

A study of chromosomal aberrations in miners exposed to diesel exhausts

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NORDENSON I, SWEINS A, DAHLGREN E, BECKMAN L. A study of chromosomal aberrations in miners exposed to diesel exhausts. *Scand j work environ health* 7 (1981) 14–17. A study was made of chromosomal aberrations in cultured lymphocytes from a group of 14 miners exposed to diesel exhausts and a reference group of office employees and construction workers. No increased frequency of chromosomal aberrations was observed. The frequency of chromosomal aberrations (both gaps and breaks) was, however, found to be associated with smoking.

Key terms: mining, smoking.

The aromatic content of motor fuels, especially benzene, is considered to be a potential health risk in a number of occupations. Increased frequencies of chromosomal aberrations (3) and leukemia (2, 7) have been observed in individuals occupationally exposed to high concentrations of benzene. Fredga et al (5) studied chromosomal aberrations in Swedish workers handling motor fuels. An increased frequency of chromosome aberrations was found in road tanker drivers and industrial workers exposed to benzene, but not in ship tanker crews and filling station staff. It was considered unlikely that exposure to benzene was the main reason for the occurrence of chromosome aberrations in the road tanker drivers, since drivers delivering gasoline and those delivering milk had the same frequency of chromosome aberrations. In another study Fredga et al (4) compared chromosome aberrations in the following four

groups of employees: (i) drivers of trucks run on diesel, (ii) drivers of trucks run on gasoline (iii) inspectors at the Swedish Motor Vehicle Inspection Company, and (iv) a control group of office employees. Chromosome aberrations were found to be associated with smoking, but not with exposure to motor fuels. A suspicion remained, however, that exposure to diesel fuel could cause chromosomal damage. In order to elucidate this question further we have studied a group of miners exposed to diesel exhausts.

Subjects and methods

A group of 14 male miners employed by the Boliden Company was selected for the study. Their ages varied between 20–57 a (mean 37.4 a), and they had worked underground as miners between 2 and 36 a (mean 11.6 a). Diesel engines have been used for loading since 1973.

The main exposure to diesel exhausts takes place during the loading procedure. Loading time is estimated to be about 30 % of the underground worktime. The yearly worktime has been estimated to be 1,700 h.

Measurements of carbon monoxide (CO) and nitrogen dioxide (NO₂) have been used

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as indicators of diesel exhausts. The maximum allowable concentrations for CO and NO₂ are 35 and 2 ppm, respectively. The concentrations at the workplace (loading) have been 5–10 ppm for CO and 0.5–1 ppm for NO₂. From available information the mean exposures to CO and NO₂ were estimated to be about 23,000 and 2,000 ppmh, respectively. The concentration of radon gas in the mines was below 0.4 Bq/l.

As referents, a group of 15 male office employees and construction workers was used. Their ages varied between 26 and 60 a with a mean of 45.9 a.

Five miners and five referents were smokers. Information on smoking habits is presented in tables 1 and 2.

A microculture technique, largely according to Hungerford (6), was used. The culture medium was RPMI 1640 supplemented with 20 % calf serum (Flow Labs), streptomycin, penicillin, and phytohemagglutinin. The incubation time was 72 h, and 90 min before harvest Velbe (Lilly) was added (0.05 mg/ml) for mitotic arrest. Air-dried slides were stained with 10 % Giemsa (Merck). From each individual 100 metaphases were analyzed. The chromosomal aberrations were recorded according to the recommendations of the World Health Organization (1).

Statistical analysis was performed by means of the chi-square test.

Results

Tables 1 and 2 show the smoking habits and chromosomal aberrations of the miners and referents, respectively. Table 3 presents the frequencies of gaps, chromatid aberrations, and chromosome aberrations with respect to exposure and smoking habits.

When the frequencies of aberrations of the groups of exposed individuals and referents, namely, in 1,400 cells from exposed individuals versus 1,500 cells from the referents, were compared, slightly higher values ($p < 0.05$) of (all) aberrations and of gaps were found among the referents. A similar comparison of smokers versus nonsmokers showed a highly significant increase of (all) aberrations among the smokers ($p < 0.001$). The frequencies of breaks ($p < 0.01$) and gaps

($p < 0.025$) tested separately were also significantly increased among the smokers.

On the individual level there were no significant differences between the diesel-exposed individuals and the referents. Thus 6 out of 14 diesel-exposed individuals had chromosome or chromatid breaks as compared to 7 out of 15 referents. Furthermore 9 of the 14 diesel-exposed workers showed gaps as compared to 13 of the 15 referents.

