JOURNAL OF PREVENTIVE MEDICINE
2008; 16(1-2): 85-91

PECULIAR ISSUES CONCERNING THE OCCUPATIONAL RISK IN AN ASBESTOS - CEMENT FACTORY

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Abstract. Aim. The risks of carcinogenic and co-carcinogenic effects from asbestos continue owing to the persistence of the fibres from mining, milling, manufacturing and its use in building materials and other products. This paper wass aimed to achieve the risk assessment in occupational exposures to asbestos in an industrial unit producing asbestos-cement for a period of 30 years and also an evaluation of the possible correlation between exposure and cytogenetic alterations in peripheral blood, which might be a quantitative comparable and reliable method of risk assessment. **Material and methods**. The retrospective evaluation of the magnitude of occupational risk has been performed on the basis of the laboratory data established through the "membrane filter" method. The enhancement of cytogenetic alterations by using the peripheral lymphocyte cultures - Evans and O'Riordan, method - on a representative group of 30 subjects, with the mean age of 42.2±8.9 years and the work length in asbestos industry of 17.5±8.7 years was performed. The results were compared with those of a matched control group through the "t" Student test and the Spearman correlation factor for $p<0.05$. The obvious correlation between the incidence of cytogenetic alterations, the cumulated exposure and the work length, respectively have been calculated. **Results.** The exposure risk, quantified in fibres/ml (l>5mm, d>3mm, l/d ≥3/1), evaluated after 1981 presented the following values: in 1981-1985 32 f/ml and in 1998 0.003 f/ml. The employees who have taken contact with the asbestos powders presented significantly higher frequencies for the structural chromosomal aberrations, compared to the control group have registered values of 3.60 ± 1.39 (IC95% 3.05-4.17) and 1.85 ± 1.05 (IC95% 1.42-2.27). **Conclusion**. Asbestos is carcinogenic for humans. The latency period for tumor occurrence is about 20 years. Our results with regard high correlation of the cytogenetic aberrations and the cancer incidence in populations occupationally or environmentally exposed to potentially genotoxic factors are in concordance with the recent literature in the field.
Key words: asbestos, occupational exposure, chromosomal aberrations, cancer

Rezumat. Scop. Riscul de producere a efectelor carcinogene sau co-carcinogene ale azbestului reprezintă încă o problemă de actualitate, datorită persistenţei fibrelor rezultate din procesele de extracţie, măcinare sau procesare şi din includerea sa în materiale de construcţie sau în alte produse. Lucrarea de faţă a urmărit să realizeze atât o evaluare a riscului în expunerile ocupaţionale la azbest în cadrul unei unităţi industriale producătoare de azbociment pentru o perioadă de 30 ani, cât şi o evaluare a posibilei corelaţii între expunere şi modificările din sângele periferic, ceea ce ar putea constitui o metodă cantitativă de evaluare a riscului comparabilă şi riguroasă. **Material şi metodă**. Evaluarea retrospectivă a riscului ocupaţional a fost efectuată pe baza datelor de laborator stabilite prin metoda „filtrului membrană". La un

grup de studiu de 30 subiecţi cu vârsta medie de 42,2±8,9 ani şi având o durată medie a activităţii de 17,5±8,7 ani în industria azbestozei, s-au evaluat alterările citogenetice folosind metoda culturilor de limfocite periferice (Evans şi O'Riordan). Rezultatele au fost comparate cu cele obţinute în urma analizei lotului martor. S-au estimat indicatorii de corelaţie între incidenţa alterărilor citogenetice, expunerea cumulativă şi durata activităţii. **Rezultate**. Riscul de expunere, cuantificat în fibre/ml (l>5mm, d>3mm, l/d ≥3/1) evaluat după 1981, a înregistrat valori de 32 f/ml în 1981-1985 şi 0,003 f/ml în 1988. Subiecţii care au fost supuşi unui contact cu pulberi de azbest au prezentat frecvenţe mai crescute în comparaţie cu lotul martor, în ce priveşte aberaţiile cromozomiale structurale: 3,60 ± 1,39 (IC95% 3,05-4,17), respectiv 1,85 ± 1,05 (IC95% 1,42-2,27). **Concluzii**. Azbestul reprezintă risc carcinogen pentru om. Perioada de latenţă în apariţia tumorii este de aproximativ 20 ani. Rezultatele noastre sunt în concordanţă cu cele din literatura recentă de specialitate, privind corelaţia crescută a aberaţiilor citogenetice şi incidenţa cancerului la populaţiile umane care sunt expuse la factori potenţiali genotoxici care provin din mediu sau de la locul de muncă.
Cuvinte cheie: azbest, expunere ocupaţională, aberaţii cromozomiale, cancer

INTRODUCTION

Asbestos, a fibrous mineral representing a family of hydrated silicates, with a great variety of physical-chemical peculiarities, has been known and used since ancient times due to its multiple qualities concerning material indestructibility and significant fire-resistance.

