J Occup Health 2017; 59: 187-193

Journal of Occupational Health

Original

Changes of median nerve conduction velocity in rayon manufacturing workers: A 6-year cohort study

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Abstract: Objectives: We conducted a 6-year cohort study to evaluate the relationship between carbon disulfide (CS_2) exposure and reductions in the motor and sensory nerve conduction velocity (MCV and SCV) of the median nerve. **Methods:** Study subjects at baseline included 432 exposed workers and 402 unexposed workers. Among the exposed workers, 145 workers terminated CS_2 exposure during the follow-up period (ex-exposed workers). MCV and SCV were measured at baseline and followed up. CS_2 personal exposure concentration was measured two times a year during a 6-year follow-up period and mean (range) CS_2 exposure concentrations (ppm) were 5.96 (0.8-16.0) and 3.93 (0.6-9.9) in the exposed and ex-exposed workers, respectively. **Results:** Reductions in MCV during the follow-up period did not differ among the exposed, ex-exposed, and unexposed workers. Reduction in SCV (m/s) of the exposed workers (-4.47±3.94) was significantly larger than that of the unexposed (-3.38±3.97) and ex-exposed workers (-3.26±3.79). For SCV reduction, a partial multiple regression coefficient of (ex-exposed workers)/(unexposed workers) was significantly positive (+0.915, $p < 0.01$) after adjustment for confounding variables. **Conclusions:** This cohort study showed that 6-year CS_2 exposure around a mean level of 6 ppm did not affect MCV reduction but induced significant SCV reduction beyond the influence of aging. The effect of CS_2 on SCV around a mean exposure level of 4 ppm may be reversible, since it disappeared in the ex-exposed workers after CS_2 exposure cessation for a mean period of 4.1 years.
(J Occup Health 2017; 59: 187-193)
doi: 10.1539/joh.16-0255-OA

Key words: Carbon disulfide, Nerve conduction velocity, Rayon

Introduction

Considerable studies have reported the adverse effects of occupational exposure to carbon disulfide (CS_2; CAS No 75-15-0) on multiple organs.

We conducted a 6-year cohort study to comprehensively evaluate CS_2 exposure concentration and health effects, including cerebrovascular, cardiovascular, ophthalmological, neurological, neurobehavioral, and endocrinological aspects, at baseline (1992-93) and follow-up (1998-99)[1-6]. The neurological system is considered to be the critical target of CS_2-induced toxicity. Toxicity is most often manifested as slower nerve conduction velocity (NCV) in the peripheral nerves[1,7] and impaired performance in psychomotor testing[8]. Peripheral nervous system effects have been reported in many cross-sectional studies on workers exposed to CS_2 in the viscose rayon industry[1,7,9-13,14]. The most common observations are characterized by slower motor (MCV) and sensory nerve conduction velocity (SCV)[1,7,9,15]. These effects have been observed in workers exposed to CS_2 at 1.0 ppm or above. In contrast, other studies on viscose rayon workers exposed to equal concentrations of CS_2 have shown little indication of an effect on NCV[11,14]. Furthermore, NCV in the

