ORIGINAL ARTICLE

Predictors of occupational exposure to styrene and styrene-7,8-oxide in the reinforced plastics industry

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Objective: To identify demographic and work related factors that predict blood levels of styrene and styrene-7,8-oxide (SO) in the fibreglass reinforced plastics (FRP) industry.
Methods: Personal breathing-zone air samples and whole blood samples were collected repeatedly from 328 reinforced plastics workers in the Unuted States between 1996 and 1999. Styrene and its major metabolite SO were measured in these samples. Multivariable linear regression analyses were applied to the subject-specific levels to explain the variation in exposure and biomarker levels.
Results: Exposure levels of styrene were approximately 500-fold higher than those of SO. Exposure levels of styrene and SO varied greatly among the types of products manufactured, with an 11-fold range of median air levels among categories for styrene and a 23-fold range for SO. Even after stratification by job title, median exposures of styrene and SO among laminators varied 14- and 31-fold across product categories. Furthermore, the relative proportions of exposures to styrene and SO varied among product categories. Multivariable regression analyses explained 70% and 63% of the variation in air levels of styrene and SO, respectively, and 72% and 34% of the variation in blood levels of styrene and SO, respectively. Overall, air levels of styrene and SO appear to have decreased substantially in this industry over the last 10–20 years in the US and were greatest among workers with the least seniority.
Conclusions: As levels of styrene and SO in air and blood varied among product categories in the FRP industry, use of job title as a surrogate for exposure can introduce unpredictable measurement errors and can confound the relation between exposure and health outcomes in epidemiology studies. Also, inverse relations between the intensity of exposure to styrene and SO and years on the job suggest that younger workers with little seniority are typically exposed to higher levels of styrene and SO than their coworkers.

Styrene, its polymers and copolymers are used in the production of polystyrene resins, styrene-butadiene rubber, and reinforced plastics.[1] Approximately 18 million metric tons of styrene were produced worldwide in 1998, with North America, Western Europe, and Asia contributing about 93% of the total production.[2] Styrene is also widespread in the environment, following its release into air by industrial processes, vehicle exhaust, cigarette smoke, consumer products and its presence in food items that either naturally contain styrene (for example, cinnamon) or are stored in polystyrene containers.[3]

Since styrene has been classified as a possible human carcinogen,[2] there are concerns that occupational and environmental exposures to this chemical may be hazardous. Although environmental exposures to styrene are low (for example, μg/m^3 levels in air), occupational exposures are much higher (mg/m^3 levels in air), particularly in the fibreglass reinforced plastics (FRP) industry, where processes are difficult to control.[4] Average air concentrations of styrene have been reported between 23 and 171 ppm (one ppm styrene = 4.26 mg/m^3) during the lamination of FRP products in different countries.[1]

Styrene uptake occurs predominantly through inhalation and virtually all of the blood dose is metabolised to styrene-7,8-oxide (SO) via cytochrome P450 enzymes (mainly via CYP2B6, CYP1A2, CYP2E1, and CYP2C8).[5] Toxic effects of styrene have been attributed to SO, which is mutagenic and a probable human carcinogen.[2] Although rarely mentioned, direct exposures to SO can also occur at low (μg/m^3) levels in styrene-using industries where SO is released into air as a byproduct of styrene oxidation.[6–11] The biological importance of SO is generally considered within the context of styrene metabolism due to the vastly greater abundance of styrene in air. However, this assumption has been questioned because, whereas metabolically derived SO is efficiently detoxified by epoxide hydrolases in the liver, inhaled SO is rapidly absorbed and distributed to the tissues where it can exert genotoxic effects.[6 8 12]

