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Genetic Markers in Relation to Different Exposures

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The increasing load of chemical effluents in soil, water and air, following industrialisation is potentially harmful to exposed populations. Many toxicants damage the cellular systems at different levels, leading to non-specific cell death. In addition, others produce deleterious effects on genetic elements when exposed at subtoxic doses. The increase in incidence of genetic disease has been attributed to environmental agents in international meetings as well (ICPEMC 1983; ICEM 1989; *INDO-US Symposium 1990*).The mutagenic effect of chemicals on human health has been shown to be high, if the extent of exposure is sufficient to produce genotoxic action on critical cellular targets (Sorsa et al. 1982; Banerjee et al. 1987; Nriagu and Pacyna 1988). The present study was carried out in two groups of populations from different industries; (i) directly exposed through occupation and (ii) indirectly exposed to effluents from factories, like various heavy metals (Cu, Zn, Ni, Pb), H_2SO_4 fumes, oil fumes, silicate, Va, Cr, Mg-oxide, carbon-monoxide, oxyacetylene gas and γ-irradiation. Certain genetic markers of protein and enzymes were studied. The influence of other factors like nutritional status, addiction and socio-economic status was assessed.

MATERIALS AND METHODS.

Blood samples were collected from one hundred and eleven healthy donors working in the two factories (A and B) near Calcutta, West Bengal. The workers were grouped into Group I and Group II.

Group I - directly exposed population such as workers engaged in electroplating, grinding and painting and fitters and millers.

Group II - indirectly exposed group comprised of workers mainly involved in storehouse, security and office work.

Table 1 gives the age and group distribution. Sera were separated by centrifugation and analyzed following vertical polyacrylamide

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disc gel electrophoresis technique of Ornstein and Davis with minor modifications for polymorphisms of haptoglobin (Hp), transferrin (Tf), lipoprotein (Lp), total protein (Tp) and the enzyme lactate dehydrogenase (LDH). The staining schedules and phenotypic patterns have been standardized in earlier publications (Ornstein 1964; Davis 1964; Baksi-Sanyal et al. 1979; Swain et al. 1980a; Swain et al. 1980b).

RESULTS AND DISCUSSION.

Data obtained on age, years of exposure, nutritional status and addiction are presented in Table 1. The majority of workers had over 20 years exposure. The nutritional status was average to good. Addiction to tobacco and alcohol was also noted. Table 2 shows the variations in genetic markers of the two groups of populations from the two factories A and B.

No appreciable differences could be observed between the two populations in the incidence of different genotypes of the stable genetic markers; total protein, haptoglobin, transferrin and the enzyme lactate dehydrogenase (Table 3). Two cases showed abnormal C-D type transferrin in the directly exposed group in Factory A and Factory B.

Screening through electrophoresis showed a higher frequency of abnormal lipoprotein (Lp) variants in directly exposed group (52.38%) of Factory A than in indirectly exposed populations (47.05%). The abnormal lipoprotein variants in directly and indirectly exposed populations of Factory B were however 10% and 14.28% respectively (Table 2).

The expression of genetic markers not related to life style or other environmental factors like Hp, Tf, Tp and LDH was more or less similar in the two populations and could not be related to the degree of industrialisation. The Hp^2 gene is predominant in this study. In transferrin, C-C type was common in most of the cases and this is the most common form observed in India (Thomas et al. 1977).

The total protein patterns maintained a general similarity except in the variable number of pre-albumin bands.

The lipoprotein variants observed were mainly type IIa in exposed group and type IIb in non-exposed population. The expression of the lipoprotein variant has been related to dietary oil to a certain extent in the Eastern India (Bhattacharya et al. 1979) which is known to intensify the genotoxic effects of certain chemicals (Gajra et al. 1981).

The distribution of haptoglobin pattern have been related with ethnic groups (Harris 1975; Roychoudhuri 1983). The differences observed here may be related to the racial groups. LDH showed normal three bands except in two cases in directly exposed group of Factory A and Factory B and in indirectly exposed group of Factory A.

Table 1. Detailed information on socio-economic status, exposures and addictions of different populations.

	Total No. of samples	Age group (in yrs.)		Years of exposure			Nutritional status		Addiction			
		20-40	41-60	5-10	11-20	> 20	Average	Good	Tobacco chewing	Smoking	Alcohol	None
Factory-A												
Group-I	22	9	13	3	7	12	11	11	7	11	1	8
Group-II	18	4	14	2	6	10	10	8	9	10	-	3
Factory-B												
Group-I	50	1	49	-	3	47	40	10	29	30	-	4
Group-II	21	2	19	-	3	18	9	12	7	13	1	6

Table 2. Different genetic markers in populations with different levels of industrial exposure

	Haptoglobin (Hp)			Lipoprotein (Lp) (Fredericksson's classification)				LDH		Transferrin (Tf)		Total protein (Tp)	
	2-2	2-1	1-1	Normal	IIa	IIb	IV	Normal	Abnormal	Normal	Abnormal	Pre-albumin Present	Pre-albumin Absent
Factory-A													
Group-I	14	7	-	10	6	4	1	20	2	16	2	7	11
Group-II	12	2	1	9	1	6	1	17	1	14	-	4	11
Factory-B													
Group-I	31	17	2	45	3	1	1	48	2	48	2	25	25
Group-II	14	6	1	18	1	1	1	21	-	21	-	11	10

Table 3. Chi-square values between different populations

Comparison between	Chi-square values				
	Hp	Lp	LDH	Tf	Tp
Factory-A					
Group- I & Group-II	2.998	0.106	0.178	1.676	0.55
Factory-B					
Group- I & Group-II	0.211	0.268	0.852	0.861	0.033

Chi-square values did not show any significance ($P > 0.05$) in all the samples tested.

The data for genetic variants thus did not show significant alterations between directly and indirectly exposed groups so far as Hp, Tf, LDH, Tp, Lp were concerned. The stability of these genetic markers may also be attributable to the protective action of better nutrition and life style of the populations due to their better socio-economic status. The rare TfD in directly exposed population has to be followed up to establish the fact that it is a fresh mutant by studying the parents. The frequencies of the relatively stable polymorphisms compared to known background population data is thus not yet altered. It is also necessary to obtain blood levels of the different pollutants (mainly metals in this case) to obtain further information. The protective effect of diet cannot be over-emphasised not only as it is based on experimental evidence but also since it may be most important factor in maintaining the low mutation load of exposed populations. Previous studies have shown that the incidence of chromosomal aberrations in industrially exposed populations is significantly higher than unexposed controls (Ghosh 1988; Roy 1987). Therefore, there is no basis of complacency based upon the present data and further studies on larger populations need to be carried out for monitoring for genotoxic effects.

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