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SODIUM FLUORIDE INDUCED GENOTOXICITY IN MICE (MUS MUSCULUS) BONE MARROW CELLS

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ABSTRACT

Fluoride is an environmental pollutant, which induce various anomalies such as skeletal fluorosis, osteoporosis, dental fluorosis and arthritic pains. In an invivo genotoxicity investigation of the action of fluoride one bone marrow cells of mice, take laboratory in bred albino swiss mice (Mus musculus) of average body weight 25-30g were fed orally 1 ppm and 2 ppm for 14 days. Among randomly 300 metaphase plates screened, both individual and gross type chromosome abnormalities were observed. Among individual type or structural type (10.66 %) and gross type (3.00%) respectively at 1 ppm and at 2 ppm (21.66%) structural and (5.33%) gross type in compare to control (2.66%) structural and (1.33%) gross type obnormalities respectively. The increase in frequency of chromosomal anomalies was mainly due to significant increase in both gross and structural or individual type in compare to control. Thus the effect was does dependent.

Key word – Fluoride, Pollutant, Anomalies.

INTRODUCTION:

Fluoride is an environmental pollutant ubiquitously distributed in our environment. It is originating from anthropogenic source as steel, aluminium, ceramics and phosphate fertilizer production as well as coal combustion (Smith & Hodge ; 1979 , Sharma, et.al., 2007), present in air water and fertilizer residue in almost every edible materials that we consume in our daily life (Sheth et.al.,1994). Water is ordinary the principle source of fluoride. More than 90% of natural fluoride content of soil is insoluble or tightly bound to soil ,particles ,which in turn disrupt the activity of microbial species (which are responsible for soil fertility), ultimately effecting the natural life supporting system (Pearce, 2006). Pollution may damage at two levels. Cell and chromosome level leading to mitotic poising and or chromosome structure changes respectively. Bio mutagens induce mitotic poisoning (Awasthy et. al., 2000), while chemical mutagens induce chromosomal abnormalities (gross and structural abnormalities). Fluoride in drinking water is easily absorbed by the intestine (Chinoy and

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Narayan; 1992) and is quickly distributed throughout the body. Ingestion and absorption of excessive amount of fluoride produce toxic effect on biota including human beings (Sharma, 2007; Poddar, 2008). There is more report of accidental intoxication caused by sodium fluoride than by any other fluoride compound (Mathur, 2013).

Fluoride ranges from 0.7 to 4.5ppm and induce various anomalies such as skeletal fluorosis, osteoporosis, cancer and arthritic pains. Fluoride predominantly affects the skeletal system, teeth, structure and function of skeletal muscle, brain and spinal cord (Shashi et. al., 1992, Chaurasia, *et.al.,* 2007). It also inhibits the synthesis of DNA protein and cell proliferation (Kaminsky *et. al.,* 1990; Godfrey and Watson 1988; National Toxicology programme (NTP); 1990). Fluoride also interferes with phagocytosis and induce release of super oxide free radicals which damage the body leading to further acceleration of ageing process (Yiamouyiannis and Gerald 1993) .The harmful effect of fluoride is not local is a global problem (Chaurasia *et.al.,* 2005). Larger problems are anticipated in other countries including china, Uzbekistan and Ethiopia etc (Pearce, 2006). In India, the problem of excess fluoride in water has been noticed in 17 states (Sharma 2007) which is prominent among them being Andhra Pradesh, Haryana, Bihar, Karnataka, Kerela. Gujrat, Madhya predesh, Maharastra, Rajasthan, Tamilnadu, Punjub and U.P According to Bihar Govt. 8188 villages in 11 district have excess fluoride in ground water (A.K.Susheela, 1987) cause toxicity. Therefore the present has been undertaken to study the genotoxicity of fluoride in mice.

MATERIALS AND METHODS:

Laboratory inbred albino Swiss mice (Mus musculus) of average body weight 25-30 g were fed orally with 1 ppm and 2 ppm NaF @ 0.5 ml/day/mice for 14 consecutive days. Animals injected with 0.04% colchicines @ 1 ml/100gm /bwt interaperitonial before sacrificing. The bone marrow from both the femurs were flushed in hypotonic solution, incubated at 37^0C for 20 minutes, centrifuged and fixed in acetomethanol fixative. The slides were prepared by flame drying Giemsa staining technique (Person et.al.,1987, Chaurasia *et.al.,* 2007), 300 well spread metaphase plates were screened randomly. Number of abnormalities were calculated and interpreted on the basis of statics.

RESULTS AND DISCUSSION

Among 300 metaphase plates the total frequency of abnormal metaphase in the NaF treated group at 1 ppm were (13.66 ± 1.98) of which structural abnormalities (10.66 ± 1.78) and gross type (3.00 ± 0.97) and at 2 ppm (27.00 ± 2.56) of which structural abnormalities (21.66 ± 2.37) and

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gross type (5.33 ±1.25) respectively. As compare to control group the frequency of total abnormalities were (4.00 ± 1.13) of which structural abnormalities (2.66 ± 0.92) and gross type (1.33 ± 0.66) respectively (Table - 01). Thus the effect was does dependent, both individual and gross type of abnormalities observed. Among the structural type abnormalities, chromatid breaks. Chromatid gap, minute fragment etc while, the gross type abnormalities were polyploidy, hypoploidy, stickiness and clumping. The chromosomal damage induced by sodium gluoride was probably resulted with induction of gross and individual type of abnormalities. This metagenic substance might be producing the damage at two different level first by affecting the internal milieu of the cell and second by affecting the chromosome morphology. The gross type of changes might be due to the damage at protein level either on spindle protein or on protein packing production of electrophilic ions and reactive such as the super oxide radical (O2) and hydroxyl radical (OH) during the metabolism of mutagenic substance (Kolpman *et.al.,* 1985). Fluoride can induce the production of free radicals which can damage DNA stands directly or by lipid peroxidation initiated by free radicals (Wang *et.al.*, 2004; Lobo *et.al.,* 2010). Thus, sodiam fluoride induced genotoxicity (damage by a physical or chemical agent to genetic material such as chromosome or DNA) in the bone marrow cells of mice.

Table – 1 Frequency of chromosomal abnormalities (%±S.E) of sodium fluoride treated bone marrow cells in mice after 14 days exposure (n=300 metaphase)

Chromosome Abnormalities						
Experiment	Individual		Gross		Grand total	
	No	%±S. E	No	%±S. E	No	%±S. E
Control	8	2.66±0.92	4	1.33±	12	4.00±1. 13
1ppm	32	10.66±1.78	9	3.00±	41	13.66±1.98*
2ppm	65	21±2.37*	16	5.33±	81	27.00±2.56*

CONCLUSION:

Fluoride pollution is a global problem and induces genotoxicity. The high level of fluoride in drinking water cause major health problems. So awareness for such harmful effect by non-permissible dose (0.5 ppm) of sodiam fluoride containing water should be made. The govt. or any authorized agency should conduct the survey of that area where such pollution exits. In order to maintain quality of water and prevention of excess fluoride content , water quality and regular health check-up awareness programme are recommended in that area. A preventing measure may be advised for people of that area to take any antioxidants regularly, which may save the population from toxic effect. Since the toxicity caused by this chemical increase formation of free reactive radicals that is totally responsible for their toxicity.

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