Different factors have been linked to increased levels of styrene exposure in the FRP industry. In a review of industrial hygiene surveys, Lemasters *et al*[13] observed significant differences in airborne styrene levels due to the type of product (highest in the boat and truck manufacturing groups) and the type of process (open moulds higher than press moulds). Other studies reported that styrene levels varied considerably in air due to differences in the following factors: job (higher exposures in hand lamination),[1 7] amount of resin used,[7] shapes of moulds (higher exposures observed with concave moulds than with convex moulds),[14] and size of the work area combined with the efficiency of the ventilation systems (lower exposures in better ventilated larger rooms).[14] In contrast to these studies of styrene exposure in the FRP industry, factors influencing SO levels in air have not been reported, other than the observation that air concentrations of SO were higher in processes using low styrene emitting resins.[7]

Abbreviations: CYP, cytochrome P450 enzymes; GC-MS, gas chromatography-mass spectrometry; FRP, fibreglass reinforced plastics; GM, geometric mean levels; LOD, limit of detection; ppb, parts per billion; ppm, parts per million; PTFE, polytetrafluoroethylene; RV, recreational vehicle; SO, styrene-7,8-oxide

The uptake of styrene in humans has also been extensively investigated in terms of styrene levels in blood and exhaled air following controlled exposures,[15–20] and in FRP workers.[18 21 22] Yet again, the rich database of styrene levels can be contrasted with the paucity of data regarding SO levels in blood or biological fluids.[8 16 21 23 24] This is due, in part, to the reactivity of SO in blood (the half life in human blood was estimated to be 42 minutes in vitro[25]) and to the low air concentrations of SO which are difficult to measure.

Given the potential importance of styrene and SO exposure to health effects, the current study was undertaken to identify demographic and work related predictors of blood levels of styrene and SO in workers exposed simultaneously to these air contaminants in the FRP industry.

METHODS

Subjects and sampling methods

This study was conducted in conjunction with another investigation of potential neurological effects of styrene exposure among FRP workers. A total of 328 subjects of both sexes were recruited from 17 similar sized FRP factories in the Pacific Northwest (Puget Sound region) of the United States. A substantial proportion of workers (45.3%, n = 148) were employed for less than one year. Repeated samples of breathing-zone air and venous blood were obtained from nearly all subjects during two or three surveys between 1996 and 1999. For each survey, a median of four (range 1–8) personal breathing zone styrene measurements (full shift) were made and a median of two (range 1–3) blood samples were collected on separate workdays over a one month period (blood was collected within 10 minutes of the end of the shift on one of these days). Surveys were generally months apart. During each survey, interviews were conducted to collect information on demographic and lifestyle factors, work history, medical history, and dietary information. Information obtained from the first survey was used in statistical analyses. More detailed descriptions of the field studies have been reported previously.[6 24 26]

Air monitoring of styrene and SO

Personal full shift breathing-zone air samples were collected using passive monitors (Cat No 3500, 3M Corp, St Paul, MN, USA). Details of the analysis of air samples have been provided elsewhere.[6] Briefly, passive monitors were capped immediately after sampling and were stored at room temperature up to 24 hours before processing. Samples were then desorbed (in their holders) with 1.5 ml ethyl acetate, transferred to 4 ml glass vials sealed with polytetrafluoroethylene (PTFE) lined caps, and stored at −20℃ for up to one month before analysis. For each sample, 1 µl of the solution was injected into a gas chromatograph (GC) coupled with either a flame ionisation detector (for styrene) or an electron impact mass spectrometry (MS) detector (for SO).

Analysis of styrene and SO in blood

Whole blood samples were collected into heparinised tubes. Details of the extraction and quantification of the analytes from blood samples were described previously.[24] Briefly, 2 ml aliquots of the blood were transferred to 8 ml vials (sealed with PTFE lined screw caps) each containing 4 ml of *n*-pentane and the internal standards (1 ng of [$^{2}H_8$]SO and 2 µg of [$^{2}H_8$]styrene), and then mixed for 30 seconds using a vortex mixer. The final extracts of styrene and SO in pentane were analysed by GC-MS with positive ion-chemical ionisation.

