

Haematological and Cytogenetic Studies in Workers Occupationally Exposed to Cement Dust

A. L. Calistus Jude, K. Sasikala, R. Ashok Kumar, S. Sudha and J. Raichel

Division of Human Genetics, Department of Zoology, Bharathiar University, Coimbatore, Tamil Nadu, India

KEY WORDS Cement dust; occupational exposure; haematological hazard; cytogenetic damage.

ABSTRACT The present study evaluates the haematological and cytogenetic risks in subjects occupationally exposed to cement dust. While cement dust caused indirect haematological damage resulting in abnormal blood cell counts, it also caused genetic damage comprising of minor chromosomal aberrations, decrease in mitotic index and increased frequency of sister chromatid exchanges.

INTRODUCTION

Air pollution is a significant factor in morbidity and mortality within industrial societies. Hazardous substances are distributed widely in ecosystems due to diverse human activities such as energy usage, industrial enterprises, agriculture, etc. The human haemopoietic system is extremely sensitive to some environmental influences because of the rapid synthesis and destruction of cells with consequent heavy metabolic demands. Human population is prone to exposure to substances that are genotoxic since some of the pollutants are carcinogens and mutagens with a capacity to affect both the structural integrity of DNA and the fidelity of its biologic expression.

For many centuries polluted air has been considered to be a hazard to health, and concern has been mounting during the last few decades about the possible deleterious effects of the introduction of an increasing number of exobiotic substances into the environment and that many of these compounds can chemically alter DNA which in-turn can lead to deleterious consequences.

In contrast to the world's experience of pollutants such as ozone, sulphur oxides, nitrous oxides and acid rains, a lot of industrial regions are confronted with problems of alkalization due to high content of various industrial alkaline dusts and ash in the pollution complex.

Toxic effects of air-borne pollutants on humans include damage to eyes, respiratory and nervous systems, and a number of teratogenic, carcinogenic and mutagenic effects (Fatima et al.

2001). Genetic damage when present in the cells of the germline, may be expressed many generations after the primary induction of the genetic change – the mutation.

Because it has direct contact with the ambient environment, the respiratory tract is often the site of injury from occupational exposures. Inhalation of potentially toxic materials in the work place can lead to all major lung diseases. Exposure to asbestos minerals has been associated with a wide variety of adverse health effects including lung cancer, pleural mesothelioma and cancer of other organs. Cement making is inevitably a dusty operation as it is much concerned with hot dry powders. Various operations where stone or raw material is crushed in the cement factory produce fugitive dust emissions. Mutagenicity in a complex mixture may often be enhanced not only by potential mutagens but also by the action of “co-mutagens” which are not themselves mutagenic.

Exposure to Portland cement dust has long been associated with the prevalence of respiratory symptoms and varying degrees of airway obstruction in man. Apart from respiratory diseases, it was also found to be the cause of lung and laryngeal cancer, gastrointestinal tumours and dermatitis (Vestbo and Rasmussen 1990; Jakobsson et al. 1993; Yang et al. 1996; Abu Dhaise et al. 1997; Noor et al. 2000; Algranti et al. 2001; Al-Neaimi et al. 2001; Laraqui et al. 2001; Stern et al. 2001).

The present study was aimed at evaluating and predicting the risk of haematological and cytogenetic abnormalities in subjects exposed to cement dust.

METHODS

Subjects for the present study were selected among the workers of a cement factory situated at Coimbatore, Tamil Nadu, India. Personnel working at the crusher, mill and packing sections were selected. The subjects were grouped according to the period of exposure to cement dust as Group I (< 10 years of exposure), Group

II (11 to 20 years of exposure) and Group III (> 20 years of exposure). 30 workers from Group I, 40 from group II and 25 workers from Group III were selected. Equal number of subjects with normal physical and mental health were selected to serve as control subjects. The blood and sera of the exposed and control subjects were used for analysis of various haematological and cytogenetic parameters. To assess the potential of cytogenetic determination on peripheral blood lymphocytes as a means of monitoring human populations subject to low level occupational and environmental exposures to chemical mutagens, the mean frequencies of chromosomal aberrations (CA), mitotic index (MI) and sister chromatid exchanges (SCE) were determined. For calculating the mitotic index (MI), 2000 cells were screened in each exposed group and the results are given as the percentage of cells in division.

