*Rev Biomed 2000; 11:87-90.*

# *Chromosomal aberrations in smokers exposed to metallic dust in mint factory.*

**Original Article**

Padmala Reddy, Ch. V. Ramana Devi, V. Vidyullatha, M. Hema Prasad, P.P. Reddy.

Department of Biochemistry, College of Science, Osmania University, Hyderabad, A.P, INDIA.

**SUMMARY.**

**Introduction.** The incidence of chromosomal aberrations was evaluated in mint factory smokers workers exposed to metal alloys of aluminium, copper, nickel and magnesium.

**Material and methods.** Heparinised blood samples were collected from 50 smokers exposed to metallic dust and fumes, 28 non smokers and 30 smokers who were not exposed to metallic dusts and fumes (belonging to same socioeconomic group as that of workers). All the samples were analysed for the incidence of chromosomal aberrations. Further, the total exposed population were categorised into 4 groups based on the duration of exposure.

**Results.** There was a significant increase of chromosomal aberrations in the smoker exposed to metal alloys when compared to the controls. An increase of chromosomal gaps, breaks, fragments, deletions was observed, as the duration of the exposure to metal dust is increased.

**Discussion.** Our study warrants that undue exposure of man to these metals in industrias might result in genetic damage and hence appropriate precautionary measures have to be taken by workers to minimise exposure in their work environment.

***(Rev Biomed 2000; 11:87-90)***

**Key words:** Occupational metal exposure, chromosomal aberrations, genetic damage.

**RESUMEN.**

**Aberraciones cromosómicas en fumadores expuestos a polvo metálico en una fábrica de monedas.**

**Introducción.** La incidencia de aberraciones cromosómicas fue evaluada en trabajadores de una fábrica de monedas expuestos a aleación de metales de aluminio, cobre, níquel y magnesio.

**Material y métodos.** Muestras de sangre heparinizada fueron colectadas de 50 personas fumadoras expuestas a polvo metálico y humo, 28 no fumadores y 30 fumadores que no estaban expuestos a polvo metálico y humo (del mismo nivel socioeconómico del grupo de trabajadores). Todas

*Corresponding address: Dr. Ch. Venkata Ramana Devi, Dept. of Biochemistry, Osmania University Hyderabad-500 007 (A.P.) India.*
*Telefax 091 - 040 - 7017044 E-mail: ramanadevi@usa.net*
*Received November 3, 1999; Accepted Frebruary 15, 2000.*
**This paper is also available at http://www.uady.mx/~biomedic/rb001121.pdf**

las muestras fueron analizadas para definir la frecuencia de aberraciones cromosómicas. La población expuesta fue categorizada en cuatro grupos de acuerdo al tiempo de exposición.

**Resultados.** Hubo un incremento significante de aberraciones cromosómicas en los fumadores expuestos a aleaciones de metales cuando se comparó con los grupos controles. Se observo incremento de brechas, rupturas, fragmentos y deleciones en relación a la duración de la exposición al polvo metálico.

**Discusión.** Nuestro estudio demuestra que la exposición del hombre a estos metales en industrias, puede resultar en un daño genético y que deben los trabajadores tomar medidas precautorias apropiadas para minimizar este tipo de exposición en su ambiente de trabajo. ***(Rev Biomed 2000; 11:87-90)***

**Palabras clave:** Exposición metálica ocupacional, aberraciones, daño genético

## INTRODUCTION.

The incidence of chromosomal aberrations was evaluated in mint factory workers exposed to metal alloys of aluminium, copper, nickel and magnesium. The alloys were further processed into currency coins of different denominations. Workers of mint factory were exposed to dusts and fumes of metal alloys and were in contact with alloys (entry is through respiratory and dermal route),

Metais like aluminium, copper, magnesium and nickel have been used as coinage metals for centuries. Aluminium is an established neurotoxin both in experimental animals and humans (1-3). Carcinogenic effects of aluminium have been reported in animals (4) and in man (5). Occupational exposure to aluminium has been reported to result in asthma, and lung disease (6).

Nickel is a well known carcinogen. Carcinogenicity of Nickei is reported in mice and in guinea pigs (7). Occupational exposure to nickel is reported to cause increase in sister chromatid exchanges (SCE's) (8) and chromosomal aberrations (9). Fatal poisoning due to Magnesium Sulphate has heen reported in both adults and children (10).

In the present study effect of exposure to metallic dusts and fumes on peripheral lymphocytes of smokers employed in mint factory was evaluated.

## MATERIALS AND METHODS.

Intravenous blood was collected aseptically using heparin (anticoagulant) from 50 smokers (age ranged from 27 to 56 years) exposed to metallic dusts and fumes. Simultaneously heparinised blood was collected aseptically from 28 non-smokers (age ranged from 26 to 40 years) (control group I) and 30 smokers (age ranged from 26 to 48 years) (control group II) who were not exposed to metallic dusts and fumes.

