

Some hygienic and clinical observations on styrene exposure

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AXELSON, O. and GUSTAVSON, J. Some hygienic and clinical observations on styrene exposure. *Scand. j. work environ. & health* 4 (1978): suppl. 2, 215—219. Styrene exposure levels in the range of 100—300 ppm as an 8-h time-weighted average have been measured in small shops manufacturing glass fiber reinforced plastic products such as boats, steeping baths, etc. Exposure control through determinations of mandelic acid in the urine at the end of the workday has been found suitable and convenient. Over the years during the 1970s, there seems to have been amendments in the exposure situation in Sweden if judged from mandelic acid monitoring. More or less severe central nervous disturbances have been observed among the workers. Elevated levels of amino-transferases have been recorded but are of unclear long-term importance to liver function.

Key words: liver function, pulmonary function, styrene exposure.

High exposure levels to styrene have occurred in the manufacturing of glass fiber reinforced plastic boats, steeping baths, swimming pools, and other products. The styrene-modified polyester plastic is either applied by hand-rolling or by spraying in combination with hand-rolling. Since the manufactured objects are often large, the evaporation of styrene is considerable from the open surfaces. The air concentration of styrene is usually increased further during spraying operations.

HYGIENIC ASPECTS

Exposure levels in the range of 100—300 ppm as 8-h time-weighted average, have occurred, and peak exposures of more than

1,000 ppm were measured in the breathing zone of spraying workers, who were able to stand the irritative styrene vapors without any protective equipment (9). Table 1 shows, in summary, some of the exposure levels measured in various small shops in Sweden during the 1970s. The relationship between mandelic acid in the urine and the 8-h time-weighted average exposure has been fairly well established (4, 6, 7, 9, 11), although some differences occur between various methods and laboratories. Therefore, we have found it convenient to utilize mandelic acid in the urine as far as possible for the routine control of workers' exposure situations.

Table 2 shows routinely gathered data from an industrial health care unit. These data might give some idea of the exposure levels reflected in grab samples, as they are usually taken, but also of adequate time-weighted measurements in relation to determinations of mandelic acid in the urine at the end of the workday. Presumably the mandelic acid values give the

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Table 1. Examples of exposure levels to styrene as measured in various shops in Sweden.

Shop	Work operation or time-weighted average	Duration (min/h)	Concentration (ppm)
1	Spraying; 1 subject	5—10 min	> 1,500
	Spraying and hand-rolling, TWA; 4 subjects	8 h	235—292
2—4	Mainly hand-rolling, TWA; 11 subjects	8 h	17—139
5	Hand-rolling; 9 samples	≈ 1.5 h	7—127
	TWA, two subjects	8 h	< 10; 50

Table 2. Grab samples on consecutive days and full period measurements of styrene in relation to mandelic acid (unspecific method) in urine (1,000 mg/l ≈ 50 ppm styrene, TWA).

Subject	Styrene in air (ppm)				Mandelic acid in urine (mg/l) ^a
	Number of samples	Mean	Minimum	Maximum	
A	3	64	48	74	1,185
	3	65	45	80	1,733
B	3	88	47	162	1,400
	2	112	48	175	1,852
C	2	72	52	91	1,140
	2	44	41	46	1,588
D	2	72	29	116	670
	4	50	19	110	792
E	2	112	85	139	1,795
	3	99	50	162	1,628
F	2	164	126	201	1,276
	3	80	19	149	1,250
G	full period	39	8	211 ^b	860
H	full period	45	13	223 ^b	980

^a Adjusted to a specific gravity of 1.024.

^b Minimum and maximum values for about 1 h.

better estimate of the average exposure (by the method used, a mandelic acid concentration of 1,000 mg/l corresponds to an average exposure of 50 ppm of styrene), since short-time measurements might poorly reflect the true situation due to fluctuations in styrene levels and the influence of the work load (1). During the last few years the exposure situation in this type of

workshop seems to have improved greatly, if judged from exposure control data based on mandelic acid in urine. Better ventilation and an effective utilization of respirators seem to contribute to the amended exposure situation, but also changes in the spraying technique might be of importance, for example, the lowering of the spraying pressure to obtain larger droplets.

CLINICAL OBSERVATIONS

General symptoms

Clinical observations made in connection with the aforementioned high exposure levels have included various, more or less ill-defined complaints, particularly increased tiredness and sleepiness and vague memory disturbances, but also an increased simple reaction time in comparison to those of age-matched referents (9). It is of interest that about a year after we finished the measurements in factory 1 (table 1), we became aware of two individuals who had both worked in this plant and had developed symptoms indicating brain injury. Both, aged 36 and 54, respectively, were given a disability pension. The man aged 54 had apparently misused alcohol for several years of his life but had quit drinking alcohol, even beer, at least five years before styrene exposure began. He worked for several years as a foreman and was considered to have a better than average work capacity. Similarly, the younger man was a good worker, but both men became abnormally tired and depressive after an exposure period of about 3–5 years and were finally unable to continue work. In clinical examinations the Rorschach test indicated a cerebral lesion, and the general clinical picture was that of neurasthenia. The almost contemporary appearance of these two

cases in a group of 10–12 highly exposed workers makes a causal relationship to styrene exposure most likely. [Further details about these cases have been given elsewhere (2).]