Received November 1, 2016; Accepted December 15, 2016
Published online in J-STAGE January 21, 2017
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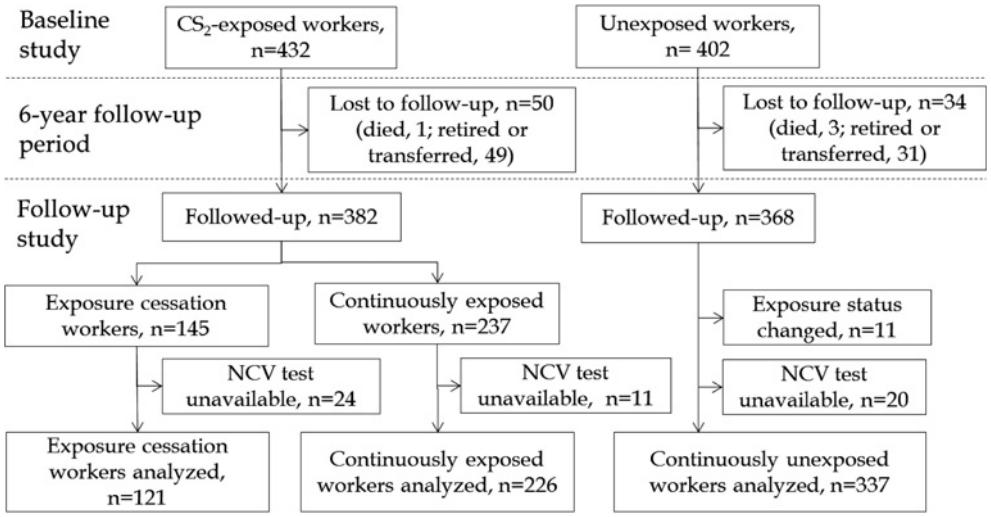

Fig. 1. Study profile and results of follow up.

present CS_2 exposed group was lower than that removed from the CS_2 exposure group on a cross-sectional observation[9]. However, all these studies were conducted under a cross-sectional design and did not evaluate whether the removal of CS_2 exposure leads to a reduction in the effects on NCV in a cohort study.

We hereby present the changes of CS_2 on NCV of the dominant median nerve observed in a 6-year cohort study.

Subjects and Methods

Study population

The cohort study design and follow-up details have been described previously[1,2,4-6]. In brief, study subjects at baseline consisted of 432 male workers exposed to CS_2 and 402 unexposed male workers, with no medical history of cerebrovascular or cardiovascular diseases as determined using company medical records and a self-administered questionnaire, in 11 Japanese viscose rayon factories. During the 6-year follow-up period, 4 factories ceased rayon production for economic reasons. At follow-up, 50 exposed (49 retired or transferred and 1 dead case) and 34 unexposed (31 retired or transferred and 3 dead cases) workers were lost to follow-up, leaving 382 exposed and 368 unexposed workers for follow-up (follow-up rates of 88.4% and 91.5%, respectively). We checked the health and personnel records of retired or transferred workers and confirmed that the cause of loss to follow-up was not related to health. Between the followed up workers and those lost to follow-up, there were no differences in either mean CS_2 exposure concentration or mean urinary 2-thiothiazolidine-4-carboxylic acid (TTCA) level in the exposed workers, as well as in the MCV and SCV of exposed and unexposed workers at baseline.

Among the 382 exposed workers, 145 workers (including workers in 4 factories that ceased rayon production) terminated CS_2 exposure (ex-exposed workers). The mean and median CS_2 cessation periods were 4.1 and 4.5 years (range 0.8-6.9 years), respectively.

MCV and SCV data of 226 exposed, 337 unexposed, and 121 ex-exposed workers were available for statistical analysis. Fig. 1 shows the study profile and Table 1 shows the basic characteristics of study subjects at baseline.

This study received approval by the ethical committee of Keio University School of Medicine (approval number 20160204) to use our old data. An opt-out consent process was conducted on the website (http://keiopublichealth.jp).

Exposure assessment

Assessment of CS_2 exposure and urinary TTCA, a metabolite of CS_2, has been described elsewhere[2]. In brief, an 8-hour time-weighted average CS_2 concentration in the workers' breathing zone was measured two times a year, beginning in the spring of 1993 and continuing throughout the study period, using a Parkin-Elmer diffusive sampler tube. As a biological exposure monitoring parameter, TTCA in urine after the workers' shift on the same day of measurement of CS_2 was also determined two times a year, beginning in the autumn of 1992, using high-performance liquid chromatography modified from the method of Ogata and Taguchi[16]. To assess an exposure-effect relationship, the exposed workers were categorized into 3 groups by tertile of individual mean CS_2 exposure concentration during the follow-up period.

