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Chromosome Aberrations in Workers Exposed to Arsenic

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The occurrence of chromosome aberrations was studied in short-term cultured lymphocytes from nine workers exposed to arsenic at the Rönnskär smeltery in northern Sweden. In the smelter workers, 87 aberrations were found in 819 mitoses. The number of aberrations varied individually from 0 to 25 aberrations per 100 cells. In a control material 13 aberrations were found in 1012 mitoses. The frequency of chromosome aberrations was significantly increased among the smelter workers, but due to the simultaneous exposure to other agents the effect of arsenic per se can not be assessed with certainty.

Carcinogenic effects of arsenic have been found in clinical and epidemiological investigations (*1*, *2*), but not in animal experiments (*3*). An increased frequency of chromosome aberrations has been found (*4*) in short-term cultured lymphocytes of wine growers and psoriatric patients treated with arsenic. Experimental studies (*5*) in *Escherichia coli* suggest that arsenic compounds may act as cocarcinogens by inhibiting DNA repair. In this paper we report some preliminary results from a study of chromosome aberrations in arsenic exposed workers at the Rönnkär smeltery in northern Sweden.

Materials and Methods

Blood samples were collected from a series of workers exposed to arsenic at the Rönnskär smeltery and sent to the Clinical Genetics laboratory in Umeå for examination. This study is a part of a larger project involving exposition to other potentially hazardous agents at Rönnskär, e.g., lead. We are presenting here data for a relatively small number of individuals. The ongoing project will not be completed until next year.

Data on urinary arsenic levels and type and duration of exposition were available on most of the examined workers. As controls apparently healthy individuals from Umeå were used. Other control material from newly employed workers at Rönnskär is now under collection.

Human lymphocytes were grown *in vitro* for a period of 68–70 hr according to a procedure described previously (*6*).

Results and Discussion

The results are shown in Table 1. The aberrations have been grouped into three categories: gaps, chromatid aberrations, and chromosome aberrations. Gaps are commonly defined as achromatic regions exceeding the width of the chromosome. Larger achromatic regions where parts of chromatids are clearly dislocated are summarized under chromatid aberrations. Chromosome aberrations include dicentric chromosomes, rings, acentric fragments, and "minutes."

Table 1. Chromosome aberrations in workers exposed to arsenic from Rönnskär, Sweden and controls.

	Arsenic workers	Controls
No. of cells	819	1012
No. of aberrant cells	71	13
No. of aberrations		
Gaps	56	9
Chromatid aberrations	12	3
Chromosome aberrations	19	1
Total	87	13
No. of aberrations per cell	0.1062	0.0128
Frequency of aberrant cells, %	8.7	1.3

The frequency of aberrations was significantly higher ($p < 0.001$) among the arsenic-exposed workers. All three types of aberrations, gaps ($p < 0.001$), chromatid aberrations ($p < 0.01$), and chromosome

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aberrations ($p < 0.001$) were significantly increased. The individual variations were large: from 0 to 25 aberrations per 100 cells.

The exact meaning of this result is difficult to evaluate at this point of the investigation. There seems to be little doubt that there is a relationship between arsenic exposition and chromosome breakage, which also is in agreement with previous findings (*4*). There was, however, no obvious relationship between exposure and frequency of aberrations in our material. Furthermore the individual with the highest frequency of aberrations had been exposed to arsenic, lead, and selenium. (Rönnskär is characterized by a multitude of potentially hazardous agents.) Therefore we feel that conclusions concerning the mutagenic-carcinogenic effect of arsenic *per se* should await further investigations. Thus a possible explanation (*5*) is that arsenic, by inhibiting DNA repair, may be deleterious mainly in combination with other agents. In cases of long-range suppression of DNA repair it is, of course, possible that the background mutagenicity may cause considerable cumulative chromosomal damage to cells with such a long life span as the lymphocytes.

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