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Analysis of Chromosomal Aberrations in the Peripheral Blood Lymphocytes of Aluminium Foundry Workers

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KEY WORDS Aluminium; occupational exposure; chromosomal aberrations.

ABSTRACT Cytogenetic effects were evaluated by analysing various types of chromosomal aberrations in the peripheral blood lymphocytes of 97 male employees involved in the manufacture of aluminium wire rods and aluminium conductors. The age range of the subjects was 20-52 years and their service in the factory ranged from 1 to 24 years. The workers were occupationally exposed to aluminium fumes and burnt gases. For comparison, 54 healthy controls belonging to the same age group and socio-economic status were also studied. Peripheral blood lymphocyte cultures were carried out for all the samples and various structural and numerical type of chromosomal aberrations were analysed. A statistically significant increase in total chromosomal aberrations was observed in the exposed group when compared to the control group.

INTRODUCTION

Aluminium finds its wide application in aircraft manufacturing, in generating atomic energy, in making construction materials, laboratory equipment, insulated cables, etc., Several aluminium compounds are used in chemical industries and some are even used in food processing and baking industries.

Aluminium obtained from the mineral bauxite is processed in smelters. While the miners are occupationally exposed to aluminium in an open bauxite mine, workers in smelters especially in potrooms are exposed to aluminium fumes, burnt gases and chemicals like polycyclic aromatic hydrocarbons (PAHs). There are studies that reported high levels of aluminium in the body fluids of the workers employed in potrooms (Rollin et al. 1996; San et al. 1998). In addition, high concentrations of PAHs in the blood (Selden et al. 1999) and NO in exhaled air were also reported in potroom workers (Lund et al. 2000).

The major health hazard associated with exposure to aluminium dust in potroom workers is bronchial asthma (Lund et al. 2000). Several studies have evaluated the lung function in the workers employed in aluminium industries and indicated impairment of pulmonary function (San et al. 1998; Radon et al. 1999).

The biological and health effects reported in the workers employed in aluminium industries reflect the intensity of occupational exposure to aluminium and other chemicals at work place. So, it is worth while to evaluate the toxic effects of such exposure in the workers. Since cytogenetic studies on these workers are meagre, we made an attempt to evaluate the chromosomal damage in the peripheral blood lymphocytes of the workers employed in a industry where aluminium is alloyed with other metals to manufacture wires, rods and conductors.

MATERIALS AND METHODS

The subjects of the present study (n=97) were employed in an aluminium industry, where they were involved in the manufacturing process of aluminium alloy rods, wires and conductors after melting the aluminium ingots and alloying with Mg and Si followed by casting, rolling and coiling. During this process the workers are exposed to high temperatures, aluminium fumes and burnt gases like CO_2, SO_2, H_2S. The workers wore uniform, boots and used scarf and gloves during work.

The age range of the subjects was 20-52 years while their period of employment in that industry ranged from 1-24 years. They worked for 8 hours a day, 6 days a week throughout the year. All the subjects of the present study were non-smokers and non-alcoholics. A control group consisting of 54 healthy nonsmoking and nonalcoholic individuals who had no exposure to any known physical or chemical agent, belonging to the same age group and socio-economic status was also studied.

Heparinised blood samples were drawn from the subjects and controls and lymphocyte cultures were initiated with 0.5ml of whole blood in RPMI 1640 medium containing 20% AB se-

rum, 0.5% phytohaemagglutinin and 0.25% antibiotic. The cultures were incubated at 37^0 C for 72 hours. Colchicine was added to the cultures at the 70^{th} hour to arrest the cell cycle at metaphase stage. Cultures were harvested, slides were prepared, coded and airdried according to the standard method of Moorhead et al. (1960). Slides were stained using giemsa and 150 well spread metaphases were screened for various structural and numerical aberrations.

Gaps: Unstained regions that are smaller than the diameter or width of the chromatid. The chromatids do not show any misalignment.

Breaks: Damage to the chromatid or isochromatid involving a discontinuity of the chromosomes greater than the width of the chromatid. It may be accompanied by a misalignment of the chromatids or chromosomes.

Fragments: Broken portions of the chromatid arms which may or may not be lying in close vicinity of the original chromosomes.

Exchanges: Exchange between chromatids of different chromosomes.

Dicentrics: These are chromosomes with two centromeres with or without associated fragments.

Polyploidy: Condition in which cells display more than double the chromosome number (2n) exact multiples of the haploid chromosome numbers.

The statistical analysis of the data was done using χ^2 test and one way ANOVA.