Statistical analyses

Statistical analyses were performed using SAS software (v8.2, SAS Institute, Cary, NC, USA). Spearman correlation coefficients were calculated using subject-specific geometric means (GMs) of exposure and biomarker levels (Proc CORR of SAS). All other analyses were performed after natural logarithmic transformation of the data to satisfy normality assumptions.

We performed preliminary analyses by applying mixed effects models to individual air and blood measurements, collected on each day of sampling. However, the resulting models were unstable because of the highly unbalanced data (1–8 repeated measurements per person of styrene and SO in air and 1–3 repeated measurements of styrene and SO in blood) plus the high percentage of censored values for SO measurements in blood. Thus, we chose the simpler approach of modeling individual mean levels (in log scale) using weighted stepwise linear regression analyses. Weighting by the number of measurements per subject, we were able to overcome problems with the unbalanced data. Since covariates were constant within subjects over time, aggregation by subject presented no other particular problems and provided independent data for multiple regression analyses. The effects of demographic and workplace characteristics were investigated using the subject-specific air and/or blood concentrations via Proc REG of SAS. Weighted multivariable linear regression methods were used, with the weights being the numbers of repeated measurements per subject. Multivariable models had the general form:

$$Y_i = \alpha + \sum_{j=1}^{p} \beta_j X_{ij} + \varepsilon_i$$

where Y_i represents the subject-specific mean of log transformed levels of styrene or SO in the air or blood for the *i*th subject, α is the intercept representing the average level of Y_i when all independent variables are zero, β_j is the regression coefficient for the *j*th independent variable X_{ij} for the *i*th subject, and ϵ_i is the error term. For models in which Y represented the blood level of styrene or SO in blood, the subject-specific mean of log transformed levels of styrene in air was used to represent the independent variable for air exposure. Because of missing values, hot tub workers were excluded from the model of SO levels in blood.

The following independent variables were considered for the regression models of air styrene and SO: product category (boat, hot tub, pipe and tank, RV, truck), job title (laminators, grinders, others), number of years in current job, gender, age, and body mass index (BMI). Additionally, the following variables were considered for the blood levels of styrene and SO: air levels of styrene, current alcohol use, number of cigarettes/day, and the use of cinnamon as spice in food or drink. The exposure and biomarker levels were first separately regressed on each potential independent variable. Those that suggested significance ($p<0.10$) were retained for further analysis. The following variables were retained (with signs of the coefficient given in parentheses):

1. Styrene in air: laminator (+), grinder (−), boat production (−), pipe and tank production (−), RV production (+), years on current job (−), and age (−).
2. SO in air: boat production (−), RV production (+), truck production (+), laminator (+), grinder (−), years on job (−), age (−), and air concentration of styrene (+).
3. Styrene in blood: boat production (−), pipe and tank production (+), RV production (+), laminator (+), grinder (−), years on job (−), male gender (−), age (−), styrene measurements in air (+), current alcohol use (+).
4. SO in blood: boat production (−), RV production (+), truck production (−), laminator (+), grinder (−), male gender (−), age (−), styrene measurements in air (+), current alcohol use (+).

Table 1 Distribution of reinforced plastics workers by product category, race, gender, and smoking status

Product	Dates surveyed (month/ year)	Workers (n)	White (%)	Males (%)	Smokers (%)
Boat building	03/1997–12/1999	138	103 (74.6)	119 (86.2)	59 (42.8)
Hot tub	02/1997–10/1997	13	12 (92.3)	12 (92.3)	7 (53.9)
Pipe and tank	06/1997–04/1999	50	47 (94.0)	43 (86.0)	32 (64.0)
RV	01/1999–06/1999	48	20 (41.7)	46 (95.8)	20 (41.7)
Truck	10/1997–10/1998	76	42 (55.3)	60 (79.0)	30 (39.5)
Total	12/1996–12/1999	328	227 (69.2)	283 (86.3)	151 (46.0)

Stepwise selection of all retained independent variables and their plausible two-way interactions (between product group and job title and between years on job and age, where applicable) was used to achieve final models (using a significance level of $p<0.05$ for retention). No interaction was significant. In final models, each job group was represented in each product category and the smallest cell contained eight subjects (grinders who manufactured RVs). Potential outliers and influential observations were investigated using Studentised residuals and Cook's distance. No influential outliers were detected. Collinearity was investigated via eigenvalues and condition indices and was not detected for any model.

