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Eight-year follow-up of viscose rayon workers exposed to carbon disulfide

by SVEN HERNBERG, M.D., MATTI TOLONEN, M.D., and MARKKU NURMINEN, Lic. Sc.¹

HERNBERG, S., TOLONEN, M. and NURMINEN, M. Eight-year follow-up of viscose rayon workers exposed to carbon disulfide. *Scand. j. work environ. & health* 2 (1976) 27—30. An 8-year follow-up of workers exposed for at least 5 years to carbon disulfide showed an excess of deaths due to coronary heart disease in reference to a comparison cohort. However, after effective preventive measures, e.g., leaving only 19 % of the original group exposed and reducing the level of exposure to less than 10 ppm, had been undertaken after the 5th year of follow-up, coronary mortality decreased and no excess mortality during the last 3 years of follow-up occurred. These results, although only suggestive — due to the small number of deaths and the short time of follow-up after the intervention — seem promising from the point of view of improving the prognosis of workers who have already accumulated an excess risk for coronary death.

Key words: Carbon disulfide, coronary heart disease, follow-up study, intervention.

A follow-up study of the effects of carbon disulfide (CS₂) exposure upon the incidence of coronary heart disease (CHD) was started in 1967. A cohort of viscose rayon workers, exposed for at least 5 years to CS₂ during any period between 1942 and 1967, and a comparison cohort from a nearby paper mill formed the groups under study. The members were matched with respect to age, birth district, and type of work and examined for the prevalence of symptoms and signs indicative of CHD (7). The results of the first 5 years indicated nearly five times as many deaths due to CHD in the exposed cohort as in the comparison cohort; also the incidence of nonfatal infarctions was elevated (3, 7). When these results became known, vigorous efforts were made to decrease the risk. They consisted of technical improvements in the work conditions, transfer of

workers with long exposure time and/or symptoms of CHD to departments without exposure, and extensive use of personal protective equipment during peak exposures. This intervention changed the premises for further follow-up, which was then continued with the purpose of possibly detecting a downward trend in CHD mortality.

MATERIAL AND METHODS

Detailed descriptions of the exposed and comparison cohorts have been given in previous publications, and will not be repeated (2, 3, 7). In short, both cohorts initially comprised 343 men, aged 25—64 years. By design the cohorts were similar with regard to age distribution, birth district and type of work. An a posteriori evaluation confirmed that they did not differ with respect to smoking habits, leisure-time physical activity, physical fitness, relative body weight, serum lipids, and glucose tolerance. The only significant difference was found for blood pressure,

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which was slightly higher in the exposed cohort, but this difference was interpreted as an effect of exposure rather than as an independent risk factor.

The causes of all deaths occurring in both cohorts during the follow-up period from June 1, 1967, through May 31, 1975, were verified from the death certificates

and classified according to the eighth revision of the international classification of diseases (8). No one was lost during the follow-up, which thus was 100 % successful.

As a basic measure of the occurrence of death we have used the 8-year cumulative incidence rate. The relative frequency of death has been expressed both as a ratio and as a difference in the rate of the exposed relative to that of the nonexposed.

According to the 5-year data the exposure status of the viscose rayon workers changed radically during the last 3 years. Table 1 shows that only 19 % of the men were still exposed to CS₂ as compared to 53 % 3 years earlier (7). During the same period the CS₂ levels fell sharply, and hence even the 19 % still exposed had much safer working conditions than before (figs. 1 and 2). The CS₂ factory had ceased to exist, and viscose film was no longer being produced.

At the end of the 8-year follow-up, the mean age of both cohorts was 53 years.

Table 1. Status of the exposed cohort in June 1975.

Status	Number	%
Still employed at the plant	165	48
Still exposed	64	19
No longer exposed	101	29
No longer employed at the plant	143	42
Retired or pensioned	66	19
Moved	77	22
Dead	35	10
Total	343	100

Fig. 1. Concentrations of CS₂ + H₂S in the rayon staple fiber factory. Note the decrease between 1972 and 1975.

Fig. 2. Concentrations of $CS_2 + H_2S$ in the rayon filament factory.

Table 2. Observed number of deaths among the exposed and comparison cohorts during the 8 years of follow-up, and estimates of crude rate ratios, by cause of death.

Mortality	Cohort		Rate ratio	95 % confidence interval
	Exposed	Comparison		
Total	35	23	1.5	0.9—2.6
CHD	20	9	2.2	1.0—4.8
During the last 3 years	6	6	1.0	..

RESULTS

Table 2 shows the accumulated total and coronary mortality of both cohorts. The 8-year cumulative incidence rate for CHD mortality was 5.8 % in the exposed group and 2.6 % in the comparison group, the rate difference being 3.2 %. For total mortality the corresponding cumulative incidence rates were 10.2 % and 6.7 %, respectively. The rate difference of 3.5 % was almost identical with that for CHD

alone. This result indicates that CHD was totally responsible for the excess mortality. However, during the last 3 years of follow-up, the same number of deaths, or 6, from CHD occurred in both cohorts, and during the 8th year there was only one CHD death in the exposed cohort against three in the comparison cohort. Thus a shift in the trend seems to have taken place.

Age-specific data indicate that the excess risk of CHD was strongest in the age range of 50 to 64 years. In all, 17 of the

20 coronary deaths of the exposed workers occurred in that age range against 5 in the comparison group. A more detailed analysis, together with a calculation of the life expectancy in different age categories, has been reported elsewhere (5).

DISCUSSION

When interpreting the data for the 6th through the 8th year of follow-up, which actually did not show any excess of CHD in the exposed group, one must consider the drastic change in exposure status. Only 19 % of the original cohort was still exposed, against 53 % in 1972. The rest of the survivors had moved away or had been removed from exposure. Furthermore, the average exposure level was well below 10 ppm for those still exposed. Thus there is no contradiction between the present results and those previously reported by us (3, 7) and others (4, 6), according to which exposure to CS₂ increases the incidence of coronary deaths. In fact, Tiller et al. (6) have already pointed out that the excess mortality found in the 1940s tended to level off with improving hygiene in the 1950s and 1960s. In the present study the small number of coronary deaths occurring during the last 3 years of follow-up (6 in both groups) prevents the drawing of definite conclusions, but the fact that the even distribution coincides with the changed conditions suggests that the excess risk is reversible. If true, such a finding would be of both practical and theoretical interest. Besides proving the preventive effect of lowering the exposure level, the data suggest that removing persons with indications of CHD from exposure really decreases the risk for future fatal attacks. The theoretical interest lies in the fact that, if the effect of CS₂ is indeed reversible, the excess mortality occurring under exposure is due to a direct toxic effect upon the myocardium rather than to an

acceleration of the atherosclerotic process (1). Further follow-up of the cohorts is now primarily motivated by the need to test this hypothesis.

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