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Obstetric histories of women occupationally exposed to styrene

by Hannu Härkönen, MD, Peter C Holmberg, MD, MSc

Environmental and occupational exposure to chemical agents during pregnancy may have a hazardous effect on the developing embryo (1, 10). For example, it has been reported that the inhalation of anesthetics may increase the rate of spontaneous abortions (13). Furthermore exposure to vinyl chloride monomer may induce excessive fetal loss among the wives of exposed workers (6). One study reported an excess of children with central nervous system (CNS) defects born to women exposed to organic solvents during early pregnancy (4).

Styrene is used as a solvent and cross-linking agent in the reinforced polyester plastics industry. This particular production process causes significant exposure to the workers involved (11). Styrene has been shown to possess a mutagenic effect on bacteria (12). Moreover the blood lymphocytes of workers exposed to styrene have also shown an increased frequency of chromosome aberrations (8). In one epidemiologic study, the rate of spontaneous abortions was reported to be significantly increased among women working in the styrene industry (2). A case report dealing with styrene exposure and congenital CNS defects has also been published (5).

The present study was undertaken to compare the obstetric histories of women occupationally exposed to styrene with those of a reference group, with the intention of evaluating the possible differences found.

Subjects and methods

The exposed subjects of the study consisted of 67 female lamination workers occupationally exposed to styrene at six plants manufacturing reinforced polyester plastic products. This group included only women under 40 a of age, their ages ranging from 19 to 40 a (mean 30, SD 6). The duration of past exposure varied from 0.5 to 10 a (mean 4.5, SD 2.6).

The referents were chosen from textile and food production workers of a similar social class, and they were matched with the exposed subjects with regard to age. They were not occupationally exposed to any solvent. The mean age of the referents was 31 a (SD 5).

A special questionnaire was designed for the study. It dealt with such questions as age at menarche, duration of menstrual cycle, changes in the menstrual cycle during the period of styrene exposure, use of contraceptives, pregnancies, spontaneous and/or induced abortions, births, smoking habits during pregnancy, consumption of alcohol during pregnancy, and possible congenital defects of children. The age and occupation of the father were also requested. To make it easier to respond, the questions were put in simple, unambiguous
terms.
The interviews were held during 1979 and 1980. The interviewer questioned each woman personally and conducted all the interviews in a similar manner. During the interview she emphasized the difference between the terms “spontaneous” and “induced” abortion.

The obstetric histories of the subjects were divided according to the time prior to the styrene exposure and the period of styrene exposure. The total duration of exposure was 302 person-years. The obstetric histories of the referents were divided to correspond to the periods of their age-matched exposed subjects.

Results
Prior to the period of styrene exposure, the number of women with pregnancies, births, spontaneous abortions, and induced abortions did not differ significantly for the exposed and reference groups. The number of women with pregnancies in the exposed group during the styrene exposure period was 12, while that for the reference group was 20. The difference was not statistically significant.

The expected number of births, calculated according to the age-specific fertility rates of the Finnish female population for 4.5 a (average length of exposure), was 20.9 for the styrene exposure period (9). During this period there were 14 births among the referents and 4 among the exposed subjects. Although the number of births among the referents during this period did not differ significantly from the expected number, that of the exposed subjects was significantly different from both the expected number ($\chi^2 = 12.87, p < 0.001$, Poisson model) and that of the referents ($\chi^2 = 6.74, p < 0.01$, Poisson model).

In both groups, the number of reported spontaneous abortions was eight for the time before the styrene exposure, and four for the exposure period. During the styrene exposure, the number of induced abortions was eight in the exposed and four in the reference group (table 1).

Two CNS birth defects were reported for the time prior to styrene exposure, one among the exposed subjects and one among the referents. For the exposure period, one oral cleft was reported among the referents, and one congenital heart defect among the exposed subjects.

Discussion
In a previous study of styrene exposure in the Finnish polyester plastics industry, the median time-weighted average exposure to styrene in lamination work was 66 ppm (68 8-h measurements) (7). In the present study no measurements were made to evaluate the level of the exposure.