For the air and blood levels, observations below the limit of detection (LOD) were assigned 2/3 of the LOD value.[27] The limit of detection for styrene and SO in air were 1.00 ppm and 1.00 ppb, respectively, as described previously.[6] The limit of detection values for styrene and SO in blood were 0.001 mg/l and 0.05 µg/l.[24] For the subject-specific measurements, the frequencies of censored (below LOD) observations were as follows: 3.96% (13/328) for styrene in air, 3.66% (12/328) for SO in air, 2.03% (6/295) for styrene in blood, and 38.7% (82/212) for SO in blood. Due to the high number of censored values of SO in blood, the corresponding multivariable regression analysis was repeated using a maximum likelihood procedure (Proc LIFEREG of SAS). Since similar parameter estimates and standard errors for the covariates were observed using Proc LIFEREG and Proc REG, results are only presented for the multivariable regression analysis using Proc REG.

RESULTS

Demographic and descriptive statistics

Demographic characteristics of the samples of workers are summarised in table 1. The largest percentage of subjects (42.1%) worked in the boat building industry, followed by truck manufacturing (23.2%), and manufacture of pipes and tanks or recreational vehicles (RVs) (about 15% each). Of all subjects, 69.2% were white, 24.7% were Hispanic, and 3.66% were Asian. Subjects were predominantly male (86.3%) and were mostly non-smokers (54%). The ages ranged between 18 and 61 years (average of 35.6 years). Subjects reported having worked in the FRP industry for an average of 6.3 years (range <1–39 years), of having been in the current job for an average of 3.8 years (range 0–30.7 years), and of working an average of five days per week (range 3–7 days/week).

Based on their job titles, subjects were stratified into three main job groups. The job group defined as ''laminators'' (including gel coaters, rollers, and fibreglass fabricators) contained most of the subjects (n = 182, 55.5%), followed by the group defined as ''other job titles'' (including maintenance workers, supervisors, workers in trades, office workers, and developers, n = 89, 27.1%), and the group defined as ''grinders/sanders'' (including drillers, finishers, patchers, pullers, and mold maintenance workers; n = 57, 17.4%).

Table 2 summarises environmental and blood levels of styrene and SO. Based on repeated personal sampling (median = 4 samples/subject), exposure to styrene in air (median = 9.14 ppm) was typically 535-fold higher than exposure to SO (median = 17.1 ppb). Blood concentrations of styrene were typically 1200-fold higher (median = 0.083 mg/l) than those of SO (median = 0.069 µg/l). Overall, the median level of styrene in air (9.14 ppm) was lower than the current TLV-TWA of 20 ppm (ACGIH, 2005). However, as shown in table 3A, air levels of styrene were unevenly distributed across product types, ranging from a median of 4.22 ppm during truck manufacture to 45.1 ppm for production of RVs. A similar pattern was observed when levels were examined for laminators only (table 3B).

Table 2 Summary of measurements (based on subject-specific GMs) of air and blood samples

Measure	Units	Median	Q1–Q3	Min-Max	N	n/subject (range)
Styrene (air)	ppm	9.14	2.71–22.2	<1–117	328	4 (1–8)
SO (air)	ppb	17.1	7.86–29.8	<1–138	328	4 (1–8)
Styrene (blood)	mg/l	0.083	0.023–0.259	<0.001–2.05	295	2 (1–3)
SO (blood)	µg/l	0.069	<0.05–0.135	<0.05–0.393	212	1 (1–2)

Q1–Q3, interquartile range (25th–75th percentile); N, number of subjects; n, median (range) of the number of measurements per subject.

