

## Analysis of Chromosomal Aberrations in Mint Factory Workers

T. Padmaja, Ch. V. Ramana Devi and P.P. Reddy

*Institute of Genetics and Hospital for Genetic Diseases, Osmania University, Begumpet  
Hyderabad 500 006, Andhra Pradesh, India*

**KEY WORDS** Metal alloys; occupational exposure; mint factory; chromosomal aberrations.

**ABSTRACT** The investigation was carried out in the peripheral lymphocytes of mint factory workers who were exposed to dusts and fumes of metal alloys like aluminium, magnesium, copper and nickel. Samples of peripheral blood were collected from 130 workers out of which 67 were smokers and 63 were nonsmokers. 54 nonsmokers and 42 smokers belonging to the same socio-economic group but not exposed to either radiation or other toxic chemicals were studied for control data. There was a significant increase in the chromosomal aberrations in the workers when compared to the controls.

### INTRODUCTION

Industrial revolution has led to the entry of various metals like lead, mercury, chromium, arsenic, nickel, cadmium, copper, manganese, and zinc into various domestic, cosmetic and consumer products that are used by man in day to day life.

Aluminium is a known neurotoxin in animals (Golub and Geshium 1992) and human (Bolla et al. 1992). Carcinogenic effects of aluminium are reported in animals (Bingham et al. 1979) and in man (Konstantinov et al. (1972). Kusiak et al. (1993) reported stomach cancer in workers occupationally exposed to aluminium. Occupational exposure to copper is reported to result in lung cancer (Chen et al. (1993) in occupational workers. Malo et al. (1993) reported severe asthma in copper miners. Nickel is a well known carcinogen (Fairhurst and Illing 1987). Carcinogenicity of nickel was reported in rats (Waalkes et al. 1987). Occupational exposure to nickel is reported to elevate sisterchromatid exchanges (Peltonen (1979)) and chromosomal aberrations (Nishimura and Umeda 1979). Though all these reports suggest deleterious effect of metals reports on the mutagenicity of these metals in occupationally exposed population are inadequate. Hence an effort has been made to study the incidence of chromosomal aberrations in the peripheral lymphocytes of mint factory workers who were

occupationally exposed to aluminium, copper, nickel and magnesium. Earlier reports suggest that smoking has adverse effect on chromosomes (Obe et al. 1978). Hence the workers were categorised into smokers and nonsmokers. Further, the workers in both categories were divided into three groups based on the duration of exposure.

### MATERIALS AND METHODS

The blood samples were collected from mint factory workers. India government mint (I.G.Mint) supplies high quality currency coins to Reserve Bank of India which are ready for circulation. The metal alloys are made from combination of aluminium and magnesium (alumag) and copper and nickel (cupronickel) by heating them to a temperature of 660°C (alumag) and 1480°C (cupronickel) in open fire furnaces. The alloys are then cooled (annealing) and made into sheets. The sheets were processed into currency coins of different denominations and supplied to Reserve Bank of India for circulation.

*Sample Collection:* Intravenous blood was collected aseptically using heparin (anticoagulant) from 63 nonsmokers (age range 24 - 56 years) and 67 smokers (age range 22 - 54 Years). Their working schedule was 8 hours per day and duration of service was from 3 - 27 years. Simultaneously heparinised blood was collected aseptically from 54 nonsmokers (control - I) and 42 smokers (control - II) who were not exposed to toxic chemicals and belonged to same socio-economic status.

Whole blood was added to RPM I 1640 medium supplemented with 25% human AB serum, 0.5% phytohemagglutinin, 0.25% dicristicin and 0.25% gentamycin. Control group cultures were maintained simultaneously under similar conditions. All the cultures were incubated at 37°C for 72 h. 0.1 µg/ml of colchicine was added 2h before harvesting the cultures, then the cultures were

harvested and slides were prepared according to standard method of Moorhead et al .(1960). Slides were coded and screened for various types of chromosomal aberrations. For each sample 150 metaphases were screened for chromosomal aberrations such as gaps, breaks, fragments, dicentrics and polyploids. The significance of total chromosomal aberrations was analysed using the chi-square ( $\chi^2$ ) test. Since gaps are not stable aberrations they were excluded from the total number of aberrations.