At present, as it is recognized by all international bodies as a certain carcinogenic substance, asbestos still raises a question mark concerning the pathogenic mechanisms through which it can induce cancer with multiple sites, especially malignant mesothelioma (pleural, peritoneal) and pulmonary cancer.

As carcinogenesis is a multistage process and because asbestos fibbers tend to gradually accumulate in the lungs – the disease has a long latency period, probably the fibbers are involved in different stages of the malignant process: initiation, promotion, progression until the development of the tumor (1, 2).

"A problem which is still unsolved is whether asbestos acts directly on the cell – mechanic action, or if its action is the result of an indirect process, his mechanism implying free reactive radicals formation and/or formation of cell growth factors" (3).

The same team of the Research Institute on Asbestos from Vermont has proposed in 1998 a hypothetical multistage mechanism of the carcinogenicity of asbestos that implies all he above mentioned factors (fig. 1).

This paper was aimed to achieve the risk assessment in occupational exposures to asbestos in an industrial unit producing asbestos-cement for a period of 30 years and also an evaluation of the possible correlation between exposure and cytogenetic alterations in peripheral blood, which might be a comparable, quantitative and reliable method of risk assessment.

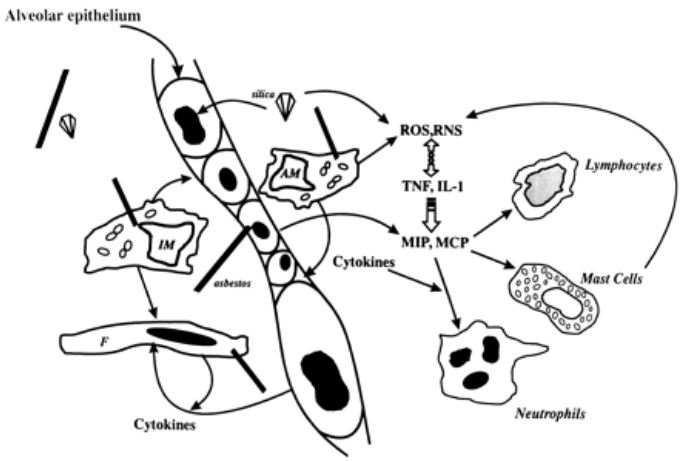

Fig. 1. A hypothetical schema of events occurring in lung after exposure to pathogenic mineral dusts (3)

AM = alveolar macrophage;
IM = interstitial macrophage;
F = fibroblast;
ROS = reactive oxygen species;
RNS = reactive nitrogen species;
TNF = tumour necrosis factor;
IL-1 = interleukin-1;
MIP = macrophage inflammatory proteins;
MCP = macrophage chemotactic proteins.

MATERIAL AND METHOD

The retrospective evaluation of the magnitude of occupational risk has been performed on the basis of the laboratory data established through the "membrane filter" method (4, 5).

The enhancement of cytogenetic alterations by using the peripheral lymphocyte cultures – Moorhead method - subsequently changed to Evans and O'Riordan, 1975 (6) - on a representative group of 30 subjects, with the mean age of 42.2±8.9 years and the work length in asbestos industry of 17.5±8.7 years; the results were compared with those of a matched control group through the "t" Student test and the Spearman correlation factor for $p<0.05$.

The enhancement of the obvious correlation between the incidence of

cytogenetic alterations, the cumulated exposure and the work length respectively had been done.

RESULTS AND DISCUSSION

A. The exposure risk, quantified in fibres/ml (l>5mm, d>3mm, l/d ≥3/1), evaluated after 1981, presented the following values:

- 1981 – 1985: 32f/ml
- 1986 – 1989 16 f/ml
- 1990 – 1994: 12 f/ml
- 1995 – 1997: 4 f/ml
- 1998: 0,003 f/ml.

For the period prior to 1981 (1970 – 1981) we took into consideration the valued corresponding to 1981

CMA (TLV) in Romania for the time of survey = 1 f/ml; TLV in USA, France, Germany = 0.1 f/ml.

The decrease of the occupational risk, especially after 1986, was conditioned by the gradual but significant diminishing of production, the closing up of Section II, and subsequently the closing up of the line of asbestos-cement tubes in Section I.

Cumulative exposure = years x f/ml evaluated during this period: 1970 – 1998 between 778 years x f/ml and 6 years x f/ml.

B1. The employees who have taken contact with the asbestos powders presented significantly higher frequencies for the structural chromosomal aberrations (see Photo 1 – 4) compared to the control group as follows:

EXPOSED GROUP:

I. Structural aberrations/subject (without chromatid gaps): 3.6±1.39
Median: 4
IC (95%): 3.05 – 4.17
II. Total aberrations/subject: 6.08±2.51
Median: 6
IC (95%): 5.06 – 7.09

CONTROL GROUP

I. Structural aberrations/subject (without chromatid gaps): 1.85±1.05
Median: 2
IC (95%): 1.42 – 2.27
II. Total aberrations/subject: 3.85±2.46
Median: 4
IC (95%): 2.85 – 4.84

THE SIGNIFICANCE OF THE DIFFERENCE:

I – I control: $t = 5.19$ $p<0.000004$
II – II control: $t = 3.23$ $p<0.002$
RR = 2.05
AR = 1.95

B2. The numerical chromosomal variations consisted in a slight polyploidy.