Correlation of air and blood levels of styrene and SO

Table 4 summarises the Spearman correlations of subject-specific GM levels of styrene and SO in air and blood. All variables were significantly correlated ($p<0.0001$), with coefficients ranging from 0.34 (SO in air *v* SO in blood) to 0.85 (styrene in air *v* styrene in blood).

Table 3 Measurements of styrene and SO in air by product category (based on subject-specific GMs)

Product group	n	Styrene in air (ppm) Median	Styrene in air (ppm) Min–Max	SO in air (ppb) Median	SO in air (ppb) Min–Max
(A) Measurements from all subjects					
Boat building	138	4.41	<1–68.6	9.47	<1–51.1
Hot tub	13	6.85	<1–62.9	16.0	<1–48.8
Pipe and tank	50	16.0	1.67–79.0	17.8	2.02–138
RV	48	45.1	6.74–117	44.0	10.4–109
Truck	76	4.22	<1–46.3	22.2	3.75–64.8
(B) Measurements from laminators only (based on subject specific GMs)					
Boat building	70	14.4	<1–68.6	13.3	<1–51.1
Hot tub	7	9.84	<1–62.9	36.4	<1–48.8
Pipe and tank	42	18.1	2.57–79.0	21.2	2.56–138
RV	30	60.5	14.4–117	59.0	10.4–109
Truck	30	21.6	3.84–46.3	27.7	15.9–64.8

Table 4 Correlation among subject-specific geometric mean levels of styrene and SO in air and blood (Spearman coefficients, p values, and numbers of observations)

	Styrene (air)	Styrene (blood)	SO (blood)
SO (air)	0.76 (<0.0001) 328	0.70 (<0.0001) 295	0.34 (<0.0001) 212
Styrene (air)		0.85 (<0.0001) 295	0.42 (<0.0001) 212
Styrene (blood)			0.49 (<0.0001) 212

Multivariable regression analyses

Results of weighted multivariable stepwise linear regression analyses of air levels of styrene and SO (under Model 1) are summarised in table 5. Regarding air levels of styrene, the final model explained 70% of the variation and included the following independent variables: work as a laminator (yes/no), working in production of RVs (yes/no), years on the current job, age, and work as a grinder/sander (yes/no). The corresponding model for SO in air explained 63% of the variation and included the same variables as for styrene in air, plus work in the production of trucks (yes/no).

Results of weighted multivariable stepwise regression analyses of blood levels of styrene and SO (under Model 1) are summarised in table 6. Final models for styrene and SO in blood explained 72% and 34% of the variation, respectively. Two variables were common to both models, namely, styrene in air and working in RV production (yes/no). In addition, working in the manufacture of trucks, and pipes and tanks (yes/no), and current alcohol use were significant predictors of styrene in blood.

DISCUSSION

Levels of styrene and SO among individual workers were extremely heterogeneous in the FRP industry, even within a single job title or product group. For example, among laminators (the largest group of subjects), the median values of individual GM air levels ranged from less than one to 117 ppm for styrene and from less than one to 138 ppb for SO (table 3). Regarding air levels of styrene in all subjects, GM values decreased with product categories in the order RVs > pipe and tank > hot tub > boat building ≅ truck; however, for SO levels, the order was RVs > trucks > pipe and tank > hot tub > boat building. It appears that different jobs, workplace characteristics, equipment, and activities involving a given type of product contributed to the large range of individual GM levels observed.

Levels of exposure to styrene and SO varied by product categories even when analyses were restricted to laminators (table 3B). This is noteworthy because retrospective epidemiological studies of FRP workers (reviewed in the IARC report[2]), which have been largely negative, have relied exclusively upon job titles across the FRP industry to classify styrene exposures and have ignored coexposures to SO. Such an approach might introduce adverse effects of measurement error (for example, attenuation bias) into the analysis because particular product categories can greatly influence the within-subject variability among workers sharing a given job title.[28 29] We recommend that epidemiologists use both product category and job title to categorise exposures in future studies of health effects in the FRP industry.