Measurement of nerve conduction velocity

We measured NCV of the median nerve of the dominant hand/arm using an evoked potential system (Neuropack four mini MEB-5304; Nihon Kohden, Tokyo, Japan). MCV was assessed at the elbow-wrist segment and

Table 1. Characteristics of study subjects at the baseline study

	CS_2 exposed workers			Unexposed workers		
	n	mean ± SD	Range	n	mean ± SD	Range
Age (years)	347	36.1 ± 7.8	19.7-47.8	337	36.2 ± 9.0	18.9-49.8
Height (cm)	343	168.6 ± 6.1	148.7-189.0	323	168.5 ± 6.2	152.6-185.9
Weight (kg)	343	63.5 ± 8.1	47.0-99.6	323	64.2 ± 8.6	43.0-97.5
BMI (kg/m^2)	343	22.3 ± 2.5	16.3-33.0	323	22.6 ± 2.8	16.3-34.4
Duration of work (years)	347	22.1 ± 8.2	7.3-36.8	337	22.6 ± 9.0	6.8-37.1
Duration of CS_2 exposure (years)	345	14.0 ± 7.9	1.2-29.0		—	
Smoking status (n, %)						
Never smoked		76 (22.0)			85 (25.2)	
Former smoker		29 (8.4)			41 (12.2)	
Current smoker		241 (69.7)			211 (62.6)	
Alcohol drinking (n, %)						
Non drinker		51 (15.0)			39 (11.8)	
Occasional drinker		94 (27.6)			87 (26.3)	
Habitual drinker		196 (57.5)			205 (61.9)	
Educational status (n, %)						
Junior high or below		129 (37.8)			103 (31.7)	
Senior high or above		212 (62.2)			222 (68.3)	

antegrade SCV conduction was assessed at the finger-wrist segment (digit 2). A surface electrode was used and supra-maximal stimulation was given. Since NCV is affected by skin temperature, surface skin temperature was maintained above 30℃ using a mantle heater or hot water bath.

Statistical analysis

NCVs at baseline and follow-up were compared among continuously exposed, ex-exposed, and unexposed workers using analysis of variance with the Tukey-Kramer method. Reductions of NCVs between baseline and follow-up were considered as aging- and exposure-related NCV changes. To assess the effects of CS_2 on NCV after adjusting possible confounders, a multiple linear regression model was applied. Potential confounding factors included in the models were age (years), body mass index (BMI; kg/m^2), education status (high school or above vs. junior high school or below), smoking status (former or current smoker vs. never smoked), and alcohol consumption (occasional or habitual drinker vs. non-drinker). JMP 12.2.0 (SAS Institute Inc., Cary, NC, USA) was used for all analyses.

Results

Table 2 shows the mean and maximal concentrations of CS_2 and TTCA during the 6-year follow-up period. The mean concentration (range) of CS_2 was 5.96 (0.8-16.0) in the exposed workers and 3.93 (0.6-9.9) ppm in the ex-exposed workers. The mean concentration (range) of CS_2 in the 1st-, 2nd-, and 3rd-tertile exposed workers was 2.84 (0.80-4.59), 5.64 (4.65-6.61), and 9.35 (6.64-16.0) ppm, respectively. The maximal concentration of CS_2 exposure was about two times the mean concentration. This suggests that inter-daily fluctuation of CS_2 exposure may not be large. TTCA was proportional to CS_2.

Table 3 shows NCV at baseline and follow-up as well as mean NCV reductions during the 6-year follow-up period.

MCV of the exposed, ex-exposed, 2nd-tertile exposed, and 3rd-tertile exposed workers were significantly slower than the unexposed workers both at baseline and follow-up, but mean reductions in MCV were not different among the exposed (-1.60±3.70 m/s), ex-exposed (-1.61±3.37 m/s), and unexposed (-1.52±3.49 m/s) workers.