## RESULTS

The incidence of gaps, breaks, fragments and polyploids in nonsmoker exposed group is shown in table 1 and in exposed smokers in table 2.

In the control group data from nonsmokers (Control - I) was compared with smokers (Control- II) data. Chromosomal aberrations were high in the smokers when compared to nonsmokers. The difference in the total chromosomal aberra-



Fig. 1. Gaps



Fig. 2. Breaks



Fig. 3. Metaphase showing exchanges



Fig. 4. Metaphase showing dicentrics

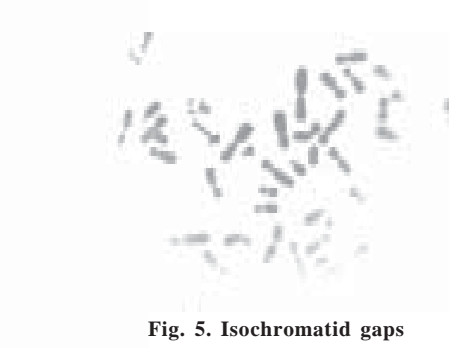


Fig. 5. Isochromatid gaps



Fig. 6. Polyploid

**Table 1: Frequency of chromosomal aberrations in nonsmokers employed in mint factory**

Group and duration of exposure in years	Number of samples	Number of metaphases screened	Chromatid type aberrations			Isochromatid aberrations			Total number of aberrations		
			Gaps	Breaks	Acentric fragments	Exchanges	Gaps	Breaks		Acentric fragments	
Control Group - I											
Exposed Group	54	8100	47 (0.59)	45 (0.55)	30 (0.37)	4 (0.04)	38 (0.46)	34 (0.41)	25 (0.30)	22 (0.27)	160 (1.97)
1 - 10 Y	25	3750	57 (1.52)	52 (1.38)	30 (0.80)	8 (0.21)	30 (0.80)	24 (0.64)	13 (0.34)	10 (0.26)	137* (3.65)
11 - 20 Y	22	3300	69 (2.09)	65 (1.90)	33 (1.00)	4 (0.12)	32 (0.96)	28 (0.84)	24 (0.72)	20 (0.60)	174* (5.27)
> 20 Y	16	2400	58 (2.41)	52 (2.16)	28 (1.16)	8 (0.33)	24 (1.00)	20 (0.83)	15 (0.62)	18 (0.75)	141* (5.87)
Total	63	9450	184 (1.94)	169 (1.78)	91 (0.96)	20 (0.21)	86 (0.91)	72 (0.76)	52 (0.55)	48 (0.50)	452* (4.78)

**Table 2: Frequency of chromosomal aberrations in smokers employed in mint factory**

Group and duration of exposure in years	Number of samples	Number of metaphases screened	Chromatid type aberrations			Isochromatid aberrations			Total number of aberrations	Number of polyploid cells	
			Gaps	Breaks	Acentric fragments	Exchanges	Gaps	Breaks			Acentric fragments
Control Group - I	54	8100	49 (0.60)	45 (0.55)	30 (0.37)	4 (0.04)	38 (0.46)	34 (0.41)	25 (0.30)	22 (0.27)	160 (0.0)
Control Group-II	42	6300	89 (1.41)	82 (1.30)	33 (0.52)	14 (0.22)	40 (0.62)	27 (0.42)	24 (0.38)	21 (0.33)	201* (3.19)
Exposed Group											
1 - 10 Y	22	3300	68 (2.06)	48 (1.36)	67 (2.03)	5 (0.15)	14 (0.42)	61 (1.84)	44 (1.33)	50 (1.51)	275* (8.33)
11-20 Y	30	4500	106 (2.35)	122 (2.71)	84 (1.86)	25 (0.55)	57 (1.26)	74 (1.64)	66 (1.46)	61 (1.35)	432* (9.60)
> 20 Y	15	2250	70 (3.11)	81 (3.60)	48 (2.13)	16 (0.71)	38 (1.68)	45 (2.00)	37 (1.64)	36 (1.60)	263* (11.68)
Total	67	10050	244 (2.42)	251 (2.49)	199 (1.98)	46 (0.45)	109 (1.08)	180 (1.79)	147 (1.46)	147 (1.46)	970* (9.65)

