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OXIDATIVE STRESS IN TRAFFIC POLICE OF KURNOOL TOWN

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ABSTRACT

The aim of present study is to estimate *Oxidative Stress* in traffic police who are occupationally exposed due to higher free radical generation. Blood samples were collected for analysis of Malondialdehyde (MDA) levels, an indicator of oxidative stress using spectrophotometer. Elevated levels of MDA were observed in traffic police. The studies revealed the presence of high levels of MDA -a biomarker of oxidative stress, in traffic police exposed to environmental air pollutants.

KEYWORDS: MDA, Oxidative Stress, Environmental Air Pollutants.

INTRODUCTION

Exposure to environmental pollutants is known to be harmful to health, in general, and to lungs in particular. The contamination of urban air by organic and inorganic toxic pollutants causes concern about the possibility of adverse health effects in resident populations.[1-4] In this respect, traffic police are at particular risk due to the nature of their job, since they are exposed to emissions from the vehicles. It has been estimated that 17% of sudden cardiac deaths observed each year in the USA are related to particulate air pollution.[1-11] Individuals with underlying cardiac or pulmonary disease proved to be at greatest risk for acute adverse effects of air pollution.[12–16]

There is considerable controversy regarding the more subtle long-term adverse effects of pollution exposure in 'chronic day to day' exposure. Numerous clinical data, have been extrapolated from studies on animals or on volunteers, in which exposures were of brief duration and ethically limited to levels having limited transient effects.[17–20] Long-term results obtained from cross-sectional and cohort studies are often difficult to compare due to practical limitations: different duration of exposure, changes in air pollution exposure and lack of adjustment for geographical, social and occupational variables.[16, 21, 22] Few of these analyses dealt with air pollution from traffic alone.[5, 23–31] In these cases possible associations between daily mean air pollution levels-sulphur dioxide (SO2), nitrogen dioxide (NO2), carbon monoxide(CO), ozone (O3) and particulate (PM10) total suspended particles were generally considered as.[12–16] Few of these analyses dealt with air pollution from traffic alone.[5, 23–31] In these cases possible associations between daily mean air pollution levels - sulphur dioxide (SO2), nitrogen dioxide (NO2), carbon monoxide (CO), ozone (O3) and particulate (PM10) total suspended particles were generally considered as indicators of exposure and respiratory and/or cardiovascular symptoms were differently reported. Policemen, taxi drivers, postmen and traffic wardens were considered the most highly exposed occupational groups in cities.[23–26, 29, 30] And it was evident that due to exposure to environmental pollutants lead to release of higher level of free radicals leading to oxidative stress.

Indirect evidence suggests that free radicals and excited state species play a key role in both normal biological function and in the pathogenesis of certain human diseases. Free radicals are molecules or molecular fragments capable of independent existence that contains one or more unpaired electrons in their outer orbital. These molecules are very reactive and tend to initiate chain reactions that result in irreversible chemical alterations of the reacting substance like lipids, proteins, enzymes, DNA, RNA with subsequent structural and functional damage. Free radicals are ubiquitous in biological systems and have been implicated as factors in the process of differentiation, aging (Southern and Powis 1988), mutagenesis and carcinogenesis (Knuutila, 1984). Furthermore, the actions of free radicals have been implicated in the pathophysiology of many diseases including ischemia, reperfusion injury involving brain

(Southern and Powis 1988), atherosclerosis, rheumatoid arthritis (Halli well 1984), myocarditis (Gaudel and Davelleroy 1984), cerebral infarction (Southern and Powis, 1988), hypertension, lung diseases, and neurological disorders.

If the antioxidant defense is not able to eliminate the ROS (Reactive Oxygen species) or inhibit their production resulting in an imbalance between pro and antioxidant, it leads to oxidative stress. ROS such as superoxide anion O_2^-, H_2O_2, OH, NO are highly reactive species, which could induce oxidative damage by initiating lipid per oxidation and DNA damage. Oxidative stress determines membrane alterations including both lipid peroxidation and modifications of membrane fluidity, accompanied by a parallel increase in the intracellular Ca^{+2} concentration (Orien 1991).

Oxidative Stress: Oxidative stress can be defined as a "disturbance in the pro oxidant - antioxidant balance in favour of the former, leading to potential damage". (Sies 1991) and Oxidative stress can result from diminished antioxidants e.g. mutations affecting antioxidant defense enzymes such as CuZnSOD, MnSOD, (or) GPx. Depletions of dietary antioxidants and other essential dietary constituents can also lead to oxidative stress. Reasearch in Jamaica has shown that children with the protein deficiency disease Kwarshiorkar suffer additional problems related to oxidative stress, including low glutathione (GSH) levels and iron overload (Golden 1987). Increased production of ROS/RNS, e.g. by exposure to elevated O_2, in the presence of toxins that are metabolized to produce ROS/RNS, or excessive activation of 'natural' ROS/RNS systems (e.g. inappropriate activation of phagocytic cells in chronic inflammatory diseases such as rheumatoid arthritis and ulcerative colitis).