RESULTS

The results on chromosomal damage in the non-smoking workers are given in Table 1. The total chromosomal aberrations observed in the exposed group was 3.09% which was statistically significant when compared with 1.78% of total chromosomal aberrations observed in the control group ($p<0.05$). The chromosomal aberrations like gaps, breaks and fragments were both of chromatid and isochromatid type. The percentage of dicentrics was 0.41 and polyploids was 0.90 in the exposed group as against 0.24% dicentrics and 0% polyploids in the control group.

Chromosomal aberrations when analysed according to duration of exposure in the workers (Table 2) revealed mean number of chromosomal aberrations of 4.267, 4.490, 5.200 and 6.375 in 1-6, 7-12, 13-18 and >=19 year groups respectively. The mean number of aberrations in each time interval significantly differed from the mean number of aberrations in the control group ($p<0.001$). Since, high variations were observed between the groups, we applied non-parametric Kruskai Wallis test and found that the differences were statistically significant ($p< 0.001$).

Table 2: Frequencies of chromosomal aberrations according to duration of exposure in the workers.

Group/duration of exposure in years	*Mean No. of chromosomal aberrations per sample ± SD*	*Sample size*
Control group	$1.000^a \pm 0.801$	70
Exposed Group		
1–6	$4.267^b \pm 1.507$	30
7–12	$4.490^b \pm 2.662$	49
13-18	$5.200^b \pm 2.781$	10
>=19	$6.375^b \pm 3.160$	8
F ratio	39.297	
Probability	$p < 0.001$	

Note: Variation in superscripts between groups indicates significance of difference ($p<0.001$)

DISCUSSION

The present study showed increased chromosomal damage in the lymphocytes of the workers. Our results are in agreement with the study of Haugen et al. (1983) who reported increased frequency of sister chromatid exchanges in aluminium plant workers exposed to aluminium dust. Since cytogenetic studies in aluminium workers are scarce we compared our results with a study (Fatima et al. 1995) that reported significant in-

Table 1: Data on different types of aberrations in aluminium workers

Group	*Samples*	*Number of Meta-phases*	*Chromatid type aberrations*			*Isochromatid type aberrations*			*Dicentrics*	*Total no. of aberr-ations*	*Poly-ploids*
			Gap	*Breaks*	*Frag-ments*	*Gaps*	*Breaks*	*Frag-ments*			
Control group	54	8100	47 (0.58)	40 (0.49)	30 (0.37)	38 (0.46)	34 (0.41)	20 (0.24)	20 (0.24)	144 (1.78)	0 (0.00)
Exposed group	97	14550	139 (0.95)	133 (0.91)	81 (0.55)	115 (0.79)	118 (0.81)	58 (0.39)	61 (0.41)	451 (3.09)	131 (0.90)

crease in the frequency of SCEs in workers occupationally exposed to cement dust that contained aluminium silicate, calcium silicate etc. Earlier, several studies have presented evidence for the clastogenic effects of aluminium and its compounds in a battery of test systems in animal models (Parada and Jaszczak 1993; Roy et al. 1991; Octive et al. 1991) and humans (Roy et al. 1990).

Although we have not estimated aluminium concentrations in the biological fluids of the workers, there are reports that indicated correlation between concentration of respirable aluminium dust in the potroom and biological indicators like concentration of aluminium in serum and urine of the workers (Rollin et al. 1996). While San et al. (1998) reported a significantly high level of aluminium in the serum of aluminium workers, Bast-Pattersen et al. (1998) reported high levels of aluminium in the urine of aluminium welders. So, chronic exposure to aluminium at the workplace may prove toxic in the long term.

High levels of urinary fluoride were also reported in workers of a primary aluminium industry after their shift compared with their preshift level (Radon et al. 1999). Selden et al. (1999) showed high correlation between levels of hexachlorobenzene and octachlorostyrene, the thermal byproducts of hexachloroethane in plasma of the workers. These studies suggest that workers have exposure to flourides and PAHs in some aluminium industries.

Several studies elucidated the increased risk for different cancers in the workers of aluminium plants. Romundstad et al. (2000) studied the association between exposure to polyaromatic hydrocarbons, fluorides and cancer incidence and mortality in aluminium plant workers. Earlier, Boffetta et al. (1997) reported increased risk for lung, skin and bladder cancer in workers with heavy exposure to PAHs. However, Romundstad et al. (2000) reported no association between exposure to PAHs and lung cancer in workers employed in aluminium reduction plants.

The results of the present study clearly indicate occupational exposure to aluminium resulting in chromosomal damage in the workers. The effects might be due to incorporation of aluminium in DNA (Karlik et al. 1980). In view of the high incidence of specific cancers, mutagenic effects and other biological and health effects reported in the workers of aluminium industries, we suggest that exposure to aluminium dust and toxic chemicals at workplace should be minimised by using appropriate protective measures.

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