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Cytogenetic analysis of peripheral blood lymphocytes in workers exposed to vinyl chloride

L. Hrivnak, Z. Rozinova, S. Korony and E. Fabianova

Regional Institute of Hygiene, 975 56 Banska Bystrica (Czechoslovakia)

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Summary

In the present study, the method of cytogenetic analysis of peripheral blood lymphocytes was used to examine 43 workers exposed to vinyl chloride monomer (average exposure 11.2 years) and 22 subjects selected from the same locality (control group). A total number of 8650 metaphases were analysed. All cytogenetic parameters examined were increased in the exposed group as compared to the control group and 3 parameters, chromatid breaks, percentage of aberrant cells and breaks per cell, were significantly increased ($P < 0.001$).

Vinyl chloride monomer (VCM) is a basic component for the production of polyvinylchloride (PVC). PVC, with an estimated yearly production of 16 million tonnes in 1980 (Groth et al., 1981), is in daily contact with the human population. In exposed workers VCM leads to an increased incidence of angiosarcomas of the liver, brain tumours, malignancies of the haema.opoetic system and cancer of the lungs (IARC, 1979; Buffer et al., 1979; Nicholson et al., 1975). Similar results were obtained in experimental animals (Suzuki, 1978, 1981).

Increased frequencies of chromosomal aberrations in exposed workers indicate that VCM has mutagenic effects in man (Ducatman et al., 1975; Kučerová et al., 1979). We analysed the chromosomes of peripheral lymphocytes of workers exposed to VCM. The maximum allowable concentration for VCM in Czechoslovakia is 10 mg/m^3.

Materials and methods

The exposed group consisted of 43 workers (average age 34.4 ± 8.5 years), with an average VCM exposure of 11.2 ± 7.2 years. According to measurements at the work place during the last 6 months before the blood was sampled the VCM concentrations varied from 2 to 40.9 mg/m^3. The 22 subjects of the control group had similar social habits and living environments, but they were not exposed to VCM or other known mutagenic agents. All subjects who had been ill or taken medicines (including X-ray examinations) during the previous 3 months were excluded.

Correspondence: Dr. L. Hrivnak, Regional Institute of Hygiene, 975 56 Banska Bystrica (Czechoslovakia).

TABLE 1

RESULTS OF CYTOGENETIC ANALYSIS OF PERIPHERAL BLOOD LYMPHOCYTES OF VCM-EXPOSED WORKERS

Group	N	Age (years)	Average exposure to VCM (years)	Number of metaphases analysed	Chromosomal aberrations				Percent of aberrant metaphases (% AB.C)	Breaks per metaphase (B/C)
					B′	B″	E′	G		
Exposed	43	34.4 ± 8.5	11.2 ± 7.2	4650	119 *	52	19	184	3.30 *	0.037 *
Control	22	40.3 ± 8.2	–	2200	24	18	5	55	1.64	0.019

B′, chromatid breaks; B″, chromosome breaks; E′, chromatid exchanges; G, gaps
* $P < 0.001$.

Peripheral blood lymphocytes were cultured according to Hungerford (1965). Heparinised blood (0.7 ml) was cultured for 48 h at 37 °C in RPMI medium (USOL, Prague), supplemented with 20% inactivated calf serum (USOL, Prague), antibiotics (penicillin, streptomycin, neomycin) and stimulated with phytohaemagglutinin (PHA, USOL, Prague). Colchicine (0.7 g/ml, Fluka) was added to the culture for the last 2 h of incubation. Cytological preparations were made using a routine protocol and the slides were stained with Giemsa. At least 100 metaphases with 46 centromeres were analysed per person for the presence of chromosomal aberrations.

The following types of chromosomal aberrations were recorded: chromatid breaks (B′), chromosome breaks (B″), chromatid exchanges (E′), gaps (G). Gaps were not considered aberrations. Differences between groups were statistically evaluated using the *t*-test. Relative frequencies were transformed by inverse trigonometric function ($2 \cdot \arcsin \sqrt{f}$) because of their small values (Reisenauer, 1970; Draper and Smith, 1981).

TABLE 2

PERCENT ABERRANT METAPHASES (% AB.C) IN SMOKERS AND NON-SMOKERS

Group	% AB.C	% AB.C	
		smokers	non-smokers
Exposed	3.3 ± 1.09	3.45 ± 1.04 *	3.23 ± 1.12 **
N	43	15	28
Range	1–6	2–5	1–6
Control	1.64 ± 0.9	1.73 ± 0.76 *	1.60 ± 1.0 **
N	22	7	15
Range	0–3	1–3	0–3

* $P < 0.05$; ** $P < 0.001$.

Results and discussion

The results are summarized in Table 1. All aberration types were increased in the exposed group as compared to the control group. The differences were highly significant for chromatid breaks (B′), percentage of aberrant cells (% AB.C) and for breaks per cell (B/C) ($P < 0.001$).

Table 2 shows the percentages of aberrant cells in the groups of exposed and control persons with respect to smoking habits. In both groups there was no difference between smokers and non-smokers. The difference in aberrations given as % AB.C between non-smokers in the exposed and the control groups was highly significant ($P < 0.001$), with respect to the smokers the difference was significant ($P < 0.05$). These data indicate that the main reason for the increased aberration frequencies is the exposure to VCM.

Our data agree with the results of Geryk and Zudová (1986) who reported 4.1% AB.C in VCM-exposed workers.

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