\*P<0.05; 150 Metaphases were analysed for each sample; Values given in parentheses are percentages; Gaps and polyploids are not included in total number of aberrations

tions between smokers and nonsmokers was found to be statistically significant.

In the nonsmokers exposed to metal alloys there was a significant increase in the chromosomal aberrations when compared to nonsmoker control group (Control - I). There was an increase in the incidence of chromosomal aberrations with duration of exposure. The increase in the chromosomal aberrations at all time intervals was significant when compared to the control subjects. The difference for chromosomal aberrations between the time intervals was also significant.

In the exposed smokers (Table 2) there was a significant increase of chromosomal aberrations when compared to control groups I and II. Totally a significant increase was observed in the chromosomal aberrations at all the time intervals, when compared to the control subjects. Further, increase in the chromosomal aberrations between time intervals was also significant. In both the control group and exposed group a high incidence of chromosomal aberrations was recorded in the smokers when compared to nonsmokers.

## DISCUSSION

The toxicants that enter into human body cause disturbance to normal state and behaviour of the chromosomes which in turn lead to reshuffling of hereditary material causing chromosomal aberrations and gene mutations in somatic and germ cells.

Several earlier reports suggest harmful effects in subjects occupationally exposed to metals. Mylius and Gullvag (1984) reported significant increase in macrophages in sputum expectorates from 84 aluminium potroom workers. Deleterious effects on peripheral nervous system were reported by Araki et al. (1993) in the workers exposed to lead, zinc and copper. Vanderwaal et al. (1990) reported high incidence of lung and skin cancers in welders and cutters exposed to chromium, nickel, copper etc. Magnesium is a relatively nontoxic metal. However acute poisoning has been observed in cows fed on diet containing high content of magnesium (O'Kelly and Fontenot, 1973).

As all these reports suggest toxic effects of metals present study was conducted in the mint factory workers who were exposed to dusts and fumes of metal alloys containing aluminium, magnesium, copper and nickel. For com-

parison studies were carried out on smokers and nonsmokers who were not exposed to toxic chemicals.

In both the control group and exposed group there was a significant increase of chromosomal aberrations in the smokers when compared to nonsmokers. Our reports are in accordance with previous reports of Obe and Herha et al. (1978), Fatima et al. (1995), Rupa et al. (1989) who reported high incidence of chromosomal aberrations in smokers.

In the present study there was a significant increase of chromosomal aberrations in the exposed group when compared to control group. The results confirm earlier reports of Deng et al. (1988) who reported increased frequency of sisterchromatid exchanges in electroplating workers exposed to nickel. Haugen et al. (1983) reported increase of sister chromatid exchanges in nickel plant workers. Though metals like aluminium, nickel, copper and magnesium are used in wide spectra of industries reports on their mutagenicity are rather scarce. It is difficult to identify the effect of individual metals when they are used as alloys. Hence our study confirms cumulative mutagenic effect of these metals. Further our study warrants that undue exposure of man to metals and their alloys in industries might result in genetic damage. Appropriate precautionary measures should be taken by the workers to minimise exposure in their work environment.

## CONCLUSION

Our study suggests that workers employed in industries using metals or their alloys and exposed to dusts or fumes of metals or metal alloys are prone to genetic damage. The high incidence of chromosomal aberrations in the smokers reveal a cumulative effect of smoking and exposure to metal alloys.