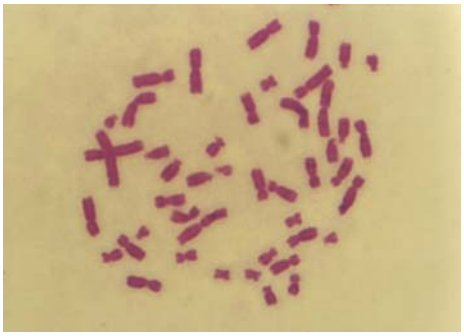

Photo 1. Normal metaphase

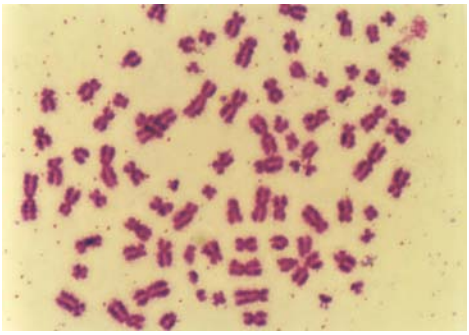

Photo 2. Numerical chromosomal variation: Polyploidy

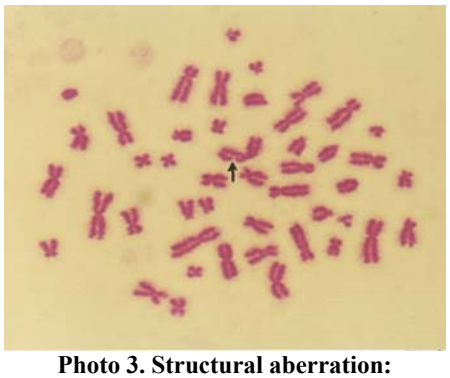

Photo 3. Structural aberration: Chromatid gap

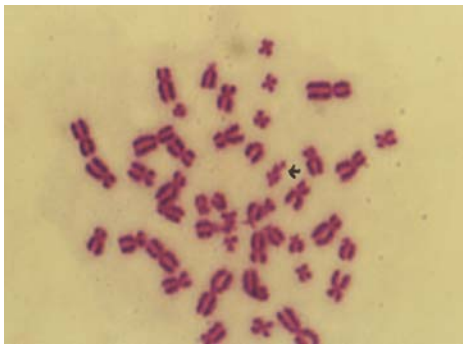

Photo 4. Structural aberration: Chromatid break

C1. The ANOVA quotient F=5.42 p=0.03 certifies the direct connection between the incidence of the chromosomal aberrations for work lengths over 25 years and the lack of this connection for work lengths that are below the mean value of the group.

C2. The correlation between the incidence of the structural aberrations and the cumulative exposure assessed through the Pearson correlation quotient is not statistically significant; the direct association is evident for big work lengths also.

According to our data, the increase of the cumulative exposure is especially due to the increase of the work length, and the probable risk is induced not only quantitatively but especially by the fibres accumulation and persistence.

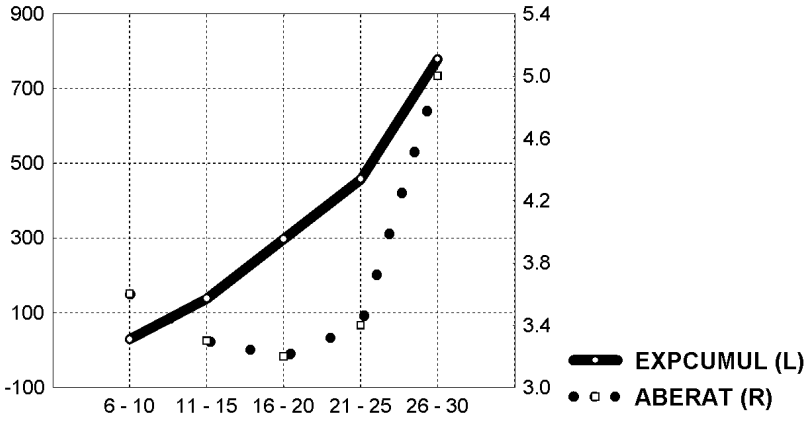

Fig. 2. The correlation between the cumulative exposure to asbestos and chromosomal aberrations

Fig. 3. The regression curve of the chromosomal aberrations related to the work length

CONCLUSIONS

If taking into account the indisputable fact that asbestos is carcinogenic for humans and that the latency period for tumour occurrence is about 20 years, the observance of relatively higher aberrations frequencies in groups with exposure length over 25 years is in accordance with certain relatively recent studies which support the existence of a serried correlation between the high frequency of the

cytogenetic aberrations and the cancer incidence in populations occupationally or environmentally exposed to potentially genotoxic factors (7, 8). The drastic decrease of the occupational risk over the last 10 years does not diminish the retroactive injuriousness and aggressiveness which lives its imprint on the caryokinetic process.

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