RESULTS

As shown in Table 1, an overall decrease in the mean erythrocyte count was observed in all the three groups and the decrease in mean erythrocyte count in the Group III exposed subjects was statistically significant. The mean leucocyte count in exposed subjects showed an increase in their count over the control subjects. In the analysis of differential count of leucocytes, the count of neutrophils and eosinophils did not reveal any statistically significant alteration whereas, the increase in lymphocyte count (in exposed subjects of Groups I and III) and the decrease in monocyte count (in exposed subjects of Group II) were statistically significant. A statistically insignificant decrease in the basophil count was also observed. The Platelet count in the exposed individuals showed mean values that were lower than that of the control subjects, which was statistically significant in Groups I and III.

A decrease in the haemoglobin content was observed in the exposed subjects of all the three groups. However, this decrease was insignificant. The packed cell volume among the various groups of cement industry workers did not show any significant alteration. The mean corpuscular volume (MCV) was increased significantly in all the exposed subjects. The total protein content did not show abnormal levels.

As shown in Table 2, chromosomal analysis of the various groups of cement industry workers and the respective controls revealed minor chromosomal aberrations (CA). The total number of CA observed in the exposed subjects of

Groups I, II and III were 6%, 10.7% and 19.3% against 2%, 1.33% and 2% observed in the respective control subjects. This result shows a dose-dependant increase in the number of CA with increase in period of exposure to cement dust.

As shown in Table 3, the MI observed in the control cultures of the mild, moderate and high risk groups were 7.9 ± 0.59 ; 7.3 ± 0.52 and 7.5 ± 0.58 , whereas the exposed samples of mild, moderate and high risk groups expressed 5.6 ± 0.48 ; 4.8 ± 0.39 and 4.9 ± 0.42 , respectively. A decreasing trend in the MI of the different groups with different periods of exposure was observed. Statistically significant results over that of the controls was observed in all the three groups. An inversely proportional relationship i. e., decrease in MI with increase in period of exposure was inferred.

As shown in Table 4, the results express increased trend in SCEs in different groups of exposed groups when compared with that of the control groups and the increase was statistically significant in the exposed groups I and III. Thus it can be inferred from the results that cement dust has an adverse effect on the frequency of SCEs when duration of exposure is increased.

DISCUSSION

Haematological parameters are a sensitive index to changes in ecological conditions and can constitute an important diagnostic tool in toxicological studies. The decrease in RBC count in exposed subjects in the present study is indicative of microcytic anemia. It was observed in the study that there was an increase in white blood cell count regardless the period of exposure and may be, only a short exposure period is necessary to stimulate the excess formation of WBC's. Immune responses and reactions may have led to lymphocytosis in the exposed groups I and III. The decrease in Monocyte count observed in the present study may be due to increased use or self-destruction or phagocytosis.

A reduced platelet count in the exposed groups of the present study may be due to the anaemic condition in the subjects. The present investigation revealed a decrease in haemoglobin concentration and packed cell volume in the blood samples of all exposed groups, which again, is a sign of anaemic condition. An increased MCV observed in the present study is indicative of anaemia of macrocytic type.

Table 1: Changes in haematological parameters in various groups of cement industry workers and controls.