Styrene has a toxic effect which may result in genetic alterations in either somatic or germ cells. These alterations may possibly effect miscarriages and spontaneous abortions (3). In the present study, no significant differences in the number of pregnancies were found between the women exposed to styrene and the reference group. But in comparison to the reference group, women from a similar social class, the women of the styrene-exposed group had fewer births during the period of exposure. One cause leading to this difference seems to be the higher number of induced abortions; the difference, however,

| Table 1. Number of women with pregnancies, births, spontaneous abortions, and induced abortions among the styrene-exposed subjects (N = 67) and the referents (N = 67). The number of occurrences is given in parentheses. |
|-------------------------------------------------|
| Before styrene exposure | During styrene exposure |
|-------------------------------|------------------------|
| Exposed subjects | Referents | Exposed subjects | Referents |
| Pregnanies | 48 (84) | 48 (80) | 12 (16) | 20 (22) |
| Births | 40 (69) | 39 (67) | 3 (4) | 14 (14) |
| Spontaneous abortions | 8 (8) | 5 (8) | 3 (4) | 4 (4) |
| Induced abortions | 6 (7) | 5 (5) | 8 (8) | 4 (4) |
Table 2. Menstruation history, use of contraceptives and drugs, and smoking habits and consumption of alcohol during pregnancy in the two groups.

|                          | Exposed subjects | Referents |
|--------------------------|------------------|-----------|
| Age (a) at menarche (mean ± SD) | 13.4 ± 1.23      | 13.3 ± 1.67 |
| Duration of menstruation cycle (mean ± SD) | 28.1 ± 2.26      | 27.7 ± 1.9  |
| Menstruation             |                  |           |
| Regular (N)              | 59               | 61        |
| Irregular (N)            | 8                | 6         |
| Changes in menstruation during the exposure period |                  |           |
| No (N)                   | 54               | 50        |
| Yes (N)                  | 13               | 17        |
| Use of contraceptives    |                  |           |
| No (N)                   | 16               | 14        |
| Sometimes (N)            | 8                | 8         |
| Regularly (N)            | 43               | 45        |
| Use of drugs             |                  |           |
| No (N)                   | 61               | 57        |
| Yes (N)                  | 6                | 10        |
| Smoker during pregnancy a |                  |           |
| No (N)                   | 53               | 79        |
| Yes (N)                  | 42               | 23        |
| Consumption of alcohol during pregnancy |                  |           |
| Not at all (N)           | 68               | 83        |
| Sometimes (N)            | 27               | 19        |

*a Data for five exposed subjects missing.

was not statistically significant.

The groups did not differ in the use of contraceptives or the use of drugs, but smoking and the consumption of alcohol during pregnancy were more common in the exposed group. There were no significant differences in the menstrual cycles of the women in the two groups, nor were there significant changes in the menstrual cycles of the women of either group during the period of exposure (table 2).

In 1976 Finland had about 170 plants manufacturing reinforced polyester plastic products. One hundred and seventy-nine women (age 20—39 a) worked at these plants and were exposed to styrene for more than 4 h per workday. It is our impression that, in recent years, the number of female workers in this industry has been decreasing in Finland. When the workplaces were visited, however, the women appeared concerned about the possible adverse health effects which exposure to different chemicals could induce.

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Acute solvent-ethanol interactions with special reference to xylene

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RIIHIMÄKI V, LAINE A, SAVOLAINEN K, SIPPEL H. Acute solvent-ethanol interactions with special reference to xylene. Scand j work environ health 8 (1982) 77—79.

Acute ethanol ingestion inhibits the metabolism of the common industrial solvents trichloroethylene and dimethylformamide. The solvents in turn may interact with ethanol metabolism as shown by an accumulation of acetaldehyde and occasional symptoms of alcohol intolerance. It was recently found that mutual metabolic interaction occurs even in the context of ethanol ingestion (0.8 g/kg in single dose) combined with subsequent inhalation exposure to m-xylene (6.0 & 11.5 mmol/m³ (140 & 280 ppm), over 4 h). Ethanol impaired the metabolic clearance of m-xylene, raised the blood xylene concentration, and decreased the urinary excretion of methylhippuric acid. Thus, ingestion of ethanol is a noticeable source of error in the biological monitoring of xylene uptake. Some people appear to be susceptible to combined ethanol-xylene exposure and may develop nausea and dermal flush.

Key terms: carbon disulfide, dimethylformamide, ethanol, industrial solvents, metabolic interaction, trichloroethylene, m-xylene.

Ethanol is known to inhibit drug metabolism acutely (14, 15), and to stimulate drug metabolism through microsomal induction when administered repeatedly (6, 7, 15). Thus the effects of ethanol on solvent metabolism may be complex, depending on the timing and frequency of ethanol ingestion. In this communication we want to call attention to the immediate metabolic consequences following ethanol ingestion in the context of solvent exposure, as reported in the literature and observed in our own studies.

Ethanol-mediated changes of solvent metabolism

Molar concentrations of ethanol in blood after social drinking are at least 100—200

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