At baseline, SCVs were not different among the exposed, ex-exposed, and unexposed workers, but SCV of the exposed workers became significantly slower than the unexposed workers at follow-up (48.82±5.49 m/s vs. 50.43±4.97 m/s). Mean reductions in SCV of the exposed workers (-4.47±3.94 m/s) and 3rd-tertile exposed workers (-4.89±4.39 m/s) were significantly larger than that in the unexposed (-3.38±3.97 m/s) and ex-exposed workers (-3.26±3.79 m/s).

Table 4 shows the results of multiple regression analyses to assess CS_2 exposure during the 6-year follow-up period on NCV after adjustment for possible confounders. For the outcome variables of SCV at follow-up and SCV reduction, partial regression coefficients of the 3rd-tertile exposed workers were significantly negative. On the other hand, for SCV reduction, a partial regression coeffi-

Table 2. CS_2 exposure profile of continuously exposed workers and exposure cessation workers.

		CS_2 (ppm)		TTCA in urine (mg/g • Cr)	
	n	mean ± SD	Range	mean ± SD	Range
Average concentration during the follow-up period					
Ex-exposed workers [$]	119	3.93 ± 2.66	0.6-9.9	1.38 ± 1.01	0.25-5.2
Exposed workers	226	5.96 ± 3.01	0.8-16.0	1.74 ± 1.12	0.25-8.2
1st Tertile exposed	75	2.84 ± 1.10	0.8-4.6	0.89 ± 0.40	0.25-2.28
2nd Tertile exposed	75	5.64 ± 0.51	4.7-6.6	1.60 - 0.54	0.33-3.01
3rd Tertile exposed	76	9.35 ± 2.04	6.6-16.0	2.71 ± 1.27	0.89-8.22
Maximal concentration during the follow-up period					
Ex-exposed workers [$]	119	5.24 ± 3.59	0.8-19	2.17 ± 1.53	0.25-6.8
Exposed workers	226	11.42 ± 6.53	1-32	4.72 ± 3.46	0.25-25.6
1st Tertile exposed	75	6.38 ± 2.95	1-20	2.74 ± 1.55	0.25-8.4
2nd Tertile exposed	75	10.00 ± 3.10	6-21	3.99 ± 1.69	0.6-10.0
3rd Tertile exposed	76	17.80 ± 6.41	8-32	7.39 ± 4.37	2.2-25.6

CS_2 and TTCA were measured twice a year during the follow-up period. Exposure cessation workers: Workers who left CS_2 jobs during the follow-up period. 1st to 3rd Tertile exposed: Workers categorized in tertiles by average CS_2 exposure concentration during the follow-up period. [$]: Exposure data of 2 exposure cessation workers were unavailable.

Table 3. Nerve conduction velocity (NCV) of the median nerve and its reduction during the 6-year follow-up period.

		Baseline study	Follow-up study	Reduction in NCV during the 6-year follow-up
	n	mean ± SD	mean ± SD	mean ± SD
Motor nerve conduction velocity (MCV) of the median nerve (m/s)				
Unexposed workers	337	58.97 ± 3.30	57.45 ± 3.31	–1.52 ± 3.49
Ex-exposed workers	121	57.59 ± 3.60 ***	55.98 ± 3.60 ***	–1.61 ± 3.37
Exposed workers	226	57.66 ± 3.65 ***	56.06 ± 3.63 ***	–1.60 ± 3.70
1st Tertile exposed	75	58.14 ± 3.67	56.53 ± 3.39	–1.62 ± 3.56
2nd Tertile exposed	75	57.18 ± 4.08 ***	55.83 ± 3.87 ***	–1.36 ± 3.92
3rd Tertile exposed	76	57.64 ± 3.11 *	55.83 ± 3.61 ***	–1.81 ± 3.64
Sensory nerve conduction velocity (SCV) of the median nerve (m/s)				
Unexposed workers	337	53.81 ± 4.32	50.43 ± 4.97	–3.38 ± 3.97
Ex-exposed workers	121	52.82 ± 5.07	49.56 ± 5.29	–3.26 ± 3.79 [#1, #2]
Exposed workers	226	53.29 ± 4.70	48.82 ± 5.49 ***	–4.47 ± 3.94 ***
1st Tertile exposed	75	53.52 ± 4.73	49.29 ± 4.88	–4.23 ± 3.76
2nd Tertile exposed	75	52.79 ± 4.77	48.52 ± 5.70 *	–4.27 ± 3.65
3rd Tertile exposed	76	53.54 ± 4.33	48.64 ± 5.88 *	–4.89 ± 4.39 *

