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Short Communication

COMPARATIVE EVALUATION OF THE FREQUENCY OF CHROMOSOMAL ABERRATIONS AND THE SCE NUMBERS IN PERIPHERAL LYMPHOCYTES OF WORKERS OCCUPATIONALLY EXPOSED TO VINYL CHLORIDE MONOMER

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There are now available plenty of authorative data on the carcinogenic [1—3, 13] and mutagenic [1,2,10] activity of vinyl chloride monomer. Increased frequency of chromosomal aberrations detectable in peripheral lymphocytes of chronically exposed workers has been reported by numerous authors [4—6, 11,12].

We have repeatedly analysed, using routine cytogenetic techniques [8], 3 blood samples from each of 9 workers occupationally exposed for 10—27 years to relatively high mean annual doses of vinyl chloride monomer (about 20—150 ppm of air). In the third blood sample series we also used the SCE technique [14] and compared the frequency of detected chromosomal aberrations with the number of SCEs in each sample. The blood samples from 8 healthy persons were used as controls and were collected with the 3rd blood sampling from the vinyl chloride (VC) workers. The control persons were matched for age and sex and they were not exposed to known mutagens during 3 months before the time of blood collection.

The results are summarized in three tables. Tables 1 and 2 show the instability and non-homogeneity in the frequency of chromosomal aberrations over a period of two years. Mostly chromatid and chromosome breaks were detected; chromatid and chromosome exchanges occurred only sporadically.

Table 2 compares the frequency of chromosomal aberrations and the number of SCE per cell in 7 blood samples. There is an apparent increase in both parameters in comparison with control levels.

The mathematical evaluation of our results was calculated only for the 3rd collection. Conventional aberrations in VC workers were non-homogeneous,

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TABLE 1

CHROMOSOMAL ANALYSES OF 9 WORKERS OCCUPATIONALLY EXPOSED TO VINYL CHLORIDE (TYPES OF ABERRATION)

Exposure (years)		Aberrant cells (%)	Number of chromatid breaks /100 cells	Number of chromatid exchanges /100 cells	Number of chromosome breaks /100 cells	Number of chromosome exchanges /100 cells	Number of dispiralizations /100 cells
E.H.	1	11	5	1	4	0	1
25–27	2	0	0	0	0	0	0
	3	11	5	0	4	3	0
J.P.	1	4	4	0	0	0	0
16–18	2	2	0	0	1	1	0
	3	2	1	0	0	1	0
K.K.	1	2	2	0	0	0	0
15–17	2	7	6	1	0	0	0
	3	3	3	0	0	0	0
A.H.	1	1	1	0	0	0	0
14–16	2	1	1	0	0	0	0
	3	6	2	0	4	0	0
M.M.	1	0	0	0	0	0	0
13–15	2	1	0	0	0	1	0
	3	7	3	0	4	0	0
E.J.	1	3	1	1	0	1	0
12–14	2	6	4	0	2	0	0
	3	11	2	1	8	0	0
J.K.	1	1	0	0	1	0	0
12–14	2	4	3	0	1	0	0
	3	3	3	0	0	0	0
F.M.	1	1	1	0	0	0	0
11–13	2	2	1	0	1	0	0
	3	1	1	0	0	0	0
A.T.	1	4	2	0	1	0	1
10–12	2	1	0	0	1	0	0
	3	3	0	0	3	0	0
8 controls (mean)		1.8	1	0	0.8	0.2	0

TABLE 2

CHROMOSOMAL ANALYSES OF 9 W
RIDE

	Exposure		1st c aberr (%) [a]
	years	ppm	
E.H.	25–27	20–150	11
J.P.	16–18	20–80	4
K.K.	15–17	15–70	2
A.H.	14–16	20–150	1
M.M.	13–15	20–90	0
E.J.	12–14	20–150	3
J.K.	12–14	20–90	1
F.M.	11–13	20–90	1
A.T.	10–12	20–90	4
Mean			
8 controls (mean)			

[a] 100 cells were scored per sample.
[b] 50 cells were scored per sample.
[c] Significantly increased in comparison w

induced chromosomal changes
the vinyl chloride monomer.

Our results seem to lead to
routine and SCE — are equall
mutagenicity in vivo. The incr
The sensitivities of the two r
about the same.

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TABLE 3

MEDICAL HISTORY, AND SMOKING

	Diagnostic radiation	Smo
E.H.	–	+
J.P.	–	±
K.K.	+	–
A.H.	–	–
M.M.	–	–
E.J.	–	+
J.K.	–	–
F.M.	–	++
A.T.	–	–

The frequency of SCEs was significantly higher in cells of VC workers in comparison with controls. The level of significance was $P < 0.001$, according to the t test.

The detailed control data are in Table 4. The medical histories, and the smoking and drinking habits of workers tested are presented in Table 3. Smoking and other habits of workers appeared to have no effect on the frequency of any chromosomal changes.

Our positive results are contradictory to findings reported by Hansteen [7] who did not observe any increase either in the frequency of aberrations or in the SCE numbers. However, the subjects examined by this author had been exposed to very low doses of vinyl chloride (0.1 ppm) and only for the relatively short period of two years. This can be taken as a further proof that VCM-

BLE 2

ROMOSOMAL ANALYSES OF 9 WORKERS OCCUPATIONALLY EXPOSED TO VINYL CHLO-

Exposure		1st collection aberrant cells (%) [a]	2nd collection aberrant cells (%) [a]	3rd collection	
ars	ppm			aberrant cells (%) [a]	SCE/cell [b]
27	20- 150	11	0	11 [c]	17 50 [c]
18	20 80	4	2	2	9 56
17	15 70	2	7	3	15 02 [c]
16	20 150	1	1	6	
15	20 90	0	1	7 [c]	15 26 [c]
14	20 150	3	6	11 [c]	16 41 [c]
11	20 90	1	4	3	10.60
13	20 90	1	2	1	12 22 [c]
12	20 90	4	1	3	
				5.2 ± 1.3	13 80 ± 1.13
(mean)				1 8 ± 0 4	9 41 ± 0 40

were scored per sample
were scored per sample
antly increased in comparison with control values

luced chromosomal changes depend both on dose and length of exposure to e vinyl chloride monomer.

Our results seem to lead to a conclusion that the two cytogenetic methods — atine and SCE — are equally suitable for testing of high-dose vinyl chloride utagenicity in vivo. The increase of SCEs in our results is more homogeneous. ie sensitivities of the two methods to detect mutagenic activity seem to be bout the same

BLE 3

DICAL HISTORY, AND SMOKING AND DRINKING HABITS OF WORKERS TESTED

	Diagnostic radiation	Smoking	Drinking	Drugs
I		+	±	Menrobamat
				Papaverin
		+	++	Chloramphenicol
				Syntophyllin
	+		+	-
H			±	Solutan
				Syntophyllin
M			+	-
		+	+	Bellaspon
			+	-
J		++	++	Superpyrin
				Thiospasmin
			+	Korizon

TABLE 1

CHROMOSOMAL ANALYSIS OF 8 CONTROLS

	Aberrant cells [a] (%)	SCE/cell [b]	Smoking	Drinking
M.S.	3	10.88		
J.K.	0	8.22		
V.B.	3	10.30	++	+
J.S.	3	10.16	++	+
K.B.	1	10.46		
M.Ku.	1	8.26	-	-
Z.P.	2	7.94	±	-
R.S.	1	9.02		+
Mean	1.8 ± 0.4	9.41 ± 0.40		

[a] 100 cells were scored per sample.
[b] 50 cells were scored per sample.
The controls were not exposed to X-rays and drugs at the time of blood collection.

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