S. No.	Particulars	No. tested	RBC (Million/ cumm)	WBC (Thousand/ cumm)	Mean values (Mean $\pm$ SD) of parameters										MCV (fl)	Total protein contents (g/dL)
					Neutros- phil	Lympho- cytes	Differential leucocyte count (%)			Planet (lakhs/cu. mm)	Hamoglobin (g/dL)	PCV (%)				
							Eosino- phils	Baso phils	Mono cytes							
1.	Group I	Control	30	5.7 $\pm$ 0.34	7750 $\pm$ 559	59.6 $\pm$ 10.99	30.2 $\pm$ 2.86	36 $\pm$ 0.54	0.6 $\pm$ 0.54	3.6 $\pm$ 0.54	3.2 $\pm$ 0.48	14.06 $\pm$ 1.04	46.98 $\pm$ 4.09	81.8 $\pm$ 2.38	5.6 $\pm$ 0.42	
		Exposed	30	5.35 $\pm$ 0.54	8800 $\pm$ 721	54 $\pm$ 8.04	39.8 $\pm$ 5.31*	3.2 $\pm$ 1.09	0.4 $\pm$ 0.54	3.2 $\pm$ 0.44	2.45 $\pm$ 0.33*	13.45 $\pm$ 0.73	45.18 $\pm$ 2.01	90.9 $\pm$ 1.81*	5.27 $\pm$ 0.92	
2.	Group II	Control	40	5.62 $\pm$ 0.11	7730 $\pm$ 852.6	60 $\pm$ 9.843	0.4 $\pm$ 3.64	3.4 $\pm$ 0.54	0.4 $\pm$ 0.54	3.2 $\pm$ 0.83	3.14 $\pm$ 0.29	15.2 $\pm$ 0.75	46.8 $\pm$ 5.76	81.25 $\pm$ 2.98	5.59 $\pm$ 0.37	
		Exposed	40	5.192 $\pm$ 0.4	8960 $\pm$ 3036.9	66.2 $\pm$ 9.443	2.6 $\pm$ 8.79	2.2 $\pm$ 1.64	0.2 $\pm$ 0.44	0.4 $\pm$ 0.54*	2.42 $\pm$ 0.82	14.44 $\pm$ 1.6	47.04 $\pm$ 3.84	90.48 $\pm$ 2.47*	5.68 $\pm$ 1.5	
3.	Group III	Control	25	5.64 $\pm$ 0.11	7800 $\pm$ 1021.6	60.166 $\pm$ 8.86	29.83 $\pm$ 3.86	3.5 $\pm$ 0.54	0.5 $\pm$ 0.54	2.83 $\pm$ 0.75	3.06 $\pm$ 0.29	14.88 $\pm$ 0.94	48.4 $\pm$ 3.5	79.6 $\pm$ 6.18	5.6 $\pm$ 0.306	
		Exposed	25	4.95 $\pm$ 0.23*	8650 $\pm$ 973.1	56.83 $\pm$ 5.70	37.66 $\pm$ 5.81*	3.66 $\pm$ 1.18	0.16 $\pm$ 0.40	1.66 $\pm$ 1.63	2.23 $\pm$ 0.28*	13.83 $\pm$ 0.52	43.98 $\pm$ 1.36	88.8 $\pm$ 3.77*	6.62 $\pm$ 1.13	

* Values significant at 5% level

1 Group I Mild Risk (>10yrs of exposure)

1 Group II Moderate Risk (11-20 yrs of exposure)

1 Group III High Risk (exposure of 21 yrs and above)

Table 2: Frequency of chromosomal aberrations in lymphocytes of cement industry workers and controls

S. No.	Particulars	No. of subjects examined	Number of metaphase plates screened	Chromatid type aberration			Total number of chromosomal aberrations
				Gaps	Breaks	Fragments	
1.	Group I						
	Control	30	150	2	-	1	3 (2%)
	Exposed	30	150	5	1	3	9 (6%)
2.	Group II						
	Control	40	150	2	-	-	2 (1.33%)
	Exposed	40	150	8	2	6	16 (10.7%)
3.	Group III						
	Control	25	150	3	-	-	3 (2%)
	Exposed	25	150	16	7	6	29 (19.3%)