Oxidative stress can result in adaptation (or) cell injury. Cell injury can be defined as "the result of a chemical or physical stimulus, either in excess or in deficiency, that transiently or permanently alters the homeostasis of the cell'' (Nicotera and Orrenius, 1994). Oxidative stress can cause damage to all types of biomolecules including lipids (lipid per oxidation), proteins and DNA. This imbalance in the oxidant/antioxidant system may lead to lung damage and is likely to cause respiratory problems in individuals exposed to air pollution. Hence the present study is aimed in knowing the correlation between oxidative stress and exposure to environmental pollutants.

MATERIALS AND METHODS

Materials: A total of 80 traffic police were taken for this study. Out of which 40 police were working in office (control group) and other 40 were traffic police who were doing their duty in traffic(experimental group). Data about their age, food habits, duration of service, other habits (smoking, etc. were taken and smokers were excluded from the study, as smoking also increases oxidative stress).

Collection of blood samples

5 ml of blood (heparinised) was collected from 80 police men and plasma was used for analyzing MDA levels.

Methods

Biochemical studies: Separated plasma was used for the estimation of lipid peroxides.

Estimation of lipid peroxidation in plasma: Malondialdehyde, the product of lipid peroxidation present in the biological samples reacts with thio barbituric acid under acidic conditions at 95^0C to form a pink coloured complex with absorbance maxima at 532 nm. The total amount of lipid peroxidation products in the plasma/amniotic fluid of the subjects was estimated by thiobarbituric acid (TBA) method (Gavino et al., 1981). Since this method measures the malondialdehyde (MDA) reactive products the final result obtained is referred as MDA equivalents.

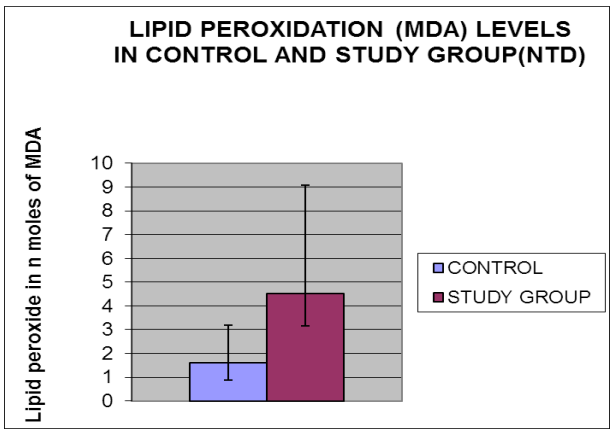
RESULTS

Estimation of Lipid peroxidation (MDA level) in plasma: Lipid peroxidation products (MDA) were measured in 80 police men off which 40 were control and 40 were study group. The mean of MDA levels in the study group was high (4.53196 ± 1.59617) compared to controls (1.596 ± 0.7087) and were expressed in ηmol of MDA equivalence.

Table –1: Showing N moles of MDA in plasma non pregnant women.

S.No	Name of the group	No. of subjects	n moles of MDA Mean ± SD
1	Controls	40	1.59617 ± 0.70877
2	Study group	40	4.53196 ± 1.36595

Lipid per oxidation in the study group was significantly raised ($P \leq 0.05$).



Graph – 1: Lipid peroxidation (MDA) levels in control and study group (NTD).

DISCUSSION

Oxidative stress can be defined as a disturbance in the prooxidant – antioxidant balance in favour of the former, leading to potential damage (Sies, 1991) which results from diminished antioxidants activity, increased production of ROS/RNS. Oxidative stress result in cell injury or cell adaptation, and can damage all types of biomolecules including DNA, proteins and lipids. Lipid peroxidation (MDA) levels in plasma is a marker of oxidative stress and is studies to know the role of oxidative stress in the pathophysiology of many diseases like Crohn's disease, Inflammatory bowel disease and NTDs. In the present study, MDA levels were high in plasma of non pregnant women which was statistically significant when compared to controls ($p \leq 0.05$).

Basal peroxidation is significantly higher in Alzheimer's cortex and this difference is also evident in the presence of exogenous iron (Subba Rao et al, 1990).

Sagger 1989 reported increased levels of MDA and oxidation of DNA and proteins in the substantia nigra of patients with Parkinson's disease.

In the present study, MDA levels were high in plasma of high risk pregnant women which was statistically significant when compared to controls ($p \leq 0.01$).
In the present study, MDA levels were high in amniotic fluid of high risk pregnant women which was statistically significant when compared to controls ($p \leq 0.01$).

Karowicz-Bilin'ska et al, (2001) reported the significantly increased level of lipid peroxidation products- malandialdehyde and Schiff bases in different tissues, amniotic fluid of pregnant woman.

Mary Loeken, 2003 reported the role of oxidative stress in the causation of NTDs. She reported that mild oxidative stress which is insufficient to cause cell death can cause NTD. Hence the present study is in accordance with the report by Mary Loeken, 2003.

Asha Ornoy, et al studied the role of ROS in the etiology of diabetes induced anomalies and these studies supported that the genetic predisposition plays an important role in inducing underdevelopment and NTD.

CONCLUSION

The results of the present study show that chronic exposure to outdoor traffic pollution may reduce resistance to physical effort and increases the risk of cardiovascular and respiratory changes in subjects with slight hypoxemia. It is also possible that these effects could be due to acute exposure, in particular to Sulphur dioxide, Ozone and suspended particulate matter. The data suggest the need to conduct further research using biological monitoring of urban pollutants in these workers, although data obtained from static monitoring can also help to assess the exposure.

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