When exposure levels were examined by product categories, we observed lower levels than those reported in previous studies.[13 14 30] Most prior studies focused upon styrene exposures. In a summary of measurements from 28 manufacturers between the years 1975 and 1980, Lemasters *et al* reported the highest average styrene exposures among manufacturers of truck parts and boats (61 and 82 ppm, respectively).[13] Studies that primarily focused on boat factories reported average styrene exposures from 15.1 to 82 ppm between the years of 1969 to 1990, with laminators having the highest levels of exposure.[7 8 13 30] Kolstad *et al* also observed higher styrene exposures in companies producing boats when compared to other products; however, they reported that styrene levels declined 10-fold between the 1960s and the 1990s.[30] In the current study, with measurements between 1996–99, we observed much lower levels of styrene in the boat manufacturing group (median = 4.41 ppm). Even the highest styrene levels that we observed (RV manufacturers, median = 45.1 ppm) were quite modest when compared to past air measurements from companies with open moulds.[13]

Only a few studies reported SO measurements in personal air samples collected from FRP workers. In two factories producing boats, car parts, and building materials, Pfaffli *et al* reported an average of 40 ppb (SD = 20) of SO in hand lay-up applications and 120 ppb (SD = 70) in spray applications.[10] These levels are similar to those reported in other studies conducted in the 1970s and 1980s in factories producing car parts, mudguards and biological toilets,[11] and boats.[8] Even though the overall range of SO levels in air in the current study was similar to previous reports (<1–138 ppb, table 2), we observed varying median levels for different product groups (ranging from 1.90 ppb to 44.0 ppb, table

Table 5 Multivariable weighted stepwise linear regression models of subject-specific means of log transformed styrene and SO levels in air (weights are the frequencies of subject-specific repeated measurements of styrene and SO, respectively)

Response variable (n = 325)	Overall r^2	Predictor variables	Parameter estimates	Δr^2	95% CI
ln(Styrene in air, ppm)	0.70	Intercept	1.37		1.01 to 1.72
		Laminator	1.77	0.46	1.58 to 1.96
		RV production	1.41	0.21	1.21 to 1.62
		Years on job	−0.024	0.02	−0.041 to −0.008
		Age (years)	−0.012	0.01	−0.020 to −0.003
		Grinder/sander	0.286	0.01	0.033 to 0.538
ln(SO in air, ppb)	0.63	Intercept	1.64		1.26 to 2.01
		RV production	1.43	0.21	1.22 to 1.65
		Laminator	1.40	0.18	1.20 to 1.59
		Truck manufacture	0.985	0.14	0.773 to 1.20
		Grinder/sander	1.08	0.08	0.824 to 1.34
		Age (years)	−0.012	0.02	−0.020 to −0.003
		Years on job	−0.020	0.01	−0.037 to −0.003

Table 6 Multivariable weighted stepwise linear regression models of subject-specific means of log transformed styrene and SO levels in blood (weights are the frequencies of subject-specific repeated measurements of styrene and SO in blood, respectively)

Response variable	Overall r^2	Predictor variables	Parameter estimates	Δr^2	95% CI
ln(Styrene in blood, mg/l, n = 292)	0.72	Intercept	−5.27		−5.52 to −5.02
		ln(Styrene in air, ppm)	0.994	0.68	0.899 to 1.09
		Truck manufacture	0.807	0.02	0.525 to 1.09
		Current alcohol use	0.358	0.01	0.135 to 0.581
		Pipe and tank production	0.535	0.01	0.204 to 0.866
		RV production	0.540	0.01	0.168 to 0.912
ln(SO in blood, µg/l, n = 211)	0.34	Intercept	−3.09		−3.26 to −2.92
		RV production	0.750	0.30	0.517 to 0.982
		ln(Styrene in air, ppm)	0.140	0.04	0.065 to 0.214

3A). Furthermore, the rankings of levels of exposure to styrene and SO were not consistent across product categories, suggesting that workers in some product categories were exposed to different proportions of the two air contaminants. Since SO is the putative toxic species related to health effects from exposure to styrene and SO, this lack of concordance in air levels of the two compounds across jobs and product categories can be an important source of confounding in epidemiological studies of the FRP industry. Thus, we encourage investigators to consider exposures to both styrene and SO in investigations of health effects in this industry.