Period of Exposure: Group I (<10 yrs) Group II (11-20 yrs) Group III (21 yrs and above)

Table 3: Mitotic Index in the exposed subjects working in the cement factory and controls

S. No.	Particulars	Total of subjects examined	Total No. of cells screened	Total No. of SCEs dividing cells	Mitotic Index
1.	Group I				
	Control	30	2000	237	7.9 ± 0.59
	Exposed	30	2000	168	5.6 ± 0.48*
2.	Group II				
	Control	40	2000	219	7.3 ± 0.52
	Exposed	40	2000	144	4.8 ± 0.39*
3.	Group III				
	Control	25	2000	225	7.5 ± 0.58
	Exposed	25	2000	147	4.9 ± 0.42*

*Values significant at 5% level

Period of Exposure: Group I (<10 yrs) Group II (11-20 yrs) Group III (21 yrs and above)

Table 4: Sister Chromatid Exchanges (SCE) in the exposed subjects working in the cement factory and the controls

S. No.	Particulars	Total no. of subjects analysed	Total no. of cells examined	Total No. of SCEs	SCE/Cell ± SD
1.	Group I				
	Control	30	450	510	1.13 ± 0.23
	Exposed	30	450	630	1.40 ± 0.29*
2.	Group II				
	Control	40	600	730	1.22 ± 0.20
	Exposed	40	600	860	1.43 ± 0.27
3.	Group III				
	Control	25	375	440	1.17 ± 0.19
	Exposed	25	375	530	1.41 ± 0.29*

*Values significant at 5% level

Period of Exposure: Group I (<10 yrs) Group II (11-20 yrs) Group III (21 yrs and above)

In the cytogenetic analysis, the frequency of chromosomal aberration was found to increase with the increase in period of exposure to cement dust. Hence there is a directly proportional relationship between the frequency of chromosomal aberrations and the period of exposure to ce-

ment dust. Hadnagy et al. (1986) emphasized that small quantities of air-borne particulate matter from polluted areas equivalent to an air-volume of about 1-2m³, are capable of inducing cytogenetic effects. A significant increase of sister chromatid exchanges and high frequency cells as well

as decreased cell kinetics and significant increase of chromosomal aberrations in men, environmentally and occupationally exposed to air-borne pollutants was reported by Motykiewicz et al. (1992). Calcium silicate, a component of most silicious dusts including that of cement, significantly increased the frequency of chromosomal aberrations and sister chromatid exchanges (Aslam et al. 1993).

It was shown previously that asbestos samples collected from an asbestos factory enhanced sister chromatid exchanges (SCEs) and chromosomal aberrations *in vitro* using human lymphocytes (Rom et al. 1983; Fatima et al. 1991; Fatima et al. 2001). Asbestos workers had a raised mean SCE rate and increased numbers of chromosomal aberrations compared with a control population. Most of the chromosomal aberrations were chromatid gap and break types (Trosic, 1997; Fatima et al. 1991; Fatima et al. 2001). All extracts of particulates induced a significant increase of sister-chromatid exchanges (SCE) in a dose-dependent manner. Samples from the industrialized area revealed the highest activities (Hadnagy et al. 1989).

Hence, it is inferred from the CA, SCE and MI studies that due to the long term exposure of the subjects to cement dust, the percentage of CA and SCE increased while that of the MI decreased. It is evident from the results recorded in this study and the same confirms the mild hemato and cytogenetic toxicity of cement dust on human subjects exposed to the toxicant.

CONCLUSIONS

On the basis of the results obtained in the present study and previously published reports, it may be concluded that in the cement factory, where the workers are exposed to cement and other dusts, there are significant haemotoxic and genotoxic changes which need to be elucidated so that the after-effects of toxicant contamination may be monitored and eliminated. This will not only help in the protection of the genetic material of future generation, but also in the prevention of malignant diseases in the present populations working in the factories as well as those residing in the industrial area.