*, **, ***: $p<0.05$, 0.01, 0.001 compared to the unexposed workers. [#1, #2]: $p<0.05$ compared to the exposed workers, and $p<0.05$ compared to the 3rd tertile exposed workers.

cient of (ex-exposed workers)/(unexposed workers) was significantly positive (+0.915, $p<0.01$) after adjustment for confounding variables.

Table 4. Regression coefficients of CS_2 exposure status and other possible confounders of nerve conduction velocity (NCV) of the median nerve on application of a multiple regression model

	NCV at the follow-up study		Reduction in NCV during the 6-year follow-up	
	MCV	SCV	MCV	SCV
Intercept	60.33 ***	59.40 ***	–1.649	–0.116
Age (years)	–0.083 ***	–0.140 ***	–0.028	–0.042
BMI (kg/m^2)	–0.051	–0.228 **	0.045	–0.115
Smoking status				
Former smoker/Never smoked	0.017	0.667	–0.257	–0.340
Current smoker/Never smoked	–0.265	0.139	–0.193	0.125
Alcohol drinking				
Occasional drinker/Non drinker	0.009	0.156	–0.233	–0.323
Habitual drinker/Non drinker	0.270	–0.218	0.260	0.152
Educational status				
High school or above/Others	0.300	0.338	–0.015	0.073
CS_2 exposure status				
Ex-exposed/Unexposed	–0.293	0.523	–0.049	0.915 **
1st Tertile exposed/Unexposed	0.185	0.231	–0.074	–0.153
2nd Tertile exposed/Unexposed	–0.377	–0.644	0.259	–0.350
3rd Tertile exposed/Unexposed	–0.611	–1.242 *	–0.187	–1.021 *

*, **, ***: $p<0.05$, 0.01 and 0.001.

Discussion

This cohort study showed that 6-year CS_2 exposure around a mean level of 6 ppm did not affect MCV reduction but induced a significant SCV reduction beyond the influence of aging. Our findings suggest that the effect of CS_2 on SCV around a mean exposure level of 4 ppm may be reversible, since SCV reduction of the ex-exposed workers was almost the same as that of the unexposed workers at follow-up and a partial regression coefficient of ex-exposed workers/unexposed workers was significantly positive when SCV reduction was used as an outcome variable.

In a cross-sectional design, we observed that CS_2 exposure was significantly associated with slowness in MCV at baseline. Various cross-sectional studies have reported an association between occupational exposure to CS_2 and slowness in MCV or SCV[1,7,9,10,12,13,15]. The most common observations are characterized by slower conduction velocity in the motor and, in some instances, sensory nerves, and effects are most pronounced in the more distal portions of the nervous system. Johnson et al.[7] associated CS_2 exposure to effects on peripheral nervous system conduction in workers in a viscose rayon factory. Workers were divided into 3 historical mean exposure level groups (1.0, 4.1, and 7.6 ppm) and found to have small but statistically significant slowness in sural SCV and peroneal MCV as compared to workers exposed at lower concentrations. Hirata et al.[15] reported significant slowness in MCV of the sural nerve as a consequence of chronic occupational exposure to low levels of CS_2 in which the average daily exposure was 1.45 ppm. These epidemiological findings are consistent with the results of our baseline study. However, the slower MCV and SCV reported in these studies were due to previous CS_2 exposure and the effect of CS_2 exposure on NCV was not assessed prospectively.

