Bull. Environ. Contam. Toxicol. (1995) 55:704–708

Environmental Contamination and Toxicology

Frequencies of Sister Chromatid Exchanges in Lymphocytes of Portland Cement Factory Workers

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Received: 6 March 1995/Accepted: 6 June 1995

Cement factory is one of the major sources of air pollution. Right from the stage of quarrying the limestone, crushing and despatch upto the stage of clinker formation, grinding of clinker and filling up of cement bags, enormous quantity of dust and particulate matter are generated at every step. Greater fraction of particles emitted are below 10 microns in size which have the potential of entering the deepest portion of respiratory system. Since most of the workers in cement factory do not take adequate protective measures they come into direct contact with cement dust. The exposure is mainly by respiratory and dermal routes and to a lesser extent by ingestion.

Portland cement dust is a mineral dust, having silicates and aluminates of lime as its main constituents. Oxides of iron, magnesium, sodium, potassium, phosphorous and sulphur are its minor constituents. Chromium is also present in Portland cement (Prodan 1971). Portland cement is different from asbestos cement as the former does not contain asbestos. There are several reports on the adverse effects of cement dust in animals (Pimentel and Gomes 1973; Wozniak and Wiecek 1984) and in occupationally exposed workers (Cortez Pimentel and Peixoto Menezes 1978; Dudkiewicz et al. 1983; *Shehla et al. 1995*). An increased risk of cancer especially laryngeal cancer (Maier et al. 1990), colorectal cancer (Jakobsson et al. 1990) and right sided colon cancer (Jakobsson et al. 1994) has been reported among cement factory workers. There exists a relationship between carcinogenesis and mutagenesis therefore, it becomes obligatory to evaluate the mutagenic potential of cement dust. An attempt has thus been made to study the frequency of sister chromatid exchanges (SCEs) in peripheral lymphocytes of the exposed workers.

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MATERIALS AND METHODS

Intravenous blood was collected from 59 non-smokers (age range 24-54) who had varied duration of exposure (1-17 years) to cement dust. They constitute the exposed group. For comparison blood was collected from 59 non-smokers (age range 22-52 years, control group) who were not exposed to cement dust. Relevant information on the background of each individual was collected using a standard questionnaire. The information included smoking history, medication and drug use, nutrition and exposure to toxic chemicals. Individuals reported to have had radiation exposure either for therapy or diagnosis or for medication and drug use were not used for the analysis. Smokers were excluded in the present study. The workers were basically non alcoholic and were on a normal diet. The workers did not use protective hand gloves or face mask and were infact heavily powdered with cement dust by the end of their work. They worked in the factory for 8 hours per day throughout the year.

Whole blood (0.5 mL) of each sample was cultured in RPMI 1640 medium supplemented with 25% human AB serum, 0.5% phytohemagglutinin-P and 0.25% antibiotic. 3ug/mL of bromodeoxyuridine (BrdU) was added to the cultures at the time of initiation. The culture vials were then wrapped in black paper and incubated at 37°C for 72 hours. Duplicate cultures were maintained for each sample. Colchicine (0.1 ug/mL) was added two hours before harvesting the cultures, to arrest the cell cycle at metaphase. Cultures were harvested and slides were prepared according to the standard method (Moorhead et al. 1960). The slides were coded and kept in dark for aging.

Three-day-old slides were stained using the fluorescence plus Giemsa technique of Perry and Wolff (1974). 50 well spread metaphases per sample were scored for sister chromatid exchanges. Statistical analysis of the results was done using Student's t test.

RESULTS AND DISCUSSION

The data on SCEs in workers exposed to cement dust are given in table. The exposed group showed a significant increase in the mean SCE rate per cell (8.88) when compared to control group (3.52). The frequency of SCEs per cell was 6.98, 9.70 and 10.74 in the groups exposed to cement dust for 1-5, 6-11 and 12-17 years respectively. The exposed groups with varying time intervals showed a significant increase in SCEs when compared to control group. A significant increase in

SCEs was observed when 1-5 years exposed group was compared with 6-11 and 12-17 years exposed groups.

Table. Frequency of sister chromatid exchanges (SCEs) in workers exposed to cement dust in cement factory.

Duration of exposure (years)	No. of samples	No. of meta-phases screened	Total No. of SCEs	SCE/Cell
Control Group (unexposed)	59	2950	10384	3.52
Exposed groups				
1-5	20	1000	6980	6.98*
6-11	33	1650	16005	9.70*
12-17	6	300	3222	10.74*
Total 1-17	59	2950	26207	8.88*

50 metaphases were analysed for each sample.
* $p < 0.05$

The results have clearly presented evidence for the clastogenic effect of cement dust. The workers are exposed to the various oxide components of cement such as calcium, aluminium, silica, iron, titanium, chromium etc. As it is difficult to pin point a particular element responsible for chromosomal damage, the mutagenic effect on the workers might be attributed to the cumulative effect of these components. Earlier reports have presented evidence for the mutagenic potential of aluminium (Ajoy et al. 1990) and silica (Sobti et al. 1991). Aluminium is reported to have a very high affinity for DNA, RNA (Karlik et al. 1980; Dyrssen et al. 1987) besides its interaction with microtubule aggregation in vitro (Mac Donald et al. 1987). In Portland cement, hexavalent chromium is present which is an established carcinogen (IARC 1980). There is sufficient evidence for carcinogenicity among cement factory workers (Rafnsson and Johannesdottir 1986; Jakobsson et al. 1993).

SCE analysis is reported to be a sensitive indicator of genetic damage (Perry and Evans 1975). It has been reported that the frequency of SCE is dramatically increased when cells, or animals, including human beings, are exposed to mutagens and carcinogens

(Latt et al. 1981; Lambert et al. 1982). As there is a close relationship between carcinogenesis and mutagenesis, there is every reason to suspect cement dust to be a potentially hazardous mutagen as well. Thus the increase in the frequency of sister chromatid exchanges in the exposed group may be a consequence of the mutagenic effect of cement dust. Our results suggest that undue exposure of man to cement dust might result in genetic damage. However, studies in different test systems using a battery of protocols are warranted to generate more data on the effects of cement dust.

Acknowledgments. The authors thank the workers and management of the cement factories for their co-operation. The authors also thank Dr. P. Usha Rani and Dr. A. Jyothi (Institute of Genetics) for their help.

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