When the smokers and nonsmokers were compared, it was found that 8 out of 10 smokers had chromosome or chromatid breaks, while among the nonsmokers only 5 out of 19 had such breaks. This difference was statistically significant ($p < 0.01$), as was the difference with respect to chromosome breaks ($p < 0.01$) and gaps ($p < 0.05$) alone.

Discussion

In a previous study from Sweden Fredga et al (4) found no significant effect of exposure to different motor fuels on the frequency of chromosomal aberrations. There remained, however, a suspicion that diesel fuel could cause chromosomal damage, and in addition chromosomal aberrations were found to be associated with smoking.

The results found in the present study are largely a confirmation of the results by Fredga et al (4). We found no increased frequency of chromosomal aberrations among miners exposed to diesel exhausts, whereas there was a significantly increased frequency of aberrations among smokers.

We have described the exposure to diesel exhausts in terms of the CO and NO₂ concentrations simply because they were the measurements available to us. Other components in motor fuels, especially benzene, have been suspected to cause chromosomal damage. The main exposure takes place during loading, but there is also some degree of exposure during the rest of the worktime. This exposure is, however, difficult to estimate. Workers sometimes report respiratory and other problems from working in diesel exhausts. One of the workers in the group examined by us has been transferred to

Table 1. Chromosomal aberrations and smoking habits of 14 miners exposed to diesel exhausts; 100 cells from each individual were examined.

Smoking habits		Gaps	Chromatid aberrations	Chromosome aberrations
Cigarettes/day	Grams of tobacco/week			
0	0	0	0	0
0	0	1	0	0
0	0	0	1	0
0	0	0	1	0
0	0	2	0	0
0	0	0	0	0
0	0	1	0	0
0	0	0	0	0
0	0	1	0	0
15	50	3	0	1
10	50	2	0	0
10—15	0	4	1	0
3	30	1	1	1
6	40	1	0	1

Table 2. Chromosomal aberrations and smoking habits of the reference group (N = 15); 100 cells from each individual were examined.

Smoking habits		Gaps	Chromatid aberrations	Chromosome aberrations
Cigarettes/day	Grams of tobacco/week			
0	0	0	0	0
0	0	4	1	0
0	0	0	0	0
0	0	1	0	0
0	0	1	0	0
0	0	1	2	0
0	0	1	0	0
0	0	2	0	0
0	0	5	0	0
0	0	3	1	0
5	50	1	0	0
0	50	4	1	1
20	50	2	1	0
0	50	4	1	0
14	0	2	2	0

Table 3. Chromosomal aberrations (per 100 cells) among miners exposed to diesel exhausts and among the referents.

	Gaps	Chromatid aberrations	Chromosome aberrations	All aberrations	Number of cells
Diesel exposure					
Smokers	2.2	0.4	0.6	3.2	500
Nonsmokers	0.6	0.2	0.0	0.8	900
All	1.1	0.3	0.2	1.6	1,400
Referents					
Smokers	2.6	1.0	0.2	3.8	500
Nonsmokers	1.8	0.4	0.0	2.2	1,000
All	2.1	0.6	0.1	2.7	1,500
All smokers	2.4	0.6	0.4	3.4	1,000
All nonsmokers	1.2	0.2	0.0	1.4	1,900

another workplace due to respiratory problems.

The negative finding of this study and those of Fredga et al (4) suggest that the levels of exposure to motor fuels in most workplaces are below the level at which chromosomal damage may be found.

The smokers in our series were neither heavy nor light smokers, but had an intermediate type of tobacco consumption. Attempts to divide the 10 smokers into 5 with higher and 5 with lower consumption resulted in no difference with respect to the frequency of chromosomal aberrations. In the study by Fredga et al (4) smokers were divided into groups according to tobacco consumption. Once again no relationship between tobacco consumption and chromosomal aberrations was found. In fact in their data there was a significantly higher ($p < 0.01$) frequency of breaks in the group with the lowest consumption in comparison to the group with the highest consumption (test performed on pooled data). Thus, while in some studies evidence for a relationship between smoking and chromosomal damage has been found, the dose-response relationship is rather poor. One possible reason may be that smoking, which is a powerful confounding factor in occupational studies, is itself confounded by a number of factors.

We have not used rank tests in the statistical analysis. One reason is that, in a study like the present, where 100 cells were counted, individual observations tend to fall into a few classes, for breaks mostly 0 or 1 in 100 cells. Thus the number of tied ranks becomes high and the parameter studied tends to be more qualitative than quantitative in nature. In this situa-

tion we feel that comparisons of distributions are appropriate, eg, 8 out of 10 smokers had breaks compared to 5 out of 19 nonsmokers.

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