Some studies have reported an improvement in NCV after removal of occupational exposure to CS_2[17-19]. Huang et al.[17] observed a tendency toward an improvement in NCV in 6 polyneuropathy patients with 3 years of follow-up. Corsi et al.[19] observed no significant improvement in the conduction velocity of slower motor fibers in 12 subjects with neuropathy as compared to those examined 4 years before. However, these studies were case series in which study subjects were patients with CS_2 poisoning due to high-level CS_2 exposure. To our knowledge, this study is the first human study to assess NCV reduction among workers exposed to relatively low CS_2 concentrations (around 5 ppm) and the first study to suggest the reversibility of CS_2 exposure-related impairment of NCV using a prospective design.

In high-level CS_2 exposure (above 100 ppm), NCV reductions were observed with histopathological alterations in the axon of both humans[17] and experimental ani-

mals[20,21]. Exposure to CS_2 can result in peripheral neuropathy for the largest and longest myelinated axons. Axonopathy is first observed in the long distal axons, where it is characterized by axonal swelling with the accumulation of neurofilament proteins in distal motor and sensory nerve tracts, with the detection most often proximal to the nodes of Ranvier. However, no study has examined histopathological alterations in the axon of humans or experimental animals exposed to low-level CS_2. In this cohort study, low-level 6-year CS_2 exposure did not affect MCV reduction but induced a significant SCV reduction. This means that other mechanisms may be involved in reversible slower SCV in low-level CS_2 exposure, and therefore, further studies are required.

Our study has several advantages, including the use of appropriately unexposed workers, a high follow-up rate, and detailed assessment of exposure during the study period. However, several limitations of this study should also be noted. First, potential loss-to-follow-up biases should be considered. A total of 84 subjects was lost to follow-up from the original cohort. Of these, there were no workers who retired due to health problems, and, between workers followed-up and loss-to-follow-up, there were no differences in CS_2 exposure concentration (5.26±3.05 vs. 5.43±3.71 ppm) and in NCV at baseline (Exposed workers: MCV; 57.63±3.62 vs. 57.7±4.26 m/s, SCV; 52.03±4.66 vs. 52.11±5.57 m/s, Unexposed workers: MCV; 58.97±3.3 vs. 59.37±3.88 m/s, SCV; 52.89±4.1 vs. 52.93±5.1 m/s, respectively). Therefore, loss-to-follow-up biases are not likely to affect the outcomes of this study. Second, there was a lack of information on previous CS_2 exposure concentration. In general, exposure concentrations to hazardous chemicals have been improved by the introduction of effective industrial hygiene control measures in occupational settings, as shown in our previous studies[4-6]. The most important question is whether the increased risk that we observed was caused by previous or recent exposure. As we have no information on previous exposure levels, it is not easy to determine to what extent the present exposure to CS_2 accounts for NCV reduction over the 6-year study period. As shown in Table 3, we continuously compared the exposed workers with ex-exposed workers and observed less SCV impairment of the median nerve in the ex-exposed workers. This result indicates that not only previous exposure but also recent exposure had some impact on SCV of the median nerve in this population. Third, although long peripheral nerves, such as the sural nerve, may be more suitable to evaluate the effects of neurotoxins, we measured NCV of the median nerve only due to the limited time allowed for comprehensive health checkups, including examinations of sclerosis of the carotid and aortic arteries, at rest and exercise ECG, ophthalmological test, neurobehavioral examinations, and NCV test at the rayon factories[4-6]. If we could examine the long nerves, we could have observed clearer outcomes than those in this study.

In conclusion, this cohort study showed that 6-year CS_2 exposure around the mean level of 6 ppm did not affect MCV reduction but induced a significant SCV reduction beyond the influence of aging. These findings indicate that the CS_2 exposure level observed in this cohort study was not sufficiently low to prevent the effects of CS_2 exposure on SCV. On the other hand, the effect of CS_2 exposure on SCV around a mean exposure level of 4 ppm may be reversible, since it disappeared in the ex-exposed workers who had ceased to be exposed for the mean period of 4.1 years.

Conflicts of interest: The authors declare that there are no conflicts of interest.

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