Referring now to broad determinants of exposure to styrene and SO (table 5), we found several common factors and some differences. The job title ‘‘laminators’’ was an important predictor of both types of exposures, consistent with previous reports.[1 7 31] Also the job title ‘‘grinders/sanders’’ was a significant predictor of exposure to both air contaminants. Although the particular origins of SO in the FRP industry are not well understood, it is common for workers in the grinders/sanders category to mix small quantities of styrene-containing resins with catalysts containing hydroperoxides for repair work. Hydroperoxides initiate the polymerisation process and can promote oxidation of styrene to SO and other reactive species.[32–34] Likewise, while we observed that production of RVs was a significant predictor of both styrene and SO levels in air, production of trucks was a significant predictor of SO levels only. It is worth investigating whether the particular formulations of resins or hydroperoxides used in truck production enhance formation of SO in situ.

Subjects who were older, or who spent more years in their current job, had significantly lower exposures to styrene and SO. The fact that both age and years on the job remained in the final models, but were only weakly correlated (Spearman $r = 0.31$), indicates that exposure was reduced with both age and seniority. As several epidemiological studies of FRP workers have noted that the prevalence of particular cancers was higher among workers with the shortest work histories,[35–39] it should be kept in mind that entry level workers in this industry appear to have been exposed to much higher air levels of styrene and SO during their work histories than their more senior coworkers.

When we examined the blood levels of styrene and SO, our models could explain much more of the variability of styrene ($r^2 = 0.72$) than of SO ($r^2 = 0.34$, table 6). This may have resulted in part from the relatively high proportion of blood measurements of SO below the LOD of our assay. Even though styrene exposure was included in both final models, types of products remained as significant predictors of the blood levels, suggesting that factors other than airborne styrene were also important. Influential product types were RVs, for both styrene and SO in blood; and trucks, and pipes and tanks, for styrene in blood only. Finally, we observed that current alcohol users had higher levels of styrene in their blood. Although it would have been reasonable to expect levels of SO to increase in blood due to the induction of CYP2E1 by alcohol,[40–42] it is not clear why alcohol consumption would increase the styrene content of the blood.

In conclusion, our results show that the type of product and job were important predictors of exposure to styrene and SO in the FRP industry, and that the type of product affected air and blood levels of these contaminants differently. Even within a given job category, we observed large variation in air levels of SO, the putative mutagenic and carcinogenic species associated with exposures in the FRP industry. This variability within and between jobs and product categories, as well as coexposure to styrene and SO, can introduce measurement errors and confounding into investigations of exposure-response relations in the FRP industry. Also, inverse relations between the intensity of exposure to styrene and SO and years on the job suggest that younger workers with little seniority are typically exposed to much higher levels of styrene and SO than their coworkers. Overall, air levels of styrene and SO appear to have decreased substantially in this industry over the last 10–20 years in the US.

Main messages

- Our results suggest that both product category and job title influence exposure levels in this industry.
- Use of job title as the only surrogate for exposure to styrene and styrene-7,8-oxide can introduce unpredictable measurement errors and confounding into investigations of exposure-response relations in the FRP industry.
- Workers with little seniority are typically exposed to much higher levels of styrene and SO than their coworkers.

Policy implications

- As epidemiological studies of health effects in the FRP industry used job titles to quantify exposures, without consideration of product category, and failed to consider coexposure to SO in air, they were subject to measurement errors and potential confounding that could have influenced results.

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