Similar cases have appeared also among workers elsewhere in the plastic boat industry. Usually they have had less severe symptoms, but for isolated cases a causal relationship to styrene exposure is more difficult to suggest. However, an abnormal tiredness and an increased demand for sleep is not uncommon among styrene workers with high exposure. In these less severe cases, a quick improvement has resulted from cessation of exposure.

Pulmonary function tests

In spite of high exposure levels (table 1) and the irritative effect of styrene on mucous membranes, we were not able to demonstrate any effects on spirometric variables (vital capacity and forced expiratory volume in 1 s in 17 subjects, studied before and after the workday (table 3).

Observations on liver function tests

The possible effects of styrene on liver function have been a matter of discussion for many years in Sweden, and transient

Table 3. Spirometric findings for styrene workers (percentage of expected values).

Category	Vital capacity		Forced expiratory capacity in 1 s	
	Mean	Range	Mean	Range
Total (N = 17)				
Before work	101	77–123	106	83–133
After work	100	72–122	105	79–135
< 150 ppm ^a (N = 11)				
Before work	103	77–123	107	83–133
After work	101	72–122	106	79–135
> 150 ppm ^a (N = 6)				
Before work	101	88–114	103	93–125
After work	101	90–114	102	92–119

^a 8-h time-weighted exposure.

Table 4. Alanine-amino-transferase (ALAT) and aspartate-amino-transferase (ASAT) among 35 workers with exposure to styrene and among 12 referents (i.e., other industrial workers).

Variable	Exposed	Referents
ALAT (μ kat/l)		
Number of samples	94	20
Geometric mean	0.50	0.40
Maximum	1.72	0.92
Student's t^a		1.60
p (one-tailed)		≈ 0.056
ASAT (μ kat/l)		
Number of samples	93	20
Geometric mean	0.53	0.41
Maximum	1.46	0.72
Student's t^a		3.13
p (one-tailed)		≈ 0.001

^a After $\ln$ transformation.

abnormalities in the serum enzyme patterns have been observed now and then by industrial health physicians. Increased gamma glutamyl transpeptidase levels have been reported in a cross-sectional study of styrene workers (12).

We compiled routinely gathered data on liver enzymes in serum, namely, aspartate-amino-transferase, ASAT (or GOT), alanine-amino-transferase, ALAT (or GPT), and alkaline phosphatase, ALP, from 35 exposed male workers in three different shops within the same company.

The exposure levels were approximately known and corresponded to the lower ranges indicated in table 1, i.e., less than or about 100 ppm of styrene as a time-weighted average. More than 90 samples were taken from these workers during a period of about a year (table 4). For comparison we used 20 samples from 12 healthy male workers in a manufacturing industry. Among the styrene workers, there were eight individuals with increased levels (above 0.67 μ kat/l according to the reference values of the laboratory) of ALAT and/or ASAT on two occasions or more. Another five individuals had elevated levels on one occasion. Among the 12 nonexposed workers there were increased levels of these enzymes in two individuals on one occasion. The alkaline phosphatases were elevated once in one

nonexposed individual versus six times (twice in one man) among the styrene workers. The average transferase levels were higher among those men with exposure, but only ASAT was clearly elevated ($p \approx 0.001$). No difference was found in the average alkaline phosphatase levels, and the means were 3.0 and 3.1 μ kat/l for the exposed and the nonexposed, respectively.

DISCUSSION

Exposure to styrene seems to have been considerable in many of the small workshops manufacturing glass fiber reinforced products from styrene-modified polyester plastic, and the standard (50 ppm) has often been highly exceeded. It is not surprising therefore that central nervous disturbances were found among the workers, and such observations are in agreement with findings reported by others, such as prolonged reaction time (8) and electroencephalographic changes (13). Furthermore, the observations of the central nervous effects of styrene reasonably concur with the indications of similar effects from other solvents (3, 10).

The elevated transferases among the styrene workers are of unclear origin and clinical importance. They seem difficult to explain only in respect to the use of alcohol, since no overconsumption was observed among these workers in spite of the close surveillance of the industrial health care unit. Perhaps low-grade alcohol consumption, e.g., beer, in combination with styrene exposure, could be of etiologic importance as it is in respect to combined exposure to alcohol and other solvents, e.g., isopropylalcohol and trichloroethylene (5, 14). Routine data, as presented in this report, should be interpreted with great caution, but they seem to indicate the need for further studies on the long-term effects of styrene exposure on the liver, especially since a potential hazard of developing cirrhosis cannot be ruled out.

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