ACKNOWLEDGEMENTS

Authorities of Bharathiar University, Coimbatore. Authorities and Medical Officer, Cement Companies, Coimbatore.

REFERENCES

- Abu Dhaise BA, Rabi AZ, al Zwaury MA, el Hader AF, el Qaderi S 1997. Pulmonary manifestations in cement workers in Jordan. *Int J Occup Med Environ Health*, **10**: 417-428.
- Al-Neaimi YI, Gomes J, Lloyd OL 2001. Respiratory illnesses and ventilatory function among workers at a cement factory in a rapidly developing country. *Occup Med (Lond)*, **51**: 367-373.
- Algranti E, Mendonca EM, DeCapitani EM, Freitas JB, Silva HC, Bussacos MA 2001. Non-malignant asbestos-related diseases in Brazilian asbestos-cement workers. *Am J Ind Med*, **40**: 240-254.
- Aslam M, Fatima N and Rahman Q. 1993. Cytotoxic and genotoxic effect of calcium silicates on human lymphocytes in vitro. *Mut Res*, **300**: 45-48.
- Fatima N, Jain AK, Rahman Q 1991. Frequency of sister chromatid exchange and chromosomal aberrations in asbestos cement workers. *Br J Ind Med*, **48**: 103-105.
- Fatima SK, Prabhavathi PA, Padmavathi P, Reddy PP 2001. Analysis of chromosomal aberrations in men occupationally exposed to cement dust. *Mut Res*, **490**: 179-186.
- Hadnagy W, Seemayer NH, Tomingas R 1986. Cytogenetic effects of airborne particulate matter in human lymphocytes in vitro. *Mut Res*, **175**: 97-101.
- Hadnagy W, Seemayer NH, Tomingas R, Ivanfy K 1989. Comparative study of sister chromatid exchanges and chromosomal aberrations induced by airborne particulates from an urban and highly industrialised location in human lymphocyte cultures *Mut Res*, **225**: 27-32.
- Jakobsson K, Horstmann V, Welinder H 1993. Mortality and cancer morbidity among cement workers. *Br J Ind Med*, **50**: 264-272.
- Laraqui CH, Laraqui O, Rahhali A, Harourate K, Tripodi D, Mounassif M, Yazidi AA 2001. Prevalence of respiratory problems in workers at two manufacturing centers of ready-made concrete in Morocco. *Int J Tuberc Lung Dis*, **5**: 1051-1058.
- Motykwicz, Michalska J, Pendzich J, Perera FP, Chorazy M 1992. A cytogenetic study of men environmentally and occupationally exposed to airborne pollutants. *Mut Res*, **280**: 253-259.
- Noor H, Yap CL, Zolkeoli O, Faridah M 2000. Effect of exposure to dust on lung function of cement factory workers *Med J Malaysia*, **55**: 51-57.
- Rom WN, Livingston GK, Casey KR, Wood SD, Egger MJ, Chiu GL, Jerominski L 1983. Sister chromatid exchange frequency in asbestos workers. *J Natl Cancer Inst*, **70**: 45-48.
- Stern F, Lehman E, Ruder A 2001. Mortality among unionized construction plasterers and cement masons. *Am J Ind Med*, **39**: 373-388.
- Trosic I, Brumen V, Horvat D 1997. in vitro assessment of asbestos fibers genotoxicity. *Zentralbl Hyg Umweltmed*, **199**: 558-567.
- Vestbo J, Rasmussen FV 1990. Long-term exposure to cement dust and later hospitalization due to respiratory disease. *Int Arch Occup Environ Health*, **62**: 217-220.
- Yang CY, Huang CC, Chiu HF, Chiu JF, Lan SJ, Ko YC 1996. Effects of occupational dust exposure on the respiratory health of Portland cement workers. *J Toxicol Environ Health*, **49**: 581-588.