern and the south-western part of the district is dotted by several hills which are thought to be an offset of the Western Ghats. Eastern ghats also pass through the
district. The soil here is considered to be very fertile. As two rivers flow through the city, the northern part of the city is more greener than other areas of the city.

1.16 POPULATION DYNAMICS

Trichy had a population of 11,39,534 (2009). Males constitute 49.97% of the population and females 50.03%. Trichy has an average literacy rate of 91.45%, and is among the highest literate cities in India. Male literacy is 94.17% and female literacy is 88.73%. In Trichy 9.59% of the population is under 6 years of age.

1.17 CLIMATOLOGY OF THE STUDY AREA

Trichy has very hot climate, with humidity slightly above normal. The city experiences mild winters and humid summers. The timing of the monsoon in this part of the country has lately become unpredictable, with the rainy season starting from mid-October until early-November and the rains then extending until early or mid-January.

<table>
<thead>
<tr>
<th></th>
<th>Summer</th>
<th>Winter</th>
</tr>
</thead>
<tbody>
<tr>
<td>Max.</td>
<td>41 °C (106 °F)</td>
<td>36 °C (97 °F)</td>
</tr>
<tr>
<td>Min.</td>
<td>21 °C (70 °F)</td>
<td>18 °C (64 °F)</td>
</tr>
</tbody>
</table>
Fig. 1 Map showing on study area of Tiruchirappalli
1.18 AIM AND OBJECTIVES

- Survey the literature for air pollution in different cities of the world, India and Tamil Nadu in particular and to compare with previous data on the air quality of Tiruchirappalli in particular.
- Analysis of the frequencies of Single Stranded Breaks (SSBs) by comet assay
- Classification of the various Chromosomal Abberations (CA) observed in the cohort study.
- Compare the frequencies of Micronucleus and chromosomal aberrations are induced by exposure to air pollutants
- Study the application of Double and Single Strand Breaks to the exposure levels in relation to age and other personal habits.

1.19 Collection of blood samples

Peripheral blood samples were obtained from normal healthy, non-smoking and non-alcoholic volunteers as well as traffic policemen of different age groups, with no previous medical exposure to radiation, within the last six months. The amount of blood sample collected from each volunteer varied between 20 and 40 ml based on the amount of the experiment. Usually the volume per aliquot of blood sample was 1.5 to 3.0
ml, depending on the number of cultures, assay type and experimental set up that was to be employed.

1.20 Physical Characteristics of Traffic Police Population.

Traffic police population of Tiruchirappalli is around 290 men in four divisions, all are physically normal (Table 1.4). In the present study, 50 blood samples were collected from traffic police population and 50 blood samples from normal people (control). The samples are divided into five groups based on their age (Bellow 20, 21-30, 31-40, 41-50, and above 51)

Table 1.4 Shows Physical status of traffic police population in Tiruchirappalli.

<table>
<thead>
<tr>
<th>S.No</th>
<th>Physical Status</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Physical Appearance</td>
<td>Normal</td>
</tr>
<tr>
<td>2.</td>
<td>Average age</td>
<td>43</td>
</tr>
<tr>
<td>3.</td>
<td>Average weight (Kg)</td>
<td>66.5</td>
</tr>
<tr>
<td>4.</td>
<td>Smoking habits (%)</td>
<td>15</td>
</tr>
<tr>
<td>5.</td>
<td>Alcoholic (%)</td>
<td>18</td>
</tr>
<tr>
<td>6.</td>
<td>Tobacco(%)</td>
<td>10</td>
</tr>
<tr>
<td>7.</td>
<td>Drugs taken within last six months</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Months (%)</td>
<td></td>
</tr>
</tbody>
</table>
1.21 Viability of cells

Blood samples were incubated at 37°C for various time periods ranging between 12 and 96 hours to check the time duration at which a good viability is found. This experiment indicated an incubation period of 48 hours could provide a reasonably good viability. The viability test was carried out by centrifuging the blood sample at 1000 rpm for 5 minutes and the resultant supernatant consisting of the buffy coat with the lymphocytes and the plasma was carefully pipetted out. To the separated cells 100 μl of 0.4% Trypan Blue solution was added. Cells that picked up the dye appeared blue were considered dead, while those which did not pick up the dye were considered viable cells (Table – 1.5). This was done to study the effect of transportation on the viability of the cells, when brought from the field collection spot to the laboratory.

Percent Viability = Number of viable cells / Total number of cells X 100
Table – 1.5  Percent viability of cells

<table>
<thead>
<tr>
<th>Serial Number</th>
<th>Incubation Duration (hrs)</th>
<th>Percent viability in normal cells</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0</td>
<td>97</td>
</tr>
<tr>
<td>2</td>
<td>12</td>
<td>93</td>
</tr>
<tr>
<td>3</td>
<td>24</td>
<td>92</td>
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<tr>
<td>4</td>
<td>36</td>
<td>91</td>
</tr>
<tr>
<td>5</td>
<td>48</td>
<td>91</td>
</tr>
<tr>
<td>6</td>
<td>60</td>
<td>75</td>
</tr>
<tr>
<td>7</td>
<td>72</td>
<td>63</td>
</tr>
<tr>
<td>8</td>
<td>84</td>
<td>52</td>
</tr>
<tr>
<td>9</td>
<td>96</td>
<td>&gt; 50</td>
</tr>
</tbody>
</table>

1.22 SEPARATION OF LYMPHOCYTES

Lymphocytes were separated from peripheral blood by density gradient method using Histopaque-1077. 3.0 ml Histopaque was taken in a conical test tube and 3 ml of blood sample was carefully layered on the above solution. This was centrifuged at 2200 rpm for 30 minutes. Erythrocytes and the granulocytes sedimented at the bottom of the centrifuge tube, while lymphocytes formed a buffy coat over the Histopaque layer. The upper layer contained plasma. The buffy coat was carefully aspirated with a Pasteur pipette and carefully transferred to a
conical tube and the plasma was used as serum supplement in all the experiments. The lymphocytes obtained were washed with 6 ml PBS and centrifuged at 1200 rpm for 10 minutes. The cell pellet was again washed with PBS for another 3 or 4 times as above. For the comet experiments, 1 ml of the blood sample was overlaid on 1 ml of Histopaque solution and lymphocytes were separated as above.