0.5 mL of heparinised whole blood was added to RPMI 1640 medium supplemented with 25% human AB serum, 0.5% phytohaemagglutinin and 0.25% discristinin and 0.25% gentamycin. Control group cultures were maintained simultaneously under similar conditions. All the cultures were incubated at 37ºC for 72 h. 0.1 mg/mL of colchicine was added 2 h before harvesting the cultures, then the cultures were harvested and slides were prepared according to the standard method describes by Moorhead, *et al*. (11). 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, deletions, dicentrics and polyploidy. The significance of the total chromosomal aberrations was analysed using the $x^2$ test. Since gaps are not stable aberrations they were excluded from the total number of aberrations.

## RESULTS.

The incidences of gaps, breaks, fragments, deletions and polyploids are shown in table 1.

Table 1 shows that chromatid type aberrations were high in normal smokers and exposed smokers when compared to the non-smokers. Isochromatid type aberrations were slightly higher in exposed and unexposed smokers when compared to the controls. The difference in the total chromosomal aberrations

**Table 1**
**Frequency of chromosomal aberrations in smokers employed in mint factory.**

| Group & duration of exposure in years | Number of samples | Total No. of metaphases screened | Chomotid type aberrations | | | | Isochromatid type aberrations | | | | Dicentrics | Total No. of aberrations | Number of polyploid cells |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Gaps | Breaks | Fragments | Deletions | Gaps | Breaks | Fragments | Deletions | | | |
| Control Group-I | 28 | 4200 | 23 (0.54) | 18 (0.42) | 8 (0.19) | 4 (0.09) | 20 (0.47) | 18 (0.42) | 13 (0.30) | 8 (0.19) | 16 (0.38) | 85 (1.92) | 0 (0.00) |
| Control Group-II | 30 | 4500 | 96 (2.13) | 50 (1.51) | 20 (0.44) | 28 (0.62) | 26 (0.57) | 16 (0.35) | 14 (0.31) | 18 (0.04) | 11 (0.24) | 157* (3.48) | 6 (0.13) |
| Expose Group | | | | | | | | | | | | | |
| 1-5yrs | 7 | 1050 | 28 (2.66) | 10 (0.95) | 10 (0.95) | 13 (1.23) | 4 (0.38) | 8 (0.76) | 9 (0.85) | 9 (0.85) | 11 (1.61) | 70* (6.66) | 20 (1.90) |
| 6-10yrs | 14 | 2100 | 37 (1.76) | 31 (1.47) | 13 (0.61) | 18 (0.85) | 16 (0.76) | 16 (0.76) | 22 (1.04) | 16 (0.76) | 35 (1.66) | 151* (7.19) | 39 (1.85) |
| 11-15yrs | 10 | 1500 | 45 (3.00) | 30 (2.85) | 14 (0.93) | 13 (0.86) | 15 (1.00) | 10 (0.66) | 14 (0.93) | 13 (0.8) | 23 (1.53) | 117* (7.80) | 28 (1.86) |
| 16-20yrs | 13 | 1950 | 56 (2.94) | 42 (2.21) | 8 (0.42) | 19 (1.00) | 16 (0.84) | 9 (0.47) | 24 (1.26) | 15 (0.78) | 38 (2.00) | 155* (8.15) | 26 (1.36) |
| Total 1-25yrs | 50 | 7500 | 192 (2.56) | 131 (1.74) | 51 (0.68) | 71 (0.94) | 61 (0.81) | 55 (0.73) | 79 (1.05) | 66 (0.88) | 122 (1.62) | 585* (7.8) | 145 (1.93) |

150 metaphases were scored for each worker * P < 0.05. Control-I - Unexposed non-smokers.
Values given in parentheses are percentages.
Gaps and polyploids are not included in total no of aberrations. Control-II - Unexposed smokers.

between smokers and non-smokers was found to be statistically significant.

The workers were categorised into four groups depending upon the duration of their service. An increase of chromosomal gaps, breaks, fragments, deletions was observed, as the duration of the exposure to metal dust is increased. The exposure of 1-5 years and 6-10 years to metal dust shows an increased chromatid and iso-chromatid type aberrations. The similar observation for 11-15 years and 16-20 years exposure was observed and a total of 1-25 years, the changes are represented in table 1.

## DISCUSSION.

Earlier studies suggest that most of the toxicants that are introduced into the body may disturb normal state and behaviour of the chromosomes and may thus reshuffle the hereditary material causing chromosomal aberrations and gene mutations in somatic and germ cells.

Though a number of earlier reports suggest aluminium to be a neurotoxic agent (l-3), it's mutagenic nature is not well known. Toxic signs of copper intakes have been known for centuries. An increase in lung cancer mortality has been observed in foundry workers exposed to nickel dusts (12). Kipling and Waterhousel reported an excess of prostatic cancer in 248 men exposed to metallic dusts in nickel-cadmium factory (13).