## REFERENCES

- Araki S, Murta K, Vchida E, Aono H 1993. Radial and media nerve conduction velocity in workers exposed to lead, copper and zinc. *Environ Res*, **61(2)**: 308-316.
- Bolla K, Briefel IG, Spector D, Wieler L 1992. Neurocognitive effects of aluminium. *Archives of Neurology*, **49(2)**: 1021-1026.
- Bingham E, Trosset RP and Warshawsky D 1979. Carcinogenic potential of petroleum hydrocarbons. *Environ J Pathol Toxicol*, **3(2)**: 483-563.
- Chen R, Wei L, Huang H 1993. Mortality from lung cancer among copper miners. *Toxicol Environ Chem*, **27(3)**: 161-166.

- Deng CZ, Lee HCH, Xian HL, Yao MC 1988. Chromosomal aberrations and sister chromatid exchanges in workers. Effect of nickel and chromium. *J Trace Elements Exp Med*, **1**: 57-62.
- Fairhurst S, Illing HPA 1987. *The Toxicity of Nickel and Its Inorganic Compounds (Health and Safety Executive Review 19)*. London: Her Majesty's stationary office.
- Fatima SK, Aruna Prabhavati P, Reddy PP 1995. Frequencies of sister chromatid exchanges in lymphocytes of portland cement factory workers. *Bull Environ Contam Toxicol*, **55(3)**:704-708.
- Golub MS, Geshuim ME 1992. Neuro development effect of aluminium in mice. Fostering studies. *Neuro Toxicol Teratol*, **14(3)**: 177-182.
- Haugen A, Becher G, Bjorseth A 1983. Biological monitoring of occupational exposure to polycyclic aromatic hydrocarbons in an aluminium plant. *Eur J Cancer Clin Oncol*, **19(1)**: 12-14.
- Konstantinov VG, Simakhina, Gotlib EV 1972. Problem of the carcinogenic hazard in aluminium electrolysis hall. In: *Occupation Cancers*, Moscow: Academy of Medical Sciences, 87-91.
- Kusiak RA, Rilchie AC, Springer J, Muller J 1993. Mortality from stomach cancer in Ontario miners. *Br Ind J Med*, **50(2)**: 117-126.
- Malo JL, Cartier A, Dolovich J. Occupational asthma due to zinc and copper. *Environ Respir J*, **6 (3)**: 447-450.
- Mylius EA, Gullvag B 1984. Alveolar macrophage count (AM-test) as indicator of lung reaction to air pollution. *Acta Cytol*, **55(1)**: 126-129.
- Moorhead PS, Nowell PS, Mellman WJ, Battups, Hungerford DA 1960. Chromosomal preparation of leukocytes cultured from human peripheral blood. *Exp Cell Res*, **20(1)**: 613-615.
- Nishimura M, Umeda M 1979. Induction of chromosomal aberrations in cultured mammalian cells by nickel compounds. *Mut Res*, **68(2)**: 337-49.
- O'Kelly RG, Fontenot JP 1993. Effects of feeding different magnesium levels to dry lot fed gestating cows. *J Anim Sci*, **36(3)**: 994 -1000.
- Obe G, Herha J 1978. Chromosomal aberrations in heavy smokers. *Hum Genet*, **41(1)**: 259-263.
- Peltonen L 1979. Nickel sensitivity in the general population. *Contact Dermatitis*, **5(3)**: 27 - 32.
- Rupa DS, Reddy PP, Reddy OS 1989. Chromosomal aberrations in peripheral lymphocytes of cotton field workers exposed to pesticides. *Environ Res*, **49(3)**: 1-6.
- Vanderwaal JF 1990. Exposure of welders to fumes and gases in dutch industries. Summary of results. *Ann Occup Hyg*, **34 (1)**: 45-54.
- Waalkes MP, Rehm S, Kaspraz KS 1987. Inflammatory, proliferative and neoplastic lesions at the site of metallic identification ear tags in Wistar rats. *Cancer Res*, **47(2)**: 2445-2450.