Magnesium is a relatively nontoxic metal. However magnesium inhalation by minerals and industrial workers is reported to cause leucocytosis, granulomas and fever (14). Acute poisoning has been observed in cows fed on a diet containing a high content of magnesium (15).

Since all these reports suggest high risk factors to man, present investigation was carried out on chromosomes of mint factory workers who were

occupationally exposed to metal alloys of aluminium, copper, nickel and magnesium while processing them into currency coins of different denominations. For comparison, studies were carried out on smokers and non- smokers who were not exposed to metals.

A significant increase in chromosomal aberrations was observed in smokers (exposed) when compared to non-smokers (unexposed). Our results confirm previous reports of Husgafvel-Pursian, *et.al.* (16), Rupa, *et. al.* (17) who showed a significant increase of chromosomal aberrations in smokers. A high incidente of SCE's was observed in cigarette smokers by Vijayalakshmi and Evans (18).

In the present investigation the frequency of chromosomal aberrations was significantly high in the workers of the mint factory. Though metals like aluminium, nickel, copper, magnesium find application in a wide spectra of industries, their role in metabolism of living organisms and their mutagenicity remains to be elucidated. Though it is difficult to identify the effect of individual metals, our study confirms a cumulative mutagenic effect of these metals. Further more our study suggests that undue exposure of man to these metals in industrias might result in genetic damage and hence appropriate precautionary measures have to be taken by workers to minimise exposure in their work environment.

**REFERENCES.**

1. Golub MS, Keen CL, Geshwin ME. Neuro developmental effect of aluminium in mice. Fostering studies. Neurotoxicol Teratol 1992; 14:177-82.

2. Russo LS, Beale G, Sandroni S, Ballinger WE. Aluminium intoxication in undialysed adults with Chronic renal failure. J Neurol Neuro Psychiat 1992; 55:697-700.

3. Shegamatsu K, McGeer PL. Accumulation of amyloid precurser protein in damaged neuronal process and microglia following intracerbral administration of aluminium salt. Brain Res 1992; 593:117-23.

4. Bingham E, Trosset RP, Warshawsky D. Carcinogenic potential of petroleum hydrocarbons. J Environ Pathol Toxicol 1979; 3:483-563.

5. Konstantinov VG, Kuzuminykh AI. Tarry substances and 3,4 benzapyrene in the air of electrolytic shops of aluminium workers and their carcinogenic significance. Hyg Sanit 1971; 36:368-71.

6. Schwarz YA, Kivity S, Ribak Y, Grief J. Esionophilic lung reaction to aluminium andhard metal. Chest 1994; 105; 1261-3.

7. Hueper L. Nickel and carcinogenecity. Eur J Cell Biol 1958; 19:276-80.

8. Peltomen L. Nickel sensitivity in the general population Contact Dermatitis 1979; 5:27-32.

9. Nishimura M, Umeda M. Induction of chromosomal aberrations in cultured marmalian cells by nickel compounds. Mutat Res 1979; 68:337-49.

10. Arena JM. Poisoning, Toxicology symptoms, Treament. 3rd ed. American Lecture Series No: 903, Thomas Spring Field.

11. Moorhead PS, Nowell PC, Mellmann WJ, Batips DM, Hungerford DA. Chromosome preparations of leucocytes cultured from human peripheral blood. Exp Cell Res 1960; 20:613-6.

12. Cornell RG, Handis Jr. Mortality patterns among nickel and chromium alloy foundary workers. Nickel in the human environment. Lyon IARC. 1984; 53:87-93.

13. Kipling MD, Waterhouse JAH. Cadmium and prostatic carcinoma. Lancet 1967; I:730-1.

14. Sollman FW. A manual of pharmacology and it's applications to therapeutics and toxicology. 8th edition. New York: W.B. Saunders Co. 1963.

15. O'Kelly RG, Fontenot JP. Effects of feeding dífferent magnesium levels to dry lot fed gestating beef, cows. J Anim Sci 1973; 36:994-1000.

16. Husgafvel, Pursianinen K. Sister chromatid exchange and cell proliferation in cultured lymphocytes of passively smoking restaurent personnel. Mutation Res 1987; 190:211-5.

17. Rupa DS, Rao PVL, Reddy PP, Reddy OS. Invitro effect of monochrotophos on human lymphocyte cultures. Bull Environ Contam Toxicol 1988; 41:737-41.

18. Vijaya Lakshmi, Evans HJ. *In vivo* and *in vitro* effects of cigarette smoke on chromosomal damage and sister chromatid exchange in human peripheral blood lymphocytes. Mutation Res 1982; 92:321-2.